부록 논문 초록집 정리

표-1. PubMed 논문번호 19602472의 내용 요약

구분	내용
PubMed ID	19602472
TITLE	Lipid and endothelium-related genes, ambient particulate matter, and heart rate variability - the VA Normative Aging Study.
JOURNAL	Journal of epidemiology and community health: 10.1136/jech.2008.083295
AUTHORS	Ren C, Baccarelli A, Wilker E, Suh H, Sparrow D, Vokonas P, Wright R, Schwartz J
BACKGROUND	Many studies have shown that exposures to air pollution are associated with cardiovascular events, although the mechanism remains to be clarified. To identify whether exposures to ambient particles act on autonomic function via the lipid/endothelial metabolism pathway, whether effects of particulate matter <2.5 mum in aerodynamic diameter (PM(2.5)) on heart rate variability (HRV) were modified by gene polymorphisms related to those pathways were evaluated.
METHODS	HRV and gene data from the Normative Aging Study and PM(2.5) from a monitor located a kilometre from the examination site were used. A mixed model was fitted to investigate the associations between PM(2.5) and repeated measurements of HRV by gene polymorphisms of apolipoprotein E (APOE), lipoprotein lipase (LPL) and vascular endothelial growth factor (VEGF) adjusting for potential confounders chosen a priori.
RESULTS	A 10 microg/m(3) increase in PM(2.5) in the 2 days before the examination was associated with 3.8% (95% CI 0.2% to 7.4%), 7.8% (95 CI 0.4% to 15.3%) and 10.6% (95% CI 1.8% to 19.4%) decreases of the standard deviation of normal-to-normal intervals, the low frequency and the high frequency, respectively. Overall, carriers of wild-type APOE, LPL and VEGF genes had stronger effects of particles on HRV than those with hetero- or homozygous types. Variations of LPL-N291S, LPL-D9N and APOE-G113C significantly modified effects of PM(2.5) on HRV.
CONCLUSION	Associations between PM(2.5) and HRV were modified by gene polymorphisms of APOE, LPL and VEGF; the biological metabolism remains to be identified.

표-2. PubMed 논문번호 19771392의 내용 요약

구분	내용
PubMed ID	19771392
TITLE	The effect of temperature on hospital admissions in nine California counties.
JOURNAL	International journal of public health: 10.1007/s00038-009-0076-0
AUTHORS	Green Rochelle S, Basu Rupa, Malig Brian, Broadwin Rachel, Kim Janice J, Ostro Bart
OBJECTIVES	This study examined the association between mean daily apparent temperature and hospital admissions for several diseases in nine California counties from May to September, 1999 to 2005.
METHODS	We conducted a time-stratified case-crossover study limited to cases with residential zip codes located within 10 km of a temperature monitor. County-specific estimates were combined, using a random effects meta-analysis. The analyses also considered the effects of ozone and particulate matter (PM(2.5)).
RESULTS	We found that a 10 degrees F increase in mean apparent temperature was associated with a 3.5% [95% confidence interval (CI) 1.5-5.6] increase in ischemic stroke and increases in several other disease-specific outcomes including all respiratory diseases (2.0%, 95% CI 0.7-3.2), pneumonia (3.7%, 95% CI 1.7-3.7), dehydration (10.8%, 95% CI 8.3-13.6), diabetes (3.1%, 95% CI 0.4-5.9), and acute renal failure (7.4%, 95% CI 4.0-10.9). There was little evidence that the temperature effects we found were due to confounding by either PM(2.5) or ozone.
CONCLUSION	Our results indicate that increases in ambient temperature have important public health impacts on morbidity.

표-3. PubMed 논문번호 19773277의 내용 요약

구분	내용
PubMed ID	19773277
TITLE	Long-term exposure to traffic-related air pollution and mortality in Shizuoka, Japan.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2008.045542
AUTHORS	Yorifuji Takashi, Kashima Saori, Tsuda Toshihide, Takao Soshi, Suzuki Etsuji, Doi Hiroyuki, Sugiyama Masumi, Ishikawa-Takata Kazuko, Ohta Toshiki
OBJECTIVES	The number of studies investigating the health effects of long-term exposure to air pollution is increasing, however, most studies have been conducted in Western countries. The health status of Asian populations may be different to that of Western populations and may, therefore, respond differently to air pollution exposure. Therefore, we evaluated the health effects of long-term exposure to traffic-related air pollution in Shizuoka, Japan.
METHODS	Individual data were extracted from participants of an ongoing cohort study. A total of 14,001 older residents, who were randomly chosen from all 74 municipalities of Shizuoka, completed questionnaires and were followed from December 1999 to March 2006. Individual nitrogen dioxide exposure data, as an index for traffic-related exposure, were modelled using a land use regression model. We assigned participants an estimated concentration of nitrogen dioxide exposure during 2000-2006. We then estimated the adjusted HR and their CI for a 10 microg/m(3) increase in exposure to nitrogen dioxide for all-cause or cause-specific mortality.
RESULTS	The adjusted HR for all-cause mortality was 1.02 (95% CI 0.96 to 1.08). Regarding cause-specific mortality, the adjusted HR for cardiopulmonary mortality was 1.16 (95% CI 1.06 to 1.26); in particular the adjusted HR for ischaemic heart disease mortality was 1.27 (95% CI 1.02 to 1.58) and for pulmonary disease mortality it was 1.19 (95% CI 1.02 to 1.38). Furthermore, among non-smokers, a 10 microg/m(3) increase in nitrogen dioxide was associated with a higher risk for lung cancer mortality (HR 1.30, 95% CI 0.85 to 1.93).
CONCLUSION	Long-term exposure to traffic-related air pollution, indexed by nitrogen dioxide concentration, increases the risk of cardiopulmonary mortality, even in a population with a relatively low body mass index and increases the risk of lung cancer mortality in non-smokers.

표-4. PubMed 논문번호 19819853의 내용 요약

구분	내용
PubMed ID	19819853
TITLE	Mortality from myocardial infarction in relation to exposure to vibration and dust among a cohort of iron-ore miners in Sweden.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2009.046599
AUTHORS	Bj□r Bodil, Burstr□m Lage, Eriksson K□re, Jonsson H□kan, Nathanaelsson Lena, Nilsson Tohr
OBJECTIVES	The aim of this study was to investigate myocardial infarction (MI) mortality in relation to exposure to hand/arm vibration (HAV) and whole body vibration (WBV) as well as exposure to dust among men employed in two Swedish iron-ore mines.
METHODS	This study comprised employed men at two iron-ore mines in Sweden who had been employed for at least 1 year from 1923 up to 1996. The causes of death were obtained from the national cause of death register from 1952 to 2001. Myocardial infarction mortality was obtained by linking personal identification numbers to the national cause of death register. Poisson regression was used for risk estimations on exposure-response relation, and analyses were made on the two age groups < or =60 years and >60 years.
RESULTS	Relative risks for MI mortality in relation to exposure were significantly increased for exposure (0/>0) to WBV (RR 1.18, 95% CI 1.06 to 1.31) and dust (RR 1.15, 95% CI 1.02 to 1.31), and the results indicated an exposure-response relation for WBV and dust separately. For 60 years and younger, exposure to HAV (0/>0) (RR 1.34, 95% CI 1.03 to 1.74) and WBV (0/>0) (RR 1.39, 95% CI 1.13 to 1.72) increased the risk of MI mortality. An exposure-response was found for HAV and WBV, as the medium and high exposed categories showed significantly increased risk estimates. None of the exposures significantly increased the risk in the group above 60 years. The increased risk estimates for exposure to WBV remained when adjusting for exposure to dust.
CONCLUSIONS	The results for the working age (< or =60 years) group showed significantly increased MI mortality for univariate exposure to HAV, WBV and dust. We found an association between increased mortality from MI and occupational exposure to WBV, and the risk remained after adjustment for dust exposure.

표-5. PubMed 논문번호 19819863의 내용 요약

구분	내용
PubMed ID	19819863
TITLE	Cause-specific mortality in British coal workers and exposure to respirable dust and quartz.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2009.046151
AUTHORS	Miller Brian G, MacCalman Laura
OBJECTIVES	In the 1950s the Pneumoconiosis Field Research (PFR) programme was set up to study the health of British coal workers. Studies included regular health surveys, an intensive characterisation of workers' individual exposures, and entry to a cohort followed up to the present for cause-specific mortality. This study reports on analyses of cause-specific mortality in a cohort of almost 18 000 men from 10 British collieries.
METHODS	External analyses used standardised mortality ratios (SMRs), comparing observed mortality with reference rates from the regions in which the collieries were situated. Causes investigated include lung and stomach cancers, chronic obstructive pulmonary disease and cardiovascular endpoints. Internal analyses used Cox regression models with time-dependent exposures adjusting for the confounding effects of age, smoking, cohort entry date and regional differences in population mortality rates.
RESULTS	Several causes showed evidence of a healthy worker effect early in the follow-up, with a deficit in the SMR diminishing over time. For most of the causes there was a significant excess in the latter part of follow-up. Internal analyses found evidence of an association between increased risks of lung cancer and increased quartz exposure, particularly at a lag of 15 years. Risks of mortality from non-malignant respiratory disease showed increases with increased exposure to respirable dust.
CONCLUSIONS	This paper adds to the evidence on the long-term effects of exposure to coalmine dust on mortality from respiratory diseases.

표-6. PubMed 논문번호 19833604의 내용 요약

구분	내용
PubMed ID	19833604
TITLE	Exposure to fine particulate matter and acute effects on blood pressure: effect modification by measures of obesity and location.
JOURNAL	Journal of epidemiology and community health: 10.1136/jech.2008.081836
AUTHORS	Kannan S, Dvonch J T, Schulz A J, Israel B A, Mentz G, House J, Max P, Reyes A G
BACKGROUND	Observational studies and controlled experiments have provided evidence that airborne particulate matter (PM) is capable of acutely increasing blood pressure (BP) in certain scenarios. The goal of this study was to evaluate whether and to what extent obesity and community location affect relationships between fine particulate matter (PM(2.5)) and blood pressure (BP) measures.
METHODS	Using data from a stratified random sample survey of adults conducted in 2002-3 in Detroit, Michigan, we tested body mass index (BMI) and waist circumference (WCIR) in separate models as effect modifiers of the relationship between PM(2.5) exposure and BP. We also tested interactions with community location. Models were adjusted for covariates with established pro-hypertensive effects.
RESULTS	PM(2.5) exposure was positively associated with increased pulse pressure (PP) for those categorised as obese (BMI> or =30) across lags 2 (beta 4.16, p<0.05) and 3 days (beta 2.55, p<0.05) prior to BP measure. WCIR similarly modified the effect of exposure to PM(2.5) on PP (beta 4.34, p<0.003). The observed effects were enhanced in the community with closer proximity to local emissions of PM(2.5), and for residents classified as obese (BMI> or =30) or with WCIR above high-risk cuts points.
CONCLUSIONS	This community-based study suggests that positive associations between PM(2.5) exposure and PP and systolic BP are enhanced in areas proximate to sources of PM (2.5) emissions. These patterns were observed for all residents, but were more visible and consistent among those who were obese. Research is needed to examine the mechanistic pathways by which air particles interact with obesity and location to affect BP, and inform community interventions to reduce the population burden of hypertension and related co-morbidities.

표-7. PubMed 논문번호 19884647의 내용 요약

구분	내용
PubMed ID	19884647
TITLE	Air pollution, obesity, genes and cellular adhesion molecules.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2009.046193
AUTHORS	Madrigano Jaime, Baccarelli Andrea, Wright Robert O, Suh Helen, Sparrow David, Vokonas Pantel S, Schwartz Joel
OBJECTIVES	Particulate matter has been associated with acute cardiovascular outcomes, but our understanding of the mechanism is incomplete. We examined the association between particulate matter and cell adhesion molecules. We also investigated the modifying effect of genotype and phenotype variation to gain insight into the relevant biological pathways for this association.
METHODS	We used mixed regression models to examine the association of PM(2.5) (particulate matter < or = 2.5 microm in diameter) and black carbon with serum concentrations of soluble intercellular adhesion molecule (sICAM-1) and soluble vascular cell adhesion molecule (sVCAM-1), markers of endothelial function and inflammation, in a longitudinal study of 809 participants in the Normative Ageing Study (1819 total observations). We also examined whether this association was modified by genotype, obesity or diabetes status. Genes selected for analyses were either related to oxidative stress, endothelial function, lipid metabolism or metal processing.
RESULTS	Black carbon during the 2 days prior to blood draw was significantly associated with increased sVCAM-1 (4.5% increase per 1 microg/m(3), 95% CI 1.1 to 8.0). Neither pollutant was associated with sICAM-1. Larger effects of black carbon on sVCAM were seen in subjects with obesity (p=0.007) and who were GSTM1 null (p=0.02).
CONCLUSIONS	Black carbon is associated with markers of endothelial function and inflammation. Genes related to oxidative defence may modify this association.

표-8. PubMed 논문번호 19945183의 내용 요약

구분	내용
PubMed ID	19945183
TITLE	Passive smoking and aortic arch calcification in older Chinese never smokers: the Guangzhou Biobank Cohort Study.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2009.11.001
AUTHORS	Xu Lin, Jiang Chao Qiang, Lam Tai Hing, Thomas G Neil, Zhang Wei Sen, Cheng Kar Keung
OBJECTIVE	To study whether passive smoking is a risk factor for aortic arch calcification (AAC) among never smokers.
BACKGROUND	We have previously reported that active smoking increases the risk of AAC, but the effect of passive smoking has not been reported.
METHODS	We used baseline data of the Phase 1 Guangzhou Biobank Cohort Study (GBCS). 7702 older Chinese never smokers from the Phase 1 GBCS were included. Information on passive smoking and potential confounders were collected by standardized interviews and laboratory assays. AAC was diagnosed from chest X-ray by two experienced radiologists. Unconditional logistic regression was used to estimate odds ratios of AAC for passive smoking with adjustment for potential confounders.
RESULTS	In women, the risk for aortic arch calcification (AAC) increased significantly with increasing duration of adulthood passive smoking exposure at home, at work and total duration of adulthood home and work exposure [adjusted odds ratio 1.24 (95% confidence interval 1.09-1.41) for high level of total exposure] (P for trend from 0.012 to 0.001). For passive smoking at home, at work and total exposure, significant trends of increasing severity of AAC with increasing duration of exposure were observed in men and women combined (P for trend from 0.05 to 0.002).
CONCLUSION	Passive smoking is a risk factor for aortic arch calcification. Studies of passive smoking and AAC, especially in developing countries can generate important local evidence to raise awareness and to support public health measures to protect non-smokers from second-hand smoke.

표-9. PubMed 논문번호 20047363의 내용 요약

구분	내용
PubMed ID	20047363
TITLE	Exposure to nitrogen dioxide is not associated with vascular dysfunction in man.
JOURNAL	Inhalation toxicology: 10.3109/08958370903144105
AUTHORS	Langrish Jeremy P, Lundb□ck Magnus, Barath Stefan, S□derberg Stefan, Mills Nicholas L, Newby David E, Sandstr□m Thomas, Blomberg Anders
BACKGROUND	Exposure to air pollution is associated with increased cardiorespiratory morbidity and mortality. It is unclear whether these effects are mediated through combustion-derived particulate matter or gaseous components, such as nitrogen dioxide.
OBJECTIVES	To investigate the effect of nitrogen dioxide exposure on vascular vasomotor and six fibrinolytic functions.
METHODS	Ten healthy male volunteers were exposed to nitrogen dioxide at 4 ppm or filtered air for 1 h during intermittent exercise in a randomized double-blind crossover study. Bilateral forearm blood flow and fibrinolytic markers were measured before and during unilateral intrabrachial infusion of bradykinin (100-1000 pmol/min), acetylcholine (5-20 microg/min), sodium nitroprusside (2-8 microg/min), and verapamil (10-100 microg/min) 4 h after the exposure. Lung function was determined before and after the exposure, and exhaled nitric oxide at baseline and 1 and 4 h after the exposure.
RESULTS	There were no differences in resting forearm blood flow after either exposure. There was a dose-dependent increase in forearm blood flow with all vasodilators but this was similar after either exposure for all vasodilators ($p > .05$ for all). Bradykinin caused a dose-dependent increase in plasma tissue-plasminogen activator, but again there was no difference between the exposures. There were no changes in lung function or exhaled nitric oxide following either exposure.
CONCLUSION	Inhalation of nitrogen dioxide does not impair vascular vasomotor or fibrinolytic function. Nitrogen dioxide does not appear to be a major arbiter of the adverse cardiovascular effects of air pollution.

표-10. PubMed 논문번호 20056584의 내용 요약

구분	내용
PubMed ID	20056584
TITLE	Baseline repeated measures from controlled human exposure studies: associations between ambient air pollution exposure and the systemic inflammatory biomarkers IL-6 and fibrinogen.
JOURNAL	Environmental health perspectives: 10.1289/ehp.0900550
AUTHORS	Thompson Aaron M S, Zanobetti Antonella, Silverman Frances, Schwartz Joel, Coull Brent, Urch Bruce, Speck Mary, Brook Jeffrey R, Manno Michael, Gold Diane R
INTRODUCTION	Systemic inflammation may be one of the mechanisms mediating the association between ambient air pollution and cardiovascular morbidity and mortality. Interleukin-6 (IL-6) and fibrinogen are biomarkers of systemic inflammation that are independent risk factors for cardio-vascular disease.
OBJECTIVE	We investigated the association between ambient air pollution and systemic inflammation using baseline measurements of IL-6 and fibrinogen from controlled human exposure studies.
METHODS	In this retrospective analysis we used repeated-measures data in 45 nonsmoking subjects. Hourly and daily moving averages were calculated for ozone, nitrogen dioxide, sulfur dioxide, and particulate matter <or= 2.5 microm in aerodynamic diameter (PM2.5). Linear mixed-model regression determined the effects of the pollutants on systemic IL-6 and fibrinogen. Effect modification by season was considered.
RESULTS	We observed a positive association between IL-6 and O3 [0.31 SD per O3 interquartile range (IQR); 95% confidence interval (CI), 0.080.54] and between IL-6 and SO2 (0.25 SD per SO2 IQR; 95% CI, 0.060.43). We observed the strongest effects using 4-day moving averages. Responses to pollutants varied by season and tended to be higher in the summer, particularly for O3 and PM2.5. Fibrinogen was not associated with pollution.
CONCLUSIONS	This study demonstrates a significant association between ambient pollutant levels and baseline levels of systemic IL-6. These findings have potential implications for controlled human exposure studies. Future research should consider whether ambient pollution exposure before chamber exposure modifies IL-6 response.

표-11. PubMed 논문번호 20060145의 내용 요약

구분	내용
PubMed ID	20060145
TITLE	Short-term effect of concentrations of fine particulate matter on hospital admissions due to cardiovascular and respiratory causes among the over-75 age group in Madrid, Spain.
JOURNAL	Public health: 10.1016/j.puhe.2009.11.007
AUTHORS	Linares C, D□az J
OBJECTIVES	This study sought to analyse the effect of daily mean concentrations of fine particulate matter (diameter 〈2.5 microm; PM(2.5)) on hospital admissions due to circulatory and respiratory causes among an elderly population (〉75 years) in Madrid between 2003 and 2005.
STUDY DESIGN	Ecological longitudinal time-series study.
METHODS	The dependent variable used was the daily number of emergency hospital admissions registered at the Gregorio Mara□□n University Teaching Hospital. The following causes were analysed: all causes [International Classification of Diseases 9th Version (ICD-9:1-799)], respiratory causes (ICD-9: 460-519) and circulatory causes (ICD-9: 390-459). Analysis focused on subjects over 75 years of age. Daily records of mean concentrations of PM(2.5), PM(10), NO(2), NO(x), SO(2) and O(3) in Madrid were used as independent variables. The control variables were seasonalities, trend, influenza epidemics, noise and pollen concentrations. Poisson regression models were constructed to calculate the relative risk (RR) and attributable risk (AR). Analyses were performed for the entire year and for the winter and summer.
RESULTS	PM(2.5) was the single primary pollutant that proved statistically significant in all models. The functional relationship with hospital admissions was linear and had no threshold. Taking the year as a whole, the RRs among people over 75 years of age for an increase of 10 microg/m(3) in PM(2.5) concentrations were: 1.038 [95% confidence interval (CI) 1.022-1.053] for all causes at lag 0; 1.062 (95% CI 1.036-1.089) for circulatory causes at lag 0; and 1.049 (95% CI 1.019-1.078) for respiratory causes at lag 3. The ARs were 3.6%, 5.9% and 4.6%, respectively. These risks increased in winter and no statistically significant associations were observed in summer. PM(2.5) was the only primary pollutant that showed a statistically significant association with hospital admissions among people over 75 years of age in Madrid across the study period.
CONCLUSION	Measures should be implemented to reduce PM(2.5) concentrations in Madrid.

표-12. PubMed 논문번호 20064787의 내용 요약

구분	내용
PubMed ID	20064787
TITLE	Long-term exposure to constituents of fine particulate air pollution and mortality: results from the California Teachers Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.0901181
AUTHORS	Ostro Bart, Lipsett Michael, Reynolds Peggy, Goldberg Debbie, Hertz Andrew, Garcia Cynthia, Henderson Katherine D, Bernstein Leslie
BACKGROUND	Several studies have reported associations between long-term exposure to ambient fine particulate matter (PM) and cardiovascular mortality. However, the health impacts of long-term exposure to specific constituents of PM(2.5) (PM with aerodynamic diameter < or = 2.5 microm) have not been explored.
METHODS	We used data from the California Teachers Study, a prospective cohort of active and former female public school professionals. We developed estimates of long-term exposures to PM(2.5) and several of its constituents, including elemental carbon, organic carbon (OC), sulfates, nitrates, iron, potassium, silicon, and zinc. Monthly averages of exposure were created using pollution data from June 2002 through July 2007. We included participants whose residential addresses were within 8 and 30 km of a monitor collecting PM(2.5) constituent data. Hazard ratios (HRs) were estimated for long-term exposure for mortality from all nontraumatic causes, cardiopulmonary disease, ischemic heart disease (IHD), and pulmonary disease.
RESULTS	Approximately 45,000 women with 2,600 deaths lived within 30 km of a monitor. We observed associations of all-cause, cardiopulmonary, and IHD mortality with PM(2.5) mass and each of its measured constituents, and between pulmonary mortality and several constituents. For example, for cardiopulmonary mortality, HRs for interquartile ranges of PM(2.5), OC, and sulfates were 1.55 [95% confidence interval (CI), 1.431.69], 1.80 (95% CI, 1.681.93), and 1.79 (95% CI, 1.582.03), respectively. Subsequent analyses indicated that, of the constituents analyzed, OC and sulfates had the strongest associations with all four outcomes.
CONCLUSIONS	Long-term exposures to PM(2.5) and several of its constituents were associated with increased risks of all-cause and cardiopulmonary mortality in this cohort. Constituents derived from combustion of fossil fuel (including diesel), as well as those of crustal origin, were associated with some of the greatest risks. These results provide additional evidence that reduction of ambient PM(2.5) may provide significant public health benefits.

표-13. PubMed 논문번호 20081539의 내용 요약

구분	내용
PubMed ID	20081539
TITLE	Second-hand smoke, cotinine levels, and risk of circulatory mortality in a large cohort study of never-smokers.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e3181c9fdad
AUTHORS	Gallo Valentina, Neasham David, Airoldi Luisa, Ferrari Pietro, Jenab Mazda, Boffetta Paolo, Overvad Kim, Tjønneland Anne, Clavel-Chapelon Francoise, Boeing Heiner, Pala Valeria, Palli Domenico, Panico Salvatore, Tumino Rosario, Arriola Larraitz, Lund Eiliv, Bueno-De-Mesquita Bas, Peeters Petra H, Melander Olle, Hallmans Goran, Riboli Elio, Saracci Rodolfo, Vineis Paolo
BACKGROUND	Exposure to second-hand smoke has been shown to be associated with increased cardiovascular mortality in several, but not all, epidemiologic studies. Our aim was to investigate the risk of circulatory death associated with exposure to second-hand smoke in never-smokers in a very large prospective study, the European Prospective Investigation into Cancer and Nutrition. A secondary aim was to use cotinine levels for cross-validating self-reported second-hand smoke exposure.
METHODS	Cox proportional hazard models were used to investigate the risk of death due to circulatory causes associated with second-hand smoke exposure in 135,233 never-smokers. Exposure to second-hand smoke was assessed through a questionnaire at enrollment and then validated against plasma cotinine measurements in a subsample.
RESULTS	Study participants who reported second-hand smoke exposure at home had higher cotinine levels (median plasma cotinine concentration in exposed = 0.82 microg/L; in those unexposed 0.02 microg/L). Second-hand smoke exposure at home was associated with an increased risk of dying from cardiovascular diseases (hazard ratio [HR] = 1.38 [95% confidence interval = 1.01-1.90]), all circulatory diseases (1.28 [0.98-1.69]), and coronary heart disease (1.31 [0.83-2.08]) after adjustment for age, sex, education, physical activity, and body mass index. Dose-response relationships were observed between exposure to second-hand smoke at home and risk of circulatory death (HR per each additional hour/d = 1.25 [1.04-1.50]). Having a partner who smokes more than 30 cigarettes per day considerably increased the risk of a circulatory death (2.94 [1.11-7.78]). Second-hand smoke exposure at home was not associated with total mortality (1.03 [0.93-1.13]).
DISCUSSION	Exposure to second-hand smoke at home (as confirmed by plasma cotinine levels) increases the risk of cardiovascular mortality.

표-14. PubMed 논문번호 20105060의 내용 요약

구분	내용
PubMed ID	20105060
TITLE	Ambient air pollution alters heart rate regulation in aged mice.
JOURNAL	Inhalation toxicology: 10.3109/08958370903349365
AUTHORS	Ramos-Bonilla Juan P, Breysse Patrick N, Dominici Francesca, Geyh Alison, Tankersley Clarke G
CONTEXT	Heart rate alterations associated with exposure to particulate matter (PM) and gaseous pollutants have been observed in epidemiological studies and animal experiments. Nevertheless, the time-lag of these associations is still unclear.
OBJECTIVE	Determine the association at different time-lags between the complex mixture of ambient concentrations of PM, carbon monoxide (CO), and nitrogen dioxide (NO(2)), and markers of cardiac function in a model of aged mice.
MATERIALS AND METHODS	AKR/J inbred mice were exposed to ambient air, 6 h daily for 40 weekdays. During this period, the animals' electrocardiogram (ECG), deep body temperature (Tdb), and body weight (BW) were registered, and concentrations of PM, CO, NO(2), as well as air temperature and relative humidity (RH) were measured. Data analysis included random effects models with lagged covariate methods.
RESULTS	CO was significantly associated with declines in heart rate (HR) and heart rate variability (HRV), PM was significantly associated with declines in HRV and BW, and NO(2) was significantly associated with declines in HR. Some significant associations occurred in the same day (PM and HRV, PM and BW, CO and HR), whereas others were delayed by 1 to 3 days (CO and HR, CO and HRV, NO(2) and HR, PM and HRV).
DISCUSSION AND CONCLUSION	Finding significant declines in heart function in aged mice associated with the combined effects of air pollutants at ambient concentrations and at different time-lags is of great importance to public health. These results further implicate the potential short term and delayed effects of air pollution on HR alterations.

표-15. PubMed 논문번호 20110671의 내용 요약

구분	내용
PubMed ID	20110671
TITLE	Passive smoke exposure and circulating carotenoids in the CARDIA study.
JOURNAL	Annals of nutrition & metabolism: 10.1159/000277662
AUTHORS	Widome Rachel, Jacobs David R, Hozawa Atsushi, Sijtsma Femke, Gross Myron, Schreiner Pamela J, Iribarren Carlos
BACKGROUND/AIMS	Our objective was to assess associations between passive smoke exposure in various venues and serum carotenoid concentrations.
METHODS	CARDIA is an ongoing longitudinal study of the risk factors for subclinical and clinical cardiovascular disease. At baseline in 1985/1986, serum carotenoids were assayed and passive smoke exposure inside and outside of the home and diet were assessed by self-report. Our analytic sample consisted of 2,633 black and white non-smoking adults aged 18-30 years.
RESULTS	Greater total passive smoke exposure was associated with lower levels of the sum of the three provitamin A carotenoids, alpha-carotene, beta-carotene, and beta-cryptoxanthin (-0.048 nmol/l per hour of passive smoke exposure, p = 0.001), unassociated with lutein/zeaxanthin, and associated with higher levels of lycopene (0.027 nmol/l per hour of passive smoke exposure, p = 0.010) after adjustment for demographics, diet, lipid profile, and supplement use. Exposure in both home and non-home spaces was also associated with lower levels of the provitamin A carotenoid index.
CONCLUSION	Cross-sectionally, in 1985/86, passive smoke exposure in various venues was associated with reduced levels of provitamin A serum carotenoids.

표-16. PubMed 논문번호 20110814의 내용 요약

구분	내용
PubMed ID	20110814
TITLE	Air pollution and homocysteine: more evidence that oxidative stress-related genes modify effects of particulate air pollution.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e3181cc8bfc
AUTHORS	Ren Cizao, Park Sung Kyun, Vokonas Pantel S, Sparrow David, Wilker Elissa, Baccarelli Andrea, Suh Helen H, Tucker Katherine L, Wright Robert O, Schwartz Joel
BACKGROUND	Ambient particles are associated with cardiovascular events and recently with total plasma homocysteine. High total plasma homocysteine is a risk for human health. However, the biologic mechanisms are not fully understood. One of the putative pathways is through oxidative stress. We aimed to examine whether associations of PM2.5 and black carbon with homocysteine were modified by genotypes including HFE H63D, C282Y, CAT (rs480575, rs1001179, rs2284367, and rs2300181), NQO1 (rs1800566), GSTP1 I105V, GSTM1, GSTT1 (deletion vs. nondeletion), and HMOX-1 (any short vs. both long). We attempted to replicate identified genes in an analysis of heart rate variability and in other outcomes reported in the literature.
METHODS	Study subjects were 1000 white non-Hispanic men in the Boston area, participating in a cohort study of aging. PM2.5, black carbon, total plasma homocysteine, and other covariates were measured at several points in time between 1995 and 2006. We fit mixed models to examine effect modification of genes on associations of pollution with total plasma homocysteine.
RESULTS	Interquartile range increases in PM2.5 and black carbon (7-day moving averages) were associated with 1.5% (95% confidence interval = 0.2% to 2.8%) and 2.2% (0.6% to 3.9%) increases in total plasma homocysteine, respectively. GSTT1 and HFE C282Y modified effects of black carbon on total plasma homocysteine, and HFE C282Y and CAT (rs2300181) modified effects of PM2.5 on homocysteine. Several genotypes marginally modified effects of PM2.5 and black carbon on various endpoints. All genes with significant interactions with particulate air pollution had modest main effects on total plasma homocysteine.
CONCLUSIONS	: Effects of PM2.5 and black carbon on various endpoints appeared to be mediated by genes related to oxidative stress pathways.

표-17. PubMed 논문번호 20123637의 내용 요약

구분	내용
PubMed ID	20123637
TITLE	Association of biomarkers of systemic inflammation with organic components and source tracers in quasi-ultrafine particles.
JOURNAL	Environmental health perspectives: 10.1289/ehp.0901407
AUTHORS	Delfino Ralph J, Staimer Norbert, Tjoa Thomas, Arhami Mohammad, Polidori Andrea, Gillen Daniel L, Kleinman Michael T, Schauer James J, Sioutas Constantinos
BACKGROUND	Evidence is needed regarding the air pollutant components and their sources responsible for associations between particle mass concentrations and human cardiovascular outcomes. We previously found associations between circulating biomarkers of inflammation and mass concentrations of quasi-ultrafine particles <or= 0.25 microm in aerodynamic diameter (PM0.25) in a panel cohort study of 60 elderly subjects with coronary artery disease living in the Los Angeles Basin.
OBJECTIVES	We reassessed biomarker associations with PM0.25 using new particle composition data.
METHODS	Weekly biomarkers of inflammation were plasma interleukin-6 (IL-6) and soluble tumor necrosis factor-alpha receptor II (sTNF-RII) (n = 578). Exposures included indoor and outdoor community organic PM0.25 constituents [polycyclic aromatic hydrocarbons (PAHs), hopanes, n-alkanes, organic acids, water-soluble organic carbon, and transition metals]. We analyzed the relation between biomarkers and exposures with mixed-effects models adjusted for potential confounders.
RESULTS	Indoor and outdoor PAHs (low-, medium-, and high-molecular-weight PAHs), followed by hopanes (vehicle emissions tracer), were positively associated with biomarkers, but other organic components and transition metals were not. sTNF-RII increased by 135 pg/mL [95% confidence interval (CI), 45-225 pg/mL], and IL-6 increased by 0.27 pg/mL (95% CI, 0.10-0.44 pg/mL) per interquartile range increase of 0.56 ng/m3 outdoor total PAHs. Two-pollutant models of PM0.25 with PAHs showed that nominal associations of IL-6 and sTNF-RII with PM0.25 mass were completely confounded by PAHs. Vehicular emission sources estimated from chemical mass balance models were strongly correlated with PAHs (R = 0.71).
CONCLUSIONS	Traffic emission sources of organic chemicals represented by PAHs are associated with increased systemic inflammation and explain associations with quasi-ultrafine particle mass.

표-18. PubMed 논문번호 20194081의 내용 요약

구분	내용
PubMed ID	20194081
TITLE	Traffic-related air pollution and QT interval: modification by diabetes, obesity, and oxidative stress gene polymorphisms in the normative aging study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.0901396
AUTHORS	Baja Emmanuel S, Schwartz Joel D, Wellenius Gregory A, Coull Brent A, Zanobetti Antonella, Vokonas Pantel S, Suh Helen H
BACKGROUND	Acute exposure to ambient air pollution has been associated with acute changes in cardiac outcomes, often within hours of exposure.
OBJECTIVES	We examined the effects of air pollutants on heart-rate-corrected QT interval (QTc), an electrocardiographic marker of ventricular repolarization, and whether these associations were modified by participant characteristics and genetic polymorphisms related to oxidative stress.
METHODS	We studied repeated measurements of QTc on 580 men from the Veterans Affairs Normative Aging Study (NAS) using mixed-effects models with random intercepts. We fitted a quadratic constrained distributed lag model to estimate the cumulative effect on QTc of ambient air pollutants including fine particulate matter <or= 2.5 microm in aerodynamic diameter (PM2.5), ozone (O3), black carbon (BC), nitrogen dioxide (NO2), carbon monoxide (CO), and sulfur dioxide (SO2) concentrations during the 10 hr before the visit. We genotyped polymorphisms related to oxidative stress and analyzed pollution-susceptibility score interactions using the genetic susceptibility score (GSS) method.
RESULTS	Ambient traffic pollutant concentrations were related to longer QTc. An interquartile range (IQR) change in BC cumulative during the 10 hr before the visit was associated with increased QTc [1.89 msec change; 95% confidence interval (CI), -0.16 to 3.93]. We found a similar association with QTc for an IQR change in 1-hr BC that occurred 4 hr before the visit (2.54 msec change; 95% CI, 0.28-4.80). We found increased QTc for IQR changes in NO2 and CO, but the change was statistically insignificant. In contrast, we found no association between QTc and PM2.5, SO2, and O3. The association between QTc and BC was stronger among participants who were obese, who had diabetes, who were nonsmokers, or who had higher GSSs.
CONCLUSIONS	Traffic-related pollutants may increase QTc among persons with diabetes, persons who are obese, and nonsmoking elderly individuals; the number of genetic variants related to oxidative stress increases this effect.

표-19. PubMed 논문번호 20197510의 내용 요약

구분	내용
PubMed ID	20197510
TITLE	Arterial intima-media thickness, endothelial function, and apolipoproteins in adolescents frequently exposed to tobacco smoke.
JOURNAL	Circulation. Cardiovascular quality and outcomes: 10.1161/CIRCOUTCOMES.109.857771
AUTHORS	Kallio Katariina, Jokinen Eero, Saarinen Maiju, H□m□l□inen Mauri, Volanen Iina, Kaitosaari Tuuli, R□nnemaa Tapani, Viikari Jorma, Raitakari Olli T, Simell Olli
BACKGROUND	Exposure to tobacco smoke is associated with markers of preclinical atherosclerosis in adults, but its effect on arterial structure in adolescents is unknown.
METHODS AND RESULTS	Healthy 13-year-old adolescents from the atherosclerosis prevention trial STRIP were studied. Maximum carotid and aortic intima-media thickness and brachial artery flow-mediated dilation were measured in 494 adolescents using high-resolution ultrasound. Serum lipid, lipoprotein, and apolipoprotein (Apo) A-I and B concentrations were determined using standard methods. Exposure to tobacco smoke was measured annually between ages 8 and 13 years using serum cotinine concentrations, analyzed with gas chromatography. To define longitudinal exposure, cotinine values of children having serum cotinine measured 2 to 6 times during follow-up were averaged and divided into tertiles (exposure groups): low (n=160), intermediate (n=171), and high (n=163). Adolescents with higher longitudinal exposure to tobacco smoke had increased carotid intima-media thickness (exposure groups [mean+/-SD]: low, 0.502+/-0.079 mm; intermediate, 0.525+/-0.070 mm; high, 0.535+/-0.066 mm; P<0.001) and increased aortic intima-media thickness (exposure groups: low, 0.527+/-0.113 mm; intermediate, 0.563+/-0.139 mm; high, 0.567+/-0.126 mm; P=0.008). The flow-mediated dilation decreased when cotinine level increased (exposure groups: low, 10.43+/-4.34%; intermediate, 9.78+/-4.38%; high, 8.82+/-4.14%; P=0.004). Moreover, ApoB (P=0.014) and ApoB/ApoA-I ratio (P=0.045) increased with increase in cotinine level. The associations between tobacco smoke exposure and ultrasound variables were unchanged after adjusting for traditional atherosclerosis risk factors and for ApoB.
CONCLUSIONS	Frequent exposure to tobacco smoke is independently associated with arterial changes of preclinical atherosclerosis and increased ApoB levels among healthy adolescents. Clinical Trial Registration- clinicaltrials.gov. Identifier: NCT00223600.

표-20. PubMed 논문번호 20339125의 내용 요약

구분	내용
PubMed ID	20339125
TITLE	Impact of outdoor air pollution on survival after stroke: population-based cohort study.
JOURNAL	Stroke: 10.1161/STROKEAHA.109.567743
AUTHORS	Maheswaran Ravi, Pearson Tim, Smeeton Nigel C, Beevers Sean D, Campbell Michael J, Wolfe Charles D
BACKGROUND AND PURPOSE	The impact of air pollution on survival after stroke is unknown. We examined the impact of outdoor air pollution on stroke survival by studying a population-based cohort.
METHODS	All patients who experienced their first-ever stroke between 1995 and 2005 in a geographically defined part of London, where road traffic contributes to spatial variation in air pollution, were followed up to mid-2006. Outdoor concentrations of nitrogen dioxide and particulate matter <10 microm in diameter modeled at a 20-m grid point resolution for 2002 were linked to residential postal codes. Hazard ratios were adjusted for age, sex, social class, ethnicity, smoking, alcohol consumption, prestroke functional ability, pre-existing medical conditions, stroke subtype and severity, hospital admission, and neighborhood socioeconomic deprivation.
RESULTS	There were 1856 deaths among 3320 patients. Median survival was 3.7 years (interquartile range, 0.1 to 10.8). Mean exposure levels were 41 microg/m(3) (SD, 3.3; range, 32.2 to 103.2) for nitrogen dioxide and 25 microg/m(3) (SD, 1.3; range, 22.7 to 52) for particulate matter <10 microm in diameter. A 10-microg/m(3) increase in nitrogen dioxide was associated with a 28% (95% CI, 11% to 48%) increase in risk of death. A 10-microg/m(3) increase in particulate matter <10 mum in diameter was associated with a 52% (6% to 118%) increase in risk of death. Reduced survival was apparent throughout the follow-up period, ruling out short-term mortality displacement.
CONCLUSIONS	Survival after stroke was lower among patients living in areas with higher levels of outdoor air pollution. If causal, a 10-microg/m(3) reduction in nitrogen dioxide exposure might be associated with a reduction in mortality comparable to that for stroke units. Improvements in outdoor air quality might contribute to better survival after stroke.

표-21. PubMed 논문번호 20407379의 내용 요약

구분	내용
PubMed ID	20407379
TITLE	Long-term exposure to air pollution and vascular damage in young adults.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e3181dec3a7
AUTHORS	Lenters Virissa, Uiterwaal Cuno S, Beelen Rob, Bots Michiel L, Fischer Paul, Brunekreef Bert, Hoek Gerard
BACKGROUND	Long-term exposure to ambient air pollution has recently been linked to atherosclerosis and cardiovascular events. There are, however, very limited data in healthy young people. We examined the association between air pollutants and indicators of vascular damage in a cohort of young adults.
METHODS	We used data from the Atherosclerosis Risk in Young Adults study. We estimated exposure to nitrogen dioxide (NO2), particulate matter less than 2.5 microm in aerodynamic diameter (PM2.5), black smoke, sulfur dioxide (SO2), and various traffic indicators for participants' 2000 home addresses. Exposure for the year 2000 was estimated by land-use regression models incorporating regional background annual air pollution levels, land-use variables, population densities, and traffic intensities on nearby roads. Outcomes were common carotid artery intima-media thickness (n = 745), aortic pulse wave velocity (n = 524), and augmentation index (n = 729).
RESULTS	Exposure contrasts were substantial for NO2, SO2, and black smoke (5th-95th percentiles = 19.7 to 44.9, 2.5 to 5.2, and 8.6 to 19.4 microg/m3, respectively) and smaller for PM2.5 (16.5 to 19.9 microg/m3). The variability of carotid artery intima-media thickness was less than for pulse wave velocity and especially augmentation index (5-95th percentiles = 0.42 to 0.58 mm, 4.9 to 7.4 m/s and -12.3% to 27.3%, respectively). No associations were found between any of the pollutants or traffic indicators and carotid artery intima-media thickness, although PM2.5 effect estimates were in line with previous studies. We observed a 4.1% (95% confidence interval = 0.1% to 8.0%) increase in pulse wave velocity and a 37.6% (2.2% to 72.9%) increase in augmentation index associated with a 25 microg/m3 increase in NO2, and a 5.3% (0.1% to 10.4%) increase in pulse wave velocity with a 5 microg/m3 increase in SO2. PM2.5 and black smoke were not associated with either of these 2 outcomes.
CONCLUSIONS	Air pollution may accelerate arterial-wall stiffening in young adults. Small outcome variability and lack of residential mobility data may have limited the power to detect an effect on intima-media thickness.

표-22. PubMed 논문번호 20408256의 내용 요약

구분	내용
PubMed ID	20408256
TITLE	Environmental determinants of blood pressure, arterial stiffness, and central hemodynamics.
JOURNAL	Journal of hypertension: 10.1097/hjh.0b013e3283369f67
AUTHORS	Adamopoulos Dionysios, Vyssoulis Gregory, Karpanou Evangelia, Kyvelou Stella-Maria, Argacha Jean-Fran□ois, Cokkinos Denis, Stefanadis Christodoulos, van de Borne Philippe
BACKGROUND	Arterial stiffness and wave reflection alterations may be implicated in the association between cardiovascular mortality, meteorological variables and ambient particulate matter air pollution. The present study explored the cross-sectional relations between ambient environmental parameters, arterial stiffness, peripheral and central hemodynamics in a large-scale cohort of hypertensive patients and normotensive controls.
METHODS	The study comprised 1222 consecutive individuals who sought medical consultation in the hypertension outpatient clinics of the Hippokration' and 'Onassis' Hospitals (Athens, Greece) during a 3-year period (2004-2006). All individuals underwent a complete clinical and lipid-profile assessment at drug-free baseline. Carotid radial, carotid-femoral pulse wave velocity measurements and aortic pressure waveform analysis were performed noninvasively to all participants. Data from the National Technical University of Athens and the air quality department of the Hellenic Ministry for the Environment were used to estimate daily meteorological conditions and PM10 exposure.
RESULTS	In the total population, multiple-linear regression analysis revealed no significant associations between environmental variables and arterial stiffness. However, in men, the mean 5 day PM10 air concentration was independently associated with the augmentation pressure [2.0 mmHg (95% confidence interval (CI) 0.56-3.39) per 43.4 microg/m3] and the aortic-pulse pressure [2.78 mmHg (95% CI 3.91-5.12)] denoting a significant effect of particulate matter on the aortic-wave reflection magnitude and central hemodynamics. In addition, globally, the mean-daily temperature was related to the aortic-pulse pressure [-2.38 mmHg (95% CI -4.51 to -0.26) per 23.6 degrees C change] and the subendocardial viability ratio [5.85% (95% CI 1.1-10.6 per 23.6 degrees C)].
CONCLUSION	The exposure to lower environmental temperatures is related to impaired hemodynamics not only to the periphery but also to the aorta. In men, PM10 air-pollution levels are associated with heightened amplitude of the reflection wave leading to significant alterations in central-pulse pressure.

표-23. PubMed 논문번호 20478865의 내용 요약

구분	내용
PubMed ID	20478865
TITLE	Cardiorespiratory and immune response to physical activity following exposure to a typical smoking environment.
JOURNAL	Heart (British Cardiac Society): 10.1136/hrt.2009.190744
AUTHORS	Flouris Andreas D, Metsios Giorgos S, Jamurtas Athanasios Z, Koutedakis Yiannis
OBJECTIVE	Millions of non-smokers suffer daily passive smoking (PS) at home or at work, many of whom then have to walk fast for several minutes or climb a few sets of stairs. We conducted a randomised single-blind crossover experiment to assess the cardiorespiratory and immune response to physical activity following PS.
DESIGN	Data were obtained from 17 (eight women) non-smoking adults during and following 30 minutes of moderate cycling administered at baseline and at 0 hour, 1 hour and 3 hours following a 1-hour PS exposure set at bar/restaurant PS levels.
RESULTS	We found that PS was associated with a 36% and 38.7% decrease in mean power output in men and women, respectively, and that this effect persisted up to 3 hours ($p<0.05$). Moreover, at 0 hour almost all cardiorespiratory and immune variables measured were markedly reduced ($p<0.05$). For instance, FEV(1) values at 0 hour dropped by 10.2% in men and 10.8% in women, while IL-5 increased by 59.2% in men and 44% in women, respectively ($p<0.05$). At 3-hour mean values of respiratory quotient, mean power, perceived exertion, cotinine, FEV(1), IL-5, IL-6 and INFgamma in both sexes, recovery diastolic and mean arterial pressure, IL-4 and TNFalpha in men, as well as percentage predicted FEV(1) in women remained different compared to baseline ($p<0.05$). Also, some of the PS effects were exacerbated in less fit individuals.
CONCLUSION	It is concluded that 1 hour of PS at bar/restaurant levels adversely affects the response to moderate physical activity in healthy non-smokers for at least 3 hours following PS.

표-24. PubMed 논문번호 20508218의 내용 요약

구분	내용
PubMed ID	20508218
TITLE	Associations of PM10 with sleep and sleep-disordered breathing in adults from seven U.S. urban areas.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.200912-1797OC
AUTHORS	Zanobetti Antonella, Redline Susan, Schwartz Joel, Rosen Dennis, Patel Sanjay, O'Connor George T, Lebowitz Michael, Coull Brent A, Gold Diane
RATIONALE	Sleep-disordered breathing (SDB), the recurrent episodic disruption of norm breathing during sleep, affects as much as 17% of U.S. adults, and may be more prevalent in poor urban environments. SDB and air pollution have bee linked to increased cardiovascular diseases and mortality, but the association between pollution and SDB is poorly understood.
OBJECTIVES	We used data from the Sleep Heart Health Study (SHHS), a U.S. multicenter cohort study assessing cardiovascular and other consequences of SDB, to examine whether particulate air matter less than 10 μm in aerodynamic diameter (PM(10)) was associated with SDB among persons 39 years of age and older.
METHODS	Using baseline data from SHHS urban sites, outcomes included the following the respiratory disturbance index (RDI); percentage of sleep time at less than 90% O(2) saturation; and sleep efficiency, measured by overnight in-home polysomnography. We applied a fixed-effect model containing a city effect, controlling for potential predictors. In all models we included both the 365-day moving averages of PM(10) and temperature (long-term effects) an the differences between the daily measures of these two predictors and their 365-day average (short-term effects).
MEASUREMENTS AND MAIN RESULTS	In summer, increases in RDI or percentage of sleep time at less than 90% O(saturation, and decreases in sleep efficiency, were all associated with increase in short-term variation in PM(10). Over all seasons, we found that increased RDI was associated with an 11.5% (95% confidence interval: 1.96, 22.01) increase per interquartile range increase (25.5° F) in temperature.
CONCLUSIONS	Reduction in air pollution exposure may decrease the severity of SDB and nocturnal hypoxemia and may improve cardiac risk.

표-25. PubMed 논문번호 20519749의 내용 요약

구분	내용
PubMed ID	20519749
TITLE	Systemic inflammation, heart rate variability and air pollution in a cohort of senior adults.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2009.050625
AUTHORS	Luttmann-Gibson Heike, Suh Helen H, Coull Brent A, Dockery Douglas W, Sarnat Stefanie Ebelt, Schwartz Joel, Stone Peter H, Gold Diane R
OBJECTIVES	Short-term elevation of ambient particulate air pollution has been associated with autonomic dysfunction and increased systemic inflammation, but the interconnections between these pathways are not well understood. We examined the association between inflammation and autonomic dysfunction and effect modification of inflammation on the association between air pollution and heart rate variability (HRV) in elderly subjects.
METHODS	25 elderly subjects in Steubenville, Ohio, were followed up to 24 times with repeated 30-min ECG Holter monitoring (545 observations). C-reactive protein (CRP), fibrinogen, interleukin-6 (IL-6), soluble inter-cellular adhesion molecule 1 (sICAM-1), and white blood cell and platelet counts were measured in peripheral blood samples collected in the first month of the study. Increased systemic inflammation was defined for subjects within the upper 20% of the distribution for each marker. A central ambient monitoring station provided daily fine particle (PM(2.5)) and sulphate (SO(4)(2-)) data. Linear mixed models were used to identify associations between inflammatory markers and HRV and to assess effect modification of the association between air pollution and HRV due to inflammatory status.
RESULTS	A 5.8 mg/l elevation in CRP was associated with decreases of between -8% and -33% for time and frequency domain HRV outcomes. A 5.1 microg/m(3) increase in SO(4)(2-) on the day before the health assessment was associated with a decrease of -6.7% in the SD of normal RR intervals (SDNN) (95% CI -11.8% to -1.3%) in subjects with elevated CRP, but not in subjects with lower CRP (p value interaction=0.04), with similar findings for PM(2.5).
CONCLUSIONS	Increased systemic inflammation is associated with autonomic dysfunction in the elderly. Air pollution effects on reduced SDNN are stronger in subjects with elevated systemic inflammation.

표-26. PubMed 논문번호 20525188의 내용 요약

구분	내용
PubMed ID	20525188
TITLE	Association of cardiac and vascular changes with ambient PM2.5 in diabetic individuals.
JOURNAL	Particle and fibre toxicology: 10.1186/1743-8977-7-14
AUTHORS	Schneider Alexandra, Neas Lucas M, Graff Don W, Herbst Margaret C, Cascio Wayne E, Schmitt Mike T, Buse John B, Peters Annette, Devlin Robert B
BACKGROUND AND OBJECTIVE	Exposure to fine airborne particles (PM2.5) has been shown to be responsible for cardiovascular and hematological effects, especially in older people with cardiovascular disease. Some epidemiological studies suggest that individuals with diabetes may be a particularly susceptible population. This study examined effects of short-term exposures to ambient PM2.5 on markers of systemic inflammation, coagulation, autonomic control of heart rate, and repolarization in 22 adults (mean age: 61 years) with type 2 diabetes.
METHODS	Each individual was studied for four consecutive days with daily assessments of plasma levels of blood markers. Cardiac rhythm and electrocardiographic parameters were examined at rest and with 24-hour ambulatory ECG monitors. PM2.5 and meteorological data were measured daily on the rooftop of the patient exam site. Data were analyzed with models adjusting for season, weekday, meteorology, and a random intercept. To identify susceptible subgroups, effect modification was analyzed by clinical characteristics associated with insulin resistance as well as with oxidative stress and by medication intake.
RESULTS	Interleukin (IL)-6 and tumor necrosis factor alpha showed a significant increase with a lag of two days (percent change of mean level: 20.2% with 95%-confidence interval [6.4; 34.1] and 13.1% [1.9; 24.4], respectively) in association with an increase of 10 mug/m3 in PM2.5. Obese participants as well as individuals with elevated glycosylated hemoglobin, lower adiponectin, higher ferritin or with glutathione S-transferase M1 null genotype showed higher IL-6 effects. Changes in repolarization were found immediately as well as up to four days after exposure in individuals without treatment with a beta-adrenergic receptor blocker.
CONCLUSIONS	Exposure to elevated levels of PM2.5 alters ventricular repolarization and thus may increase myocardial vulnerability to arrhythmias. Exposure to PM2.5 also increases systemic inflammation. Characteristics associated with insulin resistance or with oxidative stress were shown to enhance the association.

표-27. PubMed 논문번호 20530563의 내용 요약

구분	내용
PubMed ID	20530563
TITLE	Short term impact of smoke-free legislation in England: retrospective analysis of hospital admissions for myocardial infarction.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.c2161
AUTHORS	Sims Michelle, Maxwell Roy, Bauld Linda, Gilmore Anna
OBJECTIVE	To measure the short term impact on hospital admissions for myocardial infarction of the introduction of smoke-free legislation in England on 1 July 2007.
DESIGN	An interrupted time series design with routinely collected hospital episode statistics data. Analysis of admissions from July 2002 to September 2008 (providing five years' data from before the legislation and 15 months' data from after) using segmented Poisson regression.
SETTING	England. Population All patients aged 18 or older living in England with an emergency admission coded with a primary diagnosis of myocardial infarction.
MAIN OUTCOME MEASURES	Weekly number of completed hospital admissions.
RESULTS	After adjustment for secular and seasonal trends and variation in population size, there was a small but significant reduction in the number of emergency admissions for myocardial infarction after the implementation of smoke-free legislation (-2.4%, 95% confidence interval -4.06% to -0.66%, P=0.007). This equates to 1200 fewer emergency admissions for myocardial infarction (1600 including readmissions) in the first year after legislation. The reduction in admissions was significant in men (3.1%, P=0.001) and women (3.8%, P=0.007) aged 60 and over, and men (3.5%, P<0.01) but not women (2.5% P=0.38) aged under 60.
CONCLUSION	This study adds to a growing body of evidence that smoke-free legislation leads to reductions in myocardial infarctions. It builds on previous work by showing that such declines are observed even when underlying reductions in admissions and potential confounders are controlled for. The considerably smaller decline in admissions observed in England compared with many other jurisdictions probably reflects aspects of the study design and the relatively low levels of exposure to secondhand smoke in England before the legislation.

표-28. PubMed 논문번호 20550697의 내용 요약

구분	내용
PubMed ID	20550697
TITLE	Cardiovascular effects of sub-daily levels of ambient fine particles: a systematic review.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-9-26
AUTHORS	Burgan Omar, Smargiassi Audrey, Perron St□phane, Kosatsky Tom
BACKGROUND	While the effects of daily fine particulate exposure (PM) have been well reviewed, the epidemiological and physiological evidence of cardiovascular effects associated to sub-daily exposures has not. We performed a theoretical model-driven systematic non-meta-analytical literature review to document the association between PM sub-daily exposures (< or =6 hours) and arrhythmia, ischemia and myocardial infarction (MI) as well as the likely mechanisms by which sub-daily PM exposures might induce these acute cardiovascular effects. This review was motivated by the assessment of the risk of exposure to elevated sub-daily levels of PM during fireworks displays.
METHODS	Medline and Elsevier's EMBase were consulted for the years 1996-2008. Search keywords covered potential cardiovascular effects, the pollutant of interest and the short duration of the exposure. Only epidemiological and experimental studies of adult humans (age > 18 yrs) published in English were reviewed. Information on design, population and PM exposure characteristics, and presence of an association with selected cardiovascular effects or physiological assessments was extracted from retrieved articles.
RESULTS	Of 231 articles identified, 49 were reviewed. Of these, 17 addressed the relationship between sub-daily exposures to PM and cardiovascular effects: five assessed ST-segment depression indicating ischemia, eight assessed arrhythmia or fibrillation and five considered MI. Epidemiologic studies suggest that exposure to sub-daily levels of PM is associated with MI and ischemic events in the elderly. Epidemiological studies of sub-daily exposures suggest a plausible biological mechanism involving the autonomic nervous system while experimental studies suggest that vasomotor dysfunction may also relate to the occurrence of MI and ischemic events.
CONCLUSIONS	Future studies should clarify associations between cardiovascular effects of sub-daily PM exposure with PM size fraction and concurrent gaseous pollutant exposures. Experimental studies appear more promising for elucidating the physiological mechanisms, time courses and causes than epidemiological studies which employ central pollution monitors for measuring effects and for assessing their time course. Although further studies are needed to strengthen the evidence, given that exposure to sub-daily high levels of PM (for a few hours) is frequent and given the suggestive evidence that sub-daily PM exposures are associated with the occurrence of cardiovascular effects, we recommend that persons with cardiovascular diseases avoid such situations.

표-29. PubMed 논문번호 20576121의 내용 요약

구분	내용
PubMed ID	20576121
TITLE	Cumulative exposure to air pollution and long term outcomes after first acute myocardial infarction: a population-based cohort study. Objectives and methodology.
JOURNAL	BMC public health: 10.1186/1471-2458-10-369
AUTHORS	Gerber Yariv, Myers Vicki, Broday David M, Koton Silvia, Steinberg David M, Drory Yaacov
BACKGROUND	Cardiovascular disease is a leading cause of morbidity and mortality worldwide and epidemiological studies have consistently shown an increased risk for cardiovascular events in relation to exposure to air pollution. The Israel Study of First Acute Myocardial Infarction was designed to longitudinally assess clinical outcomes, psychosocial adjustment and quality of life in patients hospitalized with myocardial infarction. The current study, by introducing retrospective air pollution data, will examine the association between exposure to air pollution and outcome in myocardial infarction survivors. This report will describe the methods implemented and measures employed. The study specifically aims to examine the relationship between residential exposure to air pollution and long-term risk of recurrent coronary event, heart failure, stroke, cardiac and all-cause death in a geographically defined cohort of patients with myocardial infarction.
METHODS/DESIGN	All 1521 patients aged < or =65 years, admitted with first myocardial infarction between February 1992 and February 1993 to the 8 hospitals serving the population of central Israel, were followed for a median of 13 years. Data were collected on sociodemographic, clinical and environmental factors. Data from air quality monitoring stations will be incorporated retrospectively. Daily measures of air pollution will be summarised, allowing detailed maps to be developed in order to reflect chronic exposure for each participant.
DISCUSSION	This study addresses some of the gaps in understanding of the prognostic importance of air pollution exposure after myocardial infarction, by allowing a sufficient follow-up period, using a well-defined community cohort, adequately controlling for multiple and multilevel confounding factors and providing extensive data on various outcomes.

표-30. PubMed 논문번호 20581417의 내용 요약

구분	내용
PubMed ID	20581417
TITLE	Exposure to metal welding fume particles and risk for cardiovascular disease in Denmark: a prospective cohort study.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2009.051086
AUTHORS	Ibfelt Else, Bonde Jens Peter, Hansen Johnni
OBJECTIVES	To study welding fume particles in relation to cardiovascular diseases.
METHODS	In 1986, 10,059 male metal workers in 75 welding companies were sent a questionnaire about their welding experience and lifestyle (83.3% response rate). Of these, 5866 were available for analysis and had ever welded at baseline. Information on exposure to welding fumes after 1986 was obtained by individual linkage to the National Pension Fund. Lifelong exposure to welding fume particles was estimated from a job-exposure matrix based on more than 1000 welding-specific measures of fume particles. Hospital contacts for cardiovascular disease were obtained from the Danish National Patient Registry by individual linkage. The nine disease outcomes considered were acute myocardial infarct (AMI), angina pectoris, other acute ischaemic heart diseases, chronic ischaemic heart disease (CHD), cardiac arrythmias, cardiac arrest, heart failure, cerebral infarct, arterial embolism and thrombosis. The cohort was followed up from baseline until the end of 2006.
RESULTS	When the incidence of each of the nine cardiovascular outcomes among welders was compared with 5-year age- and calendar year-specific male national rates, the number of observed cases significantly exceeded that expected for AMI (standardised incidence ratio, 95% CI) (1.12, 1.01 to 1.24), angina pectoris (1.11, 1.01 to 1.22), CHD (1.17, 1.05 to 1.31) and cerebral infarct (1.24, 1.06 to 1.44). Internal comparisons of the cohort with adjustment for tobacco smoking, alcohol and hypertension medicines showed a significantly increasing hazard rate ratio for CHD and non-significant increases for AMI, angina pectoris and cerebral infarct with increasing exposure to particles.
CONCLUSIONS	This study supports the hypothesis that exposure to welding processed particles increases the risk for cardiovascular disease.

표-31. PubMed 논문번호 20585255의 내용 요약

구분	내용
PubMed ID	20585255
TITLE	Changes in residential proximity to road traffic and the risk of death from coronary heart disease.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e3181e89f19
AUTHORS	Gan Wen Qi, Tamburic Lillian, Davies Hugh W, Demers Paul A, Koehoorn Mieke, Brauer Michael
BACKGROUND	Residential proximity to road traffic is associated with increased coronary heart disease (CHD) morbidity and mortality. It is unknown, however, whether changes in residential proximity to traffic could alter the risk of CHD mortality.
METHODS	We used a population-based cohort study with a 5-year exposure period and a 4-year follow-up period to explore the association between changes in residential proximity to road traffic and the risk of CHD mortality. The cohort comprised all residents aged 45-85 years who resided in metropolitan Vancouver during the exposure period and without known CHD at baseline (n = 450,283). Residential proximity to traffic was estimated using a geographic information system. CHD deaths during the follow-up period were identified using provincial death registration database. The data were analyzed using logistic regression.
RESULTS	Compared with the subjects consistently living away from road traffic (>150 m from a highway or >50 m from a major road) during the 9-year study period, those consistently living close to traffic (<or=150 m from a highway or <or=50 m from a major road) had the greatest risk of CHD mortality (relative risk [RR] = 1.29 [95% confidence interval = 1.18-1.41]). By comparison, those who moved closer to traffic during the exposure period had less increased risk than those who were consistently exposed (1.20 [1.00-1.43]), and those who moved away from traffic had even less increase in the risk (1.14 [0.95-1.37]). All analyses were adjusted for baseline age, sex, pre-existing comorbidities (diabetes, chronic obstructive pulmonary disease, hypertensive heart disease), and neighborhood socioeconomic status.
CONCLUSIONS	Living close to major roadways was associated with increased risk of coronary mortality, whereas moving away from major roadways was associated with decreased risk.

표-32. PubMed 논문번호 20620712의 내용 요약

구분	내용
PubMed ID	20620712
TITLE	Objectively measured secondhand smoke exposure and risk of cardiovascular disease: what is the mediating role of inflammatory and hemostatic factors?
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2010.03.032
AUTHORS	Hamer Mark, Stamatakis Emmanuel, Kivimaki Mika, Lowe Gordon D, Batty G David
OBJECTIVES	The aim of this study was to examine the association between objectively measured secondhand smoke (SHS) exposure and incident cardiovascular disease (CVD) death and assess the extent to which this association can be explained through novel circulating markers of inflammation and hemostasis.
BACKGROUND	Existing evidence suggests there is an association between SHS and CVD risk, although the mechanisms remain poorly understood.
METHODS	In a prospective study of 13,443 participants living in England and Scotland (age 53.5 +/- 12.6 years, 52.3% women), we measured salivary cotinine (an objective marker of SHS exposure) and novel CVD biomarkers (C-reactive protein, fibrinogen) at baseline.
RESULTS	Of the sample, 20.8% had high SHS exposure on the basis of elevated levels of salivary cotinine (range 0.71 to 14.99 ng/ml). During a mean follow-up of 8 years, there were 1,221 all-cause deaths and 364 CVD deaths. High SHS was associated with all-cause (age-adjusted hazard ratio [HR]: 1.25, 95% confidence interval [CI]: 1.02 to 1.53) and CVD death (age-adjusted HR: 1.21, 95% CI: 0.85 to 1.73). High SHS was also associated with elevated CRP, which explained 48% of the association between SHS and CVD death. The excess risk of CVD associated with active smoking was exaggerated in relation to self report (age-adjusted HR: 3.27, 95% CI: 2.48 to 4.31) compared with objective assessment (age-adjusted HR: 2.44, 95% CI: 1.75 to 3.40).
CONCLUSIONS	Among a large representative sample of British adults we observed elevated levels of low-grade inflammation in otherwise healthy participants exposed to high SHS, and this partly explained their elevated risk of CVD death.

표-33. PubMed 논문번호 20653945의 내용 요약

구분	내용
PubMed ID	20653945
TITLE	Impaired vascular function after exposure to diesel exhaust generated at urba transient running conditions.
JOURNAL	Particle and fibre toxicology: 10.1186/1743-8977-7-19
AUTHORS	Barath Stefan, Mills Nicholas L, Lundb□ck Magnus, T□rnqvist H□kan, Lucking Andrew J, Langrish Jeremy P, S□derberg Stefan, Boman Christoffer Westerholm Roger, L□ndahl Jakob, Donaldson Ken, Mudway Ian S, Sandstr□m Thomas, Newby David E, Blomberg Anders
BACKGROUND	Traffic emissions including diesel engine exhaust are associated with increase respiratory and cardiovascular morbidity and mortality. Controlled human exposure studies have demonstrated impaired vascular function after inhalation of exhaust generated by a diesel engine under idling conditions.
OBJECTIVES	To assess the vascular and fibrinolytic effects of exposure to diesel exhaust generated during urban-cycle running conditions that mimic ambient 'real-world' exposures.
METHODS	In a randomised double-blind crossover study, eighteen healthy male volunteers were exposed to diesel exhaust (approximately 250 microg/m3) o filtered air for one hour during intermittent exercise. Diesel exhaust was generated during the urban part of the standardized European Transient Cycle. Six hours post-exposure, vascular vasomotor and fibrinolytic functio was assessed during venous occlusion plethysmography with intra-arterial agonist infusions.
MEASUREMENTS AND MAIN RESULTS	Forearm blood flow increased in a dose-dependent manner with both endothelial-dependent (acetylcholine and bradykinin) and endothelial-independent (sodium nitroprusside and verapamil) vasodilators. Diesel exhaust exposure attenuated the vasodilatation to acetylcholine ($P < 0.001$), bradykinin ($P < 0.05$), sodium nitroprusside ($P < 0.05$) and verapamil ($P < 0.001$). In addition, the net release of tissue plasminogen activator durin bradykinin infusion was impaired following diesel exhaust exposure ($P < 0.05$
CONCLUSION	Exposure to diesel exhaust generated under transient running conditions, as a relevant model of urban air pollution, impairs vasomotor function and endogenous fibrinolysis in a similar way as exposure to diesel exhaust generated at idling. This indicates that adverse vascular effects of diesel exhaust inhalation occur over different running conditions with varying exhaust composition and concentrations as well as physicochemical particle properties. Importantly, exposure to diesel exhaust under ETC conditions wa also associated with a novel finding of impaired of calcium channel-dependen vasomotor function. This implies that certain cardiovascular endpoints seem to be related to general diesel exhaust properties, whereas the novel calcium flux-related effect may be associated with exhaust properties more specific fo the ETC condition, for example a higher content of diesel soot particles alon with their adsorbed organic compounds.

표-34. PubMed 논문번호 20653951의 내용 요약

구분	내용
PubMed ID	20653951
TITLE	Associations between outdoor temperature and markers of inflammation: a cohort study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-9-42
AUTHORS	Halonen Jaana I, Zanobetti Antonella, Sparrow David, Vokonas Pantel S, Schwartz Joel
BACKGROUND	Associations between ambient temperature and cardiovascular mortality are well established. This study investigated whether inflammation could be part of the mechanism leading to temperature-related cardiovascular deaths.
METHODS	The study population consisted of a cohort of 673 men with mean age of 74.6 years, living in the greater Boston area. They were seen for examination roughly every 4 years, and blood samples for inflammation marker analyses were drawn in 2000-2008 (total of 1254 visits). We used a mixed effects model to estimate the associations between ambient temperature and a variety of inflammation markers (C-reactive protein, white blood cell count, soluble Vascular Cell Adhesion Molecule-1, soluble Intercellular Adhesion Molecule-1, tumor necrosis factor alpha, and interleukins -1beta, -6 and -8). Random intercept for each subject and several possible confounders, including combustion-related air pollution and ozone, were used in the models.
RESULTS	We found a 0 to 1 day lagged and up to 4 weeks cumulative responses in C-reactive protein in association with temperature. We observed a 24.9% increase [95% Confidence interval (CI): 7.36, 45.2] in C-reactive protein for a 5 degrees C decrease in the 4 weeks' moving average of temperature. We observed similar associations also between temperature and soluble Intercellular Adhesion Molecule-1 (4.52%, 95% CI: 1.05, 8.10, over 4 weeks' moving average), and between temperature and soluble Vascular Cell Adhesion Molecule-1 (6.60%, 95% CI: 1.31, 12.2 over 4 weeks' moving average). Penalized spline models showed no deviation from linearity. There were no associations between temperature and other inflammation markers.
CONCLUSIONS	Cumulative exposure to decreased temperature is associated with an increase in inflammation marker levels among elderly men. This suggests that inflammation markers are part of intermediate processes, which may lead to cold-, but not heat-, related cardiovascular deaths.

표-35. PubMed 논문번호 20656944의 내용 요약

구분	내용
PubMed ID	20656944
TITLE	Long-term ambient multipollutant exposures and mortality.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.200912-1903OC
AUTHORS	Hart Jaime E, Garshick Eric, Dockery Douglas W, Smith Thomas J, Ryan Louise, Laden Francine, Hart Jaime E, Garshick Eric, Dockery Douglas W, Smith Thomas J, Ryan Louise, Laden Francine
RATIONALE	population-based studies have demonstrated associations between ambient a pollution exposures and mortality, but few have been able to adjust for occupational exposures. Additionally, two studies have observed higher risks in individuals with occupational dust, gas, or fume exposure.
OBJECTIVES	we examined the association of ambient residential exposure to particulate matter less than 10 microm in diameter (PM(10)), particulate matter less tha 2.5 microm in diameter (PM(2.5)), NO(2), SO(2), and mortality in 53,814 men in the U.S. trucking industry.
METHODS	exposures for PM(10), NO(2), and SO(2) at each residential address were assigned using models combining spatial smoothing and geographic covariates. PM(2.5) exposures in 2000 were assigned from the nearest available monitor. Single and multipollutant Cox proportional hazard model were used to examine the association of an interquartile range (IQR) change (6 microg/m(3) for PM(10), 4 microg/m(3) for PM(2.5), 4ppb for SO(2), an 8ppb for NO(2)) and the risk of all-cause and cause-specific mortality.
MEASUREMENTS AND MAIN RESULTS	an IQR change in ambient residential exposures to PM(10) was associated with a 4.3% (95% confidence interval [CI], 1.1-7.7%) increased risk of all-cause mortality. The increase for an IQR change in SO(2) was 6.9% (95% CI, 2.3-11.6%), for NO(2) was 8.2% (95% CI, 4.5-12.1%), and for PM(2.5) was 3.9% (95% CI, 1.0-6.9%). Elevated associations with cause-specific mortality (lung cancer, cardiovascular and respiratory disease) were observed for PM(2.5), SO(2), and NO(2), but not PM(10). None of the pollutants we confounded by occupational exposures. In multipollutant models, overall, th associations were attenuated, most strongly for PM(10). In sensitivity analyse excluding long-haul drivers, who spend days away from home, larger hazard ratios were observed.
CONCLUSIONS	in this population of men, residential ambient air pollution exposures were associated with mortality.

표-36. PubMed 논문번호 20736203의 내용 요약

구분	내용
PubMed ID	20736203
TITLE	Meta-analysis of the effect of comprehensive smoke-free legislation on acute coronary events.
JOURNAL	Heart (British Cardiac Society): 10.1136/hrt.2010.199026
AUTHORS	Mackay D F, Irfan M O, Haw S, Pell J P
OBJECTIVE	To assess the evidence for a reduced risk of acute coronary events following comprehensive smoke-free legislation.
METHODS	Two independent systematic reviews were undertaken using PubMed, Embase and Science Direct with no date restrictions imposed. Meta-analysis was undertaken using a random effects model to obtain a pooled estimate of the relative risk. Linear regression was used to examine possible bias and meta-regression was used to investigate possible causes of heterogeneity.
MAIN OUTCOME MEASURE	Acute coronary events.
RESULTS	The 17 eligible studies (10 from North America, 6 from Europe and 1 from Australasia) provided 35 estimates of effect size. Apart from five subgroup analyses, all of the published results suggested a reduction in the incidence of acute coronary events following the introduction of smoke-free legislation. Meta-analysis produced a pooled estimate of the relative risk of 0.90 (95% CI 0.86 to 0.94). There was significant heterogeneity (overall I(2)=95.1%, p<0.001) but there was no evidence of small study bias (p=0.714). On univariate random effects meta-regression analysis, studies with longer data collection following legislation produced greater estimates of risk reduction and remained significant after adjustment for other study characteristics (adjusted coefficient −0.005, 95% CI −0.007 to −0.002, multiplicity adjusted p=0.006).
CONCLUSIONS	There is now a large body of evidence supporting a reduction in acute coronary events following the implementation of comprehensive smoke-free legislation, with the effect increasing over time from implementation. Countries that have not yet adopted smoke-free legislation should be encouraged to do so.

표-37. PubMed 논문번호 20822968의 내용 요약

구분	내용
PubMed ID	20822968
TITLE	Vascular effects of ultrafine particles in persons with type 2 diabetes.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002237
AUTHORS	Stewart Judith C, Chalupa David C, Devlin Robert B, Frasier Lauren M, Huang Li-Shan, Little Erika L, Lee Steven M, Phipps Richard P, Pietropaoli Anthony P, Taubman Mark B, Utell Mark J, Frampton Mark W
BACKGROUND	Diabetes confers an increased risk for cardiovascular effects of airborne particles.
OBJECTIVE	We hypothesized that inhalation of elemental carbon ultrafine particles (UFP) would activate blood platelets and vascular endothelium in people with type 2 diabetes.
METHODS	In a randomized, double-blind, crossover trial, 19 subjects with type 2 diabetes inhaled filtered air or 50 □g/m³ elemental carbon UFP (count median diameter, 32 nm) by mouthpiece for 2 hr at rest. We repeatedly measured markers of vascular activation, coagulation, and systemic inflammation before and after exposure.
RESULTS	Compared with air, particle exposure increased platelet expression of CD40 ligand (CD40L) and the number of platelet-leukocyte conjugates 3.5 hr after exposure. Soluble CD40L decreased with UFP exposure. Plasma von Willebrand factor increased immediately after exposure. There were no effects of particles on plasma tissue factor, coagulation factors VII or IX, or D-dimer.
CONCLUSIONS	Inhalation of elemental carbon UFP for 2-hr transiently activated platelets, and possibly the vascular endothelium, in people with type 2 diabetes.

표-38. PubMed 논문번호 20852305의 내용 요약

구분	내용
PubMed ID	20852305
TITLE	Mortality in small geographical areas and proximity to air polluting industries in the Basque Country (Spain).
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2009.048215
AUTHORS	Cambra K, Mart□nez-Rueda T, Alonso-Fustel E, Cirarda F B, Ib□□ez B, Esnaola S, Calvo M, Aldasoro E, Montoya I, Cambra K, Mart□nez-Rueda T, Alonso-Fustel E, Cirarda F B, Ib□□ez B, Esnaola S, Calvo M, Aldasoro E, Montoya I
OBJECTIVES	To study the association between proximity to air polluting industrial facilities and mortality in the Basque Country (Spain) in the 1996-2003 period.
METHODS	A cross-sectional ecological study with 1465 census sections (CS) as units of analysis with a mean population of 1257 inhabitants. Association of CS mortality with proximity of industries of the European Pollutant Emission Register was studied by type of industrial activity and adjusted for social deprivation. Two distance thresholds (1 km and 2 km) were used as proxies for exposure in a 'near versus far' analysis. Causes of mortality studied were: all causes; tracheal, bronchial, and lung cancer; haematological tumours; ischaemic heart disease; cerebrovascular diseases; chronic diseases of the lower respiratory tract; and breast cancer (in women). Poisson's generalised linear mixed models (GLMM) with two random effects (heterogeneity and structured spatial variability) were used in a fully Bayesian environment.
RESULTS	Men living in sections within 1 km from energy production industries had greater mortality from tracheal, bronchial, and lung cancer [CI(90%) 6% to 53%] as compared with people living further. Women had greater mortality from ischaemic heart disease [CI(90%) 1% to 17%] and respiratory illness [CI(90%) 1% to 24%] within 2 km from metal-processing industries. On the contrary, within the 1 km buffer from mineral industries, mortality was lower for all causes [CI(90%) -20% to -6%] and for ischaemic heart disease [CI(90%) -40% to -10%] in women, and from respiratory diseases in men [CI(90%) -39% to -4%], while it was greater for breast cancer in women [CI(90%) 2% to 28%] within the 2 km buffer.
CONCLUSIONS	Analysis of mortality by census sections is a helpful exploratory tool for investigating environmental risk factors and directing actions to sites and risk factors with a greater impact on health. Further epidemiological and environmental investigations around metal-processing and energy-producing plants are required.

표-39. PubMed 논문번호 20864465의 내용 요약

구분	내용
PubMed ID	20864465
TITLE	Relationship between outdoor temperature and blood pressure.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2010.056507
AUTHORS	Halonen Jaana I, Zanobetti Antonella, Sparrow David, Vokonas Pantel S, Schwartz Joel, Halonen Jaana I, Zanobetti Antonella, Sparrow David, Vokonas Pantel S, Schwartz Joel
OBJECTIVES	Cardiovascular mortality has been linked to changes in outdoor temperature. However, the mechanisms behind these effects are not well established. We aimed to study the effect of outdoor temperature on blood pressure, as increased blood pressure is a risk factor for cardiovascular death.
METHODS	The study population consisted of men aged 53-100 years living in the Boston area. We used a mixed effects model to estimate the effect of three temperature variables: ambient, apparent and dew point temperature (DPT), on repeated measures (every 3-5 years) of diastolic (DBP) and systolic blood pressure (SBP). Random intercepts for subjects and several possible confounders were used in the models, including black carbon and barometric pressure.
RESULTS	We found modest associations between DBP and ambient and apparent temperature. In the basic models, DBP in association with a 5 ° C decrease in 7-day moving averages of temperatures increased by 1.01% (95% CI -0.06% to 2.09%) and 1.55% (95% CI 0.61% to 2.49%) for ambient and apparent temperature, respectively. Excluding extreme temperatures strengthened these associations (2.13%, 95% CI 0.66% to 3.63%, and 1.65%, 95% CI 0.41% to 2.90%, for ambient and apparent temperature, respectively). Effect estimates for DPT were close to null. The effect of apparent temperature on SBP was similar (1.30% increase (95% CI 0.32% to 2.29%) for a 5 ° C decrease in 7-day moving average).
CONCLUSIONS	Cumulative exposure to decreasing ambient and apparent temperature may increase blood pressure. These findings suggest that an increase in blood pressure could be a mechanism behind cold-related, but not heat-related, cardiovascular mortality.

표-40. PubMed 논문번호 20870755의 내용 요약

구분	내용
PubMed ID	20870755
TITLE	Chronic obstructive pulmonary disease and long-term exposure to traffic-related air pollution: a cohort study.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201006-0937OC
AUTHORS	Andersen Zorana J, Hvidberg Martin, Jensen Steen S, Ketzel Matthias, Loft Steffen, S ø rensen Mette, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole, Andersen Zorana J, Hvidberg Martin, Jensen Steen S Ketzel Matthias, Loft Steffen, S ø rensen Mette, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
RATIONALE	Short-term exposure to air pollution has been associated with exacerbation chronic obstructive pulmonary disease (COPD), whereas the role of long-ter exposures on the development of COPD is not yet fully understood.
OBJECTIVES	We assessed the effect of exposure to traffic-related air pollution over 35 yea on the incidence of COPD in a prospective cohort study.
METHODS	We followed 57,053 participants in the Danish Diet, Cancer, and Health cohort in the Hospital Discharge Register for their first hospital admission fo COPD between 1993 and 2006. We estimated the annual mean levels of nitrogen dioxide (NO_2) and nitrogen oxides (NO(x)) at all residential addresses of the cohort participants since 1971 to an event or 2006 and used indicators of traffic near the residential address at recruitment. We assessed the association between exposure to air pollution and COPD incidence by Co regression analyses for the full cohort, and for participants with and without comorbid conditions, including asthma, diabetes, or cardiovascular disease.
MEASUREMENTS AND MAIN RESULTS	A first hospital admission for COPD was recorded for 1,786 (3.4%) of 52,79 eligible subjects between recruitment (1993-1997) and 2006. COPD incidenc was associated with the 35-year mean NO_2 level (hazard ratio, 1.08; 95% confidence interval, 1.02-1.14, per interquartile range of 5.8 $\mu g/m^3$), with stronger associations in subjects with diabetes (1.29; 1.05-1.50) and asthma (1.19; 1.03-1.38).
CONCLUSIONS	Long-term exposure to traffic-related air pollution may contribute to the development of COPD with possibly enhanced susceptibility in people with diabetes and asthma.

표-41. PubMed 논문번호 20881600의 내용 요약

구분	내용
PubMed ID	20881600
TITLE	Aircraft noise, air pollution, and mortality from myocardial infarction.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e3181f4e634
AUTHORS	Huss Anke, Spoerri Adrian, Egger Matthias, R□□sli Martin
OBJECTIVE	Myocardial infarction has been associated with both transportation noise and air pollution. We examined residential exposure to aircraft noise and mortality from myocardial infarction, taking air pollution into account.
METHODS	We analyzed the Swiss National Cohort, which includes geocoded information on residence. Exposure to aircraft noise and air pollution was determined based on geospatial noise and air-pollution (PM10) models and distance to major roads. We used Cox proportional hazard models, with age as the timescale. We compared the risk of death across categories of A-weighted sound pressure levels (dB(A)) and by duration of living in exposed corridors, adjusting for PM10 levels, distance to major roads, sex, education, and socioeconomic position of the municipality.
RESULTS	We analyzed 4.6 million persons older than 30 years who were followed from near the end of 2000 through December 2005, including 15,532 deaths from myocardial infarction (ICD-10 codes I 21, I 22). Mortality increased with increasing level and duration of aircraft noise. The adjusted hazard ratio comparing ≥60 dB(A) with <45 dB(A) was 1.3 (95% confidence interval = 0.96-1.7) overall, and 1.5 (1.0-2.2) in persons who had lived at the same place for at least 15 years. None of the other endpoints (mortality from all causes, all circulatory disease, cerebrovascular disease, stroke, and lung cancer) was associated with aircraft noise.
CONCLUSION	Aircraft noise was associated with mortality from myocardial infarction, with a dose-response relationship for level and duration of exposure. The association does not appear to be explained by exposure to particulate matter air pollution, education, or socioeconomic status of the municipality.

표-42. PubMed 논문번호 20929904의 내용 요약

구분	내용
PubMed ID	20929904
TITLE	Direct health costs of environmental tobacco smoke exposure and indirect health benefits due to smoking ban introduction.
JOURNAL	European journal of public health: 10.1093/eurpub/ckq142
AUTHORS	Hauri Dimitri D, Lieb Christoph M, Rajkumar Sarah, Kooijman Cornelis, Sommer Heini L, R□□sli Martin, Hauri Dimitri D, Lieb Christoph M, Rajkumar Sarah, Kooijman Cornelis, Sommer Heini L, R□□sli Martin
BACKGROUND	Introducing comprehensive smoke-free policies to public places is expected to reduce health costs. This includes prevented health damages by avoiding environmental tobacco smoke (ETS) exposure as well as indirect health benefits from reduced tobacco consumption.
METHODS	The aim of this study was to estimate direct health costs of ETS exposure in public places and indirect health benefits from reduced tobacco consumption. We calculated attributable hospital days and years of life lost (YLL), based on the observed passive smoking and disease rates in Switzerland. The exposure-response associations of all relevant health outcomes were derived by meta-analysis from prospective cohort studies in order to calculate the direct health costs. To assess the indirect health benefits, a meta-analysis of smoking ban studies on hospital admissions for acute myocardial infarction was conducted.
RESULTS	ETS exposure in public places in Switzerland causes 32,000 preventable hospital days (95% CI: 10,000-61,000), 3000 YLL (95% CI: 1000-5000), corresponding to health costs of 330 Mio CHF. The number of hospital days for ischaemic heart disease attributable to passive smoking is much larger if derived from smoking ban studies (41,000) than from prospective cohort studies (3200), resulting in additional health costs of 89 Mio CHF, which are attributed to the indirect health benefits of a smoking ban introduction.
CONCLUSION	The example of smoking ban studies on ischaemic heart disease hospitalization rates suggests that total health costs that can be prevented with smoking bans are considerably larger than the costs arising from the direct health impact of ETS exposure in public places.

표-43. PubMed 논문번호 20962342의 내용 요약

구분	내용
PubMed ID	20962342
TITLE	Diesel exhaust inhalation does not affect heart rhythm or heart rate variability.
JOURNAL	Heart (British Cardiac Society): 10.1136/hrt.2010.199042
AUTHORS	Mills Nicholas L, Finlayson Alexander E, Gonzalez Manuel C, T□rnqvist H□kan, Barath Stefan, Vink Elen, Goudie Colin, Langrish Jeremy P, S□derberg Stefan, Boon Nicholas A, Fox Keith A A, Donaldson Ken, Sandstr□m Thomas, Blomberg Anders, Newby David E, Mills Nicholas L, Finlayson Alexander E, Gonzalez Manuel C, T□rnqvist H□kan, Barath Stefan, Vink Elen, Goudie Colin, Langrish Jeremy P, S□derberg Stefan, Boon Nicholas A, Fox Keith A A, Donaldson Ken, Sandstr□m Thomas, Blomberg Anders, Newby David E
OBJECTIVE	Exposure to air pollution is associated with increases in cardiovascular morbidity and mortality. This study was undertaken to determine the effect of diesel exhaust inhalation on heart rhythm and heart rate variability in healthy volunteers and patients with coronary heart disease.
DESIGN AND SETTING	Double-blind randomised crossover studies in a university teaching hospital.
PATIENTS	32 healthy non-smoking volunteers and 20 patients with prior myocardial infarction.
INTERVENTIONS	All 52 subjects were exposed for 1 h to dilute diesel exhaust (particle concentration 300 $\mu g/m^3$) or filtered air.
MAIN OUTCOME MEASURES	Heart rhythm and heart rate variability were monitored during and for 24 h after the exposure using continuous ambulatory electrocardiography and assessed using standard time and frequency domain analysis.
RESULTS	No significant arrhythmias occurred during or following exposures. Patients with coronary heart disease had reduced autonomic function in comparison to healthy volunteers, with reduced standard deviations of the NN interval (SDNN, $p < 0.001$) and triangular index ($p < 0.001$). Diesel exhaust did not affect heart rate variability compared with filtered air ($p > 0.05$ for all) in healthy volunteers (SDNN 101 ± 6 vs 91 ± 6, triangular index 20 ± 1 vs 21 ± 1) or patients with coronary heart disease (SDNN 47 ± 5 vs 38 ± 4, triangular index 8 ± 1 vs 7 ± 1).
CONCLUSIONS	Brief exposure to dilute diesel exhaust does not alter heart rhythm or heart rate variability in healthy volunteers or well-treated patients with stable coronary heart disease. Autonomic dysfunction does not appear to be a dominant mechanism that can explain the observed excess in cardiovascular events following exposure to combustion-derived air pollution.

표-44. PubMed 논문번호 20965803의 내용 요약

구분	내용
PubMed ID	20965803
TITLE	Electrocardiographic ST-segment depression and exposure to traffic-related aerosols in elderly subjects with coronary artery disease.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002372
AUTHORS	Delfino Ralph J, Gillen Daniel L, Tjoa Thomas, Staimer Norbert, Polidori Andrea, Arhami Mohammad, Sioutas Constantinos, Longhurst John, Delfino Ralph J, Gillen Daniel L, Tjoa Thomas, Staimer Norbert, Polidori Andrea, Arhami Mohammad, Sioutas Constantinos, Longhurst John
BACKGROUND	Air pollutants have not been associated with ambulatory electrocardiographic evidence of ST-segment depression ≥ 1 mm (probable cardiac ischemia). We previously found that markers of primary (combustion-related) organic aerosols and gases were positively associated with circulating biomarkers of inflammation and ambulatory blood pressure in the present cohort panel study of elderly subjects with coronary artery disease.
OBJECTIVES	We specifically aimed to evaluate whether exposure markers of primary organic aerosols and ultrafine particles were more strongly associated with ST-segment depression of ≥ 1 mm than were secondary organic aerosols or PM2.5 (particulate matter with aerodynamic diameter ≤ 2.5 □m) mass.
METHODS	We evaluated relations of air pollutants to ambulatory electrocardiographic evidence of cardiac ischemia over 10 days in 38 subjects without ST depression on baseline electrocardiographs. Exposures were measured outdoors in retirement communities in the Los Angeles basin, including daily size-fractionated particle mass and hourly markers of primary and secondary organic aerosols and gases. Generalized estimating equations were used to estimate odds of hourly ST-segment depression (≥ 1 mm) from hourly air pollution exposures and to estimate relative rates of daily counts of ST-segment depression from daily average exposures, controlling for potential confounders.
RESULTS	We found significant positive associations of hourly ST-segment depression with markers of combustion-related aerosols and gases averaged 1-hr through 3-4 days, but not secondary (photochemically aged) organic aerosols or ozone. The odds ratio per interquartile increase in 2-day average primary organic carbon (5.2 □g/m3) was 15.4 (95% confidence interval, 3.5-68.2). Daily counts of ST-segment depression were consistently associated with primary combustion markers and 2-day average quasi-ultrafine particles < 0.25 □m.
CONCLUSIONS	Results suggest that exposure to quasi-ultrafine particles and combustion-related pollutants (predominantly from traffic) increase the risk of myocardial ischemia, coherent with our previous findings for systemic inflammation and blood pressure.

표-45. PubMed 논문번호 20980001의 내용 요약

구분	내용
PubMed ID	20980001
TITLE	Lifestyle and cardiovascular risk factors in 2001 child-parent pairs: the PEP Family Heart Study.
JOURNAL	Atherosclerosis: 10.1016/j.atherosclerosis.2010.09.032
AUTHORS	Schwandt Peter, Haas Gerda-Maria, Liepold Evelyn
OBJECTIVE	Genes and environment are the main determinants of cardiovascular disease (CVD) risk factor clustering in families. Since individual risk factor profiles are easily predicted within families, we examined whether CVD risk factors are affected by lifestyle factors in these families.
METHODS	Nutrition, physical activity, and smoking habits were assessed in 2001 biological child-parent pairs from 852 families participating in the Prevention Education Program (PEP). Height, weight, body mass index, waist circumference, blood pressure, and fasting lipid levels were measured. Within-family associations were calculated using generalized estimating equations (GEE).
RESULTS	Fathers possessed the most adverse risk profile. Daily energy consumption above the recommended levels was higher in children (daughters +35.4%, sons +26.7%) than in parents (fathers +15.7%, mothers +10.6%). Higher energy consumption was significantly associated with hypertension in mothers (OR 2.5) and in fathers (OR 1.7). Hyper-caloric nutrition of the parents predicted the energy intake of the children in: mother-daughter (OR 7.5), mother-son (OR 3.0), and father-son (OR 2.8) pairs. Low mono-unsaturated fatty acid intake was significantly associated with a high LDL/HDL-C ratio (OR 3.4) and hypertriglyceridemia (OR 2.2) in fathers. Approximately 25% of parents and children reported at least two physical activities twice a week. The 23% of children who were passive smokers presented a far more adverse risk profile than children without exposure to second-hand smoke.
CONCLUSION	Intergenerational lifestyle habits affect cardiovascular risk factors within biological families. As lifestyle habits are predictable, they may be used for implementation of family-based CVD prevention strategies.

표-46. PubMed 논문번호 21029174의 내용 요약

구분	내용
PubMed ID	21029174
TITLE	Pollution sources and mortality rates across rural-urban areas in the United States.
JOURNAL	The Journal of rural health : official journal of the American Rural Health Association and the National Rural Health Care Association: 10.1111/j.1748-0361.2010.00305.x
AUTHORS	Hendryx Michael, Fedorko Evan, Halverson Joel
PURPOSE	To conduct an assessment of rural environmental pollution sources and associated population mortality rates.
METHODS	The design is a secondary analysis of county-level data from the Environmental Protection Agency (EPA), Department of Agriculture, National Land Cover Dataset, Energy Information Administration, Centers for Disease Control and Prevention, the US Census, and others. We described the types of pollution sources present in metropolitan and nonmetropolitan counties and examined the associations between these sources and rates of all-cause, cardiovascular, respiratory, and cancer mortality while controlling for age, race, and other covariates.
FINDINGS	Rural counties had 65,055 EPA-monitored pollution discharge sites. As expected, rural counties had significantly greater exposure to potential agriculture-related pollution. Regression models specific to rural counties indicated that greater density of water pollution sources was significantly associated with greater total and cancer mortality. Rural air pollution sources were associated with greater cancer mortality rates. Rural coal mining areas had higher total, cancer, and respiratory disease mortality rates. Agricultural production was generally associated with lower mortality rates. Greater levels of human development were significantly related to higher adjusted total and cancer mortality.
CONCLUSIONS	The association between pollution sources and mortality risk is not a phenomenon limited to metropolitan areas. Results carry policy implications regarding the need for effective environmental standards and monitoring. Further research is needed to better understand the types and distributions of pollution in rural areas, and the health consequences that result.

표-47. PubMed 논문번호 21036692의 내용 요약

구분	내용
PubMed ID	21036692
TITLE	Ambient particulate matter air pollution and venous thromboembolism in the Women's Health Initiative Hormone Therapy trials.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002256
AUTHORS	Shih Regina A, Griffin Beth Ann, Salkowski Nicholas, Jewell Adria, Eibner Christine, Bird Chloe E, Liao Duanping, Cushman Mary, Margolis Helene G, Eaton Charles B, Whitsel Eric A, Shih Regina A, Griffin Beth Ann, Salkowski Nicholas, Jewell Adria, Eibner Christine, Bird Chloe E, Liao Duanping, Cushman Mary, Margolis Helene G, Eaton Charles B, Whitsel Eric A
BACKGROUND	The putative effects of postmenopausal hormone therapy on the association between particulate matter (PM) air pollution and venous thromboembolism (VTE) have not been assessed in a randomized trial of hormone therapy, despite its widespread use among postmenopausal women.
OBJECTIVE	In this study, we examined whether hormone therapy modifies the association of PM with VTE risk.
METHODS	Postmenopausal women 50-79 years of age (n = 26,450) who did not have a history of VTE and who were not taking anticoagulants were enrolled in the Women's Health Initiative Hormone Therapy trials at 40 geographically diverse U.S. clinical centers. The women were randomized to treatment with estrogen versus placebo (E trial) or to estrogen plus progestin versus placebo (E + P trial). We used age-stratified Cox proportional hazard models to examine the association between time to incident, centrally adjudicated VTE, and daily mean PM concentrations spatially interpolated at geocoded addresses of the participants and averaged over 1, 7, 30, and 365 days.
RESULTS	During the follow-up period (mean, 7.7 years), 508 participants (2.0%) had VTEs at a rate of 2.6 events per 1,000 person-years. Unadjusted and covariate-adjusted VTE risk was not associated with concentrations of PM 〈 2.5 □m (PM(2.5)) or 〈 10 □m (PM(10))] in aerodynamic diameter and PM × active treatment interactions were not statistically significant (p 〉 0.05) regardless of PM averaging period, either before or after combining data from both trials [e.g., combined trial-adjusted hazard ratios (95% confidence intervals) per 10 □g/m(3) increase in annual mean PM(2.5) and PM(10), were 0.93 (0.54-1.60) and 1.05 (0.72-1.53), respectively]. Findings were insensitive to alternative exposure metrics, outcome definitions, time scales, analytic methods, and censoring dates.
CONCLUSIONS	In contrast to prior research, our findings provide little evidence of an association between short-term or long-term PM exposure and VTE, or clinically important modification by randomized exposure to exogenous estrogens among postmenopausal women.

표-48. PubMed 논문번호 21059260의 내용 요약

구분	내용
PubMed ID	21059260
TITLE	Acute effects of fine particulate air pollution on ST segment height: a longitudinal study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-9-68
AUTHORS	He Fan, Shaffer Michele L, Rodriguez-Colon Sol, Bixler Edward O, Vgontzas Alexandros N, Williams Ronald W, Wu Rongling, Cascio Wayne E, Liao Duanping
BACKGROUND	The mechanisms for the relationship between particulate air pollution and cardiac disease are not fully understood. Air pollution-induced myocardial ischemia is one of the potentially important mechanisms.
METHODS	We investigate the acute effects and the time course of fine particulate pollution (PM2.5) on myocardium ischemic injury as assessed by ST-segment height in a community-based sample of 106 healthy non-smokers. Twenty-four hour beat-to-beat electrocardiogram (ECG) data were obtained using a high resolution 12-lead Holter ECG system. After visually identifying and removing all the artifacts and arrhythmic beats, we calculated beat-to-beat ST-height from ten leads (inferior leads II, III, and aVF; anterior leads V3 and V4; septal leads V1 and V2; lateral leads I, V5, and V6,). Individual-level 24-hour real-time PM2.5 concentration was obtained by a continuous personal PM2.5 monitor. We then calculated, on a 30-minute basis, the corresponding time-of-the-day specific average exposure to PM2.5 for each participant. Distributed lag models under a linear mixed-effects models framework were used to assess the regression coefficients between 30-minute PM2.5 and ST-height measures from each lead; i.e., one lag indicates a 30-minute separation between the exposure and outcome.
RESULTS	The mean (SD) age was 56 (7.6) years, with 41% male and 74% white. The mean (SD) PM2.5 exposure was 14 (22) μg/m3. All inferior leads (II, III, and aVF) and two out of three lateral leads (I and V6), showed a significant association between higher PM2.5 levels and higher ST-height. Most of the adverse effects occurred within two hours after PM2.5 exposure. The multivariable adjusted regression coefficients β (95% CI) of the cumulative effect due to a 10 μg/m3 increase in Lag 0-4 PM2.5 on ST-I, II, III, aVF and ST-V6 were 0.29 (0.01-0.56) μV, 0.79 (0.20-1.39) μV, 0.52 (0.01-1.05) μV, 0.65 (0.11-1.19) μV, and 0.58 (0.07-1.09) μV, respectively, with all $p < 0.05$.
CONCLUSIONS	Increased PM2.5 concentration is associated with immediate increase in ST-segment height in inferior and lateral leads, generally within two hours. Such an acute effect of PM2.5 may contribute to increased potential for regional myocardial ischemic injury among healthy individuals.

표-49. PubMed 논문번호 21081301의 내용 요약

구분	내용
PubMed ID	21081301
TITLE	Long-term exposure to traffic-related air pollution and the risk of coronary heart disease hospitalization and mortality.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002511
AUTHORS	Gan Wen Qi, Koehoorn Mieke, Davies Hugh W, Demers Paul A, Tamburic Lillian, Brauer Michael, Gan Wen Qi, Koehoorn Mieke, Davies Hugh W, Demers Paul A, Tamburic Lillian, Brauer Michael
BACKGROUND	Epidemiologic studies have demonstrated that exposure to road traffic is associated with adverse cardiovascular outcomes.
OBJECTIVES	We aimed to identify specific traffic-related air pollutants that are associated with the risk of coronary heart disease (CHD) morbidity and mortality to support evidence-based environmental policy making.
METHODS	This population-based cohort study included a 5-year exposure period and a 4-year follow-up period. All residents 45-85 years of age who resided in Metropolitan Vancouver during the exposure period and without known CHD at baseline were included in this study (n=452,735). Individual exposures to traffic-related air pollutants including black carbon, fine particles [aerodynamic diameter ≤ 2.5 □m (PM(2.5))], nitrogen dioxide (NO(2)), and nitric oxide were estimated at residences of the subjects using land-use regression models and integrating changes in residences during the exposure period. CHD hospitalizations and deaths during the follow-up period were identified from provincial hospitalization and death registration records.
RESULTS	An interquartile range elevation in the average concentration of black carbon (0.94 × 10(-5)/m filter absorbance, equivalent to approximately 0.8 □g/m(3) elemental carbon) was associated with a 3% increase in CHD hospitalization (95% confidence interval, 1-5%) and a 6% increase in CHD mortality (3-9%) after adjusting for age, sex, preexisting comorbidity, neighborhood socioeconomic status, and copollutants (PM(2.5) and NO(2)). There were clear linear exposure-response relationships between black carbon and coronary events.
CONCLUSIONS	Long-term exposure to traffic-related fine particulate air pollution, indicated by black carbon, may partly explain the observed associations between exposure to road traffic and adverse cardiovascular outcomes.

표-50. PubMed 논문번호 21087707의 내용 요약

구분	내용
PubMed ID	21087707
TITLE	Urban particulate matter air pollution is associated with subclinical atherosclerosis: results from the HNR (Heinz Nixdorf Recall) study.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2010.04.065
AUTHORS	Bauer Marcus, Moebus Susanne, M□hlenkamp Stefan, Dragano Nico, Nonnemacher Michael, Fuchsluger Miriam, Kessler Christoph, Jakobs Hermann, Memmesheimer Michael, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara
OBJECTIVES	The aim of this study was to investigate the association of long-term residential exposure to fine particles with carotid intima-media thickness (CIMT).
BACKGROUND	Experimental and epidemiological evidence suggest that long-term exposure to air pollution might have a causal role in atherogenesis, but epidemiological findings are still inconsistent. We investigate whether urban particulate matter (PM) air pollution is associated with CIMT, a marker of subclinical atherosclerosis.
METHODS	We used baseline data (2000 to 2003) from the HNR (Heinz Nixdorf Recall) study, a population-based cohort of 4,814 participants, 45 to 75 years of age. We assessed residential long-term exposure to PM with a chemistry transport model and measured distance to high traffic. Multiple linear regression was used to estimate associations of air pollutants and traffic with CIMT, adjusting for each other, city of residence, age, sex, diabetes, and lifestyle variables.
RESULTS	Median CIMT of the 3,380 analyzed participants was 0.66 mm (interquartile range 0.16 mm). An interdecile range increase in PM(2.5) (4.2 μg/m(3)), PM(10) (6.7 μg/m(3)), and distance to high traffic (1,939 m) was associated with a 4.3% (95% confidence interval [CI]: 1.9% to 6.7%), 1.7% (95% CI: -0.7% to 4.1%), and 1.2% (95% CI: -0.2% to 2.6%) increase in CIMT, respectively.
CONCLUSIONS	Our study shows a clear association of long-term exposure to PM(2.5) with atherosclerosis. This finding strengthens the hypothesized role of PM(2.5) as a risk factor for atherogenesis.

표-51. PubMed 논문번호 21112082의 내용 요약

구분	내용
PubMed ID	21112082
TITLE	Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(10)61388-8
AUTHORS	Oberg Mattias, Jaakkola Maritta S, Woodward Alistair, Peruga Armando, Pr□ss-Ust□n Annette
BACKGROUND	Exposure to second-hand smoke is common in many countries but the magnitude of the problem worldwide is poorly described. We aimed to estimate the worldwide exposure to second-hand smoke and its burden of disease in children and adult non-smokers in 2004.
METHODS	The burden of disease from second-hand smoke was estimated as deaths and disability-adjusted life-years (DALYs) for children and adult non-smokers. The calculations were based on disease-specific relative risk estimates and area-specific estimates of the proportion of people exposed to second-hand smoke, by comparative risk assessment methods, with data from 192 countries during 2004.
FINDINGS	Worldwide, 40% of children, 33% of male non-smokers, and 35% of female non-smokers were exposed to second-hand smoke in 2004. This exposure was estimated to have caused 379,000 deaths from ischaemic heart disease, 165,000 from lower respiratory infections, 36,900 from asthma, and 21,400 from lung cancer. 603,000 deaths were attributable to second-hand smoke in 2004, which was about 1·0% of worldwide mortality. 47% of deaths from second-hand smoke occurred in women, 28% in children, and 26% in men. DALYs lost because of exposure to second-hand smoke amounted to 10·9 million, which was about 0·7% of total worldwide burden of diseases in DALYs in 2004. 61% of DALYs were in children. The largest disease burdens were from lower respiratory infections in children younger than 5 years (5,939,000), ischaemic heart disease in adults (2,836,000), and asthma in adults (1,246,000) and children (651,000).
INTERPRETATION	These estimates of worldwide burden of disease attributable to second-hand smoke suggest that substantial health gains could be made by extending effective public health and clinical interventions to reduce passive smoking worldwide.
FUNDING	Swedish National Board of Health and Welfare and Bloomberg Philanthropies.

표-52. PubMed 논문번호 21118784의 내용 요약

구분	내용
PubMed ID	21118784
TITLE	Are particulate matter exposures associated with risk of type 2 diabetes?
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002344
AUTHORS	Puett Robin C, Hart Jaime E, Schwartz Joel, Hu Frank B, Liese Angela D, Laden Francine, Puett Robin C, Hart Jaime E, Schwartz Joel, Hu Frank B, Liese Angela D, Laden Francine
BACKGROUND	Although studies have found that diabetes mellitus (DM) modifies the impact of exposures from air pollution on cardiovascular outcomes, information is limited regarding DM as an air pollution-associated outcome.
OBJECTIVES	Using two prospective cohorts, the Nurses' Health Study (NHS) and the Health Professionals Follow-Up Study (HPFS), we investigated the relationship of incident type 2 DM with exposures to particulate matter (PM) < 2.5 □m (PM2.5), PM < 10 □m (PM10), and PM between 2.5 and 10 □m in aerodynamic diameter (PM10-2.5) in the previous 12 months and the distance to roadways.
METHODS	Cases were reported and confirmed through biennial and supplemental questionnaires of diagnosis and treatment information. During follow-up from 1989 to 2002, questionnaires provided information on time-varying covariates and updated addresses. Addresses were geocoded and used to assign air pollution exposures from spatiotemporal statistical models.
RESULTS	Among participants living in metropolitan areas of the northeastern and midwestern United States, there were 3,784 incident cases of DM in the NHS, and 688 cases in the HPFS. Pooled results from random effects meta-analysis of cohort-specific models adjusted for body mass index and other known risk factors produced hazard ratios (HRs) for incident DM with interquartile range (IQR) increases in average PM during the 12 months before diagnosis of 1.03 [95% confidence interval (CI), 0.96-1.10] for PM2.5, 1.04 (95% CI, 0.99-1.09) for PM10, and 1.04 (95% CI, 0.99-1.09) for PM10-2.5. Among women, the fully adjusted HR for living < 50 m versus ≥ 200 m from a roadway was 1.14 (95% CI, 1.03-1.27).
CONCLUSIONS	Overall, results did not provide strong evidence of an association between exposure to PM in the previous 12 months and incident DM; however, an association with distance to road (a proxy marker of exposure to traffic-related pollution) was shown among women.

표-53. PubMed 논문번호 21131253의 내용 요약

구분	내용
PubMed ID	21131253
TITLE	Ambient air pollution and risk of congenital anomalies: a systematic review and meta-analysis.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002946
AUTHORS	Vrijheid Martine, Martinez David, Manzanares Sandra, Dadvand Payam, Schembari Anna, Rankin Judith, Nieuwenhuijsen Mark, Vrijheid Martine, Martinez David, Manzanares Sandra, Dadvand Payam, Schembari Anna, Rankin Judith, Nieuwenhuijsen Mark
OBJECTIVE	We systematically reviewed epidemiologic studies on ambient air pollution and congenital anomalies and conducted meta-analyses for a number of air pollutant-anomaly combinations.
DATA SOURCES AND EXTRACTION	From bibliographic searches we extracted 10 original epidemiologic studies that examined the association between congenital anomaly risk and concentrations of air pollutants. Meta-analyses were conducted if at least four studies published risk estimates for the same pollutant and anomaly group. Summary risk estimates were calculated for a) risk at high versus low exposure level in each study and b) risk per unit increase in continuous pollutant concentration.
DATA SYNTHESIS	Each individual study reported statistically significantly increased risks for some combinations of air pollutants and congenital anomalies, among many combinations tested. In meta-analyses, nitrogen dioxide (NO_2) and sulfur dioxide (SO_2) exposures were related to increases in risk of coarctation of the aorta [odds ratio (OR) per 10 ppb NO_2 = 1.17; 95% confidence interval (CI), 1.00-1.36; OR per 1 ppb SO_2 = 1.07; 95% CI, 1.01-1.13] and tetralogy of Fallot (OR per 10 ppb NO_2 = 1.20; 95% CI, 1.02-1.42; OR per 1 ppb SO_2 = 1.03; 95% CI, 1.01-1.05), and PM_1 □ (particulate matter ≤ 10 □m) exposure was related to an increased risk of atrial septal defects (OR per 10 $\mu g/m^3$ = 1.14; 95% CI, 1.01-1.28). Meta-analyses found no statistically significant increase in risk of other cardiac anomalies and oral clefts.
CONCLUSIONS	We found some evidence for an effect of ambient air pollutants on congenital cardiac anomaly risk. Improvements in the areas of exposure assessment, outcome harmonization, assessment of other congenital anomalies, and mechanistic knowledge are needed to advance this field.

표-54. PubMed 논문번호 21152417의 내용 요약

구분	내용
PubMed ID	21152417
TITLE	Air Pollution and the microvasculature: a cross-sectional assessment of in vivo retinal images in the population-based multi-ethnic study of atherosclerosis (MESA).
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1000372
AUTHORS	Adar Sara D, Klein Ronald, Klein Barbara E K, Szpiro Adam A, Cotch Mary Frances, Wong Tien Y, O'Neill Marie S, Shrager Sandi, Barr R Graham, Siscovick David S, Daviglus Martha L, Sampson Paul D, Kaufman Joel D
BACKGROUND	Long- and short-term exposures to air pollution, especially fine particulate matter (PM(2.5)), have been linked to cardiovascular morbidity and mortality. One hypothesized mechanism for these associations involves microvascular effects. Retinal photography provides a novel, in vivo approach to examine the association of air pollution with changes in the human microvasculature.
METHODS AND FINDINGS	Chronic and acute associations between residential air pollution concentrations and retinal vessel diameters, expressed as central retinal arteriolar equivalents (CRAE) and central retinal venular equivalents (CRVE), were examined using digital retinal images taken in Multi-Ethnic Study of Atherosclerosis (MESA) participants between 2002 and 2003. Study participants (46 to 87 years of age) were without clinical cardiovascular disease at the baseline examination (2000-2002). Long-term outdoor concentrations of PM(2.5) were estimated at each participant's home for the 2 years preceding the clinical exam using a spatio-temporal model. Short-term concentrations were assigned using outdoor measurements on the day preceding the clinical exam. Residential proximity to roadways was also used as an indicator of long-term traffic exposures. All associations were examined using linear regression models adjusted for subject-specific age, sex, race/ethnicity, education, income, smoking status, alcohol use, physical activity, body mass index, family history of cardiovascular disease, diabetes status, serum cholesterol, glucose, blood pressure, emphysema, C-reactive protein, medication use, and fellow vessel diameter. Short-term associations were further controlled for weather and seasonality. Among the 4,607 participants with complete data, CRAE were found to be narrower among persons residing in regions with increased long- and short-term levels of PM(2.5). These relationships were observed in a joint exposure model with −0.8 □m (95% confidence interval [CI] −1.1 to −0.5) and −0.4 □m (95% CI −0.8 to 0.1) decreases in CRAE per interquartile increases in long- (3 □g/m(3)) and short-term (9 □g/m(3)) PM(2.5) levels, respectively. These reductions in CRAE are equivalent to 7- and 3-year increases in age in the same cohort. Similarly, living near a major road was also associated with a −0.7 □m decrease (95% CI −1.4 to 0.1) in CRAE. Although the chronic association with CRAE was largely influenced by differences in exposure between cities, this relationship was generally robust to control for city-level covariates and no significant differences were observed between cities. Wider CRVE were associated with living in areas of higher PM(2.5) concentrations, but these findings were less robust and not supported by the presence of consistent acute associations with PM(2.5).
CONCLUSIONS	Residing in regions with higher air pollution concentrations and experiencing daily increases in air pollution were each associated with narrower retinal arteriolar diameters in older individuals. These findings support the hypothesis that important vascular phenomena are associated with small increases in short-term or long-term air pollution exposures, even at current exposure levels, and further corroborate reported associations between air pollution and

표-55. PubMed 논문번호 21167480의 내용 요약

구분	내용
PubMed ID	21167480
TITLE	Local determinants of road traffic noise levels versus determinants of air pollution levels in a Mediterranean city.
JOURNAL	Environmental research: 10.1016/j.envres.2010.10.013
AUTHORS	Foraster Maria, Deltell Alexandre, Basaga□a Xavier, Medina-Ram□n Mercedes, Aguilera Inmaculada, Bouso Laura, Grau Mar□a, Phuleria Harish C, Rivera Marcela, Slama R□my, Sunyer Jordi, Targa Jaume, K□nzli Nino, Foraster Maria, Deltell Alexandre, Basaga□a Xavier, Medina-Ram□n Mercedes, Aguilera Inmaculada, Bouso Laura, Grau Mar□a, Phuleria Harish C, Rivera Marcela, Slama R□my, Sunyer Jordi, Targa Jaume, K□nzli Nino
BACKGROUND	Both traffic-related noise and air pollution have been associated with cardiovascular disease (CVD). Spatial correlations between these environmental stressors may entail mutual confounding in epidemiological studies investigating their long-term effects. Few studies have investigated their correlation - none in Spain - and results differ among cities.
OBJECTIVES	We assessed the contribution of urban land-use and traffic variables to the noise-air pollution correlation in Girona town, where an investigation of the chronic effects of air pollution and noise on CVD takes place (REGICOR-AIR).
METHODOLOGY	Outdoor annual mean concentrations of nitrogen dioxide (NO(2)) derived from monthly passive sampler measurements were obtained at 83 residential locations. Long-term average traffic-related noise levels from a validated model were assigned to each residence. Linear regression models were fitted both for NO(2) and noise.
RESULTS	The correlation between NO(2) and noise (L(24h)) was 0.62. However, the correlation differed across the urban space, with lower correlations at sites with higher traffic density and in the modern downtown. Traffic density, distance from the location to the sidewalk and building density nearby explained 35.6% and 73.2% of the variability of NO(2) and noise levels, respectively. The correlation between the residuals of the two models suggested the presence of other unmeasured common variables.
CONCLUSIONS	The substantial correlation between traffic-related noise and NO(2), endorsed by common determinants, and the dependence of this correlation on complex local characteristics call for careful evaluations of both factors to ultimately assess their cardiovascular effects.

표-56. PubMed 논문번호 21172696의 내용 요약

구분	내용
PubMed ID	21172696
TITLE	Outdoor temperature is associated with serum HDL and LDL.
JOURNAL	Environmental research: 10.1016/j.envres.2010.12.001
AUTHORS	Halonen Jaana I, Zanobetti Antonella, Sparrow David, Vokonas Pantel S, Schwartz Joel, Halonen Jaana I, Zanobetti Antonella, Sparrow David, Vokonas Pantel S, Schwartz Joel
BACKGROUND	While exposures to high and low air temperatures are associated with cardiovascular mortality, the underlying mechanisms are poorly understood. The risk factors for cardiovascular disease include high levels of total cholesterol and low-density lipoprotein (LDL), and low levels of high-density lipoprotein (HDL). We investigated whether temperature was associated with changes in circulating lipid levels, and whether this might explain part of the association with increased cardiovascular events.
METHODS	The study cohort consisted of 478 men in the greater Boston area with a mean age of 74.2 years. They visited the clinic every 3-5 years between 1995 and 2008 for physical examination and to complete questionnaires. We excluded from analyses all men taking statin medication and all days with missing data, resulting in a total of 862 visits. Associations between three temperature variables (ambient, apparent, and dew point temperature) and serum lipid levels (total cholesterol, HDL, LDL, and triglycerides) were studied with linear mixed models that included possible confounders such as air pollution and a random intercept for each subject.
RESULTS	We found that HDL decreased -1.76% (95% CI: from -3.17 to -0.32, lag 2 days), and -5.58% (95% CI: from -8.87 to -2.16, moving average of 4 weeks) for each 5° C increase in mean ambient temperature. For the same increase in mean ambient temperature, LDL increased by 1.74% (95% CI: 0.07-3.44, lag 1 day) and 1.87% (95% CI: 0.14-3.63, lag 2 days). These results were also similar for apparent and dew point temperatures. No changes were found in total cholesterol or triglycerides in relation to temperature increase.
CONCLUSIONS	Changes in HDL and LDL levels associated with an increase in ambient temperature may be among the underlying mechanisms of temperature-related cardiovascular mortality.

표-57. PubMed 논문번호 21180892의 내용 요약

구분	내용
PubMed ID	21180892
TITLE	Sidestream cigarette smoke exposure effects on baroreflex in adult rats.
JOURNAL	Arquivos brasileiros de cardiologia: 10.1590/s0066-782x2010005000164
AUTHORS	Valenti Vitor E, Abreu Luiz Carlos de, Ferreira Celso, Valenti Vitor E, Abreu Luiz Carlos de, Ferreira Celso
BACKGROUND	It has been evidenced in the literature that exposure to cigarette smoke causes hypertension in rats; however, it has not been demonstrated if the baroreflex function is impaired before the animal becomes hypertensive.
OBJECTIVE	We evaluated short-term effects of sidestream cigarette smoke (SSCS) exposure on baroreflex function in Wistar normotensive rats.
METHODS	Rats were exposed to SSCS during three weeks, 180 minutes, five days per week, at a concentration of monoxide carbon between 100-300 ppm. Mean arterial pressure (MAP) and heart rate (HR) were evaluated through cannulation of the femoral vein and artery.
RESULTS	There was no significant difference between control and SSCS groups regarding basal mean arterial pressure and heart rate, sympathetic and parasympathetic components of the baroreflex function.
CONCLUSION	Our data suggest that three weeks of exposure to SSCS is not enough to significantly impair cardiovascular parameters and baroreflex sensitivity in normotensive Wistar rats.

표-58. PubMed 논문번호 21211691의 내용 요약

구분	내용
PubMed ID	21211691
TITLE	The effect of air pollution on spatial dispersion of myocardial repolarization in healthy human volunteers.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2010.08.625
AUTHORS	Sivagangabalan Gopal, Spears Danna, Masse Stephane, Urch Bruce, Brook Robert D, Silverman Frances, Gold Diane R, Lukic Karl Z, Speck Mary, Kusha Marjan, Farid Talha, Poku Kwaku, Shi Evelyn, Floras John, Nanthakumar Kumaraswamy
OBJECTIVES	We tested the hypothesis that exposure to concentrated ambient particles (CAP) and/or ozone (O(3)) would increase dispersion of ventricular repolarization.
BACKGROUND	Elevated levels of air pollution are associated with cardiac arrhythmias through mechanisms yet to be elucidated.
METHODS	Each of 25 volunteers (18 to 50 years of age) had four 2-h exposures to 150 μg/m(3) CAP; 120 parts per billion O(3); CAP + O(3); and filtered air (FA). Exposure-induced changes (Δ = 5-min epochs at end-start) in spatial dispersion of repolarization were determined from continuous 12-lead electrocardiographic recording.
RESULTS	Spatial dispersion of repolarization assessed by corrected ΔT-wave peak to T-wave end interval increased significantly for CAP + O(3) (0.17 ± 0.03, p < 0.0001) exposure only, remaining significant when factoring FA (CAP + O(3) - FA) as control (0.11 ± 0.04, p = 0.013). The influence on repolarization was further verified by a significant increase in ΔQT dispersion (for CAP + O(3) compared with FA (5.7 ± 1.4, p = 0.0002). When the low-frequency to high-frequency ratio of heart rate variability (a conventional representation of sympathetic-parasympathetic balances) was included as a covariate, the effect estimate was positive for both corrected ΔT-wave peak to T-wave end interval (p = 0.002) and ΔQT dispersion (p = 0.038). When the high-frequency component (parasympathetic heart rate modulation) was included as a covariate with corrected ΔT-wave peak to T-wave end interval, the effect estimate for high frequency was inverse (p = 0.02).
CONCLUSIONS	CAP + O(3) exposure alters dispersion of ventricular repolarization in part by increasing sympathetic and decreasing parasympathetic heart rate modulation. Detection of changes in repolarization parameters, even in this small cohort of healthy individuals, suggests an underappreciated role for air pollutants in urban arrhythmogenesis.

표-59. PubMed 논문번호 21245016의 내용 요약

구분	내용
PubMed ID	21245016
TITLE	Long-term exposure to airborne particles and arterial stiffness: the Multi-Ethnic Study of Atherosclerosis (MESA).
JOURNAL	Environmental health perspectives: 10.1289/ehp.0901524
AUTHORS	O'Neill Marie S, Diez-Roux Ana V, Auchincloss Amy H, Shen Mingwu, Lima Jo□o A, Polak Joseph F, Barr R Graham, Kaufman Joel, Jacobs David R
BACKGROUND	Increased arterial stiffness could represent an intermediate subclinical outcome in the mechanistic pathway underlying associations between average long-term pollution exposure and cardiovascular events.
OBJECTIVE	We hypothesized that 20 years of exposure to particulate matter (PM) ≤ 2.5 and 10 μm in aerodynamic diameter (PM2.5 and PM10, respectively) would be positively associated with arterial stiffness in 3,996 participants from the Multi-Ethnic Study of Atherosclerosis (MESA) who were seen at six U.S. study sites.
METHODS	We assigned pollution exposure during two decades preceding a clinical exam (2000-2002) using observed PM10 from monitors nearest participants' residences and PM10 and PM2.5 imputed from a space-time model. We examined three log-transformed arterial stiffness outcome measures: Young's modulus (YM) from carotid artery ultrasound and large (C1) and small (C2) artery vessel compliance from the radial artery pulse wave. All associations are expressed per 10 μg/m3 increment in PM and were adjusted for weather, age, sex, race, glucose, triglycerides, diabetes, waist:hip ratio, seated mean arterial pressure, smoking status, pack-years, cigarettes per day, environmental tobacco smoke, and physical activity. C1 and C2 models were further adjusted for heart rate, weight, and height.
RESULTS	Long-term average particle exposure was not associated with greater arterial stiffness measured by YM, C1, or C2, and the few associations observed were not robust across metrics and adjustment schemes.
CONCLUSIONS	Long-term particle mass exposure did not appear to be associated with greater arterial stiffness in this study sample.

표-60. PubMed 논문번호 21255249의 내용 요약

구분	내용
PubMed ID	21255249
TITLE	Traffic exposure and incident venous thromboembolism in the Atherosclerosis Risk in Communities (ARIC) Study.
JOURNAL	Journal of thrombosis and haemostasis : JTH: 10.1111/j.1538-7836.2011.04210.x
AUTHORS	Kan H, Folsom A R, Cushman M, Rose K M, Rosamond W D, Liao D, Lurmann F, London S J
BACKGROUND	Two recent case-control studies in Italy reported that long-term exposure to particulate air pollution or living near major traffic roads was associated with an increased risk of deep vein thrombosis (DVT). No prospective evidence exists on the possible association between long-term traffic-related air pollution and incident venous thromboembolism (VTE).
OBJECTIVES	To examine the association between long-term traffic exposure and incident VTE in a population-based prospective cohort study.
METHODS	We studied 13,143 middle-aged men and women in the Atherosclerosis Risk in Communities Study without a history of DVT or pulmonary embolism at baseline examination (1987-1989). The Geographical Information System-mapped traffic density and distance to major roads in the four study communities served as measures of traffic exposure. We examined the association between traffic exposure and incident VTE with proportional hazards regression models.
RESULTS	A total of 405 subjects developed VTE in 2005. Traffic density was not significantly associated with VTE. Relative to those in the lowest quartile of traffic density, the adjusted hazard ratios across increasing quartiles were 1.18 (95% confidence interval [CI] 0.88-1.57), 0.99 (95% CI 0.74-1.34) and 1.14 (95% CI 0.86-1.51) (P-value for trend across quartiles = 0.64). For residents living within 150 m of major roads, as compared with subjects living further away, the adjusted hazard ratio was 1.16 (95% CI 0.95-1.42, P = 0.14).
CONCLUSIONS	This first prospective study in the general population does not support an association between air pollution exposure or traffic proximity and risk of DVT. More data may be needed to clarify whether traffic or air pollution influences the risk of VTE.

표-61. PubMed 논문번호 21257787의 내용 요약

구분	내용
PubMed ID	21257787
TITLE	An air filter intervention study of endothelial function among healthy adults a woodsmoke-impacted community.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201010-1572OC
AUTHORS	Allen Ryan W, Carlsten Chris, Karlen Barbara, Leckie Sara, van Eeden Stephan, Vedal Sverre, Wong Imelda, Brauer Michael
RATIONALE	Particulate air pollution is associated with cardiovascular morbidity. One hypothesized mechanism involves oxidative stress, systemic inflammation, an endothelial dysfunction.
OBJECTIVES	To assess an intervention's impact on particle exposures and endothelial function among healthy adults in a woodsmoke-impacted community. We also investigated the underlying role of oxidative stress and inflammation in relation to exposure reductions.
METHODS	Portable air filters were used in a randomized crossover intervention study of 45 healthy adults exposed to consecutive 7-day periods of filtered and nonfiltered air.
MEASUREMENTS AND MAIN RESULTS	Reactive hyperemia index was measured as an indicator of endothelial function via peripheral artery tonometry, and markers of inflammation (C-reactive protein, interleukin-6, and band cells) and lipid peroxidation (malondialdehyde and 8-iso-prostaglandin F(2α)) were quantified. Air filters reduced indoor fine particle concentrations by 60%. Filtration was associated with a 9.4% (95% confidence interval, 0.9-18%) increase in reactive hyperemia index and a 32.6% (4.4-60.9%) decrease in C-reactive protein. Decreases in particulate matter and the woodsmoke tracer levoglucosan were associated with reduced band cell counts. There was limited evidence of more pronounced effects on endothelial function and leve of systemic inflammation among males, overweight participants, younger participants, and residents of wood-burning homes. No associations were noted for oxidative stress markers.
CONCLUSIONS	Air filtration was associated with improved endothelial function and decrease concentrations of inflammatory biomarkers but not markers of oxidative stress. Our results support the hypothesis that systemic inflammation and impaired endothelial function, both predictors of cardiovascular morbidity, can be favorably influenced by reducing indoor particle concentrations.

표-62. PubMed 논문번호 21266374의 내용 요약

구분	내용
PubMed ID	21266374
TITLE	Road traffic noise and stroke: a prospective cohort study.
JOURNAL	European heart journal: 10.1093/eurheartj/ehq466
AUTHORS	S ø rensen Mette, Hvidberg Martin, Andersen Zorana J, Nordsborg Rikke B, Lillelund Kenneth G, Jakobsen J ø rgen, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
AIMS	Epidemiological studies suggest that long-term exposure to road traffic noise increases the risk of cardiovascular disorders. The aim of this study was to investigate the relation between exposure to road traffic noise and risk for stroke, which has not been studied before.
METHODS AND RESULTS	In a population-based cohort of 57,053 people, we identified 1881 cases of first-ever stroke in a national hospital register between 1993-1997 and 2006. Exposure to road traffic noise and air pollution during the same period was estimated for all cohort members from residential address history. Associations between exposure to road traffic noise and stroke incidence were analysed in a Cox regression model with stratification for gender and calendar-year and adjustment for air pollution and other potential confounders. We found an incidence rate ratio (IRR) of 1.14 for stroke [95% confidence interval (CI): 1.03-1.25] per 10 dB higher level of road traffic noise (L(den)). There was a statistically significant interaction with age (P < 0.001), with a strong association between road traffic noise and stroke among cases over 64.5 years (IRR: 1.27; 95% CI: 1.13-1.43) and no association for those under 64.5 years (IRR: 1.02; 95% CI: 0.91-1.14).
CONCLUSION	Exposure to residential road traffic noise was associated with a higher risk for stroke among people older than 64.5 years of age.

표-63. PubMed 논문번호 21353301의 내용 요약

구분	내용
PubMed ID	21353301
TITLE	Public health importance of triggers of myocardial infarction: a comparative risk assessment.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(10)62296-9
AUTHORS	Nawrot Tim S, Perez Laura, K□nzli Nino, Munters Elke, Nemery Benoit
BACKGROUND	Acute myocardial infarction is triggered by various factors, such as physical exertion, stressful events, heavy meals, or increases in air pollution. However, the importance and relevance of each trigger are uncertain. We compared triggers of myocardial infarction at an individual and population level.
METHODS	We searched PubMed and the Web of Science citation databases to identify studies of triggers of non-fatal myocardial infarction to calculate population attributable fractions (PAF). When feasible, we did a meta-regression analysis for studies of the same trigger.
FINDINGS	Of the epidemiologic studies reviewed, 36 provided sufficient details to be considered. In the studied populations, the exposure prevalence for triggers in the relevant control time window ranged from 0.04% for cocaine use to 100% for air pollution. The reported odds ratios (OR) ranged from 1.05 to 23.7. Ranking triggers from the highest to the lowest OR resulted in the following order: use of cocaine, heavy meal, smoking of marijuana, negative emotions, physical exertion, positive emotions, anger, sexual activity, traffic exposure, respiratory infections, coffee consumption, air pollution (based on a difference of 30 μg/m3 in particulate matter with a diameter <10 μm [PM10]). Taking into account the OR and the prevalences of exposure, the highest PAF was estimated for traffic exposure (7.4%), followed by physical exertion (6.2%), alcohol (5.0%), coffee (5.0%), a difference of 30 μg/m3 in PM10 (4.8%), negative emotions (3.9%), anger (3.1%), heavy meal (2.7%), positive emotions (2.4%), sexual activity (2.2%), cocaine use (0.9%), marijuana smoking (0.8%) and respiratory infections (0.6%). Interpretation In view of both the magnitude of the risk and the prevalence in the population, air pollution is an important trigger of myocardial infarction, it is of similar magnitude (PAF 5-7%) as other well accepted triggers such as physical exertion, alcohol, and coffee. Our work shows that ever-present small risks might have considerable public health relevance.
FUNDING	The research on air pollution and health at Hasselt University is supported by a grant from the Flemish Scientific Fund (FWO, Krediet aan navorsers/G.0873.11), tUL-impulse financing, and bijzonder onderzoeksfonds (BOF) and at the Katholieke Universiteit Leuven by the sustainable development programme of BELSPO (Belgian Science Policy).

표-64. PubMed 논문번호 21356620의 내용 요약

구분	내용
PubMed ID	21356620
TITLE	Ambient air pollution and lipoprotein-associated phospholipase A₂ in survivors of myocardial infarction.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002681
AUTHORS	Br□ske Irene, Hampel Regina, Baumg□rtner Zita, R□ckerl Regina, Greven Sonja, Koenig Wolfgang, Peters Annette, Schneider Alexandra
BACKGROUND	Increasing evidence suggests a proatherogenic role for lipoprotein-associated phospholipase A₂ (Lp-PLA2). A meta-analysis of published cohorts has shown that Lp-PLA2 is an independent predictor of coronary heart disease events and stroke.
OBJECTIVE	In this study, we investigated whether the association between air pollution and cardiovascular disease might be partly explained by increased Lp-PLA2 mass in response to exposure.
METHODS	A prospective longitudinal study of 200 patients who had had a myocardial infarction was performed in Augsburg, Germany. Up to six repeated clinical examinations were scheduled every 4-6 weeks between May 2003 and March 2004. Supplementary to the multicenter AIRGENE protocol, we assessed repeated plasma Lp-PLA2 concentrations. Air pollution data from a fixed monitoring site representing urban background concentrations were collected. We measured hourly means of particle mass [particulate matter (PM) < 10 □m (PM_{1} □) and PM < 2.5 □m (PM(2.5)) in aerodynamic diameter] and particle number concentrations (PNCs), as well as the gaseous air pollutants carbon monoxide (CO), sulfur dioxide (SO_2), ozone (O_3), nitric oxide (NO), and nitrogen dioxide (NO_2). Data were analyzed using mixed models with random patient effects.
RESULTS	Lp-PLA2 showed a positive association with PM_{1} □, PM(2.5), and PNCs, as well as with CO, NO_2, NO, and SO_2 4-5 days before blood withdrawal (lag 4-5). A positive association with O_3 was much more immediate (lag 0). However, inverse associations with some pollutants were evident at shorter time lags.
CONCLUSION	These preliminary findings should be replicated in other study populations because they suggest that the accumulation of acute and subacute effects or the chronic exposure to ambient particulate and gaseous air pollution may result in the promotion of atherosclerosis, mediated, at least in part, by increased levels of Lp-PLA2.

표-65. PubMed 논문번호 21385671의 내용 요약

구분	내용
PubMed ID	21385671
TITLE	Prolonged exposure to particulate pollution, genes associated with glutathione pathways, and DNA methylation in a cohort of older men.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002773
AUTHORS	Madrigano Jaime, Baccarelli Andrea, Mittleman Murray A, Wright Robert O, Sparrow David, Vokonas Pantel S, Tarantini Letizia, Schwartz Joel
BACKGROUND	DNA methylation is a potential pathway linking environmental exposures to disease. Exposure to particulate air pollution has been associated with increased cardiovascular morbidity and mortality, and lower blood DNA methylation has been found in processes related to cardiovascular morbidity.
OBJECTIVE	We hypothesized that prolonged exposure to particulate pollution would be associated with hypomethylation of repetitive DNA elements and that this association would be modified by genes involved in glutathione metabolism and other host characteristics.
METHODS	DNA methylation of the long interspersed nucleotide element-1 (LINE-1) and the short interspersed nucleotide element Alu were measured by quantitative polymerase chain reaction pyrosequencing in 1,406 blood samples from 706 elderly participants in the Normative Aging Study. We estimated changes in repetitive element DNA methylation associated with ambient particles (particulate matter ≤ 2.5 □m in aerodynamic diameter), black carbon (BC), and sulfates (SO_4), with mixed models. We examined multiple exposure windows (1-6 months) before DNA methylation measurement. We investigated whether this association was modified by genotype and phenotype.
RESULTS	An interquartile range (IQR) increase in BC over a 90-day period was associated with a decrease of 0.31% 5-methylcytosine (5mC) (95% confidence interval, 0.12-0.50%) in Alu. An IQR increase in SO_4 over a 90-day period was associated with a decrease of 0.27% 5mC (0.02-0.52%) in LINE-1. The glutathione S-transferase mu-1-null genotype strengthened the association between BC and Alu hypomethylation.
CONCLUSION	Prolonged exposure to BC and SO_4 particles was associated with hypomethylation of two types of repetitive elements.

표-66. PubMed 논문번호 21399501의 내용 요약

구분	내용
PubMed ID	21399501
TITLE	Fine particulate air pollution (PM2.5) and the risk of acute ischemic stroke.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e3182126580
AUTHORS	O'Donnell Martin J, Fang Jiming, Mittleman Murray A, Kapral Moira K, Wellenius Gregory A
BACKGROUND	Short-term changes in levels of fine ambient particulate matter (PM2.5) may increase the risk of acute ischemic stroke; however, results from prior studies have been inconsistent. We examined this hypothesis using data from a multicenter prospective stroke registry.
METHODS	We analyzed data from 9202 patients hospitalized with acute ischemic stroke, having a documented date and time of stroke onset, and residing within 50 km of a PM2.5 monitor in 8 cities in Ontario, Canada. We evaluated the risk of ischemic stroke onset associated with PM2.5 in each city using a time-stratified case-crossover design, matching on day of week and time of day. We then combined these city-specific estimates using random-effects meta-analysis techniques. We examined whether the effects of PM2.5 differed across strata defined by patient characteristics and ischemic stroke etiology.
RESULTS	Overall, PM2.5 was associated with a -0.7% change in ischemic stroke risk per 10- μg/m increase in PM2.5 (95% confidence interval = -6.3% to 5.1%). These overall negative results were robust to a number of sensitivity analyses. Among patients with diabetes mellitus, PM2.5 was associated with an 11% increase in ischemic stroke risk (1% to 22%). The association between PM2.5 and ischemic stroke risk varied according to stroke etiology, with the strongest associations observed for strokes due to large-artery atherosclerosis and small-vessel occlusion.
CONCLUSIONS	These results do not support the hypothesis that short-term increases in PM2.5 levels are associated with ischemic stroke risk overall. However, specific patient subgroups may be at increased risk of particulate-related ischemic strokes.

표-67. PubMed 논문번호 21406695의 내용 요약

구분	내용
PubMed ID	21406695
TITLE	The use of superoxidized aqueous solution versus saline as a replacement solution in the versajet lavage system in chronic diabetic foot ulcers: a pilot study.
JOURNAL	Journal of the American Podiatric Medical Association: 10.7547/1010124
AUTHORS	Bowling Frank L, Crews Ryan T, Salgami Eleanna, Armstrong David G, Boulton Andrew J M
BACKGROUND	The removal of necrotic tissue from chronic wounds is required for wound healing to occur. Hydrodebridement (jet lavage) and superoxidized aqueous solution have been independently used for debriding wounds. We sought to investigate the use of superoxidized aqueous solution with a jet lavage system.
METHODS	Twenty patients with diabetic foot ulcers were randomly assigned in a 1:1 ratio to receive jet lavage debridement with either superoxidized aqueous solution or standard saline weekly.
RESULTS	There was no significant difference between the two treatments in the reduction of bacterial load or wound size in 4 weeks. No adverse reactions were reported for either treatment.
CONCLUSIONS	The use of superoxidized aqueous solution for jet lavage debridement seemed to be as safe and effective as saline. Future investigations should concentrate on whether superoxidized aqueous solution may reduce the bacterial air contamination associated with hydrodebridement.

표-68. PubMed 논문번호 21415130의 내용 요약

구분	내용
PubMed ID	21415130
TITLE	The effect of secondhand smoke exposure on markers of elastin degradation.
JOURNAL	Chest: 10.1378/chest.10-2298
AUTHORS	Slowik Natalie, Ma Shuren, He Jiangtao, Lin Yong Y, Soldin Offie P, Robbins Richard A, Turino Gerard M
BACKGROUND	Tobacco smoke is a major risk factor in the development of COPD. Secondhand smoke (SHS) exposure is a known risk factor in asthma, bronchitis, and coronary artery disease. Elastin is a recognized target for injury in COPD, and the amino acids desmosine and isodesmosine (D/I), which are specific for elastin degradation, are elevated in COPD. This study determined whether exposure to SHS affects elastin degradation in asymptomatic individuals.
METHODS	Two cohorts of asymptomatic individuals without evidence of respiratory or circulatory disease, exposed to SHS, were studied. Both cohorts comprised normal nonsmokers, active smokers, and those exposed to SHS. D/I were measured in plasma and quantified by high-performance liquid chromatography and tandem mass spectrometry by published methods. Plasma cotinine, a metabolite of nicotine, was also measured.
RESULTS	In each cohort, the levels of D/I in plasma were statistically significantly higher in secondhand-smoke-exposed subjects than in the normal nonexposed subjects. Smokers had the highest levels of D/I but their levels were not statistically significantly higher than those of the secondhand-smoke-exposed. Cotinine levels were elevated in secondhand-smoke-exposed subjects and active smokers but not in most nonsmoking control subjects.
CONCLUSIONS	Results indicate a tissue matrix effect of degradation of body elastin from SHS exposure and possible lung structure injury, which may result in COPD. Long-term studies of individuals exposed to SHS for the development of COPD are warranted.

표-69. PubMed 논문번호 21422014의 내용 요약

구분	내용
PubMed ID	21422014
TITLE	Meta-analysis of the association between secondhand smoke exposure and stroke.
JOURNAL	Journal of public health (Oxford, England): 10.1093/pubmed/fdr025
AUTHORS	Oono I P, Mackay D F, Pell J P
BACKGROUND	Active smoking is a recognized risk factor for stroke. We determined the evidence for an association with secondhand smoke exposure.
METHODS	A systematic review was undertaken according to PRISMA guidelines. Random effects meta-analysis provided a pooled estimate of risk, and heterogeneity quantified using I(2) values. Potential publication and study bias were assessed using a funnel plot and Egger's test. Meta-regression analyses were used to investigate sources of heterogeneity.
RESULTS	The 20 eligible studies provided 35 estimates of risk derived from 885 307 participants, of whom 5894 (0.7%) suffered a stroke. The pooled estimate of risk was 1.25 (95% CI: 1.12-1.38) with no evidence of significant publication or small-study bias. There was moderate heterogeneity (I(2) = 54.2%, P < 0.001) but no study characteristics were statistically significant in the meta-regression analysis. There was a non-linear dose relationship. The relative risk increased from 1.16 (95% CI: 1.06-1.27) for exposure to 5 cigarettes/day to 1.56 (95% CI: 1.25-1.96) for exposure to 40 cigarettes/day.
CONCLUSIONS	There is evidence of a strong, consistent and dose-dependent association between exposure to secondhand smoke and risk of stroke, suggestive of a causal relationship, with disproportionately high risk at low levels of exposure suggesting no safe lower limit of exposure.

표-70. PubMed 논문번호 21435200의 내용 요약

구분	내용
PubMed ID	21435200
TITLE	Mortality and morbidity among people living close to incinerators: a cohort study based on dispersion modeling for exposure assessment.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-10-22
AUTHORS	Ranzi Andrea, Fano Valeria, Erspamer Laura, Lauriola Paolo, Perucci Carlo A, Forastiere Francesco
BACKGROUND	Several studies have been conducted on the possible health effects for people living close to incinerators and well-conducted reviews are available. Nevertheless, several uncertainties limit the overall interpretation of the findings. We evaluated the health effects of emissions from two incinerators in a pilot cohort study.
METHODS	The study area was defined as the 3.5 km radius around two incinerators located near Forl□ (Italy). People who were residents in 1/1/1990, or subsequently became residents up to 31/12/2003, were enrolled in a longitudinal study (31,347 individuals). All the addresses were geocoded. Follow-up continued until 31/12/2003 by linking the mortality register, cancer registry and hospital admissions databases. Atmospheric Dispersion Model System (ADMS) software was used for exposure assessment; modelled concentration maps of heavy metals (annual average) were considered the indicators of exposure to atmospheric pollution from the incinerators, while concentration maps of nitrogen dioxide (NO2) were considered for exposure to other pollution sources. Age and area-based socioeconomic status adjusted rate ratios and 95% Confidence Intervals were estimated with Poisson regression, using the lowest exposure category to heavy metals as reference.
RESULTS	The mortality and morbidity experience of the whole cohort did not differ from the regional population. In the internal analysis, no association between pollution exposure from the incinerators and all-cause and cause-specific mortality outcomes was observed in men, with the exception of colon cancer. Exposure to the incinerators was associated with cancer mortality among women, in particular for all cancer sites (RR for the highest exposure level = 1.47, 95% CI: 1.09, 1.99), stomach, colon, liver and breast cancer. No clear trend was detected for cancer incidence. No association was found for hospitalizations related to major diseases. NO2 levels, as a proxy from other pollution sources (traffic in particular), did not exert an important confounding role.
CONCLUSIONS	No increased risk of mortality and morbidity was found in the entire area. The internal analysis of the cohort based on dispersion modeling found excesses of mortality for some cancer types in the highest exposure categories, especially in women. The interpretation of the findings is limited given the pilot nature of the study.

표-71. PubMed 논문번호 21441173의 내용 요약

구분	내용
PubMed ID	21441173
TITLE	Enhancement of systemic and sputum granulocyte response to inhaled endotoxin in people with the GSTM1 null genotype.
JOURNAL	Occupational and environmental medicine: 10.1136/oem.2010.061747
AUTHORS	Dillon Madeline A, Harris Bradford, Hernandez Michelle L, Zou Baiming, Reed William, Bromberg Philip A, Devlin Robert B, Diaz-Sanchez David, Kleeberger Steven, Zhou Haibo, Lay John C, Alexis Neil E, Peden David B
OBJECTIVE	To determine if the GSTM1 null genotype is a risk factor for increased inflammatory response to inhaled endotoxin.
METHODS	35 volunteers who had undergone inhalation challenge with a 20 000 endotoxin unit dose of Clinical Center Reference Endotoxin (CCRE) were genotyped for the GSTM1 null polymorphism. Parameters of airway and systemic inflammation observed before and after challenge were compared in GSTM1 null (n=17) and GSTM1 (n=18) sufficient volunteers.
RESULTS	GSTM1 null volunteers had significantly increased circulating white blood cells (WBCs), polymorphonuclear neutrophils (PMNs), platelets and sputum PMNs (% sputum PMNs and PMNs/mg sputum) after CCRE challenge. GSTM1 sufficient volunteers had significant, but lower increases in circulating WBCs, PMNs and % sputum PMNs, and no increase in platelets or PMNs/mg sputum. Linear regression analysis adjusted for baseline values of the entire cohort revealed that the GSTM1 null genotype significantly increased circulating WBCs, platelets and % sputum PMNs after challenge.
CONCLUSION	These data support the hypothesis that the GSTM1 null genotype is a risk factor for increased acute respiratory and systemic inflammatory response to inhaled CCRE. These data are consistent with other observations that the GSTM1 null genotype is associated with increased respiratory, systemic and cardiovascular effects linked to ambient air particulate matter exposure and indicate that the GSTM1 null genotype should be considered a risk factor for adverse health effects associated with exposure to environmental endotoxin.

표-72. PubMed 논문번호 21454146의 내용 요약

구분	내용
PubMed ID	21454146
TITLE	Particulate matter exposures, mortality, and cardiovascular disease in the health professionals follow-up study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002921
AUTHORS	Puett Robin C, Hart Jaime E, Suh Helen, Mittleman Murray, Laden Francine
BACKGROUND	The association of all-cause mortality and cardiovascular outcomes with air pollution exposures has been well established in the literature. The number of studies examining chronic exposures in cohorts is growing, with more recent studies conducted among women finding risk estimates of greater magnitude. Questions remain regarding sex differences in the relationship of chronic particulate matter (PM) exposures with mortality and cardiovascular outcomes.
OBJECTIVES	In this study we explored these associations in the all-male Health Professionals Follow-Up Study prospective cohort.
METHODS	The same spatiotemporal exposure estimation models, similar outcomes, and biennially updated covariates were used as those previously applied in the female Nurses' Health Study cohort.
RESULTS	Among 17,545 men residing in the northeastern and midwestern United States, there were 2,813 deaths, including 746 cases of fatal coronary heart disease (CHD). An interquartile range change (4 □g/m3) in average exposure to PM ≤ 2.5 □m in diameter in the 12 previous months was not associated with all-cause mortality [hazard ratio (HR) = 0.94; 95% confidence interval (CI), 0.87-1.00] or fatal CHD (HR = 0.99; 95% CI, 0.87-1.13) in fully adjusted models. Findings were similar for separate models of exposure to PM ≤ 10 □m in diameter and PM between 2.5 and 10 □m in diameter and for copollutant models.
CONCLUSIONS	Among this cohort of men with high socioeconomic status living in the midwestern and northeastern United States, the results did not support an association of chronic PM exposures with all-cause mortality and cardiovascular outcomes in models with time-varying covariates. Whether these findings suggest sex differences in susceptibility or the protective impact of healthier lifestyles and higher socioeconomic status requires additional investigation.

표-73. PubMed 논문번호 21465749의 내용 요약

구분	내용
PubMed ID	21465749
TITLE	Annual ambient black carbon associated with shorter telomeres in elderly men: Veterans Affairs Normative Aging Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.0901831
AUTHORS	McCracken John, Baccarelli Andrea, Hoxha Mirjam, Dioni Laura, Melly Steve, Coull Brent, Suh Helen, Vokonas Pantel, Schwartz Joel
BACKGROUND	Telomere length reflects biological age and is inversely associated with risk of cardiovascular disease (CVD). Ambient air pollution is associated with CVD, but its effect on telomere length is unknown.
OBJECTIVE	We investigated whether ambient black carbon (BC), a marker for traffic-related particles, is associated with telomere length in the Normative Aging Study (NAS).
METHODS	Among 165 never-smoking men from the NAS, leukocyte telomere length (LTL) was measured repeatedly approximately every 3 years from 1999 through 2006 using quantitative real-time polymerase chain reaction (qRT-PCR). BC concentration at their residences during the year before each LTL measurement was estimated based on a spatiotemporal model calibrated with BC measurements from 82 locations within the study area.
RESULTS	The median [interquartile range (IQR)] annual moving-average BC concentration was 0.32 (0.20-0.45) microg/m3. LTL, expressed as population-standardized ratio of telomere repeat to single-copy gene copy numbers, had a geometric mean (geometric SD) of 1.25 (1.42). We used linear mixed-effects models including random subject intercepts and adjusted for several potential confounders. We used inverse probability of response weighting to adjust for potential selection bias due to loss to follow-up. An IQR increase in annual BC (0.25 microg/m3) was associated with a 7.6% decrease (95% confidence interval, -12.8 to -2.1) in LTL. We found evidence of effect modification, with a stronger association among subjects 〉 or = 75 years of age compared with younger participants (p = 0.050) and statin medications appearing protective of the effects of BC on LTL (p = 0.050).
CONCLUSIONS	Telomere attrition, linked to biological aging, may be associated with long-term exposures to airborne particles, particularly those rich in BC, which are primarily related to automobile traffic.

표-74. PubMed 논문번호 21573961의 내용 요약

구분	내용
PubMed ID	21573961
TITLE	Effects of fine particulate on heart rate variability in Beijing: a panel study of healthy elderly subjects.
JOURNAL	International archives of occupational and environmental health: 10.1007/s00420-011-0646-3
AUTHORS	Jia Xiaofeng, Song Xiaoming, Shima Masayuki, Tamura Kenji, Deng Furong, Guo Xinbiao, Jia Xiaofeng, Song Xiaoming, Shima Masayuki, Tamura Kenji, Deng Furong, Guo Xinbiao
PURPOSE	This study aims to investigate the effects of ambient fine particulate (particulate matter with an aerodynamic diameter of 2.5 μm or less, PM(2.5)) exposure within several minutes on Heart Rate Variability (HRV) of the healthy elderly subjects in the general environments (indoor and outdoor).
METHODS	This study is conducted by measuring the real-time indoor and outdoor exposure variables (PM(2.5), Temperature, and relative humidity) and heart rate variability (HRV), a marker of cardiac autonomic function measured by 24-h ambulatory electrocardiogram monitoring in a panel of 30 healthy elderly subjects in Beijing. Associations between personal 5-min PM(2.5) concentrations and concurrent 5-min HRV frequency indices are investigated using the mixed linear model.
RESULTS	High Frequency (HF) and Low Frequency (LF) increase, respectively by 1.30% (95% CI, 0.16-2.45%) and 1.34% (95% CI, 0.38-2.30%) per 10 μg/m(3) increases of PM(2.5) in the polled data analysis after the potential confounders are adjusted. When the indoor and outdoor periods are separated, positive associations are found between PM(2.5) and HRV when the subjects are indoors; however, there is no association when the subjects are outdoors.
CONCLUSIONS	We conclude that PM(2.5) exposure within several minutes leads to increases of HRV of the healthy older subjects, which may increase the cardiac risks. Prominent effect of PM(2.5) on HRV is found when they are indoors, while the effect is not obvious in outdoor environment.

표-75. PubMed 논문번호 21600003의 내용 요약

구분	내용
PubMed ID	21600003
TITLE	Ambient pollutants, polymorphisms associated with microRNA processing and adhesion molecules: the Normative Aging Study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-10-45
AUTHORS	Wilker Elissa H, Alexeeff Stacey E, Suh Helen, Vokonas Pantel S, Baccarelli Andrea, Schwartz Joel
BACKGROUND	Particulate air pollution has been associated with cardiovascular morbidity and mortality, but it remains unclear which time windows and pollutant sources are most critical. MicroRNA (miRNA) is thought to be involved in cardiovascular regulation. However, little is known about whether polymorphisms in genes that process microRNAs influence response to pollutant exposure. We hypothesized that averaging times longer than routinely measured one or two day moving averages are associated with higher soluble intercellular adhesion molecule-1 (sICAM-1) and vascular cell adhesion molecule-1 (sVCAM-1) levels, and that stationary and mobile sources contribute differently to these effects. We also investigated whether single nucleotide polymorphisms (SNPs) in miRNA-processing genes modify these associations.
METHODS	sICAM-1 and sVCAM-1 were measured from 1999-2008 and matched to air pollution monitoring for fine particulate matter (PM2.5) black carbon, and sulfates (SO42-). We selected 17 SNPs in five miRNA-processing genes. Mixed-effects models were used to assess effects of pollutants, SNPs, and interactions under recessive inheritance models using repeated measures.
RESULTS	723 participants with 1652 observations and 1-5 visits were included in our analyses for black carbon and PM2.5. Sulfate data was available for 672 participants with 1390 observations. An interquartile range change in seven day moving average of PM2.5 (4.27 μg/m3) was associated with 3.1% (95%CI: 1.6, 4.6) and 2.5% (95%CI: 0.6, 4.5) higher sICAM-1 and sVCAM-1. Interquartile range changes in sulfates (1.39 μg/m3) were associated with 1.4% higher (95%CI: 0.04, 2.7) and 1.6% (95%CI: -0.4, 3.7) higher sICAM-1 and sVCAM-1 respectively. No significant associations were observed for black carbon. In interaction models with PM2.5, both sICAM-1 and sVCAM-1 levels were lower in rs1062923 homozygous carriers. These interactions remained significant after multiple comparisons adjustment.
CONCLUSIONS	PM2.5 seven day moving averages are associated with higher sICAM-1 and sVCAM-1 levels. SO4-2 seven day moving averages are associated with higher sICAM-1 and a suggestive association was observed with sVCAM-1 in aging men. SNPs in miRNA-processing genes may modify associations between ambient pollution and sICAM-1 and sVCAM-1, which are correlates of atherosclerosis and cardiovascular disease.

표-76. PubMed 논문번호 21623477의 내용 요약

구분	내용
PubMed ID	21623477
TITLE	Incidence of acute myocardial infarction after implementation of a public smoking ban in Graub□nden, Switzerland: two year follow-up.
JOURNAL	Swiss medical weekly: 10.4414/smw.2011.13206
AUTHORS	Bonetti Piero O, Trachsel Lukas D, Kuhn Max U, Schulzki Thomas, Erne Paul, Radovanovic Dragana, Reinhart Walter H
QUESTION UNDER STUDY	In the first year after implementation of a public smoking ban a significant decrease in the incidence of acute myocardial infarction (AMI) was observed in Graub□nden. In the present study we analyzed the incidence of AMI in the second year of the ban. In addition, we investigated the contribution of smoking ban-unrelated factors to the reduced incidence of AMI incidence observed after enactment of the ban.
METHODS	Data of all AMI patients who underwent coronary angiography at the Kantonsspital Graub□nden, the only tertiary care hospital with a cardiac catheterization laboratory in Graub□nden, between March 1st, 2009 and February 28th, 2010 were collected prospectively. Data were compared with those of the three preceding 12-month periods. We also estimated AMI incidence during the corresponding time period in Lucerne, a region with no smoke-free legislation, using data of the AMIS Plus registry. The influence of outdoor air pollution was analyzed with the help of official measurements of PM(10)- and NO(2)-concentrations in Graub□nden. The prescription of lipid-lowering drugs was estimated by using sales figures in Graub□nden and Lucerne.
RESULTS	In Graub□nden, the number of patients with AMI in the second year after adoption of the smoking ban was similar to that in the first year of the ban (188 vs. 183; P = ns) and significantly lower than in each of the two years preceding the ban (229 and 242, respectively; P 〈0.05 vs. each of the 12-month periods after the ban). Overall, the number of AMI patients in the two post-ban years was 21% lower than in the two pre-ban years. The reduction in the number of patients with AMI was most pronounced in non-smokers and individuals with known coronary artery disease. During the corresponding time period, no similar decrease in the incidence of AMI was observed in Lucerne. No association was found between the magnitude of outdoor air pollution and the incidence of AMI. During the observation period, the use of lipid-lowering drugs increased similarly in Graub□nden and Lucerne.
CONCLUSIONS	Compared with the two years preceding the implementation of a smoking ban, the incidence of AMI remained significantly reduced in the second year of the ban in Graub□nden, whereas no similar reduction was seen in a comparable area without smoke-free legislation. Changes in outdoor air pollution or the use of lipid-lowering drugs did not substantially contribute to the decrease in the incidence of AMI that occurred after adoption of the ban in Graub□nden.

표-77. PubMed 논문번호 21649609의 내용 요약

구분	내용
PubMed ID	21649609
TITLE	The gender gap in stroke: a meta-analysis.
JOURNAL	Acta neurologica Scandinavica: 10.1111/j.1600-0404.2011.01514.x
AUTHORS	Giralt D, Domingues-Montanari S, Mendioroz M, Ortega L, Maisterra O, Perea-Gainza M, Delgado P, Rosell A, Montaner J, Giralt D, Domingues-Montanari S, Mendioroz M, Ortega L, Maisterra O, Perea-Gainza M, Delgado P, Rosell A, Montaner J
UNLABELLED	Several studies have indicated that gender differences might exist in stroke.
OBJECTIVES AND METHODS	Our goal was to perform a comprehensive meta-analysis in order to evaluate and quantify stroke gender disparities through a systematic search of relevant articles published up to October 2009 and addressing gender related differences in ischemic stroke risk factors, stroke subtype and severity, diagnostic tests, and acute phase and secondary prevention treatments.
RESULTS	Forty-five articles were included in the analysis, representing a total of 673,935 patients. Women were globally older than men (+5.2 years) and suffered more hypertension (P = 0.017) and atrial fibrillation (P < 0.001), although they were less likely to drink alcohol (P < 0.001), smoke cigarettes (P < 0.001), present hyperlipidemia (P = 0.033) or diabetes (P = 0.003) than men. Baseline stroke severity was not different between genders. Women suffered more cardioembolic strokes, while men had more atherothrombotic strokes. Moreover, women were less likely to receive stroke-related treatments, such as antiplatelets (P < 0.001), statins (P < 0.001), and tPA (P < 0.001) than men. Although meta-regression did not identify age or stroke etiology as sources of heterogeneity, caution should be taken as that analysis was possible only for gender differences in secondary prevention with antiplatelets because of limited data for other end points.
CONCLUSIONS	Gender differences have been identified on the risk factors profile and diagnostic and therapeutic management of patients with ischemic stroke. Active measures should thus be taken to avoid bias in clinical practice.

표-78. PubMed 논문번호 21652290의 내용 요약

구분	내용
PubMed ID	21652290
TITLE	Impact of reduced maternal exposures to wood smoke from an introduced chimney stove on newborn birth weight in rural Guatemala.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002928
AUTHORS	Thompson Lisa M, Bruce Nigel, Eskenazi Brenda, Diaz Anaite, Pope Daniel, Smith Kirk R
BACKGROUND	A growing body of evidence indicates a relationship between household indoor air pollution from cooking fires and adverse neonatal outcomes, such as low birth weight (LBW), in resource-poor countries.
OBJECTIVE	We examined the effect of reduced wood smoke exposure in pregnancy on LBW of Guatemalan infants in RESPIRE (Randomized Exposure Study of Pollution Indoors and Respiratory Effects).
METHODS	Pregnant women (n = 266) either received a chimney stove (intervention) or continued to cook over an open fire (control). Between October 2002 and December 2004 we weighed 174 eligible infants (69 to mothers who used a chimney stove and 105 to mothers who used an open fire during pregnancy) within 48 hr of birth. Multivariate linear regression and adjusted odds ratios (ORs) were used to estimate differences in birth weight and LBW (< 2,500 g) associated with chimney-stove versus open-fire use during pregnancy.
RESULTS	Pregnant women using chimney stoves had a 39% reduction in mean exposure to carbon monoxide compared with those using open fires. LBW prevalence was high at 22.4%. On average, infants born to mothers who used a stove weighed 89 g more [95% confidence interval (CI), -27 to 204 g] than infants whose mothers used open fires after adjusting for maternal height, diastolic blood pressure, gravidity, and season of birth. The adjusted OR for LBW was 0.74 (95% CI, 0.33-1.66) among infants of stove users compared with open-fire users. Average birth weight was 296 g higher (95% CI, 109-482 g) in infants born during the cold season (after harvest) than in other infants; this unanticipated finding may reflect the role of maternal nutrition on birth weight in an impoverished region.
CONCLUSIONS	A chimney stove reduced wood smoke exposures and was associated with reduced LBW occurrence. Although not statistically significant, the estimated effect was consistent with previous studies.

표-79. PubMed 논문번호 21654409의 내용 요약

구분	내용
PubMed ID	21654409
TITLE	Development of a French epidemiological surveillance system of workers producing or handling engineered nanomaterials in the workplace.
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0b013e31821b1d68
AUTHORS	Boutou-Kempf Odile, Marchand Jean-Luc, Radauceanu Anca, Witschger Olivier, Imbernon Ellen
OBJECTIVE	Concern has been raised about the potential impact of nanomaterials exposure on human health, and France has decided to implement a timely epidemiological surveillance tool of workers likely to be exposed to engineered nanomaterials that could accompany the development of nanotechnologies.
METHODS	A comprehensive review of the toxicological and epidemiological literature has been conducted together with an exploratory study among French companies producing or handling nanoobjects.
RESULTS	A double surveillance system is proposed consisting of a prospective cohort survey and repeated cross-sectional studies. The aim of the cohort is (1) to monitor long-term health effects and (2) to allow of further research. Setting-up an exposure registry is the first planned step.
CONCLUSIONS	The protocol is about to be submitted to the French Government for approval and funding.

표-80. PubMed 논문번호 21654419의 내용 요약

구분	내용
PubMed ID	21654419
TITLE	Epidemiologic challenges for studies of occupational exposure to engineered nanoparticles; a commentary.
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0b013e31821bde98
AUTHORS	Eisen Ellen A, Costello Sadie, Chevrier Jonathan, Picciotto Sally
OBJECTIVE	Identify most likely health effects of occupational exposure to engineered nanoparticles (ENP). Recommend analytic approaches to address epidemiologic challenges.
METHODS	Review air pollution and occupational literature on health effects of fine particulate matter (PM). Provide example of mortality study of exposure to PM composed of metalworking fluid. Apply standard Cox models and g-estimation to adjust for potential healthy worker survival effect (HWSE).
RESULTS	In contrast with standard methods, g-estimation suggests that exposure to PM may cause chronic heart and lung disease; longer exposure reduces survival. HWSE appears stronger for chronic disease than for cancer.
CONCLUSIONS	We recommend hazard surveillance, short-term panel studies of biomarkers, and prospective cohort studies of cardiovascular and respiratory diseases. Building research capacity in g-estimation methods to reduce HWSE is necessary for future studies of chronic disease and ENP.

표-81. PubMed 논문번호 21659039의 내용 요약

구분	내용
PubMed ID	21659039
TITLE	Three measures of forest fire smoke exposure and their associations with respiratory and cardiovascular health outcomes in a population-based cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002288
AUTHORS	Henderson Sarah B, Brauer Michael, Macnab Ying C, Kennedy Susan M
BACKGROUND	During the summer of 2003 numerous fires burned in British Columbia, Canada.
OBJECTIVES	We examined the associations between respiratory and cardiovascular physician visits and hospital admissions, and three measures of smoke exposure over a 92-day study period (1 July to 30 September 2003).
METHODS	A population-based cohort of 281,711 residents was identified from administrative data. Spatially specific daily exposure estimates were assigned to each subject based on total measurements of particulate matter (PM) ≤ 10 μm in aerodynamic diameter (PM10) from six regulatory tapered element oscillating microbalance (TEOM) air quality monitors, smoke-related PM10 from a CALPUFF dispersion model run for the study, and a SMOKE exposure metric for plumes visible in satellite images. Logistic regression with repeated measures was used to estimate associations with each outcome.
RESULTS	The mean (± SD) exposure based on TEOM-measured PM10 was 29 ± 31 μg/m3, with an interquartile range of 14-31 μg/m3. Correlations between the TEOM, smoke, and CALPUFF metrics were moderate (0.37-0.76). Odds ratios (ORs) for a 30- μg/m3 increase in TEOM-based PM10 were 1.05 [95% confidence interval (CI), 1.03-1.06] for all respiratory physician visits, 1.16 (95% CI, 1.09-1.23) for asthma-specific visits, and 1.15 (95% CI, 1.00-1.29) for respiratory hospital admissions. Associations with cardiovascular outcomes were largely null.
CONCLUSIONS	Overall we found that increases in TEOM-measured PM10 were associated with increased odds of respiratory physician visits and hospital admissions, but not with cardiovascular health outcomes. Results indicating effects of fire smoke on respiratory outcomes are consistent with previous studies, as are the null results for cardiovascular outcomes. Some agreement between TEOM and the other metrics suggests that exposure assessment tools that are independent of air quality monitoring may be useful with further refinement.

표-82. PubMed 논문번호 21669557의 내용 요약

구분	내용
PubMed ID	21669557
TITLE	Intervention to lower household wood smoke exposure in Guatemala reduces ST-segment depression on electrocardiograms.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1002834
AUTHORS	McCracken John, Smith Kirk R, Stone Peter, D□az Anait□, Arana Byron, Schwartz Joel
BACKGROUND	A large body of evidence suggests that fine particulate matter (PM) air pollution is a cause of cardiovascular disease, but little is known in particular about the cardiovascular effects of indoor air pollution from household use of solid fuels in developing countries. RESPIRE (Randomized Exposure Study of Pollution Indoors and Respiratory Effects) was a randomized trial of a chimney woodstove that reduces wood smoke exposure.
OBJECTIVES	We tested the hypotheses that the stove intervention, compared with open fire use, would reduce ST-segment depression and increase heart rate variability (HRV).
METHODS	We used two complementary study designs: a) between-groups comparisons based on randomized stove assignment, and b) before-and-after comparisons within control subjects who used open fires during the trial and received chimney stoves after the trial. Electrocardiogram sessions that lasted 20 hr were repeated up to three times among 49 intervention and 70 control women 38-84 years of age, and 55 control subjects were also assessed after receiving stoves. HRV and ST-segment values were assessed for each 30-min period. ST-segment depression was defined as an average value below -1.00 mm. Personal fine PM [aerodynamic diameter ≤ 2.5 μm (PM_{2} .□] exposures were measured for 24 hr before each electrocardiogram.
RESULTS	PM_{2} .□ exposure means were 266 and 102 $\mu g/m^{3}$ during the trial period in the control and intervention groups, respectively. During the trial, the stove intervention was associated with an odds ratio of 0.26 (95% confidence interval, 0.08-0.90) for ST-segment depression. We found similar associations with the before-and-after comparison. The intervention was not significantly associated with HRV.
CONCLUSIONS	The stove intervention was associated with reduced occurrence of nonspecific ST-segment depression, suggesting that household wood smoke exposures affect ventricular repolarization and potentially cardiovascular health.

표-83. PubMed 논문번호 21693475의 내용 요약

구분	내용
PubMed ID	21693475
TITLE	Maternal cigarette smoking is associated with reduced high-density lipoprotein cholesterol in healthy 8-year-old children.
JOURNAL	European heart journal: 10.1093/eurheartj/ehr174
AUTHORS	Ayer Julian G, Belousova Elena, Harmer Jason A, David Clementine, Marks Guy B, Celermajer David S
AIMS	Smoking in pregnancy is common. Its effects on lipoprotein levels and arterial structure in childhood are not well characterized. We aimed to determine the effects of maternal smoking in pregnancy on lipoprotein levels and arterial wall thickness in healthy pre-pubertal children.
METHODS AND RESULTS	A community-based longitudinal study with prospective ascertainment of exposure to smoking in pregnancy and environmental tobacco smoke (ETS) since birth and then lipoprotein and arterial measurements at age 8 years. In 616 newborn infants (gestation >36 weeks and birth weight >2.5 kg) data were collected prospectively by questionnaire on smoking in pregnancy and ETS exposure in childhood. At age 8-years, 405 of the children had measurements of lipoproteins, blood pressure (BP) and carotid intima-media thickness. Children born to mothers who smoked in pregnancy had lower HDL cholesterol [1.32 vs. 1.50 mmol/L, 95% confidence interval (CI) for difference -0.28 to -0.08, P = 0.0005], higher triglycerides (1.36 vs. 1.20 mmol/L, 95% CI for ratio 1.01-1.30, P = 0.04) and higher systolic BP (102.1 vs. 99.9 mmHg, 95% CI for difference 0.6-3.8, P = 0.006). After adjustment for maternal passive smoking, post-natal ETS exposure, gender, breast feeding duration, physical inactivity, and adiposity, smoking in pregnancy remained significantly associated with lower HDL cholesterol (difference = -0.22 mmol/L, 95% CI -0.36 to -0.08, P = 0.003) but not with higher systolic BP. Neither smoking in pregnancy nor post-natal ETS exposure was associated with alterations of carotid artery wall thickness.
CONCLUSION	Smoking in pregnancy is independently associated with significantly lower HDL cholesterol in healthy 8-year-old children.

표-84. PubMed 논문번호 21695220의 내용 요약

구분	내용
PubMed ID	21695220
TITLE	Long-term exposure to ambient air pollution and mortality due to cardiovascular disease and cerebrovascular disease in Shenyang, China.
JOURNAL	PloS one: 10.1371/journal.pone.0020827
AUTHORS	Zhang Pengfei, Dong Guanghui, Sun Baijun, Zhang Liwen, Chen Xi, Ma Nannan, Yu Fei, Guo Huimin, Huang Hui, Lee Yungling Leo, Tang Naijun Chen Jie
BACKGROUND	The relationship between ambient air pollution exposure and mortality of cardiovascular and cerebrovascular diseases in human is controversial, and there is little information about how exposures to ambient air pollution contribution to the mortality of cardiovascular and cerebrovascular disease among Chinese. The aim of the present study was to examine whether exposure to ambient-air pollution increases the risk for cardiovascular and cerebrovascular disease.
METHODOLOGY/PRINCIPAL FINDINGS	We conducted a retrospective cohort study among humans to examine the association between compound-air pollutants [particulate matter <10 □m aerodynamic diameter (PM(10)), sulfur dioxide (SO(2)) and nitrogen dioxi (NO(2))] and mortality in Shenyang, China, using 12 years of data (1998-2009). Also, stratified analysis by sex, age, education, and income w conducted for cardiovascular and cerebrovascular mortality. The results showed that an increase of 10 □g/m(3) in a year average concentration of PM(10) corresponds to 55% increase in the risk of a death cardiovascular disease (hazard ratio [HR], 1.55; 95% confidence interval [CI], 1.51 to 1.6 and 49% increase in cerebrovascular disease (HR, 1.49; 95% CI, 1.45 to 1.53), respectively. The corresponding figures of adjusted HR (95%CI) for 10 □g/m(3) increase in NO(2) was 2.46 (2.31 to 2.63) for cardiovascular mortality and 2.44 (2.27 to 2.62) for cerebrovascular mortality, respectively The effects of air pollution were more evident in female that in male, and nonsmokers and residents with BMI<18.5 were more vulnerable to outdoor air pollution.
CONCLUSION/SIGNIFICANCE	Long-term exposure to ambient air pollution is associated with the death c cardiovascular and cerebrovascular diseases among Chinese populations.

표-85. PubMed 논문번호 21700913의 내용 요약

구분	내용
PubMed ID	21700913
TITLE	Long-term exposure to air pollution and cardiorespiratory disease in the California teachers study cohort.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201012-2082OC
AUTHORS	Lipsett Michael J, Ostro Bart D, Reynolds Peggy, Goldberg Debbie, Hertz Andrew, Jerrett Michael, Smith Daniel F, Garcia Cynthia, Chang Ellen T, Bernstein Leslie
RATIONALE	Several studies have linked long-term exposure to particulate air pollution with increased cardiopulmonary mortality; only two have also examined incident circulatory disease.
OBJECTIVES	To examine associations of individualized long-term exposures to particulate and gaseous air pollution with incident myocardial infarction and stroke, as well as all-cause and cause specific mortality.
METHODS	We estimated long-term residential air pollution exposure for more than 100,000 participants in the California Teachers Study, a prospective cohort o female public school professionals.We linked geocoded residential addresses with inverse distance-weighted monthly pollutant surfaces for two measures of particulate matter and for several gaseous pollutants. We examined associations between exposure to these pollutants and risks of incident myocardial infarction and stroke, and of all-cause and cause-specific mortality, using Cox proportional hazards models.
MEASUREMENTS AND MAIN RESULTS	We found elevated hazard ratios linking long-term exposure to particulate matter less than 2.5 μm in aerodynamic diameter (PM2.5), scaled to an increment of 10 μg/m3 with mortality from ischemic heart disease (IHD) (1.20; 95% confidence interval [CI], 1.02-1.41) and, particularly among postmenopausal women, incident stroke (1.19; 95% CI, 1.02-1.38). Long-term exposure to particulate matter less than 10 μm in aerodynamic diameter (PM10) was associated with elevated risks for IHD mortality (1.06; 95% CI, 0.99-1.14) and incident stroke (1.06; 95% CI, 1.00-1.13), while exposure to nitrogen oxides was associated with elevated risks for IHD and a cardiovascular mortality.
CONCLUSIONS	This study provides evidence linking long-term exposure to PM2.5 and PM1 with increased risks of incident stroke as well as IHD mortality; exposure to nitrogen oxides was also related to death from cardiovascular diseases.

표-86. PubMed 논문번호 21724522의 내용 요약

구분	내용
PubMed ID	21724522
TITLE	Indoor air pollution and blood pressure in adult women living in rural China.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1003371
AUTHORS	Baumgartner Jill, Schauer James J, Ezzati Majid, Lu Lin, Cheng Chun, Patz Jonathan A, Bautista Leonelo E
BACKGROUND	Almost half of the world's population uses coal and biomass fuels for domestic energy. Limited evidence suggests that exposure to air pollutants from indoor biomass combustion may be associated with elevated blood pressure (BP).
OBJECTIVE	Our aim was to assess the relationship between air pollution exposure from indoor biomass combustion and BP in women in rural China.
METHODS	We measured 24-hr personal integrated gravimetric exposure to fine particles < 2.5 □m in aerodynamic diameter (PM2.5) and systolic BP (SBP) and diastolic BP (DBP) in the winter and summer among 280 women ≥ 25 years of age living in rural households using biomass fuels in Yunnan, China. We investigated the association between PM2.5 exposure and SBP and DBP using mixed-effects models with random intercepts to account for correlation among repeated measures.
RESULTS	Personal average 24-hr exposure to PM2.5 ranged from 22 to 634 □g/m3 in winter and from 9 to 492 □g/m3 in summer. A 1-log-□g/m3 increase in PM2.5 exposure was associated with 2.2 mm Hg higher SBP [95% confidence interval (CI), 0.8 to 3.7; p = 0.003] and 0.5 mm Hg higher DBP (95% CI, -0.4 to 1.3; p = 0.31) among all women; estimated effects varied by age group. Among women > 50 years of age, a 1-log-□g/m3 increase in PM2.5 exposure was associated with 4.1 mm Hg higher SBP (95% CI, 1.5 to 6.6; p = 0.002) and 1.8 mm Hg higher DBP (95% CI, 0.4 to 3.2; p = 0.01). PM2.5 exposure was positively associated with SBP among younger women, but the association was not statistically significant.
CONCLUSION	PM2.5 exposure from biomass combustion may be a risk factor for elevated BP and hence for cardiovascular events. Our findings should be corroborated in longitudinal studies.

표-87. PubMed 논문번호 21730862의 내용 요약

구분	내용
PubMed ID	21730862
TITLE	Short-term impact of ambient air pollution and air temperature on blood pressure among pregnant women.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e318226e8d6
AUTHORS	Hampel Regina, Lepeule Johanna, Schneider Alexandra, Bottagisi S□bastien, Charles Marie-Aline, Ducimeti□re Pierre, Peters Annette, Slama R□my
BACKGROUND	Epidemiologic studies have reported inconsistent findings for the association between air pollution levels and blood pressure (BP), which has been studied mainly in elderly subjects. Short-term air pollution effects on BP have not been investigated in pregnant women, who may constitute a vulnerable population.
METHODS	Between 2002 and 2006, 1500 pregnant women from a mother-child cohort study conducted in Nancy and Poitiers, France, underwent 11,220 repeated BP measurements (average, 7.5 measurements/woman). Nitrogen dioxide (NO_2), particulate matter with an aerodynamic diameter below 10 μm (PM_{10}), and meteorologic variables were measured on an hourly basis at permanent monitoring sites. We studied changes of BP in relation to short-term variations of air pollution and temperature with mixed models adjusted for meteorologic and personal characteristics.
RESULTS	A 10°C decrease in temperature led to an increase in systolic BP of 0.5% (95% confidence interval = 0.1% to 1.0%). Elevated NO_2-levels 1 day, 5 days and averaged over 7 days before the BP measurement were associated with reduced systolic BP. The strongest decrease was observed for the 7-day NO_2 average (-0.4% [-0.7% to -0.2%] change for an 11 $\mu g/m^3$ increase in NO_2). PM_{10} □ effects on systolic BP differed according to pregnancy trimester: PM_{10} □ concentration was associated with systolic BP increases during the first trimester and systolic BP decreases later in pregnancy.
CONCLUSIONS	We observed short-term associations of air pollution and of temperature with BP in pregnant women. Whether such changes in BP have clinical implications remains to be investigated.

표-88. PubMed 논문번호 21742575의 내용 요약

구분	내용
PubMed ID	21742575
TITLE	Secondhand smoke exposure and coronary artery calcification among nonsmoking participants of a population-based cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1003347
AUTHORS	Peinemann Frank, Moebus Susanne, Dragano Nico, M□hlenkamp Stefan, Lehmann Nils, Zeeb Hajo, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara
BACKGROUND	Secondhand smoke (SHS) consists of fine particulate matter, carcinogens, and various toxins that affect large parts of the population. SHS increases the risk for acute cardiovascular events and may contribute to the development of atherosclerosis.
OBJECTIVES	We investigated the association of SHS with coronary artery calcification (CAC).
METHODS	In this cross-sectional analysis, we used baseline data (2000-2003) from 1,766 never-smokers without clinically manifested coronary heart disease, 45-75 years of age, from the Heinz Nixdorf Recall Study, an ongoing, prospective, population-based cohort study in Germany. Self-reported frequent SHS at home, at work, and in other places was assessed by questionnaire. CAC scores were derived based on electron-beam computed tomography. We conducted multiple linear regression analysis using exposure to SHS as the explanatory variable and ln(CAC+1) as the response variable. We conducted logistic regression to estimate the odds ratio (OR) for presence of any CAC.
RESULTS	Frequent exposure to SHS was reported by 21.5% of participants. After adjustment for age, sex, and socioeconomic status, CAC + 1 was 21.1% [95% confidence interval (CI): -5.5%, 55.2%] higher in exposed than in unexposed participants. After adjusting for other cardiovascular risk factors, the association was attenuated (15.4%; 95% CI: -9.6%, 47.2%). SHS exposure was also associated with a CAC score > 0 (fully adjusted OR = 1.38; 95% CI: 1.03, 1.84).
CONCLUSIONS	Self-reported frequent exposure to SHS was associated with subclinical coronary atherosclerosis in our cross-sectional study population. Considering the widespread exposure and the clinical relevance of coronary atherosclerosis, this result, if confirmed, is of public health importance.

표-89. PubMed 논문번호 21753226의 내용 요약

구분	내용
PubMed ID	21753226
TITLE	Combustion-derived nanoparticulate induces the adverse vascular effects of diesel exhaust inhalation.
JOURNAL	European heart journal: 10.1093/eurheartj/ehr195
AUTHORS	Mills Nicholas L, Miller Mark R, Lucking Andrew J, Beveridge Jon, Flint Laura, Boere A John F, Fokkens Paul H, Boon Nicholas A, Sandstrom Thomas, Blomberg Anders, Duffin Rodger, Donaldson Ken, Hadoke Patrick W F, Cassee Flemming R, Newby David E
AIM	Exposure to road traffic and air pollution may be a trigger of acute myocardial infarction, but the individual pollutants responsible for this effect have not been established. We assess the role of combustion-derived-nanoparticles in mediating the adverse cardiovascular effects of air pollution.
METHODS AND RESULTS	To determine the in vivo effects of inhalation of diesel exhaust components, 16 healthy volunteers were exposed to (i) dilute diesel exhaust, (ii) pure carbon nanoparticulate, (iii) filtered diesel exhaust, or (iv) filtered air, in a randomized double blind cross-over study. Following each exposure, forearm blood flow was measured during intra-brachial bradykinin, acetylcholine, sodium nitroprusside, and verapamil infusions. Compared with filtered air, inhalation of diesel exhaust increased systolic blood pressure (145 ± 4 vs. 133 ± 3 mmHg, $P < 0.05$) and attenuated vasodilatation to bradykinin ($P = 0.005$), acetylcholine ($P = 0.008$), and sodium nitroprusside ($P < 0.001$). Exposure to pure carbon nanoparticulate or filtered exhaust had no effect on endothelium-dependent or -independent vasodilatation. To determine the direct vascular effects of nanoparticulate, isolated rat aortic rings (n= 6-9 per group) were assessed in vitro by wire myography and exposed to diesel exhaust particulate, pure carbon nanoparticulate and vehicle. Compared with vehicle, diesel exhaust particulate (but not pure carbon nanoparticulate) attenuated both acetylcholine ($P < 0.001$) and sodium-nitroprusside ($P = 0.019$)-induced vasorelaxation. These effects were partially attributable to both soluble and insoluble components of the particulate.
CONCLUSION	Combustion-derived nanoparticulate appears to predominately mediate the adverse vascular effects of diesel exhaust inhalation. This provides a rationale for testing environmental health interventions targeted at reducing traffic-derived particulate emissions.

표-90. PubMed 논문번호 21761428의 내용 요약

구분	내용
PubMed ID	21761428
TITLE	Coal dust exposure and mortality from ischemic heart disease among a cohort of U.S. coal miners.
JOURNAL	American journal of industrial medicine: 10.1002/ajim.20986
AUTHORS	Landen Deborah D, Wassell James T, McWilliams Linda, Patel Ami
BACKGROUND	Particulate exposure from air pollution increases the risk of ischemic heart disease (IHD) mortality. Although coal miners are highly exposed to coal dust particulate, studies of IHD mortality risk among coal miners have had inconsistent results. Previous studies may have been biased by the healthy worker effect.
METHODS	We examined the dose-response relationship between cumulative coal dust exposure, coal rank, and IHD mortality among a cohort of underground coal miners who participated in the National Study of Coal Workers' Pneumoconiosis.
RESULTS	After adjusting for age, smoking, and body mass index, risk of IHD mortality increased at higher levels of coal dust exposure. Mortality risk was also associated with coal rank region.
CONCLUSION	There was an increased risk of mortality from IHD associated with cumulative exposure to coal dust, and with coal rank. The effect of coal rank may be due differences in the composition of coal mine dust particulate. The association of risk of IHD mortality with cumulative particulate exposure is consistent with air pollution studies.

표-91. PubMed 논문번호 21768054의 내용 요약

구분	내용
PubMed ID	21768054
TITLE	Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: shape of the exposure-response relationships.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103639
AUTHORS	Pope C Arden, Burnett Richard T, Turner Michelle C, Cohen Aaron, Krewski Daniel, Jerrett Michael, Gapstur Susan M, Thun Michael J
BACKGROUND	Lung cancer and cardiovascular disease (CVD) mortality risks increase with smoking, secondhand smoke (SHS), and exposure to fine particulate matter < 2.5 μm in diameter (PM_{2} .□) from ambient air pollution. Recent research indicates that the exposure-response relationship for CVD is nonlinear, with a steep increase in risk at low exposures and flattening out at higher exposures. Comparable estimates of the exposure-response relationship for lung cancer are required for disease burden estimates and related public health policy assessments.
OBJECTIVES	We compared exposure-response relationships of PM_{2} .□ with lung cancer and cardiovascular mortality and considered the implications of the observed differences for efforts to estimate the disease burden of PM2.5.
METHODS	Prospective cohort data for 1.2 million adults were collected by the American Cancer Society as part of the Cancer Prevention Study II. We estimated relative risks (RRs) for increments of cigarette smoking, adjusting for various individual risk factors. RRs were plotted against estimated daily dose of PM_{2} .□ from smoking along with comparison estimates for ambient air pollution and SHS.
RESULTS	For lung cancer mortality, excess risk rose nearly linearly, reaching maximum RRs > 40 among long-term heavy smokers. Excess risks for CVD mortality increased steeply at low exposure levels and leveled off at higher exposures, reaching RRs of approximately 2-3 for cigarette smoking.
CONCLUSIONS	The exposure-response relationship associated with PM_{2} .□ is qualitatively different for lung cancer versus cardiovascular mortality. At low exposure levels, cardiovascular deaths are projected to account for most of the burden of disease, whereas at high levels of PM_{2} .□, lung cancer becomes proportionately more important.

표-92. PubMed 논문번호 21778006의 내용 요약

구분	내용
PubMed ID	21778006
TITLE	Ambient air pollutants and risk of fatal coronary heart disease among kidney transplant recipients.
JOURNAL	American journal of kidney diseases : the official journal of the National Kidney Foundation: 10.1053/j.ajkd.2011.05.017
AUTHORS	Spencer-Hwang Rhonda, Knutsen Synnove Fonnebo, Soret Sam, Ghamsary Mark, Beeson W Lawrence, Oda Keiji, Shavlik David, Jaipaul Navin
BACKGROUND	There is increasing evidence that specific ambient air pollutants are associated with coronary heart disease (CHD) morbidity and mortality. Because kidney transplant recipients have prevalent traditional and nontraditional risk factors, they may constitute a sensitive subgroup.
STUDY DESIGN	Retrospective cohort.
SETTING & PARTICIPANTS	This study includes 32,239 nonsmoking adult kidney transplant recipients who underwent transplant in 1997-2003, identified through the US Renal Data System and living in the United States within 50 km of an air pollution monitoring station.
PREDICTOR	Long-term ambient pollutant ozone and particulate matter ≤10 μm (PM(10)), assessed from monthly concentrations of ozone and PM(10) calculated from ambient monitoring data by the US Environmental Protection Agency Air Quality System and interpolated to zip code centroids according to patients' residence.
OUTCOMES	Outcomes of interest were death from CHD and natural-cause mortality.
RESULTS	For the entire transplant cohort, average pollutant levels for ozone and PM(10) were 25.5 ± 4.4 parts per billion (ppb) and 25.3 ± 6.4 μg/m(3), respectively. Correlation between ozone and PM(10) values was low, but statistically significant (P < 0.001). There were deaths from CHD (n = 267) and natural causes (n = 2,076) during the 7-year study period. For each 10-ppb increase in ozone, the risk of fatal CHD increased by 35% (RR, 1.35; 95% CI, 1.04-1.77) in the single-pollutant model and 34% (RR, 1.34; 95% CI, 1.03-1.76) in the 2-pollutant model. No independent association was found between CHD and PM(10). No significant association was identified for PM(10) or ozone level and natural-cause mortality (RR, 1.09; 95% CI, 0.99-1.21).
LIMITATIONS	Exposure assignment based on only residential location.
CONCLUSIONS	For kidney transplant recipients, ambient ozone levels potentially are associated with higher risk of fatal CHD. These findings may have implications for regulations governing air pollution and the development of individual CHD risk-reduction strategies.

표-93. PubMed 논문번호 21816396의 내용 요약

구분	내용
PubMed ID	21816396
TITLE	Emergency ambulance dispatches and apparent temperature: a time series analysis in Emilia-Romagna, Italy.
JOURNAL	Environmental research: 10.1016/j.envres.2011.07.005
AUTHORS	Alessandrini Ester, Zauli Sajani Stefano, Scotto Fabiana, Miglio Rossella, Marchesi Stefano, Lauriola Paolo
INTRODUCTION	Increases in mortality associated with oppressive weather have been widely investigated in several epidemiological studies. However, to properly understand the full public health significance of heat-related health effects, as well as to develop an effective surveillance system, it is also important to investigate the impact of stressful meteorological conditions on non-fatal events. The objective of our study was to evaluate the exposure-response relationship of ambulance dispatch data in association with biometeorological conditions using time series techniques similar to those used in previous studies on mortality.
METHODS	Daily data of emergency ambulance dispatches for people aged 35 or older in the summer periods from 2002 to 2006 were collected for the major towns in the Emilia-Romagna region. In the first stage of the analysis, the city-specific relationship between daily ambulance dispatches and increasing apparent temperature was explored using Generalized Additive Models while controlling for air pollution, seasonality, long-term trend, holidays and weekends. The relationship between ambulance dispatches and apparent temperature was approximated by linear splines. The effects of high temperatures on health were evaluated for respiratory and cardiovascular diseases as well as for all non-traumatic conditions. In the second stage of the analysis, city-specific effects were combined in fixed or random effect meta-analyses.
RESULTS	The percent change in the ambulance dispatches associated with every 1 ° C increase in the mean apparent temperature between 25 and 30 ° C was 1.45% (95% confidence interval: 0.95, 1.95) for non-traumatic diseases and 2.74% (95% CI: 1.34, 4.14) for respiratory diseases. The percent increase in risk was greater on days in which the mean apparent temperature exceeded 30 ° C (8.85%, 95% CI: 7.12, 10.58 for non-traumatic diseases). In this interval of biometeorological conditions, cardiovascular diseases became positively associated with the apparent temperature. The risks increased with age. The increase in risk for the non-traumatic diseases reached 13.34% for people aged 75 or older compared to 4.75% for those aged 35-64.
CONCLUSION	Time series analysis techniques were adopted for the first time to investigate emergency ambulance dispatches to evaluate the risks associated with biometeorological discomfort. Our findings show a strong relationship between biometeorological conditions and ambulance dispatches.

표-94. PubMed 논문번호 21827977의 내용 요약

구분	내용
PubMed ID	21827977
TITLE	Long-term urban particulate air pollution, traffic noise, and arterial blood pressure.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103564
AUTHORS	Fuks Kateryna, Moebus Susanne, Hertel Sabine, Viehmann Anja, Nonnemacher Michael, Dragano Nico, M□hlenkamp Stefan, Jakobs Hermann, Kessler Christoph, Erbel Raimund, Hoffmann Barbara
BACKGROUND	Recent studies have shown an association of short-term exposure to fine particulate matter (PM) with transient increases in blood pressure (BP), but it is unclear whether long-term exposure has an effect on arterial BP and hypertension.
OBJECTIVES	We investigated the cross-sectional association of residential long-term PM exposure with arterial BP and hypertension, taking short-term variations of PM and long-term road traffic noise exposure into account.
METHODS	We used baseline data (2000-2003) on 4,291 participants, 45-75 years of age, from the Heinz Nixdorf Recall Study, a population-based prospective cohort in Germany. Urban background exposure to PM with aerodynamic diameter ≤ 2.5 μm (PM(2.5)) and ≤ 10 μm (PM(10)) was assessed with a dispersion and chemistry transport model. We used generalized additive models, adjusting for short-term PM, meteorology, traffic proximity, and individual risk factors.
RESULTS	An interquartile increase in PM2.5 (2.4 μg/m(3)) was associated with estimated increases in mean systolic and diastolic BP of 1.4 mmHg [95% confidence interval (CI): 0.5, 2.3] and 0.9 mmHg (95% CI: 0.4, 1.4), respectively. The observed relationship was independent of long-term exposure to road traffic noise and robust to the inclusion of many potential confounders. Residential proximity to high traffic and traffic noise exposure showed a tendency toward higher BP and an elevated prevalence of hypertension.
CONCLUSIONS	We found an association of long-term exposure to PM with increased arterial BP in a population-based sample. This finding supports our hypothesis that long-term PM exposure may promote atherosclerosis, with air-pollution-induced increases in BP being one possible biological pathway.

표-95. PubMed 논문번호 21835436의 내용 요약

구분	내용
PubMed ID	21835436
TITLE	The effect of particle size on cardiovascular disorders - the smaller the worse.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2011.05.049
AUTHORS	Franck Ulrich, Odeh Siad, Wiedensohler Alfred, Wehner Birgit, Herbarth Olf
BACKGROUND	Previous studies observed associations between airborne particles and cardio-vascular disease. Questions, however, remain as to which size of the inhalable particles (coarse, fine, or ultrafine) exerts the most significant impact on health.
METHODS	For this retrospective study, data of the total number of 23,741 emergency service calls, registered between February 2002 and January 2003 in the City of Leipzig, were analysed, identifying 5326 as being related to cardiovascular incidences. Simultaneous particle exposure was determined for the particle sizes classes <100 nm (UFP), <2.5 μm (PM2.5) and <10 μm (PM10). We used a time resolution of 1 day for both parameters, emergency calls and exposure.
RESULTS	Within the group of cardiovascular diseases, the diagnostic category of hypertensive crisis showed a significant association with particle exposure. The significant effect on hypertensive crisis was found for particles with a size of <100 nm in diameter and starting with a lag of 2 days after exposure. No consistent influence could be observed for PM2.5 and PM10. The Odds Ratios on hypertensive crisis were significant for the particle size <100 nm in diameter from day 2 post exposure OR=1.06 (95%CI: 1.02-1.10, p=0.002) up to day 7 OR=1.05 (95%CI 1.02-1.09, p=0.005).
CONCLUSION	Ultrafine particles affect cardiovascular disease adversely, particularly hypertensive crises. Their effect is significant compared with PM2.5 and PM10. It appears necessary, from a public health point of view, to consider regulating this type of particles using appropriate measurands as particle number.

표-96. PubMed 논문번호 21885382의 내용 요약

구분	내용
PubMed ID	21885382
TITLE	Transportation noise and blood pressure in a population-based sample of adults.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103448
AUTHORS	Dratva Julia, Phuleria Harish C, Foraster Maria, Gaspoz Jean-Michel, Keidel Dirk, K□nzli Nino, Liu L-J Sally, Pons Marco, Zemp Elisabeth, Gerbase Margaret W, Schindler Christian, Dratva Julia, Phuleria Harish C, Foraster Maria, Gaspoz Jean-Michel, Keidel Dirk, K□nzli Nino, Liu L-J Sally, Pons Marco, Zemp Elisabeth, Gerbase Margaret W, Schindler Christian
BACKGROUND	There is some evidence for an association between traffic noise and ischemic heart disease; however, associations with blood pressure have been inconsistent, and little is known about health effects of railway noise.
OBJECTIVES	We aimed to investigate the effects of railway and traffic noise exposure on blood pressure; a secondary aim was to address potentially susceptible subpopulations.
METHODS	We performed adjusted linear regression analyses using data from 6,450 participants of the second survey of the Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA 2) to estimate the associations of daytime and nighttime railway and traffic noise (A-weighted decibels) with systolic blood pressure (SBP) and diastolic blood pressure (DBP; millimeters of mercury). Noise data were provided by the Federal Office for the Environment. Stratified analyses by self-reported hypertension, cardiovascular disease (CVD), and diabetes were performed.
RESULTS	Mean noise exposure during the day and night was 51 dB(A) and 39 dB(A) for traffic noise, respectively, and 19 dB(A) and 17 dB(A) for railway noise. Adjusted regression models yielded significant effect estimates for a 10 dB(A) increase in railway noise during the night [SBP β = 0.84; 95% confidence interval (CI): 0.22, 1.46; DBP β = 0.44; 95% CI: 0.06, 0.81] and day (SBP β = 0.60; 95% CI: 0.07, 1.13). Additional adjustment for nitrogen dioxide left effect estimates almost unchanged. Stronger associations were estimated for participants with chronic disease. Significant associations with traffic noise were seen only among participants with diabetes.
CONCLUSION	We found evidence of an adverse effect of railway noise on blood pressure in this cohort population. Traffic noise was associated with higher blood pressure only in diabetics, possibly due to low exposure levels. The study results imply more severe health effects by transportation noise in vulnerable populations, such as adults with hypertension, diabetes, or CVD.

표-97. PubMed 논문번호 21890052의 내용 요약

구분	내용
PubMed ID	21890052
TITLE	Mortality among survivors of the Sept 11, 2001, World Trade Center disaster: results from the World Trade Center Health Registry cohort.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(11)60966-5
AUTHORS	Jordan Hannah T, Brackbill Robert M, Cone James E, Debchoudhury Indira, Farfel Mark R, Greene Carolyn M, Hadler James L, Kennedy Joseph, Li Jiehui, Liff Jonathan, Stayner Leslie, Stellman Steven D
BACKGROUND	The Sept 11, 2001 (9/11) World Trade Center (WTC) disaster has been associated with several subacute and chronic health effects, but whether excess mortality after 9/11 has occurred is unknown. We tested whether excess mortality has occurred in people exposed to the WTC disaster.
METHODS	In this observational cohort study, deaths occurring in 2003-09 in WTC Health Registry participants residing in New York City were identified through linkage to New York City vital records and the National Death Index. Eligible participants were rescue and recovery workers and volunteers; lower Manhattan area residents, workers, school staff and students; and commuters and passers-by on 9/11. Study participants were categorised as rescue and recovery workers (including volunteers), or non-rescue and non-recovery participants. Standardised mortality ratios (SMR) were calculated with New York City rates from 2000-09 as the reference. Within the cohort, proportional hazards were used to examine the relation between a three-tiered WTC-related exposure level (high, intermediate, or low) and total mortality.
FINDINGS	We identified 156 deaths in 13,337 rescue and recovery workers and 634 deaths in 28,593 non-rescue and non-recovery participants. All-cause SMRs were significantly lower than that expected for rescue and recovery participants (SMR 0·45, 95% CI 0·38-0·53) and non-rescue and non-recovery participants (0·61, 0·56-0·66). No significantly increased SMRs for diseases of the respiratory system or heart, or for haematological malignancies were found. In non-rescue and non-recovery participants, both intermediate and high levels of WTC-related exposure were significantly associated with mortality when compared with low exposure (adjusted hazard ratio 1·22, 95% CI 1·01-1·48, for intermediate exposure and 1·56, 1·15-2·12, for high exposure). High levels of exposure in non-rescue and non-recovery individuals, when compared with low exposed non-rescue and non-recovery individuals, were associated with heart-disease-related mortality (adjusted hazard ratio 2·06, 1·10-3·86). In rescue and recovery participants, level of WTC-related exposure was not significantly associated with all-cause mortality (adjusted hazard ratio 1·25, 95% CI 0·56-2·78, for high exposure and 1·03, 0·52-2·06, for intermediate exposure when compared with low exposure).
INTERPRETATION	This exploratory study of mortality in a well defined cohort of 9/11 survivors provides a baseline for continued surveillance. Additional follow-up is needed to establish whether these associations persist and whether a similar association over time will occur in rescue and recovery participants.
FUNDING	US Centers for Disease Control and Prevention (National Institute for Occupational Safety and Health, Agency for Toxic Substances and Disease Registry, and National Center for Environmental Health); New York City Department of Health and Mental Hygiene.

표-98. PubMed 논문번호 21975181의 내용 요약

구분	내용
PubMed ID	21975181
TITLE	A retrospective cohort study of stroke onset: implications for characterizing short term effects from ambient air pollution.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-10-87
AUTHORS	Johnson Julie Y M, Villeneuve Paul J, Pasichnyk Dion, Rowe Brian H
BACKGROUND	Case-crossover studies used to investigate associations between an environmental exposure and an acute health response, such as stroke, will often use the day an individual presents to an emergency department (ED) or is admitted to hospital to infer when the stroke occurred. Similarly, they will use patient's place of residence to assign exposure. The validity of using these two data elements, typically extracted from administrative databases or patient charts, to define the time of stroke onset and to assign exposure are critical in this field of research as air pollutant concentrations are temporally and spatially variable. Our a priori hypotheses were that date of presentation differs from the date of stroke onset for a substantial number of patients, and that assigning exposure to ambient pollution using place of residence introduces an important source of exposure measurement error. The objective of this study was to improve our understanding on how these sources of errors influence risk estimates derived using a case-crossover study design.
METHODS	We sought to collect survey data from stroke patients presenting to hospital EDs in Edmonton, Canada on the date, time, location and nature of activities at onset of stroke symptoms. The daily mean ambient concentrations of NO_2 and PM(2.5) on the self-reported day of stroke onset was estimated from continuous fixed-site monitoring stations.
RESULTS	Of the 336 participating patients, 241 were able to recall when their stroke started and 72.6% (95% confidence interval [CI]: 66.9-78.3%) experienced stroke onset the same day they presented to the ED. For subjects whose day of stroke onset differed from the day of presentation to the ED, this difference ranged from 1 to 12 days (mean = 1.8; median = 1). In these subjects, there were no systematic differences in assigned pollution levels for either NO_2 or PM(2.5) when day of presentation rather than day of stroke onset was used. At the time of stroke onset, 89.9% (95% CI: 86.6-93.1%) reported that they were inside, while 84.5% (95% CI: 80.6 - 88.4%) reported that for most of the day they were within a 15 minute drive from home. We estimated that due to the mis-specification of the day of stroke onset, the risk of hospitalization for stroke would be understated by 15% and 20%, for NO_2 and PM(2.5), respectively.
CONCLUSIONS	Our data suggest that day of presentation and residential location data obtained from administrative records reasonably captures the time and location of stroke onset for most patients. Under these conditions, any associated errors are unlikely to be an important source of bias when estimating air pollution risks in this population.

표-99. PubMed 논문번호 22004949의 내용 요약

구분	내용
PubMed ID	22004949
TITLE	Health impacts of the built environment: within-urban variability in physical inactivity, air pollution, and ischemic heart disease mortality.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103806
AUTHORS	Hankey Steve, Marshall Julian D, Brauer Michael, Hankey Steve, Marshall Julian D, Brauer Michael
BACKGROUND	Physical inactivity and exposure to air pollution are important risk factors for death and disease globally. The built environment may influence exposures to these risk factors in different ways and thus differentially affect the health of urban populations.
OBJECTIVE	We investigated the built environment's association with air pollution and physical inactivity, and estimated attributable health risks.
METHODS	We used a regional travel survey to estimate within-urban variability in physical inactivity and home-based air pollution exposure [particulate matter with aerodynamic diameter ≤ 2.5 μm (PM2.5), nitrogen oxides (NOx), and ozone (O3)] for 30,007 individuals in southern California. We then estimated the resulting risk for ischemic heart disease (IHD) using literature-derived dose-response values. Using a cross-sectional approach, we compared estimated IHD mortality risks among neighborhoods based on "walkability" scores.
RESULTS	The proportion of physically active individuals was higher in high- versus low-walkability neighborhoods (24.9% vs. 12.5%); however, only a small proportion of the population was physically active, and between-neighborhood variability in estimated IHD mortality attributable to physical inactivity was modest (7 fewer IHD deaths/100,000/year in high- vs. low-walkability neighborhoods). Between-neighborhood differences in estimated IHD mortality from air pollution were comparable in magnitude (9 more IHD deaths/100,000/year for PM2.5 and 3 fewer IHD deaths for O3 in high- vs. low-walkability neighborhoods), suggesting that population health benefits from increased physical activity in high-walkability neighborhoods may be offset by adverse effects of air pollution exposure.
POLICY IMPLICATIONS	Currently, planning efforts mainly focus on increasing physical activity through neighborhood design. Our results suggest that differences in population health impacts among neighborhoods are similar in magnitude for air pollution and physical activity. Thus, physical activity and exposure to air pollution are critical aspects of planning for cleaner, health-promoting cities.

표-100. PubMed 논문번호 22020729의 내용 요약

구분	내용
PubMed ID	22020729
TITLE	Opposing effects of particle pollution, ozone, and ambient temperature on arterial blood pressure.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103647
AUTHORS	Hoffmann Barbara, Luttmann-Gibson Heike, Cohen Allison, Zanobetti Antonella, de Souza Celine, Foley Christopher, Suh Helen H, Coull Brent A, Schwartz Joel, Mittleman Murray, Stone Peter, Horton Edward, Gold Diane R, Hoffmann Barbara, Luttmann-Gibson Heike, Cohen Allison, Zanobetti Antonella, de Souza Celine, Foley Christopher, Suh Helen H, Coull Brent A, Schwartz Joel, Mittleman Murray, Stone Peter, Horton Edward, Gold Diane R
BACKGROUND	Diabetes increases the risk of hypertension and orthostatic hypotension and raises the risk of cardiovascular death during heat waves and high pollution episodes.
OBJECTIVE	We examined whether short-term exposures to air pollution (fine particles, ozone) and heat resulted in perturbation of arterial blood pressure (BP) in persons with type 2 diabetes mellitus (T2DM).
METHODS	We conducted a panel study in 70 subjects with T2DM, measuring BP by automated oscillometric sphygmomanometer and pulse wave analysis every 2 weeks on up to five occasions (355 repeated measures). Hourly central site measurements of fine particles, ozone, and meteorology were conducted. We applied linear mixed models with random participant intercepts to investigate the association of fine particles, ozone, and ambient temperature with systolic, diastolic, and mean arterial BP in a multipollutant model, controlling for season, meteorological variables, and subject characteristics.
RESULTS	An interquartile increase in ambient fine particle mass [particulate matter (PM) with an aerodynamic diameter of ≤ 2.5 μm (PM2.5)] and in the traffic component black carbon in the previous 5 days (3.54 and 0.25 μg/m3, respectively) predicted increases of 1.4 mmHg [95% confidence interval (CI): 0.0, 2.9 mmHg] and 2.2 mmHg (95% CI: 0.4, 4.0 mmHg) in systolic BP (SBP) at the population geometric mean, respectively. In contrast, an interquartile increase in the 5-day mean of ozone (13.3 ppb) was associated with a 5.2 mmHg (95% CI: -8.6, -1.8 mmHg) decrease in SBP. Higher temperatures were associated with a marginal decrease in BP.
CONCLUSIONS	In subjects with T2DM, PM was associated with increased BP, and ozone was associated with decreased BP. These effects may be clinically important in patients with already compromised autoregulatory function.

표-101. PubMed 논문번호 22034939의 내용 요약

구분	내용
PubMed ID	22034939
TITLE	Exposure to road traffic and railway noise and associations with blood pressure and self-reported hypertension: a cohort study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-10-92
AUTHORS	S ø rensen Mette, Hvidberg Martin, Hoffmann Barbara, Andersen Zorana J, Nordsborg Rikke B, Lillelund Kenneth G, Jakobsen J ø rgen, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
BACKGROUND	Epidemiological studies suggest that long-term exposure to transport noise increases the risk for cardiovascular disorders. The effect of transport noise on blood pressure and hypertension is uncertain.
METHODS	In 1993-1997, 57,053 participants aged 50-64 year were enrolled in a population-based cohort study. At enrollment, systolic and diastolic blood pressure was measured. Incident hypertension during a mean follow-up of 5.3 years was assessed by questionnaire. Residential long-term road traffic noise (Lden) was estimated for 1- and 5-year periods preceding enrollment and preceding diagnosis of hypertension. Residential exposure to railway noise was estimated at enrollment. We conducted a cross-sectional analysis of associations between road traffic and railway noise and blood pressure at enrollment with linear regression, adjusting for long-term air pollution, meteorology and potential lifestyle confounders (N = 44,083). Incident self-reported hypertension was analyzed with Cox regression, adjusting for long-term air pollution and potential lifestyle confounders.
RESULTS	We found a 0.26 mm Hg higher systolic blood pressure (95% confidence intervals (CI): -0.11; 0.63) per 10 dB(A) increase in 1-year mean road traffic noise levels, with stronger associations in men (0.59 mm Hg (CI: 0.13; 1.05) per 10 dB(A)) and older participants (0.65 mm Hg (0.08; 1.22) per 10 dB(A)). Road traffic noise was not associated with diastolic blood pressure or hypertension. Exposure to railway noise above 60 dB was associated with 8% higher risk for hypertension (95% CI: -2%; 19%, P = 0.11).
CONCLUSIONS	While exposure to road traffic noise was associated with systolic blood pressure in subgroups, we were not able to identify associations with hypertension.

표-102. PubMed 논문번호 22046271의 내용 요약

구분	내용
PubMed ID	22046271
TITLE	Lung function is associated with arterial stiffness in children.
JOURNAL	PloS one: 10.1371/journal.pone.0026303
AUTHORS	Ayer Julian G, Belousova Elena G, Harmer Jason A, Toelle Brett, Celermajer David S, Marks Guy B
BACKGROUND	In older adults, an independent association exists between impaired lung function and cardiovascular disease. This interaction might be related to the effects of aging and/or smoking. In order to explore possible childhood antecedents to this association, we hypothesized that decreased lung function and vascular stiffness might be related, in early life.
OBJECTIVE	To determine the relationship between lung function and carotid augmentation index (AIx), a measure of vascular stiffness, in 8-year old children.
METHODS	Data on brachial blood pressure, lung function (FEV(1), FVC, FEV(1)/FVC, obtained by spirometry) and carotid AIx75 (AIx standardised to an arbitrary heart rate of 75 beats per minute, obtained by applanation tonometry) was available in 249 community-based 8-year old children. These healthy children had been subjects in a randomised controlled trial of two interventions (omega-3 fatty acid supplementation and house-dust mite avoidance) to prevent asthma. Smoking in pregnancy and childhood environmental tobacco smoke (ETS) exposure was prospectively collected by questionnaire. The association between lung function and carotid AIx75 was assessed in multivariate models that included sex, height, smoking status during pregnancy, ETS exposure and randomisation groups (house dust mite avoidance and dietary intervention) as covariates.
RESULTS	In the fully adjusted models, Carotid AIx75 was independently associated with FEV1 (standardised β = −0.17,b = −6.72, partial R(2) = .02, p = 0.03), FVC (standardised β = −0.29, b = −9.31, partial R(2) = 0.04, p<0.001) and FEV1/FVC (standardised β = .13, b = 18.4, partial R(2) = 0.02, p = 0.04).
CONCLUSION	Lower lung volumes are associated with increased vascular stiffness at an early age. The interaction between lung function and vascular stiffness may thus represent more than just age-related alterations in both the pulmonary and vascular systems.

표-103. PubMed 논문번호 22047619의 내용 요약

구분	내용
PubMed ID	22047619
TITLE	High frequency of diastolic dysfunction in a population-based cohort of elderly women-but poor association with the symptom dyspnea.
JOURNAL	BMC geriatrics: 10.1186/1471-2318-11-71
AUTHORS	Germing Alfried, Gotzmann Michael, Schikowski Tamara, Vierk□tter Andrea, Ranft Ulrich, Kr□mer Ursula, M□gge Andreas
BACKGROUND	The European Society of Cardiology recently proposed a new algorithm "How to diagnose heart failure with normal ejection fraction". Central element of the diagnostic strategy is the demonstration of diastolic dysfunction, either by tissue Doppler-derived indices in first line, or in second line by a combination of elevated blood levels of natriuretic peptide with abnormal tissue Doppler findings. We thought to use this diagnostic flowchart in a population-based cohort of elderly women, in whom the prevalence of diastolic dysfunction and heart failure is believed to be high. The purpose was to evaluate the association of dyspnea with the presence of diastolic dysfunction.
METHODS	The study cohort recruited from a cross-sectional follow-up examination of the SALIA cohort (study on the influence of air pollution on lung function, inflammation, and aging). Participants with cardiac or pulmonary disease were excluded, 291 participants formed the final study group (all women, age range 69 to 79 years, all in sinus rhythm, LV ejection fraction > 50%, LV enddiastolic volume index < 97 mL/m2). Quality of life was assessed by the Minnesota living with heart failure questionnaire, and actual symptoms by a structural questionnaire; the examination consisted of a physical examination, measurement of B-type natriuretic peptide, ECG and tissue Doppler echocardiography. Diastolic dysfunction was assumed when the E/E' ratio exceeded 15 as derived from tissue Doppler. In case, tissue Doppler yielded an E/E' ratio ranging from 8 to 15, additional non-invasive parameters had to be fulfilled: left atrial volume index > 40 ml/m2 body surface, or left ventricular mass index > 122 g/m2 body surface, or transmitral E/A ratio < 0.5 plus deceleration time > 280 ms, or blood level of brain natriuretic peptide (BNP) > 200 pg/mL.
RESULTS	The examinations were concordant with the presence of diastolic dysfunction in 122/291 participants (41.9%). The diagnosis based in 94% of cases on two criteria: in 50 cases on the criterion "E/E' ratio > 15", and in 65 cases on the criterion "15 > E/E'>8 and LV mass index > 122 g/m2". The participants with diastolic dysfunction had on average a higher body mass index, more frequent a history of arterial hypertension and of hospitalization for congestive heart failure, poorer quality of life, and higher BNP blood levels as compared to those participants without signs of diastolic dysfunction. The number of participants complaining exertional dyspnea, however, was similar distributed among the subgroups with and without signs of diastolic dysfunction (40.2 vs 40.8%; p = n.s). In a logistic regression model, the symptom dyspnea was best predicted by systolic pulmonary artery pressure, followed by left atrial volume index, BNP, and body mass index.
CONCLUSION	The demonstration of diastolic dysfunction showed only a poor association with the symptom dyspnea in a cohort of elderly women with otherwise normal systolic function. Additional structural or hemodynamic changes are necessary to "explain" the symptom dyspnea. It is unclear whether these additional factors are secondary to a more advanced stage of diastolic dysfunction, or are related to cardiovascular co-morbidities, or both.

표-104. PubMed 논문번호 22052517의 내용 요약

구분	내용
PubMed ID	22052517
TITLE	Stroke and long-term exposure to outdoor air pollution from nitrogen dioxide: a cohort study.
JOURNAL	Stroke: 10.1161/STROKEAHA.111.629246
AUTHORS	Andersen Zorana J, Kristiansen Luise C, Andersen Klaus K, Olsen Tom S, Hvidberg Martin, Jensen Steen S, Ketzel Matthias, Loft Steffen, S ø rensen Mette, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole, Andersen Zorana J, Kristiansen Luise C, Andersen Klaus K, Olsen Tom S, Hvidberg Martin, Jensen Steen S, Ketzel Matthias, Loft Steffen, S ø rensen Mette, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
BACKGROUND AND PURPOSE	Years of exposure to tobacco smoke substantially increase the risk for stroke. Whether long-term exposure to outdoor air pollution can lead to stroke is not yet established. We examined the association between long-term exposure to traffic-related air pollution and incident and fatal stroke in a prospective cohort study.
METHODS	We followed 57,053 participants of the Danish Diet, Cancer and Health cohort in the Hospital Discharge Register for the first-ever hospital admission for stroke (incident stroke) between baseline (1993-1997) and 2006 and defined fatal strokes as death within 30 days of admission. We associated the estimated mean levels of nitrogen dioxide at residential addresses since 1971 to incident and fatal stroke by Cox regression analyses and examined the effects by stroke subtypes: ischemic, hemorrhagic, and nonspecified stroke.
RESULTS	Over a mean follow-up of 9.8 years of 52,215 eligible subjects, there were 1984 (3.8%) first-ever (incident) hospital admissions for stroke of whom 142 (7.2%) died within 30 days. We detected borderline significant associations between mean nitrogen dioxide levels at residence since 1971 and incident stroke (hazard ratio, 1.05; 95% CI, 0.99-1.11, per interquartile range increase) and stroke hospitalization followed by death within 30 days (1.22; 1.00-1.50). The associations were strongest for nonspecified and ischemic strokes, whereas no association was detected with hemorrhagic stroke.
CONCLUSIONS	Long-term exposure to traffic-related air pollution may contribute to the development of ischemic but not hemorrhagic stroke, especially severe ischemic strokes leading to death within 30 days.

표-105. PubMed 논문번호 22075540의 내용 요약

구분	내용
PubMed ID	22075540
TITLE	Exposure to particulate matter and long-term risk of cardiovascular mortality in Japan: NIPPON DATA80.
JOURNAL	Journal of atherosclerosis and thrombosis: 10.5551/jat.9506
AUTHORS	Ueda Kayo, Nagasawa Shin-Ya, Nitta Hiroshi, Miura Katsuyuki, Ueshima Hirotsugu, Ueda Kayo, Nagasawa Shin-Ya, Nitta Hiroshi, Miura Katsuyuki, Ueshima Hirotsugu
AIM	It remains uncertain whether chronic exposure to particulate air pollution is associated with increased mortality in Japan because Japan has a different distribution pattern of cardiovascular disease and its risk factors compared to Western countries. We investigated the association between long-term exposure to particulate matter (PM) and cardiovascular mortality risk using a representative Japanese cohort.
METHODS	A total of 7,250 participants aged 30 years and older from 232 districts throughout Japan were followed from 1980 to 2004. We linked the averaged annual concentrations of PM from 1985 to 2004 to each cohort participant who resided in the district at the time of the baseline survey. Study participants were divided into quintiles of average PM concentration. We applied the Cox proportional hazard model adjusting for sex, age, body mass index, blood pressure, total cholesterol, blood glucose, smoking categories, drinking categories, and the municipality population size.
RESULTS	During follow-up, there were 1,716 deaths from all causes; 571 from cardiovascular disease, 116 from coronary heart disease, and 250 from stroke. Hazard ratios were not different among the quintiles and those for trend per 10 □g/m3 increase in annual PM concentration were 0.98 (95% confidence interval, 0.92-1.04) for all-cause mortality and 0.90 (95% confidence interval, 0.81-1.00) for cardiovascular mortality.
CONCLUSION	Long-term exposure to PM was not associated with increased cardiovascular mortality risk in this population-based cohort in Japan.

표-106. PubMed 논문번호 22092933의 내용 요약

구분	내용
PubMed ID	22092933
TITLE	The relation between coronary artery calcification in asymptomatic subjects and both traditional risk factors and living in the city centre: a DanRisk substudy.
JOURNAL	Journal of internal medicine: 10.1111/j.1365-2796.2011.02486.x
AUTHORS	Lambrechtsen J, Gerke O, Egstrup K, Sand N P, N ø rgaard B L, Petersen H, Mickley H, Diederichsen A C P, Lambrechtsen J, Gerke O, Egstrup K, Sand N P, N ø rgaard B L, Petersen H, Mickley H, Diederichsen A C P
OBJECTIVE	To evaluate the association between the risk factor for living in the city centre as a surrogate for air pollution and the presence of coronary artery calcification (CAC) in a population of asymptomatic Danish subjects.
DESIGN AND SUBJECTS	A random sample of 1825 men and women of either 50 or 60 years of age were invited to take part in a screening project designed to assess risk factors for cardiovascular disease (CVD). Noncontrast cardiac computed tomography was performed on all subjects, and their Agatston scores were calculated to evaluate the presence of subclinical coronary atherosclerosis. The relationship between CAC and several demographic and clinical parameters was evaluated using multivariate logistic regression.
RESULTS	A total of 1225 individuals participated in the study, of whom 250 (20%) were living in the centres of major Danish cities. Gender and age showed the greatest association with the presence of CAC: the odds ratio (OR) for men compared with women was 3.2 [95% confidence interval (CI) 2.5-4.2; P < 0.0001], and the OR for subjects aged 60 versus those aged 50 years was 2.2 (95% CI 1.7-2.8; P < 0.0001). Other variables independently associated with the presence of CAC were diabetes and smoking with ORs of 2.0 (95% CI 1.1-3.5; P = 0.03) and 1.9 (95% CI 1.4-2.5, P < 0.0001), respectively. The adjusted OR for subjects living in city centres compared to those living outside was 1.8 (95% CI 1.3-2.4; P = 0.0003).
CONCLUSION	Both conventional risk factors for CVD and living in a city centre are independently associated with the presence of CAC in asymptomatic middle-aged subjects.

표-107. PubMed 논문번호 22123794의 내용 요약

구분	내용
PubMed ID	22123794
TITLE	Secondhand smoke exposure and quality of life in patients with heart failure.
JOURNAL	Archives of internal medicine: 10.1001/archinternmed.2011.518
AUTHORS	Weeks Sarah G, Glantz Stanton A, De Marco Teresa, Rosen Alisa B, Fleischmann Kirsten E
BACKGROUND	Secondhand smoke (SHS) exposure is associated with an increased risk of atherosclerotic heart disease and cardiac events. We sought to assess the effect of SHS on health-related quality of life (HRQOL) in patients with heart failure.
METHODS	Current nonsmokers with heart failure (N = 205) were enrolled in a cohort study. Exposure to SHS was assessed with a validated exposure questionnaire and a high-sensitivity assay for urinary cotinine level. Multidimensional HRQOL was evaluated with the RAND 36-Item Short Form Health Survey, which assesses 8 domains on a scale of 0 (worst) to 100 (best): physical functioning, bodily pain, role limitations due to physical health problems (role physical), role limitations due to emotional/personal problems (role emotional), emotional well-being, social functioning, energy/fatigue, and general health perceptions. A subset of patients (n = 75) agreed to assessment of functional status with a 6-minute walk test.
RESULTS	Self-reported exposure to SHS was associated with generally lower HRQOL scores in univariate analysis, with statistically and clinically significant reductions in 3 subscale scores: role physical (22.2 points), emotional well-being (11.0 points), and role emotional (16.2 points). Even after adjustment for clinical factors, such as age, sex, New York Heart Association class of heart failure, comorbidities, and medications, exposure to SHS remained an independent predictor of HRQOL scores in these domains. When increasing quartiles of urinary cotinine level were used as the exposure measure, qualitatively similar results were obtained.
CONCLUSIONS	Even low levels of SHS are associated with lower scores in several aspects of HRQOL. Physicians should advise patients with heart failure and their families to avoid SHS exposure.

표-108. PubMed 논문번호 22128036의 내용 요약

구분	내용
PubMed ID	22128036
TITLE	In utero exposure to maternal tobacco smoke and subsequent obesity, hypertension, and gestational diabetes among women in the MoBa cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103789
AUTHORS	Cupul-Uicab Lea A, Skjaerven Rolv, Haug Kjell, Melve Kari K, Engel Stephanie M, Longnecker Matthew P, Cupul-Uicab Lea A, Skjaerven Rolv, Haug Kjell, Melve Kari K, Engel Stephanie M, Longnecker Matthew P
BACKGROUND	Environmental factors influencing the developmental origins of health and disease need to be identified and investigated. In utero exposure to tobacco smoke has been associated with obesity and a small increase in blood pressure in children; however, whether there is a corresponding increased risk of conditions such as diabetes and hypertension during adulthood remains unclear.
OBJECTIVE	Our goal was to assess the association of self-reported in utero exposure to tobacco smoke with the prevalence of obesity, hypertension, type 2 diabetes mellitus (T2DM), and gestational diabetes mellitus (GDM) in women 14-47 years of age.
METHODS	We conducted a cross-sectional analysis of the Norwegian Mother and Child Cohort Study, which enrolled pregnant women in Norway from 1999 thorough 2008. Exposure to tobacco smoke in utero (yes vs. no) was ascertained on the baseline questionnaire (obtained at ~ 17 weeks' gestation); the outcomes were ascertained from the Medical Birth Registry of Norway and the questionnaire. Our analysis included 74,023 women.
RESULTS	Women exposed to tobacco smoke in utero had 1.53 times the odds of obesity [95% confidence interval (CI): 1.45, 1.61] relative to those unexposed, after adjusting for age, education, and personal smoking. After further adjustment for body mass index, the odds ratio for hypertension was 1.68 (95% CI: 1.19, 2.39); for T2DM 1.14 (95% CI: 0.79, 1.65); and for GDM 1.32 (95% CI: 1.10, 1.58) among exposed compared with unexposed.
CONCLUSIONS	Exposure to tobacco smoke in utero was associated with obesity, hypertension, and GDM in adult women. The possibility that the associations were attributable to unmeasured confounding cannot be excluded.

표-109. PubMed 논문번호 22138703의 내용 요약

구분	내용
PubMed ID	22138703
TITLE	Overt and latent cardiac effects of ozone inhalation in rats: evidence for autonomic modulation and increased myocardial vulnerability.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1104244
AUTHORS	Farraj Aimen K, Hazari Mehdi S, Winsett Darrell W, Kulukulualani Anthony, Carll Alex P, Haykal-Coates Najwa, Lamb Christina M, Lappi Edwin, Terrell Dock, Cascio Wayne E, Costa Daniel L, Farraj Aimen K, Hazari Mehdi S, Winsett Darrell W, Kulukulualani Anthony, Carll Alex P, Haykal-Coates Najwa, Lamb Christina M, Lappi Edwin, Terrell Dock, Cascio Wayne E, Costa Daniel L
BACKGROUND	Ozone (O_3) is a well-documented respiratory oxidant, but increasing epidemiological evidence points to extrapulmonary effects, including positive associations between ambient O_3 concentrations and cardiovascular morbidity and mortality.
OBJECTIVE	With preliminary reports linking O_3 exposure with changes in heart rate (HR), we investigated the hypothesis that a single inhalation exposure to O_3 will cause concentration-dependent autonomic modulation of cardiac function in rats.
METHODS	Rats implanted with telemeters to monitor HR and cardiac electrophysiology [electrocardiography (ECG)] were exposed once by whole-body inhalation for 4 hr to 0.2 or 0.8 ppm O_3 or filtered air. A separate cohort was tested for vulnerability to aconitine-induced arrhythmia 24 hr after exposure.
RESULTS	Exposure to 0.8 ppm O_3 caused bradycardia, PR prolongation, ST depression, and substantial increases in atrial premature beats, sinoatrial block, and atrioventricular block, accompanied by concurrent increases in several HR variability parameters that were suggestive of increased parasympathetic tone. Low-O_3 exposure failed to elicit any overt changes in autonomic tone, heart rhythm, or ECG. However, both 0.2 and 0.8 ppm O_3 increased sensitivity to aconitine-induced arrhythmia formation, suggesting a latent O_3 -induced alteration in myocardial excitability.
CONCLUSIONS	O_3 exposure causes several alterations in cardiac electrophysiology that are likely mediated by modulation of autonomic input to the heart. Moreover, exposure to low O_3 concentrations may cause subclinical effects that manifest only when triggered by a stressor, suggesting that the adverse health effects of ambient levels of air pollutants may be insidious and potentially underestimated.

표-110. PubMed 논문번호 22214647의 내용 요약

구분	내용
PubMed ID	22214647
TITLE	Long-term exposure to traffic-related air pollution associated with blood pressure and self-reported hypertension in a Danish cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103631
AUTHORS	S ø rensen Mette, Hoffmann Barbara, Hvidberg Martin, Ketzel Matthias, Jensen Steen Solvang, Andersen Zorana Jovanovic, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
BACKGROUND	Short-term exposure to air pollution has been associated with changes in blood pressure (BP) and emergency department visits for hypertension, but little is known about the effects of long-term exposure to traffic-related air pollution on BP and hypertension.
OBJECTIVES	We studied whether long-term exposure to air pollution is associated with BP and hypertension.
METHODS	In 1993-1997, 57,053 participants 50-64 years of age were enrolled in a population-based cohort study. Systolic and diastolic BP (SBP and DBP, respectively) were measured at enrollment. Self-reported incident hypertension during a mean follow-up of 5.3 years was assessed by questionnaire. We used a validated dispersion model to estimate residential long-term nitrogen oxides (NO(x)), a marker of traffic-related air pollution, for the 1- and 5-year periods prior to enrollment and before a diagnosis of hypertension. We conducted a cross-sectional analysis of associations between air pollution and BP at enrollment with linear regression, adjusting for traffic noise, measured short-term NO(x), temperature, relative humidity, and potential lifestyle confounders (n = 44,436). We analyzed incident hypertension with Cox regression, adjusting for traffic noise and potential confounders.
RESULTS	A doubling of NO(x) exposure during 1- and 5-year periods preceding enrollment was associated with 0.53-mmHg decreases [95% confidence interval (CI): -0.88, -0.19 mmHg] and 0.50-mmHg decreases (95% CI: -0.84, -0.16 mmHg) in SBP, respectively. Long-term exposure also was associated with a lower prevalence of baseline self-reported hypertension (per doubling of 5-year mean NO(x): odds ratio = 0.96; 95% CI: 0.91, 1.00), whereas long-term NO(x) exposure was not associated with incident self-reported hypertension during follow-up.
CONCLUSIONS	Long-term exposure to traffic-related air pollution was associated with a slightly lower prevalence of BP at baseline, but was not associated with incident hypertension.

표-111. PubMed 논문번호 22219348의 내용 요약

구분	내용
PubMed ID	22219348
TITLE	Air pollution and incidence of hypertension and diabetes mellitus in black women living in Los Angeles.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.111.052753
AUTHORS	Coogan Patricia F, White Laura F, Jerrett Michael, Brook Robert D, Su Jason G, Seto Edmund, Burnett Richard, Palmer Julie R, Rosenberg Lynn
BACKGROUND	Evidence suggests that longer-term exposure to air pollutants over years confers higher risks of cardiovascular morbidity and mortality than shorter-term exposure. One explanation is that the cumulative adverse effects that develop over longer durations lead to the genesis of chronic disease. Preliminary epidemiological and clinical evidence suggests that air pollution may contribute to the development of hypertension and type 2 diabetes mellitus.
METHODS AND RESULTS	We used Cox proportional hazards models to assess incidence rate ratios (IRRs) and 95% confidence intervals (CIs) for incident hypertension and diabetes mellitus associated with exposure to fine particulate matter (PM(2.5)) and nitrogen oxides in a cohort of black women living in Los Angeles. Pollutant levels were estimated at participants' residential addresses with land use regression models (nitrogen oxides) and interpolation from monitoring station measurements (PM(2.5)). Over follow-up from 1995 to 2005, 531 incident cases of hypertension and 183 incident cases of diabetes mellitus occurred. When pollutants were analyzed separately, the IRR for hypertension for a 10-μg/m(3) increase in PM(2.5) was 1.48 (95% CI, 0.95-2.31), and the IRR for the interquartile range (12.4 parts per billion) of nitrogen oxides was 1.14 (95% CI, 1.03-1.25). The corresponding IRRs for diabetes mellitus were 1.63 (95% CI, 0.78-3.44) and 1.25 (95% CI, 1.07-1.46). When both pollutants were included in the same model, the IRRs for PM(2.5) were attenuated and the IRRs for nitrogen oxides were essentially unchanged for both outcomes.
CONCLUSION	Our results suggest that exposure to air pollutants, especially traffic-related pollutants, may increase the risk of type 2 diabetes mellitus and possibly of hypertension.

표-112. PubMed 논문번호 22237295의 내용 요약

구분	내용
PubMed ID	22237295
TITLE	Air pollution and markers of coagulation, inflammation, and endothelial function: associations and epigene-environment interactions in an elderly cohort.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e31824523f0
AUTHORS	Bind Marie-Abele, Baccarelli Andrea, Zanobetti Antonella, Tarantini Letizia, Suh Helen, Vokonas Pantel, Schwartz Joel
BACKGROUND	Previous studies suggest that air pollution is related to thrombosis, inflammation, and endothelial dysfunction. Mechanisms and sources of susceptibility are still unclear. One possibility is that these associations can be modified by DNA methylation states.
METHODS	We conducted a cohort study with repeated measurements of fibrinogen, C-reactive protein, intercellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1) in 704 elderly men participating in the Veterans Administration Normative Aging Study (2000-2009). We investigated short- and intermediate-term air pollution effects on these blood markers, and epigene-environment interactions by DNA methylation of Alu, LINE-1, tissue factor (F3), Toll-like receptor 2 (TLR-2), and ICAM-1.
RESULTS	We found effects of particle number, black carbon, nitrogen dioxide (NO(2)), and carbon monoxide (CO) on fibrinogen. Ozone was a predictor of C-reactive protein and ICAM-1. Particle number, black carbon, NO(2), CO, PM(2.5), and sulfates were associated with ICAM-1 and VCAM-1. An interquartile range increase in 24-hour exposure for NO(2) was associated with a 1.7% (95% confidence interval = 0.2%-3.3%) increase in fibrinogen for ozone; a 10.8% (2.2%-20.0%) increase in C-reactive protein for particle number; a 5.9% (3.6%-8.3%) increase in ICAM-1; and for PM(2.5), a 3.7% (1.7%-5.8%) increase in VCAM-1. The air pollution effect was stronger among subjects having higher Alu, lower LINE-1, tissue factor, or TLR-2 methylation status.
CONCLUSION	We observed associations of traffic-related pollutants on fibrinogen, and both traffic and secondary particles on C-reactive protein, ICAM-1, and VCAM-1. There was effect modification by DNA methylation status, indicating that epigenetic states can convey susceptibility to air pollution.

표-113. PubMed 논문번호 22241632의 내용 요약

구분	내용
PubMed ID	22241632
TITLE	Occupational exposure to particles and incidence of stroke.
JOURNAL	Scandinavian journal of work, environment & health: 10.5271/sjweh.3271
AUTHORS	Sj□gren Bengt, L□nn Maria, Fremling Karin, Feychting Maria, Nise Gun, Kauppinen Timo, Plato Nils, Wiebert Pernilla, Gustavsson Per, Sj□gren Bengt, L□nn Maria, Fremling Karin, Feychting Maria, Nise Gun, Kauppinen Timo, Plato Nils, Wiebert Pernilla, Gustavsson Per
OBJECTIVES	This paper aims to investigate the relation between occupational exposure to particles, particle size, and the incidence of ischemic and hemorrhagic stroke.
METHODS	The cohort included all manual workers identified from the Swedish National Census in 1980, who were alive as of 1 January 1987. First time events of ischemic or hemorrhagic stroke during the period 1987-2005 were identified through linkage to the Hospital Discharge Register and the National Cause of Death Register. A job-exposure matrix for exposure to small (<1 □m) and large (>1 □m) particles was developed and applied. Hazard ratios (HR) were estimated by Cox regression with adjustment for age, socioeconomic group, and residential area.
RESULTS	Increased HR of ischemic stroke were found among both women and men occupationally exposed to small as well as large particles for ≥5 years. The risks were higher for workers exposed for ≥5 years compared to "ever exposed" participants indicating a dose-response relationship, but no trend with exposure intensity was observed. A tentative association between particle exposure and hemorrhagic stroke was also found.
CONCLUSIONS	Occupational exposure to small and large particles was associated with increased risks of ischemic stroke. Further studies are needed to explore the relationships between exposure to different types of particles and various doses and the occurrence of stroke among women as well as men.

표-114. PubMed 논문번호 22309254의 내용 요약

구분	내용
PubMed ID	22309254
TITLE	Lack of association between air pollutant exposure and short-term risk of ischaemic stroke in Lyon, France.
JOURNAL	International journal of stroke : official journal of the International Stroke Society: 10.1111/j.1747-4949.2011.00737.x
AUTHORS	Mechtouff Laura, Canoui-Poitrine Florence, Schott Anne-Marie, Nighoghossian Norbert, Trouillas Paul, Termoz Anne, Porthault-Chatard Sylvie, David Jean-Stephane, Chasles Virginie, Derex Laurent
BACKGROUND	Some observational and experimental studies have suggested a short-term relationship between air pollutants and ischaemic stroke; however, the results conflict.
AIMS	The objective of this study was to investigate the association between particulate matter less than 2·5 and 10 microns in aerodynamic diameter, nitrogen dioxide, sulphur dioxide and ozone, and short-term risk of ischaemic stroke in Lyon, France.
METHODS	The AVC69 study was a multicenter cohort study in which all consecutive adult patients admitted to one of the emergency or neurological departments of the Rh□ne area for suspicion of stroke were included during a seven-month period. Only patients with ischaemic stroke living within the study area, composed of Lyon and 18 neighbouring communities with homogenous air pollutants exposure, formed the basis of our study. We adopted a time-stratified case-crossover design to analyse the short-term effect (up to two-days) of air pollutants on ischaemic stroke incidence. Models were adjusted for temperature, variation of atmospheric pressure, minimal relative humidity, influenza epidemics, pollen count, and holidays. Stratified analyses by gender and class age were performed. Different lag times were analysed.
RESULTS	376 patients were included. Mean age was 76·6 years (± 13·7). 53·7% were women. No association was observed between air pollutants and short-term risk of ischaemic stroke after adjustment for main confounding factors. Results remained unchanged whatever the gender or age.
CONCLUSIONS	These results suggest a lack of association between air pollutant exposure and short-term risk of ischaemic stroke in a French urban area.

표-115. PubMed 논문번호 22313724의 내용 요약

구분	내용
PubMed ID	22313724
TITLE	Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1104049
AUTHORS	Crouse Dan L, Peters Paul A, van Donkelaar Aaron, Goldberg Mark S, Villeneuve Paul J, Brion Orly, Khan Saeeda, Atari Dominic Odwa, Jerrett Michael, Pope C Arden, Brauer Michael, Brook Jeffrey R, Martin Randall V, Stieb David, Burnett Richard T
BACKGROUND	Few cohort studies have evaluated the risk of mortality associated with long-term exposure to fine particulate matter [≤ 2.5 μm in aerodynamic diameter (PM(2.5))]. This is the first national-level cohort study to investigate these risks in Canada.
OBJECTIVE	We investigated the association between long-term exposure to ambient PM(2.5) and cardiovascular mortality in nonimmigrant Canadian adults.
METHODS	We assigned estimates of exposure to ambient PM(2.5) derived from satellite observations to a cohort of 2.1 million Canadian adults who in 1991 were among the 20% of the population mandated to provide detailed census data. We identified deaths occurring between 1991 and 2001 through record linkage. We calculated hazard ratios (HRs) and 95% confidence intervals (CIs) adjusted for available individual-level and contextual covariates using both standard Cox proportional survival models and nested, spatial random-effects survival models.
RESULTS	Using standard Cox models, we calculated HRs of 1.15 (95% CI: 1.13, 1.16) from nonaccidental causes and 1.31 (95% CI: 1.27, 1.35) from ischemic heart disease for each 10-μg/m(3) increase in concentrations of PM(2.5). Using spatial random-effects models controlling for the same variables, we calculated HRs of 1.10 (95% CI: 1.05, 1.15) and 1.30 (95% CI: 1.18, 1.43), respectively. We found similar associations between nonaccidental mortality and PM2.5 based on satellite-derived estimates and ground-based measurements in a subanalysis of subjects in 11 cities.
CONCLUSIONS	In this large national cohort of nonimmigrant Canadians, mortality was associated with long-term exposure to PM(2.5). Associations were observed with exposures to PM(2.5) at concentrations that were predominantly lower (mean, 8.7 μg/m(3); interquartile range, 6.2 μg/m(3)) than those reported previously.

표-116. PubMed 논문번호 22337682의 내용 요약

구분	내용
PubMed ID	22337682
TITLE	Main air pollutants and myocardial infarction: a systematic review and meta-analysis.
JOURNAL	JAMA: 10.1001/jama.2012.126
AUTHORS	Mustafic Hazrije, Jabre Patricia, Caussin Christophe, Murad Mohammad H, Escolano Sylvie, Tafflet Muriel, P□rier Marie-C□cile, Marijon Eloi, Vernerey Dewi, Empana Jean-Philippe, Jouven Xavier
CONTEXT	Short-term exposure to high levels of air pollution may trigger myocardial infarction (MI), but this association remains unclear.
OBJECTIVE	To assess and quantify the association between short-term exposure to major air pollutants (ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, and particulate matter ≤10 μm [PM(10)] and ≤2.5 μm [PM(2.5)] in diameter) on MI risk.
DATA SOURCES	EMBASE, Ovid MEDLINE in-process and other nonindexed citations, and Ovid MEDLINE (between 1948 and November 28, 2011), and EBM Reviews-Cochrane Central Register of Controlled Trials and EBM Reviews-Cochrane Database of Systematic Reviews (between 2005 and November 28, 2011) were searched for a combination of keywords related to the type of exposure (air pollution, ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, PM(10), and PM(2.5)) and to the type of outcome (MI, heart attack, acute coronary syndrome).
STUDY SELECTION	Two independent reviewers selected studies of any study design and in any language, using original data and investigating the association between short-term exposure (for up to 7 days) to 1 or more air pollutants and subsequent MI risk. Selection was performed from abstracts and titles and pursued by reviewing the full text of potentially eligible studies.
DATA EXTRACTION	Descriptive and quantitative information was extracted from each selected study. Using a random effects model, relative risks (RRs) and 95% CIs were calculated for each increment of 10 μg/m(3) in pollutant concentration, with the exception of carbon monoxide, for which an increase of 1 mg/m(3) was considered.
DATA SYNTHESIS	After a detailed screening of 117 studies, 34 studies were identified. All the main air pollutants, with the exception of ozone, were significantly associated with an increase in MI risk (carbon monoxide: 1.048; 95% CI, 1.026-1.070; nitrogen dioxide: 1.011; 95% CI, 1.006-1.016; sulfur dioxide: 1.010; 95% CI, 1.003-1.017; PM(10): 1.006; 95% CI, 1.002-1.009; and PM(2.5): 1.025; 95% CI, 1.015-1.036). For ozone, the RR was 1.003 (95% CI, 0.997-1.010; P = .36). Subgroup analyses provided results comparable with those of the overall analyses. Population attributable fractions ranged between 0.6% and 4.5%, depending on the air pollutant.
CONCLUSION	All the main air pollutants, with the exception of ozone, were significantly associated with a near-term increase in MI risk.

표-117. PubMed 논문번호 22345167의 내용 요약

구분	내용
PubMed ID	22345167
TITLE	Parental smoking in childhood and brachial artery flow-mediated dilatation in young adults: the Cardiovascular Risk in Young Finns study and the Childhood Determinants of Adult Health study.
JOURNAL	Arteriosclerosis, thrombosis, and vascular biology: 10.1161/ATVBAHA.111.243261
AUTHORS	Juonala Markus, Magnussen Costan G, Venn Alison, Gall Seana, K□h□nen Mika, Laitinen Tomi, Taittonen Leena, Lehtim□ki Terho, Jokinen Eero, Sun Cong, Viikari Jorma S A, Dwyer Terence, Raitakari Olli T
OBJECTIVE	Passive smoking has been associated with increased cardiovascular morbidity. The present study aimed to examine the long-term effects of childhood exposure to tobacco smoke on endothelium-dependent vasodilation in adults.
METHODS AND RESULTS	The analyses were based on 2171 participants in the population-based Cardiovascular Risk in Young Finns (N=2067) and Childhood Determinants of Adult Health (N=104) studies who had measures of conventional risk factors (lipids, blood pressure, adiposity, socioeconomic status) and self-reported parental smoking status when aged 3 to 18 years at baseline. They were re-examined 19 to 27 years later when aged 28 to 45 years. Brachial artery flow-mediated dilatation was measured at follow-up with ultrasound. In analyses adjusting for age, sex, and childhood risk factors, flow-mediated dilatation was reduced among participants who had parents that smoked in youth compared to those whose parents did not smoke (Young Finns: 9.2 ± 0.1% (mean ± SEM) versus 8.6 ± 0.1%, P=0.001; Childhood Determinants of Adult Health: 7.4 ± 0.6% versus 4.9 ± 0.9%, P=0.04). These effects remained after adjustment for adult risk factors including own smoking status (Young Finns, P=0.003; Childhood Determinants of Adult Health, P=0.03).
CONCLUSIONS	Parental smoking in youth is associated with reduced flow-mediated dilatation in young adulthood measured over 20 years later. These findings suggest that passive exposure to cigarette smoke among children might cause irreversible impairment in endothelium-dependent vasodilation.

표-118. PubMed 논문번호 22357786의 내용 요약

구분	내용
PubMed ID	22357786
TITLE	Work-related cardiovascular disease risk factors using a socioecological approach: implications for practice and research.
JOURNAL	European journal of cardiovascular nursing : journal of the Working Group on Cardiovascular Nursing of the European Society of Cardiology: 10.1177/1474515111430890
AUTHORS	Hwang Won Ju, Hong Oisaeng
BACKGROUND	Cardiovascular disease (CVD) is a leading cause of mortality. Numerous investigations have linked occupational factors and CVD. Occupational factors such as overtime work have an enormous effect on the CVD risk of industrial workers. However, risk factors for CVD are not systematically reviewed in the workplace. The purpose of the paper is to review work-related risk factors for CVD.
METHODS	A systematic review of work-related CVD risk factors was performed, yielding 180 articles. All articles were assessed in relation to inclusion and exclusion criteria, resulting in 44 articles being reviewed. The sole inclusion criteria was work-related environmental factors and intra/inter-personal factors (psychosocial factors), which is based on the socioecological perspective. The articles were also assessed regarding the quality of each study using the scoring methods developed by Cesario et al. and Brown et al.
CONCLUSION	The literature review demonstrated that work environment factors such as shift work, overtime work, and noise and chemical exposures; and psychosocial factors such as job stress, social support, and socioeconomic status cannot be explained or intervened by one single risk factor. Furthermore, certain occupational factors were shown to aggravate or attenuate other risk factors. The implication of these findings is to incorporate work-related environmental and psychosocial factors into assessment of the patient's CVD risks and intervention plan. Future research should also incorporate a well-defined conceptual framework to address the effects of work-related environmental and psychosocial factors on CVD among CVD patients.

표-119. PubMed 논문번호 22383587의 내용 요약

구분	내용
PubMed ID	22383587
TITLE	Association between long-term exposure to traffic particles and blood pressure in the Veterans Administration Normative Aging Study.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2011-100268
AUTHORS	Schwartz Joel, Alexeeff Stacey E, Mordukhovich Irina, Gryparis Alexandros, Vokonas Pantel, Suh Helen, Coull Brent A
OBJECTIVES	Particulate air pollution is associated with cardiovascular events, but the mechanisms are not fully understood. The main objective was to assess the relationship between long-term exposure to traffic-related air pollution and blood pressure (BP).
METHODS	The authors used longitudinal data from 853 elderly men participating in the Veterans Administration Normative Aging Study, followed during 1996-2008. Long-term average exposures to traffic particles were created from daily predictions of black carbon (BC) exposure at the geocoded address of each subject, using a validated spatiotemporal model based on ambient monitoring at 82 Boston-area locations. The authors examined the association of these exposures with BP using a mixed model. The authors included the following covariates: age, body mass index, smoking, alcohol, fasting glucose, creatinine clearance, use of cardiovascular medication, education, census-level poverty, day of week and season of clinical visit.
RESULTS	The authors found significant positive associations between 1-year average BC exposure and both systolic and diastolic blood pressure. An IQR increase in 1-year average BC exposure (0.32 μg/m(3)) was associated with a 2.64 mm Hg increase in systolic blood pressure (95% CI 1.47 to 3.80) and a 2.41 mm Hg increase in diastolic blood pressure (95% CI 1.77 to 3.05).
CONCLUSIONS	Long-term exposure to traffic particles is associated with increased BP, which may explain part of the association with myocardial infarctions and cardiovascular deaths reported in cohort studies.

표-120. PubMed 논문번호 22383660의 내용 요약

구분	내용
PubMed ID	22383660
TITLE	Acute pulmonary admissions following implementation of a national workplace smoking ban.
JOURNAL	Chest: 10.1378/chest.11-2757
AUTHORS	Kent Brian D, Sulaiman Imran, Nicholson Trevor T, Lane Stephen J, Moloney Edward D
BACKGROUND	The implementation of workplace smoking bans has contributed to a significant reduction in the incidence of acute coronary syndrome admissions, but their influence on adult acute pulmonary disease admissions is unclear. We sought to assess the impact of a national smoking ban on nationwide admissions of individuals of working age with acute pulmonary illness.
METHODS	Data relating to emergency hospital admissions of subjects aged 20 to 70 years preceding and succeeding the implementation of the Irish smoking ban were obtained from a central registry. Population, weather, pollution, and influenza data were obtained from the relevant authorities. Poisson regression analysis was used to assess adjusted risk of emergency hospital admission following implementation of the smoking ban.
RESULTS	Overall admissions with pulmonary illness decreased from 439 per 100,000 population per annum to 396 per 100,000 population per annum following the ban (unadjusted relative risk [RR], 0.91; 95% CI, 0.83-0.99; P = .048). This persisted following adjustment for confounding factors (adjusted RR, 0.85; 95% CI, 0.72-0.99; P = .04) and was most marked among younger age groups and in admissions due to asthma (adjusted RR, 0.60; 95% CI, 0.39-0.91; P = .016). Admissions with acute coronary syndromes (adjusted RR, 0.82; 95% CI, 0.70-0.97; P = .02), but not stroke (adjusted RR, 0.93; 95% CI, 0.73-1.20; P = .60), were also reduced.
CONCLUSIONS	The implementation of a nationwide workplace smoking ban is associated with a decline in admissions with acute pulmonary disease among specific age groups and an overall reduction in asthma admissions. This may result from reduced exposure of vulnerable individuals to environmental tobacco smoke, emphasizing the potential benefit of legislation reducing second-hand smoke exposure.

표-121. PubMed 논문번호 22389220의 내용 요약

구분	내용
PubMed ID	22389220
TITLE	Reducing personal exposure to particulate air pollution improves cardiovascular health in patients with coronary heart disease.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1103898
AUTHORS	Langrish Jeremy P, Li Xi, Wang Shengfeng, Lee Matthew M Y, Barnes Gareth D, Miller Mark R, Cassee Flemming R, Boon Nicholas A, Donaldson Ken, Li Jing, Li Liming, Mills Nicholas L, Newby David E, Jiang Lixin
BACKGROUND	Air pollution exposure increases cardiovascular morbidity and mortality and is a major global public health concern.
OBJECTIVES	We investigated the benefits of reducing personal exposure to urban air pollution in patients with coronary heart disease.
METHODS	In an open randomized crossover trial, 98 patients with coronary heart disease walked on a predefined route in central Beijing, China, under different conditions: once while using a highly efficient face mask, and once while not using the mask. Symptoms, exercise, personal air pollution exposure, blood pressure, heart rate, and 12-lead electrocardiography were monitored throughout the 24-hr study period.
RESULTS	Ambient air pollutants were dominated by fine and ultrafine particulate matter (PM) that was present at high levels [74 $\mu g/m^3$ for PM(2.5) (PM with aerodynamic diamater 〈2.5 □m)]. Consistent with traffic-derived sources, this PM contained organic carbon and polycyclic aromatic hydrocarbons and was highly oxidizing, generating large amounts of free radicals. The face mask was well tolerated, and its use was associated with decreased self-reported symptoms and reduced maximal ST segment depression (-142 vs. -156 μV, p = 0.046) over the 24-hr period. When the face mask was used during the prescribed walk, mean arterial pressure was lower (93 ± 10 vs. 96 ± 10 mmHg, p = 0.025) and heart rate variability increased (high-frequency power: 54 vs. 40 $msec^2$, p = 0.005; high-frequency normalized power: 23.5 vs. 20.5 msec, p = 0.001; root mean square successive differences: 16.7 vs. 14.8 msec, p = 0.007). However, mask use did not appear to influence heart rate or energy expenditure.
CONCLUSIONS	Reducing personal exposure to air pollution using a highly efficient face mask appeared to reduce symptoms and improve a range of cardiovascular health measures in patients with coronary heart disease. Such interventions to reduce personal exposure to PM air pollution have the potential to reduce the incidence of cardiovascular events in this highly susceptible population.

표-122. PubMed 논문번호 22450694의 내용 요약

구분	내용
PubMed ID	22450694
TITLE	PM mass concentration and PM oxidative potential in relation to carotid intima-media thickness.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e31824e613e
AUTHORS	Tonne Cathryn, Yanosky Jeff D, Beevers Sean, Wilkinson Paul, Kelly Frank J
BACKGROUND	There is limited evidence on whether particulate matter (PM) can augment the progression of atherosclerosis; furthermore, the specific attributes of PM responsible for health effects are unclear. We developed models to predict exposure to PM <10 μm (PM10) and also to predict a measure of oxidative potential (the capacity of particles to induce oxidative damage). Our objectives were (1) to estimate the association between PM10 and carotid intima-media thickness, a measure of subclinical atherosclerosis, and (2) to compare this association with that of PM10 weighted by its oxidative potential (PM10*OP).
METHODS	Analysis was based on 2348 participants of the Whitehall II cohort of British civil servants who had intima-media thickness measured between 2003 and 2005 and lived in Greater London. Weekly PM10 and PM10*OP were predicted at each participant's residence. Primary exposure metrics were defined as PM10 and PM10*OP averaged over the year before scan. We estimated associations between exposure metrics and intima-media thickness using generalized linear regression models.
RESULTS	An interquartile range increase (5.2 μgm(-3)) in PM10 was associated with a 5.0% (95% confidence interval = 1.9% to 8.3%) increase in intima-media thickness after covariate adjustment. The association for an interquartile range change in PM10*OP (1.5 m(-3)) was weaker: 1.2% (0.2% to 2.2%).
CONCLUSIONS	These findings support a relationship between PM exposure and atherosclerosis. PM weighted by this particular measure of oxidative potential was not more predictive of the extent of atherosclerosis than PM mass concentration.

표-123. PubMed 논문번호 22490471의 내용 요약

구분	내용
PubMed ID	22490471
TITLE	Integrating a quantitative risk appraisal in a health impact assessment: analysis of the novel smoke-free policy in Hungary.
JOURNAL	European journal of public health: 10.1093/eurpub/cks018
AUTHORS	□d□m Bal□zs, Moln□r □gnes, Gulis Gabriel, □d□ny R□za, □d□m Bal□zs, Moln□r □gnes, Gulis Gabriel, □d□ny R□za
BACKGROUND	Although the quantification of health outcomes in a health impact assessment (HIA) is scarce in practice, it is preferred by policymakers, as it assists various aspects of the decision-making process. This article provides an example of integrating a quantitative risk appraisal in an HIA performed for the recently adopted Hungarian anti-smoking policy which introduced a smoking ban in closed public places, workplaces and public transport vehicles, and is one of the most effective measures to decrease smoking-related ill health.
METHODS	A comprehensive, prospective HIA was conducted to map the full impact chain of the proposal. Causal pathways were prioritized in a transparent process with special attention given to those pathways for which measures of disease burden could be calculated for the baseline and predicted future scenarios.
RESULTS	The proposal was found to decrease the prevalence of active and passive smoking and result in a considerably positive effect on several diseases, among which lung cancer, chronic pulmonary diseases, coronary heart diseases and stroke have the greatest importance. The health gain calculated for the quantifiable health outcomes is close to 1700 deaths postponed and 16,000 life years saved annually in Hungary.
CONCLUSION	The provision of smoke-free public places has an unambiguously positive impact on the health of the public, especially in a country with a high burden of smoking-related diseases. The study described offers a practical example of applying quantification in an HIA, thereby promoting its incorporation into political decision making.

표-124. PubMed 논문번호 22507957의 내용 요약

구분	내용
PubMed ID	22507957
TITLE	The biological effects of subacute inhalation of diesel exhaust following addition of cerium oxide nanoparticles in atherosclerosis-prone mice.
JOURNAL	Environmental research: 10.1016/j.envres.2012.03.004
AUTHORS	Cassee Flemming R, Campbell Arezoo, Boere A John F, McLean Steven G, Duffin Rodger, Krystek Petra, Gosens Ilse, Miller Mark R
BACKGROUND	Cerium oxide (CeO(2)) nanoparticles improve the burning efficiency of fuel, however, little is known about health impacts of altered emissions from the vehicles.
METHODS	Atherosclerosis-prone apolipoprotein E knockout (ApoE(-/-)) mice were exposed by inhalation to diluted exhaust (1.7 mg/m(3), 20, 60 or 180 min, 5 day/week, for 4 weeks), from an engine using standard diesel fuel (DE) or the same diesel fuel containing 9 ppm cerium oxide nanoparticles (DCeE). Changes in hematological indices, clinical chemistry, atherosclerotic burden, tissue levels of inflammatory cytokines and pathology of the major organs were assessed.
RESULTS	Addition of CeO(2) to fuel resulted in a reduction of the number (30%) and surface area (10%) of the particles in the exhaust, whereas the gaseous co-pollutants were increased (6-8%). There was, however, a trend towards an increased size and complexity of the atherosclerotic plaques following DE exposure, which was not evident in the DCeE group. There were no clear signs of altered hematological or pathological changes induced by either treatment. However, levels of proinflammatory cytokines were modulated in a brain region and liver following DCeE exposure.
CONCLUSIONS	These results imply that addition of CeO(2) nanoparticles to fuel decreases the number of particles in exhaust and may reduce atherosclerotic burden associated with exposure to standard diesel fuel. From the extensive assessment of biological parameters performed, the only concerning effect of cerium addition was a slightly raised level of cytokines in a region of the central nervous system. Overall, the use of cerium as a fuel additive may be a potentially useful way to limit the health effects of vehicle exhaust. However, further testing is required to ensure that such an approach is not associated with a chronic inflammatory response which may eventually cause long-term health effects.

표-125. PubMed 논문번호 22514211의 내용 요약

구분	내용
PubMed ID	22514211
TITLE	Omega-3 fatty acid supplementation appears to attenuate particulate air pollution-induced cardiac effects and lipid changes in healthy middle-aged adults.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1104472
AUTHORS	Tong Haiyan, Rappold Ana G, Diaz-Sanchez David, Steck Susan E, Berntsen Jon, Cascio Wayne E, Devlin Robert B, Samet James M
BACKGROUND	Air pollution exposure has been associated with adverse cardiovascular health effects. Findings of a recent epidemiological study suggested that omega-3 fatty acid (fish oil) supplementation blunted cardiac responses to air pollution exposure.
OBJECTIVES	We conducted a randomized, controlled exposure study to evaluate the efficacy of fish oil supplements in attenuating adverse cardiac effects of exposure to concentrated ambient fine and ultrafine particulate matter (CAP).
METHODS	Twenty-nine healthy middle-aged participants (mean, 58 ± 1 years of age) were supplemented in a randomized, double-blinded manner with 3 g/day of either fish oil or olive oil for 4 weeks before sequential chamber exposure to filtered air and CAP (mean mass concentration 278 ± 19 □g/m3) for 2 hr. Cardiac responses were assessed by comparing time and frequency domain changes in heart rate variability (HRV) and electrocardiographic repolarization changes measured before, immediately after, and 20 hr after exposure. Changes in plasma lipids were also evaluated at these time points.
RESULTS	Fish oil supplementation appeared to attenuate CAP-induced reductions in high-frequency/low-frequency ratio, as well as elevations in normalized low-frequency HRV and prolongation of the QT interval corrected for heart rate (QTc). Very low-density lipoprotein and triglyceride concentrations increased significantly immediately after exposure to CAP in participants supplemented with olive oil, but not in those supplemented with fish oil.
CONCLUSIONS	Exposure of healthy middle-aged adults to CAP for 2 hr induced acute cardiac and lipid changes after supplementation with olive oil, but not fish oil. Our findings suggest that omega-3 fatty acid supplements offer protection against the adverse cardiac and lipid effects associated with air pollution exposure.

표-126. PubMed 논문번호 22529751의 내용 요약

구분	내용
PubMed ID	22529751
TITLE	Long-term exposure to silica dust and risk of total and cause-specific mortality in Chinese workers: a cohort study.
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1001206
AUTHORS	Chen Weihong, Liu Yuewei, Wang Haijiao, Hnizdo Eva, Sun Yi, Su Liangping, Zhang Xiaokang, Weng Shaofan, Bochmann Frank, Hearl Frank J, Chen Jingqiong, Wu Tangchun
BACKGROUND	Human exposure to silica dust is very common in both working and living environments. However, the potential long-term health effects have not been well established across different exposure situations.
METHODS AND FINDINGS	We studied 74,040 workers who worked at 29 metal mines and pottery factories in China for 1 y or more between January 1, 1960, and December 31, 1974, with follow-up until December 31, 2003 (median follow-up of 33 y). We estimated the cumulative silica dust exposure (CDE) for each worker by linking work history to a job-exposure matrix. We calculated standardized mortality ratios for underlying causes of death based on Chinese national mortality rates. Hazard ratios (HRs) for selected causes of death associated with CDE were estimated using the Cox proportional hazards model. The population attributable risks were estimated based on the prevalence of workers with silica dust exposure and HRs. The number of deaths attributable to silica dust exposure among Chinese workers was then calculated using the population attributable risk and the national mortality rate. We observed 19,516 deaths during 2,306,428 person-years of follow-up. Mortality from all causes was higher among workers exposed to silica dust than among non-exposed workers (993 versus 551 per 100,000 person-years). We observed significant positive exposure-response relationships between CDE (measured in milligrams/cubic meter-years, i.e., the sum of silica dust concentrations multiplied by the years of silica exposure) and mortality from all causes (HR 1.026, 95% confidence interval 1.023-1.029), respiratory diseases (1.069, 1.064-1.074), respiratory tuberculosis (1.065, 1.059-1.071), and cardiovascular disease (1.031, 1.025-1.036). Significantly elevated standardized mortality ratios were observed for all causes (1.06, 95% confidence interval 1.01-1.11), ischemic heart disease (1.65, 1.35-1.99), and pneumoconiosis (11.01, 7.67-14.95) among workers exposed to respirable silica concentrations equal to or lower than 0.1 mg/m(3). After adjustment for potential confounders, including smoking, silica dust exposure accounted for 15.2% of all deaths in this study. We estimated that 4.2% of deaths (231,104 cases) among Chinese workers were attributable to silica dust exposure. The limitations of this study included a lack of data on dietary patterns and leisure time physical activity, possible underestimation of silica dust exposure for individuals who worked at the mines/factories before 1950, and a small number of deaths (4.3%) where the cause of death was based on oral reports from relatives.
CONCLUSIONS	Long-term silica dust exposure was associated with substantially increased mortality among Chinese workers. The increased risk was observed not only for deaths due to respiratory diseases and lung cancer, but also for deaths due to cardiovascular disease. Please see later in the article for the Editors' Summary.

표-127. PubMed 논문번호 22534026의 내용 요약

구분	내용
PubMed ID	22534026
TITLE	Modeling the residential infiltration of outdoor PM(2.5) in the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air).
JOURNAL	Environmental health perspectives: 10.1289/ehp.1104447
AUTHORS	Allen Ryan W, Adar Sara D, Avol Ed, Cohen Martin, Curl Cynthia L, Larson Timothy, Liu L-J Sally, Sheppard Lianne, Kaufman Joel D
BACKGROUND	Epidemiologic studies of fine particulate matter [aerodynamic diameter ≤ 2.5 μm (PM(2.5))] typically use outdoor concentrations as exposure surrogates. Failure to account for variation in residential infiltration efficiencies (F(inf)) will affect epidemiologic study results.
OBJECTIVE	We aimed to develop models to predict F(inf) for > 6,000 homes in the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air), a prospective cohort study of PM(2.5) exposure, subclinical cardiovascular disease, and clinical outcomes.
METHODS	We collected 526 two-week, paired indoor-outdoor PM(2.5) filter samples from a subset of study homes. PM(2.5) elemental composition was measured by X-ray fluorescence, and F(inf) was estimated as the indoor/outdoor sulfur ratio. We regressed F(inf) on meteorologic variables and questionnaire-based predictors in season-specific models. Models were evaluated using the R^2 and root mean square error (RMSE) from a 10-fold cross-validation.
RESULTS	The mean ± SD F(inf) across all communities and seasons was 0.62 ± 0.21, and community-specific means ranged from 0.47 ± 0.15 in Winston-Salem, North Carolina, to 0.82 ± 0.14 in New York, New York. F(inf) was generally greater during the warm (> 18° C) season. Central air conditioning (AC) use, frequency of AC use, and window opening frequency were the most important predictors during the warm season; outdoor temperature and forced-air heat were the best cold-season predictors. The models predicted 60% of the variance in 2-week F(inf), with an RMSE of 0.13.
CONCLUSIONS	We developed intuitive models that can predict F(inf) using easily obtained variables. Using these models, MESA Air will be the first large epidemiologic study to incorporate variation in residential F(inf) into an exposure assessment.

표-128. PubMed 논문번호 22569477의 내용 요약

구분	내용
PubMed ID	22569477
TITLE	Mortality among population with exposure to industrial air pollution containing nickel and other toxic metals.
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0b013e3182492050
AUTHORS	Pasanen Kari, Pukkala Eero, Turunen Anu W, Patama Toni, Jussila Ilkka, Makkonen Sari, Salonen Raimo O, Verkasalo Pia K
OBJECTIVE	To assess disease mortality among people with exposure to metal-rich particulate air pollution.
METHODS	We conducted a cohort study on mortality from 1981 to 2005 among 33,573 people living near a nickel/copper smelter in Harjavalta, Finland. Nickel concentration in soil humus was selected as an indicator for long-term exposure. Relative risks - adjusted for age, socioeconomic status, and calendar period - were calculated for three exposure zones.
RESULTS	The relative risks for diseases of the circulatory system by increasing exposure were 0.93 (95% confidence interval = 0.79 to 1.09), 1.20 (1.04 to 1.39), and 1.18 (1.00 to 1.39) among men and 1.01 (0.88 to 1.17), 1.20 (1.04 to 1.38), and 1.14 (0.97 to 1.33) among women. Exclusion of smelter workers from the cohort did not materially change the results.
CONCLUSIONS	Long-term environmental exposure to metal-rich air pollution was associated with increased mortality from circulatory diseases.

표-129. PubMed 논문번호 22592907의 내용 요약

구분	내용
PubMed ID	22592907
TITLE	Air pollution interventions and their impact on public health.
JOURNAL	International journal of public health: 10.1007/s00038-012-0369-6
AUTHORS	Henschel Susann, Atkinson Richard, Zeka Ariana, Le Tertre Alain, Analitis Antonis, Katsouyanni Klea, Chanel Olivier, Pascal Mathilde, Forsberg Bertil, Medina Sylvia, Goodman Patrick G
INTRODUCTION	Numerous epidemiological studies have found a link between air pollution and health. We are reviewing a collection of published intervention studies with particular focus on studies assessing both improvements in air quality and associated health effects.
METHODS	Interventions, defined as events aimed at reducing air pollution or where reductions occurred as a side effect, e.g. strikes, German reunification, from the 1960s onwards were considered for inclusion. This review is not a complete record of all existing air pollution interventions. In total, 28 studies published in English were selected based on a systematic search of internet databases.
RESULTS	Overall air pollution interventions have succeeded at improving air quality. Consistently published evidence suggests that most of these interventions have been associated with health benefits, mainly by the way of reduced cardiovascular and/or respiratory mortality and/or morbidity. The decrease in mortality from the majority of the reviewed interventions has been estimated to exceed the expected predicted figures based on the estimates from time-series studies.
CONCLUSION	There is consistent evidence that decreased air pollution levels following an intervention resulted in health benefits for the assessed population.

표-130. PubMed 논문번호 22628493의 내용 요약

구분	내용
PubMed ID	22628493
TITLE	Secondhand smoke exposure predicted COPD and other tobacco-related mortality in a 17-year cohort study in China.
JOURNAL	Chest: 10.1378/chest.11-2884
AUTHORS	He Yao, Jiang Bin, Li Liang Shou, Li Lan Sun, Ko Lisanne, Wu Lei, Sun Dong Ling, He Shu Fang, Liang Bao Qing, Hu Frank B, Lam Tai Hing
BACKGROUND	Prospective evidence on the association between secondhand smoke (SHS) and COPD and ischemic stroke is scarce.
METHODS	We prospectively examined the relationship between SHS and major tobacco-related deaths, particularly COPD and stroke, in 910 Chinese (439 men, 471 women) who never smoked from a 17-year follow-up study in Xi'an, China. SHS exposure was defi ned as exposure to another person's tobacco smoke at home or in the workplace.
RESULTS	At baseline among the 910 subjects, 44.2% were exposed to SHS at home, 52.9% in the workplace, and 67.1% at home, work, or both. From March 1, 1994, to July 1, 2011, 249 (150 men,99 women) died within 14,016 person-years. Those who were exposed to SHS had increased mortality due to coronary heart disease (adjusted relative risk [RR], 2.15; 95% CI, 1.00-4.61), ischemic stroke (RR, 2.88; 95% CI, 1.10-7.55), lung cancer (RR, 2.00; 95% CI, 0.62-6.40), COPD (RR, 2.30;95% CI, 1.06-5.00), and all causes (RR, 1.72; 95% CI, 1.29-2.20), with significant dose-response relationships between cumulative SHS exposure at home and work and the increased risk of cause-specific and total mortality (P for linear trend ranged from .045 to , .001).
CONCLUSIONS	This study shows dose-response relationships between SHS and major tobacco-related mortality and provides new evidence to support causation for COPD and ischemic stroke.

표-131. PubMed 논문번호 22628541의 내용 요약

구분	내용
PubMed ID	22628541
TITLE	An epidemiological appraisal of the association between heart rate variability and particulate air pollution: a meta-analysis.
JOURNAL	Heart (British Cardiac Society): 10.1136/heartjnl-2011-301505
AUTHORS	Pieters Nicky, Plusquin Michelle, Cox Bianca, Kicinski Michal, Vangronsveld Jaco, Nawrot Tim S
OBJECTIVE	Studies on the association between short-term exposure to ambient air pollution and heart rate variability (HRV) suggest that particulate matter (PM) exposure is associated with reductions in measures of HRV, but there is heterogeneity in the nature and magnitude of this association between studies. The authors performed a meta-analysis to determine how consistent this association is.
DATA SOURCE	The authors searched the Pubmed citation database and Web of Knowledge to identify studies on HRV and PM.
STUDY SELECTION	Of the epidemiologic studies reviewed, 29 provided sufficient details to be considered. The meta-analysis included 18667 subjects recruited from the population in surveys, studies from patient groups, and from occupationally exposed groups.
DATA EXTRACTION	Two investigators read all papers and computerised all relevant information.
RESULTS	The authors computed pooled estimates from a random-effects model. In the combined studies, an increase of 10 μg/m(3) in PM(2.5) was associated with significant reductions in the time-domain measurements, including low frequency (-1.66%, 95% CI -2.58% to -0.74%) and high frequency (-2.44%, 95% CI -3.76% to -1.12%) and in frequency-domain measurements, for SDNN (-0.12%, 95% CI -0.22% to -0.03%) and for rMSSD (-2.18%, 95% CI -3.33% to -1.03%). Funnel plots suggested that no publication bias was present and a sensitivity analysis confirmed the robustness of our combined estimates.
CONCLUSION	The meta-analysis supports an inverse relationship between HRV, a marker for a worse cardiovascular prognosis, and particulate air pollution.

표-132. PubMed 논문번호 22672304의 내용 요약

구분	내용
PubMed ID	22672304
TITLE	Meta-analysis of association between particulate matter and stroke attack.
JOURNAL	CNS neuroscience & therapeutics: 10.1111/j.1755-5949.2012.00325.x
AUTHORS	Li Xiu-Yang, Yu Xiao-Bo, Liang Wei-Wei, Yu Nan, Wang Li, Ye Xu-Jun, Chen Kun, Bian Ping-Da
AIMS	We conducted systematic review as well as meta-analyses on the association between particulate matter and daily stroke attack from a number of epidemiologic studies.
METHODS	Twelve quantitative studies about the associations between particulate matter and stroke attack met the inclusive criteria. We evaluated the odds ratio (OR) of stroke attack associated with per 10 μg/m(3) increase of the concentration of PM(10) (particulate matter with aerodynamic diameter ≤ 10 μm) or PM(2.5) (particulate matter with aerodynamic diameter ≤ 2.5 μm) as effect scale, and a sensitivity analysis for the results was conducted.
RESULTS	In the time-series design, PM(10) exposure wasn't related to an increased risk of daily stroke attack [OR per 10 μg/m(3) = 1.002, 95% confidence interval (CI): 0.999~1.005], PM(2.5) exposure were related to an increased risk of daily stroke attack (OR per 10 μg/m(3) = 1.006, 95%CI: 1.002~1.010]; but in the case-crossover studies, PM(10) exposure was related to increase in risk of daily stroke attack (OR per 10 μg/m(3) = 1.028, 95%CI: 1.001~1.057). PM(2.5) exposure was not significant association with daily stroke attack (OR per 10 μg/m(3) = 1.016, 95%CI: 0.937~1.097). Sensitivity analysis showed that the results for PM(10) , PM(2.5) and daily stroke attack were robust in the time-series design.
CONCLUSIONS	We found some evidence for an effect of air pollutants on stroke attack risk.

표-133. PubMed 논문번호 22674867의 내용 요약

구분	내용
PubMed ID	22674867
TITLE	A comparison of short-term and long-term air pollution exposure associations with mortality in two cohorts in Scotland.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1104509
AUTHORS	Beverland Iain J, Cohen Geoffrey R, Heal Mathew R, Carder Melanie, Yap Christina, Robertson Chris, Hart Carole L, Agius Raymond M
BACKGROUND	Air pollution-mortality risk estimates are generally larger at longer-term, compared with short-term, exposure time scales.
OBJECTIVE	We compared associations between short-term exposure to black smoke (BS) and mortality with long-term exposure-mortality associations in cohort participants and with short-term exposure-mortality associations in the general population from which the cohorts were selected.
METHODS	We assessed short-to-medium-term exposure-mortality associations in the Renfrew-Paisley and Collaborative cohorts (using nested case-control data sets), and compared them with long-term exposure-mortality associations (using a multilevel spatiotemporal exposure model and survival analyses) and short-to-medium-term exposure-mortality associations in the general population (using time-series analyses).
RESULTS	For the Renfrew-Paisley cohort (15,331 participants), BS exposure-mortality associations were observed in nested case-control analyses that accounted for spatial variations in pollution exposure and individual-level risk factors. These cohort-based associations were consistently greater than associations estimated in time-series analyses using a single monitoring site to represent general population exposure {e.g., 1.8% [95% confidence interval (CI): 0.1, 3.4%] vs. 0.2% (95% CI: 0.0, 0.4%) increases in mortality associated with 10-$\mu g/m^3$ increases in 3-day lag BS, respectively}. Exposure-mortality associations were of larger magnitude for longer exposure periods [e.g., 3.4% (95% CI: -0.7, 7.7%) and 0.9% (95% CI: 0.3, 1.5%) increases in all-cause mortality associated with 10-$\mu g/m^3$ increases in 31-day BS in case-control and time-series analyses, respectively; and 10% (95% CI: 4, 17%) increase in all-cause mortality associated with a 10-$\mu g/m^3$ increase in geometic mean BS for 1970-1979, in survival analysis].
CONCLUSIONS	After adjusting for individual-level exposure and potential confounders, short-term exposure-mortality associations in cohort participants were of greater magnitude than in comparable general population time-series study analyses. However, short-term exposure-mortality associations were substantially lower than equivalent long-term associations, which is consistent with the possibility of larger, more persistent cumulative effects from long-term exposures.

표-134. PubMed 논문번호 22693266의 내용 요약

구분	내용
PubMed ID	22693266
TITLE	Occupational exposure to particles and incidence of acute myocardial infarction and other ischaemic heart disease.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2011-100285
AUTHORS	Wiebert Pernilla, L□nn Maria, Fremling Karin, Feychting Maria, Sj□gren Bengt, Nise Gun, Kauppinen T, Plato Nils, Gustavsson Per
BACKGROUND	Ambient particulate air pollution has been linked to cardiovascular disease. Occupational particle exposure levels may be several times higher than ambient levels but has been less studied.
OBJECTIVES	The authors investigated the association between occupational exposure to particles and the incidence of ischaemic heart disease (IHD).
METHODS	The cohort included all manual workers in the Swedish national census of 1980 with information on demographic data and occupation. Information on hospital admissions for acute myocardial infarction or other IHDs and cause of death were obtained from nation-wide registers. A job-exposure matrix for exposure to small (<1 μm) and large (>1 μm) particles was developed. HRs were calculated with Cox regression with adjustment for sex, age, socioeconomic group and urban/rural residential area.
RESULTS	Exposure to small particles was associated with an increased HR for acute myocardial infarction of 1.12 (95% CI 1.09 to 1.15), and HR for exposure to large particles was 1.14 (95% CI 1.10 to 1.18). The association was somewhat stronger for workers exposed to small particles for more than 5 years, 1.21 (95% CI 1.11 to 1.31), but no trend with exposure intensity was found. The risk associated with exposure to small particles was higher among women than among men, 1.30 (95% CI 1.12 to 1.51) and 1.10 (95% CI 1.07 to 1.14), respectively. Findings were essentially similar for other IHDs.
CONCLUSIONS	This explorative study gives some support to the hypothesis that occupational exposure to particles increases the risk of acute myocardial infarction and other IHD. The findings must be interpreted cautiously due to lack of smoking data.

표-135. PubMed 논문번호 22693269의 내용 요약

구분	내용
PubMed ID	22693269
TITLE	Lack of effect of nitrogen dioxide exposure on heart rate variability in patients with stable coronary heart disease and impaired left ventricular systolic function.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2011-100126
AUTHORS	Scaife Alison, Barclay Justin, Hillis Graham S, Srinivasan Janaki, Macdonald David W, Ross John A S, Ayres Jon G
OBJECTIVES	Epidemiological studies of air pollution on cardiovascular health show associations of cardiac mortality and admissions with exposure to nitrogen dioxide (NO(2)) at low concentrations. These associations could be causal or NO(2) could be acting as a surrogate measure for another air pollutant, most likely ultrafine particles. No studies of cardiac susceptibility to acute exposure to NO(2) have been undertaken.
METHODS	Randomised controlled exposures to NO(2) (400 ppb for 1 h) and air in subjects with coronary heart disease and impaired left ventricular systolic function not taking β adrenoceptor blocking drugs.
RESULTS	There were no significant changes in heart rate, blood pressure, leucocyte coping capacity or any heart rate variability measure following NO(2) exposure compared with air.
CONCLUSION	These findings suggest that NO(2) does not affect heart rate variability at these concentrations (which are high for urban background levels) and in the absence of other pollutants. While a synergistic effect has not been ruled out, these data lend support to the idea that the epidemiological data associating cardiac outcomes with NO(2) are more likely due to an associated pollutant rather than NO(2) itself.

표-136. PubMed 논문번호 22732313의 내용 요약

구분	내용
PubMed ID	22732313
TITLE	Controlled exposure of healthy young volunteers to ozone causes cardiovascular effects.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.112.094359
AUTHORS	Devlin Robert B, Duncan Kelly E, Jardim Melanie, Schmitt Michael T, Rappold Ana G, Diaz-Sanchez David
BACKGROUND	Recent epidemiology studies have reported associations between short-term ozone exposure and mortality. Such studies have previously reported associations between airborne particulate matter pollution and mortality, and support for a causal relationship has come from controlled-exposure studies that describe pathophysiological mechanisms by which particulate matter could induce acute mortality. In contrast, for ozone, almost no controlled-human-exposure studies have tested whether ozone exposure can modulate the cardiovascular system.
METHODS AND RESULTS	Twenty-three young healthy individuals were exposed in a randomized crossover fashion to clean air and to 0.3-ppm ozone for 2 hours while intermittently exercising. Blood was obtained immediately before exposure, immediately afterward, and the next morning. Continuous Holter monitoring began immediately before exposure and continued for 24 hours. Lung function was performed immediately before and immediately after exposure, and bronchoalveolar lavage was performed 24 hours after exposure. Immediately after ozone exposure, we observed a 98.9% increase in interleukin-8, a 21.4% decrease in plasminogen activator inhibitor-1, a 51.3% decrease in the high-frequency component of heart rate variability, and a 1.2% increase in QT duration. Changes in interleukin-1B and plasminogen activator inhibitor-1 were apparent 24 hours after exposure. In agreement with previous studies, we also observed ozone-induced drops in lung function and an increase in pulmonary inflammation.
CONCLUSIONS	This controlled-human-exposure study shows that ozone can cause an increase in vascular markers of inflammation and changes in markers of fibrinolysis and markers that affect autonomic control of heart rate and repolarization. We believe that these findings provide biological plausibility for the epidemiology studies that associate ozone exposure with mortality.
CLINICAL TRIAL REGISTRATION	URL: http://www.clinicaltrials.gov. Unique identifier: NCT01492517.

표-137. PubMed 논문번호 22745727의 내용 요약

구분	내용
PubMed ID	22745727
TITLE	Road traffic noise and incident myocardial infarction: a prospective cohort study.
JOURNAL	PloS one: 10.1371/journal.pone.0039283
AUTHORS	S ø rensen Mette, Andersen Zorana J, Nordsborg Rikke B, Jensen Steen S, Lillelund Kenneth G, Beelen Rob, Schmidt Erik B, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
BACKGROUND	Both road traffic noise and ambient air pollution have been associated with risk for ischemic heart disease, but only few inconsistent studies include both exposures.
METHODS	In a population-based cohort of 57 053 people aged 50 to 64 years at enrolment in 1993-1997, we identified 1600 cases of first-ever MI between enrolment and 2006. The mean follow-up time was 9.8 years. Exposure to road traffic noise and air pollution from 1988 to 2006 was estimated for all cohort members from residential address history. Associations between exposure to road traffic noise and incident MI were analysed in a Cox regression model with adjustment for air pollution (NO(x)) and other potential confounders: age, sex, education, lifestyle confounders, railway and airport noise.
RESULTS	We found that residential exposure to road traffic noise (L(den)) was significantly associated with MI, with an incidence rate ratio IRR of 1.12 per 10 dB for both of the two exposure windows: yearly exposure at the time of diagnosis (95% confidence interval (CI): 1.02-1.22) and 5-years time-weighted mean (95% CI: 1.02-1.23) preceding the diagnosis. Visualizing of the results using restricted cubic splines showed a linear dose-response relationship.
CONCLUSIONS	Exposure to long-term residential road traffic noise was associated with a higher risk for MI, in a dose-dependent manner.

표-138. PubMed 논문번호 22820680의 내용 요약

구분	내용
PubMed ID	22820680
TITLE	Long-term exposure to traffic-related PM(10) and decreased heart rate variability: is the association restricted to subjects taking ACE inhibitors?
JOURNAL	Environment international: 10.1016/j.envint.2012.06.016
AUTHORS	Adam Martin, Felber Dietrich Denise, Schaffner Emmanuel, Carballo David, Barth□l□my Jean-Claude, Gaspoz Jean-Michel, Tsai Ming-Yi, Rapp Regula, Phuleria Harish Chandra, Schindler Christian, Schwartz Joel, K□nzli Nino, Probst-Hensch Nicole M
BACKGROUND	Alterations in heart rate variability (HRV) are a potential link between exposure to traffic-related air pollution and cardiovascular mortality.
OBJECTIVES	We investigated whether long-term exposure to traffic-related PM(10) (TPM(10)) is associated with HRV in older subjects and/or in participants taking specific cardiovascular treatment or with self-reported heart disease.
METHODS	We included 1607 subjects from the general population aged 50 to 72 years. These participants from the SAPALDIA cohort underwent ambulatory 24-hr electrocardiogram monitoring. Associations of average annual exposure to TPM(10) over 10 years with HRV parameters from time and frequency domains were estimated using multivariable mixed linear models. Effect estimates are expressed as percent changes in geometric means.
RESULTS	HRV was only associated with TPM(10) in participants under ACE inhibitor therapy (N=94). A 1 μg/m(3) increment, approximately equivalent to an interquartile range, in 10 year average TPM(10) was associated with decrements of 14.5% (95% confidence interval (CI), -25.9 to -1.3) in high frequency (HF) power, of 4.5% (-8.2 to -0.5) in the standard deviation of all normal-to-normal RR intervals (SDNN), of 10.6% (-18.5 to -1.9) in total power (TP) and an increase of 9.2% (0.8 to 20.2) in the LF/HF power ratio.
CONCLUSIONS	In the absence of an overall effect our results suggest that alterations in HRV, a measure of autonomic control of the cardiac rhythm, may not be a central mechanism by which long-term exposure to TPM(10) increases cardiovascular mortality. Novel evidence on an effect in persons under ACE inhibitor treatment needs to be confirmed in future studies.

표-139. PubMed 논문번호 22835955의 내용 요약

구분	내용
PubMed ID	22835955
TITLE	Ambient air pollution exposure and blood pressure changes during pregnancy.
JOURNAL	Environmental research: 10.1016/j.envres.2012.05.011
AUTHORS	Lee Pei-Chen, Talbott Evelyn O, Roberts James M, Catov Janet M, Bilonick Richard A, Stone Roslyn A, Sharma Ravi K, Ritz Beate
BACKGROUND	Maternal exposure to ambient air pollution has been associated with adverse birth outcomes such as preterm delivery. However, only one study to date has linked air pollution to blood pressure changes during pregnancy, a period of dramatic cardiovascular function changes.
OBJECTIVES	We examined whether maternal exposures to criteria air pollutants, including particles of less than 10 μm (PM(10)) or 2.5 μm diameter (PM(2.5)), carbon monoxide (CO), nitrogen dioxide (NO(2)), sulfur dioxide (SO(2)), and ozone (O(3)), in each trimester of pregnancy are associated with magnitude of rise of blood pressure between the first 20 weeks of gestation and late pregnancy in a prospectively followed cohort of 1684 pregnant women in Allegheny County, PA.
METHODS	Air pollution measures for maternal ZIP code areas were derived using Kriging interpolation. Using logistic regression analysis, we evaluated the associations between air pollution exposures and blood pressure changes between the first 20 weeks of gestation and late pregnancy.
RESULTS	First trimester PM(10) and ozone exposures were associated with blood pressure changes between the first 20 weeks of gestation and late pregnancy, most strongly in non-smokers. Per interquartile increases in first trimester PM(10) and O(3) concentrations were associated with mean increases in systolic blood pressure of 1.88 mm Hg (95% CI=0.84 to 2.93) and 1.84 (95% CI=1.05 to 4.63), respectively, and in diastolic blood pressure of 0.63 mm Hg (95% CI=-0.50 to 1.76) and 1.13 (95% CI=-0.46 to 2.71) in non-smokers.
CONCLUSIONS	Our novel finding suggests that first trimester PM(10) and O(3) air pollution exposures increase blood pressure in the later stages of pregnancy. These changes may play a role in mediating the relationships between air pollution and adverse birth outcomes.

표-140. PubMed 논문번호 22859900의 내용 요약

구분	내용
PubMed ID	22859900
TITLE	Frequency of pulmonary hypertension in patients with COPD due to biomass smoke and tobacco smoke.
JOURNAL	International journal of medical sciences: 10.7150/ijms.4715
AUTHORS	Sertogullarindan Bunyamin, Gumrukcuoglu Hasan Ali, Sezgi Cengizhan, Akil Mehmet Ata
OBJECTIVES	Pulmonary hypertension (PH) is a common and well established complication of chronic obstructive pulmonary disease (COPD). Its presence is associated with decreased survival. This study was designed to investigate the PH frequency and its relations in hospitalized tobacco and biomass related COPD patients.
METHODS AND RESULTS	The study was a retrospective review of inpatients with COPD defined as a history of tobacco or biomass smoking, Pulmonary function tests (PFT) within stable status, an echocardiogram within stable status. PH was defined as systolic pulmonary artery pressure (sPAP) >35 mmHg. Of the 694 individuals, 600 had suitable aspects for inclusion of study. All Females were biomass exposer and males were tobacco smoker. The Prevalence of PH was found more frequent in females than males. It was more prominent in moderate level COPD cases (56,2% and 37,5%, P<0,002). Both groups had airflow limitation, hypercapnia and hypoxemia, but no differences were found in terms of PaCO(2) and PaO2. However, FEV1 % was lower in males than females (p<0,005). On the other hand, FVC % was lower in the females compared with the males (p < 0.02). When analyzing the influence of PFT and demographic parameters on PH in separate COPD level groups, the results a bit varied among the groups.
CONCLUSION	Our study demonstrated that PH frequency is higher in female COPD cases due to biomass smoke than in male COPD cases due to tobacco smoke. The influence of FVC % on the risk of a person having PH increased with increasing COPD level.

표-141. PubMed 논문번호 22896588의 내용 요약

구분	내용
PubMed ID	22896588
TITLE	Childhood air pollutant exposure and carotid artery intima-media thickness in young adults.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.112.096164
AUTHORS	Breton Carrie V, Wang Xinhui, Mack Wendy J, Berhane Kiros, Lopez Milena, Islam Talat S, Feng Mei, Lurmann Fred, McConnell Rob, Hodis Howard N, K□nzli Nino, Avol Ed
BACKGROUND	Exposure to ambient air pollutants increases risk for cardiovascular health outcomes in adults. The contribution of childhood air pollutant exposure to cardiovascular health has not been thoroughly evaluated.
METHODS AND RESULTS	The Testing Responses on Youth study consists of 861 college students recruited from the University of Southern California in 2007 to 2009. Participants attended 1 study visit during which blood pressure, heart rate, and carotid artery intima-media thickness (CIMT) were assessed. Self-administered questionnaires collected information about health and sociodemographic characteristics, and a 12-hour fasting blood sample was drawn for lipid and biomarker analyses. Residential addresses were geocoded and used to assign cumulative air pollutant exposure estimates based on data derived from the U.S. Environmental Protection Agency's Air Quality System database. The associations between CIMT and air pollutants were assessed using linear regression analysis. Mean CIMT was 603 μm (±54 SD). A 2 standard deviation (SD) increase in childhood (aged 0-5 years) or elementary school (aged 6-12 years) O(3) exposure was associated with a 7.8- μm (95% confidence interval, -0.3-15.9) or 10.1- μm (95% confidence interval, 1.8-18.5) higher CIMT, respectively. Lifetime exposure to O(3) showed similar but nonsignificant associations. No associations were observed for PM(2.5), PM(10), or NO(2), although adjustment for these pollutants strengthened the childhood O(3) associations.
CONCLUSIONS	Childhood exposure to O(3) may be a novel risk factor for CIMT in a healthy population of college students. Regulation of air pollutants and efforts that focus on limiting childhood exposures continue to be important public health goals.

표-142. PubMed 논문번호 22912729의 내용 요약

구분	내용
PubMed ID	22912729
TITLE	SERPINA1 PiZ and PiS heterozygotes and lung function decline in the SAPALDIA cohort.
JOURNAL	PloS one: 10.1371/journal.pone.0042728
AUTHORS	Thun Gian-Andri, Ferrarotti Ilaria, Imboden Medea, Rochat Thierry, Margaret, Kronenberg Florian, Bridevaux Pierre-Olivier, Zemp Elisal Zorzetto Michele, Ottaviani Stefania, Russi Erich W, Luisetti Maurizi Probst-Hensch Nicole M
BACKGROUND	Severe alpha1-antitrypsin (AAT) deficiency is a strong risk factor for But the impact of gene variants resulting in mild or intermediate AAT deficiency on the longitudinal course of respiratory health remains controversial. There is indication from experimental studies that pro-inflammatory agents like cigarette smoke can interact with these and thus increase the risk of adverse respiratory health effects. Theref tested the effect of the presence of a protease inhibitor (Pi) S or Z alle and PiMZ) on the change in lung function in different inflammation- subgroups of a large, population-based cohort study.
METHODOLOGY AND PRINCIPAL FINDINGS	The SAPALDIA population includes over 4600 subjects from whom SERPINA1 genotypes for S and Z alleles, spirometry and respiratory symptoms at baseline and after 11 years follow-up, as well as proxies inflammatory conditions, such as detailed smoking history, obesity an sensitivity C-reactive protein (hs-CRP), were available. All analyses performed by applying multivariate regression models. There was no unfavourable effect of PiMS or PiMZ genotype on lung function char found indication that PiZ heterozygosity interacted with inflammatory leading to an accelerated decline in measures in use as indices for asse mild airway obstruction. Obese individuals with genotype PiMM had average annual decline in the forced mid expiratory flow (ΔFEF25-7 58.4 ml whereas in obese individuals with PiMZ it amounted to 92.2 0.03). Corresponding numbers for persistent smokers differed even m strongly (66.8 ml (PiMM) vs. 108.2 ml (PiMZ), p = 0.005). Equivale less strong associations were observed for the change in the FEV1/FV
CONCLUSIONS	We suggest that, in addition to the well established impact of the rare genotype, one Z allele may be sufficient to accelerate lung function de population subgroups characterized by elevated levels of low grade inflammation.

표-143. PubMed 논문번호 22948092의 내용 요약

구분	내용
PubMed ID	22948092
TITLE	Therapeutic effect of forest bathing on human hypertension in the elderly.
JOURNAL	Journal of cardiology: 10.1016/j.jjcc.2012.08.003
AUTHORS	Mao Gen-Xiang, Cao Yong-Bao, Lan Xiao-Guang, He Zhi-Hua, Chen Zhuo-Mei, Wang Ya-Zhen, Hu Xi-Lian, Lv Yuan-Dong, Wang Guo-Fu, Yan Jing
OBJECTIVE	To provide scientific evidence supporting the efficacy of forest bathing as a natural therapy for human hypertension.
METHODS	Twenty-four elderly patients with essential hypertension were randomly divided into two groups of 12. One group was sent to a broad-leaved evergreen forest to experience a 7-day/7-night trip, and the other was sent to a city area in Hangzhou for control. Blood pressure indicators, cardiovascular disease-related pathological factors including endothelin-1, homocysteine, renin, angiotensinogen, angiotensin II, angiotensin II type 1 receptor, angiotensin II type 2 receptor as well as inflammatory cytokines interleukin-6 and tumor necrosis factor α were detected. Meanwhile, profile of mood states (POMS) evaluation was used to assess the change of mood state of subjects. In addition, the air quality in the two experimental sites was monitored during the 7-day duration, simultaneously.
RESULTS	The baselines of the indicators of the subjects were not significantly different. Little alteration in the detected indicators in the city group was observed after the experiment. While subjects exposed to the forest environment showed a significant reduction in blood pressure in comparison to that of the city group. The values for the bio-indicators in subjects exposed to the forest environment were also lower than those in the urban control group and the baseline levels of themselves. POMS evaluation showed that the scores in the negative subscales were lowered after exposure to the forest environment. Besides, the air quality in the forest environment was much better than that of the urban area evidenced by the quantitative detection of negative ions and PM10 (particulate matter $<$ 10 μm in aerodynamic diameter).
CONCLUSION	Our results provided direct evidence that forest bathing has therapeutic effects on human hypertension and induces inhibition of the renin-angiotensin system and inflammation, and thus inspiring its preventive efficacy against cardiovascular disorders.

표-144. PubMed 논문번호 22950554의 내용 요약

구분	내용
PubMed ID	22950554
TITLE	Traffic air pollution and mortality from cardiovascular disease and all causes: a Danish cohort study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-11-60
AUTHORS	Raaschou-Nielsen Ole, Andersen Zorana Jovanovic, Jensen Steen Solvang, Ketzel Matthias, S ø rensen Mette, Hansen Johnni, Loft Steffen, Tj ø nneland Anne, Overvad Kim
BACKGROUND	Traffic air pollution has been linked to cardiovascular mortality, which might be due to co-exposure to road traffic noise. Further, personal and lifestyle characteristics might modify any association.
METHODS	We followed up 52 061 participants in a Danish cohort for mortality in the nationwide Register of Causes of Death, from enrollment in 1993-1997 through 2009, and traced their residential addresses from 1971 onwards in the Central Population Registry. We used dispersion-modelled concentration of nitrogen dioxide (NO_2) since 1971 as indicator of traffic air pollution and used Cox regression models to estimate mortality rate ratios (MRRs) with adjustment for potential confounders.
RESULTS	Mean levels of NO_2 at the residence since 1971 were significantly associated with mortality from cardiovascular disease (MRR, 1.26; 95% confidence interval [CI], 1.06-1.51, per doubling of NO_2 concentration) and all causes (MRR, 1.13; 95% CI, 1.04-1.23, per doubling of NO_2 concentration) after adjustment for potential confounders. For participants who ate < 200 g of fruit and vegetables per day, the MRR was 1.45 (95% CI, 1.13-1.87) for mortality from cardiovascular disease and 1.25 (95% CI, 1.11-1.42) for mortality from all causes.
CONCLUSIONS	Traffic air pollution is associated with mortality from cardiovascular diseases and all causes, after adjustment for traffic noise. The association was strongest for people with a low fruit and vegetable intake.

표-145. PubMed 논문번호 22968969의 내용 요약

구분	내용
PubMed ID	22968969
TITLE	Occupational exposures and mortality from cardiovascular disease among women textile workers in Shanghai, China.
JOURNAL	American journal of industrial medicine: 10.1002/ajim.22113
AUTHORS	Gallagher Lisa G, Ray Roberta M, Li Wenjin, Psaty Bruce M, Gao Dao Li, Thomas David B, Checkoway Harvey
BACKGROUND	Exposure to textile fiber dusts, like particulate air pollution, may be associated with cardiovascular disease (CVD) mortality. Bacterial endotoxin, a potent inflammagen found in cotton dust, may be a specific risk factor.
METHODS	Female textile workers (N = 267,400) in Shanghai, China were followed for CVD mortality (1989-2000). Factory exposures were approximated by sector classifications based on materials and processes. Quantitative endotoxin and cotton dust measures were available for a subcohort (n = 3,188). Cox proportional hazards modeling was used to estimate hazard ratios (HR) and 95% confidence interval (CI).
RESULTS	Slightly elevated mortality risk for the cotton sector was seen for ischemic stroke (HR = 1.12, 95% CI: 0.97-1.31) and hemorrhagic stroke (HR = 1.12, 95% CI: 1.02-1.23). Similar hemorrhagic stroke mortality risk was observed in high dust sectors (HR = 1.12, 95% CI: 1.02-1.24). No association was observed for ischemic heart disease.
CONCLUSIONS	Exposures in textile factories may have contributed to CVD mortality among this cohort. The specific components of these exposures that may be harmful are not clear and should be further investigated.

표-146. PubMed 논문번호 22978720의 내용 요약

구분	내용
PubMed ID	22978720
TITLE	In vitro and clinical studies examining the expression of osteopontin in cigarette smoke-exposed endothelial cells and cigarette smokers.
JOURNAL	BMC cardiovascular disorders: 10.1186/1471-2261-12-75
AUTHORS	Bishop Emma, Theophilus Eugenia H, Fearon Ian M
BACKGROUND	Cigarette smoking is a leading cause of mortality and morbidity and is associated with cardiovascular disease via contributory processes such as endothelial dysfunction, inflammation and thrombosis. Cigarette smoke both contains and stimulates the production of cellular oxidants and it may also promote vascular inflammation. Osteopontin is a non-collagenous matrix protein first identified in bone and there is increasing evidence for its role in inflammation and cardiovascular disease via its action as a soluble cytokine.
METHODS	In this study we have examined the mechanisms underlying the expression of osteopontin in human vascular endothelial cells in vitro following exposure to cigarette smoke particulate matter (PM), using PCR, electrochemiluminescence, immunostaining and Western blotting. We further determined if serum osteopontin levels changed in humans who quit smoking.
RESULTS	Non-cytotoxic concentrations of PM increased osteopontin levels in cultured human endothelial cells and this effect was reduced in the presence of ascorbate, suggesting a role for oxidants in the response to PM. However, oxidant production played no role in the PM-evoked induction MMP-3, an enzyme which cleaves osteopontin. In smokers who quit smoking for 5 days, serum osteopontin levels were significantly lowered compared to those measured prior to smoking cessation.
CONCLUSIONS	In vitro cigarette smoke extract exposure induced osteopontin expression in human endothelial cells in an oxidative stress-dependent manner, which may involve MMP-3 cleavage. In humans, serum osteopontin was decreased with short-term smoking cessation. Endothelial-derived osteopontin may contribute to inflammation in smokers, and may also contribute to atherosclerosis and cardiovascular disease-related processes.

표-147. PubMed 논문번호 22992341의 내용 요약

구분	내용
PubMed ID	22992341
TITLE	Ischaemic heart disease mortality and years of work in trucking industry workers.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2011-100017
AUTHORS	Hart Jaime E, Garshick Eric, Smith Thomas J, Davis Mary E, Laden Francine, Hart Jaime E, Garshick Eric, Smith Thomas J, Davis Mary E, Laden Francine
OBJECTIVES	Evidence from general population-based studies and occupational cohorts has identified air pollution from mobile sources as a risk factor for cardiovascular disease. In a cohort of US trucking industry workers, with regular exposure to vehicle exhaust, the authors previously observed elevated standardised mortality ratios for ischaemic heart disease (IHD) compared with members of the general US population. Therefore, the authors examined the association of increasing years of work in jobs with vehicle exhaust exposure and IHD mortality within the cohort.
METHODS	The authors calculated years of work in eight job groups for 30,758 workers using work records from four nationwide companies. Proportional hazard regression was used to examine relationships between IHD mortality, 1985-2000, and employment duration in each job group.
RESULTS	HRs for at least 1 year of work in each job were elevated for dockworkers, long haul drivers, pick-up and delivery drivers, combination workers, hostlers, and shop workers. There was a suggestion of an increased risk of IHD mortality with increasing years of work as a long haul driver, pick-up and delivery driver, combination worker, and dockworker.
CONCLUSION	These results suggest an elevated risk of IHD mortality in workers with a previous history of regular exposure to vehicle exhaust.

표-148. PubMed 논문번호 23018970의 내용 요약

구분	내용
PubMed ID	23018970
TITLE	Short-term effects of air pollution in a cohort of patients with chronic obstructive pulmonary disease.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e31826767c2
AUTHORS	Faustini Annunziata, Stafoggia Massimo, Cappai Giovanna, Forastiere Francesco
BACKGROUND	Although damage to the respiratory system from air pollutants has been recognized, research on susceptibility to air pollution in patients with chronic obstructive respiratory disease (COPD) has produced contradictory results. We studied the short-term effects of particulate matter (PM10, PM2.5), nitrogen dioxide (NO2), and ozone (O3) on cardiac and respiratory mortality in a COPD cohort. We assessed age, sex, and previous diseases as effect modifiers.
METHODS	Using hospital data (1998-2009) and pharmaceutical data (2005-2009), we enrolled 145,681 COPD subjects, aged 35+ years and residents of Rome, and followed them from 2005 to 2009. A comparison group of people without COPD (1,710,557 subjects) was also studied. We analyzed deaths due to all natural causes (International Classification of Diseases - Ninth Revision codes 1-799). Statistical analyses were carried out using Poisson regression and a case-crossover approach.
RESULTS	PM10, PM2.5, and NO2 (0- to 5-day lag) were associated with daily mortality, with stronger effects in people with COPD. The mortality associated with PM10 (per interquartile range [IQR] = 16 μg/m) was five times more in COPD patients (3.5% [95% confidence interval = -0.1% to 7.2%]) than in other subjects (0.7% [-0.8% to 2.2%]). Effects on respiratory mortality among COPD subjects were particularly elevated from PM2.5 (IQR = 11 μg/m) (11.6% [2.0% to 22.2%]) and NO2 (IQR = 24 μg/m) (19.6% [3.5% to 38.2%]). Older age, male sex, preexisting heart conduction disorders, and cerebrovascular diseases were associated with stronger effects in COPD subjects.
CONCLUSIONS	COPD patients are more susceptible to air pollutants, especially PM10 and NO2. These results suggest a need for more protective air pollution standards for susceptible groups.

표-149. PubMed 논문번호 23024248의 내용 요약

구분	내용
PubMed ID	23024248
TITLE	Impact of a comprehensive smoke-free law following a partial smoke-free law on incidence of heart attacks at a rural community hospital.
JOURNAL	Nicotine & tobacco research : official journal of the Society for Research on Nicotine and Tobacco: 10.1093/ntr/nts216
AUTHORS	Johnson Eric L, Beal James R, Johnson Eric L, Beal James R
INTRODUCTION	Secondhand smoking (passive smoking) is associated with many negative health effects, primarily respiratory and cardiovascular diseases. Approximately, 46,000 deaths from cardiovascular disease are associated with secondhand smoke exposure annually in the United States, which is roughly 150 deaths in North Dakota. Studies show that passage of smoke-free laws at the community level can reduce the incidence of heart attack.
METHODS	We conducted a retrospective review of electronic medical records of patients admitted for heart attacks 4 months prior (April 15, 2010 through August 14, 2010) to implementation of the smoke-free ordinance and 4 months following (August 15, 2010 through December 14, 2010) implementation of the comprehensive smoke-free ordinance in Grand Forks, ND, United States.
RESULTS	We found an association between the heart attack rate and implementation of the comprehensive smoke-free law. The heart attack rate prior to the ban was 0.5% (83/16,702) compared with 0.3% (63/18,513) after the ban (p = .023). Thus, the rate of heart attacks decreased 30.6% and number of heart attack admissions decreased 24.1%, from 83 to 63, after implementation of a comprehensive smoke-free law.
CONCLUSIONS	We found an implementation of the comprehensive smoke-free law was associated with a decrease in the heart attack rate. The heart attack rate decreased 30.6%. Our finding was similar to previous community level smoke-free law implementation studies and notable for the change going from a partial smoke-free law to a comprehensive smoke-free law.

표-150. PubMed 논문번호 23048073의 내용 요약

구분	내용
PubMed ID	23048073
TITLE	Premature atrial contractions in the general population: frequency and risk factors.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.112.112300
AUTHORS	Conen David, Adam Martin, Roche Frederic, Barthelemy Jean-Claude, Felber Dietrich Denise, Imboden Medea, K□nzli Nino, von Eckardstein Arnold, Regenass Stephan, Hornemann Thorsten, Rochat Thierry, Gaspoz Jean-Michel, Probst-Hensch Nicole, Carballo David
BACKGROUND	Premature atrial contractions (PACs) are independent predictors of atrial fibrillation, stroke, and death. However, little is known about PAC frequency in the general population and its association with other cardiovascular risk factors.
METHODS AND RESULTS	We performed a cross-sectional analysis among participants of the population-based Swiss cohort Study on Air Pollution and Lung Diseases in Adults (SAPALDIA). 24-hour Holter electrocardiograms to assess PAC prevalence and frequency were performed in a random sample of 1742 participants aged ≥50 years. The median (interquartile range) number of PACs per hour was 0.8 (0.4-1.8), 1.1 (0.5-2.4), 1.4 (0.7-4.6), 2.3 (0.8-6.9), and 2.6 (1.2-6.5) among participants aged 50 to 55, 55 to 60, 60 to 65, 65 to 70, and ≥70 years, respectively (P<0.0001). Only 18 (1.0%) participants did not have at least 1 PAC during Holter monitoring. In multivariable negative binomial regression models, PAC frequency was significantly associated with age (risk ratio [RR] per SD 1.80; P<0.0001), height (RR per SD 1.52; P<0.0001), prevalent cardiovascular disease (RR 2.40; P<0.0001), log-transformed N-terminal pro B-type natriuretic peptides (RR per SD 1.27; P<0.0001), physical activity ≥2 hours per day (RR 0.69; P=0.002), and high-density lipoprotein cholesterol (RR per SD 0.80; P=0.0002). Hypertension and body mass index were not significantly related to PAC frequency.
CONCLUSIONS	To our knowledge, this is the first study to assess risk factors for PAC frequency in the general population aged ≥50 years. PACs are common, and their frequency is independently associated with age, height, history of cardiovascular disease, natriuretic peptide levels, physical activity, and high-density lipoprotein cholesterol. The underlying mechanisms of these relationships need to be addressed in future studies.

표-151. PubMed 논문번호 23067103의 내용 요약

구분	내용
PubMed ID	23067103
TITLE	Short-term effects of ambient particulates and gaseous pollutants on the incidence of transient ischaemic attack and minor stroke: a case-crossover study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-11-77
AUTHORS	Bedada Getahun Bero, Smith Craig J, Tyrrell Pippa J, Hirst Adrian A, Agius Raymond
BACKGROUND	While several studies have investigated the effects of short-term air pollution on cardiovascular disease, less is known about its effects on cerebrovascular disease, including stroke and transient ischaemic attack (TIA). The aim of the study was to assess the effects of short-term variation in air pollutants on the onset of TIA and minor stroke.
METHODS	We performed secondary analyses of data collected prospectively in the North West of England in a multi-centre study (NORTHSTAR) of patients with recent TIA or minor stroke. A case-crossover study was conducted to determine the association between occurrence of TIA and the concentration of ambient PM10 or gaseous pollutants.
RESULTS	A total of 709 cases were recruited from the Manchester (n = 335) and Liverpool (n = 374) areas. Data for the Manchester cohort showed an association between ambient nitric oxide (NO) and risk of occurrence of TIA and minor stroke with a lag of 3 days (odds ratio 1.06, 95% CI: 1.01 - 1.11), whereas negative association was found for the patients from Liverpool. Effects of similar magnitude, although not statistically significant, were generally observed with other pollutants. In a two pollutant model the effect of NO remained stronger and statistically significant when analysed in combination with CO or SO2, but was marginal in combination with NO2 or ozone and non-significant with PM10. There was evidence of effect modification by age, gender and season.
CONCLUSIONS	Our data suggest an association between NO and occurrence of TIA and minor stroke in Greater Manchester.

표-152. PubMed 논문번호 23069326의 내용 요약

구분	내용
PubMed ID	23069326
TITLE	Cigarette smoke retention and bronchodilation in patients with COPD. A controlled randomized trial.
JOURNAL	Respiratory medicine: 10.1016/j.rmed.2012.09.019
AUTHORS	van Dijk Wouter D, Heijdra Yvonne, Lenders Jacques W M, Klerx Walther, Akkermans Reinier, van der Pouw Anouschka, van Weel Chris, Scheepers Paul T J, Schermer Tjard R J, van Dijk Wouter D, Heijdra Yvonne, Lenders Jacques W M, Klerx Walther, Akkermans Reinier, van der Pouw Anouschka, van Weel Chris, Scheepers Paul T J, Schermer Tjard R J
INTRODUCTION	Bronchodilators are the cornerstone for symptomatic treatment of chronic obstructive pulmonary disease (COPD). Many patients use these agents while persisting in their habit of cigarette smoking. We hypothesized that bronchodilators increase pulmonary retention of cigarette smoke and hence the risk of smoking-related (cardiovascular) disease. Our aim was to investigate if bronchodilation causes increased pulmonary retention of cigarette smoke in patients with COPD.
METHODS	A double-blinded, placebo-controlled, randomized crossover trial, in which COPD patients smoked cigarettes during undilated conditions at one session and maximal bronchodilated conditions at the other session. Co-primary outcomes were pulmonary tar and nicotine retention. We performed a secondary analysis that excludes errors due to possible contamination. Secondary outcomes included the biomarkers C-reactive protein and fibrinogen, and smoke inhalation patterns.
RESULTS	Of 39 randomized patients, 35 patients completed the experiment and were included in the final analysis. Bronchodilation did not significantly increase tar retention (-4.5%, p = 0.20) or nicotine retention (-2.6%, p = 0.11). Secondary analysis revealed a potential reduction of retention due to bronchodilation: tar retention (-3.8%, p = 0.13), and nicotine retention (-3.4%, p = 0.01). Bronchodilation did not modify our secondary outcomes.
CONCLUSIONS	Our results do not support the hypothesis that cigarette tar and nicotine retention in COPD patients is increased by bronchodilation, whereas we observed a possibility towards less retention.
TRIAL REGISTRATION	www.clinicaltrials.gov: NCT00981851.

표-153. PubMed 논문번호 23103035의 내용 요약

구분	내용
PubMed ID	23103035
TITLE	Vascular responses to long- and short-term exposure to fine particulate matter: MESA Air (Multi-Ethnic Study of Atherosclerosis and Air Pollution).
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2012.08.973
AUTHORS	Krishnan Ranjini M, Adar Sara D, Szpiro Adam A, Jorgensen Neal W, Van Hee Victor C, Barr R Graham, O'Neill Marie S, Herrington David M, Polak Joseph F, Kaufman Joel D
OBJECTIVES	This study evaluated the association of long- and short-term air pollutant exposures with flow-mediated dilation (FMD) and baseline arterial diameter (BAD) of the brachial artery using ultrasound in a large multicity cohort.
BACKGROUND	Exposures to ambient air pollution, especially long-term exposure to particulate matter <2.5 μm in aerodynamic diameter (PM(2.5)), are linked with cardiovascular mortality. Short-term exposure to PM(2.5) has been associated with decreased FMD and vasoconstriction, suggesting that adverse effects of PM(2.5) may involve endothelial dysfunction. However, long-term effects of PM(2.5) on endothelial dysfunction have not been investigated.
METHODS	FMD and BAD were measured by brachial artery ultrasound at the initial examination of the Multi-Ethnic Study of Atherosclerosis. Long-term PM(2.5) concentrations were estimated for the year 2000 at each participant's residence (n = 3,040) using a spatio-temporal model informed by cohort-specific monitoring. Short-term PM(2.5) concentrations were based on daily central-site monitoring in each of the 6 cities.
RESULTS	An interquartile increase in long-term PM(2.5) concentration (3 μg/m(3)) was associated with a 0.3% decrease in FMD (95% confidence interval [CI] of difference: -0.6 to -0.03; p = 0.03), adjusting for demographic characteristics, traditional risk factors, sonographers, and 1/BAD. Women, nonsmokers, younger participants, and those with hypertension seemed to show a greater association of PM(2.5) with FMD. FMD was not significantly associated with short-term variation in PM(2.5) (-0.1% per 12 μg/m(3) daily increase [95% CI: -0.2 to 0.04] on the day before examination).
CONCLUSIONS	Long-term PM(2.5) exposure was significantly associated with decreased endothelial function according to brachial ultrasound results. These findings may elucidate an important pathway linking air pollution and cardiovascular mortality.

표-154. PubMed 논문번호 23104730의 내용 요약

구분	내용
PubMed ID	23104730
TITLE	Association between long-term exposure to air pollution and specific causes of mortality in Scotland.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2011-100600
AUTHORS	Yap Christina, Beverland Iain J, Heal Mathew R, Cohen Geoffrey R, Robertson Chris, Henderson Deborah E J, Ferguson Neil S, Hart Carole L, Morris George, Agius Raymond M
OBJECTIVE	This study investigated the association between long-term exposure to black smoke (BS) air pollution and mortality in two related Scottish cohorts with 25 years of follow-up.
METHODS	Risk factors were collected during 1970-1976 for 15331 and 6680 participants in the Renfrew/Paisley and Collaborative cohorts respectively. Exposure to BS during 1970-1979 was estimated by inverse-distance weighted averages of observed concentrations at monitoring sites and by two alternative spatial modelling approaches which included local air quality predictors (LAQP).
RESULTS	Consistent BS-mortality associations (per 10 μg m(-3) increment in 10-year average BS) were observed in the Renfrew/Paisley cohort using LAQP-based exposure models (all-cause mortality HR 1.10 (95% CI 1.04 to 1.17); cardiovascular HR 1.11 (1.01 to 1.22); ischaemic heart disease HR 1.13 (1.02 to 1.25); respiratory HR 1.26 (1.02 to 1.28)). The associations were largely unaffected by additional adjustment for area-level deprivation category. A less consistent and generally implausible pattern of cause-specific BS-mortality associations was found for inverse-distance averaging of BS concentrations at nearby monitoring sites. BS-mortality associations in the Collaborative cohort were weaker and not statistically significant.
CONCLUSIONS	The association between mortality and long-term exposure to BS observed in the Renfrew/Paisley cohort is consistent with hypotheses of how air pollution may affect human health. The dissimilarity in pollution-mortality associations for different exposure models highlights the critical importance of reliable estimation of exposures on intraurban spatial scales to avoid potential misclassification bias.

표-155. PubMed 논문번호 23109514의 내용 요약

구분	내용
PubMed ID	23109514
TITLE	Association between smoke-free legislation and hospitalizations for cardiac, cerebrovascular, and respiratory diseases: a meta-analysis.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.112.121301
AUTHORS	Tan Crystal E, Glantz Stanton A
BACKGROUND	Secondhand smoke causes cardiovascular and respiratory disease. Smoke-free legislation is associated with a lower risk of hospitalization and death from these diseases.
METHODS AND RESULTS	Random-effects meta-analysis was conducted by law comprehensiveness to determine the relationship between smoke-free legislation and hospital admission or death from cardiac, cerebrovascular, and respiratory diseases. Studies were identified by using a systematic search for studies published before November 30, 2011 with the use of the Science Citation Index, Google Scholar, PubMed, and Embase and references in identified articles. Change in hospital admissions (or deaths) in the presence of a smoke-free law, duration of follow-up, and law comprehensiveness (workplaces only; workplaces and restaurants; or workplaces, restaurants, and bars) were recorded. Forty-five studies of 33 smoke-free laws with median follow-up of 24 months (range, 2-57 months) were included. Comprehensive smoke-free legislation was associated with significantly lower rates of hospital admissions (or deaths) for all 4 diagnostic groups: coronary events (relative risk, 0.848; 95% confidence interval 0.816-0.881), other heart disease (relative risk, 0.610; 95% confidence interval, 0.440-0.847), cerebrovascular accidents (relative risk, 0.840; 95% confidence interval, 0.753-0.936), and respiratory disease (relative risk, 0.760; 95% confidence interval, 0.682-0.846). The difference in risk following comprehensive smoke-free laws does not change with longer follow-up. More comprehensive laws were associated with larger changes in risk.
CONCLUSIONS	Smoke-free legislation was associated with a lower risk of smoking-related cardiac, cerebrovascular, and respiratory diseases, with more comprehensive laws associated with greater changes in risk.

표-156. PubMed 논문번호 23210563의 내용 요약

구분	내용
PubMed ID	23210563
TITLE	A randomized double-blind crossover study of indoor air filtration and acute changes in cardiorespiratory health in a First Nations community.
JOURNAL	Indoor air: 10.1111/ina.12019
AUTHORS	Weichenthal S, Mallach G, Kulka R, Black A, Wheeler A, You H, St-Jean M, Kwiatkowski R, Sharp D
UNLABELLED	Few studies have examined indoor air quality in First Nations communities and its impact on cardiorespiratory health. To address this need, we conducted a crossover study on a First Nations reserve in Manitoba, Canada, including 37 residents in 20 homes. Each home received an electrostatic air filter and a placebo filter for 1 week in random order, and lung function, blood pressure, and endothelial function measures were collected at the beginning and end of each week. Indoor air pollutants were monitored throughout the study period. Indoor PM2.5 decreased substantially during air filter weeks relative to placebo (mean difference: 37 μg/m(3) , 95% CI: 10, 64) but remained approximately five times greater than outdoor concentrations owing to a high prevalence of indoor smoking. On average, air filter use was associated with a 217-ml (95% CI: 23, 410) increase in forced expiratory volume in 1 s, a 7.9-mm Hg (95% CI: -17, 0.82) decrease in systolic blood pressure, and a 4.5-mm Hg (95% CI: -11, 2.4) decrease in diastolic blood pressure. Consistent inverse associations were also observed between indoor PM2.5 and lung function. In general, our findings suggest that reducing indoor PM2.5 may contribute to improved lung function in First Nations communities.
PRACTICAL IMPLICATIONS	Indoor air quality is known to contribute to adverse cardiorespiratory health, but few studies have examined indoor air quality in First Nations communities. Our findings suggest that indoor PM2.5 may contribute to reduced lung function and that portable air filters may help to alleviate these effects by effectively reducing indoor levels of particulate matter.

표-157. PubMed 논문번호 23222514의 내용 요약

구분	내용
PubMed ID	23222514
TITLE	Long-term exposure to outdoor air pollution and incidence of cardiovascular diseases.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e318276ccb8
AUTHORS	Atkinson Richard W, Carey Iain M, Kent Andrew J, van Staa Tjeerd P, Anderson H Ross, Cook Derek G
BACKGROUND	Evidence based largely on US cohorts suggests that long-term exposure to fine particulate matter is associated with cardiovascular mortality. There is less evidence for other pollutants and for cardiovascular morbidity. By using a cohort of 836,557 patients age 40 to 89 years registered with 205 English general practices in 2003, we investigated relationships between ambient outdoor air pollution and incident myocardial infarction, stroke, arrhythmia, and heart failure over a 5-year period.
METHODS	Events were identified from primary care records, hospital admissions, and death certificates. Annual average concentrations in 2002 for particulate matter with a median aerodynamic diameter <10 (PM10) and <2.5 microns, nitrogen dioxide (NO2), ozone, and sulfur dioxide at a 1 × 1 km resolution were derived from emission-based models and linked to residential postcode. Analyses were performed using Cox proportional hazards models adjusting for relevant confounders, including social and economic deprivation and smoking.
RESULTS	While evidence was weak for relationships with myocardial infarction, stroke, or arrhythmia, we found consistent associations between pollutant concentrations and incident cases of heart failure. An interquartile range change in PM10 and in NO2 (3.0 and 10.7 □g/m, respectively) both produced a hazard ratio of 1.06 (95% confidence interval = 1.01-1.11) after adjustment for confounders. There was some evidence that these effects were greater in more affluent areas.
CONCLUSIONS	This study of an English national cohort found evidence linking long-term exposure to particulate matter and NO2 with the development of heart failure. We did not, however, replicate associations for other cardiovascular outcomes that have been reported elsewhere.

표-158. PubMed 논문번호 23222554의 내용 요약

구분	내용
PubMed ID	23222554
TITLE	Long-term exposure to traffic-related air pollution and cardiovascular mortality.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e318276c005
AUTHORS	Chen Hong, Goldberg Mark S, Burnett Richard T, Jerrett Michael, Wheeler Amanda J, Villeneuve Paul J
BACKGROUND	Findings from previous cohort studies suggest a positive association between traffic-related air pollution and cardiovascular mortality. However, few studies have assessed intraurban variation in traffic-related pollution or evaluated cardiovascular effects at lower levels of pollution that are typically seen in Canadian cities.
METHODS	We conducted a cohort study of traffic-related air pollution and cardiovascular mortality among adults who lived in three cities in Ontario, Canada. Study members of the cohort were a random sample from the federal family income tax database, comprising 205,440 adults age 35-85 years, who lived in Toronto, Hamilton, or Windsor between 1982 and 1986. Follow-up ended on 31 December 2004. Mortality from cardiovascular and cerebrovascular diseases was ascertained using the Canadian Mortality Database. We estimated time-dependent concentrations of ambient nitrogen dioxide (NO2) from land-use regression models and assigned exposures to residences of subjects. Rate ratios (RRs) were estimated from Cox proportional hazard model adjusted for individual risk factors and selected contextual covariables. We adjusted indirectly for smoking and obesity.
RESULTS	The spatial distributions of NO2 did not change appreciably over the follow-up period. Cumulative exposure to NO2 was associated with a 12% increase in mortality from cardiovascular disease for each increase of 5 parts per billion of NO2 (95% confidence interval [CI] = 7%-17%) and a 15% increase (8%-21%) in mortality from ischemic heart disease. Risks of cardiovascular mortality were also increased with shorter term exposures, but the RRs were somewhat smaller. No association was found for cerebrovascular mortality (RR = 0.99 [95% CI = 0.91-1.08]).
CONCLUSION	Traffic-related air pollution at relatively low concentrations in Ontario was associated with increased mortality from cardiovascular disease.

표-159. PubMed 논문번호 23231781의 내용 요약

구분	내용
PubMed ID	23231781
TITLE	Chemical constituents of ambient particulate air pollution and biomarkers of inflammation, coagulation and homocysteine in healthy adults: a prospective panel study.
JOURNAL	Particle and fibre toxicology: 10.1186/1743-8977-9-49
AUTHORS	Wu Shaowei, Deng Furong, Wei Hongying, Huang Jing, Wang Hongyi, Shima Masayuki, Wang Xin, Qin Yu, Zheng Chanjuan, Hao Yu, Guo Xinbiao
BACKGROUND	Ambient air pollution has been associated with activation of systemic inflammation and hypercoagulability and increased plasma homocysteine, but the chemical constituents behind the association are not well understood. We examined the relations of various chemical constituents of fine particles (PM(2.5)) and biomarkers of inflammation, coagulation and homocysteine in the context of traffic-related air pollution.
METHODS	A panel of 40 healthy college students underwent biweekly blood collection for 12 times before and after their relocation from a suburban campus to an urban campus with changing air pollution contents in Beijing. Blood samples were measured for circulatory biomarkers of high-sensitivity C reactive protein (hs-CRP), tumor necrosis factor alpha (TNF-α), fibrinogen, plasminogen activator inhibitor type 1 (PAI-1), tissue-type plasminogen activator (t-PA), von Willebrand factor (vWF), soluble platelet selectin (sP-selectin), and total homocysteine (tHcy). Various air pollutants were measured in a central air-monitoring station in each campus and 32 PM(2.5) chemical constituents were determined in the laboratory. We used three different mixed-effects models (single-constituent model, constituent-PM(2.5) joint model and constituent residual model) controlling for potential confounders to estimate the effects of PM(2.5) chemical constituents on circulatory biomarkers.
RESULTS	We found consistent positive associations between the following biomarkers and PM(2.5) chemical constituents across different models: TNF-α with secondary organic carbon, chloride, zinc, molybdenum and stannum; fibrinogen with magnesium, iron, titanium, cobalt and cadmium; PAI-1 with titanium, cobalt and manganese; t-PA with cadmium and selenium; vWF with aluminum. We also found consistent inverse associations of vWF with nitrate, chloride and sodium, and sP-selectin with manganese. Two positive associations of zinc with TNF-α and of cobalt with fibrinogen, and two inverse associations of nitrate with vWF, and of manganese with sP-selectin, were independent of the other constituents in two-constituent models using constituent residual data. We only found weak air pollution effects on hs-CRP and tHcy.
CONCLUSIONS	Our results provide clues for the potential roles that PM(2.5) chemical constituents may play in the biological mechanisms through which air pollution may influence the cardiovascular system.

표-160. PubMed 논문번호 23245609의 내용 요약

구분	내용
PubMed ID	23245609
TITLE	A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(12)61766-8
AUTHORS	Lim Stephen S, Vos Theo, Flaxman Abraham D, Danaei Goodarz, Shibuya Kenji, Adair-Rohani Heather, Amann Markus, Anderson H Ross, Andrews Kathryn G, Aryee Martin, Atkinson Charles, Bacchus Loraine J, Bahalim Adil N, Balakrishnan Kalpana, Balmes John, Barker-Collo Suzanne, Baxter Amanda, Bell Michelle L, Blore Jed D, Blyth Fiona, Bonner Carissa, Borges Guilherme, Bourne Rupert, Boussinesq Michel, Brauer Michael, Brooks Peter, Bruce Nigel G, Brunekreef Bert, Bryan-Hancock Claire, Bucello Chiara, Buchbinder Rachelle, Bull Fiona, Burnett Richard T, Byers Tim E, Calabria Bianca, Carapetis Jonathan, Carnahan Emily, Chafe Zoe, Charlson Fiona, Chen Honglei, Chen Jian Shen, Cheng Andrew Tai-Ann, Child Jennifer Christine, Cohen Aaron, Colson K Ellicott, Cowie Benjamin C, Darby Sarah, Darling Susan, Davis Adrian, Degenhardt Louisa, Dentener Frank, Des Jarlais Don C, Devries Karen, Dherani Mukesh, Ding Eric L, Dorsey E Ray, Driscoll Tim, Edmond Karen, Ali Suad Eltahir, Engell Rebecca E, Erwin Patricia J, Fahimi Saman, Falder Gail, Farzadfar Farshad, Ferrari Alize, Finucane Mariel M, Flaxman Seth, Fowkes Francis Gerry R, Freedman Greg, Freeman Michael K, Gakidou Emmanuela, Ghosh Santu, Giovannucci Edward, Gmel Gerhard, Graham Kathryn, Grainger Rebecca, Grant Bridget, Gunnell David, Gutierrez Hialy R, Hall Wayne, Hoek Hans W, Hogan Anthony, Hosgood H Dean, Hoy Damian, Hu Howard, Hubbell Bryan J, Hutchings Sally J, Ibeanusi Sydney E, Jacklyn Gemma L, Jasrasaria Rashmi, Jonas Jost B, Kan Haidong, Kanis John A, Kassebaum Nicholas, Kawakami Norito, Khang Young-Ho, Khatibzadeh Shahab, Khoo Jon-Paul, Kok Cindy, Laden Francine, Lalloo Ratilal, Lan Qing, Lathlean Tim, Leasher Janet L, Leigh James, Li Yang, Lin John Kent, Lipshultz Steven E, London Stephanie, Lozano Rafael, Lu Yuan, Mak Joelle, Malekzadeh Reza, Mallinger Leslie, Marcenes Wagner, March Lyn, Marks Robin, Martin Randall, McGale Paul, McGrath John, Mehta Sumi, Mensah George A, Merriman Tony R, Micha Renata, Michaud Catherine, Mishra Vinod, Mohd Hanafiah Khayriyyah, Mokdad Ali A, Morawska Lidia, Mozaffarian Dariush, Murphy Tasha, Naghavi Mohsen, Neal Bruce, Nelson Paul K, Nolla Joan Miquel, Norman Rosana, Olives Casey, Omer Saad B, Orchard Jessica, Osborne Richard, Ostro Bart, Page Andrew, Pandey Kiran D, Parry Charles D H, Passmore Erin, Patra Jayadeep, Pearce Neil, Pelizzari Pamela M, Petzold Max, Phillips Michael R, Pope Dan, Pope C Arden, Powles John, Rao Mayuree, Razavi Homie, Rehfuess Eva A, Rehm J□rgen T, Ritz Beate, Rivara Frederick P, Roberts Thomas, Robinson Carolyn, Rodriguez-Portales Jose A, Romieu Isabelle, Room Robin, Rosenfeld Lisa C, Roy Ananya, Rushton Lesley, Salomon Joshua A, Sampson Uchechukwu, Sanchez-Riera Lidia, Sanman Ella, Sapkota Amir, Seedat Soraya, Shi Peilin, Shield Kevin, Shivakoti Rupak, Singh Gitanjali M, Sleet David A, Smith Emma, Smith Kirk R, Stapelberg Nicolas J C, Steenland Kyle, St□ckl Heidi, Stovner Lars Jacob, Straif Kurt, Straney Lahn, Thurston George D, Tran Jimmy H, Van Dingenen Rita, van Donkelaar Aaron, Veerman J Lennert, Vijayakumar Lakshmi, Weintraub Robert, Weissman Myrna M, White Richard A, Whiteford Harvey, Wiersma Steven T, Wilkinson James D, Williams Hywel C, Williams Warwick, Wilson Nicholas, Woolf Anthony D, Yip Paul, Zielinski Jan M, Lopez Alan D, Murray Christopher J L, Ezzati Majid, AlMazroa Mohammad A, Memish Ziad A

표-161. PubMed 논문번호 23268570의 내용 요약

구분	내용
PubMed ID	23268570
TITLE	Low level maternal smoking and infant birthweight reduction: genetic contributions of GSTT1 and GSTM1 polymorphisms.
JOURNAL	BMC pregnancy and childbirth: 10.1186/1471-2393-12-161
AUTHORS	Danileviciute Asta, Grazuleviciene Regina, Paulauskas Algimantas, Nadisauskiene Ruta, Nieuwenhuijsen Mark J
BACKGROUND	Genetic susceptibility to tobacco smoke might modify the effect of smoking on pregnancy outcomes.
METHODS	We conducted a case-control study of 543 women who delivered singleton live births in Kaunas (Lithuania), examining the association between low-level tobacco smoke exposure (mean: 4.8 cigarettes/day) during pregnancy, GSTT1 and GSTM1 polymorphisms and birthweight of the infant. Multiple linear-regression analysis was performed adjusting for gestational age, maternal education, family status, body mass index, blood pressure, and parity. Subsequently, we tested for the interaction effect of maternal smoking, GSTT1 and GSTM1 genes polymorphisms with birthweight by adding all the product terms in the regression models.
RESULTS	The findings suggested a birthweight reduction among light-smoking with the GSTT1-null genotype (-162.9 g, P = 0.041) and those with the GSTM1-null genotype (-118.7 g, P = 0.069). When a combination of these genotypes was considered, birthweight was significantly lower for infants of smoking women the carriers of the double-null genotypes (-311.2 g, P = 0.008). The interaction effect of maternal smoking, GSTM1 and GSTT1 genotypes was marginally significant on birthweight (-234.5 g, P = 0.078). Among non-smokers, genotype did not independently confer an adverse effect on infant birthweight.
CONCLUSIONS	The study shows the GSTT1-null genotype, either presents only one or both with GSTM1-null genotype in a single subject, have a modifying effect on birthweight among smoking women even though their smoking is low level. Our data also indicate that identification of the group of susceptible subjects should be based on both environmental exposure and gene polymorphism. Findings of this study add additional evidence on the interplay among two key GST genes and maternal smoking on birth weight of newborns.

표-162. PubMed 논문번호 23280679의 내용 요약

구분	내용
PubMed ID	23280679
TITLE	Ischemic heart disease mortality and PM(3.5) in a cohort of autoworkers.
JOURNAL	American journal of industrial medicine: 10.1002/ajim.22152
AUTHORS	Costello Sadie, Garcia Erika, Hammond S Katharine, Eisen Ellen A, Costello Sadie, Garcia Erika, Hammond S Katharine, Eisen Ellen A
BACKGROUND	Increased risk of ischemic heart disease (IHD) has been associated with particulate matter (PM) from air pollution. Yet evidence of increased risk associated with higher workplace exposures is scant.
METHODS	We examined the exposure-response relationship between IHD mortality and PM(3.5) (<3.5 □m diameter) from current and cumulative exposure to straight metalworking fluid in a cohort of 39,412 autoworkers followed from 1941 to 1995. Age, calendar year of follow up, sex, race, and plant were included in each model.
RESULTS	To address the decrease in polycyclic-aromatic hydrocarbon (PAH) content in the straight metalworking fluid over time, analyses were stratified by calendar time. Increased risk of IHD mortality was associated with current exposure to PM(3.5) before 1971 and with cumulative exposure to PM(3.5) after 1971.
CONCLUSIONS	Results provide modest evidence that occupational exposure to fine PM from straight fluids, especially fluid with higher PAH, may increase the risk of IHD mortality.

표-163. PubMed 논문번호 23299843의 내용 요약

구분	내용
PubMed ID	23299843
TITLE	Evaluation of interventions to reduce air pollution from biomass smoke on mortality in Launceston, Australia: retrospective analysis of daily mortality, 1994-2007.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.e8446
AUTHORS	Johnston Fay H, Hanigan Ivan C, Henderson Sarah B, Morgan Geoffrey G
OBJECTIVE	To assess the effect of reductions in air pollution from biomass smoke on daily mortality.
DESIGN	Age stratified time series analysis of daily mortality with Poisson regression models adjusted for the effects of temperature, humidity, day of week, respiratory epidemics, and secular mortality trends, applied to an intervention and control community.
SETTING	Central Launceston, Australia, a town in which coordinated strategies were implemented to reduce pollution from wood smoke and central Hobart, a comparable city in which there were no specific air quality interventions.
PARTICIPANTS	67,000 residents of central Launceston and 148,000 residents of central Hobart (at 2001 census).
INTERVENTIONS	Community education campaigns, enforcement of environmental regulations, and a wood heater replacement programme to reduce ambient pollution from residential wood stoves started in the winter of 2001.
MAIN OUTCOME MEASURES	Changes in daily all cause, cardiovascular, and respiratory mortality during the 6.5 year periods before and after June 2001 in Launceston and Hobart.
RESULTS	Mean daily wintertime concentration of PM(10) (particulate matter with particle size <10 □m diameter) fell from 44 □g/m(3) during 1994-2000 to 27 □g/m(3) during 2001-07 in Launceston. The period of improved air quality was associated with small non-significant reductions in annual mortality. In males the observed reductions in annual mortality were larger and significant for all cause (-11.4%, 95% confidence interval -19.2% to -2.9%; P=0.01), cardiovascular (-17.9%, -30.6% to -2.8%; P=0.02), and respiratory (-22.8%, -40.6% to 0.3%; P=0.05) mortality. In wintertime reductions in cardiovascular (-19.6%, -36.3% to 1.5%; P=0.06) and respiratory (-27.9%, -49.5% to 3.1%; P=0.07) mortality were of borderline significance (males and females combined). There were no significant changes in mortality in the control city of Hobart.
CONCLUSIONS	Decreased air pollution from ambient biomass smoke was associated with reduced annual mortality in males and with reduced cardiovascular and respiratory mortality during winter months.

표-164. PubMed 논문번호 23308401의 내용 요약

구분	내용
PubMed ID	23308401
TITLE	Long-term exposure to urban air pollution and mortality in a cohort of more than a million adults in Rome.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1205862
AUTHORS	Cesaroni Giulia, Badaloni Chiara, Gariazzo Claudio, Stafoggia Massimo, Sozzi Roberto, Davoli Marina, Forastiere Francesco
BACKGROUND	Few European studies have investigated the effects of long-term exposure to both fine particulate matter (≤ 2.5 □m; PM2.5) and nitrogen dioxide (NO2) on mortality.
OBJECTIVES	We studied the association of exposure to NO2, PM2.5, and traffic indicators on cause-specific mortality to evaluate the form of the concentration-response relationship.
METHODS	We analyzed a population-based cohort enrolled at the 2001 Italian census with 9 years of follow-up. We selected all 1,265,058 subjects ≥ 30 years of age who had been living in Rome for at least 5 years at baseline. Residential exposures included annual NO2 (from a land use regression model) and annual PM2.5 (from a Eulerian dispersion model), as well as distance to roads with > 10,000 vehicles/day and traffic intensity. We used Cox regression models to estimate associations with cause-specific mortality adjusted for individual (sex, age, place of birth, residential history, marital status, education, occupation) and area (socioeconomic status, clustering) characteristics.
RESULTS	Long-term exposures to both NO2 and PM2.5 were associated with an increase in nonaccidental mortality [hazard ratio (HR) = 1.03 (95% CI: 1.02, 1.03) per 10-□g/m3 NO2; HR = 1.04 (95% CI: 1.03, 1.05) per 10-□g/m3 PM2.5]. The strongest association was found for ischemic heart diseases (IHD) [HR = 1.10 (95% CI: 1.06, 1.13) per 10-□g/m3 PM2.5], followed by cardiovascular diseases and lung cancer. The only association showing some deviation from linearity was that between NO2 and IHD. In a bi-pollutant model, the estimated effect of NO2 on mortality was independent of PM2.5.
CONCLUSIONS	This large study strongly supports an effect of long-term exposure to NO2 and PM2.5 on mortality, especially from cardiovascular causes. The results are relevant for the next European policy decisions regarding air quality.

표-165. PubMed 논문번호 23384207의 내용 요약

구분	내용
PubMed ID	23384207
TITLE	In-line filtration minimizes organ dysfunction: new aspects from a prospective, randomized, controlled trial.
JOURNAL	BMC pediatrics: 10.1186/1471-2431-13-21
AUTHORS	Boehne Martin, Jack Thomas, K□ditz Harald, Seidemann Kathrin, Schmidt Florian, Abura Michaela, Bertram Harald, Sasse Michael
BACKGROUND	Infused particles induce thrombogenesis, impair microcirculation and modulate immune response. We have previously shown in critically ill children, that particle-retentive in-line filtration reduced the overall complication rate of severe events, length of stay and duration of mechanical ventilation. We now evaluated the influence of in-line filtration on different organ function and thereby elucidated the potential underlying pathophysiological effects of particle infusion.
METHODS	In this single-centre, prospective, randomized controlled trial 807 critically ill children were assigned to either control (n = 406) or filter group (n = 401), the latter receiving in-line filtration for complete infusion therapy. Both groups were compared regarding the differences of incidence rates and its 95% confidence interval (CI) of different organ dysfunction as defined by the International Pediatric Sepsis Consensus Conference 2005.
RESULTS	The incidence rates of respiratory (-5.06%; 95% CI, -9.52 to -0.59%), renal (-3.87%; 95% CI, -7.58 to -0.15%) and hematologic (-3.89%; 95% CI, -7.26 to -0.51%) dysfunction were decreased in the filter group. No difference was demonstrated for the occurrence rates of cardiovascular, hepatic, or neurologic dysfunction between both groups.
CONCLUSIONS	In-line filtration has beneficial effects on the preservation of hematologic, renal and respiratory function in critically ill patients. The presented clinical data further support our hypothesis regarding potential harmful effects of particles. In critically ill patients infused particles may lead to further deterioration of the microcirculation, induce a systemic hypercoagulability and inflammation with consecutive negative effects on organ function.
TRIAL REGISTRATION	ClinicalTrials.gov number; NCT00209768.

표-166. PubMed 논문번호 23384708의 내용 요약

구분	내용
PubMed ID	23384708
TITLE	Association between long-term exposure to traffic-related air pollution and subclinical atherosclerosis: the REGICOR study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1205146
AUTHORS	Rivera Marcela, Basaga□a Xavier, Aguilera Inmaculada, Foraster Maria, Agis David, de Groot Eric, Perez Laura, Mendez Michelle A, Bouso Laura, Targa Jaume, Ramos Rafael, Sala Joan, Marrugat Jaume, Elosua Roberto, K□nzli Nino, Rivera Marcela, Basaga□a Xavier, Aguilera Inmaculada, Foraster Maria, Agis David, de Groot Eric, Perez Laura, Mendez Michelle A, Bouso Laura, Targa Jaume, Ramos Rafael, Sala Joan, Marrugat Jaume, Elosua Roberto, K□nzli Nino
BACKGROUND	Epidemiological evidence of the effects of long-term exposure to air pollution on the chronic processes of atherogenesis is limited.
OBJECTIVE	We investigated the association of long-term exposure to traffic-related air pollution with subclinical atherosclerosis, measured by carotid intima media thickness (IMT) and ankle-brachial index (ABI).
METHODS	We performed a cross-sectional analysis using data collected during the reexamination (2007-2010) of 2,780 participants in the REGICOR (Registre Giron□ del Cor: the Gerona Heart Register) study, a population-based prospective cohort in Girona, Spain. Long-term exposure across residences was calculated as the last 10 years' time-weighted average of residential nitrogen dioxide (NO2) estimates (based on a local-scale land-use regression model), traffic intensity in the nearest street, and traffic intensity in a 100 m buffer. Associations with IMT and ABI were estimated using linear regression and multinomial logistic regression, respectively, controlling for sex, age, smoking status, education, marital status, and several other potential confounders or intermediates.
RESULTS	Exposure contrasts between the 5th and 95th percentiles for NO2 (25 □g/m3), traffic intensity in the nearest street (15,000 vehicles/day), and traffic load within 100 m (7,200,000 vehicle-m/day) were associated with differences of 0.56% (95% CI: -1.5, 2.6%), 2.32% (95% CI: 0.48, 4.17%), and 1.91% (95% CI: -0.24, 4.06) percent difference in IMT, respectively. Exposures were positively associated with an ABI of > 1.3, but not an ABI of < 0.9. Stronger associations were observed among those with a high level of education and in men ≥ 60 years of age.
CONCLUSIONS	Long-term traffic-related exposures were associated with subclinical markers of atherosclerosis. Prospective studies are needed to confirm associations and further examine differences among population subgroups.

표-167. PubMed 논문번호 23406673의 내용 요약

구분	내용
PubMed ID	23406673
TITLE	A case-crossover analysis of out-of-hospital cardiac arrest and air pollution.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.113.000027
AUTHORS	Ensor Katherine B, Raun Loren H, Persse David
BACKGROUND	Evidence of an association between the exposure to air pollution and overall cardiovascular morbidity and mortality is increasingly found in the literature. However, results from studies of the association between acute air pollution exposure and risk of out-of-hospital cardiac arrest (OHCA) are inconsistent for fine particulate matter, and, although pathophysiological evidence indicates a plausible link between OHCA and ozone, none has been reported. Approximately 300 000 persons in the United States experience an OHCA each year, of which >90% die. Understanding the association provides important information to protect public health.
METHODS AND RESULTS	The association between OHCA and air pollution concentrations hours and days before onset was assessed by using a time-stratified case-crossover design using 11 677 emergency medical service-logged OHCA events between 2004 and 2011 in Houston, Texas. Air pollution concentrations were obtained from an extensive area monitor network. An average increase of 6 □g/m(3) in fine particulate matter 2 days before onset was associated with an increased risk of OHCA (1.046; 95% confidence interval, 1.012-1.082). A 20-ppb ozone increase for the 8-hour average daily maximum was associated with an increased risk of OHCA on the day of the event (1.039; 95% confidence interval, 1.005-1.073). Each 20-ppb increase in ozone in the previous 1 to 3 hours was associated with an increased risk of OHCA (1.044; 95% confidence interval, 1.004-1.085). Relative risk estimates were higher for men, blacks, or those aged >65 years.
CONCLUSIONS	The findings confirm the link between OHCA and fine particulate matter and introduce evidence of a similar link with ozone.

표-168. PubMed 논문번호 23413885의 내용 요약

구분	내용
PubMed ID	23413885
TITLE	Modification by hemochromatosis gene polymorphisms of the association between traffic-related air pollution and cognition in older men: a cohort study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-12-16
AUTHORS	Power Melinda C, Weisskopf Marc G, Alexeeff Stacey E, Wright Robert O, Coull Brent A, Spiro Avron, Schwartz Joel
BACKGROUND	Previous studies found effect modification of associations between traffic-related air pollution and cardiovascular outcomes by polymorphisms in the hemochromatosis gene (HFE). As traffic-related air pollution may impact cognition through effects on cardiovascular health or through mechanisms which may also influence cardiovascular outcomes, we hypothesized that HFE polymorphisms would also modify a previously observed association between traffic-related air pollution exposure and cognition in older men.
METHODS	We considered data from 628 participants of the VA Normative Aging Study. We estimated long term exposure to black carbon (BC), a marker of traffic related air pollution, using a spatio-temporal land use regression model. We assessed cognition using the Mini-Mental State Examination (MMSE), a test of global function, and performance on a battery of other tests, covering a wide range of domains. We investigated whether variants of HFE C282Y and H63D modified the association between BC and having a low MMSE score using logistic models with generalized estimating equations and multiplicative interaction terms. Similarly, we assessed whether HFE variants modified the association between BC and performance on the cognitive battery using linear mixed models with multiplicative interaction terms.
RESULTS	Our results suggest modification of the BC-cognition association by HFE C282Y, although the test of interaction did not achieve statistical significance. In multivariable-adjusted models, participants who lacked a HFE C282Y variant (CC) exhibited an adverse association between BC and total cognition z-score (beta for a doubling in BC concentration: -0.061, 95% CI: -0.115, -0.007), while we did not observe an association in participants with at least one variant genotype (CY or YY) (beta for a doubling in BC concentration: 0.073, 95% CI: -0.081, 0.228; p-value for interaction: 0.11). The pattern of association was similar for analyses considering performance on the Mini-Mental State Examination. There was little evidence to support effect modification of the BC-cognition association by the HFE H63D genotype.
CONCLUSIONS	Our data suggest that older adults who lack an HFE C282Y variant may be more susceptible to an adverse effect of traffic-related air pollution exposure on cognition. This finding and the proposed biological mechanism require confirmation.

표-169. PubMed 논문번호 23423735의 내용 요약

구분	내용
PubMed ID	23423735
TITLE	Long-term exposure to air pollution is associated with survival following acute coronary syndrome.
JOURNAL	European heart journal: 10.1093/eurheartj/ehs480
AUTHORS	Tonne Cathryn, Wilkinson Paul
AIMS	The aim of this study was to determine (i) whether long-term exposure to air pollution was associated with all-cause mortality using the Myocardial Ischaemia National Audit Project (MINAP) data for England and Wales, and (ii) the extent to which exposure to air pollution contributed to socioeconomic inequalities in prognosis.
METHODS AND RESULTS	Records of patients admitted to hospital with acute coronary syndrome (ACS) in MINAP collected under the National Institute for Cardiovascular Outcomes Research were linked to modelled annual average air pollution concentrations for 2004-10. Hazard ratios for mortality starting 28 days after admission were estimated using Cox proportional hazards models. Among the 154 204 patients included in the cohort, the average follow-up was 3.7 years and there were 39 863 deaths. Mortality rates were higher for individuals exposed to higher levels of particles with a diameter of ≤2.5 □m (PM2.5; PM, particulate matter): the fully adjusted hazard ratio for a 10 □g/m(3) increase in PM2.5 was 1.20 (95% CI 1.04-1.38). No associations were observed for larger particles or oxides of nitrogen. Air pollution explained socioeconomic inequalities in survival to only a small extent.
CONCLUSION	Mortality from all causes was higher among individuals with greater exposure to PM2.5 in survivors of hospital admission for ACS in England and Wales. Despite higher exposure to PM2.5 among those from more deprived areas, such exposure was a minor contribution to the socioeconomic inequalities in prognosis following ACS. Our findings add to the evidence of mortality associated with long-term exposure to fine particles.

표-170. PubMed 논문번호 23429405의 내용 요약

구분	내용
PubMed ID	23429405
TITLE	Maternal active and passive smoking and hypertensive disorders of pregnancy: risk with trimester-specific exposures.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e3182873a73
AUTHORS	Engel Stephanie M, Scher Erica, Wallenstein Sylvan, Savitz David A, Alsaker Elin R, Trogstad Lill, Magnus Per
BACKGROUND	The inverse association between prenatal smoking and preeclampsia is puzzling, given the increased risks of prematurity and low birthweight associated with both smoking and preeclampsia. We analyzed the Norwegian Mother and Child Birth Cohort (MoBa) to determine whether the associations varied by timing of prenatal smoking.
METHODS	We conducted an analysis of 74,439 singleton pregnancies with completed second- and third- trimester questionnaires. Active and passive smoke exposure by trimester were determined by maternal self-report, and covered the period of preconception through approximately 30 weeks' gestation. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were calculated.
RESULTS	Rates of active smoking declined dramatically during pregnancy: for trimester 1, 23%; trimester 2, 9%; and trimester 3, 8%. Active smoking in the third trimester was associated with reduced odds of preeclampsia and gestational hypertension, with the strongest association among continuous smokers (for preeclampsia, OR= 0.57 [95% CI = 0.46-0.70]). Women who quit smoking before the third trimester had approximately the same risk of preeclampsia and gestational hypertension as nonsmokers. There was some evidence of dose-response, with the heaviest smokers (more than eight cigarettes per day) having the lowest risks of preeclampsia (0.48 [0.32-0.73]) and gestational hypertension (0.51 [0.28-0.95]). There was little evidence of an association with passive smoking exposure.
CONCLUSION	The association between smoking and preeclampsia varies substantially according to the timing and intensity of exposure. A better understanding of the biologic pathways that underlie these associations may provide important clues to the etiology of preeclampsia and the development of effective clinical interventions.

표-171. PubMed 논문번호 23490845의 내용 요약

구분	내용
PubMed ID	23490845
TITLE	Second-hand tobacco smoke in never smokers is a significant risk factor for coronary artery calcification.
JOURNAL	JACC. Cardiovascular imaging: 10.1016/j.jcmg.2013.02.004
AUTHORS	Yankelevitz David F, Henschke Claudia I, Yip Rowena, Boffetta Paolo, Shemesh Joseph, Cham Matthew D, Narula Jagat, Hecht Harvey S
OBJECTIVES	The aim of this study was to assess the relationship of the extent of subclinical atherosclerosis measured by coronary artery calcification (CAC) to the extent of second-hand tobacco smoke (SHTS) exposure in asymptomatic people who never smoked.
BACKGROUND	An association between SHTS and CAC was recently reported in a single study, but the quantitative aspects of the relationship are not known.
METHODS	A cohort of 3,098 never smokers 40 to 80 years of age, enrolled in the FAMRI-IELCAP (Flight Attendant Medical Research Institute International Early Lung Cancer Action Program) screening program, completed a SHTS questionnaire, and had a low-dose nongated computed tomography scan. The questionnaire provided a quantitative score for total SHTS exposure, as well as separately as a child and as an adult at home and at work; 4 categories of exposure to SHTS were identified (minimal, low, moderate, and high exposure). CAC was graded using a previously validated ordinal scale score that ranged from 0 to 12. Logistic regression analysis of the prevalence and ordered logistic regression analysis of the extent of CAC were performed to assess the independent contribution of SHTS adjusted for age, sex, diabetes, hypercholesterolemia, hypertension, and renal disease. Linear and quadratic regression analyses of CAC and SHTS were performed.
RESULTS	The prevalence of CAC was 24.3% (n = 754) and was significantly higher in those with more than minimal SHTS exposure compared with those with minimal SHTS exposure (26.4% vs. 18.5%, $p < 0.0001$). The adjusted odds ratios for CAC prevalence were 1.54 (95% confidence interval: 1.17 to 2.20) for low SHTS exposure, 1.60 (95% confidence interval: 1.21 to 2.10) for moderate exposure, and 1.93 (95% confidence interval: 1.49 to 2.51) for high exposure. The association of the extent of SHTS with the extent of CAC was confirmed by the adjusted odds ratio ($p < 0.0001$).
CONCLUSIONS	The presence and extent of CAC were associated with extent of SHTS exposure even when adjusted for other risk factors for CAC, suggesting that SHTS exposure causes CAC.

표-172. PubMed 논문번호 23503825의 내용 요약

구분	내용
PubMed ID	23503825
TITLE	The oxidative response of mouse hearts is modulated by genetic background.
JOURNAL	Arquivos brasileiros de cardiologia: 10.5935/abc.20130029
AUTHORS	Santos-Silva Marco Aur□lio, Nagato Akinori Cardozo, Trajano Eduardo Tavares Lima, Alves Jackson Nogueira, Bandeira Ana Carla Balthar, Porto Lu□s Crist□v□o, Bezerra Frank Silva
BACKGROUND	Smoking plays an important role in cardiovascular diseases. However, the reasons why some individuals develop those diseases and others do not remain to be explained.
OBJECTIVE	This study aimed at assessing the redox profile of the heart of different mouse strains after exposure to cigarette smoke.
METHODS	Male mice of the Swiss (n = 10), C3H (n = 10), BALB/c (n = 10) and C57BL/6 (n = 10) strains were exposed to cigarette smoke (12 cigarettes/day), while their respective controls (n = 10) were exposed to ambient air for 60 days. After being euthanized, their heart was removed for biochemical analyses.
RESULTS	Although the malondialdehyde content did not increase in any of the groups, catalase activity decreased in the Swiss ($p < 0.05$) and BALB/c ($p < 0.05$) strain mice as compared with their respective control groups, while myeloperoxidase decreased in the C3H ($p < 0.05$) and C57BL/6 ($p < 0.001$) strain mice as compared with their respective control groups. The reduced glutathione content decreased in the Swiss, C3H, C57BL/6 ($p < 0.05$) and BALB/c ($p < 0,001$) strain mice as compared with their respective control groups. Regarding reduced glutathione content, an increase was observed in the Swiss strain mice ($p < 0.05$), while a decrease was observed in the C3H ($p < 0.05$) and BALB/c ($p < 0.001$) strain mice as compared with their respective control groups. The reduced glutathione/reduced glutathione ratio showed a reduction in the Swiss and C57BL/6 ($p < 0.05$) strain mice as compared with their respective control groups.
CONCLUSIONS	The genetic background of mice can influence the antioxidant response after exposure to cigarette smoke and seems to be a determinant factor for redox imbalance in Swiss and C57BL/6 strain mice. Understanding antioxidant responses and genetic background of C3H and BALB/c strain mice might provide important information regarding cardiac resistance to cigarette smoke.

표-173. PubMed 논문번호 23522615의 내용 요약

구분	내용
PubMed ID	23522615
TITLE	Associations between ambient air pollution and Hypertensive Disorders of Pregnancy.
JOURNAL	Environmental research: 10.1016/j.envres.2013.01.006
AUTHORS	Mobasher Zahra, Salam Muhammad T, Goodwin T Murphy, Lurmann Frederick, Ingles Sue A, Wilson Melissa L
BACKGROUND	Exposure to ambient air pollution is linked to adverse pregnancy outcomes. Previous reports examining the relationship between ambient air pollution and Hypertensive Disorders of Pregnancy have been inconsistent.
OBJECTIVES	We evaluated the effects of ambient air pollution on the odds of Hypertensive Disorder of Pregnancy and whether these associations varied by body mass index (BMI).
METHODS	We conducted a retrospective, case-control study among 298 predominantly Hispanic women (136 clinically confirmed cases) who attended the Los Angeles County+University of Southern California Women's and Children's Hospital during 1996-2008. Trimester-specific carbon monoxide (CO), nitrogen dioxide (NO2), ozone (O3), and particulate matter with aerodynamic diameter <10 μm and <2.5 μm (PM10, PM2.5) exposure were estimated based on 24-hour exposure level at residential address. Logistic regression models were fitted to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for two standard deviation increase in exposure levels.
RESULTS	Exposures to CO and PM2.5 in the 1st trimester were significantly associated with Hypertensive Disorders of Pregnancy, and these associations were modified by BMI. In non-obese women (BMI <30), 1st trimester exposures to PM2.5 and CO were significantly associated with increased odds of Hypertensive Disorder of Pregnancy (ORs per 2-standard deviation increase in PM2.5 (7 μg/m(3)) and CO (1 ppm) exposures were 9.10 [95% CI: 3.33-24.6] and 4.96 [95% CI: 1.85-13.31], respectively). Additionally, there was a significantly positive association between exposure to O3 in the 2nd trimester and Hypertensive Disorder of Pregnancy (OR per 15 ppb=2.05; 95% CI: 1.22-3.46).
CONCLUSION	Among non-obese women, 1st trimester exposure to PM2.5 and carbon monoxide are associated with increased odds of Hypertensive Disorder of Pregnancy.

표-174. PubMed 논문번호 23525434의 내용 요약

구분	내용
PubMed ID	23525434
TITLE	Altered nitric oxide bioavailability contributes to diesel exhaust inhalation-induced cardiovascular dysfunction in man.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.112.004309
AUTHORS	Langrish Jeremy P, Unosson Jon, Bosson Jenny, Barath Stefan, Muala Ala, Blackwell Scott, S□derberg Stefan, Pourazar Jamshid, Megson Ian L, Treweeke Andrew, Sandstr□m Thomas, Newby David E, Blomberg Anders, Mills Nicholas L
BACKGROUND	Diesel exhaust inhalation causes cardiovascular dysfunction including impaired vascular reactivity, increased blood pressure, and arterial stiffness. We investigated the role of nitric oxide (NO) bioavailability in mediating these effects.
METHODS AND RESULTS	In 2 randomized double-blind crossover studies, healthy nonsmokers were exposed to diesel exhaust or filtered air. Study 1: Bilateral forearm blood flow was measured during intrabrachial infusions of acetylcholine (ACh; 5 to 20 μg/min) and sodium nitroprusside (SNP; 2 to 8 μg/min) in the presence of the NO clamp (NO synthase inhibitor N(G)-monomethyl-l-arginine (l-NMMA) 8 μg/min coinfused with the NO donor SNP at 90 to 540 ng/min to restore basal blood flow). Study 2: Blood pressure, arterial stiffness, and cardiac output were measured during systemic NO synthase inhibition with intravenous l-NMMA (3 mg/kg). Following diesel exhaust inhalation, plasma nitrite concentrations were increased (68±48 versus 41±32 nmol/L; P=0.006) despite similar l-NMMA-induced reductions in basal blood flow (-20.6±14.7% versus -21.1±14.6%; P=0.559) compared to air. In the presence of the NO clamp, ACh and SNP caused dose-dependent vasodilatation that was not affected by diesel exhaust inhalation (P>0.05 for both). Following exposure to diesel exhaust, l-NMMA caused a greater increase in blood pressure (P=0.048) and central arterial stiffness (P=0.007), but reductions in cardiac output and increases in systemic vascular resistance (P>0.05 for both) were similar to those seen with filtered air.
CONCLUSIONS	Diesel exhaust inhalation disturbs normal vascular homeostasis with enhanced NO generation unable to compensate for excess consumption. We suggest the adverse cardiovascular effects of air pollution are, in part, mediated through reduced NO bioavailability.
CLINICAL TRIAL REGISTRATION	URL: http://www.ClinicalTrials.gov. Unique identifiers: NCT00845767 and NCT01060930.

표-175. PubMed 논문번호 23531317의 내용 요약

구분	내용
PubMed ID	23531317
TITLE	A randomized cross-over study of inhalation of diesel exhaust, hematological indices, and endothelial markers in humans.
JOURNAL	Particle and fibre toxicology: 10.1186/1743-8977-10-7
AUTHORS	Krishnan Ranjini M, Sullivan Jeffrey H, Carlsten Chris, Wilkerson Hui-Wen, Beyer Richard P, Bammler Theo, Farin Fred, Peretz Alon, Kaufman Joel D
BACKGROUND	Exposure to traffic-related air pollution (TRAP) is considered a trigger for acute cardiovascular events. Diesel Exhaust (DE) is a major contributor to TRAP in the world. We evaluated the effect of DE inhalation on circulating blood cell populations, hematological indices, and systemic inflammatory cytokines in humans using a specialized facility.
METHODS	In a randomized double-blind crossover study balanced to order, 17 metabolic syndrome (MetS) and 15 healthy subjects inhaled filtered air (FA) or DE exposure in two-hour sessions on different days with a minimum 2-week washout period. We collected blood pre-exposure, 7, and 22 hours after exposure initiation and measured the complete blood count and differential. We performed multiplex cytokine assay to measure the changes in the systemic inflammatory cytokines, and endothelial adhesion molecules (n=15). A paired analysis compared the effect of DE and FA exposures for the change from pre-exposure to the subsequent time points.
RESULTS	A significant increase in the hematocrit was noted 7 hrs after DE [1.4% (95% CI: 0.9 to 1.9%)] compared to FA exposure [0.5% (95% CI: -0.09 to 1.0%); p=0.008. The hemoglobin levels increased non-significantly at 7 hrs post DE [0.3 gm/dL (95% CI: 0.2 to 0.5 gm/dL)] versus FA exposure [0.2 gm/dL (95% CI: 0 to 0.3 gm/dL)]; p=0.06. Furthermore, the platelet count increased 22 hrs after DE exposure in healthy, but not in MetS subjects [DE: 16.6 (95% CI: 10.2 to 23) thousand platelets/mL versus [FA: 3.4 (95% CI: -9.5 to 16.3) thousand platelets/mL)]; p=0.04. No DE effect was observed for WBC, neutrophils, lymphocytes or erythrocytes. Using the multiplex assay, small borderline significant increases in matrix metalloproteinase-9, interleukins (IL)-1 beta, 6 and 10 occurred 7 hrs post exposure initiation, whereas E-selectin, intercellular adhesion molecule-1, and vascular cell adhesion molecule -1, and myeloperoxidase 22 hrs post exposure.
CONCLUSIONS	Our results suggest that short-term DE exposure results in hemoconcentration and thrombocytosis, which are important determinants of acute cardiovascular events. Multiplex assay showed a non-significant increase in IL-1β and IL-6 immediately post exposure followed by myeloperoxidase and endothelial activation molecules. Further specific assays in a larger population will improve our understanding of the systemic inflammatory mechanisms following acute exposure to TRAP.

표-176. PubMed 논문번호 23578893의 내용 요약

구분	내용
PubMed ID	23578893
TITLE	Increased aortic wave reflection and smaller pulse pressure amplification in smokers and passive smokers confirmed by urinary cotinine levels: the Nagahama Study.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2013.03.028
AUTHORS	Tabara Yasuharu, Takahashi Yoshimitsu, Setoh Kazuya, Muro Shigeo, Kawaguchi Takahisa, Terao Chikashi, Kosugi Shinji, Sekine Akihiro, Yamada Ryo, Mishima Michiaki, Nakayama Takeo, Matsuda Fumihiko
BACKGROUND	Central blood pressure (cSBP) is suggested to be a better predictor of cardiovascular risk than brachial BP. Although brachial BP levels among smokers have been reported to be the same or somewhat lower than those in nonsmokers, it is suggested that smoking might have a substantial impact on cSBP.
METHODS	We conducted a cross-sectional study to clarify the association of smoking habit with arterial tone and cSBP in a general population of 8557 participants using urinary cotinine levels as an objective marker of smoking intensity. Absolute pressure of the late systolic peak (SBP2) was obtained by calibrating the radial waveform with brachial systolic BP (bSBP) and considered to be the cSBP.
RESULTS	Confounding factor-adjusted mean pulse pressure amplification (PPa = bSBP-cSBP) was significantly smaller in habitual smokers (current, 9.3 ± 0.15; past, 10.2 ± 0.13; never, 10.6 ± 0.10 mmHg; $p<0.001$). Further, among smokers, PPa was linearly decreased with increasing urinary cotinine quartile (Q1, 10.9 ± 0.38; Q2, 10.9 ± 0.39; Q3, 10.4 ± 0.39; Q4, 9.7 ± 0.41 mmHg; $p = 0.020$). Multiple linear regression analysis identified both smoking habit ($p = 0.003$) and urinary cotinine levels ($p = 0.008$) as independent determinants of PPa. Urinary cotinine was also detected in a small fraction of never smokers (1.8%). These passive smokers showed a smaller PPa (passive smoker, 9.4 ± 0.4; never smoker, 10.4 ± 0.12 mmHg, $p = 0.020$) but not bSBP (122.7 ± 0.6, 123.1 ± 0.2 mmHg, $p = 0.474$).
CONCLUSIONS	Not only habitual smoking but also passive smoking had harmful effects on AIx and central BP. Our results strongly emphasize the importance of avoiding passive smoking to the prevention of cardiovascular risks of which the subject is likely unaware.

표-177. PubMed 논문번호 23584848의 내용 요약

구분	내용
PubMed ID	23584848
TITLE	Secondhand smoke exposure is associated with proteinuria in children with chronic kidney disease.
JOURNAL	Pediatric nephrology (Berlin, Germany): 10.1007/s00467-013-2456-1
AUTHORS	Omoloja Abiodun, Jerry-Fluker Judith, Ng Derek K, Abraham Alison G, Furth Susan, Warady Bradley A, Mitsnefes Mark
BACKGROUND	In adults with chronic kidney disease (CKD), cigarette smoking is associated with an increased risk for CKD progression and transplant failure. In children, secondhand smoke (SHS) exposure has been associated with elevated blood pressure. There are no studies on the prevalence and effect of SHS exposure in CKD.
METHODS	Subjects were enrolled in the Chronic Kidney Disease in Children (CKiD) Study, an observational cohort of 366 children aged 1 to 16 years with CKD. Secondhand smoke exposure was obtained via questionnaire. SHS exposure was also determined based on urine cotinine (Ucot) measurements (1 ng/mL ≤ Ucot 〈 75 ng/mL). The cross-sectional association of SHS exposure with proteinuria was assessed.
RESULTS	Using Ucot, 22 % of subjects were exposed to SHS. SHS exposure was significantly associated with lower maternal education and African American race, and a greater prevalence of nephrotic range proteinuria and left ventricular hypertrophy. In a multivariate model (including sex, age, race, maternal education, income level, private insurance status, abnormal birth history and CKD diagnosis), the prevalence odds of nephrotic range proteinuria was 2.64, (95 % confidence interval 1.08, 6.42) higher in children exposed to SHS compared to those unexposed.
CONCLUSIONS	In our cohort of children with CKD, SHS exposure was common (22 %) and independently associated with nephrotic range proteinuria. Exposure to SHS may be an important factor to consider in CKD progression.

표-178. PubMed 논문번호 23587116의 내용 요약

구분	내용
PubMed ID	23587116
TITLE	Effects of secondhand smoke on the birth weight of term infants and the demographic profile of Saudi exposed women.
JOURNAL	BMC public health: 10.1186/1471-2458-13-341
AUTHORS	Wahabi Hayfaa A, Alzeidan Rasmieh A, Fayed Amel A, Mandil Ahmed, Al-Shaikh Ghadeer, Esmaeil Samia A
BACKGROUND	Maternal exposure to tobacco smoke during pregnancy is associated with detrimental effects on the mother and the fetus including; impaired fetal growth, low birth weight and preterm delivery. In utero exposure to tobacco is implicated in the etiology of many adults' diseases including obesity, diabetes and hypertension.The objectives of this study were to evaluate the effects of Secondhand Tobacco Smoke (SHS) exposure on newborns' anthropometric measurements and to compare the demographic profile of the women exposed to SHS to those who were not.
METHOD	This is a retrospective cohort study investigating the effects of SHS during pregnancy on newborns' anthropometry. Women who self-reported SHS exposure were compared with those not exposed. The primary outcomes were birth weight, newborn length and head circumference. Univariate analysis and multivariate regression analysis were performed. Adjusted differences with 95% confidence intervals were calculated.
RESULTS	Mothers exposed to SHS constituted 31% of the cohort. The mean birth weight of infants of exposed mothers was significantly lower by 35 g, 95% CI: 2-68 g, (P = 0.037) and the mean length was shorter by 0.261 cm, 95% CI 0.058-0.464 cm, (P = 0.012) compared to the infants of unexposed mothers. Women exposed to SHS, were younger, of lower parity and more likely to be illiterate than those who were not exposed in addition, exposed women were less likely to be primiparous.
CONCLUSION	The prevalence of exposure of Saudi pregnant women to SHS is high at 31% and it is associated with reduced birth weight, and shorter length of the newborn.

표-179. PubMed 논문번호 23590261의 내용 요약

구분	내용
PubMed ID	23590261
TITLE	Mortality associations with long-term exposure to outdoor air pollution in a national English cohort.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201210-1758OC
AUTHORS	Carey Iain M, Atkinson Richard W, Kent Andrew J, van Staa Tjeerd, Cook Derek G, Anderson H Ross
RATIONALE	Cohort evidence linking long-term exposure to outdoor particulate air pollution and mortality has come largely from the United States. There is relatively little evidence from nationally representative cohorts in other countries.
OBJECTIVES	To investigate the relationship between long-term exposure to a range of pollutants and causes of death in a national English cohort.
METHODS	A total of 835,607 patients aged 40-89 years registered with 205 general practices were followed from 2003-2007. Annual average concentrations in 2002 for particulate matter with a median aerodynamic diameter less than 10 (PM(10)) and less than 2.5 μm (PM(2.5)), nitrogen dioxide (NO(2)), ozone and sulfur dioxide (SO(2)) at 1 km(2) resolution, estimated from emission-based models, were linked to residential postcode. Deaths (n = 83,103) were ascertained from linkage to death certificates, and hazard ratios (HRs) for all- and cause-specific mortality for pollutants were estimated for interquartile pollutant changes from Cox models adjusting for age, sex, smoking, body mass index, and area-level socioeconomic status markers.
MEASUREMENTS AND MAIN RESULTS	Residential concentrations of all pollutants except ozone were positively associated with all-cause mortality (HR, 1.02, 1.03, and 1.04 for PM(2.5), NO(2), and SO(2), respectively). Associations for PM(2.5), NO(2), and SO(2) were larger for respiratory deaths (HR, 1.09 each) and lung cancer (HR, 1.0 1.06, and 1.05) but nearer unity for cardiovascular deaths (1.00, 1.00, and 1.04).
CONCLUSIONS	These results strengthen the evidence linking long-term ambient air pollution exposure to increased all-cause mortality. However, the stronger association with respiratory mortality are not consistent with most US studies in which associations with cardiovascular causes of death tend to predominate.

표-180. PubMed 논문번호 23594435의 내용 요약

구분	내용
PubMed ID	23594435
TITLE	Meta-analysis of adverse health effects due to air pollution in Chinese populations.
JOURNAL	BMC public health: 10.1186/1471-2458-13-360
AUTHORS	Lai Hak-Kan, Tsang Hilda, Wong Chit-Ming
BACKGROUND	Pooled estimates of air pollution health effects are important drivers of environmental risk communications and political willingness. In China, there is a lack of review studies to provide such estimates for health impact assessments.
METHODS	We systematically searched the MEDLINE database using keywords of 80 major Chinese cities in Mainland China, Hong Kong and Taiwan on 30 June 2012, yielding 350 abstracts with 48 non-duplicated reports either in English or Chinese after screening. We pooled the relative risks (RR) per 10 μg/m3 of particulate matter (PM10), nitrogen dioxide (NO2), sulphur dioxide (SO2) and ozone (O3).
RESULTS	For short-term effects, the pooled RR ($p<0.05$) ranges were: 1.0031 (PM10) to 1.0140 (NO2) for all-cause mortality, 1.0034 (cardiopulmonary, PM10) to 1.0235 (influenza and pneumonia, SO2) for 9 specific-causes mortality, 1.0021 (cardiovascular, PM10) to 1.0162 (asthma, O3) for 5 specific-causes hospital admissions. For birth outcomes, the RR ($p<0.05$) ranged from 1.0051 (stillbirth, O3) to 1.1189 (preterm-birth, SO2) and for long-term effect on mortality from 1.0150 (respiratory, SO2) to 1.0297 (respiratory, NO2). Publication bias was absent (Egger test: p=0.326 to 0.624). Annual PM10 and NO2 concentrations were inversely associated with RR of mortality (p=0.017-0.028).
CONCLUSIONS	Evidence on short-term effects of air pollution is consistent and sufficient for health impact assessment but that on long-term effects is still insufficient.

표-181. PubMed 논문번호 23596186의 내용 요약

구분	내용
PubMed ID	23596186
TITLE	Incidence of myocardial infarction in Swedish chimney sweeps 1991-2005: a prospective cohort study.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2013-101371
AUTHORS	Gustavsson Per, Jansson Catarina, Hogstedt Christer
OBJECTIVES	Previous studies of chimney sweeps have shown an excess mortality from cardiovascular diseases, although the extent of confounding from tobacco smoking is uncertain. The present study used referents of similar socioeconomic background as the chimney sweeps in order to reduce confounding, included both lethal and surviving cases of myocardial infarction, and investigated dose-response in terms of duration of employment.
METHODS	A cohort of 4436 male chimney sweeps was identified from nationwide trade union records from 1918 to 2006. Myocardial infarctions during 1991-2005 were identified from the Swedish nationwide register of first-time myocardial infarctions. Standardised incidence ratios (SIRs) were estimated using skilled manual workers in the service sector in Sweden to calculate expected numbers.
RESULTS	There was a strong and statistically significant excess of myocardial infarction among the chimney sweeps, SIR 1.39 (95% CI 1.24 to 1.55). The excess was observed among both short- and long-term employed.
CONCLUSIONS	While the excess of myocardial infarction among the short-term employed may be due to tobacco and, possibly, alcohol use, it is likely that the excess noted among the long-term employed was caused by the high exposure to combustion products, particles or metals still occurring among chimney sweeps. Preventive measures to reduce hazardous occupational exposures as well as smoking and alcohol use among chimney sweeps are urgently needed.

표-182. PubMed 논문번호 23598582의 내용 요약

구분	내용
PubMed ID	23598582
TITLE	Smoking is associated with remodeling of gap junction in the rat heart: smoker's paradox explanation?
JOURNAL	Arquivos brasileiros de cardiologia: 10.5935/abc.20130065
AUTHORS	Novo Rosangela, Freire Cristiana M, Felisbino Sergio, Minicucci Marcos F, Azevedo Paula S, Zornoff Leonardo A M, Paiva Sergio A R
BACKGROUND	In a previous study utilizing the rat model, exposure to tobacco smoke for 5 weeks increased survival after AMI, despite similar age and infarct size between the smokers and nonsmokers, and absence of reperfusion.
OBJECTIVE	Thus, this study aimed to analyze the effects of exposure to tobacco smoke on intensity, distribution or phosphorylation of connexin 43 in the rat heart.
METHODS	Wistar rats weighing 100 g were randomly allocated into 2 groups: 1) CONTROL (n = 25); 2) Exposed to tobacco smoke (ETS), n = 23. After 5 weeks, left ventricular morphometric analysis, immunohistochemistry and western blotting for connexin 43 (Cx43) were performed.
RESULTS	Collagen volume fraction, cross-sectional areas, and ventricular weight were not statistically different between control and ETS. ETS showed lower stain intensity of Cx43 at intercalated disks (
CONTROL	2.32 ± 0.19; ETS: 1.73 ± 0.18; p = 0.04). The distribution of CX43 at intercalated disks did not differ between the groups (
CONTROL	3.73 ± 0.12; ETS: 3.20 ± 0.17; p = 0.18). ETS rats showed higher levels of dephosphorylated form of Cx43 (CONTROL: 0.45 ± 0.11; ETS: 0.90 ± 0.11; p = 0.03). On the other hand, total Cx43 did not differ between control and ETS groups (
CONTROL	0.75 ± 0.19; ETS: 0.93 ± 0.27; p = 0.58).
CONCLUSION	Exposure to tobacco smoke resulted in cardiac gap junction remodeling, characterized by alterations in the quantity and phosphorylation of the Cx43, in rats hearts. This finding could explain the smoker's paradox observed in some studies.

표-183. PubMed 논문번호 23619984의 내용 요약

구분	내용
PubMed ID	23619984
TITLE	Chronic exposure to biomass fuel is associated with increased carotid artery intima-media thickness and a higher prevalence of atherosclerotic plaque.
JOURNAL	Heart (British Cardiac Society): 10.1136/heartjnl-2012-303440
AUTHORS	Painschab Matthew S, Davila-Roman Victor G, Gilman Robert H, Vasquez-Villar Angel D, Pollard Suzanne L, Wise Robert A, Miranda J Jaime, Checkley William
BACKGROUND	Biomass fuels are used for cooking in the majority of rural households worldwide. While their use is associated with an increased risk of lung diseases and all-cause mortality, the effects on cardiovascular disease (CVD) are not well characterised. Exposure to biomass fuel smoke has been associated with lung-mediated inflammation and oxidative stress, which may increase the risk of atherosclerosis as evaluated by carotid intima-media thickness (CIMT), carotid atherosclerotic plaque prevalence and blood pressure.
METHODS	A cross-sectional study was performed in 266 adults aged ≥35 years in Puno, Peru (3825 m above sea level). We stratified participants by their long-term history of exposure to clean fuel (n=112) or biomass fuel (n=154) and measured 24 h indoor particulate matter (PM2.5) in a random subset (n=84). Participants completed questionnaires and underwent a clinical assessment, laboratory analyses and carotid artery ultrasound. The main outcome measures were CIMT, carotid plaque and blood pressure.
RESULTS	The groups were similar in age and gender. The biomass fuel group had greater unadjusted mean CIMT (0.66 vs 0.60 mm; p<0.001), carotid plaque prevalence (26% vs 14%; p=0.03), systolic blood pressure (118 vs 111 mm Hg; p<0.001) and median household PM2.5 (280 vs 14 □g/m(3); p<0.001). In multivariable regression, the biomass fuel group had greater mean CIMT (mean difference=0.03 mm, 95% CI 0.01 to 0.06; p=0.02), a higher prevalence of carotid plaques (OR=2.6, 95% CI 1.1 to 6.0; p=0.03) and higher systolic blood pressure (mean difference=9.2 mm Hg, 95% CI 5.4 to 13.0; p<0.001).
CONCLUSIONS	Chronic exposure to biomass fuel was associated with increased CIMT, increased prevalence of atherosclerotic plaques and higher blood pressure. These findings identify biomass fuel use as a risk factor for CVD, which may have important global health implications.

표-184. PubMed 논문번호 23633198의 내용 요약

구분	내용
PubMed ID	23633198
TITLE	Gender difference in the relationship between passive smoking exposure and HDL-cholesterol levels in late adolescence.
JOURNAL	The Journal of clinical endocrinology and metabolism: 10.1210/jc.2013-1016
AUTHORS	Le-Ha Chi, Beilin Lawrence J, Burrows Sally, Huang Rae-Chi, Oddy Wendy H, Hands Beth, Mori Trevor A
BACKGROUND	High-density lipoprotein-cholesterol (HDL-C) levels are influenced by gender and by genetic and environmental factors. We aimed to assess the impact of passive smoking exposure since birth on HDL-C levels of nonsmoking adolescents at age 17 years and to determine whether there was a gender difference in the relationship between smoking exposure and HDL-C.
METHODS	A total of 804 nonsmoking adolescents with biochemical, anthropometric, and lifestyle data from a cohort of 1754 adolescents (mean age, 17 ± 0.25 y) of the Western Australian Pregnancy Cohort (Raine) Study had data of maternal smoking during pregnancy and smoking exposure in the household over 17 years. HDL-C was analyzed using multivariable linear regression, with adjustment for early-life, adiposity, and current lifestyle confounders.
RESULTS	HDL-C levels were significantly lower in girls exposed to passive smoking compared to those not exposed (regression coefficient b = −0.09 [95% confidence interval, −0.15, −0.03]); this was not observed in boys (b = 0.02 [95% confidence interval, −0.04, 0.08]), with a significant sex interaction P = .009. The effects of passive smoking in girls persisted after adjusting for oral contraceptive use.
CONCLUSIONS	This study has shown a gender difference in the relationship between passive smoking exposure since birth and HDL-C in late adolescence. Exposure to passive smoking in girls could have adverse consequences on their risk of cardiovascular disease in adulthood. These findings reinforce the need for future public health measures to reduce children's exposure to passive smoking.

표-185. PubMed 논문번호 23637576의 내용 요약

구분	내용
PubMed ID	23637576
TITLE	Fine particulate air pollution and the progression of carotid intima-medial thickness: a prospective cohort study from the multi-ethnic study of atherosclerosis and air pollution.
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1001430
AUTHORS	Adar Sara D, Sheppard Lianne, Vedal Sverre, Polak Joseph F, Sampson Paul D, Diez Roux Ana V, Budoff Matthew, Jacobs David R, Barr R Graham, Watson Karol, Kaufman Joel D
BACKGROUND	Fine particulate matter (PM2.5) has been linked to cardiovascular disease, possibly via accelerated atherosclerosis. We examined associations between the progression of the intima-medial thickness (IMT) of the common carotid artery, as an indicator of atherosclerosis, and long-term PM2.5 concentrations in participants from the Multi-Ethnic Study of Atherosclerosis (MESA).
METHODS AND RESULTS	MESA, a prospective cohort study, enrolled 6,814 participants at the baseline exam (2000-2002), with 5,660 (83%) of those participants completing two ultrasound examinations between 2000 and 2005 (mean follow-up: 2.5 years). PM2.5 was estimated over the year preceding baseline and between ultrasounds using a spatio-temporal model. Cross-sectional and longitudinal associations were examined using mixed models adjusted for confounders including age, sex, race/ethnicity, smoking, and socio-economic indicators. Among 5,362 participants (5% of participants had missing data) with a mean annual progression of 14 □m/y, 2.5 □g/m(3) higher levels of residential PM2.5 during the follow-up period were associated with 5.0 □m/y (95% CI 2.6 to 7.4 □m/y) greater IMT progressions among persons in the same metropolitan area. Although significant associations were not found with IMT progression without adjustment for metropolitan area (0.4 □m/y [95% CI -0.4 to 1.2 □m/y] per 2.5 □g/m(3)), all of the six areas showed positive associations. Greater reductions in PM2.5 over follow-up for a fixed baseline PM2.5 were also associated with slowed IMT progression (-2.8 □m/y [95% CI -1.6 to -3.9 □m/y] per 1 □g/m(3) reduction). Study limitations include the use of a surrogate measure of atherosclerosis, some loss to follow-up, and the lack of estimates for air pollution concentrations prior to 1999.
CONCLUSIONS	This early analysis from MESA suggests that higher long-term PM2.5 concentrations are associated with increased IMT progression and that greater reductions in PM2.5 are related to slower IMT progression. These findings, even over a relatively short follow-up period, add to the limited literature on air pollution and the progression of atherosclerotic processes in humans. If confirmed by future analyses of the full 10 years of follow-up in this cohort, these findings will help to explain associations between long-term PM2.5 concentrations and clinical cardiovascular events.

표-186. PubMed 논문번호 23641873의 내용 요약

구분	내용
PubMed ID	23641873
TITLE	Particulate matter components and subclinical atherosclerosis: common approaches to estimating exposure in a Multi-Ethnic Study of Atherosclerosis cross-sectional study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-12-39
AUTHORS	Sun Min, Kaufman Joel D, Kim Sun-Young, Larson Timothy V, Gould Timothy R, Polak Joseph F, Budoff Matthew J, Diez Roux Ana V, Vedal Sverre
BACKGROUND	Concentrations of outdoor fine particulate matter (PM2.5) have been associated with cardiovascular disease. PM2.5 chemical composition may be responsible for effects of exposure to PM2.5.
METHODS	Using data from the Multi-Ethnic Study of Atherosclerosis (MESA) collected in 2000-2002 on 6,256 US adults without clinical cardiovascular disease in six U.S. metropolitan areas, we investigated cross-sectional associations of estimated long-term exposure to total PM2.5 mass and PM2.5 components (elemental carbon [EC], organic carbon [OC], silicon and sulfur) with measures of subclinical atherosclerosis (coronary artery calcium [CAC] and right common carotid intima-media thickness [CIMT]). Community monitors deployed for this study from 2007 to 2008 were used to estimate exposures at baseline addresses using three commonly-used approaches: (1) nearest monitor (the primary approach), (2) inverse-distance monitor weighting and (3) city-wide average.
RESULTS	Using the exposure estimate based on nearest monitor, in single-pollutant models, increased OC (effect estimate [95% CI] per IQR: 35.1 μm [26.8, 43.3]), EC (9.6 μm [3.6,15.7]), sulfur (22.7 μm [15.0,30.4]) and total PM2.5 (14.7 μm [9.0,20.5]) but not silicon (5.2 μm [-9.8,20.1]), were associated with increased CIMT; in two-pollutant models, only the association with OC was robust to control for the other pollutants. Findings were generally consistent across the three exposure estimation approaches. None of the PM measures were positively associated with either the presence or extent of CAC. In sensitivity analyses, effect estimates for OC and silicon were particularly sensitive to control for metropolitan area.
CONCLUSION	Employing commonly-used exposure estimation approaches, all of the PM2.5 components considered, except silicon, were associated with increased CIMT, with the evidence being strongest for OC; no component was associated with increased CAC. PM2.5 chemical components, or other features of the sources that produced them, may be important in determining the effect of PM exposure on atherosclerosis. These cross-sectional findings await confirmation in future work employing longitudinal outcome measures and using more sophisticated approaches to estimating exposure.

표-187. PubMed 논문번호 23669275의 내용 요약

구분	내용
PubMed ID	23669275
TITLE	Residential proximity to major roadways and renal function.
JOURNAL	Journal of epidemiology and community health: 10.1136/jech-2012-202307
AUTHORS	Lue Shih-Ho, Wellenius Gregory A, Wilker Elissa H, Mostofsky Elizabeth, Mittleman Murray A
BACKGROUND	Living near major roadways has been associated with increased risk of cardiovascular events, but little is known about its impact on renal function.
METHODS	We calculated the estimated glomerular filtration rate (eGFR) for 1103 consecutive Boston-area patients hospitalised with confirmed acute ischaemic stroke between 1999 and 2004. We used linear regression to evaluate the association between eGFR and categories of residential distance to major roadway (0 to ≤50, >50 to ≤100, >100 to ≤200, >200 to ≤400, >400 to ≤1000 and >1000 m) adjusting for age, sex, race, smoking, comorbid conditions, treatment with ACE inhibitor and neighbourhood-level socioeconomic characteristics. In a second analysis, we considered the log of distance to major roadway as a continuous variable.
RESULTS	Patients living closer to a major roadway had lower eGFR than patients living farther away (Ptrend=0.01). Comparing patients living 50 m versus 1000 m from a major roadway was associated with a 3.9 ml/min/1.73 m(2) lower eGFR (95% CI 1.0 to 6.7; p=0.007): a difference comparable in magnitude to the reduction in eGFR observed for a 4-year increase in age in population-based studies. The magnitude of this association did not differ significantly across categories of age, sex, race, history of hypertension, diabetes or socioeconomic status.
CONCLUSIONS	Living near a major roadway is associated with lower eGFR in a cohort of patients presenting with acute ischaemic stroke. If causal, these results imply that exposures associated with living near a major roadway contribute to reduced renal function, an important risk factor for cardiovascular events.

표-188. PubMed 논문번호 23709640의 내용 요약

구분	내용
PubMed ID	23709640
TITLE	Ambient fine particulate matter alters cerebral hemodynamics in the elderly.
JOURNAL	Stroke: 10.1161/STROKEAHA.111.000395
AUTHORS	Wellenius Gregory A, Boyle Luke D, Wilker Elissa H, Sorond Farzaneh A, Coull Brent A, Koutrakis Petros, Mittleman Murray A, Lipsitz Lewis A
BACKGROUND AND PURPOSE	Short-term elevations in fine particulate matter air pollution (PM2.5) are associated with increased risk of acute cerebrovascular events. Evidence from the peripheral circulation suggests that vascular dysfunction may be a central mechanism. However, the effects of PM2.5 on cerebrovascular function and hemodynamics are unknown.
METHODS	We used transcranial Doppler ultrasound to measure beat-to-beat blood flow velocity in the middle cerebral artery at rest and in response to changes in end-tidal CO2 (cerebral vasoreactivity) and arterial blood pressure (cerebral autoregulation) in 482 participants from the Maintenance of Balance, Independent Living, Intellect, and Zest in the Elderly (MOBILIZE) of Boston study. We used linear mixed effects models with random subject intercepts to evaluate the association between cerebrovascular hemodynamic parameters and mean PM2.5 levels 1 to 28 days earlier adjusting for age, race, medical history, meteorologic covariates, day of week, temporal trends, and season.
RESULTS	An interquartile range increase (3.0 □g/m(3)) in mean PM2.5 levels during the previous 28 days was associated with an 8.6% (95% confidence interval, 3.7%-13.8%; P<0.001) higher cerebral vascular resistance and a 7.5% (95% confidence interval, 4.2%-10.6%; P<0.001) lower blood flow velocity at rest. Measures of cerebral vasoreactivity and autoregulation were not associated with PM2.5 levels.
CONCLUSIONS	In this cohort of community-dwelling seniors, exposure to PM2.5 was associated with higher resting cerebrovascular resistance and lower cerebral blood flow velocity. If replicated, these findings suggest that alterations in cerebrovascular hemodynamics may underlie the increased risk of particle-related acute cerebrovascular events.

표-189. PubMed 논문번호 23717615의 내용 요약

구분	내용
PubMed ID	23717615
TITLE	Mitochondrial genetic background modifies the relationship between traffic-related air pollution exposure and systemic biomarkers of inflammation.
JOURNAL	PloS one: 10.1371/journal.pone.0064444
AUTHORS	Wittkopp Sharine, Staimer Norbert, Tjoa Thomas, Gillen Daniel, Daher Nancy, Shafer Martin, Schauer James J, Sioutas Constantinos, Delfino Ralph J
BACKGROUND	Mitochondria are the main source of reactive oxygen species (ROS). Human mitochondrial haplogroups are linked to differences in ROS production and oxidative-stress induced inflammation that may influence disease pathogenesis, including coronary artery disease (CAD). We previously showed that traffic-related air pollutants were associated with biomarkers of systemic inflammation in a cohort panel of subjects with CAD in the Los Angeles air basin.
OBJECTIVE	We tested whether air pollutant exposure-associated inflammation was stronger in mitochondrial haplogroup H than U (high versus low ROS production) in this panel (38 subjects and 417 observations).
METHODS	Inflammation biomarkers were measured weekly in each subject (≤ 12 weeks), including interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), C-reactive protein, interleukin-6 soluble receptor and tumor necrosis factor-soluble receptor II. We determined haplogroup by restriction fragment length polymorphism analysis. Air pollutants included nitrogen oxides (NOx), carbon monoxide (CO), organic carbon, elemental and black carbon (EC, BC); and particulate matter mass, three size fractions (<0.25 □m, 0.25-2.5 □m, and 2.5-10 □m in aerodynamic diameter). Particulate matter extracts were analyzed for organic compounds, including polycyclic aromatic hydrocarbons (PAH), and in vitro oxidative potential of aqueous extracts. Associations between exposures and biomarkers, stratified by haplogroup, were analyzed by mixed-effects models.
RESULTS	IL-6 and TNF-α were associated with traffic-related air pollutants (BC, CO, NOx and PAH), and with mass and oxidative potential of quasi-ultrafine particles <0.25 □m. These associations were stronger for haplogroup H than haplogroup U.
CONCLUSIONS	Results suggest that mitochondrial haplogroup U is a novel protective factor for air pollution-related systemic inflammation in this small group of subjects.

표-190. PubMed 논문번호 23721370의 내용 요약

구분	내용
PubMed ID	23721370
TITLE	The effects of smoke-free legislation on acute myocardial infarction: a systematic review and meta-analysis.
JOURNAL	BMC public health: 10.1186/1471-2458-13-529
AUTHORS	Lin Hualiang, Wang Hongchun, Wu Wei, Lang Lingling, Wang Qinzhou, Tian Linwei
BACKGROUND	Comprehensive smoke-free legislation has been implemented in many countries. The current study quantitatively examined the reduction in risk of acute myocardial infarction (MI) occurrence following the legislations and the relationship with the corresponding smoking prevalence decrease.
METHODS	PubMed, EMBASE, and Google Scholar databases and bibliographies of relevant studies and reviews were searched for potential original studies published from January 1, 2004, through October 31, 2011. Meta-analysis was performed using a random effect model to estimate the overall effects of the smoking-free legislations. Meta-regression was used to investigate possible causes of heterogeneity in risk estimates.
RESULTS	A total of 18 eligible studies with 44 estimates of effect size were used in this study. Meta-analysis produced a pooled estimate of the relative risk of 0.87 (95% confidence interval (CI): 0.84 to 0.91). There was significant heterogeneity in the risk estimates (overall I^2 = 96.03%, $p<0.001$). In meta-regression analysis, studies with greater smoking prevalence decrease produced larger relative risk (adjusted coefficient -0.027, 95% CI: -0.049 to -0.006, p=0.014).
CONCLUSION	Smoke-free legislations in public and work places were associated with significant reduction in acute MI risk, which might be partly attributable to reduced smoking prevalence.

표-191. PubMed 논문번호 23721619의 내용 요약

구분	내용
PubMed ID	23721619
TITLE	Residential proximity to high-traffic roadways and poststroke mortality.
JOURNAL	Journal of stroke and cerebrovascular diseases : the official journal of National Stroke Association: 10.1016/j.jstrokecerebrovasdis.2013.03.034
AUTHORS	Wilker Elissa H, Mostofsky Elizabeth, Lue Shih-Ho, Gold Diane, Schwartz Joel, Wellenius Gregory A, Mittleman Murray A
BACKGROUND	Living in areas with higher levels of ambient air pollution has been associated with a higher incidence of ischemic stroke and all-cause mortality but less is known about the relationship between traffic-related pollution and long-term survival after stroke.
METHODS	We identified consecutive patients admitted to Beth Israel Deaconess Medical Center with ischemic stroke between 1999 and 2008 and determined the distance to the nearest roadway with an average daily traffic count of more than 10,000 vehicles/day. Categories of residential proximity were defined as 100 m or less, 100-200 m, 200-400 m or less, or more than 400 m from a busy roadway. We identified deaths through June 2012 using the Social Security Death Index and used Cox proportional hazards models adjusted for medical history and socioeconomic factors to calculate hazard ratios for the association between residential proximity to a high-traffic roadway and all-cause mortality.
RESULTS	Among 1683 stroke patients with complete data, there were 950 deaths (median follow-up=4.6 years). We observed higher poststroke mortality among people living closer to high-traffic roadways. Patients living 100 m or less from high-traffic roadways had a 20% (95% confidence interval: 1%, 43%) higher rate of poststroke mortality than patients living more than 400 m away (P trend=.02).
CONCLUSIONS	In this study, living close to a high-traffic roadway was associated with an elevated mortality rate. This relationship remained statistically significant after adjustment for individual- and neighborhood-level factors, providing evidence that traffic-related pollution is associated with a higher mortality rate among stroke survivors.

표-192. PubMed 논문번호 23742058의 내용 요약

구분	내용
PubMed ID	23742058
TITLE	Exposure to wood smoke increases arterial stiffness and decreases heart rate variability in humans.
JOURNAL	Particle and fibre toxicology: 10.1186/1743-8977-10-20
AUTHORS	Unosson Jon, Blomberg Anders, Sandstr□m Thomas, Muala Ala, Boman Christoffer, Nystr□m Robin, Westerholm Roger, Mills Nicholas L, Newby David E, Langrish Jeremy P, Bosson Jenny A
BACKGROUND	Emissions from biomass combustion are a major source of indoor and outdoor air pollution, and are estimated to cause millions of premature deaths worldwide annually. Whilst adverse respiratory health effects of biomass exposure are well established, less is known about its effects on the cardiovascular system. In this study we assessed the effect of exposure to wood smoke on heart rate, blood pressure, central arterial stiffness and heart rate variability in otherwise healthy persons.
METHODS	Fourteen healthy non-smoking subjects participated in a randomized, double-blind crossover study. Subjects were exposed to dilute wood smoke (mean particle concentration of 314±38 μg/m3) or filtered air for three hours during intermittent exercise. Heart rate, blood pressure, central arterial stiffness and heart rate variability were measured at baseline and for one hour post-exposure.
RESULTS	Central arterial stiffness, measured as augmentation index, augmentation pressure and pulse wave velocity, was higher after wood smoke exposure as compared to filtered air ($p < 0.01$ for all), and heart rate was increased ($p < 0.01$) although there was no effect on blood pressure. Heart rate variability (SDNN, RMSSD and pNN50; $p = 0.003$, $p < 0.001$ and $p < 0.001$ respectively) was decreased one hour following exposure to wood smoke compared to filtered air.
CONCLUSIONS	Acute exposure to wood smoke as a model of exposure to biomass combustion is associated with an immediate increase in central arterial stiffness and a simultaneous reduction in heart rate variability. As biomass is used for cooking and heating by a large fraction of the global population and is currently advocated as a sustainable alternative energy source, further studies are required to establish its likely impact on cardiovascular disease.
TRIAL REGISTRATION	ClinicalTrials.gov, NCT01488500.

표-193. PubMed 논문번호 23757428의 내용 요약

구분	내용
PubMed ID	23757428
TITLE	Childhood and adult secondhand smoke and type 2 diabetes in women.
JOURNAL	Diabetes care: 10.2337/dc12-2173
AUTHORS	Lajous Martin, Tondeur Laura, Fagherazzi Guy, de Lauzon-Guillain Blandine, Boutron-Ruaualt Marie-Christine, Clavel-Chapelon Fran□oise
OBJECTIVE	The objective of this study was to evaluate the relationship between childhood and adult secondhand smoke and type 2 diabetes.
RESEARCH DESIGN AND METHODS	We conducted a prospective cohort study among 37,343 French women from the E3N-EPIC (Etude Epid□miologique aupr□s des femmes de la Mutuelle G□n□rale de l'Education Nationale-European Prospective Investigation into Cancer and Nutrition) who never smoked and who were free of type 2 diabetes, cancer, or cardiovascular disease at baseline in 1992. Self-reported childhood secondhand smoke exposure was defined as having at least one parent who smoked. Adult secondhand smoke was defined as the sum of self-reported hours recorded at baseline of exposure to tobacco smoke from a spouse who smoked (or domestic close contact) and from outside the home.
RESULTS	Between 1992 and 2007, 795 cases of incident type 2 diabetes were identified and validated through a drug reimbursement dataset and a specific questionnaire. Women with at least one parent who smoked appeared to have an 18% higher rate of type 2 diabetes than women with parents who did not smoke (age-adjusted hazard ratio 1.18 [95% CI 1.02-1.36]). Adult secondhand smoke exposure (no exposure versus ≥ 4 h/day) was associated with an increased rate of type 2 diabetes (1.36 [1.05-1.77], P = 0.002 for trend) after adjusting for parental history of diabetes, education, body silhouette at age 8, childhood secondhand smoke exposure, physical activity, body mass index, hypertension, hypercholesterolemia, menopausal status and hormone use, alcohol intake, and processed red meat and coffee consumption.
CONCLUSIONS	This prospective analysis suggests that secondhand smoke exposure in childhood and adulthood are associated with a higher rate of type 2 diabetes.

표-194. PubMed 논문번호 23770178의 내용 요약

구분	내용
PubMed ID	23770178
TITLE	Acute exposure to air pollution triggers atrial fibrillation.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2013.05.043
AUTHORS	Link Mark S, Luttmann-Gibson Heike, Schwartz Joel, Mittleman Murray A, Wessler Benjamin, Gold Diane R, Dockery Douglas W, Laden Francine
OBJECTIVES	This study sought to evaluate the association of air pollution with the onset of atrial fibrillation (AF).
BACKGROUND	Air pollution in general and more specifically particulate matter has been associated with cardiovascular events. Although ventricular arrhythmias are traditionally thought to convey the increased cardiovascular risk, AF may also contribute.
METHODS	Patients with dual chamber implantable cardioverter-defibrillators (ICDs) were enrolled and followed prospectively. The association of AF onset with air quality including ambient particulate matter <2.5 μm aerodynamic diameter (PM2.5), black carbon, sulfate, particle number, NO2, SO2, and O3 in the 24 h prior to the arrhythmia was examined utilizing a case-crossover analysis. In sensitivity analyses, associations with air pollution between 2 and 48 h prior to the AF were examined.
RESULTS	Of 176 patients followed for an average of 1.9 years, 49 patients had 328 episodes of AF lasting ≥ 30 s. Positive but nonsignificant associations were found for PM2.5 in the prior 24 h, but stronger associations were found with shorter exposure windows. The odds of AF increased by 26% (95% confidence interval: 8% to 47%) for each 6.0 μg/m(3) increase in PM2.5 in the 2 h prior to the event (p = 0.004). The odds of AF were highest at the upper quartile of mean PM2.5.
CONCLUSIONS	PM was associated with increased odds of AF onset within hours following exposure in patients with known cardiac disease. Air pollution is an acute trigger of AF, likely contributing to the pollution-associated adverse cardiac outcomes observed in epidemiological studies.

표-195. PubMed 논문번호 23777671의 내용 요약

구분	내용
PubMed ID	23777671
TITLE	Cumulative exposure to particulate matter air pollution and long-term post-myocardial infarction outcomes.
JOURNAL	Preventive medicine: 10.1016/j.ypmed.2013.06.009
AUTHORS	Koton Silvia, Molshatzki Noa, Myers Vicki, Broday David M, Drory Yaacov, Steinberg David M, Gerber Yariv
INTRODUCTION	Chronic environmental exposure to particulate matter <2.5 μm in diameter (PM2.5) has been associated with cardiovascular disease; however, the effect of air pollution on myocardial infarction (MI) survivors is not clear. We studied the association of chronic exposure to PM2.5 with death and recurrent cardiovascular events in MI survivors.
METHODS	Consecutive patients aged ≤65years admitted to all medical centers in central Israel after first-MI in 1992-1993 were followed through 2005 for cardiovascular events and 2011 for survival. Data on sociodemographic and prognostic factors were collected at baseline and during follow-up. Residential exposure to PM2.5 was estimated for each patient based on data recorded at air quality monitoring stations. Cox and Andersen-Gill proportional hazards models were used to study the pollution-outcome association.
RESULTS	Among the 1120 patients, 469 (41.9%) died and 541 (48.3%) experienced one or more recurrent cardiovascular event. The adjusted hazard ratios associated with a 10 μg/m(3) increase in PM2.5 exposure were 1.3 (95% CI 0.8-2.1) for death and 1.5 (95% CI 1.1-1.9) for multiple recurrences of cardiovascular events (MI, heart failure and stroke).
CONCLUSION	When adjustment for socio-demographic factors is performed, cumulative chronic exposure to PM2.5 is positively associated with recurrence of cardiovascular events in patients after a first MI.

표-196. PubMed 논문번호 23777832의 내용 요약

구분	내용
PubMed ID	23777832
TITLE	Short-term associations between fine and coarse particulate matter and hospitalizations in Southern Europe: results from the MED-PARTICLES project.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1206151
AUTHORS	Stafoggia Massimo, Samoli Evangelia, Alessandrini Ester, Cadum Ennio, Ostro Bart, Berti Giovanna, Faustini Annunziata, Jacquemin Benedicte, Linares Cristina, Pascal Mathilde, Randi Giorgia, Ranzi Andrea, Stivanello Elisa, Forastiere Francesco
BACKGROUND	Evidence on the short-term effects of fine and coarse particles on morbidity in Europe is scarce and inconsistent.
OBJECTIVES	We aimed to estimate the association between daily concentrations of fine and coarse particles with hospitalizations for cardiovascular and respiratory conditions in eight Southern European cities, within the MED-PARTICLES project.
METHODS	City-specific Poisson models were fitted to estimate associations of daily concentrations of particulate matter with aerodynamic diameter ≤ 2.5 μm (PM2.5), ≤ 10 μm (PM10), and their difference (PM2.5-10) with daily counts of emergency hospitalizations for cardiovascular and respiratory diseases. We derived pooled estimates from random-effects meta-analysis and evaluated the robustness of results to co-pollutant exposure adjustment and model specification. Pooled concentration-response curves were estimated using a meta-smoothing approach.
RESULTS	We found significant associations between all PM fractions and cardiovascular admissions. Increases of 10 μg/m3 in PM2.5, 6.3 μg/m3 in PM2.5-10, and 14.4 μg/m3 in PM10 (lag 0-1 days) were associated with increases in cardiovascular admissions of 0.51% (95% CI: 0.12, 0.90%), 0.46% (95% CI: 0.10, 0.82%), and 0.53% (95% CI: 0.06, 1.00%), respectively. Stronger associations were estimated for respiratory hospitalizations, ranging from 1.15% (95% CI: 0.21, 2.11%) for PM10 to 1.36% (95% CI: 0.23, 2.49) for PM2.5 (lag 0-5 days).
CONCLUSIONS	PM2.5 and PM2.5-10 were positively associated with cardiovascular and respiratory admissions in eight Mediterranean cities. Information on the short-term effects of different PM fractions on morbidity in Southern Europe will be useful to inform European policies on air quality standards.

표-197. PubMed 논문번호 23782920의 내용 요약

구분	내용
PubMed ID	23782920
TITLE	DNA hypomethylation, ambient particulate matter, and increased blood pressure: findings from controlled human exposure experiments.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.113.000212
AUTHORS	Bellavia Andrea, Urch Bruce, Speck Mary, Brook Robert D, Scott Jeremy A, Albetti Benedetta, Behbod Behrooz, North Michelle, Valeri Linda, Bertazzi Pier Alberto, Silverman Frances, Gold Diane, Baccarelli Andrea A
BACKGROUND	Short-term exposures to fine (<2.5 μm aerodynamic diameter) ambient particulate-matter (PM) have been related with increased blood pressure (BP) in controlled-human exposure and community-based studies. However, whether coarse (2.5 to 10 μm) PM exposure increases BP is uncertain. Recent observational studies have linked PM exposures with blood DNA hypomethylation, an epigenetic alteration that activates inflammatory and vascular responses. No experimental evidence is available to confirm those observational data and demonstrate the relations between PM, hypomethylation, and BP.
METHODS AND RESULTS	We conducted a cross-over trial of controlled-human exposure to concentrated ambient particles (CAPs). Fifteen healthy adult participants were exposed for 130 minutes to fine CAPs, coarse CAPs, or HEPA-filtered medical air (control) in randomized order with ≥2-week washout. Repetitive-element (Alu, long interspersed nuclear element-1 [LINE-1]) and candidate-gene (TLR4, IL-12, IL-6, iNOS) blood methylation, systolic and diastolic BP were measured pre- and postexposure. After adjustment for multiple comparisons, fine CAPs exposure lowered Alu methylation (β-standardized=-0.74, adjusted-P=0.03); coarse CAPs exposure lowered TLR4 methylation (β-standardized=-0.27, adjusted-P=0.04). Both fine and coarse CAPs determined significantly increased systolic BP (β=2.53 mm Hg, P=0.001; β=1.56 mm Hg, P=0.03, respectively) and nonsignificantly increased diastolic BP (β=0.98 mm Hg, P=0.12; β=0.82 mm Hg, P=0.11, respectively). Decreased Alu and TLR4 methylation was associated with higher postexposure DBP (β-standardized=0.41, P=0.04; and β-standardized=0.84, P=0.02; respectively). Decreased TLR4 methylation was associated with higher postexposure SBP (β-standardized=1.45, P=0.01).
CONCLUSIONS	Our findings provide novel evidence of effects of coarse PM on BP and confirm effects of fine PM. Our results provide the first experimental evidence of PM-induced DNA hypomethylation and its correlation to BP.

표-198. PubMed 논문번호 23790344의 내용 요약

구분	내용
PubMed ID	23790344
TITLE	Exposure to particulate air pollution and long-term incidence of frailty after myocardial infarction.
JOURNAL	Annals of epidemiology: 10.1016/j.annepidem.2013.05.001
AUTHORS	Myers Vicki, Broday David M, Steinberg David M, Drory Yaacov, Gerber Yariv
PURPOSE	Frailty, a multidimensional syndrome of increased vulnerability, is prevalent post-myocardial infarction (MI) and predicts mortality and recurrent events. We investigated whether chronic exposure to particulate matter ≤2.5 μm in diameter (PM2.5) is associated with the development of post-MI frailty.
METHODS	Participants (n = 1120) were aged 65 or less and admitted to hospital in central Israel with first MI in 1992 and 1993. Daily measures of PM2.5 recorded at air quality monitoring stations were summarized and chronic exposure was estimated individually using the geo-coded residential location. Frailty assessment was conducted via an index based on deficit accumulation, and those defined as frail (applying a threshold of ≥0.25) at baseline were excluded. Remaining participants who survived to follow-up 10 to 13 years post-MI (n = 848) were reassessed for frailty. Logistic regression models were constructed to evaluate the role of PM2.5 exposure in frailty risk prediction.
RESULTS	Mean exposure to PM2.5 was 24.2 μg/m(3) (range, 16.9-28.6). A total of 301 participants (35.5%) developed frailty during follow-up. Adjusting for sociodemographic and clinical variables, PM2.5 exposure was associated with increased odds of developing frailty (odds ratio, 1.53; 95% confidence interval, 1.22-1.91, comparing the 75th vs. 25th percentiles). Addition of PM2.5 exposure to the multivariable model resulted in an integrated discrimination improvement of 1.60% (P = .005) and a net reclassification index of 6.51% (P = .02).
CONCLUSIONS	An association was observed between exposure to PM2.5 and incidence of frailty, providing a potential intermediary between air pollution and post-MI outcomes.

표-199. PubMed 논문번호 23838152의 내용 요약

구분	내용
PubMed ID	23838152
TITLE	Particulate air pollution, ambulatory heart rate variability, and cardiac arrhythmia in retirement community residents with coronary artery disease.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1205914
AUTHORS	Bartell Scott M, Longhurst John, Tjoa Thomas, Sioutas Constantinos, Delfino Ralph J
BACKGROUND	Decreased heart rate variability (HRV) has been associated with future cardiac morbidity and mortality and is often used as a marker of altered cardiac autonomic balance in studies of health effects of airborne particulate matter. Fewer studies have evaluated associations between air pollutants and cardiac arrhythmia.
OBJECTIVES	We examined relationships between cardiac arrhythmias, HRV, and exposures to airborne particulate matter.
METHODS	We measured HRV and arrhythmia with ambulatory electrocardiograms in a cohort panel study for up to 235 hr per participant among 50 nonsmokers with coronary artery disease who were ≥ 71 years of age and living in four retirement communities in the Los Angeles, California, Air Basin. Exposures included hourly outdoor gases, hourly traffic-related and secondary organic aerosol markers, and daily size-fractionated particle mass. We used repeated measures analyses, adjusting for actigraph-derived physical activity and heart rate, temperature, day of week, season, and community location.
RESULTS	Ventricular tachycardia was significantly increased in association with increases in markers of traffic-related particles, secondary organic carbon, and ozone. Few consistent associations were observed for supraventricular tachycardia. Particulates were significantly associated with decreased ambulatory HRV only in the 20 participants using ACE (angiotensin I-converting enzyme) inhibitors.
CONCLUSIONS	Although these data support the hypothesis that particulate exposures may increase the risk of ventricular tachycardia for elderly people with coronary artery disease, HRV was not associated with exposure in most of our participants. These results are consistent with previous findings in this cohort for systemic inflammation, blood pressure, and ST segment depression.

표-200. PubMed 논문번호 23842036의 내용 요약

구분	내용
PubMed ID	23842036
TITLE	Exposure to parental smoking and child growth and development: a cohort study.
JOURNAL	BMC pediatrics: 10.1186/1471-2431-13-104
AUTHORS	Yang Seungmi, Decker Adriana, Kramer Michael S
BACKGROUND	Studies on adverse childhood health and development outcomes associated with parental smoking have shown inconsistent results. Using a cohort of Belarusian children, we examined differences in cognition, behaviors, growth, adiposity, and blood pressure at 6.5 years according to prenatal and postnatal exposure to parental smoking.
METHODS	Using cluster-adjusted multivariable regression, effects of exposure to prenatal smoking were examined by comparing (1) children whose mothers smoked during pregnancy with those of mothers who smoked neither during nor after pregnancy and (2) children whose mothers smoked during and after pregnancy with those whose mothers smoked after pregnancy only; effects of postnatal smoking were examined by comparing (1) children whose mothers smoked after pregnancy only with those of mothers who smoked neither during nor after pregnancy and (2) children whose fathers smoked with those whose fathers did not smoke among children of non-smoking mothers after adjusting for a wide range of socioeconomic and family characteristics.
RESULTS	After adjusting for confounders, children exposed vs unexposed to prenatal maternal smoking had no differences in mean IQ, teacher-rated behavioral problems, adiposity, or blood pressure. Children exposed to maternal postnatal smoking had slightly increased behavioral problems [0.9, 95% CI: 0.6, 1.2 for total difficulties], higher body mass index [0.2, 95% CI: 0.1, 0.3], greater total skinfold thickness [0.4, 95% CI: 0.04, 0.71], and higher odds of overweight or obesity [1.4, 95% CI; 1.1, 1.7]. Similar magnitudes of association were observed with postnatal paternal smoking.
CONCLUSIONS	No adverse cognitive, behavioral and developmental outcomes were associated with exposure to maternal prenatal smoking. Observed associations with postnatal smoking of both parents may reflect residual confounding by genetic and family environmental factors.

표-201. PubMed 논문번호 23849322의 내용 요약

구분	내용
PubMed ID	23849322
TITLE	Global association of air pollution and heart failure: a systematic review and meta-analysis.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(13)60898-3
AUTHORS	Shah Anoop S V, Langrish Jeremy P, Nair Harish, McAllister David A, Hunter Amanda L, Donaldson Ken, Newby David E, Mills Nicholas L
BACKGROUND	Acute exposure to air pollution has been linked to myocardial infarction, but its effect on heart failure is uncertain. We did a systematic review and meta-analysis to assess the association between air pollution and acute decompensated heart failure including hospitalisation and heart failure mortality.
METHODS	Five databases were searched for studies investigating the association between daily increases in gaseous (carbon monoxide, sulphur dioxide, nitrogen dioxide, ozone) and particulate (diameter <2·5 μm [PM2·5] or <10 μm [PM10]) air pollutants, and heart failure hospitalisations or heart failure mortality. We used a random-effects model to derive overall risk estimates per pollutant.
FINDINGS	Of 1146 identified articles, 195 were reviewed in-depth with 35 satisfying inclusion criteria. Heart failure hospitalisation or death was associated with increases in carbon monoxide (3·52% per 1 part per million; 95% CI 2·52-4·54), sulphur dioxide (2·36% per 10 parts per billion; 1·35-3·38), and nitrogen dioxide (1·70% per 10 parts per billion; 1·25-2·16), but not ozone (0·46% per 10 parts per billion; -0·10 to 1·02) concentrations. Increases in particulate matter concentration were associated with heart failure hospitalisation or death (PM2·5 2·12% per 10 μg/m(3), 95% CI 1·42-2·82; PM10 1·63% per 10 μg/m(3), 95% CI 1·20-2·07). Strongest associations were seen on the day of exposure, with more persistent effects for PM2·5. In the USA, we estimate that a mean reduction in PM2·5 of 3·9 μg/m(3) would prevent 7978 heart failure hospitalisations and save a third of a billion US dollars a year.
INTERPRETATION	Air pollution has a close temporal association with heart failure hospitalisation and heart failure mortality. Although more studies from developing nations are required, air pollution is a pervasive public health issue with major cardiovascular and health economic consequences, and it should remain a key target for global health policy.
FUNDING	British Heart Foundation.

표-202. PubMed 논문번호 23874781의 내용 요약

구분	내용
PubMed ID	23874781
TITLE	Venous thromboembolism in an industrial north american city: temporal distribution and association with particulate matter air pollution.
JOURNAL	PloS one: 10.1371/journal.pone.0068829
AUTHORS	Chiu Holly H, Whittaker Peter
BACKGROUND	Emerging evidence, mainly from Europe and Asia, indicates that venous thromboembolism (VTE) occurs most often in winter. Factors implicated in such seasonality are low temperature-mediated exacerbation of coagulation and high levels of particulate matter (PM) air pollution. However, in contrast to most European and Asian cities, particulate matter pollution peaks in the summer in many North American cities.
OBJECTIVES	We aimed to exploit this geographical difference and examine the temporal distribution of VTE in a cold-weather, North American city, Detroit, with a summer PM peak. Our goal was thereby to resolve the influence of temperature and PM levels on VTE.
METHODS	Our retrospective, analytical semi-ecological study used chart review to confirm 1,907 acute, ambulatory VTE cases, divided them by location (Detroit versus suburban), and plotted monthly VTE frequency distributions. We used Environmental Protection Agency data to determine the temporal distribution of PM pollution components in Detroit. Suburban PM air pollution is presumed negligible and therefore not monitored.
RESULTS	Acute VTE cases in Detroit (1,490) exhibited a summer peak (June 24(th)) and differed from both a uniform distribution (P<0.01) and also that of 1,123 no-VTE cases (P<0.02). Levels of 10 □m diameter PM and coarse particle (2.5 to 10 □m) PM also exhibited summer peaks versus a winter peak for 2.5 □m diameter PM. Contrary to their urban counterparts, suburban cases of acute VTE (417) showed no monthly variation.
CONCLUSIONS	The summer peak of acute VTE in Detroit indicates that low temperature is not a major factor in VTE pathogenesis. In contrast, concordance of the 10 □m diameter PM, coarse particle, and the Detroit VTE monthly distributions, combined with no monthly suburban VTE variation, is consistent with a role for PM pollution. Furthermore, divergence of the VTE and 2.5 □m PM distributions suggests that particle size may play a role.

표-203. PubMed 논문번호 23877047의 내용 요약

구분	내용
PubMed ID	23877047
TITLE	Changes in traffic exposure and the risk of incident myocardial infarction and all-cause mortality.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e31829d5dae
AUTHORS	Hart Jaime E, Rimm Eric B, Rexrode Kathryn M, Laden Francine
BACKGROUND	Traffic-related exposures, such as air pollution and noise, have been associated with increased cardiovascular morbidity and mortality. Few studies, however, have been able to examine the effects of changes in exposure on changes in risk. Our objective was to explore the associations of changes in traffic exposure with changes in risk between 1990 and 2008 in the Nurses' Health Study.
METHODS	Incident myocardial infarction (MI) and all-cause mortality were prospectively identified. As a proxy for traffic exposure, we calculated residential distance to roads at all residential addresses 1986-2006 and considered addresses to be "close" or "far" based on distance and road type. To examine the effect of changes in exposure, each consecutive pair of addresses was categorized as: (1) consistently close, (2) consistently far, (3) change from close to far, and (4) change from far to close. We also examined the change in NO2 levels between address pairs.
RESULTS	In time-varying Cox proportional hazards models adjusted for a variety of risk factors, women living at residences consistently close to traffic were at a higher risk of an incident MI (hazard ratio [HR] = 1.11; 95% confidence interval [CI] = 1.01-1.22) and a higher risk of all-cause mortality (1.05; 1.00-1.10), compared with those consistently far. The highest risks were seen among women who moved from being far from traffic to close (incident MI: HR = 1.50 [95% CI = 1.11-2.03]; all-cause mortality: HR = 1.17 [95% CI = 1.00-1.37]). Each 1 ppb increase in NO2 compared with the previous address was associated with a HR = 1.22 for incident MI (95% CI = 0.99-1.50) and 1.03 for all-cause mortality (95% CI = 0.92-1.15).
CONCLUSIONS	Our results suggest that changes in traffic exposure (measured as roadway proximity or change in NO2 levels) are associated with changes in risk of MI and all-cause mortality.

표-204. PubMed 논문번호 23887610의 내용 요약

구분	내용
PubMed ID	23887610
TITLE	Cardiovascular health, traffic-related air pollution and noise: are associations mutually confounded? A systematic review.
JOURNAL	International journal of public health: 10.1007/s00038-013-0489-7
AUTHORS	T□treault Louis-Fran□ois, Perron St□phane, Smargiassi Audrey
OBJECTIVES	This review assessed the confounding effect of one traffic-related exposure (noise or air pollutants) on the association between the other exposure and cardiovascular outcomes.
METHODS	A systematic review was conducted with the databases Medline and Embase. The confounding effects in studies were assessed by using change in the estimate with a 10 % cutoff point. The influence on the change in the estimate of the quality of the studies, the exposure assessment methods and the correlation between road noise and air pollutions were also assessed.
RESULTS	Nine publications were identified. For most studies, the specified confounders produced changes in estimates <10 %. The correlation between noise and pollutants, the quality of the study and of the exposure assessment do not seem to influence the confounding effects.
CONCLUSIONS	Results from this review suggest that confounding of cardiovascular effects by noise or air pollutants is low, though with further improvements in exposure assessment, the situation may change. More studies using pollution indicators specific to road traffic are needed to properly assess if noise and air pollution are subjected to confounding.

표-205. PubMed 논문번호 23892931의 내용 요약

구분	내용
PubMed ID	23892931
TITLE	Systematic review of the effects of black carbon on cardiovascular disease among individuals with pre-existing disease.
JOURNAL	International journal of public health: 10.1007/s00038-013-0492-z
AUTHORS	Nichols Jennifer L, Owens Elizabeth Oesterling, Dutton Steven J, Luben Thomas J
OBJECTIVES	Recent interest has developed in understanding the health effects attributable to different components of particulate matter. This review evaluates the effects of black carbon (BC) on cardiovascular disease in individuals with pre-existing disease using evidence from epidemiologic and experimental studies.
METHODS	A systematic literature search was conducted to identify epidemiologic and experimental studies examining the relationship between BC and cardiovascular health effects in humans with pre-existing diseases. Nineteen epidemiologic and six experimental studies were included. Risk of bias was evaluated for each study.
RESULTS	Evidence across studies suggested ambient BC is associated with changes in subclinical cardiovascular health effects in individuals with diabetes and coronary artery disease (CAD). Limited evidence demonstrated that chronic respiratory disease does not modify the effect of BC on cardiovascular health.
CONCLUSIONS	Results in these studies consistently demonstrated that diabetes is a risk factor for BC-related cardiovascular effects, including increased interleukin-6 and ECG parameters. Cardiovascular effects were associated with BC in individuals with CAD, but few comparisons to individuals without CAD were provided in the literature.

표-206. PubMed 논문번호 23911139의 내용 요약

구분	내용
PubMed ID	23911139
TITLE	Exposure to air pollution near a steel plant and effects on cardiovascular physiology: a randomized crossover study.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2013.06.007
AUTHORS	Liu Ling, Kauri Lisa Marie, Mahmud Mamun, Weichenthal Scott, Cakmak Sabit, Shutt Robin, You Hongyu, Thomson Errol, Vincent Renaud, Kumarathasan Premkumari, Broad Gayle, Dales Robert, Liu Ling, Kauri Lisa Marie, Mahmud Mamun, Weichenthal Scott, Cakmak Sabit, Shutt Robin, You Hongyu, Thomson Errol, Vincent Renaud, Kumarathasan Premkumari, Broad Gayle, Dales Robert
BACKGROUND	Iron and steel industry is an important source of air pollution emissions. Few studies have investigated cardiovascular effects of air pollutants emitted from steel plants.
OBJECTIVE	We examined the influence of outdoor air pollution in the vicinity of a steel plant on cardiovascular physiology in Sault Ste. Marie, Canada.
METHODS	Sixty-one healthy, non-smoking subjects (females/males=33/28, median age 22 years) spent 5 consecutive 8-hour days outdoors in a residential area neighbouring a steel plant, or on a college campus approximately 5 kilometres away from the plant, and then crossed over to the other site with a 9-day washout. Mid day, subjects underwent daily 30-minute moderate intensity exercise. Blood pressure (BP) and pulse rate were determined daily and post exercise at both sites. Flow-mediated vasodilation (FMD) was determined at the site near the plant. Air pollution was monitored at both sites. Mixed-effects regressions were run for statistical associations, adjusting for weather variables.
RESULTS	Concentrations of ultrafine particles, sulphur dioxide (SO2), nitrogen dioxide (NO2) and carbon monoxide (CO) were 50-100% higher at the site near the plant than at the college site, with minor differences in temperature, humidity, and concentrations of particulate matter ≤2.5 μm in size (PM2.5) and ozone (O3). Resting pulse rate [mean (95% confidence interval)] was moderately higher near the steel plant [+1.53 bpm (0.31, 2.78)] than at the college site, male subjects having the highest pulse rate elevation [+2.77 bpm (0.78, 4.76)]. Resting systolic and diastolic BP and pulse pressure, and post-exercise BP and pulse rate were not significantly different between two sites. Interquartile range concentrations of SO2 (2.9 ppb), NO2 (5.0 ppb) and CO (0.2 ppm) were associated with increased pulse rate [0.19 bpm (-0.00, 0.38), 0.86 bpm (0.03, 1.68), and 0.11 bpm (0.00, 0.22), respectively], ultrafine particles (10,256 count/cm(3)) associated with increased pulse pressure [0.85 mmHg (0.23, 1.48)], and NO2 and CO inversely associated with FMD [-0.14% (-0.31, 0.02), -0.02% (-0.03, -0.00), respectively]. SO2 during exercise was associated with increased pulse rate [0.26 bpm (0.01, 0.51)].
CONCLUSION	Air quality in residential areas near steel plants may influence cardiovascular physiology.

표-207. PubMed 논문번호 23911218의 내용 요약

구분	내용
PubMed ID	23911218
TITLE	Dietary fiber intake modifies the association between secondhand smoke exposure and coronary heart disease mortality among Chinese non-smokers in Singapore.
JOURNAL	Nutrition (Burbank, Los Angeles County, Calif.): 10.1016/j.nut.2013.04.003
AUTHORS	Clark Maggie L, Butler Lesley M, Koh Woon-Puay, Wang Renwei, Yuan Jian-Min
OBJECTIVE	Secondhand smoke (SHS) exposure increases the risk for coronary heart disease (CHD) by an estimated 25% to 30% via oxidative stress and inflammatory mechanisms that may be ameliorated by dietary components. The aim of this study was to evaluate the hypothesized modifying role of nutrients with known antioxidant and/or anti-inflammatory properties on the relationship between SHS exposure and CHD mortality.
METHODS	Detailed SHS exposure and dietary information was collected among 29,579 non-smokers in the Singapore Chinese Health Study, a prospective population-based cohort. The evaluation of whether or not dietary factors (β-cryptoxanthin, lutein, ω-3 polyunsaturated fatty acids, fiber, isothiocyanates, and soy isoflavones) modified the relationship between SHS exposure and CHD mortality was conducted within multivariable Cox proportional hazards models by creating an interaction term between the potential dietary effect modifier (lowest quartile of intake versus the second through fourth quartiles of intake) and the SHS exposure (none versus living with at least one smoker[s]).
RESULTS	Evidence for a main-effects association between SHS exposure and risk for CHD mortality was not observed. In stratified analyses by levels of selected dietary nutrient intake, fiber modified the effects of SHS exposure on risk for CHD mortality (P for interaction = 0.02). The adjusted hazards ratio for SHS exposure (living with at least one smoker[s] versus living with no smokers) and CHD mortality was 1.62 (95% confidence interval, 1.00-2.63) for those with low-fiber intake. In contrast, among those with high-fiber intake, there was no association with SHS exposure.
CONCLUSION	We provide evidence that a diet high in fiber may ameliorate the harmful effects of SHS exposure on risk for CHD mortality.

표-208. PubMed 논문번호 23918257의 내용 요약

구분	내용
PubMed ID	23918257
TITLE	Changes in ambulance calls after implementation of a smoke-free law and its extension to casinos.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.113.003455
AUTHORS	Glantz Stanton A, Gibbs Erin
BACKGROUND	Casinos are often exempted from legislation mandating smoke-free environments, potentially putting employees and patrons at risk for adverse events triggered by secondhand smoke exposure.
METHODS AND RESULTS	We used an interrupted time series analysis of ambulance calls not originating and originating from casinos in Gilpin County, Colorado, a rural Colorado county with a large casino presence, from January 2000 through December 2012 to determine whether there was a change in ambulance calls originating from casinos when a state smoke-free law was extended to include them. Initial implementation of the smoke-free law (which exempted casinos) was followed by a significant 22.8% drop in ambulance calls (incidence rate ratio, 0.772; 95% confidence interval, 0.685-0.871; P<0.001) from locations other than casinos but no significant change in calls from casinos (P>0.9). The law requiring smoke-free casinos taking effect was followed by a 19.1% (incidence rate ratio, 0.809; 95% confidence interval, 0.724-0.905; P<0.001) drop in ambulance calls from casinos but no change in calls originating outside casinos (P>0.1).
CONCLUSIONS	The observation that ambulance calls not coming from casinos dropped when the smoke-free law was initially implemented (excluding casinos) with no change in calls from casinos, followed by a comparable drop in calls originating from casinos (but not calls from elsewhere) when the law was extended to casinos, suggests that the important effects of secondhand smoke exposure occur acutely. These results also suggest that exempting casinos from smoke-free laws means that more people will suffer medical emergencies.

표-209. PubMed 논문번호 23919441의 내용 요약

구분	내용
PubMed ID	23919441
TITLE	The effect of acute exposure to coarse particulate matter air pollution in a rural location on circulating endothelial progenitor cells: results from a randomized controlled study.
JOURNAL	Inhalation toxicology: 10.3109/08958378.2013.814733
AUTHORS	Brook Robert D, Bard Robert L, Kaplan Mariana J, Yalavarthi Srilakshmi, Morishita Masako, Dvonch J Timothy, Wang Lu, Yang Hui-Yu, Spino Catherine, Mukherjee Bhramar, Oral Elif A, Sun Qinghua, Brook Jeffrey R, Harkema Jack, Rajagopalan Sanjay
CONTEXT	Fine particulate matter (PM) air pollution has been associated with alterations in circulating endothelial progenitor cell (EPC) levels, which may be one mechanism whereby exposures promote cardiovascular diseases. However, the impact of coarse PM on EPCs is unknown.
OBJECTIVE	We aimed to determine the effect of acute exposure to coarse concentrated ambient particles (CAP) on circulating EPC levels.
METHODS	Thirty-two adults (25.9 ± 6.6 years) were exposed to coarse CAP (76.2 ± 51.5 μg m(-3)) in a rural location and filtered air (FA) for 2 h in a randomized double-blind crossover study. Peripheral venous blood was collected 2 and 20 h post-exposures for circulating EPC (n = 21), white blood cell (n = 24) and vascular endothelial growth factor (VEGF) (n = 16-19) levels. The changes between exposures were compared by matched Wilcoxon signed-rank tests.
RESULTS	Circulating EPC levels were elevated 2 [108.29 (6.24-249.71) EPC mL(-1); median (25th-75th percentiles), p = 0.052] and 20 h [106.86 (52.91-278.35) EPC mL(-1), p = 0.008] post-CAP exposure compared to the same time points following FA [38.47 (0.00-84.83) and 50.16 (0.00-104.79) EPC mL(-1)]. VEGF and white blood cell (WBC) levels did not differ between exposures.
CONCLUSIONS	Brief inhalation of coarse PM from a rural location elicited an increase in EPCs that persisted for at least 20 h. The underlying mechanism responsible may reflect a systemic reaction to an acute "endothelial injury" and/or a circulating EPC response to sympathetic nervous system activation.

표-210. PubMed 논문번호 23928921의 내용 요약

구분	내용
PubMed ID	23928921
TITLE	When to prevent cardiovascular disease? As early as possible: lessons from prospective cohorts beginning in childhood.
JOURNAL	Current opinion in cardiology: 10.1097/HCO.0b013e32836428f4
AUTHORS	Magnussen Costan G, Smith Kylie J, Juonala Markus
PURPOSE OF REVIEW	To detail recent developments linking modifiable youth risk factors with preclinical markers of cardiovascular disease such as carotid artery intima-media thickness, pulse-wave velocity (PVW) and large artery stiffness, brachial artery flow-mediated dilatation, left ventricular geometry, and coronary artery calcification in adulthood.
RECENT FINDINGS	Population-based data from prospective cohort studies beginning in youth with follow-up into adulthood have shown that the modifiable youth risk factors of elevated blood lipids, blood pressure, and adiposity, smoking (active and passive), metabolic disorders, physical inactivity, low cardiorespiratory fitness, and diet associate with preclinical markers of cardiovascular disease in adulthood. The data suggest that, in some instances, those who amend their trajectory by not maintaining these risk factors into adulthood experience reductions in preclinical markers to levels associated with never having had the risk factor.
SUMMARY	Though avoidance of risk factors in youth is ideal, there is still a window for intervention where long-lasting cardiovascular effects might be avoided. Health-enhancing changes in the rates of active and passive smoking, adiposity, increased physical activity, accentuated fitness, modified diet, and socioeconomic position in the transition from youth to adulthood might be important in modifying an individual's trajectory from high risk in youth to low risk in adulthood.

표-211. PubMed 논문번호 23940250의 내용 요약

구분	내용
PubMed ID	23940250
TITLE	Modelling the effects of low indoor temperatures on the lung function of children with asthma.
JOURNAL	Journal of epidemiology and community health: 10.1136/jech-2013-202632
AUTHORS	Pierse Nevil, Arnold Richard, Keall Michael, Howden-Chapman Philippa, Crane Julian, Cunningham Malcolm
INTRODUCTION	While many epidemiological studies have shown that low outdoor temperatures are associated with increased rates of hospitalisation and mortality (especially for respiratory or cardiovascular disease), very few studies have looked at the association between indoor temperatures and health. Such studies are clearly warranted, as people have greater exposure to the indoor environment than the outdoor environment.
OBJECTIVES	To examine the relationship between various metrics of indoor temperature and lung function in children with asthma. Our specific research questions were: (1) In which room of the home is temperature most strongly associated with lung function? (2) Which exposure metric best describes the relationship between indoor temperature and lung function? (3) Over what lag/time period does indoor air temperature affect lung function most strongly?
METHODS	The Heating Housing and Health Study was a randomised controlled trial that investigated the effect of installing heaters in the homes of children with asthma. This study collected measurements of lung function (daily) and indoor temperature (hourly). Lung function and indoor temperature were measured for 309 children over 12 049 child-days. Statistical models were fitted to identify the best measures and metrics.
RESULTS	The strongest association with lung function was found for the severity of exposure to low bedroom temperatures averaged over the preceding periods of 0-7 to 0-12 days. A 1° C increase in temperature was associated with an average increase of 0.010, 0.008, 10.06, 12.06, in our four measures of lung function (peak expiratory flow rate (PEFR) morning, PEFR evening, forced expiratory volume in 1 s (FEV1) morning and FEV1 evening).
CONCLUSIONS	Indoor temperatures have a small, but significant, association with short-term variations in the lung function of children with asthma.

표-212. PubMed 논문번호 24011457의 내용 요약

구분	내용
PubMed ID	24011457
TITLE	Ozone short-term exposure and acute coronary events: a multicities study in Tuscany (Italy).
JOURNAL	Environmental research: 10.1016/j.envres.2013.08.002
AUTHORS	Nuvolone Daniela, Balzi Daniela, Pepe Pasquale, Chini Marco, Scala Danila, Giovannini Franco, Cipriani Francesco, Barchielli Alessandro
OBJECTIVE	Many studies have investigated the potential role of ozone exposure in cardiovascular mortality and morbidity. The effects on specific cardiovascular outcome and the role of individual susceptibility are less studied. This paper focuses on the short-term effects of ozone on acute coronary events and it investigates comorbidities as indicators of personal susceptibility.
SETTING AND PATIENTS	This study was conducted in five urban areas of the Tuscany region (Italy) covering the period January 2002-December 2005. Air quality and meteorological data from urban background monitoring sites were collected. Hospital admissions for acute myocardial infarction and out-of-hospital coronary deaths were extracted from administrative database.
DESIGN	Both time series and case-crossover designs were applied. The confounding effects of some time-dependent variables, such as temperature, were taken into account. Some potential susceptibility factors were investigated. Pooled estimates were derived from random-effect meta-analysis.
RESULTS	During the warm season 4555 hospitalized acute myocardial infarctions and 1931 out-of-hospital coronary deaths occurred. Authors estimated a 6.3% (95% confidence interval, 1.2%, 11.7%) increase in out-of-hospital coronary deaths for a 10 μg/m3 increase in ozone (lag 0-5). Results also suggested higher risks for females, elderly, and patients previously hospitalized for cerebrovascular and artery diseases.
CONCLUSIONS	This study adds further evidence to the relation between cardiovascular diseases and ozone exposure, showing an adverse effect on out-of-hospital coronary deaths, but not on hospitalized acute myocardial infarctions. Some susceptible subgroups, such as females, elderly, and patients affected by some chronic diseases, are likely to be at major risk.

표-213. PubMed 논문번호 24025094의 내용 요약

구분	내용
PubMed ID	24025094
TITLE	Chronic exposure to emissions from photocopiers in copy shops causes oxidative stress and systematic inflammation among photocopier operators in India.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-12-78
AUTHORS	Elango Nithya, Kasi Vallikkannu, Vembhu Bhuvaneswari, Poornima Jeyanthi Govindasamy
BACKGROUND	We assessed indoor air quality in photocopier centers and investigated whether occupational exposure to emissions from photocopiers is associated with decline in lung function or changes in haematological parameters, oxidative stress and inflammatory status.
METHODS	Indoor air quality was monitored in five photocopier centers. Pulmonary function was assessed by spirometry in 81 photocopier operators (64 male and 17 female) and 43 healthy control (31 male and 12 female) subjects. Hematological status, serum thio-barbituric acid reactive substances (TBARS), total ferric reducing antioxidant capacity (FRAC), leukotriene B4 (LTB4), 8-isoprostane, C reactive protein (CRP), interleukin 8 (IL-8), clara cell protein (CC-16), intercellular adhesion molecule 1 (ICAM-1) and eosinophilic cationic protein (ECP) were analyzed. Relationships between cumulative exposure, lung function and inflammatory markers were assessed.
RESULTS	PM10 and PM2.5 were above the permissible levels in all the photocopier centers, whereas the levels of carbon monoxide, nitrogen oxides, ozone, sulphur dioxide, lead, arsenic, nickel, ammonia, benzene and benzo(a)pyrene were within Indian ambient air quality standards. Lung function was similar in the photocopier operators and control subjects. Serum TBARS was significantly higher and FRAC was lower among photocopier operators when compared to healthy controls. Plasma IL-8, LTB4, ICAM-1 and ECP were significantly higher in the photocopier exposed group.
CONCLUSIONS	Photocopiers emit high levels of particulate matter. Long term exposure to emissions from photocopiers was not associated with decreased lung function, but resulted in high oxidative stress and systemic inflammation leading to high risk of cardiovascular diseases.

표-214. PubMed 논문번호 24038014의 내용 요약

구분	내용
PubMed ID	24038014
TITLE	Hypothesis: cadmium explains, in part, why smoking increases the risk of cardiovascular disease.
JOURNAL	Journal of cardiovascular pharmacology and therapeutics: 10.1177/1074248413494815
AUTHORS	Hecht Eric M, Landy David C, Ahn Soyeon, Hlaing WayWay M, Hennekens Charles H
INTRODUCTION	Cigarette smoking is a major risk factor for cardiovascular disease via acute and chronic mechanisms, some of which remain unclear. One plausible but untested hypothesis concerns cadmium (Cd), a component of cigarette smoke, which is injurious to vascular endothelial cells and is independently associated with cardiovascular disease. To contribute to the formulation of this hypothesis, we performed a meta-analysis of the available data that consisted of cross-sectional studies useful to formulate but not test hypotheses.
METHODS	PubMed and Google Scholar were searched by combining the terms smoking, Cd, correlation, blood, human, and tobacco. Following abstract review, 10 cross-sectional studies were identified. We compared serum Cd levels between smokers and nonsmokers using standardized mean differences (SMDs) as well as correlation coefficients between smoking and Cd.
RESULTS	The estimated overall random effects SMD in Cd between smokers and nonsmokers was 1.13 (95% confidence interval [CI], .70-1.56) with significant heterogeneity (Q = 8.6, P < .001). The estimated overall random effects correlation coefficient between smoking and Cd was .54 (95% CI, .30-.72) with significant heterogeneity (Q = 71.3, P < .01).
CONCLUSIONS	Despite major inherent limitations of meta-analyses of cross-sectional studies, we believe that the data contribute to the formulation of the hypothesis that Cd explains, in part, why smokers have an increased risk of cardiovascular disease. Further research, including analytic studies designed a priori are necessary to test the hypothesis.

표-215. PubMed 논문번호 24047569의 내용 요약

구분	내용
PubMed ID	24047569
TITLE	Traffic-related air pollution and noise and children's blood pressure: results from the PIAMA birth cohort study.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487313505821
AUTHORS	Bilenko Natalya, van Rossem Lenie, Brunekreef Bert, Beelen Rob, Eeftens Marloes, Hoek Gerard, Houthuijs Danny, de Jongste Johan C, van Kempen Elise, Koppelman Gerard H, Meliefste Kees, Oldenwening Marieke, Smit Henriette A, Wijga Alet H, Gehring Ulrike, Bilenko Natalya, van Rossem Lenie, Brunekreef Bert, Beelen Rob, Eeftens Marloes, Hoek Gerard, Houthuijs Danny, de Jongste Johan C, van Kempen Elise, Koppelman Gerard H, Meliefste Kees, Oldenwening Marieke, Smit Henriette A, Wijga Alet H, Gehring Ulrike
AIMS	Elevation of a child's blood pressure may cause possible health risks in later life. There is evidence for adverse effects of exposure to air pollution and noise on blood pressure in adults. Little is known about these associations in children. We investigated the associations of air pollution and noise exposure with blood pressure in 12-year-olds.
METHODS	Blood pressure was measured at age 12 years in 1432 participants of the PIAMA birth cohort study. Annual average exposure to traffic-related air pollution [NO2, mass concentrations of particulate matter with diameters of less than 2.5 □m (PM2.5) and less than 10 □m (PM10), and PM2.5 absorbance] at the participants' home and school addresses at the time of blood pressure measurements was estimated by land-use regression models. Air pollution exposure on the days preceding blood pressure measurements was estimated from routine air monitoring data. Long-term noise exposure was assessed by linking addresses to modelled equivalent road traffic noise levels. Associations of exposures with blood pressure were analysed by linear regression. Effects are presented for an interquartile range increase in exposure.
RESULTS	Long-term exposure to NO2 and PM2.5 absorbance were associated with increased diastolic blood pressure, in children who lived at the same address since birth [adjusted mean difference (95% confidence interval) [mmHg] 0.83 (0.06 to 1.61) and 0.75 (-0.08 to 1.58), respectively], but not with systolic blood pressure. We found no association of blood pressure with short-term air pollution or noise exposure.
CONCLUSIONS	Long-term exposure to traffic-related air pollution may increase diastolic blood pressure in children.

표-216. PubMed 논문번호 24075310의 내용 요약

구분	내용
PubMed ID	24075310
TITLE	Are day-to-day variations of airborne particles associated with emergency ambulance dispatches?
JOURNAL	International journal of occupational and environmental health: 10.1179/2049396713Y.0000000045
AUTHORS	Zauli Sajani Stefano, Alessandrini Ester, Marchesi Stefano, Lauriola Paolo
BACKGROUND	Much of the evidence on the health effects of airborne particles is based on mortality and hospital admissions, while the evidence from other morbidity indicators is still limited.
OBJECTIVE	To measure the relationship between particles with diameter below 10 microm (PM10) and emergency ambulance dispatches (EAD).
METHODS	Daily EAD for six towns of the Emilia-Romagna region (Italy) were obtained from a database collecting real-time data. Time series analyses were performed, and city-specific estimates were combined using meta-analytic techniques.
RESULTS	We found a significant percentage change of EAD associated with a 10 microg/m3 increase of PM10 for non-traumatic diseases (0.86%, 95% CI: 0.61,1.1%). A positive relationship was also found for cardiovascular and respiratory diseases without reaching statistical significance. The risks were much higher in the warm (April-September) than in the cold season (January-March and October-December).
CONCLUSIONS	Emergency ambulance dispatches provide useful insight into the health effects of air pollution and may be useful to establish surveillance systems.

표-217. PubMed 논문번호 24076625의 내용 요약

구분	내용
PubMed ID	24076625
TITLE	Air pollution and individual and neighborhood socioeconomic status: evidence from the Multi-Ethnic Study of Atherosclerosis (MESA).
JOURNAL	Environmental health perspectives: 10.1289/ehp.1206337
AUTHORS	Hajat Anjum, Diez-Roux Ana V, Adar Sara D, Auchincloss Amy H, Lovasi Gina S, O'Neill Marie S, Sheppard Lianne, Kaufman Joel D
BACKGROUND	Although research has shown that low socioeconomic status (SES) and minority communities have higher exposure to air pollution, few studies have simultaneously investigated the associations of individual and neighborhood SES with pollutants across multiple sites.
OBJECTIVES	We characterized the distribution of ambient air pollution by both individual and neighborhood SES using spatial regression methods.
METHODS	The study population comprised 6,140 participants from the Multi-Ethnic Study of Atherosclerosis (MESA). Year 2000 annual average ambient PM2.5 and NOx concentrations were calculated for each study participant's home address at baseline examination. We investigated individual and neighborhood (2000 U.S. Census tract level) SES measures corresponding to the domains of income, wealth, education, and occupation. We used a spatial intrinsic conditional autoregressive model for multivariable analysis and examined pooled and metropolitan area-specific models.
RESULTS	A 1-unit increase in the z-score for family income was associated with 0.03- μg/m3 lower PM2.5 (95% CI: -0.05, -0.01) and 0.93% lower NOx (95% CI: -1.33, -0.53) after adjustment for covariates. A 1-SD-unit increase in the neighborhood's percentage of persons with at least a high school degree was associated with 0.47- μg/m3 lower mean PM2.5 (95% CI: -0.55, -0.40) and 9.61% lower NOx (95% CI: -10.85, -8.37). Metropolitan area-specific results exhibited considerable heterogeneity. For example, in New York, high-SES neighborhoods were associated with higher concentrations of pollution.
CONCLUSIONS	We found statistically significant associations of SES measures with predicted air pollutant concentrations, demonstrating the importance of accounting for neighborhood- and individual-level SES in air pollution health effects research.

표-218. PubMed 논문번호 24083379의 내용 요약

구분	내용
PubMed ID	24083379
TITLE	Secondhand tobacco smoke exposure and heart rate variability and inflammation among non-smoking construction workers: a repeated measures study.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-12-83
AUTHORS	Zhang Jinming, Fang Shona C, Mittleman Murray A, Christiani David C, Cavallari Jennifer M
BACKGROUND	Although it has been well recognized that exposure to secondhand tobacco smoke (SHS) is associated with cardiovascular mortality, the mechanisms and time course by which SHS exposure may lead to cardiovascular effects are still being explored.
METHODS	Non-smoking workers were recruited from a local union and monitored inside a union hall while exposed to SHS over approximately 6 hours. Participants were fitted with a continuous electrocardiographic monitor upon enrollment which was removed at the end of a 24-hr monitoring period. A repeated measures study design was used where resting ECGs and blood samples were taken from individuals before SHS exposure (baseline), immediately following SHS exposure (post) and the morning following SHS exposure (next-morning).Inflammatory markers, including high sensitivity C-reactive protein (CRP) and white blood cell count (WBC) were analyzed. Heart rate variability (HRV) was analyzed from the ECG recordings in time (SDNN, rMSSD) and frequency (LF, HF) domain parameters over 5-minute periods. SHS exposure was quantified using a personal fine particulate matter (PM2.5) monitor.Linear mixed effects regression models were used to examine within-person changes in inflammatory and HRV parameters across the 3 time periods. Exposure-response relationships with PM2.5 were examined using mixed effects models. All models were adjusted for age, BMI and circadian variation.
RESULTS	A total of 32 male non-smokers were monitored between June 2010 and June 2012. The mean PM2.5 from SHS exposure was 132 μg/m3. Immediately following SHS exposure, a 100 μg/m3 increase in PM2.5 was associated with declines in HRV (7.8% [standard error (SE) =3%] SDNN, 8.0% (SE = 3.9%) rMSSD, 17.2% (SE = 6.3%) LF, 29.0% (SE = 10.1%) HF) and increases in WBC count 0.42 (SE = 0.14) k/μl. Eighteen hours following SHS exposure, a 100 μg/m3 increase in PM2.5 was associated with 24.2% higher CRP levels.
CONCLUSIONS	Our study suggest that short-term SHS exposure is associated with significantly lower HRV and higher levels of inflammatory markers. Exposure-associated declines in HRV were observed immediately following exposure while higher levels of CRP were not observed until 18 hours following exposure. Cardiovascular autonomic and inflammation responses may contribute to the pathophysiologic pathways that link SHS exposure with adverse cardiovascular outcomes.

표-219. PubMed 논문번호 24090339의 내용 요약

구분	내용
PubMed ID	24090339
TITLE	Highway proximity associated with cardiovascular disease risk: the influence of individual-level confounders and exposure misclassification.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-12-84
AUTHORS	Brugge Doug, Lane Kevin, Padr□-Mart□nez Luz T, Stewart Andrea, Hoesterey Kyle, Weiss David, Wang Ding Ding, Levy Jonathan I, Patton Allison P, Zamore Wig, Mwamburi Mkaya
BACKGROUND	Elevated cardiovascular disease risk has been reported with proximity to highways or busy roadways, but proximity measures can be challenging to interpret given potential confounders and exposure error.
METHODS	We conducted a cross sectional analysis of plasma levels of C-Reactive Protein (hsCRP), Interleukin-6 (IL-6), Tumor Necrosis Factor alpha receptor II (TNF-RII) and fibrinogen with distance of residence to a highway in and around Boston, Massachusetts. Distance was assigned using ortho-photo corrected parcel matching, as well as less precise approaches such as simple parcel matching and geocoding addresses to street networks. We used a combined random and convenience sample of 260 adults >40 years old. We screened a large number of individual-level variables including some infrequently collected for assessment of highway proximity, and included a subset in our final regression models. We monitored ultrafine particle (UFP) levels in the study areas to help interpret proximity measures.
RESULTS	Using the orthophoto corrected geocoding, in a fully adjusted model, hsCRP and IL-6 differed by distance category relative to urban background: 43% (-16%,141%) and 49% (6%,110%) increase for 0-50 m; 7% (-39%,45%) and 41% (6%,86%) for 50-150 m; 54% (-2%,142%) and 18% (-11%,57%) for 150-250 m, and 49% (-4%, 131%) and 42% (6%, 89%) for 250-450 m. There was little evidence for association for TNF-RII or fibrinogen. Ortho-photo corrected geocoding resulted in stronger associations than traditional methods which introduced differential misclassification. Restricted analysis found the effect of proximity on biomarkers was mostly downwind from the highway or upwind where there was considerable local street traffic, consistent with patterns of monitored UFP levels.
CONCLUSION	We found associations between highway proximity and both hsCRP and IL-6, with non-monotonic patterns explained partly by individual-level factors and differences between proximity and UFP concentrations. Our analyses emphasize the importance of controlling for the risk of differential exposure misclassification from geocoding error.

표-220. PubMed 논문번호 24096183의 내용 요약

구분	내용
PubMed ID	24096183
TITLE	Plasma cotinine indicates an increased risk of preeclampsia in previous and passive smokers.
JOURNAL	American journal of obstetrics and gynecology: 10.1016/j.ajog.2013.09.041
AUTHORS	Luo Zhong-Cheng, Julien Pierre, Wei Shu-Qin, Audibert Francois, Smith Graeme N, Fraser William D, Luo Zhong-Cheng, Julien Pierre, Wei Shu-Qin, Audibert Francois, Smith Graeme N, Fraser William D
OBJECTIVE	Self-reported tobacco smoking in pregnancy has been consistently associated with a decreased risk of developing preeclampsia, but the evidence has been limited and inconsistent for previous and passive smokers. Misclassifications and inaccuracies of self-reported tobacco exposure may disguise the true relationship. This study aimed to assess the association of gestational hypertension and preeclampsia with maternal smoking status as ascertained by plasma cotinine.
STUDY DESIGN	This was a prospective study of 605 pregnant women without chronic hypertension. Maternal smoking status at 24-26 weeks' gestation was defined by plasma cotinine: >3.0 ng/mL "current smokers," 0.20-3.00 ng/mL "previous and passive smokers," and <0.20 ng/mL "nonsmokers."
RESULTS	Compared to nonsmokers, the risk of developing preeclampsia did not change significantly for current smokers, but increased significantly (adjusted odds ratio, 6.06; 95% confidence interval, 2.32-15.85; P < .001) for previous and passive smokers. There were no significant differences in the risk of developing gestational hypertension only.
CONCLUSION	Previous and passive smoking may increase the risk of preeclampsia. Avoidance of exposure to environmental tobacco smoke in pregnancy may decrease the risk of preeclampsia.

표-221. PubMed 논문번호 24101168의 내용 요약

구분	내용
PubMed ID	24101168
TITLE	A systematic review of air pollution and incidence of out-of-hospital cardiac arrest.
JOURNAL	Journal of epidemiology and community health: 10.1136/jech-2013-203116
AUTHORS	Teng Tiew-Hwa Katherine, Williams Teresa A, Bremner Alexandra, Tohira Hideo, Franklin Peter, Tonkin Andrew, Jacobs Ian, Finn Judith, Teng Tiew-Hwa Katherine, Williams Teresa A, Bremner Alexandra, Tohira Hideo, Franklin Peter, Tonkin Andrew, Jacobs Ian, Finn Judith
INTRODUCTION	Studies have linked air pollution with the incidence of acute coronary artery events and cardiovascular mortality but the association with out-of-hospital cardiac arrest (OHCA) is less clear.
AIM	To examine the association of air pollution with the occurrence of OHCA.
METHODS	Electronic bibliographic databases (until February 2013) were searched. Search terms included common air pollutants and OHCA. Studies of patients with implantable cardioverter defibrillators and OHCA not attended by paramedics were excluded. Two independent reviewers (THKT and TAW) identified potential studies. Methodological quality was assessed by the Newcastle-Ottawa Scale.
RESULTS	Of 849 studies, 8 met the selection criteria. Significant associations between particulate matter (PM) exposure (especially PM(2.5)) and OHCA were found in 5 studies. An increase of OHCA risk ranged from 2.4% to 7% per interquartile increase in average PM exposure on the same day and up to 4 days prior to the event. A large study found ozone increased the risk of OHCA within 3 h prior to the event. The strongest risk OR of 3.8-4.6% per 20 parts per billion ozone increase of the average level was within 2 h prior to the event. Similarly, another study found an increased risk of 18% within 2 days prior to the event.
CONCLUSIONS	Larger studies have suggested an increased risk of OHCA with air pollution exposure from PM(2.5) and ozone.

표-222. PubMed 논문번호 24103537의 내용 요약

구분	내용
PubMed ID	24103537
TITLE	Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.f5432
AUTHORS	Hansell Anna L, Blangiardo Marta, Fortunato Lea, Floud Sarah, de Hoogh Kees, Fecht Daniela, Ghosh Rebecca E, Laszlo Helga E, Pearson Clare, Beale Linda, Beevers Sean, Gulliver John, Best Nicky, Richardson Sylvia, Elliott Paul
OBJECTIVE	To investigate the association of aircraft noise with risk of stroke, coronary heart disease, and cardiovascular disease in the general population.
DESIGN	Small area study.
SETTING	12 London boroughs and nine districts west of London exposed to aircraft noise related to Heathrow airport in London.
POPULATION	About 3.6 million residents living near Heathrow airport. Risks for hospital admissions were assessed in 12 110 census output areas (average population about 300 inhabitants) and risks for mortality in 2378 super output areas (about 1500 inhabitants).
MAIN OUTCOME MEASURES	Risk of hospital admissions for, and mortality from, stroke, coronary heart disease, and cardiovascular disease, 2001-05.
RESULTS	Hospital admissions showed statistically significant linear trends (P<0.001 to P<0.05) of increasing risk with higher levels of both daytime (average A weighted equivalent noise 7 am to 11 pm, L(Aeq),16 h) and night time (11 pm to 7 am, Lnight) aircraft noise. When areas experiencing the highest levels of daytime aircraft noise were compared with those experiencing the lowest levels (>63 dB v ≤ 51 dB), the relative risk of hospital admissions for stroke was 1.24 (95% confidence interval 1.08 to 1.43), for coronary heart disease was 1.21 (1.12 to 1.31), and for cardiovascular disease was 1.14 (1.08 to 1.20) adjusted for age, sex, ethnicity, deprivation, and a smoking proxy (lung cancer mortality) using a Poisson regression model including a random effect term to account for residual heterogeneity. Corresponding relative risks for mortality were of similar magnitude, although with wider confidence limits. Admissions for coronary heart disease and cardiovascular disease were particularly affected by adjustment for South Asian ethnicity, which needs to be considered in interpretation. All results were robust to adjustment for particulate matter (PM10) air pollution, and road traffic noise, possible for London boroughs (population about 2.6 million). We could not distinguish between the effects of daytime or night time noise as these measures were highly correlated.
CONCLUSION	High levels of aircraft noise were associated with increased risks of stroke, coronary heart disease, and cardiovascular disease for both hospital admissions and mortality in areas near Heathrow airport in London. As well as the possibility of causal associations, alternative explanations such as residual confounding and potential for ecological bias should be considered.

표-223. PubMed 논문번호 24103538의 내용 요약

구분	내용
PubMed ID	24103538
TITLE	Residential exposure to aircraft noise and hospital admissions for cardiovascular diseases: multi-airport retrospective study.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.f5561
AUTHORS	Correia Andrew W, Peters Junenette L, Levy Jonathan I, Melly Steven, Dominici Francesca
OBJECTIVE	To investigate whether exposure to aircraft noise increases the risk of hospitalization for cardiovascular diseases in older people (≥ 65 years) residing near airports.
DESIGN	Multi-airport retrospective study of approximately 6 million older people residing near airports in the United States. We superimposed contours of aircraft noise levels (in decibels, dB) for 89 airports for 2009 provided by the US Federal Aviation Administration on census block resolution population data to construct two exposure metrics applicable to zip code resolution health insurance data: population weighted noise within each zip code, and 90th centile of noise among populated census blocks within each zip code.
SETTING	2218 zip codes surrounding 89 airports in the contiguous states.
PARTICIPANTS	6 027 363 people eligible to participate in the national medical insurance (Medicare) program (aged ≥ 65 years) residing near airports in 2009.
MAIN OUTCOME MEASURES	Percentage increase in the hospitalization admission rate for cardiovascular disease associated with a 10 dB increase in aircraft noise, for each airport and on average across airports adjusted by individual level characteristics (age, sex, race), zip code level socioeconomic status and demographics, zip code level air pollution (fine particulate matter and ozone), and roadway density.
RESULTS	Averaged across all airports and using the 90th centile noise exposure metric, a zip code with 10 dB higher noise exposure had a 3.5% higher (95% confidence interval 0.2% to 7.0%) cardiovascular hospital admission rate, after controlling for covariates.
CONCLUSIONS	Despite limitations related to potential misclassification of exposure, we found a statistically significant association between exposure to aircraft noise and risk of hospitalization for cardiovascular diseases among older people living near airports.

표-224. PubMed 논문번호 24112419의 내용 요약

구분	내용
PubMed ID	24112419
TITLE	Feasibility intervention trial of two types of improved cookstoves in three resource-limited settings: study protocol for a randomized controlled trial.
JOURNAL	Trials: 10.1186/1745-6215-14-327
AUTHORS	Klasen Elizabeth, Miranda J Jaime, Khatry Subarna, Menya Diana, Gilman Robert H, Tielsch James M, Kennedy Caitlin, Dreibelbis Robert, Naithani Neha, Kimaiyo Sylvester, Chiang Marilu, Carter E Jane, Sherman Charles B, Breysse Patrick N, Checkley William
BACKGROUND	Exposure to biomass fuel smoke is one of the leading risk factors for disease burden worldwide. International campaigns are currently promoting the widespread adoption of improved cookstoves in resource-limited settings, yet little is known about the cultural and social barriers to successful improved cookstove adoption and how these barriers affect environmental exposures and health outcomes.
DESIGN	We plan to conduct a one-year crossover, feasibility intervention trial in three resource-limited settings (Kenya, Nepal and Peru). We will enroll 40 to 46 female primary cooks aged 20 to 49 years in each site (total 120 to 138).
METHODS	At baseline, we will collect information on sociodemographic characteristics and cooking practices, and measure respiratory health and blood pressure for all participating women. An initial observational period of four months while households use their traditional, open-fire design cookstoves will take place prior to randomization. All participants will then be randomized to receive one of two types of improved, ventilated cookstoves with a chimney: a commercially-constructed cookstove (Envirofit G3300/G3355) or a locally-constructed cookstove. After four months of observation, participants will crossover and receive the other improved cookstove design and be followed for another four months. During each of the three four-month study periods, we will collect monthly information on self-reported respiratory symptoms, cooking practices, compliance with cookstove use (intervention periods only), and measure peak expiratory flow, forced expiratory volume at 1 second, exhaled carbon monoxide and blood pressure. We will also measure pulmonary function testing in the women participants and 24-hour kitchen particulate matter and carbon monoxide levels at least once per period.
DISCUSSION	Findings from this study will help us better understand the behavioral, biological, and environmental changes that occur with a cookstove intervention. If this trial indicates that reducing indoor air pollution is feasible and effective in resource-limited settings like Peru, Kenya and Nepal, trials and programs to modify the open burning of biomass fuels by installation of low-cost ventilated cookstoves could significantly reduce the burden of illness and death worldwide.
TRIAL REGISTRATION	ClinicalTrials.gov NCT01686867.

표-225. PubMed 논문번호 24142983의 내용 요약

구분	내용
PubMed ID	24142983
TITLE	Long-term effects of aluminium dust inhalation.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2013-101487
AUTHORS	Peters Susan, Reid Alison, Fritschi Lin, de Klerk Nicholas, Musk A W Bill
OBJECTIVES	During the 1950s and 1960s, aluminium dust inhalation was used as a potential prophylaxis against silicosis in underground miners, including in Australia. We investigated the association between aluminium dust inhalation and cardiovascular, cerebrovascular and Alzheimer's diseases in a cohort of Australian male underground gold miners. We additionally looked at pneumoconiosis mortality to estimate the effect of the aluminium therapy.
METHODS	SMRs and 95% CI were calculated to compare mortality of the cohort members with that of the Western Australian male population (1961-2009). Internal comparisons on duration of aluminium dust inhalation were examined using Cox regression.
RESULTS	Aluminium dust inhalation was reported for 647 out of 1894 underground gold miners. During 42 780 person-years of follow-up, 1577 deaths were observed. An indication of increased mortality of Alzheimer's disease among miners ever exposed to aluminium dust was found (SMR=1.38), although it was not statistically significant (95% CI 0.69 to 2.75). Rates for cardiovascular and cerebrovascular death were above population levels, but were similar for subjects with or without a history of aluminium dust inhalation. HRs suggested an increasing risk of cardiovascular disease with duration of aluminium dust inhalation (HR=1.02, 95% CI 1.00 to 1.04, per year of exposure). No difference in the association between duration of work underground and pneumoconiosis was observed between the groups with or without aluminium dust exposure.
CONCLUSIONS	No protective effect against silicosis was observed from aluminium dust inhalation. Conversely, exposure to aluminium dust may possibly increase the risk of cardiovascular disease and dementia of the Alzheimer's type.

표-226. PubMed 논문번호 24142987의 내용 요약

구분	내용
PubMed ID	24142987
TITLE	Associations between arrhythmia episodes and temporally and spatially resolved black carbon and particulate matter in elderly patients.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2013-101526
AUTHORS	Zanobetti Antonella, Coull Brent A, Gryparis Alexandros, Kloog Itai, Sparrow David, Vokonas Pantel S, Wright Robert O, Gold Diane R, Schwartz Joel, Zanobetti Antonella, Coull Brent A, Gryparis Alexandros, Kloog Itai, Sparrow David, Vokonas Pantel S, Wright Robert O, Gold Diane R, Schwartz Joel
OBJECTIVES	Ambient air pollution has been associated with sudden deaths, some of which are likely due to ventricular arrhythmias. Defibrillator discharge studies have examined the association of air pollution with arrhythmias in sensitive populations. No studies have assessed this association using residence-specific estimates of air pollution exposure.
METHODS	In the Normative Aging Study, we investigated the association between temporally resolved and spatially resolved black carbon (BC) and PM2.5 and arrhythmia episodes (bigeminy, trigeminy or couplets episodes) measured as ventricular ectopy (VE) by 4 min ECG monitoring in repeated measures of 701 subjects, during the years 2000-2010. We used a binomial distribution (having or not a VE episode) in a mixed effect model with a random intercept for subject, controlling for seasonality, temperature, day of the week, medication use, smoking, having diabetes, body mass index and age. We also examined whether these associations were modified by genotype or phenotype.
RESULTS	We found significant increases in VE with both pollutants and lags; for the estimated concentration averaged over the 3 days prior to the health assessment, we found increases in the odds of having VE with an OR of 1.52 (95% CI 1.19 to 1.94) for an IQR (0.30 μg/m(3)) increase in BC and an OR of 1.39 (95% CI 1.12 to 1.71) for an IQR (5.63 μg/m(3)) increase in PM2.5. We also found higher effects in subjects with the glutathione S-transferase theta-1 and glutathione S-transferase mu-1 variants and in obese ($p<0.05$).
CONCLUSIONS	Increased levels of short-term traffic-related pollutants may increase the risk of ventricular arrhythmia in elderly subjects.

표-227. PubMed 논문번호 24143234의 내용 요약

구분	내용
PubMed ID	24143234
TITLE	Global metabolomic profiling reveals an association of metal fume exposure and plasma unsaturated fatty acids.
JOURNAL	PloS one: 10.1371/journal.pone.0077413
AUTHORS	Wei Yongyue, Wang Zhaoxi, Chang Chiung-yu, Fan Tianteng, Su Li, Chen Feng, Christiani David C
BACKGROUND	Welding-associated air pollutants negatively affect the health of exposed workers; however, their molecular mechanisms in causing disease remain largely unclear. Few studies have systematically investigated the systemic toxic effects of welding fumes on humans.
OBJECTIVES	To explore the effects of welding fumes on the plasma metabolome, and to identify biomarkers for risk assessment of welding fume exposure.
METHODS	The two-stage, self-controlled exploratory study included 11 boilermakers from a 2011 discovery panel and 8 boilermakers from a 2012 validation panel. Plasma samples were collected pre- and post-welding fume exposure and analyzed by chromatography/mass spectrometry.
RESULTS	Eicosapentaenoic or docosapentaenoic acid metabolic changes post-welding were significantly associated with particulate (PM2.5) exposure ($p<0.05$). The combined analysis by linear mixed-effects model showed that exposure was associated with a statistically significant decline in metabolite change of eicosapentaenoic acid [β (95% CI) = −0.013(−0.022 ≈ −0.004); p = 0.005], docosapentaenoic acid n3 [β (95% CI) = −0.010(−0.018 ≈ −0.002); p = 0.017], and docosapentaenoic acid n6 [β (95% CI) = −0.007(−0.013 ≈ −0.001); p = 0.021]. Pathway analysis identified an association of the unsaturated fatty acid pathway with exposure (p Study-2011 = 0.025; p Study-2012 = 0.021; p Combined = 0.009). The functional network built by these fatty acids and their interactive genes contained significant enrichment of genes associated with various diseases, including neoplasms, cardiovascular diseases, and lipid metabolism disorders.
CONCLUSIONS	High-dose exposure of metal welding fumes decreases unsaturated fatty acids with an exposure-response relationship. This alteration in fatty acids is a potential biological mediator and biomarker for exposure-related health disorders.

표-228. PubMed 논문번호 24159077의 내용 요약

구분	내용
PubMed ID	24159077
TITLE	Parental smoking during pregnancy and cardiovascular structures and function in childhood: the Generation R Study.
JOURNAL	International journal of epidemiology: 10.1093/ije/dyt178
AUTHORS	Taal H Rob, de Jonge Layla L, van Osch-Gevers Lennie, Steegers Eric A P, Hofman Albert, Helbing Willem A, van der Heijden Albert J, Jaddoe Vincent W V
BACKGROUND	Foetal smoke exposure might lead to foetal developmental adaptations that permanently affect the cardiovascular system. We assessed the associations of both maternal and paternal smoking during pregnancy with childhood cardiovascular structures and function.
METHOD	In a prospective cohort study among 5565 children, we examined whether maternal and paternal smoking during pregnancy are associated with blood pressure, carotid-femoral pulse wave velocity and left cardiac structures and function in 6-year-old children.
RESULTS	As compared with children from non-smoking mothers, children from mothers who smoked more than 10 cigarettes per day had a higher diastolic blood pressure [difference 1.43 mmHg (95% confidence interval: 0.22, 2.63)]. Maternal smoking during pregnancy was not associated with systolic blood pressure, childhood carotid-femoral pulse wave velocity or left cardiac structures. Maternal smoking of 10 or more cigarettes per day was associated with a higher fractional shortening in childhood [difference 1.01% (95% confidence interval: 0.18, 1.84)]. Among mothers who did not smoke during pregnancy, paternal smoking was associated with aortic root diameter but not with other cardiovascular outcomes.
CONCLUSIONS	Maternal smoking during pregnancy is associated with higher diastolic blood pressure and fractional shortening, although the effect estimates are small. The stronger effect estimates for maternal smoking compared with paternal smoking might suggest that direct intrauterine adaptive responses are involved as underlying mechanisms.

표-229. PubMed 논문번호 24162013의 내용 요약

구분	내용
PubMed ID	24162013
TITLE	Effects of heat waves on mortality: effect modification and confounding by air pollutants.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0b013e31828ac01b
AUTHORS	Analitis Antonis, Michelozzi Paola, D'Ippoliti Daniela, De'Donato Francesca, Menne Bettina, Matthies Franziska, Atkinson Richard W, I□iguez Carmen, Basaga□a Xavier, Schneider Alexandra, Lefranc Agn□s, Paldy Anna, Bisanti Luigi, Katsouyanni Klea
BACKGROUND	Heat waves and air pollution are both associated with increased mortality. Their joint effects are less well understood.
METHODS	We explored the role of air pollution in modifying the effects of heat waves on mortality, within the EuroHEAT project. Daily mortality, meteorologic, and air pollution data from nine European cities for the years 1990-2004 were assembled. We defined heat waves by taking both intensity and duration into account. The city-specific effects of heat wave episodes were estimated using generalized estimating equation models, adjusting for potential confounders with and without inclusion of air pollutants (particles, ozone, nitrogen dioxide, sulphur dioxide, carbon monoxide). To investigate effect modification, we introduced an interaction term between heat waves and each single pollutant in the models. Random effects meta-analysis was used to summarize the city-specific results.
RESULTS	The increase in the number of daily deaths during heat wave episodes was 54% higher on high ozone days compared with low, among people age 75-84 years. The heat wave effect on high PM10 days was increased by 36% and 106% in the 75-84 year and 85+ year age groups, respectively. A similar pattern was observed for effects on cardiovascular mortality. Effect modification was less evident for respiratory mortality, although the heat wave effect itself was greater for this cause of death. The heat wave effect was smaller (15-30%) after adjustment for ozone or PM10.
CONCLUSIONS	The heat wave effect on mortality was larger during high ozone or high PM10 days. When assessing the effect of heat waves on mortality, lack of adjustment for ozone and especially PM10 overestimates effect parameters. This bias has implications for public health policy.

표-230. PubMed 논문번호 24182671의 내용 요약

구분	내용
PubMed ID	24182671
TITLE	Watching soccer is not associated with an increase in cardiac events.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2013.10.066
AUTHORS	Niederseer David, Thaler Christoph W, Egger Andreas, Niederseer Michaela C, Pl□derl Martin, Niebauer Josef
BACKGROUND	It remains controversial, whether spectators of soccer matches are exposed to an increased risk of cardiac events. In 2006, the Soccer World Cup (SWC) took place in Germany and provided an excellent opportunity to assess the effects of emotional stress on cardiac events in a large cohort of soccer enthusiasts in the region of Bavaria.
METHODS	We analyzed data from the Bavarian Council for Statistics and Data Management for the period of SWC (June 9-July 9, 2006) and reference periods (SWCRef; May 1-July 31, 2005; May 1-June 8, 2006 and July 10-31, 2006) for the following diagnoses: myocardial infarction; myocardial re-infarction; cardiac arrest; paroxysmal tachycardia; atrial fibrillation, atrial flutter; all remaining tachyarrhythmias. Data were compared to the seven days during the tournament, on which the German team played (SWCGerman), the rest of the SWC period (i.e. the days the German team did not play, 24 days, SWCRest) and SWCRef (61 days).
RESULTS	There was neither a significant increase (p>0.433) in total cardiac events in Bavaria per day during SWCGerman (161.1 ± 46.7) or SWCRest (170.5 ± 52.3) as compared to the SWCRef (176.2 ± 51.8), nor in any investigated diagnosis. After controlling for age, gender, loss of a match, outside temperature and nitric-dioxide air pollution levels the results remained essentially unchanged.
CONCLUSION	Watching soccer was not associated with an increased incidence of cardiac events, regardless of whether the home team played or not. These data further support the hypothesis that spectators of sporting events are not exposed to an increased risk of cardiac events.

표-231. PubMed 논문번호 24190962의 내용 요약

구분	내용
PubMed ID	24190962
TITLE	Spatial association between ambient fine particulate matter and incident hypertension.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.113.003532
AUTHORS	Chen Hong, Burnett Richard T, Kwong Jeffrey C, Villeneuve Paul J, Goldberg Mark S, Brook Robert D, van Donkelaar Aaron, Jerrett Michael, Martin Randall V, Kopp Alexander, Brook Jeffrey R, Copes Ray, Chen Hong, Burnett Richard T, Kwong Jeffrey C, Villeneuve Paul J, Goldberg Mark S, Brook Robert D, van Donkelaar Aaron, Jerrett Michael, Martin Randall V, Kopp Alexander, Brook Jeffrey R, Copes Ray
BACKGROUND	Laboratory studies suggest that exposure to fine particulate matter (≤2.5 μm in diameter) (PM2.5) can trigger a combination of pathophysiological responses that may induce the development of hypertension. However, epidemiological evidence relating PM2.5 and hypertension is sparse. We thus conducted a population-based cohort study to determine whether exposure to ambient PM2.5 is associated with incident hypertension.
METHODS AND RESULTS	We assembled a cohort of 35 303 nonhypertensive adults from Ontario, Canada, who responded to 1 of 4 population-based health surveys between 1996 and 2005 and were followed up until December 31, 2010. Incident diagnoses of hypertension were ascertained from the Ontario Hypertension Database, a validated registry of persons diagnosed with hypertension in Ontario (sensitivity=72%, specificity=95%). Estimates of long-term exposure to PM2.5 at participants' postal-code residences were derived from satellite observations. We used Cox proportional hazards models, adjusting for various individual and contextual risk factors including body mass index, smoking, physical activity, and neighbourhood-level unemployment rates. We conducted various sensitivity analyses to assess the robustness of the effect estimate, such as investigating several time windows of exposure and controlling for potential changes in the risk of hypertension over time. Between 1996 and 2010, we identified 8649 incident cases of hypertension and 2296 deaths. For every 10-□g/m(3) increase of PM2.5, the adjusted hazard ratio of incident hypertension was 1.13 (95% confidence interval, 1.05-1.22). Estimated associations were comparable among all sensitivity analyses.
CONCLUSIONS	This study supports an association between PM2.5 and incident hypertension.

표-232. PubMed 논문번호 24194529의 내용 요약

구분	내용
PubMed ID	24194529
TITLE	Are air pollution and traffic noise independently associated with atherosclerosis: the Heinz Nixdorf Recall Study.
JOURNAL	European heart journal: 10.1093/eurheartj/eht426
AUTHORS	K□lsch Hagen, Hennig Frauke, Moebus Susanne, M□hlenkamp Stefan, Dragano Nico, Jakobs Hermann, Memmesheimer Michael, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara, K□lsch Hagen, Hennig Frauke, Moebus Susanne, M□hlenkamp Stefan, Dragano Nico, Jakobs Hermann, Memmesheimer Michael, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara
AIMS	Living close to high traffic has been linked to subclinical atherosclerosis, however it is not clear, whether fine particulate matter (PM) air pollution or noise, two important traffic-related exposures, are responsible for the association. We investigate the independent associations of long-term exposure to fine PM and road traffic noise with thoracic aortic calcification (TAC), a reliable measure of subclinical atherosclerosis.
METHODS AND RESULTS	We used baseline data (2000-2003) from the German Heinz Nixdorf Recall Study, a population-based cohort of 4814 randomly selected participants. We assessed residential long-term exposure to PM with a chemistry transport model, and to road traffic noise using fa□ade levels from noise models as weighted 24 h mean noise (Lden) and night-time noise (Lnight). Thoracic aortic calcification was quantified from non-contrast enhanced electron beam computed tomography. We used multiple linear regression to estimate associations of environmental exposures with ln(TAC+1), adjusting for each other, individual, and neighbourhood characteristics. In 4238 participants (mean age 60 years, 49.9% male), PM2.5 (aerodynamic diameter ≤2.5 □m) and Lnight are both associated with an increasing TAC-burden of 18.1% (95% CI: 6.6; 30.9%) per 2.4 □g/m(3) PM2.5 and 3.9% (95% CI 0.0; 8.0%) per 5dB(A) Lnight, respectively, in the full model and after mutual adjustment. We did not observe effect measure modification of the PM2.5 association by Lnight or vice versa.
CONCLUSION	Long-term exposure to fine PM and night-time traffic noise are both independently associated with subclinical atherosclerosis and may both contribute to the association of traffic proximity with atherosclerosis.

표-233. PubMed 논문번호 24257509의 내용 요약

구분	내용
PubMed ID	24257509
TITLE	Ambient particulate air pollution and microRNAs in elderly men.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000026
AUTHORS	Fossati Serena, Baccarelli Andrea, Zanobetti Antonella, Hoxha Mirjam, Vokonas Pantel S, Wright Robert O, Schwartz Joel
BACKGROUND	Ambient particulate matter (PM) has been associated with mortality and morbidity for cardiovascular disease. MicroRNAs control gene expression at a posttranscriptional level. Altered microRNA expression has been reported in processes related to cardiovascular disease and PM exposure, such as systemic inflammation, endothelial dysfunction, and atherosclerosis. Polymorphisms in microRNA-related genes could influence response to PM.
METHODS	We investigated the association of exposure to ambient particles in several time windows (4-hour to 28-day moving averages) and blood leukocyte expression changes in 14 candidate microRNAs in 153 elderly males from the Normative Aging Study (examined 2005-2009). Potential effect modification by six single nucleotide polymorphisms (SNPs) in three microRNA-related genes was investigated. Fine PM (PM2.5), black carbon, organic carbon, and sulfates were measured at a stationary ambient monitoring site. Linear regression models, adjusted for potential confounders, were used to assess effects of particles and SNP-by-pollutant interaction. An in silico pathway analysis was performed on target genes of microRNAs associated with the pollutants.
RESULTS	We found a negative association for pollutants in all moving averages and miR-1, -126, -135a, -146a, -155, -21, -222, and -9. The strongest associations were observed with the 7-day moving averages for PM2.5 and black carbon and with the 48-hour moving averages for organic carbon. The association with sulfates was stable across the moving averages. The in silico pathway analysis identified 18 pathways related to immune response shared by at least two microRNAs; in particular, the "high-mobility group protein B1/advanced glycosylation end product-specific receptor signaling pathway" was shared by miR-126, -146a, -155, -21, and -222. No important associations were observed for miR-125a-5p, -125b, -128, -147, -218, and -96. We found significant SNP-by-pollutant interactions for rs7813, rs910925, and rs1062923 in GEMIN4 and black carbon and PM2.5 for miR-1, -126, -146a, -222, and -9, and for rs1640299 in DGCR8 and SO4 for miR-1 and -135a.
CONCLUSIONS	Exposure to ambient particles could cause a downregulation of microRNAs involved in processes related to PM exposure. Polymorphisms in GEMIN4 and DGCR8 could modify these associations.

표-234. PubMed 논문번호 24267265의 내용 요약

구분	내용
PubMed ID	24267265
TITLE	Variability and reproducibility of carotid structural and functional parameters assessed with transcutaneous ultrasound - results from the SAPALDIA Cohort Study.
JOURNAL	Atherosclerosis: 10.1016/j.atherosclerosis.2013.10.010
AUTHORS	Caviezel Seraina, Dratva Julia, Schaffner Emmanuel, Teynor Alexandra, Baumstark Manfred W, Schindler Christian, de Groot Eric, Burdet Luc, Rothe Thomas, Pons Marco, Gaspoz Jean-Michel, Rochat Thierry, K□nzli Nino, Probst-Hensch Nicole, Schmidt-Trucks□ss Arno
OBJECTIVE	Carotid intima media thickness (CIMT) and local stiffness are vascular biomarkers of atherosclerotic burden. We investigated the variability and reproducibility of clinically relevant structural (CIMT, lumen diameter) and functional parameters (strain, distensibility, compliance, β-stiffness index, Peterson's elastic modulus and Young's elastic modulus) measured in B-mode ultrasound sequences of the common carotid artery in the second follow up of the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases In Adults (SAPALDIA3).
METHODS	Ultrasound sequential images were examined twice over a 1 cm segment across at least one heart cycle in 165 SAPALDIA3 participants. To assess variability and reproducibility of structural and functional parameters, individual coefficients of variation (CV), intraclass correlation (ICC), Bland-Altman plots and mixed effect regressions were used.
RESULTS	ICCs of repeated examinations ranged between 0.67 and 0.77 for blood pressure indices, between 0.87 and 0.97 for structural properties and between 0.75 and 0.79 for functional parameters. CV was lowest in structural parameters (1.6-4.6%), followed by blood pressure (5.1-7.9%) and functional indices (11.0-13.1%). Variations in all parameters were predominantly explained by subjects (>74% in functional, >82% in structural properties). Bland-Altman plots for functional indices showed mean and standard deviation of the respective mean value of 4.2(19.6)% for strain, 1.9(24.4)% for distensibility, 2.4(22.2)% for compliance, 3.0(24.4)% for β-stiffness index, 0.9(25.7)% for Peterson's elastic modulus and 1.2(27.9)% for Young's elastic modulus.
CONCLUSION	The results show that SAPALDIA3 measurements of transcutaneous ultrasound examinations have an excellent reproducibility of structural parameters and a good reproducibility of functional indices.

표-235. PubMed 논문번호 24295879의 내용 요약

구분	내용
PubMed ID	24295879
TITLE	Implementation of the NHLBI integrated guidelines for cardiovascular health and risk reduction in children and adolescents: rationale and study design for young hearts, strong starts, a cluster-randomized trial targeting body mass index, blood pressure, and tobacco.
JOURNAL	Contemporary clinical trials: 10.1016/j.cct.2013.11.011
AUTHORS	LaBresh Kenneth A, Lazorick Suzanne, Ariza Adolfo J, Furberg Robert D, Whetstone Lauren, Hobbs Connie, de Jesus Janet, Bender Randall H, Salinas Ilse G, Binns Helen J, LaBresh Kenneth A, Lazorick Suzanne, Ariza Adolfo J, Furberg Robert D, Whetstone Lauren, Hobbs Connie, de Jesus Janet, Bender Randall H, Salinas Ilse G, Binns Helen J
BACKGROUND	Cardiovascular disease (CVD) and the underlying atherosclerosis begin in childhood, and their presence and intensity are related to known cardiovascular disease risk factors. Attention to risk factor control in childhood has the potential to reduce subsequent risk of CVD.
OBJECTIVE	The Young Hearts Strong Starts Study was designed to test strategies facilitating adoption of the National, Heart, Lung and Blood Institute supported Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents. This study compares guideline-based quality measures for body mass index, blood pressure, and tobacco using two strategies: a multifaceted, practice-directed intervention versus standard dissemination.
STUDY DESIGN	Two primary care research networks recruited practices and provided support for the intervention and outcome evaluations. Individual practices were randomly assigned to the intervention or control groups using a cluster randomized design based on network affiliation, number of clinicians per practice, urban versus nonurban location, and practice type. The units of observation are individual children because measure adherence is abstracted from individual patient's medical records. The units of randomization are physician practices. This results in a multilevel design in which patients are nested within practices. The intervention practices received toolkits and supported guideline implementation including academic detailing, an ongoing e-learning group. This project is aligned with the American Board of Pediatrics Maintenance of Certification requirements including monthly physician self-abstraction, webinars, and other elements of the trial.
SIGNIFICANCE	This trial will provide an opportunity to demonstrate tools and strategies to enhance CV prevention in children by guideline-based interventions.

표-236. PubMed 논문번호 24339911의 내용 요약

구분	내용
PubMed ID	24339911
TITLE	Association between exposure to environmental tobacco smoke and biomarkers of oxidative stress among patients hospitalised with acute myocardial infarction.
JOURNAL	PloS one: 10.1371/journal.pone.0081209
AUTHORS	Megson Ian L, Haw Sally J, Newby David E, Pell Jill P
OBJECTIVE	To determine whether exposure to environmental tobacco smoke was associated with oxidative stress among patients hospitalised for acute myocardial infarction.
DESIGN	An existing cohort study of 1,261 patients hospitalised for acute myocardial infarction.
SETTING	Nine acute hospitals in Scotland.
PARTICIPANTS	Sixty never smokers who had been exposed to environmental tobacco smoke (admission serum cotinine ≥3.0 ng/mL) were compared with 60 never smokers who had not (admission serum cotinine ≤0.1 ng/mL).
INTERVENTION	None.
MAIN OUTCOME MEASURES	Three biomarkers of oxidative stress (protein carbonyl, malondialdehyde (MDA) and oxidised low-density lipoprotein (ox-LDL)) were measured on admission blood samples and adjusted for potential confounders.
RESULTS	After adjusting for baseline differences in age, sex and socioeconomic status, exposure to environmental tobacco smoke was associated with serum concentrations of both protein carbonyl (beta coefficient 7.96, 95% CI 0.76, 15.17, p=0.031) and MDA (beta coefficient 10.57, 95% CI 4.32, 16.81, p=0.001) but not ox-LDL (beta coefficient 2.14, 95% CI -8.94, 13.21, p=0.703).
CONCLUSIONS	Exposure to environmental tobacco smoke was associated with increased oxidative stress. Further studies are requires to explore the role of oxidative stress in the association between environmental tobacco smoke and myocardial infarction.

표-237. PubMed 논문번호 24373585의 내용 요약

구분	내용
PubMed ID	24373585
TITLE	An indoor air filtration study in homes of elderly: cardiovascular and respiratory effects of exposure to particulate matter.
JOURNAL	Environmental health : a global access science source: 10.1186/1476-069X-12-116
AUTHORS	Karottki Dorina Gabriela, Spilak Michal, Frederiksen Marie, Gunnarsen Lars, Brauner Elvira Vaclavik, Kolarik Barbara, Andersen Zorana Jovanovic, Sigsgaard Torben, Barregard Lars, Strandberg Bo, Sallsten Gerd, M ø ller Peter, Loft Steffen
BACKGROUND	Exposure to particulate air pollution increases respiratory and cardiovascular morbidity and mortality, especially in elderly, possibly through inflammation and vascular dysfunction.
METHODS	We examined potential beneficial effects of indoor air filtration in the homes of elderly, including people taking vasoactive drugs.Forty-eight nonsmoking subjects (51 to 81 years) in 27 homes were included in this randomized, double-blind, crossover intervention study with consecutive two-week periods with or without the inclusion of a high-efficiency particle air filter in re-circulating custom built units in their living room and bedroom. We measured blood pressure, microvascular and lung function and collected blood samples for hematological, inflammation, monocyte surface and lung cell damage markers before and at day 2, 7 and 14 during each exposure scenario.
RESULTS	The particle filters reduced the median concentration of PM2.5 from approximately 8 to 4 μg/m3 and the particle number concentration from 7669 to 5352 particles/cm3. No statistically significant effects of filtration as category were observed on microvascular and lung function or the biomarkers of systemic inflammation among all subjects, or in the subgroups taking (n = 11) or not taking vasoactive drugs (n = 37). However, the filtration efficacy was variable and microvascular function was within 2 days significantly increased with the actual PM2.5 decrease in the bedroom, especially among 25 subjects not taking any drugs.
CONCLUSION	Substantial exposure contrasts in the bedroom and no confounding by drugs appear required for improved microvascular function by air filtration, whereas no other beneficial effect was found in this elderly population.

표-238. PubMed 논문번호 24382024의 내용 요약

구분	내용
PubMed ID	24382024
TITLE	Ambient fine particulate air pollution triggers ST-elevation myocardial infarction, but not non-ST elevation myocardial infarction: a case-crossover study.
JOURNAL	Particle and fibre toxicology: 10.1186/1743-8977-11-1
AUTHORS	Gardner Blake, Ling Frederick, Hopke Philip K, Frampton Mark W, Utell Mark J, Zareba Wojciech, Cameron Scott J, Chalupa David, Kane Cathleen, Kulandhaisamy Suresh, Topf Michael C, Rich David Q
BACKGROUND	We and others have shown that increases in particulate air pollutant (PM) concentrations in the previous hours and days have been associated with increased risks of myocardial infarction, but little is known about the relationships between air pollution and specific subsets of myocardial infarction, such as ST-elevation myocardial infarction (STEMI) and non ST-elevation myocardial infarction (NSTEMI).
METHODS	Using data from acute coronary syndrome patients with STEMI (n = 338) and NSTEMI (n = 339) and case-crossover methods, we estimated the risk of STEMI and NSTEMI associated with increased ambient fine particle (<2.5 um) concentrations, ultrafine particle (10-100 nm) number concentrations, and accumulation mode particle (100-500 nm) number concentrations in the previous few hours and days.
RESULTS	We found a significant 18% increase in the risk of STEMI associated with each 7.1 $\mu g/m^3$ increase in PM_2 .□ concentration in the previous hour prior to acute coronary syndrome onset, with smaller, non-significantly increased risks associated with increased fine particle concentrations in the previous 3, 12, and 24 hours. We found no pattern with NSTEMI. Estimates of the risk of STEMI associated with interquartile range increases in ultrafine particle and accumulation mode particle number concentrations in the previous 1 to 96 hours were all greater than 1.0, but not statistically significant. Patients with pre-existing hypertension had a significantly greater risk of STEMI associated with increased fine particle concentration in the previous hour than patients without hypertension.
CONCLUSIONS	Increased fine particle concentrations in the hour prior to acute coronary syndrome onset were associated with an increased risk of STEMI, but not NSTEMI. Patients with pre-existing hypertension and other cardiovascular disease appeared particularly susceptible. Further investigation into mechanisms by which PM can preferentially trigger STEMI over NSTEMI within this rapid time scale is needed.

표-239. PubMed 논문번호 24398072의 내용 요약

구분	내용
PubMed ID	24398072
TITLE	Brachial artery responses to ambient pollution, temperature, and humidity in people with type 2 diabetes: a repeated-measures study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1206136
AUTHORS	Zanobetti Antonella, Luttmann-Gibson Heike, Horton Edward S, Cohen Allison, Coull Brent A, Hoffmann Barbara, Schwartz Joel D, Mittleman Murray A, Li Yongsheng, Stone Peter H, de Souza Celine, Lamparello Brooke, Koutrakis Petros, Gold Diane R
BACKGROUND	Extreme weather and air pollution are associated with increased cardiovascular risk in people with diabetes.
OBJECTIVES	In a population with diabetes, we conducted a novel assessment of vascular brachial artery responses both to ambient pollution and to weather (temperature and water vapor pressure, a measure of humidity).
METHODS	Sixty-four 49- to 85-year-old Boston residents with type 2 diabetes completed up to five study visits (279 repeated measures). Brachial artery diameter (BAD) was measured by ultrasound before and after brachial artery occlusion [i.e., flow-mediated dilation (FMD)] and before and after nitroglycerin-mediated dilation (NMD). Ambient concentrations of fine particulate mass (PM2.5), black carbon (BC), organic carbon (OC), elemental carbon, particle number, and sulfate were measured at our monitoring site; ambient concentrations of carbon monoxide, nitrogen dioxide, and ozone were obtained from state monitors. Particle exposure in the home and during each trip to the clinic (home/trip exposure) was measured continuously and as a 5-day integrated sample. We used linear models with fixed effects for participants, adjusting for date, season, temperature, and water vapor pressure on the day of each visit, to estimate associations between our outcomes and interquartile range increases in exposure.
RESULTS	Baseline BAD was negatively associated with particle pollution, including home/trip-integrated BC (-0.02 mm; 95% CI: -0.04, -0.003, for a 0.28 μg/m3 increase in BC), OC (-0.08 mm; 95% CI: -0.14, -0.03, for a 1.61 μg/m3 increase) as well as PM2.5, 5-day average ambient PM2.5, and BC. BAD was positively associated with ambient temperature and water vapor pressure. However, exposures were not consistently associated with FMD or NMD.
CONCLUSION	Brachial artery diameter, a predictor of cardiovascular risk, decreased in association with particle pollution and increased in association with ambient temperature in our study population of adults with type 2 diabetes.
CITATION	Zanobetti A, Luttmann-Gibson H, Horton ES, Cohen A, Coull BA, Hoffmann B, Schwartz JD, Mittleman MA, Li Y, Stone PH, de Souza C, Lamparello B, Koutrakis P, Gold DR. 2014. Brachial artery responses to ambient pollution, temperature, and humidity in people with type 2 diabetes: a repeated-measures study. Environ Health Perspect 122:242-248; http://dx.doi.org/10.1289/ehp.1206136.

표-240. PubMed 논문번호 24407475의 내용 요약

구분	내용
PubMed ID	24407475
TITLE	Air quality modeling and mortality impact of fine particles reduction policies in Spain.
JOURNAL	Environmental research: 10.1016/j.envres.2013.10.009
AUTHORS	Boldo Elena, Linares Cristina, Aragon□s Nuria, Lumbreras Julio, Borge Rafael, de la Paz David, P□rez-G□mez Beatriz, Fern□ndez-Navarro Pablo, Garc□a-P□rez Javier, Poll□n Marina, Ramis Rebeca, Moreno Teresa, Karanasiou Angeliki, L□pez-Abente Gonzalo, Boldo Elena, Linares Cristina, Aragon□s Nuria, Lumbreras Julio, Borge Rafael, de la Paz David, P□rez-G□mez Beatriz, Fern□ndez-Navarro Pablo, Garc□a-P□rez Javier, Poll□n Marina, Ramis Rebeca, Moreno Teresa, Karanasiou Angeliki, L□pez-Abente Gonzalo
BACKGROUND	In recent years, Spain has implemented a number of air quality control measures that are expected to lead to a future reduction in fine particle concentrations and an ensuing positive impact on public health.
OBJECTIVES	We aimed to assess the impact on mortality attributable to a reduction in fine particle levels in Spain in 2014 in relation to the estimated level for 2007.
METHODS	To estimate exposure, we constructed fine particle distribution models for Spain for 2007 (reference scenario) and 2014 (projected scenario) with a spatial resolution of 16×16km(2). In a second step, we used the concentration-response functions proposed by cohort studies carried out in Europe (European Study of Cohorts for Air Pollution Effects and Rome longitudinal cohort) and North America (American Cancer Society cohort, Harvard Six Cities study and Canadian national cohort) to calculate the number of attributable annual deaths corresponding to all causes, all non-accidental causes, ischemic heart disease and lung cancer among persons aged over 25 years (2005-2007 mortality rate data). We examined the effect of the Spanish demographic shift in our analysis using 2007 and 2012 population figures.
RESULTS	Our model suggested that there would be a mean overall reduction in fine particle levels of 1□g/m(3) by 2014. Taking into account 2007 population data, between 8 and 15 all-cause deaths per 100,000 population could be postponed annually by the expected reduction in fine particle levels. For specific subgroups, estimates varied from 10 to 30 deaths for all non-accidental causes, from 1 to 5 for lung cancer, and from 2 to 6 for ischemic heart disease. The expected burden of preventable mortality would be even higher in the future due to the Spanish population growth. Taking into account the population older than 30 years in 2012, the absolute mortality impact estimate would increase approximately by 18%.
CONCLUSIONS	Effective implementation of air quality measures in Spain, in a scenario with a short-term projection, would amount to an appreciable decline in fine particle concentrations, and this, in turn, would lead to notable health-related benefits. Recent European cohort studies strengthen the evidence of an association between long-term exposure to fine particles and health effects, and could enhance the health impact quantification in Europe. Air quality models can contribute to improved assessment of air pollution health impact estimates, particularly in study areas without air pollution monitoring data.

표-241. PubMed 논문번호 24452269의 내용 요약

구분	내용
PubMed ID	24452269
TITLE	Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE Project.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.f7412
AUTHORS	Cesaroni Giulia, Forastiere Francesco, Stafoggia Massimo, Andersen Zorana J, Badaloni Chiara, Beelen Rob, Caracciolo Barbara, de Faire Ulf, Erbel Raimund, Eriksen Kirsten T, Fratiglioni Laura, Galassi Claudia, Hampel Regina, Heier Margit, Hennig Frauke, Hilding Agneta, Hoffmann Barbara, Houthuijs Danny, J□ckel Karl-Heinz, Korek Michal, Lanki Timo, Leander Karin, Magnusson Patrik K E, Migliore Enrica, Ostenson Caes-G□ran, Overvad Kim, Pedersen Nancy L, J Juha Pekkanen, Penell Johanna, Pershagen G□ran, Pyko Andrei, Raaschou-Nielsen Ole, Ranzi Andrea, Ricceri Fulvio, Sacerdote Carlotta, Salomaa Veikko, Swart Wim, Turunen Anu W, Vineis Paolo, Weinmayr Gudrun, Wolf Kathrin, de Hoogh Kees, Hoek Gerard, Brunekreef Bert, Peters Annette
OBJECTIVES	To study the effect of long term exposure to airborne pollutants on the incidence of acute coronary events in 11 cohorts participating in the European Study of Cohorts for Air Pollution Effects (ESCAPE).
DESIGN	Prospective cohort studies and meta-analysis of the results.
SETTING	Cohorts in Finland, Sweden, Denmark, Germany, and Italy.
PARTICIPANTS	100 166 people were enrolled from 1997 to 2007 and followed for an average of 11.5 years. Participants were free from previous coronary events at baseline.
MAIN OUTCOME MEASURES	Modelled concentrations of particulate matter <2.5 μm (PM2.5), 2.5-10 μm (PMcoarse), and <10 μm (PM10) in aerodynamic diameter, soot (PM2.5 absorbance), nitrogen oxides, and traffic exposure at the home address based on measurements of air pollution conducted in 2008-12. Cohort specific hazard ratios for incidence of acute coronary events (myocardial infarction and unstable angina) per fixed increments of the pollutants with adjustment for sociodemographic and lifestyle risk factors, and pooled random effects meta-analytic hazard ratios.
RESULTS	5157 participants experienced incident events. A 5 μg/m(3) increase in estimated annual mean PM2.5 was associated with a 13% increased risk of coronary events (hazard ratio 1.13, 95% confidence interval 0.98 to 1.30), and a 10 μg/m(3) increase in estimated annual mean PM10 was associated with a 12% increased risk of coronary events (1.12, 1.01 to 1.25) with no evidence of heterogeneity between cohorts. Positive associations were detected below the current annual European limit value of 25 μg/m(3) for PM2.5 (1.18, 1.01 to 1.39, for 5 μg/m(3) increase in PM2.5) and below 40 μg/m(3) for PM10 (1.12, 1.00 to 1.27, for 10 μg/m(3) increase in PM10). Positive but non-significant associations were found with other pollutants.
CONCLUSIONS	Long term exposure to particulate matter is associated with incidence of coronary events, and this association persists at levels of exposure below the current European limit values.

표-242. PubMed 논문번호 24504155의 내용 요약

구분	내용
PubMed ID	24504155
TITLE	The effect of workplace smoking bans on heart rate variability and pulse wave velocity of non-smoking hospitality workers.
JOURNAL	International journal of public health: 10.1007/s00038-014-0545-y
AUTHORS	Rajkumar Sarah, Schmidt-Trucks□ss Arno, Wellenius Gregory A, Bauer Georg F, Huynh Cong Khanh, Moeller Alexander, R□□sli Martin
OBJECTIVES	To investigate the effect of a change in second-hand smoke (SHS) exposure on heart rate variability (HRV) and pulse wave velocity (PWV), this study utilized a quasi-experimental setting when a smoking ban was introduced.
METHODS	HRV, a quantitative marker of autonomic activity of the nervous system, and PWV, a marker of arterial stiffness, were measured in 55 non-smoking hospitality workers before and 3-12 months after a smoking ban and compared to a control group that did not experience an exposure change. SHS exposure was determined with a nicotine-specific badge and expressed as inhaled cigarette equivalents per day (CE/d).
RESULTS	PWV and HRV parameters significantly changed in a dose-dependent manner in the intervention group as compared to the control group. A one CE/d decrease was associated with a 2.3% (95% CI 0.2-4.4; p = 0.031) higher root mean square of successive differences (RMSSD), a 5.7% (95% CI 0.9-10.2; p = 0.02) higher high-frequency component and a 0.72% (95% CI 0.40-1.05; p < 0.001) lower PWV.
CONCLUSIONS	PWV and HRV significantly improved after introducing smoke-free workplaces indicating a decreased cardiovascular risk.

표-243. PubMed 논문번호 24531056의 내용 요약

구분	내용
PubMed ID	24531056
TITLE	Association of long-term exposure to traffic-related air pollution with blood pressure and hypertension in an adult population-based cohort in Spain (the REGICOR study).
JOURNAL	Environmental health perspectives: 10.1289/ehp.1306497
AUTHORS	Foraster Maria, Basaga□a Xavier, Aguilera Inmaculada, Rivera Marcela, Agis David, Bouso Laura, Deltell Alexandre, Marrugat Jaume, Ramos Rafel, Sunyer Jordi, Vila Joan, Elosua Roberto, K□nzli Nino
BACKGROUND	Long-term exposure to traffic-related air pollution may increase blood pressure (BP) and induce hypertension. However, evidence supporting these associations is limited, and they may be confounded by exposure to traffic noise and biased due to inappropriate control for use of BP-lowering medications.
OBJECTIVES	We evaluated the associations of long-term traffic-related air pollution with BP and prevalent hypertension, adjusting for transportation noise and assessing different methodologies to control for BP-lowering medications.
METHODS	We measured systolic (SBP) and diastolic BP (DBP) at baseline (years 2003-2005) in 3,700 participants, 35-83 years of age, from a population-based cohort in Spain. We estimated home outdoor annual average concentrations of nitrogen dioxide (NO2) with a land-use regression model. We used multivariate linear and logistic regression.
RESULTS	A 10- μg/m3 increase in NO2 levels was associated with 1.34 mmHg (95% CI: 0.14, 2.55) higher SBP in nonmedicated individuals, after adjusting for transportation noise. Results were similar in the entire population after adjusting for medication, as commonly done, but weaker when other methods were used to account for medication use. For example, when 10 mmHg were added to the measured SBP levels of medicated participants, the association was β = 0.78 (95% CI: -0.43, 2.00). NO2 was not associated with hypertension. Associations of NO2 with SBP and DBP were stronger in participants with cardiovascular disease, and the association with SBP was stronger in those exposed to high traffic density and traffic noise levels $\geq$ 55 dB(A).
CONCLUSIONS	We observed a positive association between long-term exposure to NO2 and SBP, after adjustment for transportation noise, which was sensitive to the methodology used to account for medication.

표-244. PubMed 논문번호 24561271의 내용 요약

구분	내용
PubMed ID	24561271
TITLE	Long-term exposure to elemental constituents of particulate matter and cardiovascular mortality in 19 European cohorts: results from the ESCAPE and TRANSPHORM projects.
JOURNAL	Environment international: 10.1016/j.envint.2014.01.026
AUTHORS	Wang Meng, Beelen Rob, Stafoggia Massimo, Raaschou-Nielsen Ole, Andersen Zorana Jovanovic, Hoffmann Barbara, Fischer Paul, Houthuijs Danny, Nieuwenhuijsen Mark, Weinmayr Gudrun, Vineis Paolo, Xun Wei W, Dimakopoulou Konstantina, Samoli Evangelia, Laatikainen Tiina, Lanki Timo, Turunen Anu W, Oftedal Bente, Schwarze Per, Aamodt Geir, Penell Johanna, De Faire Ulf, Korek Michal, Leander Karin, Pershagen G□ran, Pedersen Nancy L, □stenson Claes-G□ran, Fratiglioni Laura, Eriksen Kirsten Thorup, S ø rensen Mette, Tj ø nneland Anne, Bueno-de-Mesquita Bas, Eeftens Marloes, Bots Michiel L, Meliefste Kees, Kr□mer Ursula, Heinrich Joachim, Sugiri Dorothea, Key Timothy, de Hoogh Kees, Wolf Kathrin, Peters Annette, Cyrys Josef, Jaensch Andrea, Concin Hans, Nagel Gabriele, Tsai Ming-Yi, Phuleria Harish, Ineichen Alex, K□nzli Nino, Probst-Hensch Nicole, Schaffner Emmanuel, Vilier Alice, Clavel-Chapelon Fran□oise, Declerq Christophe, Ricceri Fulvio, Sacerdote Carlotta, Marcon Alessandro, Galassi Claudia, Migliore Enrica, Ranzi Andrea, Cesaroni Giulia, Badaloni Chiara, Forastiere Francesco, Katsoulis Michail, Trichopoulou Antonia, Keuken Menno, Jedynska Aleksandra, Kooter Ingeborg M, Kukkonen Jaakko, Sokhi Ranjeet S, Brunekreef Bert, Katsouyanni Klea, Hoek Gerard
BACKGROUND	Associations between long-term exposure to ambient particulate matter (PM) and cardiovascular (CVD) mortality have been widely recognized. However, health effects of long-term exposure to constituents of PM on total CVD mortality have been explored in a single study only.
AIMS	The aim of this study was to examine the association of PM composition with cardiovascular mortality.
METHODS	We used data from 19 European ongoing cohorts within the framework of the ESCAPE (European Study of Cohorts for Air Pollution Effects) and TRANSPHORM (Transport related Air Pollution and Health impacts - Integrated Methodologies for Assessing Particulate Matter) projects. Residential annual average exposure to elemental constituents within particle matter smaller than 2.5 and 10 μm (PM2.5 and PM10) was estimated using Land Use Regression models. Eight elements representing major sources were selected a priori (copper, iron, potassium, nickel, sulfur, silicon, vanadium and zinc). Cohort-specific analyses were conducted using Cox proportional hazards models with a standardized protocol. Random-effects meta-analysis was used to calculate combined effect estimates.
RESULTS	The total population consisted of 322,291 participants, with 9545 CVD deaths. We found no statistically significant associations between any of the elemental constituents in PM2.5 or PM10 and CVD mortality in the pooled analysis. Most of the hazard ratios (HRs) were close to unity, e.g. for PM10 Fe the combined HR was 0.96 (0.84-1.09). Elevated combined HRs were found for PM2.5 Si (1.17, 95% CI: 0.93-1.47), and S in PM2.5 (1.08, 95% CI: 0.95-1.22) and PM10 (1.09, 95% CI: 0.90-1.32).
CONCLUSION	In a joint analysis of 19 European cohorts, we found no statistically significant association between long-term exposure to 8 elemental constituents of particles and total cardiovascular mortality.

표-245. PubMed 논문번호 24589872의 내용 요약

구분	내용
PubMed ID	24589872
TITLE	Long-term exposure to air pollution and cardiovascular mortality: an analysis of 22 European cohorts.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000076
AUTHORS	Beelen Rob, Stafoggia Massimo, Raaschou-Nielsen Ole, Andersen Zorana Jovanovic, Xun Wei W, Katsouyanni Klea, Dimakopoulou Konstantina, Brunekreef Bert, Weinmayr Gudrun, Hoffmann Barbara, Wolf Kathrin, Samoli Evangelia, Houthuijs Danny, Nieuwenhuijsen Mark, Oudin Anna, Forsberg Bertil, Olsson David, Salomaa Veikko, Lanki Timo, Yli-Tuomi Tarja, Oftedal Bente, Aamodt Geir, Nafstad Per, De Faire Ulf, Pedersen Nancy L, □stenson Claes-G□ran, Fratiglioni Laura, Penell Johanna, Korek Michal, Pyko Andrei, Eriksen Kirsten Thorup, Tj ø nneland Anne, Becker Thomas, Eeftens Marloes, Bots Michiel, Meliefste Kees, Wang Meng, Bueno-de-Mesquita Bas, Sugiri Dorothea, Kr□mer Ursula, Heinrich Joachim, de Hoogh Kees, Key Timothy, Peters Annette, Cyrys Josef, Concin Hans, Nagel Gabriele, Ineichen Alex, Schaffner Emmanuel, Probst-Hensch Nicole, Dratva Julia, Ducret-Stich Regina, Vilier Alice, Clavel-Chapelon Fran□oise, Stempfelet Morgane, Grioni Sara, Krogh Vittorio, Tsai Ming-Yi, Marcon Alessandro, Ricceri Fulvio, Sacerdote Carlotta, Galassi Claudia, Migliore Enrica, Ranzi Andrea, Cesaroni Giulia, Badaloni Chiara, Forastiere Francesco, Tamayo Ibon, Amiano Pilar, Dorronsoro Miren, Katsoulis Michail, Trichopoulou Antonia, Vineis Paolo, Hoek Gerard
BACKGROUND	Air pollution has been associated with cardiovascular mortality, but it remains unclear as to whether specific pollutants are related to specific cardiovascular causes of death. Within the multicenter European Study of Cohorts for Air Pollution Effects (ESCAPE), we investigated the associations of long-term exposure to several air pollutants with all cardiovascular disease (CVD) mortality, as well as with specific cardiovascular causes of death.
METHODS	Data from 22 European cohort studies were used. Using a standardized protocol, study area-specific air pollution exposure at the residential address was characterized as annual average concentrations of the following: nitrogen oxides (NO2 and NOx); particles with diameters of less than 2.5 μm (PM2.5), less than 10 μm (PM10), and 10 μm to 2.5 μm (PMcoarse); PM2.5 absorbance estimated by land-use regression models; and traffic indicators. We applied cohort-specific Cox proportional hazards models using a standardized protocol. Random-effects meta-analysis was used to obtain pooled effect estimates.
RESULTS	The total study population consisted of 367,383 participants, with 9994 deaths from CVD (including 4,992 from ischemic heart disease, 2264 from myocardial infarction, and 2484 from cerebrovascular disease). All hazard ratios were approximately 1.0, except for particle mass and cerebrovascular disease mortality; for PM2.5, the hazard ratio was 1.21 (95% confidence interval = 0.87-1.69) per 5 μg/m and for PM10, 1.22 (0.91-1.63) per 10 μg/m.
CONCLUSION	In a joint analysis of data from 22 European cohorts, most hazard ratios for the association of air pollutants with mortality from overall CVD and with specific CVDs were approximately 1.0, with the exception of particulate mass and cerebrovascular disease mortality for which there was suggestive evidence for an association.

표-246. PubMed 논문번호 24593877의 내용 요약

구분	내용
PubMed ID	24593877
TITLE	Traffic-related air pollution and the right ventricle. The multi-ethnic study o atherosclerosis.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201312-2298OC
AUTHORS	Leary Peter J, Kaufman Joel D, Barr R Graham, Bluemke David A, Curl Cynthia L, Hough Catherine L, Lima Joao A, Szpiro Adam A, Van Hee Victo C, Kawut Steven M
RATIONALE	Right heart failure is a cause of morbidity and mortality in common and rare heart and lung diseases. Exposure to traffic-related air pollution is linked to left ventricular hypertrophy, heart failure, and death. Relationships between traffic-related air pollution and right ventricular (RV) structure and function have not been studied.
OBJECTIVES	To characterize the relationship between traffic-related air pollutants and RV structure and function.
METHODS	We included men and women with magnetic resonance imaging assessment o RV structure and function and estimated residential outdoor nitrogen dioxide (NO2) concentrations from the Multi-ethnic Study of Atherosclerosis, a stud of individuals free of clinical cardiovascular disease at baseline. Multivariable linear regression estimated associations between NO2 exposure (averaged ov the year prior to magnetic resonance imaging) and measures of RV structure and function after adjusting for demographics, anthropometrics, smoking status, diabetes mellitus, and hypertension. Adjustment for corresponding lef ventricular parameters, traffic-related noise, markers of inflammation, and lung disease were considered in separate models. Secondary analyses considered oxides of nitrogen (NOx) as the exposure.
MEASUREMENTS AND MAIN RESULTS	The study sample included 3,896 participants. In fully adjusted models, high NO2 was associated with greater RV mass and larger RV end-diastolic volume with or without further adjustment for corresponding left ventricular parameters, traffic-related noise, inflammatory markers, or lung disease (all < 0.05). There was no association between NO2 and RV ejection fraction. Relationships between NOx and RV morphology were similar.
CONCLUSIONS	Higher levels of NO2 exposure were associated with greater RV mass and larger RV end-diastolic volume.

표-247. PubMed 논문번호 24595866의 내용 요약

구분	내용
PubMed ID	24595866
TITLE	Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults: evidence from the Cardiovascular Risk in Young Finns study and the Childhood Determinants of Adult Health Study.
JOURNAL	European heart journal: 10.1093/eurheartj/ehu049
AUTHORS	Gall Seana, Huynh Quan Long, Magnussen Costan G, Juonala Markus, Viikari Jorma S A, K□h□nen Mika, Dwyer Terence, Raitakari Olli T, Venn Alison
AIM	Recent evidence suggests that the exposure of children to their parents' smoking adversely effects endothelial function in adulthood. We investigated whether the association was also present with carotid intima-media thickness (IMT) up to 25 years later.
METHODS AND RESULTS	The study comprised participants from the Cardiovascular Risk in Young Finns Study (YFS, n = 2401) and the Childhood Determinants of Adult Health (CDAH, n = 1375) study. Exposure to parental smoking (none, one, or both) was assessed at baseline by questionnaire. B-mode ultrasound of the carotid artery determined IMT in adulthood. Linear regression on a pooled dataset accounting for the hierarchical data and potential confounders including age, sex, parental education, participant smoking, education, and adult cardiovascular risk factors was conducted. Carotid IMT in adulthood was greater in those exposed to both parents smoking than in those whose parents did not smoke [adjusted marginal means: 0.647 mm ± 0.022 (mean ± SE) vs. 0.632 mm ± 0.021, P = 0.004]. Having both parents smoke was associated with vascular age 3.3 years greater at follow-up than having neither parent smoke. The effect was independent of participant smoking at baseline and follow-up and other confounders and was uniform across categories of age, sex, adult smoking status, and cohort.
CONCLUSIONS	These results show the pervasive effect of exposure to parental smoking on children's vascular health up to 25 years later. There must be continued efforts to reduce smoking among adults to protect young people and to reduce the burden of cardiovascular disease across the population.

표-248. PubMed 논문번호 24598414의 내용 요약

구분	내용
PubMed ID	24598414
TITLE	Mortality related to air pollution with the moscow heat wave and wildfire of 2010.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000090
AUTHORS	Shaposhnikov Dmitry, Revich Boris, Bellander Tom, Bedada Getahun Bero, Bottai Matteo, Kharkova Tatyana, Kvasha Ekaterina, Lezina Elena, Lind Tomas, Semutnikova Eugenia, Pershagen G□ran
BACKGROUND	Prolonged high temperatures and air pollution from wildfires often occur together, and the two may interact in their effects on mortality. However, there are few data on such possible interactions.
METHODS	We analyzed day-to-day variations in the number of deaths in Moscow, Russia, in relation to air pollution levels and temperature during the disastrous heat wave and wildfire of 2010. Corresponding data for the period 2006-2009 were used for comparison. Daily average levels of PM10 and ozone were obtained from several continuous measurement stations. The daily number of nonaccidental deaths from specific causes was extracted from official records. Analyses of interactions considered the main effect of temperature as well as the added effect of prolonged high temperatures and the interaction with PM10.
RESULTS	The major heat wave lasted for 44 days, with 24-hour average temperatures ranging from 24° C to 31° C and PM10 levels exceeding 300 μg/m on several days. There were close to 11,000 excess deaths from nonaccidental causes during this period, mainly among those older than 65 years. Increased risks also occurred in younger age groups. The most pronounced effects were for deaths from cardiovascular, respiratory, genitourinary, and nervous system diseases. Continuously increasing risks following prolonged high temperatures were apparent during the first 2 weeks of the heat wave. Interactions between high temperatures and air pollution from wildfires in excess of an additive effect contributed to more than 2000 deaths.
CONCLUSIONS	Interactions between high temperatures and wildfire air pollution should be considered in risk assessments regarding health consequences of climate change.

표-249. PubMed 논문번호 24618231의 내용 요약

구분	내용
PubMed ID	24618231
TITLE	Hemodynamic, autonomic, and vascular effects of exposure to coarse particulate matter air pollution from a rural location.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1306595
AUTHORS	Brook Robert D, Bard Robert L, Morishita Masako, Dvonch J Timothy, Wang Lu, Yang Hui-Yu, Spino Catherine, Mukherjee Bhramar, Kaplan Mariana J, Yalavarthi Srilakshmi, Oral Elif A, Ajluni Nevin, Sun Qinghua, Brook Jeffrey R, Harkema Jack, Rajagopalan Sanjay
BACKGROUND	Fine particulate matter (PM) air pollution is associated with numerous adverse health effects, including increased blood pressure (BP) and vascular dysfunction. Coarse PM substantially contributes to global air pollution, yet differs in characteristics from fine particles and is currently not regulated. However, the cardiovascular (CV) impacts of coarse PM exposure remain largely unknown.
OBJECTIVES	Our goal was to elucidate whether coarse PM, like fine PM, is itself capable of eliciting adverse CV responses.
METHODS	We performed a randomized double-blind crossover study in which 32 healthy adults (25.9 ± 6.6 years of age) were exposed to concentrated ambient coarse particles (CAP; 76.2 ± 51.5 μg/m(3)) in a rural location and filtered air (FA) for 2 hr. We measured CV outcomes during, immediately after, and 2 hr postexposures.
RESULTS	Both systolic (mean difference = 0.32 mmHg; 95% CI: 0.05, 0.58; p = 0.021) and diastolic BP (0.27 mmHg; 95% CI: 0.003, 0.53; p = 0.05) linearly increased per 10 min of exposure during the inhalation of coarse CAP when compared with changes during FA exposure. Heart rate was on average higher (4.1 bpm; 95% CI: 3.06, 5.12; p < 0.0001) and the ratio of low-to-high frequency heart rate variability increased (0.24; 95% CI: 0.07, 0.41; p = 0.007) during coarse particle versus FA exposure. Other outcomes (brachial flow-mediated dilatation, microvascular reactive hyperemia index, aortic hemodynamics, pulse wave velocity) were not differentially altered by the exposures.
CONCLUSIONS	Inhalation of coarse PM from a rural location is associated with a rapid elevation in BP and heart rate during exposure, likely due to the triggering of autonomic imbalance. These findings add mechanistic evidence supporting the biological plausibility that coarse particles could contribute to the triggering of acute CV events.

표-250. PubMed 논문번호 24633320의 내용 요약

구분	내용
PubMed ID	24633320
TITLE	Long-term exposure to fine particulate matter: association with nonaccidental and cardiovascular mortality in the agricultural health study cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307277
AUTHORS	Weichenthal Scott, Villeneuve Paul J, Burnett Richard T, van Donkelaar Aaron, Martin Randall V, Jones Rena R, DellaValle Curt T, Sandler Dale P, Ward Mary H, Hoppin Jane A
BACKGROUND	Few studies have examined the relationship between long-term exposure to ambient fine particulate matter (PM2.5) and nonaccidental mortality in rural populations.
OBJECTIVE	We examined the relationship between PM2.5 and nonaccidental and cardiovascular mortality in the U.S. Agricultural Health Study cohort.
METHODS	The cohort (n = 83,378) included farmers, their spouses, and commercial pesticide applicators residing primarily in Iowa and North Carolina. Deaths occurring between enrollment (1993-1997) and 30 December 2009 were identified by record linkage. Six-year average (2001-2006) remote-sensing derived estimates of PM2.5 were assigned to participants' residences at enrollment, and Cox proportional hazards models were used to estimate hazard ratios (HR) in relation to a 10- μg/m(3) increase in PM2.5 adjusted for individual-level covariates.
RESULTS	In total, 5,931 nonaccidental and 1,967 cardiovascular deaths occurred over a median follow-up time of 13.9 years. PM2.5 was not associated with nonaccidental mortality in the cohort as a whole (HR = 0.95; 95% CI: 0.76, 1.20), but consistent inverse relationships were observed among women. Positive associations were observed between ambient PM2.5 and cardiovascular mortality among men, and these associations were strongest among men who did not move from their enrollment address (HR = 1.63; 95% 0.94, 2.84). In particular, cardiovascular mortality risk in men was significantly increased when analyses were limited to nonmoving participants with the most precise exposure geocoding (HR = 1.87; 95% CI: 1.04, 3.36).
CONCLUSIONS	Rural PM2.5 may be associated with cardiovascular mortality in men; however, similar associations were not observed among women. Further evaluation is required to explore these sex differences.

표-251. PubMed 논문번호 24639433의 내용 요약

구분	내용
PubMed ID	24639433
TITLE	Obesity and the cardiovascular health effects of fine particulate air pollution.
JOURNAL	Obesity (Silver Spring, Md.): 10.1002/oby.20748
AUTHORS	Weichenthal Scott, Hoppin Jane A, Reeves Francois
OBJECTIVE	This review examines evidence related to the potential impact of obesity on the cardiovascular health effects of fine particulate air pollution (PM_2 .□).
METHODS	A PubMed search was conducted in December, 2013 and studies were included if they examined the relationship between PM_2 .□ and cardiovascular health as well as effect modification by obesity.
RESULTS	One hundred twenty-one citations were reviewed; three large prospective cohort studies and 14 panel studies with short-term follow-up met the above criteria. All three cohort studies reported stronger associations between PM_2 .□ and cardiovascular mortality among obese subjects and one reported a significant trend of increased risk with increased body mass index. Similarly, 11 of 14 panel studies reported stronger associations between PM_2 .□ and acute changes in physiological measures of cardiovascular health among obese subjects including outcomes such as blood pressure and arrhythmia. Although interactions were not always statistically significant, the consistent pattern of stronger associations among obese subjects suggests that obesity may modify the impact of PM2.5 on cardiovascular health.
CONCLUSIONS	Epidemiological evidence suggests that obesity may increase susceptibility to the cardiovascular health effects of PM_2 .□. This an important area of research as the public health impacts of air pollution could increase with increasing prevalence of obesity.

표-252. PubMed 논문번호 24706041의 내용 요약

구분	내용
PubMed ID	24706041
TITLE	Epidemiological time series studies of PM2.5 and daily mortality and hospital admissions: a systematic review and meta-analysis.
JOURNAL	Thorax: 10.1136/thoraxjnl-2013-204492
AUTHORS	Atkinson R W, Kang S, Anderson H R, Mills I C, Walton H A
BACKGROUND	Short-term exposure to outdoor fine particulate matter (particles with a median aerodynamic diameter <2.5 μm (PM2.5)) air pollution has been associated with adverse health effects. Existing literature reviews have been limited in size and scope.
METHODS	We conducted a comprehensive, systematic review and meta-analysis of 110 peer-reviewed time series studies indexed in medical databases to May 2011 to assess the evidence for associations between PM2.5 and daily mortality and hospital admissions for a range of diseases and ages. We stratified our analyses by geographical region to determine the consistency of the evidence worldwide and investigated small study bias.
RESULTS	Based upon 23 estimates for all-cause mortality, a 10 □g/m(3) increment in PM2.5 was associated with a 1.04% (95% CI 0.52% to 1.56%) increase in the risk of death. Worldwide, there was substantial regional variation (0.25% to 2.08%). Associations for respiratory causes of death were larger than for cardiovascular causes, 1.51% (1.01% to 2.01%) vs 0.84% (0.41% to 1.28%). Positive associations with mortality for most other causes of death and for cardiovascular and respiratory hospital admissions were also observed. We found evidence for small study bias in single-city mortality studies and in multicity studies of cardiovascular disease.
CONCLUSIONS	The consistency of the evidence for adverse health effects of short-term exposure to PM2.5 across a range of important health outcomes and diseases supports policy measures to control PM2.5 concentrations. However, reasons for heterogeneity in effect estimates in different regions of the world require further investigation. Small study bias should also be considered in assessing and quantifying health risks from PM2.5.

표-253. PubMed 논문번호 24710134의 내용 요약

구분	내용
PubMed ID	24710134
TITLE	Long-term exposure to traffic-related air pollution and progression of artery atherosclerosis: a prospective cohort study.
JOURNAL	BMJ open: 10.1136/bmjopen-2013-004743
AUTHORS	Gan Wen Qi, Allen Ryan W, Brauer Michael, Davies Hugh W, Mancin John, Lear Scott A
OBJECTIVES	Epidemiological studies have demonstrated associations between long-t exposure to traffic-related air pollution and coronary heart disease (CH Atherosclerosis is the principal pathological process responsible for CH events, but effects of traffic-related air pollution on progression of atherosclerosis are not clear. This study aimed to investigate association between long-term exposure to traffic-related air pollution and progre of carotid artery atherosclerosis.
SETTING	Healthy volunteers in metropolitan Vancouver, Canada.
PARTICIPANTS AND OUTCOME MEASURES	509 participants aged 30-65 years were recruited and followed for approximately 5 years. At baseline and end of follow-up, participants underwent carotid artery ultrasound examinations to assess atheroscler severity, including carotid intima-media thickness, plaque area, plaque number and total area. Annual change of each atherosclerosis marker d the follow-up period was calculated as the difference between these tw measurements divided by years of follow-up. Living close to major roa defined as ≤150 m from a highway or ≤50 m from a major road. Resi exposures to traffic-related air pollutants including black carbon, fine particles, nitrogen dioxide and nitric oxide were estimated using high-resolution land-use regression models. The data were analysed us general linear models adjusting for various covariates.
RESULTS	At baseline, there were no significant differences in any atherosclerosis markers between participants living close to and those living away from roads. After follow-up, the differences in annual changes of these mark between these two groups were small and not statistically significant. A significant associations were observed with concentrations of traffic-rel air pollutants including black carbon, fine particles, nitrogen dioxide an nitric oxide.
CONCLUSIONS	This study did not find significant associations between traffic-related a pollution and progression of carotid artery atherosclerosis in a region w lower levels and smaller contrasts of ambient air pollution.

표-254. PubMed 논문번호 24742726의 내용 요약

구분	내용
PubMed ID	24742726
TITLE	Associations between personal exposure to air pollutants and lung function tests and cardiovascular indices among children with asthma living near an industrial complex and petroleum refineries.
JOURNAL	Environmental research: 10.1016/j.envres.2014.03.030
AUTHORS	Smargiassi Audrey, Goldberg Mark S, Wheeler Amanda J, Plante C□line, Valois Marie-France, Mallach Gary, Kauri Lisa Marie, Shutt Robin, Bartlett Susan, Raphoz Marie, Liu Ling
OBJECTIVE	The acute cardiorespiratory effects of air quality among children living in areas with considerable heavy industry have not been well investigated. We conducted a panel study of children with asthma living in proximity to an industrial complex housing two refineries in Montreal, Quebec, in order to assess associations between their personal daily exposure to air pollutants and changes in pulmonary function and selected indicators of cardiovascular health.
METHODS	Seventy-two children with asthma age 7-12 years in 2009-2010 participated in this panel study for a period of 10 consecutive days. They carried a small backpack for personal monitoring of sulphur dioxide (SO2), benzene, fine particles (PM2.5), nitrogen dioxide (NO2) and polycyclic aromatic hydrocarbons (PAHs) and underwent daily spirometry and cardiovascular testing (blood pressure, pulse rate and oxygen saturation). To estimate these associations, we used mixed regression models, adjusting for within-subject serial correlation, and for the effects of a number of personal and environmental variables (e.g., medication use, ethnicity, temperature).
RESULTS	Children with asthma involved in the study had relatively good pulmonary function test results (mean FEV1 compared to standard values: 89.8%, mean FVC: 97.6%, mean FEF25-75: 76.3%). Median diastolic, systolic blood pressures and oxygen saturation were 60/94 mmHg and 99%, respectively. Median personal concentrations of pollutants were NO2, 5.5 ppb; benzene, 2.1 □g/m(3); PM2.5, 5.7 □g/m(3); and total PAH, 130 □g/m(3). Most personal concentrations of SO2 were below the level of detection. No consistent associations were observed between cardio-pulmonary indices and personal exposure to PM2.5, NO2 and benzene, although there was a suggestion for a small decrease in respiratory function with total concentrations of PAHs (e.g., adjusted association with FVC: -9.9 ml per interquartile range 95%CI: -23.4, 3.7).
CONCLUSIONS	This study suggests that at low daily average levels of exposure to industrial emissions, effects on pulmonary and cardiovascular functions in children with asthma may be difficult to detect over 10 consecutive days.

표-255. PubMed 논문번호 24755038의 내용 요약

구분	내용
PubMed ID	24755038
TITLE	Association between source-specific particulate matter air pollution and hs-CRP: local traffic and industrial emissions.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307081
AUTHORS	Hennig Frauke, Fuks Kateryna, Moebus Susanne, Weinmayr Gudrun, Memmesheimer Michael, Jakobs Hermann, Br□cker-Preuss Martina, F□hrer-Sakel Dagmar, M□hlenkamp Stefan, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara
BACKGROUND	Long-term exposures to particulate matter air pollution (PM2.5 and PM10) and high traffic load have been associated with markers of systemic inflammation. Epidemiological investigations have focused primarily on total PM, which represents a mixture of pollutants originating from different sources.
OBJECTIVE	We investigated associations between source-specific PM and high-sensitive C-reactive protein (hs-CRP), an independent predictor of cardiovascular disease.
METHODS	We used data from the first (2000-2003) and second examination (2006-2008) of the Heinz Nixdorf Recall study, a prospective population-based German cohort of initially 4,814 participants (45-75 years of age). We estimated residential long-term exposure to local traffic- and industry-specific fine particulate matter (PM2.5) at participants' residences using a chemistry transport model. We used a linear mixed model with a random participant intercept to estimate associations of source-specific PM and natural log-transformed hs-CRP, controlling for age, sex, education, body mass index, low- and high-density lipoprotein cholesterol, smoking variables, physical activity, season, humidity, and city (8,204 total observations).
RESULTS	A 1- μg/m3 increase in total PM2.5 was associated with a 4.53% increase in hs-CRP concentration (95% CI: 2.76, 6.33%). hs-CRP was 17.89% (95% CI: 7.66, 29.09%) and 7.96% (95% CI: 3.45, 12.67%) higher in association with 1- μg/m3 increases in traffic- and industry-specific PM2.5, respectively. RESULTS for PM10 were similar.
CONCLUSIONS	Long-term exposure to local traffic-specific PM (PM2.5, PM10) was more strongly associated with systemic inflammation than total PM. Associations of local industry-specific PM were slightly stronger but not significantly different from associations with total PM.

표-256. PubMed 논문번호 24792413의 내용 요약

구분	내용
PubMed ID	24792413
TITLE	Chronic PM2.5 exposure and inflammation: determining sensitive subgroups in mid-life women.
JOURNAL	Environmental research: 10.1016/j.envres.2014.03.042
AUTHORS	Ostro Bart, Malig Brian, Broadwin Rachel, Basu Rupa, Gold Ellen B, Bromberger Joyce T, Derby Carol, Feinstein Steven, Greendale Gail A, Jackson Elizabeth A, Kravitz Howard M, Matthews Karen A, Sternfeld Barbara, Tomey Kristin, Green Robin R, Green Rochelle
BACKGROUND	Several cohort studies report associations between chronic exposure to ambient fine particles (PM2.5) and cardiovascular mortality. Uncertainty exists about biological mechanisms responsible for this observation, but systemic inflammation has been postulated. In addition, the subgroups susceptible to inflammation have not been fully elucidated.
METHODS	We investigated whether certain subgroups are susceptible to the effects of long-term exposure to PM2.5 on C-reactive protein (CRP), a marker of inflammation directly linked to subsequent cardiovascular disease. We used data from the SWAN cohort of 1923 mid-life women with up to five annual repeated measures of CRP. Linear mixed and GEE models accounting for repeated measurements within an individual were used to estimate the effects of prior-year PM2.5 exposure on CRP. We examined CRP as a continuous and as binary outcome for CRP greater than 3mg/l, a level of clinical significance.
RESULTS	We found strong associations between PM2.5 and CRP among several subgroups. For example a 10 □g/m(3) increase in annual PM2.5 more than doubled the risk of CRP greater than 3mg/l in older diabetics, smokers and the unmarried. Larger effects were also observed among those with low income, high blood pressure, or who were using hormone therapy, with indications of a protective effects for those using statins or consuming moderate amounts of alcohol.
CONCLUSIONS	In this study, we observed significant associations between long-term exposure to PM2.5 and CRP in several susceptible subgroups. This suggests a plausible pathway by which exposure to particulate matter may be associated with increased risk of cardiovascular disease.

표-257. PubMed 논문번호 24802512의 내용 요약

구분	내용
PubMed ID	24802512
TITLE	Short-term effects of particulate matter on stroke attack: meta-regression and meta-analyses.
JOURNAL	PloS one: 10.1371/journal.pone.0095682
AUTHORS	Yu Xiao-Bo, Su Jun-Wei, Li Xiu-Yang, Chen Gao
BACKGROUND AND PURPOSE	Currently there are more and more studies on the association between short-term effects of exposure to particulate matter (PM) and the morbidity of stroke attack, but few have focused on stroke subtypes. The objective of this study is to assess the relationship between PM and stroke subtypes attack, which is uncertain now.
METHODS	Meta-analyses, meta-regression and subgroup analyses were conducted to investigate the association between short-term effects of exposure to PM and the morbidity of different stroke subtypes from a number of epidemiologic studies (from 1997 to 2012).
RESULTS	Nineteen articles were identified. Odds ratio (OR) of stroke attack associated with particular matter ("thoracic particles" [PM10]<10 □m in aerodynamic diameter, "fine particles" [PM2.5]<2.5 □m in aerodynamic diameter) increment of 10 □g/m3 was as effect size. PM10 exposure was related to an increase in risk of stroke attack (OR per 10 □g/m3 = 1.004, 95%CI: 1.001 ~ 1.008) and PM2.5 exposure was not significantly associated with stroke attack (OR per 10 □g/m3 = 0.999, 95%CI: 0.994 ~ 1.003). But when focused on stroke subtypes, PM2.5 (OR per 10 □g/m3 = 1.025; 95%CI, 1.001~1.049) and PM10 (OR per 10 □g/m3 = 1.013; 95%CI, 1.001 ~ 1.025) exposure were statistically significantly associated with an increased risk of ischemic stroke attack, while PM2.5 (all the studies showed no significant association) and PM10 (OR per 10 □g/m3 = 1.007; 95%CI, 0.992 ~ 1.022) exposure were not associated with an increased risk of hemorrhagic stroke attack. Meta-regression found study design and area were two effective covariates.
CONCLUSION	PM2.5 and PM10 had different effects on different stroke subtypes. In the future, it's worthwhile to study the effects of PM to ischemic stroke and hemorrhagic stroke, respectively.

표-258. PubMed 논문번호 24835336의 내용 요약

구분	내용
PubMed ID	24835336
TITLE	Long-term exposure to ambient air pollution and incidence of cerebrovascular events: results from 11 European cohorts within the ESCAPE project.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307301
AUTHORS	Stafoggia Massimo, Cesaroni Giulia, Peters Annette, Andersen Zorana J, Badaloni Chiara, Beelen Rob, Caracciolo Barbara, Cyrys Josef, de Faire Ulf, de Hoogh Kees, Eriksen Kirsten T, Fratiglioni Laura, Galassi Claudia, Gigante Bruna, Havulinna Aki S, Hennig Frauke, Hilding Agneta, Hoek Gerard, Hoffmann Barbara, Houthuijs Danny, Korek Michal, Lanki Timo, Leander Karin, Magnusson Patrik K, Meisinger Christa, Migliore Enrica, Overvad Kim, Ostenson Claes-G□ran, Pedersen Nancy L, Pekkanen Juha, Penell Johanna, Pershagen Goran, Pundt Noreen, Pyko Andrei, Raaschou-Nielsen Ole, Ranzi Andrea, Ricceri Fulvio, Sacerdote Carlotta, Swart Wim J R, Turunen Anu W, Vineis Paolo, Weimar Christian, Weinmayr Gudrun, Wolf Kathrin, Brunekreef Bert, Forastiere Francesco
BACKGROUND	Few studies have investigated effects of air pollution on the incidence of cerebrovascular events.
OBJECTIVES	We assessed the association between long-term exposure to multiple air pollutants and the incidence of stroke in European cohorts.
METHODS	Data from 11 cohorts were collected, and occurrence of a first stroke was evaluated. Individual air pollution exposures were predicted from land-use regression models developed within the European Study of Cohorts for Air Pollution Effects (ESCAPE). The exposures were: PM2.5 [particulate matter (PM) ≤ 2.5 μm in diameter], coarse PM (PM between 2.5 and 10 μm), PM10 (PM ≤ 10 μm), PM2.5 absorbance, nitrogen oxides, and two traffic indicators. Cohort-specific analyses were conducted using Cox proportional hazards models. Random-effects meta-analysis was used for pooled effect estimation.
RESULTS	A total of 99,446 study participants were included, 3,086 of whom developed stroke. A 5-μg/m3 increase in annual PM2.5 exposure was associated with 19% increased risk of incident stroke [hazard ratio (HR) = 1.19, 95% CI: 0.88, 1.62]. Similar findings were obtained for PM10. The results were robust to adjustment for an extensive list of cardiovascular risk factors and noise coexposure. The association with PM2.5 was apparent among those ≥ 60 years of age (HR = 1.40, 95% CI: 1.05, 1.87), among never-smokers (HR = 1.74, 95% CI: 1.06, 2.88), and among participants with PM2.5 exposure < 25 μg/m3 (HR = 1.33, 95% CI: 1.01, 1.77).
CONCLUSIONS	We found suggestive evidence of an association between fine particles and incidence of cerebrovascular events in Europe, even at lower concentrations than set by the current air quality limit value.

표-259. PubMed 논문번호 24835507의 내용 요약

구분	내용
PubMed ID	24835507
TITLE	Arterial blood pressure and long-term exposure to traffic-related air pollution: an analysis in the European Study of Cohorts for Air Pollution Effects (ESCAPE).
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307725
AUTHORS	Fuks Kateryna B, Weinmayr Gudrun, Foraster Maria, Dratva Julia, Hampel Regina, Houthuijs Danny, Oftedal Bente, Oudin Anna, Panasevich Sviatlana, Penell Johanna, Sommar Johan N, S ø rensen Mette, Tiittanen Pekka, Wolf Kathrin, Xun Wei W, Aguilera Inmaculada, Basaga□a Xavier, Beelen Rob, Bots Michiel L, Brunekreef Bert, Bueno-de-Mesquita H Bas, Caracciolo Barbara, Cirach Marta, de Faire Ulf, de Nazelle Audrey, Eeftens Marloes, Elosua Roberto, Erbel Raimund, Forsberg Bertil, Fratiglioni Laura, Gaspoz Jean-Michel, Hilding Agneta, Jula Antti, Korek Michal, Kr□mer Ursula, K□nzli Nino, Lanki Timo, Leander Karin, Magnusson Patrik K E, Marrugat Jaume, Nieuwenhuijsen Mark J, Ostenson Claes-G□ran, Pedersen Nancy L, Pershagen G□ran, Phuleria Harish C, Probst-Hensch Nicole M, Raaschou-Nielsen Ole, Schaffner Emmanuel, Schikowski Tamara, Schindler Christian, Schwarze Per E, S ø gaard Anne J, Sugiri Dorothea, Swart Wim J R, Tsai Ming-Yi, Turunen Anu W, Vineis Paolo, Peters Annette, Hoffmann Barbara
BACKGROUND	Long-term exposure to air pollution has been hypothesized to elevate arterial blood pressure (BP). The existing evidence is scarce and country specific.
OBJECTIVES	We investigated the cross-sectional association of long-term traffic-related air pollution with BP and prevalent hypertension in European populations.
METHODS	We analyzed 15 population-based cohorts, participating in the European Study of Cohorts for Air Pollution Effects (ESCAPE). We modeled residential exposure to particulate matter and nitrogen oxides with land use regression using a uniform protocol. We assessed traffic exposure with traffic indicator variables. We analyzed systolic and diastolic BP in participants medicated and nonmedicated with BP-lowering medication (BPLM) separately, adjusting for personal and area-level risk factors and environmental noise. Prevalent hypertension was defined as ≥ 140 mmHg systolic BP, or ≥ 90 mmHg diastolic BP, or intake of BPLM. We combined cohort-specific results using random-effects meta-analysis.
RESULTS	In the main meta-analysis of 113,926 participants, traffic load on major roads within 100 m of the residence was associated with increased systolic and diastolic BP in nonmedicated participants [0.35 mmHg (95% CI: 0.02, 0.68) and 0.22 mmHg (95% CI: 0.04, 0.40) per 4,000,000 vehicles × m/day, respectively]. The estimated odds ratio (OR) for prevalent hypertension was 1.05 (95% CI: 0.99, 1.11) per 4,000,000 vehicles × m/day. Modeled air pollutants and BP were not clearly associated.
CONCLUSIONS	In this first comprehensive meta-analysis of European population-based cohorts, we observed a weak positive association of high residential traffic exposure with BP in nonmedicated participants, and an elevated OR for prevalent hypertension. The relationship of modeled air pollutants with BP was inconsistent.

표-260. PubMed 논문번호 24862977의 내용 요약

구분	내용
PubMed ID	24862977
TITLE	Plasma nitrite is an indicator of acute changes in ambient air pollutant concentrations.
JOURNAL	Inhalation toxicology: 10.3109/08958378.2014.913216
AUTHORS	Gandhi Sampada K, Rich David Q, Ohman-Strickland Pamela A, Kipen Howard M, Gow Andrew
CONTEXT	Endothelial dysfunction has been suggested as a potential mechanism by which ambient air pollution may cause acute cardiovascular events. Recently, plasma nitrite has been developed as a marker of endothelial dysfunction.
OBJECTIVES	We examined the changes in plasma nitrite concentration associated with increases in ambient air pollutant concentrations in the previous 7 d.
MATERIALS AND METHODS	We linked up to three measurements of plasma nitrite concentrations obtained from 49 students to 24-h average concentrations of five criteria air pollutants [particle mass < 2.5 □m in aerodynamic diameter (PM(2.5)), carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone (O_3)] measured at two monitoring sites closest to Rutgers University campus (6-15 miles) in New Jersey during the years 2006-2009. We examined the change in plasma nitrite associated with each interquartile-range (IQR) increase in pollutant concentration in the previous 24 h and six preceding 24- h periods, using linear mixed models.
RESULTS	IQR increases in mean PM(2.5) (7.0 □g/m^3) and CO (161.7 parts per billion) concentrations in the first 24 h before the plasma nitrite measurement were associated with increased plasma nitrite concentrations (PM(2.5): 15.5 nanomolar; 95% confidence interval (CI): 2.4, 28.5; CO: 15.6 nanomolar; 95% CI: 2.4, 28.9). Increased plasma nitrite associated with IQR increases in O_3 and SO_2 concentrations over longer lags were observed.
DISCUSSION AND CONCLUSION	Rapid increases in plasma nitrite following exposure to ambient air pollutants support the hypothesis that ambient air pollution is associated with inducible nitric oxide synthase-mediated systemic inflammation in humans.

표-261. PubMed 논문번호 24866079의 내용 요약

구분	내용
PubMed ID	24866079
TITLE	An evidence-based appraisal of global association between air pollution and risk of stroke.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2014.05.044
AUTHORS	Yang Wan-Shui, Wang Xin, Deng Qin, Fan Wen-Yan, Wang Wei-Ye
BACKGROUND	The aim of this study was to evaluate the transient effects of air pollutants on stroke morbidity and mortality using the meta-analytic approach.
METHODS	Three databases were searched for case-crossover and time series studies assessing associations between daily increases in particles with diameter<2.5 μm (PM2.5) and diameter<10 μm (PM10), sulfur dioxide (SO2), carbon monoxide (CO), nitrogen dioxide (NO2), ozone, and risks of stroke hospitalizations and mortality. Risk estimates were combined using random-effects model.
RESULTS	A total of 34 studies were included in the meta-analysis. Stroke hospitalizations or mortality increased 1.20% (95%CI: 0.22-2.18) per 10 μg/m3 increase in PM2.5, 0.58% (95%CI: 0.31-0.86) per 10 μg/m3 increase in PM10, 1.53% (95%CI: 0.66-2.41) per 10 parts per billion (ppb) increase in SO2, 2.96% (95%CI: 0.70-5.27) per 1 ppm increase in CO, and 2.24% (95%CI: 1.16-3.33) per 10ppb increase in NO2. These positive associations were the strongest on the same day of exposure, and appeared to be more apparent for ischemic stroke (for all 4 gaseous pollutants) and among Asian countries (for all 6 pollutants). In addition, an elevated risk (2.45% per 10 ppb; 95%CI: 0.35-4.60) of ischemic stroke associated with ozone was found, but not for hemorrhagic stroke.
CONCLUSION	Our study indicates that air pollution may transiently increase the risk of stroke hospitalizations and stroke mortality. Although with a weak association, these findings if validated may be of both clinical and public health importance given the great global burden of stroke and air pollution.

표-262. PubMed 논문번호 24886243의 내용 요약

구분	내용
PubMed ID	24886243
TITLE	A cross-sectional analysis of the effects of residential greenness on blood pressure in 10-year old children: results from the GINIplus and LISAplus studies.
JOURNAL	BMC public health: 10.1186/1471-2458-14-477
AUTHORS	Markevych Iana, Thiering Elisabeth, Fuertes Elaine, Sugiri Dorothea, Berdel Dietrich, Koletzko Sibylle, von Berg Andrea, Bauer Carl-Peter, Heinrich Joachim
BACKGROUND	According to Ulrich's psychoevolutionary theory, contact with green environments mitigates stress by activating the parasympathetic system, (specifically, by decreasing blood pressure (BP)). Experimental studies have confirmed this biological effect. However, greenness effects on BP have not yet been explored using an observational study design. We assessed whether surrounding residential greenness is associated with BP in 10 year-old German children.
METHODS	Systolic and diastolic BPs were assessed in 10 year-old children residing in the Munich and Wesel study areas of the German GINIplus and LISAplus birth cohorts. Complete exposure, outcome and covariate data were available for 2,078 children. Residential surrounding greenness was defined as the mean of Normalized Difference Vegetation Index (NDVI) values, derived from Landsat 5 TM satellite images, in circular 500-m buffers around current home addresses of participants. Generalized additive models assessed pooled and area-specific associations between BP and residential greenness categorized into area-specific tertiles.
RESULTS	In the pooled adjusted model, the systolic BP of children living at residences with low and moderate greenness was 0.90 ± 0.50 mmHg (p-value = 0.073) and 1.23 ± 0.50 mmHg (p-value = 0.014) higher, respectively, than the systolic BP of children living in areas of high greenness. Similarly, the diastolic BP of children living in areas with low and moderate greenness was 0.80 ± 0.38 mmHg (p-value = 0.033) and 0.96 ± 0.38 mmHg (p-value = 0.011) higher, respectively, than children living in areas with high greenness. These associations were not influenced by environmental stressors (temperature, air pollution, noise annoyance, altitude and urbanisation level). When stratified by study area, associations were significant among children residing in the urbanised Munich area but null for those in the rural Wesel area.
CONCLUSIONS	Lower residential greenness was positively associated with higher BP in 10 year-old children living in an urbanised area. Further studies varying in participants' age, geographical area and urbanisation level are required.

표-263. PubMed 논문번호 24895081의 내용 요약

구분	내용
PubMed ID	24895081
TITLE	Reduction of ST-elevation myocardial infarction in Canton Ticino (Switzerland) after smoking bans in enclosed public places - No Smoke Pub Study.
JOURNAL	European journal of public health: 10.1093/eurpub/cku067
AUTHORS	Di Valentino Marcello, Muzzarelli Stefano, Limoni Costanzo, Porretta Alessandra P, Rigoli Aldo, Barazzoni Fabrizio, Kaiser Christoph, Pedrazzini Giovanni, Osswald Stefan, Moccetti Tiziano, Gallino Augusto, Di Valentino Marcello, Muzzarelli Stefano, Limoni Costanzo, Porretta Alessandra P, Rigoli Aldo, Barazzoni Fabrizio, Kaiser Christoph, Pedrazzini Giovanni, Osswald Stefan, Moccetti Tiziano, Gallino Augusto
BACKGROUND	Second-hand smoke increases the risk of acute myocardial infarction. Canton Ticino (CT) first introduced a smoking ban in public places in 2007. This offered the opportunity to assess the long-term impact of a smoking ban on the incidence of ST-elevation myocardial infarctions (STEMI) compared with a population where the law was not yet implemented.
METHODS	We assessed the incidence of STEMI hospitalizations per 100 000 inhabitants both during 3 years before and after the ban application in CT and in Canton Basel City (CBC), where this law was not yet applied. Data were obtained from the codified hospital registry (ICD-10 codes).
RESULTS	In CT, the mean incidence of STEMI admissions during the 3 pre-ban years (123.7) was significantly higher than the incidence of admissions in each of the 3 post-ban years (92.9, 101.6 and 89.6 respectively; P <.024). Analysing population subsets, a post-ban reduction was observed among ≥65-year-old people of both sexes in each of the 3 post-ban years and in the <65-year age group during the first post-ban year (P = 0.02). Conversely, the mean incidence of STEMI hospitalizations in CBC (92.4) didn't change significantly in each of the 3 post-ban years (83.9, 83.3 and 79.5, P = NS) during the same period. However, a significant long-term reduction in STEMI admissions was observed in CBC among the male group with ≥65 years (P < 0.01).
CONCLUSION	Our work suggests a significant impact of the smoke-free policy on the number of annual STEMI. Specific population subsets (i.e. ≥65-year-old females) were particularly affected by the smoking ban, showing a significant reduction in STEMI hospitalizations.

표-264. PubMed 논문번호 24906062의 내용 요약

구분	내용
PubMed ID	24906062
TITLE	Peak expiratory flow, breath rate and blood pressure in adults with changes in particulate matter air pollution during the Beijing Olympics: a panel study.
JOURNAL	Environmental research: 10.1016/j.envres.2014.05.006
AUTHORS	Mu Lina, Deng Furong, Tian Lili, Li Yanli, Swanson Mya, Ying Jingjing, Browne Richard W, Rittenhouse-Olson Kate, Zhang Junfeng Jim, Zhang Zuo-Feng, Bonner Matthew R
OBJECTIVES	This study aims to examine whether changes in short-term exposures to particulate matter are associated with changes in lung function, breath rate, and blood pressure among healthy adults and whether smoking status modifies the association.
METHODS	We took advantage of the artificially controlled changes in air pollution levels that occurred during the 2008 Olympic Games in Beijing, China and conducted a panel study of 201 Beijing residents. Data were collected before, during, and after the Olympics, respectively. Linear mixed-effect models and generalized estimating equation models were used to compare measurements of peak expiratory flow, breath rate and blood pressure across three time points.
RESULTS	The mean values of peak expiratory flow were 346.0 L/min, 399.3 L/min, and 364.1L/min over the three study periods. Peak expiratory flow levels increased in 78% of the participants when comparing the during- with pre- Olympics time points, while peak expiratory flow levels decreased in 80% of participants for the post- and during-Olympic periods comparison. In subgroup analyses comparing the during-Olympic to pre-Olympic time points, we found a larger percentage change in peak expiratory flow (+17%) among female, younger and non-smoking participants than among male, elderly and smoking participants (+12%). The percentage of participants with a fast breath rate (>20/min) changed from 9.7% to 4.9% to 30.1% among females, and from 7.9% to 2.6% to 27.3% among males over the three time points. The changes in blood pressure over the three study periods were not very clear, although there is an increase in diastolic pressure and a decrease in pulse pressure among males during the games.
CONCLUSIONS	The results suggest that exposure to different air pollution levels has significant effects on respiratory function. Smoking, age and gender appear to modify participants' biological response to changes in air quality.

표-265. PubMed 논문번호 24906066의 내용 요약

구분	내용
PubMed ID	24906066
TITLE	Total and cardiovascular mortality rates in relation to discharges from Toxics Release Inventory sites in the United States.
JOURNAL	Environmental research: 10.1016/j.envres.2014.05.010
AUTHORS	Hendryx Michael, Luo Juhua, Chen Bo-Chiuan
BACKGROUND	This study analyzed Toxics Release Inventory (TRI) discharges in association with covariate-adjusted total and cardiovascular mortality rates for males and females in US counties.
METHODS	Average annual county-level reported releases from TRI facilities measured in pounds per square mile which were calculated for the years 1990-1999, and tested for associations with age-adjusted mortality rates for 2006-2010. Chemicals were grouped into four categories: 1) carcinogens, 2) metals, 3) hazardous air pollutants, and 4) chemicals in the Comprehensive Environmental Response, Compensation and Liability Act. For each of these chemical groups the reported total, water, and air emissions were measured. Age-adjusted mortality rates were found separately for males and females from the Centers for Disease Control and Prevention for total and cardiovascular disease. Covariates included rates of smoking, obesity, high school and college education, race/ethnicity, poverty, unemployment, percent without health insurance, and urban-rural setting. Data were analyzed using multiple linear regression models.
RESULTS	Greater average annual TRI releases in 1990-1999 in all four chemical categories were significantly associated with higher mortality rates in 2006-2010 for both total and cardiovascular mortality, and for both males and females, adjusted for covariates. Associations were stronger for air releases than for water releases.
CONCLUSIONS	This study provides the first evidence that greater amounts of TRI releases are related to higher population mortality rates for cardiovascular disease. In addition, the study showed that adverse TRI effects were broadly present for both males and females for multiple chemical groups. Further progress is needed to reduce the use and release of harmful chemicals from TRI facilities in the United States.

표-266. PubMed 논문번호 24909892의 내용 요약

구분	내용
PubMed ID	24909892
TITLE	Short-term exposure to high levels of air pollution as a risk factor for acute isolated pulmonary embolism.
JOURNAL	Thrombosis research: 10.1016/j.thromres.2014.05.011
AUTHORS	Spiezia Luca, Campello Elena, Bon Maria, Maggiolo Sara, Pelizzaro Elena, Simioni Paolo
BACKGROUND	The association between air pollution exposure and occurrence of venous thromboembolism is a matter of debate. This retrospective case-control study investigated the associations between one month's exposure to elevated levels of different pollutants (i.e. PM10, CO, NOx, O3, SO2, Benzene, Benzoapyrene, Nickel, Lead Arsenic) and the development of acute isolated pulmonary embolism (PE).
METHODS	The cases included 33 patients consecutively admitted to Padua Hospital with an objectively proven diagnosis of acute unprovoked (i.e. without predisposing conditions) isolated (i.e. without deep vein thrombosis) PE. The control group consisted of 72 consecutive patients with objectively proven acute provoked (i.e. associated to predisposing conditions) isolated PE. Average mean concentrations of pollutants in the month before PE diagnosis were computed by monitors located at 2 different sites throughout the city of Padua, and were obtained from the Regional Agency for Environmental Protection.
RESULTS	Individuals who had PM10, NOx, Benzene, Benzoapyrene, Cadmium, and Lead exposure equal/above the 2nd tertile, measured in controls, showed a significant increase in the risk of unprovoked PE. In case of PM10 and Benzoapyrene this risk was not affected after adjustment for possible confounders. In fact, in the multivariate logistic regression analysis, the OR values were 5.24 (95% CI: 1.52-18.12) for PM10 and 3.95 (95% CI: 1.06-14.71) for Benzoapyrene exposure in the month before PE diagnosis.
CONCLUSIONS	Our results, although preliminary, identify short-term (i.e. one month) exposure to elevate levels of air pollutants as a possible risk factor for the development of acute isolated PE. Larger studies are needed to confirm our results.

표-267. PubMed 논문번호 24932584의 내용 요약

구분	내용
PubMed ID	24932584
TITLE	Traffic-related air pollution and the onset of myocardial infarction: disclosing benzene as a trigger? A small-area case-crossover study.
JOURNAL	PloS one: 10.1371/journal.pone.0100307
AUTHORS	Bard Denis, Kihal Wahida, Schillinger Charles, Fermanian Christophe, S□gala Claire, Glorion Sophie, Arveiler Dominique, Weber Christiane
BACKGROUND AND OBJECTIVES	Exposure to traffic is an established risk factor for the triggering of myocardial infarction (MI). Particulate matter, mainly emitted by diesel vehicles, appears to be the most important stressor. However, the possible influence of benzene from gasoline-fueled cars has not been explored so far.
METHODS AND RESULTS	We conducted a case-crossover study from 2,134 MI cases recorded by the local Coronary Heart Disease Registry (2000-2007) in the Strasbourg Metropolitan Area (France). Available individual data were age, gender, previous history of ischemic heart disease and address of residence at the time of the event. Nitrogen dioxide, particles of median aerodynamic diameter 〈10 □m (PM10), ozone, carbon monoxide and benzene air concentrations were modeled on an hourly basis at the census block level over the study period using the deterministic ADMS-Urban air dispersion model. Model input data were emissions inventories, background pollution measurements, and meteorological data. We have found a positive, statistically significant association between concentrations of benzene and the onset of MI: per cent increase in risk for a 1 □g/m3 increase in benzene concentration in the previous 0, 0-1 and 1 day was 10.4 (95% confidence interval 3-18.2), 10.7 (2.7-19.2) and 7.2 (0.3-14.5), respectively. The associations between the other pollutants and outcome were much lower and in accordance with the literature.
CONCLUSION	We have observed that benzene in ambient air is strongly associated with the triggering of MI. This novel finding needs confirmation. If so, this would mean that not only diesel vehicles, the main particulate matter emitters, but also gasoline-fueled cars - main benzene emitters-, should be taken into account for public health action.

표-268. PubMed 논문번호 24983889의 내용 요약

구분	내용
PubMed ID	24983889
TITLE	Stroke mortality associated with environmental tobacco smoke among never-smoking Japanese women: a prospective cohort study.
JOURNAL	Preventive medicine: 10.1016/j.ypmed.2014.06.029
AUTHORS	Nishino Yoshikazu, Tsuji Ichiro, Tanaka Hideo, Nakayama Tomio, Nakatsuka Haruo, Ito Hidemi, Suzuki Takaichiro, Katanoda Kota, Sobue Tomotaka, Tominaga Suketami
OBJECTIVE	This study examined the association of exposure to environmental tobacco smoke (ETS) during adulthood with stroke and its subtypes using data from a large-scale prospective cohort study in Japan.
METHODS	The study population included 36,021 never-smoking Japanese women who were enrolled between 1983 and 1985 and were followed-up for 15 years. We used Cox proportional hazard regression models to estimate hazard ratios (HRs) for stroke death associated with ETS exposure at home during adulthood.
RESULTS	A total of 906 cases of stroke death were observed during 437,715 person-years of follow-up. Compared with never-smoking women without smoking family members, HRs for stroke mortality among never-smoking women living with smoking family members in all subjects, in those aged 40-79 years, and in those aged ≥ 80 years were 1.14 (95% confidence interval: 0.99-1.31), 1.24 (95% CI: 1.05-1.46), and 0.89 (95% CI: 0.66-1.19), respectively, after adjustment for possible confounders. The risk was most evident for subarachnoid hemorrhage [HR: 1.66 (95% CI: 1.02-2.70) in all subjects].
CONCLUSION	This study suggests that exposure to ETS at home during adulthood is associated with an increased risk of stroke among never-smoking Japanese women.

표-269. PubMed 논문번호 24998583의 내용 요약

구분	내용
PubMed ID	24998583
TITLE	A cross-country comparison of knowledge, attitudes and practices about tobacco use: findings from the global adult tobacco survey.
JOURNAL	Asian Pacific journal of cancer prevention : APJCP: 10.7314/apjcp.2014.15.12.5035
AUTHORS	Gupta Bhawna, Kumar Narinder
BACKGROUND	Knowledge and individual perceptions about adverse effects of all forms of tobacco exert direct influence on the level of tobacco consumption in various socio-demographic groups. The objective of this study was to determine the nature, extent and demographic correlates of knowledge, attitudes and perceptions of use of tobacco among adults in low and middle income countries.
MATERIALS AND METHODS	The Global Adult Tobacco Survey, conducted in fourteen different countries from 2008-2010, was sourced for the data analyzed in this study. Descriptive statistical analyses were conducted to determine the prevalent knowledge and individual perceptions amongst adults about all forms of tobacco consumption.
RESULTS	There was relatively high awareness about the harmful effects of smoking tobacco with main awareness being about its relationship with lung cancer (>90% in most countries). In contrast, there was relatively low awareness about harmful effects of smokeless tobacco (< 90% in all countries except India and Bangladesh), and observed correlations of smoking tobacco with heart attacks (40.6% in China, 65.1% in India) and stroke (28.2% in China, 50.5% in India).
CONCLUSIONS	A large proportion of adults living in low and middle income countries possess adequate knowledge about smoking tobacco but have inadequate awareness as well as false perceptions about smokeless forms of tobacco. Popular beliefs of inverse relationships of tobacco consumption with knowledge, attitudes and perception of populations towards tobacco are challenged by the findings of this study.

표-270. PubMed 논문번호 25003348의 내용 요약

구분	내용
PubMed ID	25003348
TITLE	High blood pressure and long-term exposure to indoor noise and air pollution from road traffic.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307156
AUTHORS	Foraster Maria, K□nzli Nino, Aguilera Inmaculada, Rivera Marcela, Agis David, Vila Joan, Bouso Laura, Deltell Alexandre, Marrugat Jaume, Ramos Rafel, Sunyer Jordi, Elosua Roberto, Basaga□a Xavier
BACKGROUND	Traffic noise has been associated with prevalence of hypertension, but reports are inconsistent for blood pressure (BP). To ascertain noise effects and to disentangle them from those suspected to be from traffic-related air pollution, it may be essential to estimate people's noise exposure indoors in bedrooms.
OBJECTIVES	We analyzed associations between long-term exposure to indoor traffic noise in bedrooms and prevalent hypertension and systolic (SBP) and diastolic (DBP) BP, considering long-term exposure to outdoor nitrogen dioxide (NO2).
METHODS	We evaluated 1,926 cohort participants at baseline (years 2003-2006; Girona, Spain). Outdoor annual average levels of nighttime traffic noise (Lnight) and NO2 were estimated at postal addresses with a detailed traffic noise model and a land-use regression model, respectively. Individual indoor traffic Lnight levels were derived from outdoor Lnight with application of insulations provided by reported noise-reducing factors. We assessed associations for hypertension and BP with multi-exposure logistic and linear regression models, respectively.
RESULTS	Median levels were 27.1 dB(A) (indoor Lnight), 56.7 dB(A) (outdoor Lnight), and 26.8 μg/m3 (NO2). Spearman correlations between outdoor and indoor Lnight with NO2 were 0.75 and 0.23, respectively. Indoor Lnight was associated both with hypertension (OR = 1.06; 95% CI: 0.99, 1.13) and SBP (β = 0.72; 95% CI: 0.29, 1.15) per 5 dB(A); and NO2 was associated with hypertension (OR = 1.16; 95% CI: 0.99, 1.36), SBP (β = 1.23; 95% CI: 0.21, 2.25), and DBP ($\beta \geq$ 0.56; 95% CI: -0.03, 1.14) per 10 μg/m3. In the outdoor noise model, Lnight was associated only with hypertension and NO2 with BP only. The indoor noise-SBP association was stronger and statistically significant with a threshold at 30 dB(A).
CONCLUSION	Long-term exposure to indoor traffic noise was associated with prevalent hypertension and SBP, independently of NO2. Associations were less consistent for outdoor traffic Lnight and likely affected by collinearity.

표-271. PubMed 논문번호 25028096의 내용 요약

구분	내용
PubMed ID	25028096
TITLE	Inhalation of ultrafine carbon particles alters heart rate and heart rate variability in people with type 2 diabetes.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-014-0031-y
AUTHORS	Vora Rathin, Zareba Wojciech, Utell Mark J, Pietropaoli Anthony P, Chalupa David, Little Erika L, Oakes David, Bausch Jan, Wiltshire Jelani, Frampton Mark W
BACKGROUND	Diabetes may confer an increased risk for the cardiovascular health effects of particulate air pollution, but few human clinical studies of air pollution have included people with diabetes. Ultrafine particles (UFP, ≤100 nm in diameter) have been hypothesized to be an important component of particulate air pollution with regard to cardiovascular health effects.
METHODS	17 never-smoker subjects 30-60 years of age, with stable type 2 diabetes but otherwise healthy, inhaled either filtered air (0-10 particles/cm3) or elemental carbon UFP (~107 particles/cm3, ~50 ug/m3, count median diameter 32 nm) by mouthpiece, for 2 hours at rest, in a double-blind, randomized, crossover study design. A digital 12-lead electrocardiogram (ECG) was recorded continuously for 48 hours, beginning 1 hour prior to exposure.
RESULTS	Analysis of 5-minute segments of the ECG during quiet rest showed reduced high-frequency heart rate variability with UFP relative to air exposure (p = 0.014), paralleled by non-significant reductions in time-domain heart rate variability parameters. In the analysis of longer durations of the ECG, we found that UFP exposure increased the heart rate relative to air exposure. During the 21- to 45-hour interval after exposure, the average heart rate increased approximately 8 beats per minute with UFP, compared to 5 beats per minute with air (p = 0.045). There were no UFP effects on cardiac rhythm or repolarization.
CONCLUSIONS	Inhalation of elemental carbon ultrafine particles alters heart rate and heart rate variability in people with type 2 diabetes. Our findings suggest that effects may occur and persist hours after a single 2-hour exposure.

표-272. PubMed 논문번호 25047981의 내용 요약

구분	내용
PubMed ID	25047981
TITLE	Risk of ischemic heart disease following occupational exposure to welding fumes: a systematic review with meta-analysis.
JOURNAL	International archives of occupational and environmental health: 10.1007/s00420-014-0965-2
AUTHORS	Mocevic Emina, Kristiansen Pernille, Bonde Jens Peter, Mocevic Emina, Kristiansen Pernille, Bonde Jens Peter
PURPOSE	Air pollution has been linked to an increased risk of ischemic heart disease (IHD), but less is known about occupational exposure to welding fumes and the risk of IHD. The objective of this paper was to review the epidemiological evidence on causal links between welding fume exposure and risk of IHD and to investigate whether the risk of IHD depends on specific welding characteristics.
METHODS	A systematic search in Medline 1979-2013 and EMBASE 1974-2013 identified 18 epidemiological studies with at least one risk estimate of IHD morbidity or mortality among workers exposed to welding fumes. Following an assessment of completeness of reporting, confounding, and bias, each risk estimate was characterized as more or less reliable. Pooled risk estimates were computed across studies by random effect meta-analyses.
RESULTS	The weighted relative risk (RR) for IHD following exposure to welding fumes was 1.09 [95 % confidence interval (CI) 1.00, 1.19]. We calculated a RR of 1.39 (95 % CI 0.96, 2.02) among studies using an internal reference group and 1.08 (95 % CI 0.99, 1.18) for studies using an external reference group. An increased risk was observed for acute myocardial infarction RR = 1.69 (95 % CI 1.18, 2.42) and other IHDs RR = 1.06 (95 % CI 0.98, 1.14). There was too limited evidence to evaluate the risk of IHD related to specific welding characteristics.
CONCLUSION	Several studies indicate that welding is associated with a moderately increased risk of IHD; however, bias and confounding cannot be ruled out with reasonable confidence.

표-273. PubMed 논문번호 25103204의 내용 요약

구분	내용
PubMed ID	25103204
TITLE	Short-term changes in ambient particulate matter and risk of stroke: a systematic review and meta-analysis.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.114.000983
AUTHORS	Wang Yi, Eliot Melissa N, Wellenius Gregory A
BACKGROUND	Stroke is a leading cause of death and long-term disability in the United States. There is a well-documented association between ambient particulate matter air pollution (PM) and cardiovascular disease morbidity and mortality. Given the pathophysiologic mechanisms of these effects, short-term elevations in PM may also increase the risk of ischemic and/or hemorrhagic stroke morbidity and mortality, but the evidence has not been systematically reviewed.
METHODS AND RESULTS	We provide a comprehensive review of all observational human studies (January 1966 to January 2014) on the association between short-term changes in ambient PM levels and cerebrovascular events. We also performed meta-analyses to evaluate the evidence for an association between each PM size fraction (PM2.5, PM10, PM2.5-10) and each outcome (total cerebrovascular disease, ischemic stroke/transient ischemic attack, hemorrhagic stroke) separately for mortality and hospital admission. We used a random-effects model to estimate the summary percent change in relative risk of the outcome per 10- μg/m(3) increase in PM.
CONCLUSIONS	We found that PM2.5 and PM10 are associated with a 1.4% (95% CI 0.9% to 1.9%) and 0.5% (95% CI 0.3% to 0.7%) higher total cerebrovascular disease mortality, respectively, with evidence of inconsistent, nonsignificant associations for hospital admission for total cerebrovascular disease or ischemic or hemorrhagic stroke. Current limited evidence does not suggest an association between PM2.5-10 and cerebrovascular mortality or morbidity. We discuss the potential sources of variability in results across studies, highlight some observations, and identify gaps in literature and make recommendations for future studies.

표-274. PubMed 논문번호 25127437의 내용 요약

구분	내용
PubMed ID	25127437
TITLE	Incense use and cardiovascular mortality among Chinese in Singapore: the Singapore Chinese Health Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307662
AUTHORS	Pan An, Clark Maggie L, Ang Li-Wei, Yu Mimi C, Yuan Jian-Min, Koh Woon-Puay
BACKGROUND	Incense burning is common in many parts of the world. Although it is perceived that particulate matter from incense smoke is deleterious to health, there is no epidemiologic evidence linking domestic exposure to cardiovascular mortality.
OBJECTIVE	We examined the association between exposure to incense burning and cardiovascular mortality in the Singapore Chinese Health Study.
METHODS	We enrolled a total of 63,257 Singapore Chinese 45-74 years of age during 1993-1998. All participants were interviewed in person to collect information about lifestyle behaviors, including the practice of burning incense at home. We identified cardiovascular deaths via record linkage with the nationwide death registry through 31 December 2011.
RESULTS	In this cohort, 76.9% were current incense users, and most of the current users (89.9%) had burned incense daily for ≥ 20 years. Relative to noncurrent users, current users had a 12% higher risk of cardiovascular mortality [multivariable adjusted hazard ratio (HR) = 1.12; 95% CI: 1.04, 1.20]. The HR was 1.19 (95% CI: 1.03, 1.37) for mortality due to stroke and 1.10 (95% CI: 1.00, 1.21) for mortality due to coronary heart disease. The association between current incense use and cardiovascular mortality appeared to be limited to participants without a history of cardiovascular disease at baseline (HR = 1.16; 95% CI: 1.07, 1.26) but not linked to those with a history (HR = 1.00; 95% CI: 0.86, 1.17). In addition, the association was stronger in never-smokers (HR = 1.12; 95% CI: 1.02, 1.23) and former smokers (HR = 1.19; 95% CI: 1.00, 1.42) than in current smokers (HR = 1.05; 95% CI: 0.91, 1.22).
CONCLUSIONS	Long-term exposure to incense burning in the home environment was associated with an increased risk of cardiovascular mortality in the study population.

표-275. PubMed 논문번호 25133672의 내용 요약

구분	내용
PubMed ID	25133672
TITLE	Modifying effect of a common polymorphism in the interleukin-6 promoter on the relationship between long-term exposure to traffic-related particulate matter and heart rate variability.
JOURNAL	PloS one: 10.1371/journal.pone.0104978
AUTHORS	Adam Martin, Imboden Medea, Boes Eva, Schaffner Emmanuel, K□nzli Nino, Phuleria Harish Chandra, Kronenberg Florian, Gaspoz Jean-Michel, Carballo David, Probst-Hensch Nicole
BACKGROUND	Exposure to particulate matter (PM) has been associated with an increase in many inflammatory markers, including interleukin 6 (IL6). Air pollution exposure has also been suggested to induce an imbalance in the autonomic nervous system (ANS), such as a decrease in heart rate variability (HRV). In this study we aimed to investigate the modifying effect of polymorphisms in a major proinflammatory marker gene, interleukin 6 (IL6), on the relationship between long-term exposure to traffic-related PM10 (TPM10) and HRV.
METHODS	For this cross-sectional study we analysed 1552 participants of the SAPALDIA cohort aged 50 years and older. Included were persons with valid genotype data, who underwent ambulatory 24-hr electrocardiogram monitoring, and reported on medical history and lifestyle. Main effects of annual average TPM10 and IL6 gene variants (rs1800795; rs2069827; rs2069840; rs10242595) on HRV indices and their interaction with average annual exposure to TPM10 were tested, applying a multivariable mixed linear model.
RESULTS	No overall association of TPM10 on HRV was found. Carriers of two proinflammatory G-alleles of the functional IL6 -174 G/C (rs1800795) polymorphism exhibited lower HRV. An inverse association between a 1 □g/m3 increment in yearly averaged TPM10 and HRV was restricted to GG genotypes at this locus with a standard deviation of normal-to-normal intervals (SDNN) (GG-carriers: -1.8%; 95% confidence interval -3.5 to 0.01; pinteraction(additive)=0.028); and low frequency power (LF) (GG-carriers: -5.7%; 95%CI: -10.4 to -0.8; pinteraction(dominant)=0.049).
CONCLUSIONS	Our results are consistent with the hypothesis that traffic-related air pollution decreases heart rate variability through inflammatory mechanisms.

표-276. PubMed 논문번호 25135844의 내용 요약

구분	내용
PubMed ID	25135844
TITLE	Mortality from internal and external radiation exposure in a cohort of male German uranium millers, 1946-2008.
JOURNAL	International archives of occupational and environmental health: 10.1007/s00420-014-0973-2
AUTHORS	Kreuzer M, Dufey F, Laurier D, Nowak D, Marsh J W, Schnelzer M, Sogl M, Walsh L, Kreuzer M, Dufey F, Laurier D, Nowak D, Marsh J W, Schnelzer M, Sogl M, Walsh L
PURPOSE	To examine exposure-response relationships between ionizing radiation and several mortality outcomes in a subgroup of 4,054 men of the German uranium miner cohort study, who worked between 1946 and 1989 in milling facilities, but never underground or in open pit mines.
METHODS	Mortality follow-up was from 1946 to 2008, accumulating 158,383 person-years at risk. Cumulative exposure to radon progeny in working level months (WLM) (mean = 8, max = 127), long-lived radionuclides from uranium ore dust in kBqh/m(3) (mean = 3.9, max = 132), external gamma radiation in mSv (mean = 26, max = 667) and silica dust was estimated by a comprehensive job-exposure matrix. Internal Poisson regression models were applied to estimate the linear excess relative risk (ERR) per unit of cumulative exposure.
RESULTS	Overall, a total of 457, 717 and 111 deaths occurred from malignant cancer, cardiovascular diseases and non-malignant respiratory diseases, respectively. Uranium ore dust and silica dust were not associated with mortality from any of these disease groups. A statistically significant relationship between cumulative radon exposure and mortality from all cancers (ERR/100 WLM = 1.71; p = 0.02), primarily due to lung cancer (n = 159; ERR/100 WLM = 3.39; p = 0.05), was found. With respect to cumulative external gamma radiation, an excess of mortality of solid cancers (n = 434; ERR/Sv = 1.86; p = 0.06), primarily due to stomach cancer (n = 49, ERR/Sv = 10.0; p = 0.12), was present.
CONCLUSION	The present findings show an excess mortality from lung cancer due to radon exposure and from solid cancers due to external gamma radiation in uranium millers that was not statistically significant. Exposure to uranium was not associated with any cause of death, but absorbed organ doses were estimated to be low.

표-277. PubMed 논문번호 25138392의 내용 요약

구분	내용
PubMed ID	25138392
TITLE	Passive smoking assessed by salivary cotinine and self-report in relation to cause-specific mortality: 17-year follow-up of study participants in the UK Health and Lifestyle Survey.
JOURNAL	Journal of epidemiology and community health: 10.1136/jech-2014-203870
AUTHORS	Batty G David, Gale Catharine R, Jefferis Barbara, Kvaavik Elisabeth
BACKGROUND	Evidence that passive smoking is a risk factor for cardiovascular disease and selected cancers is largely derived from studies in which this exposure is self-reported. Objective assessment using biochemical techniques may yield a more accurate estimate of risk, although each approach has its strengths and weaknesses. We examined the association of salivary cotinine, a widely utilised biomarker for passive smoking, and self-reported passive smoking in the home, with mortality from all causes, cardiovascular disease and all cancers combined.
METHODS	In 1992, investigators on the UK Health and Lifestyle Survey collected data on salivary cotinine, self-reported smoking (direct and passive) and a range of covariates in 3731 men and women aged 25 years and over. Mortality was ascertained using linkage to national death records.
RESULTS	Analyses were based on 2523 individuals (1433 [57%] women) who classified themselves as non-smokers (never and former). Seventeen years of follow-up gave rise to 588 deaths (253 from cardiovascular disease and 146 from cancer). In men, adjusted hazard ratios (HR) for the association between cotinine levels (1.3-15.0 [high] vs ≤0.3 [low] ng/mL) and the various mortality outcomes were weak for total mortality (HR; 95% CI: 1.22; 0.91 to 1.64) and cardiovascular disease (1.25; 0.78 to 1.99) and absent for all cancers combined (1.10; 0.61 to 2.00). Corresponding associations were generally stronger when self-reported passive smoking (some vs none) was the exposure of interest: 1.53 (1.12 to 2.08), 1.88 (1.20 to 2.96) and 1.58 (0.85 to 2.93). The pattern of association for women in both sets of analyses was less consistent.
CONCLUSIONS	In men in the present study, compared with our biochemical marker of passive smoking, cotinine, mortality was generally more consistently associated with self-reported passive smoking.

표-278. PubMed 논문번호 25188557의 내용 요약

구분	내용
PubMed ID	25188557
TITLE	Outdoor fine particles and nonfatal strokes: systematic review and meta-analysis.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000162
AUTHORS	Shin Hwashin H, Fann Neal, Burnett Richard T, Cohen Aaron, Hubbell Bryan J
BACKGROUND	Epidemiologic studies find that long- and short-term exposure to fine particles (PM2.5) is associated with adverse cardiovascular outcomes, including ischemic and hemorrhagic strokes. However, few systematic reviews or meta-analyses have synthesized these results.
METHODS	We reviewed epidemiologic studies that estimated the risks of nonfatal strokes attributable to ambient PM2.5. To pool risks among studies we used a random-effects model and 2 Bayesian approaches. The first Bayesian approach assumes a normal prior that allows risks to be zero, positive or negative. The second assumes a gamma prior, where risks can only be positive. This second approach is proposed when the number of studies pooled is small, and there is toxicological or clinical literature to support a causal relation.
RESULTS	We identified 20 studies suitable for quantitative meta-analysis. Evidence for publication bias is limited. The frequentist meta-analysis produced pooled risk ratios of 1.06 (95% confidence interval = 1.00-1.13) and 1.007 (1.003-1.010) for long- and short-term effects, respectively. The Bayesian meta-analysis found a posterior mean risk ratio of 1.08 (95% posterior interval = 0.96-1.26) and 1.008 (1.003-1.013) from a normal prior, and of 1.05 (1.02-1.10) and 1.008 (1.004-1.013) from a gamma prior, for long- and short-term effects, respectively, per 10 μg/m PM2.5.
CONCLUSIONS	Sufficient evidence exists to develop a concentration-response relation for short- and long-term exposures to PM2.5 and stroke incidence. Long-term exposures to PM2.5 result in a higher risk ratio than short-term exposures, regardless of the pooling method. The evidence for short-term PM2.5-related ischemic stroke is especially strong.

표-279. PubMed 논문번호 25249036의 내용 요약

구분	내용
PubMed ID	25249036
TITLE	Combined impact of negative lifestyle factors on cardiovascular risk in children: a randomized prospective study.
JOURNAL	The Journal of adolescent health : official publication of the Society for Adolescent Medicine: 10.1016/j.jadohealth.2014.07.007
AUTHORS	Meyer Ursina, Schindler Christian, Bloesch Tamara, Schmocker Eliane, Zahner Lukas, Puder Jardena J, Kriemler Susi
PURPOSE	Negative lifestyle factors are known to be associated with increased cardiovascular risk (CVR) in children, but research on their combined impact on a general population of children is sparse. Therefore, we aimed to quantify the combined impact of easily assessable negative lifestyle factors on the CVR scores of randomly selected children after 4 years.
METHODS	Of the 540 randomly selected 6- to 13-year-old children, 502 children participated in a baseline health assessment, and 64% were assessed again after 4 years. Measures included anthropometry, fasting blood samples, and a health assessment questionnaire. Participants scored one point for each negative lifestyle factor at baseline: overweight; physical inactivity; high media consumption; little outdoor time; skipping breakfast; and having a parent who has ever smoked, is inactive, or overweight. A CVR score at follow-up was constructed by averaging sex- and age-related z-scores of waist circumference, blood pressure, glucose, inverted high-density lipoprotein, and triglycerides.
RESULTS	The age-, sex-, pubertal stage-, and social class-adjusted probabilities (95% confidence interval) for being in the highest CVR score tertile at follow-up for children who had at most one (n = 48), two (n = 64), three (n = 56), four (n = 41), or five or more (n = 14) risky lifestyle factors were 15.4% (8.9-25.3), 24.3% (17.4-32.8), 36.0% (28.6-44.2), 49.8% (38.6-61.0), and 63.5% (47.2-77.2), respectively.
CONCLUSIONS	Even in childhood, an accumulation of negative lifestyle factors is associated with higher CVR scores after 4 years. These negative lifestyle factors are easy to assess in clinical practice and allow early detection and prevention of CVR in childhood.

표-280. PubMed 논문번호 25250520의 내용 요약

구분	내용
PubMed ID	25250520
TITLE	Effect of exposure to PM2.5 on blood pressure: a systematic review and meta-analysis.
JOURNAL	Journal of hypertension: 10.1097/HJH.0000000000000342
AUTHORS	Liang Ruijuan, Zhang Biao, Zhao Xiaoyi, Ruan Yanping, Lian Hui, Fan Zhongjie
BACKGROUND	Comprehensive studies have confirmed that particulate matter air pollution could trigger myocardial infarction, heart failure and reduce heart rate variability; however, its effect on blood pressure (BP) remains controversial. Therefore, we did a systematic review and meta-analysis to investigate the association and its magnitude between exposure to PM2.5 and BP.
METHODS	The databases of PubMed, Ovid Medline and Embase between 1948 and 15 November 2013 were searched to identify the studies exploring the association between particulate matters (diameter <2.5 μm) (PM2.5) and BP. Selection was performed by screening abstracts and titles and then reviewing the full text of potentially eligible studies. We extracted descriptive and quantitative information from each study and used a random-effects model to calculate BP change and 95% confidence interval (95% CI) for each increment of 10 μg/m in PM2.5. Meta-regression and subgroup analyses were conducted to explore the source of heterogeneity and the impact of possible confounding factors.
RESULTS	Of 1028 identified articles, after screening and reviewing in detail, 22 studies were included in our meta-analysis. The overall analysis suggested that BP was positively related to PM2.5 exposure with an elevation of 1.393 mmHg, 95% CI (0.874-1.912) and 0.895 mmHg, 95% CI (0.49-1.299) per 10 μg/m increase for SBP and DBP, respectively. Long-term exposure showed the strongest associations with BP. And for short-term effect, the largest magnitude was seen at the lag of the previous 5 days average prior to BP measurement. Subgroup analyses yielded consistent results with the overall analyses. Meta-regression of SBP did not identify any significant potential causes of heterogeneity. For DBP, study design, the method of BP monitoring, publication year, study design, study period and sample size were significant modifiers of the relationship between DBP and PM2.5.
CONCLUSION	Exposure to PM2.5 had a statistically significant impact on BP and the magnitude of this effect may have substantially clinical implication.

표-281. PubMed 논문번호 25252923의 내용 요약

구분	내용
PubMed ID	25252923
TITLE	The Wuhan-Zhuhai (WHZH) cohort study of environmental air particulate matter and the pathogenesis of cardiopulmonary diseases: study design, methods and baseline characteristics of the cohort.
JOURNAL	BMC public health: 10.1186/1471-2458-14-994
AUTHORS	Song Yuanchao, Hou Jian, Huang Xiji, Zhang Xiaomin, Tan Aijun, Rong Yi, Sun Huizhen, Zhou Yun, Cui Xiuqing, Yang Yuqing, Guo Yanjun, Zhang Zhihong, Luo Xin, Zhang Bing, Hou Fan, He Xiaosheng, Xie Jungang, Wu Tangchun, Chen Weihong, Yuan Jing
BACKGROUND	Particulate air pollution has been recognized to be associated with a wide range of adverse health effects, including increased mortality, morbidity, exacerbation of respiratory conditions. However, earlier physiological or pathological changes or long-term bodies' reaction to air pollutants have not been studied in depth in China. The Wuhan-Zhuhai (WHZH) cohort study is designed to investigate the association between air pollutants exposure and physiological or pathological reactions on respiratory and cardiovascular system.
METHODS/DESIGN	The cohort is a community-based prospective study that includes 4812 individuals aged 18-80 years. The collections of data were conducted from April to May 2011 in Wuhan city and in May 2012 in Zhuhai city. At baseline, data on demographic and socioeconomic information, occupational history, family disease history, lifestyle, cooking mode, daily travel mode, physical activity and living condition have been collected by questionnaires. Participants underwent an extensive physical examination, including anthropometry, spirometry, electrocardiography, and measurements of blood pressure, heart rate, exhaled nitric oxide and carbon monoxide. Potential conditions in the lung, heart, liver, spleen, and skin were synchronously performed. In addition, samples of morning urine, fasting blood serum and plasma were collected during physical health examination. DNA were extracted and were stored at -80° C. Environment concentrations of particulate matter and chemicals were determined for 15 days in each of four seasons. Participants are followed for physiological or pathological changes or incidence of cardiopulmonary diseases every 3 years.
DISCUSSION	The results obtained in WHZH cohort study may increase a better understanding of the relationship between particulate air pollution and its components and possible health damages. And the potential mechanisms underlying the development of cardiopulmonary diseases has implications for the development of prevention and treatment strategies.

표-282. PubMed 논문번호 25272970의 내용 요약

구분	내용
PubMed ID	25272970
TITLE	Acute cardiovascular autonomic responses to inhaled particulates.
JOURNAL	European journal of applied physiology: 10.1007/s00421-014-2998-3
AUTHORS	Evans Joyce M, Jenkins Roger A, Ilgner Ralph H, Knapp Charles F, Zhang Qingguang, Patwardhan Abhijit R, Evans Joyce M, Jenkins Roger A, Ilgner Ralph H, Knapp Charles F, Zhang Qingguang, Patwardhan Abhijit R
PURPOSE	Harmful effects of inhaled particulates have been established in epidemiologic studies of ambient air pollution. In particular, heart rate variability responses to high levels of environmental tobacco smoke (ETS), similar to responses observed during direct smoking, have been reported. We sought to determine whether such responses could be observed at lower particulate concentrations.
METHODS	We monitored cardiovascular responses of non-smoking 21 women and 19 men to work-place-relevant levels of: ETS, cooking oil fumes (Coil), wood smoke (WS), and water vapor as sham control. Responses, tested on three consecutive days (random order of aerosol presentation), were averaged for each subject.
RESULTS	Low frequency spectral powers of heart rate and blood pressure rose during recovery from exposure to particulate, but not to sham exposures. At breathing frequencies, spectral power of men's systolic pressure doubled, and baroreflex effectiveness increased, following ETS exposure. An index of sympathetic control of heart rate was more pronounced in men than women, in response to ETS and Coil, compared to WS and sham.
CONCLUSIONS	When measured under controlled conditions, autonomic activities in non-smoking men and women exposed to low level, short term, particulate concentrations were similar to those observed during longer term, higher level exposures to ETS and to direct smoking. These increased indexes of sympathetic control of heart rate and peripheral vasomotion followed introduction of particulates by about 15 min. Finally, coupling of heart rate and systolic pressure indicated an increase in baroreflex activity in the response to breathing ETS that was less effective in men than women.

표-283. PubMed 논문번호 25275243의 내용 요약

구분	내용
PubMed ID	25275243
TITLE	Is there a differential impact of parity on blood pressure by age?
JOURNAL	Journal of hypertension: 10.1097/HJH.0000000000000325
AUTHORS	Dratva Julia, Schneider Cornelia, Schindler Christian, Stolz Daiana, Gerbase Margaret, Pons Marco, Bettschart Robert, Gaspoz Jean-Michel, K□nzli Nino, Zemp Elisabeth, Probst-Hensch Nicole
OBJECTIVE	In pregnancy, women experience metabolic and hemodynamic changes of potential long-term impact. Conflicting evidence exists on the impact on blood pressure (BP). We investigated the association between parity and BP in the Swiss Study on Air Pollution And Lung and Heart Disease In Adults cohort.
METHODS	Multilevel linear and logistic regression analyses were performed in 2837 women aged 30-73 years, with data on parity, number of births, BP, and doctor-diagnosed hypertension adjusting for potential confounders. Hypertension was defined as at least 140/90mmHg, doctor diagnosed or taking relevant treatment. Stratified analyses were performed by age (<40, 40-59, and ≥60 years) and menopausal status.
RESULTS	Parous women had a mean of 2.3 pregnancies (SD 0.95, range 1-7). A total of 26% were nulliparous. Mean BP was 119/76mmHg in nulliparous and 121/76mmHg in parous women. Parity had a significant adverse effect on BP in women at least 60 years [SBP 5.6mmHg, 95% confidence interval (CI) 2.3 to 8.9; DBP 1.8mmHg, 95% CI 0.1 to 3.6] and protective effect in women below 40 years (SBP −3.4mmHg, 95% CI −5.8 to −1.0; DBP −0.2mmHg, 95% CI −1.0 to 0.6). With increasing number of births, SBP (mmHg/birth; 95% CI) increased in older (1.2, 95% CI 0.2 to 2.2) and decreased in younger women (−1.6, 95% CI −2.6 to −0.5). Opposite effects of parity were also found for diagnosed hypertension. No interaction by menopausal status was found.
CONCLUSION	Our analyses yield differential effects of parity on BP in older vs. younger women. Reductions in BP in younger parous women have been described before; the opposite impact in older women is new. The findings may constitute biological mechanisms in an aging population or reflect birth cohort effects.

표-284. PubMed 논문번호 25298377의 내용 요약

구분	내용
PubMed ID	25298377
TITLE	Main air pollutants and diabetes-associated mortality: a systematic review and meta-analysis.
JOURNAL	European journal of endocrinology: 10.1530/EJE-14-0287
AUTHORS	Li Chengqian, Fang Dongdong, Xu Donghua, Wang Bin, Zhao Shihua, Yan Shengli, Wang Yangang
OBJECTIVE	Exposure to high levels of air pollutants may be linked to diabetes-associated mortality, but the associations remain unclear. To assess the associations between main air pollutants and diabetes-associated mortality, a systematic review and meta-analysis was performed.
METHODS	PubMed, Embase and Web of Science were searched for studies investigating the associations between increments in gaseous (nitrogen dioxide (NO2), sulphur dioxide, ozone (O3) and carbon monoxide) and particulate matter (PM; diameter<2.5 μm (PM2.5) or <10 μm (PM10)) air pollutants and diabetes-associated mortality. Using a random-effects model, relative risks (RRs) and 95% CIs were calculated per interquartile range (IQR) increment or per 10 μg/m3 increment in pollutant concentrations.
RESULTS	Out of 925 identified articles, 36 were reviewed in depth and 12 studies from 13 articles satisfying the inclusion criteria (five time-series, five case-crossovers and two cohorts) were finally included. Increased risk of diabetes-associated mortality was associated with higher levels of PM2.5 (per 10 μg/m3: RR=1.123, 95% CI 1.036-1.217, P=0.005, I2=96.1%), PM10 (per 10 μg/m3: RR=1.008, 95% CI 1.004-1.013, P<0.001, I2=0%), NO2 (per 10 μg/m3: RR=1.024, 95% CI 1.007-1.041, P=0.006, I2=49.7%) and O3 (per IQR increment: RR=1.065, 95% CI 1.017-1.115, P=0.007, I2=0.0%). No obvious risk of publication bias was observed.
CONCLUSIONS	Exposure to high levels of air pollutants is significantly associated with an increased risk of diabetes-associated mortality.

표-285. PubMed 논문번호 25318591의 내용 요약

구분	내용
PubMed ID	25318591
TITLE	Ozone co-exposure modifies cardiac responses to fine and ultrafine ambient particulate matter in mice: concordance of electrocardiogram and mechanical responses.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-014-0054-4
AUTHORS	Kurhanewicz Nicole, McIntosh-Kastrinsky Rachel, Tong Haiyan, Walsh Leon, Farraj Aimen K, Hazari Mehdi S
BACKGROUND	Studies have shown a relationship between air pollution and increased risk of cardiovascular morbidity and mortality. Due to the complexity of ambient air pollution composition, recent studies have examined the effects of co-exposure, particularly particulate matter (PM) and gas, to determine whether pollutant interactions alter (e.g. synergistically, antagonistically) the health response. This study examines the independent effects of fine (FCAPs) and ultrafine (UFCAPs) concentrated ambient particles on cardiac function, and determine the impact of ozone (O_3) co-exposure on the response. We hypothesized that UFCAPs would cause greater decrement in mechanical function and electrical dysfunction than FCAPs, and that O_3 co-exposure would enhance the effects of both particle-types.
METHODS	Conscious/unrestrained radiotelemetered mice were exposed once whole-body to either 190 $\mu g/m^3$ FCAPs or 140 $\mu g/m^3$ UFCAPs with/without 0.3 ppm O_3; separate groups were exposed to either filtered air (FA) or O_3 alone. Heart rate (HR) and electrocardiogram (ECG) were recorded continuously before, during and after exposure, and cardiac mechanical function was assessed using a Langendorff perfusion preparation 24 hrs post-exposure.
RESULTS	FCAPs alone caused a significant decrease in baseline left ventricular developed pressure (LVDP) and contractility, whereas UFCAPs did not; neither FCAPs nor UFCAPs alone caused any ECG changes. O_3 co-exposure with FCAPs caused a significant decrease in heart rate variability when compared to FA but also blocked the decrement in cardiac function. On the other hand, O_3 co-exposure with UFCAPs significantly increased QRS-interval, QTc and non-conducted P-wave arrhythmias, and decreased LVDP, rate of contractility and relaxation when compared to controls.
CONCLUSIONS	These data suggest that particle size and gaseous interactions may play a role in cardiac function decrements one day after exposure. Although FCAPs + O_3 only altered autonomic balance, UFCAPs + O_3 appeared to be more serious by increasing cardiac arrhythmias and causing mechanical decrements. As such, O_3 appears to interact differently with FCAPs and UFCAPs, resulting in varied cardiac changes, which suggests that the cardiovascular effects of particle-gas co-exposures are not simply additive or even generalizable. Additionally, the mode of toxicity underlying this effect may be subtle given none of the exposures described here impaired post-ischemia recovery.

표-286. PubMed 논문번호 25332277의 내용 요약

구분	내용
PubMed ID	25332277
TITLE	Roadway proximity and risk of sudden cardiac death in women.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.114.011489
AUTHORS	Hart Jaime E, Chiuve Stephanie E, Laden Francine, Albert Christine M
BACKGROUND	Sudden cardiac death (SCD) is a major source of mortality and is the first manifestation of heart disease for the majority of cases. Thus, there is a definite need to identify risk factors for SCD that can be modified at the population level. Exposure to traffic, measured by residential roadway proximity, has been shown to be associated with an increased risk of cardiovascular disease. Our objective was to determine whether roadway proximity was associated with an increased risk of SCD and to compare that risk with the risk of other coronary heart disease outcomes.
METHODS AND RESULTS	A total of 523 cases of SCD were identified over 26 years of follow-up among 107 130 members of the prospective Nurses' Health Study. We calculated residential distance to roadways at all residential addresses from 1986 to 2012. In age- and race-adjusted models, women living within 50 m of a major roadway had an elevated risk of SCD (hazard ratio=1.56; 95% confidence interval, 1.18-2.05). The association was attenuated but still statistically significant after controlling for potential confounders and mediators (hazard ratio=1.38; 95% confidence interval, 1.04-1.82). The equivalent adjusted hazard ratios for nonfatal myocardial infarction and fatal coronary heart disease were 1.08 (95% confidence interval, 0.96-1.23) and 1.24 (95% confidence interval, 1.03-1.50), respectively.
CONCLUSIONS	Among this sample of middle-aged and older women, roadway proximity was associated with elevated and statistically significant risks of SCD and fatal coronary heart disease, even after controlling for other cardiovascular risk factors.

표-287. PubMed 논문번호 25348167의 내용 요약

구분	내용
PubMed ID	25348167
TITLE	Relationships between fine particulate air pollution, cardiometabolic disorders, and cardiovascular mortality.
JOURNAL	Circulation research: 10.1161/CIRCRESAHA.116.305060
AUTHORS	Pope C Arden, Turner Michelle C, Burnett Richard T, Jerrett Michael, Gapstur Susan M, Diver W Ryan, Krewski Daniel, Brook Robert D, Pope C Arden, Turner Michelle C, Burnett Richard T, Jerrett Michael, Gapstur Susan M, Diver W Ryan, Krewski Daniel, Brook Robert D
RATIONALE	Growing evidence suggests that long-term exposure to fine particulate matter (PM2.5) air pollution contributes to risk of cardiovascular disease (CVD) morbidity and mortality. There is uncertainty about who are most susceptible. Individuals with underlying cardiometabolic disorders, including hypertension, diabetes mellitus, and obesity, may be at greater risk. PM2.5 pollution may also contribute to cardiometabolic disorders, augmenting CVD risk.
OBJECTIVE	This analysis evaluates relationships between long-term PM2.5 exposure and cardiometabolic disease on risk of death from CVD and cardiometabolic conditions.
METHODS AND RESULTS	Data on 669 046 participants from the American Cancer Society Cancer Prevention Study II cohort were linked to modeled PM2.5 concentrations at geocoded home addresses. Cox proportional hazards regression models were used to estimate adjusted hazards ratios for death from CVD and cardiometabolic diseases based on death-certificate information. Effect modification by pre-existing cardiometabolic risk factors on the PM2.5-CVD mortality association was examined. PM2.5 exposure was associated with CVD mortality, with the hazards ratios (95% confidence interval) per 10 μg/m(3) increase in PM2.5 equal to 1.12 (1.10-1.15). Deaths linked to hypertension and diabetes mellitus (mentioned on death certificate as either primary or contributing cause of death) were also associated with PM2.5. There was no consistent evidence of effect modification by cardiometabolic disease risk factors on the PM2.5-CVD mortality association.
CONCLUSIONS	Pollution-induced CVD mortality risk is observed for those with and without existing cardiometabolic disorders. Long-term exposure may also contribute to the development or exacerbation of cardiometabolic disorders, increasing risk of CVD, and cardiometabolic disease mortality.

표-288. PubMed 논문번호 25361615의 내용 요약

구분	내용
PubMed ID	25361615
TITLE	A controlled trial of acute effects of human exposure to traffic particles on pulmonary oxidative stress and heart rate variability.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-014-0045-5
AUTHORS	Laumbach Robert J, Kipen Howard M, Ko Susan, Kelly-McNeil Kathie, Cepeda Clarimel, Pettit Ashley, Ohman-Strickland Pamela, Zhang Lin, Zhang Junfeng, Gong Jicheng, Veleeparambil Manoj, Gow Andrew J
BACKGROUND	For many individuals, daily commuting activities on roadways account for a substantial proportion of total exposure, as well as peak-level exposures, to traffic-related air pollutants (TRAPS) including ultrafine particles, but the health impacts of these exposures are not well-understood. We sought to determine if exposure to TRAPs particles during commuting causes acute oxidative stress in the respiratory tract or changes in heart rate variability (HRV), a measure of autonomic activity.
METHODS	We conducted a randomized, cross-over trial in which twenty-one young adults took two 1.5-hr rides in a passenger vehicle in morning rush-hour traffic. The subjects wore a powered-air-purifying respirator, and were blinded to high-efficiency particulate air (HEPA) filtration during one of the rides. At time points before and after the rides, we measured HRV and markers of oxidative stress in exhaled breath condensate (EBC) including nitrite, the sum of nitrite and nitrate, malondialdehyde, and 8-isoprostane. We used mixed linear models to evaluate the effect of exposure on EBC and HRV outcomes, adjusting for pre-exposure response levels. We used linear models to examine the effects of particle concentrations on EBC outcomes at post-exposure time points.
RESULTS	Mean EBC nitrite and the sum of nitrite and nitrate were increased from baseline at immediately post-exposure comparing unfiltered to filtered rides (2.11 μM vs 1.70 μM, p = 0.02 and 19.1 μM vs 10.0 μM, p = 0.02, respectively). Mean EBC malondialdehyde (MDA) concentrations were about 10% greater following the unfiltered vs. filtered exposures, although this result was not statistically significant. We found no significant associations between exposure to traffic particles and HRV outcomes at any of the time points. At immediately post-exposure, an interquartile range increase in particle number concentration was associated with statistically significant increases in nitrite (99.4%, 95% CI 32.1% to 166.7%) and nitrite + nitrate (75.7%, 95% CI 21.5% to 130.0%).
CONCLUSIONS	Increases in markers of oxidative stress in EBC may represent early biological responses to widespread exposures to TRAPs particles that affect passengers in vehicles on heavily trafficked roadways.

표-289. PubMed 논문번호 25398188의 내용 요약

구분	내용
PubMed ID	25398188
TITLE	A unified spatiotemporal modeling approach for predicting concentrations of multiple air pollutants in the multi-ethnic study of atherosclerosis and air pollution.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408145
AUTHORS	Keller Joshua P, Olives Casey, Kim Sun-Young, Sheppard Lianne, Sampson Paul D, Szpiro Adam A, Oron Assaf P, Lindstr□m Johan, Vedal Sverre, Kaufman Joel D, Keller Joshua P, Olives Casey, Kim Sun-Young, Sheppard Lianne, Sampson Paul D, Szpiro Adam A, Oron Assaf P, Lindstr□m Johan, Vedal Sverre, Kaufman Joel D
BACKGROUND	Cohort studies of the relationship between air pollution exposure and chronic health effects require predictions of exposure over long periods of time.
OBJECTIVES	We developed a unified modeling approach for predicting fine particulate matter, nitrogen dioxide, oxides of nitrogen, and black carbon (as measured by light absorption coefficient) in six U.S. metropolitan regions from 1999 through early 2012 as part of the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air).
METHODS	We obtained monitoring data from regulatory networks and supplemented those data with study-specific measurements collected from MESA Air community locations and participants' homes. In each region, we applied a spatiotemporal model that included a long-term spatial mean, time trends with spatially varying coefficients, and a spatiotemporal residual. The mean structure was derived from a large set of geographic covariates that was reduced using partial least-squares regression. We estimated time trends from observed time series and used spatial smoothing methods to borrow strength between observations.
RESULTS	Prediction accuracy was high for most models, with cross-validation R2 (R2CV) > 0.80 at regulatory and fixed sites for most regions and pollutants. At home sites, overall R2CV ranged from 0.45 to 0.92, and temporally adjusted R2CV ranged from 0.23 to 0.92.
CONCLUSIONS	This novel spatiotemporal modeling approach provides accurate fine-scale predictions in multiple regions for four pollutants. We have generated participant-specific predictions for MESA Air to investigate health effects of long-term air pollution exposures. These successes highlight modeling advances that can be adopted more widely in modern cohort studies.

표-290. PubMed 논문번호 25415971의 내용 요약

구분	내용
PubMed ID	25415971
TITLE	Social disparities in heart disease risk and survivor bias among autoworkers: an examination based on survival models and g-estimation.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2014-102168
AUTHORS	Costello Sadie, Picciotto Sally, Rehkopf David H, Eisen Ellen A, Costello Sadie, Picciotto Sally, Rehkopf David H, Eisen Ellen A
OBJECTIVES	To examine gender and racial disparities in ischaemic heart disease (IHD) mortality related to metalworking fluid exposures and in the healthy worker survivor effect.
METHODS	A cohort of white and black men and women autoworkers in the USA was followed from 1941 to 1995 with quantitative exposure to respirable particulate matter from water-based metalworking fluids. Separate analyses used proportional hazards models and g-estimation.
RESULTS	The HR for IHD among black men was 3.29 (95% CI 1.49 to 7.31) in the highest category of cumulative synthetic fluid exposure. The HR for IHD among white women exposed to soluble fluid reached 2.44 (95% CI 0.96 to 6.22). However, no increased risk was observed among white men until we corrected for the healthy worker survivor effect. Results from g-estimation indicate that if white male cases exposed to soluble or synthetic fluid had been unexposed to that fluid type, then 1.59 and 1.20 years of life would have been saved on average, respectively.
CONCLUSIONS	We leveraged the strengths of two different analytic approaches to examine the IHD risks of metalworking fluids. All workers may have the same aetiological risk; however, black and female workers may experience more IHD from water-based metalworking fluid exposure because of a steeper exposure-response or weaker healthy worker survivor effect.

표-291. PubMed 논문번호 25440802의 내용 요약

구분	내용
PubMed ID	25440802
TITLE	Lack of association between chronic exposure to biomass fuel smoke and markers of right ventricular pressure overload at high altitude.
JOURNAL	American heart journal: 10.1016/j.ahj.2014.06.030
AUTHORS	Caravedo Maria A, Painschab Matthew S, Davila-Roman Victor G, De Ferrari Aldo, Gilman Robert H, Vasquez-Villar Angel D, Pollard Suzanne L, Miranda J Jaime, Checkley William
BACKGROUND	Chronic exposure to biomass fuel smoke has been implicated in the development of pulmonary hypertension and right ventricular pressure/volume overload through activation of inflammation, increase in vascular resistance, and endothelial dysfunction. We sought to compare N-terminal pro-B-type natriuretic peptide (NT-pro-BNP) and echocardiography-derived pulmonary artery systolic pressure (PASP) levels in a high-altitude population-based study in Peru with and without chronic exposure to biomass fuel smoke.
METHODS	NT-pro-BNP levels were measured in 519 adults (275 with and 244 without chronic exposure to biomass fuel smoke). Participants answered sociodemographics and clinical history questionnaires, underwent a clinical examination and blood testing for cardiopulmonary biomarkers. PASP was measured in a subgroup of 153 (31%) subjects.
RESULTS	The study group consisted of 280 men (54%) and 239 women (46%). Average age was 56 years and average body mass index was 27 kg/m(2). In multivariable analysis, there was no association between chronic exposure to biomass fuel smoke and NT-pro-BNP (P = .31) or PASP (P = .31). In the subgroup in which both NT-pro-BNP levels and PASP were measured, there was strong evidence of an association between these two variables (ρ = 0.24, 95% CI 0.09-0.39; P = .003). We found that age, high sensitivity C-reactive protein, being male, and systolic blood pressure were positively associated with NT-pro-BNP levels whereas body mass index, low-density/high-density lipoprotein ratio, and Homeostasis Model of Assessment-Insulin Resistance were negatively associated (all P ≤ .02).
CONCLUSIONS	In this population-based study in a high-altitude setting, neither NT-pro-BNP levels nor echocardiography-derived PASP were associated with chronic exposure to biomass fuel smoke.

표-292. PubMed 논문번호 25442690의 내용 요약

구분	내용
PubMed ID	25442690
TITLE	Effect of antenatal multiple micronutrient supplementation on anthropometry and blood pressure in mid-childhood in Nepal: follow-up of a double-blind randomised controlled trial.
JOURNAL	The Lancet. Global health: 10.1016/S2214-109X(14)70314-6
AUTHORS	Devakumar Delan, Chaube Shiva Shankar, Wells Jonathan C K, Saville Naomi M, Ayres Jon G, Manandhar Dharma S, Costello Anthony, Osrin David
BACKGROUND	In 2002-04, we did a randomised controlled trial in southern Nepal, and reported that children born to mothers taking multiple micronutrient supplements during pregnancy had a mean birthweight 77 g greater than children born to mothers taking iron and folic acid supplements. Children born to mothers in the study group were a mean 204 g heavier at 2·5 years of age and their systolic blood pressure was a mean 2·5 mm Hg lower than children born to mothers in the control group. We aimed to follow up the same children to mid-childhood (age 8·5 years) to investigate whether these differences would be sustained.
METHODS	For this follow-up study, we identified children from the original trial and measured anthropometry, body composition with bioelectrical impedance (with population-specific isotope calibration), blood pressure, and renal dimensions by ultrasound. We documented socioeconomic status, household food security, and air pollution. Main outcomes of the follow-up at 8 years were Z scores for weight-for-age, height-for-age, and body-mass index (BMI)-for-age according to WHO Child Growth Standards for children aged 5-19 years, and blood pressure. This study is registered with the International Standard Randomised Controlled Trial register, number ISRCTN88625934.
FINDINGS	Between Sept 21, 2011, and Dec 7, 2012, we assessed 841 children (422 in the control group and 419 in the intervention group). Unadjusted differences (intervention minus control) in Z scores were 0·05 for weight-for-age (95% CI −0·09 to 0·19), 0·02 in height-for-age (−0·10 to 0·15), and 0·04 in BMI-for-age (−0·09 to 0·18). We recorded no difference in blood pressure. Adjusted differences were similar for all outcomes.
INTERPRETATION	We recorded no differences in phenotype between children born to mothers who received antenatal multiple micronutrient or iron and folate supplements at age 8·5 years. Our findings did not extend to physiological differences or potential longer-term effects.
FUNDING	The Wellcome Trust.

표-293. PubMed 논문번호 25460628의 내용 요약

구분	내용
PubMed ID	25460628
TITLE	A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke.
JOURNAL	Environmental research: 10.1016/j.envres.2014.10.015
AUTHORS	Liu Jia C, Pereira Gavin, Uhl Sarah A, Bravo Mercedes A, Bell Michelle L, Liu Jia C, Pereira Gavin, Uhl Sarah A, Bravo Mercedes A, Bell Michelle L
BACKGROUND	Climate change is likely to increase the threat of wildfires, and little is known about how wildfires affect health in exposed communities. A better understanding of the impacts of the resulting air pollution has important public health implications for the present day and the future.
METHOD	We performed a systematic search to identify peer-reviewed scientific studies published since 1986 regarding impacts of wildfire smoke on health in exposed communities. We reviewed and synthesized the state of science of this issue including methods to estimate exposure, and identified limitations in current research.
RESULTS	We identified 61 epidemiological studies linking wildfire and human health in communities. The U.S. and Australia were the most frequently studied countries (18 studies on the U.S., 15 on Australia). Geographic scales ranged from a single small city (population about 55,000) to the entire globe. Most studies focused on areas close to fire events. Exposure was most commonly assessed with stationary air pollutant monitors (35 of 61 studies). Other methods included using satellite remote sensing and measurements from air samples collected during fires. Most studies compared risk of health outcomes between 1) periods with no fire events and periods during or after fire events, or 2) regions affected by wildfire smoke and unaffected regions. Daily pollution levels during or after wildfire in most studies exceeded U.S. EPA regulations. Levels of PM10, the most frequently studied pollutant, were 1.2 to 10 times higher due to wildfire smoke compared to non-fire periods and/or locations. Respiratory disease was the most frequently studied health condition, and had the most consistent results. Over 90% of these 45 studies reported that wildfire smoke was significantly associated with risk of respiratory morbidity.
CONCLUSION	Exposure measurement is a key challenge in current literature on wildfire and human health. A limitation is the difficulty of estimating pollution specific to wildfires. New methods are needed to separate air pollution levels of wildfires from those from ambient sources, such as transportation. The majority of studies found that wildfire smoke was associated with increased risk of respiratory and cardiovascular diseases. Children, the elderly and those with underlying chronic diseases appear to be susceptible. More studies on mortality and cardiovascular morbidity are needed. Further exploration with new methods could help ascertain the public health impacts of wildfires under climate change and guide mitigation policies.

표-294. PubMed 논문번호 25460637의 내용 요약

구분	내용
PubMed ID	25460637
TITLE	Systematic review and meta-analysis of the adverse health effects of ambient PM2.5 and PM10 pollution in the Chinese population.
JOURNAL	Environmental research: 10.1016/j.envres.2014.06.029
AUTHORS	Lu Feng, Xu Dongqun, Cheng Yibin, Dong Shaoxia, Guo Chao, Jiang Xue, Zheng Xiaoying, Lu Feng, Xu Dongqun, Cheng Yibin, Dong Shaoxia, Guo Chao, Jiang Xue, Zheng Xiaoying
INTRODUCTION	As the largest developing country, China has some of the worst air quality in the world. Heavy smog in January 2013 led to unprecedented public concern about the health impact of exposure to particulate matter. Conducting health impact assessments of particulate matter has thus become an urgent task for public health practitioners. Combined estimates of the health effects of exposure to particulate matter from quantitative reviews could provide vital information for epidemiology-based health impact assessments, but estimates for the Chinese population are limited.
METHODS	On December 31, 2013, we systematically searched the PubMed, Web of Science, and China National Knowledge Infrastructure databases using as keywords names of 127 major cities in Mainland China, Hong Kong, and Taiwan. From among the 1464 articles identified, 59 studies were manually screened. Random-effects or fixed-effects models were used to combine their risk estimates, the funnel plots with Egger test were performed to evaluate the publication bias and Meta regression were run to explore the association between exposure to particulate matter with aerodynamic diameters less than 10 and 2.5 □m (PM10 and PM2.5) and the resulting health effects by the Comprehensive Meta Analysis.
RESULTS	In terms of short-term effects, the combined excess risks of total non-accidental mortality, mortality due to cardiovascular disease, and mortality due to respiratory disease were 0.36% (95% confidence interval [95%CI]: 0.26%, 0.46%), 0.36% (95%CI: 0.24%, 0.49%), and 0.42% (95%CI: 0.28%, 0.55%), for each 10 μg/m(3) increase in PM10. A 10 μg/m(3) increase in PM2.5 was associated with a 0.40% (95%CI: 0.22%, 0.59%) increase in total non-accidental mortality, a 0.63% (95%CI: 0.35%, 0.91%) increase in mortality due to cardiovascular disease, and a 0.75% (95%CI: 01.39%, 1.11%) increase in mortality due to respiratory disease. For constituent-specific mortality, increases of 0.40-3.11% were associated with an increase of 10 ng/m(3) for nickel in PM. The summary estimate ranges of hospital utilization were 0.08% ~ 0.72% and -0.58% ~ 1.32% for a 10 μg/m(3) increase in PM10 and PM2.5. In terms of long-term effects, a 10 μg/m(3) increase of PM10 corresponded to 23-67% increase in the risk of mortality.
CONCLUSION	Short exposures to PM10 and PM2.5 are associated with increases in mortality, but evidence of constituent-associated health effects, long-term effects and morbidity in China is still inadequate.

표-295. PubMed 논문번호 25461424의 내용 요약

구분	내용
PubMed ID	25461424
TITLE	Short-term effects of particulate matter constituents on daily hospitalizations and mortality in five South-European cities: results from the MED-PARTICLES project.
JOURNAL	Environment international: 10.1016/j.envint.2014.11.011
AUTHORS	Basaga□a Xavier, Jacquemin B□n□dicte, Karanasiou Angeliki, Ostro Bart, Querol Xavier, Agis David, Alessandrini Ester, Alguacil Juan, Arti□ano Bego□a, Catrambone Maria, de la Rosa Jes□s D, D□az Julio, Faustini Annunziata, Ferrari Silvia, Forastiere Francesco, Katsouyanni Klea, Linares Cristina, Perrino Cinzia, Ranzi Andrea, Ricciardelli Isabella, Samoli Evangelia, Zauli-Sajani Stefano, Sunyer Jordi, Stafoggia Massimo, Basaga□a Xavier, Jacquemin B□n□dicte, Karanasiou Angeliki, Ostro Bart, Querol Xavier, Agis David, Alessandrini Ester, Alguacil Juan, Arti□ano Bego□a, Catrambone Maria, de la Rosa Jes□s D, D□az Julio, Faustini Annunziata, Ferrari Silvia, Forastiere Francesco, Katsouyanni Klea, Linares Cristina, Perrino Cinzia, Ranzi Andrea, Ricciardelli Isabella, Samoli Evangelia, Zauli-Sajani Stefano, Sunyer Jordi, Stafoggia Massimo
BACKGROUND	Few recent studies examined acute effects on health of individual chemical species in the particulate matter (PM) mixture, and most of them have been conducted in North America. Studies in Southern Europe are scarce. The aim of this study is to examine the relationship between particulate matter constituents and daily hospital admissions and mortality in five cities in Southern Europe.
METHODS	The study included five cities in Southern Europe, three cities in Spain: Barcelona (2003-2010), Madrid (2007-2008) and Huelva (2003-2010); and two cities in Italy: Rome (2005-2007) and Bologna (2011-2013). A case-crossover design was used to link cardiovascular and respiratory hospital admissions and total, cardiovascular and respiratory mortality with a pre-defined list of 16 PM10 and PM2.5 constituents. Lags 0 to 2 were examined. City-specific results were combined by random-effects meta-analysis.
RESULTS	Most of the elements studied, namely EC, SO4(2-), SiO2, Ca, Fe, Zn, Cu, Ti, Mn, V and Ni, showed increased percent changes in cardiovascular and/or respiratory hospitalizations, mainly at lags 0 and 1. The percent increase by one interquartile range (IQR) change ranged from 0.69% to 3.29%. After adjustment for total PM levels, only associations for Mn, Zn and Ni remained significant. For mortality, although positive associations were identified (Fe and Ti for total mortality; EC and Mg for cardiovascular mortality; and NO3(-) for respiratory mortality) the patterns were less clear.
CONCLUSIONS	The associations found in this study reflect that several PM constituents, originating from different sources, may drive previously reported results between PM and hospital admissions in the Mediterranean area.

표-296. PubMed 논문번호 25468504의 내용 요약

구분	내용
PubMed ID	25468504
TITLE	Spatial variation of heart failure and air pollution in Warwickshire, UK: an investigation of small scale variation at the ward-level.
JOURNAL	BMJ open: 10.1136/bmjopen-2014-006028
AUTHORS	Bennett Oscar, Kandala Ngianga-Bakwin, Ji Chen, Linnane John, Clarke Aileen
OBJECTIVES	To map using geospatial modelling techniques the morbidity and mortality caused by heart failure within Warwickshire to characterise and quantify any influence of air pollution on these risks.
DESIGN	Cross-sectional.
SETTING	Warwickshire, UK.
PARTICIPANTS	Data from all of the 105 current Warwickshire County wards were collected on hospital admissions and deaths due to heart failure.
RESULTS	In multivariate analyses, the presence of higher mono-nitrogen oxide (NOx) in a ward (3.35:1.89, 4.99), benzene (Ben) (31.9:8.36, 55.85) and index of multiple deprivation (IMD; 0.02: 0.01, 0.03), were consistently associated with a higher risk of heart failure morbidity. Particulate matter (Pm; -12.93: -20.41, -6.54) was negatively associated with the risk of heart failure morbidity. No association was found between sulfur dioxide (SO2) and heart failure morbidity. The risk of heart failure mortality was higher in wards with a higher NOx (4.30: 1.68, 7.37) and wards with more inhabitants 50+ years old (1.60: 0.47, 2.92). Pm was negatively associated (-14.69: -23.46, -6.50) with heart failure mortality. SO2, Ben and IMD scores were not associated with heart failure mortality. There was a prominent variation in heart failure morbidity and mortality risk across wards, the highest risk being in the regions around Nuneaton and Bedworth.
CONCLUSIONS	This study showed distinct spatial patterns in heart failure morbidity and mortality, suggesting the potential role of environmental factors beyond individual-level risk factors. Air pollution levels should therefore be taken into account when considering the wider determinants of public health and the impact that changes in air pollution might have on the health of a population.

표-297. PubMed 논문번호 25487196의 내용 요약

구분	내용
PubMed ID	25487196
TITLE	Effect of wood smoke exposure on vascular function and thrombus formation in healthy fire fighters.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-014-0062-4
AUTHORS	Hunter Amanda L, Unosson Jon, Bosson Jenny A, Langrish Jeremy P, Pourazar Jamshid, Raftis Jennifer B, Miller Mark R, Lucking Andrew J, Boman Christoffer, Nystr□m Robin, Donaldson Kenneth, Flapan Andrew D, Shah Anoop S V, Pung Louis, Sadiktsis Ioannis, Masala Silvia, Westerholm Roger, Sandstr□m Thomas, Blomberg Anders, Newby David E, Mills Nicholas L
BACKGROUND	Myocardial infarction is the leading cause of death in fire fighters and has been linked with exposure to air pollution and fire suppression duties. We therefore investigated the effects of wood smoke exposure on vascular vasomotor and fibrinolytic function, and thrombus formation in healthy fire fighters.
METHODS	In a double-blind randomized cross-over study, 16 healthy male fire fighters were exposed to wood smoke (~1 mg/m³ particulate matter concentration) or filtered air for one hour during intermittent exercise. Arterial pressure and stiffness were measured before and immediately after exposure, and forearm blood flow was measured during intra-brachial infusion of endothelium-dependent and -independent vasodilators 4-6 hours after exposure. Thrombus formation was assessed using the ex vivo Badimon chamber at 2 hours, and platelet activation was measured using flow cytometry for up to 24 hours after the exposure.
RESULTS	Compared to filtered air, exposure to wood smoke increased blood carboxyhaemoglobin concentrations (1.3% versus 0.8%; $P < 0.001$), but had no effect on arterial pressure, augmentation index or pulse wave velocity ($P > 0.05$ for all). Whilst there was a dose-dependent increase in forearm blood flow with each vasodilator ($P < 0.01$ for all), there were no differences in blood flow responses to acetylcholine, sodium nitroprusside or verapamil between exposures ($P > 0.05$ for all). Following exposure to wood smoke, vasodilatation to bradykinin increased ($P = 0.003$), but there was no effect on bradykinin-induced tissue-plasminogen activator release, thrombus area or markers of platelet activation ($P > 0.05$ for all).
CONCLUSIONS	Wood smoke exposure does not impair vascular vasomotor or fibrinolytic function, or increase thrombus formation in fire fighters. Acute cardiovascular events following fire suppression may be precipitated by exposure to other air pollutants or through other mechanisms, such as strenuous physical exertion and dehydration.

표-298. PubMed 논문번호 25487431의 내용 요약

구분	내용
PubMed ID	25487431
TITLE	Exposure to traffic-related air pollution during physical activity and acute changes in blood pressure, autonomic and micro-vascular function in women: a cross-over study.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-014-0070-4
AUTHORS	Weichenthal Scott, Hatzopoulou Marianne, Goldberg Mark S
BACKGROUND	Traffic-related air pollution may contribute to cardiovascular morbidity. In urban areas, exposures during physical activity are of interest owing to increased breathing rates and close proximity to vehicle emissions.
METHODS	We conducted a cross-over study among 53 healthy non-smoking women in Montreal, Canada during the summer of 2013. Women were exposed to traffic pollutants for 2-hours on three separate occasions during cycling on high and low-traffic routes as well as indoors. Personal air pollution exposures (PM(2.5), ultrafine particles (UFP), black carbon, NO_2, and O_3) were evaluated along each route and linear mixed-effects models with random subject intercepts were used to estimate the impact of air pollutants on acute changes in blood pressure, heart rate variability, and micro-vascular function in the hours immediately following exposure. Single and multi-pollutant models were examined and potential effect modification by mean regional air pollution concentrations (PM(2.5), NO_2, and O_3) was explored for the 24-hour and 5-day periods preceding exposure.
RESULTS	In total, 143 exposure routes were completed. Each interquartile increase (10,850/cm^3) in UFP exposure was associated with a 4.91% (95% CI: -9.31, -0.512) decrease in reactive hyperemia index (a measure of micro-vascular function) and each 24 ppb increase in O_3 exposure corresponded to a 2.49% (95% CI: 0.141, 4.84) increase in systolic blood pressure and a 3.26% (95% CI: 0.0117, 6.51) increase in diastolic blood pressure 3-hours after exposure. Personal exposure to PM(2.5) was associated with decreases in HRV measures reflecting parasympathetic modulation of the heart and regional PM(2.5) concentrations modified these relationships ($p < 0.05$). In particular, stronger inverse associations were observed when regional PM(2.5) was higher on the days prior to the study period. Regional PM(2.5) also modified the impact of personal O_3 on the standard deviation of normal to normal intervals (SDNN) ($p < 0.05$): a significant inverse relationship was observed when regional PM(2.5) was low prior to study periods and a significant positive relationship was observed when regional PM(2.5) was high.
CONCLUSION	Exposure to traffic pollution may contribute to acute changes in blood pressure, autonomic and micro-vascular function in women. Regional air pollution concentrations may modify the impact of these exposures on autonomic function.

표-299. PubMed 논문번호 25568517의 내용 요약

구분	내용
PubMed ID	25568517
TITLE	Air pollution in pristina, influence on cardiovascular hospital morbid
JOURNAL	Medical archives (Sarajevo, Bosnia and Herzegovina): 10.5455/medarh.2013.67.438-441
AUTHORS	Uk□haxhaj Antigona, Gjorgjev Dragan, Ramadani Maser, Krasniqi Gjergji Tahire, Zogaj Drita
INTRODUCTION	Numerous studies observed health effects of particulate air pollution. air quality is particularly bad in Pristina. The principal sources of contaminants are sulfur dioxide (SO2), nitrogen oxides NO and NO ozone (O3), lead (Pb), carbon dioxide (CO2), particulate matter (PM
OBJECTIVE	to investigate effects of concentrations of pollutants in ambient air or admissions for cardiovascular disease in UCCK- Pristina.
METHODS	Retrospective ecological study. During the three year analytical resea predict the potential benefit of decreasing for concentration of PM 2. were measured in two station in Pristina. The study population consi all hospitalization patient in intern clinic for 2010,2011 and 2012 yea pollution measurements will be used by KHMI data for the year of 20 and 2012 for the municipality of.
PRISTINA IN THE MEASUREMENTS POINT IN	KHMI-MESP which is equipped with automatic analyzer- Air Com Monitoring System (Version 2.2) recordum MESSTECHNIK GmbH. Statistical data processing will be done with SPSS 17.0 statistical pacl
RESULTS	Based on the results obtained during the study period concentrated P higher level than standards value. The results showed that the numbe hospital admissions for cardiovascular disease are positively correlate concentration pollutants. Results show clear seasonal variation in the of PM on hospital admissions in Kosovo. The study period was shor mean daily admissions for cardiovascular illnesses were quite large.
CONCLUSION	The main source for air pollution was coal-burned power plant and (old vehicles) in Kosovo.

표-300. PubMed 논문번호 25577313의 내용 요약

구분	내용
PubMed ID	25577313
TITLE	Air pollution and activation of mobile medical team for out-of-hospital cardiac arrest.
JOURNAL	The American journal of emergency medicine: 10.1016/j.ajem.2014.12.007
AUTHORS	Pradeau Catherine, Rondeau Virginie, L□v□que Emilie, Guernion Pierre-Yves, Tentillier Eric, Thicoip□ Michel, Brochard Patrick, Pradeau Catherine, Rondeau Virginie, L□v□que Emilie, Guernion Pierre-Yves, Tentillier Eric, Thicoip□ Michel, Brochard Patrick
BACKGROUND	The association between air pollution exposure and cardiovascular events is well established, and the effect of short-term exposure on out-of-hospital cardiac arrest (OHCA) has received some attention. The effect of air pollution exposure and the activation of mobile intensive care units (MICUs) for cardiac arrest have never been studied.
OBJECTIVE	We analyzed associations between air pollutants and MICU activation for OHCA.
METHOD	This is a retrospective study including 4558 patients with OHCA and MICU activation from 2007 to 2012. A time-stratified case crossover design was used. Particulate matter (PM) of median aerodynamic diameter less than 2.5 μm (PM2.5), less than 10 μm, and ozone were the 3 main pollutants used to determine the effects of pollution exposure on the event.
RESULTS	A daily average increase of 27.6 μg/m(3) in ozone was associated with an increase of MICU activation for OHCA the following day (odds ratio [OR], 1.13; 95% confidence interval [CI], 1.03-1.22). For women, a daily average increase of 27.6 μg/m(3) in ozone was associated with an increase of MICU activation for OHCA the following day (OR, 1.19; 95% CI, 1.01-1.37). An hourly average increase of 10.5 μg/m(3) in PM2.5 was associated with an increase of MICU activation for OHCA in the current hour (OR, 1.11; 95% CI, 1.02-1.19). For men, an increase in PM2.5 was associated with an increase in MICU activation for OHCA the current hour (OR, 1.10; 95% CI, 1.01-1.20). No association was found with PM of median aerodynamic diameter less than 10 μm.
CONCLUSION	An association was found between air pollution and MICU activation for OHCA (ozone and PM2.5).

표-301. PubMed 논문번호 25594797의 내용 요약

구분	내용
PubMed ID	25594797
TITLE	Reproducibility of oscillometrically measured arterial stiffness indices: Results of the SAPALDIA 3 cohort study.
JOURNAL	Scandinavian journal of clinical and laboratory investigation: 10.3109/00365513.2014.993692
AUTHORS	Endes Simon, Caviezel Seraina, Dratva Julia, Schaffner Emmanuel, Schindler Christian, Rothe Thomas, Rochat Thierry, K□nzli Nino, Probst-Hensch Nicole, Schmidt-Trucks□ss Arno
BACKGROUND	There is an increasing interest in oscillometric arterial stiffness measurement for cardiovascular risk stratification. We assessed reproducibility of the cuff-based arterial stiffness measures cardio-ankle vascular index (CAVI), brachial-ankle pulse wave velocity (baPWV) and peripheral augmentation index (pAI) in a subsample of the second follow-up of the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases in Adults (SAPALDIA 3).
METHODS	CAVI, baPWV and pAI were measured twice within 90 days in a representative subsample (n = 105) of SAPALDIA 3 with a mean age of 63 years (52.4% female).
RESULTS	The mean coefficient of variation for CAVI was 4.4%, baPWV 3.9%, and pAI 7.4%. The intraclass correlation coefficient ranged from 0.6 for pAI to 0.8 for CAVI, and 0.9 for baPWV. The mixed linear model revealed that 68.7%/80.1%/55.0% of the CAVI/baPWV/pAI variance was accounted for by the subject, 5.2%/8.1%/ < 0.01% by the fieldworker, 6.7%/7.8%/28.5% by variation between measurement days, and 19.4%/4%/16.5% by measurement error. Bland-Altman plots showed no particular dispersion patterns except for pAI.
CONCLUSIONS	Oscillometric arterial stiffness measurement by CAVI and baPWV has proved to be highly reproducible in Caucasians. Results of the pAI showed lower reproducibility. CAVI and baPWV can be implemented as easy-to-apply arterial stiffness measures in population wide cardiovascular risk assessment in Caucasians.

표-302. PubMed 논문번호 25600221의 내용 요약

구분	내용
PubMed ID	25600221
TITLE	Cardiovascular effects of ozone in healthy subjects with and without deletion of glutathione-S-transferase M1.
JOURNAL	Inhalation toxicology: 10.3109/08958378.2014.996272
AUTHORS	Frampton Mark W, Pietropaoli Anthony, Dentler Michael, Chalupa David, Little Erika L, Stewart Judith, Frasier Lauren, Oakes David, Wiltshire Jelani, Vora Rathin, Utell Mark J
CONTEXT	Exposure to ozone has acute respiratory effects, but few human clinical studies have evaluated cardiovascular effects.
OBJECTIVE	We hypothesized that ozone exposure alters pulmonary and systemic vascular function, and cardiac function, with more pronounced effects in subjects with impaired antioxidant defense from deletion of the glutathione-S-transferase M1 gene (GSTM1 null).
METHODS	Twenty-four young, healthy never-smoker subjects (12 GSTM1 null) inhaled filtered air, 100 ppb ozone and 200 ppb ozone for 3 h, with intermittent exercise, in a double-blind, randomized, crossover fashion. Exposures were separated by at least 2 weeks. Vital signs, spirometry, arterial and venous blood nitrite levels, impedance cardiography, peripheral arterial tonometry, estimation of pulmonary capillary blood volume (Vc), and blood microparticles and platelet activation were measured at baseline and during 4 h after each exposure.
RESULTS	Ozone inhalation decreased lung function immediately after exposure (mean ± standard error change in FEV1, air: −0.03 ± 0.04 L; 200 ppb ozone: −0.30 ± 0.07 L; $p < 0.001$). The immediate post-exposure increase in blood pressure, caused by the final 15-min exercise period, was blunted by 200 ppb ozone exposure (mean ± standard error change for air: 16.7 ± 2.6 mmHg; 100 ppb ozone: 14.5 ± 2.4 mmHg; 200 ppb ozone: 8.5 ± 2.5 mmHg; $p = 0.02$). We found no consistent effects of ozone on any other measure of cardiac or vascular function. All results were independent of the GSTM1 genotype.
CONCLUSIONS	We did not find convincing evidence for early acute adverse cardiovascular consequences of ozone exposure in young healthy adults. The ozone-associated blunting of the blood pressure response to exercise is of unclear clinical significance.

표-303. PubMed 논문번호 25604176의 내용 요약

구분	내용
PubMed ID	25604176
TITLE	Effect of particulate matter less than 10□m (PM10) on mortality in Bogota, Colombia: a time-series analysis, 1998-2006.
JOURNAL	Salud publica de Mexico: 10.21149/spm.v56i4.7356
AUTHORS	Blanco-Becerra Luis Camilo, Miranda-Soberanis V□ctor, Hern□ndez-Cadena Leticia, Barraza-Villarreal Albino, Junger Washington, Hurtado-D□az Magali, Romieu Isabelle
OBJECTIVE	To analyze the association between daily mortality from different causes and acute exposure to particulate matter less than 10 microns in aerodynamic diameter (PM10), in Bogota, Colombia.
MATERIALS AND METHODS	A time-series ecological study was conducted from 1998 to 2006. The association between mortality (due to different causes) and exposure was analyzed using single and distributed lag models and adjusting for potential confounders.
RESULTS	For all ages, the cumulative effect of acute mortality from all causes and respiratory causes increased 0.71% (95%CI 0.46-0.96) and 1.43% (95%CI 0.85-2.00), respectively, per 10□g/m^3 increment in daily average PM10 with a lag of three days before death. Cumulative effect of mortality from cardiovascular causes was -0.03% (95%CI -0.49-0.44%) with the same lag.
CONCLUSIONS	The results suggest an association between an increase in PM10 concentrations and acute mortality from all causes and respiratory causes.

표-304. PubMed 논문번호 25616223의 내용 요약

구분	내용
PubMed ID	25616223
TITLE	Effects of ambient coarse, fine, and ultrafine particles and their biological constituents on systemic biomarkers: a controlled human exposure study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408387
AUTHORS	Liu Ling, Urch Bruce, Poon Raymond, Szyszkowicz Mieczyslaw, Speck Mary, Gold Diane R, Wheeler Amanda J, Scott James A, Brook Jeffrey R, Thorne Peter S, Silverman Frances S
BACKGROUND	Ambient coarse, fine, and ultrafine particles have been associated with mortality and morbidity. Few studies have compared how various particle size fractions affect systemic biomarkers.
OBJECTIVES	We examined changes of blood and urinary biomarkers following exposures to three particle sizes.
METHODS	Fifty healthy nonsmoking volunteers, mean age of 28 years, were exposed to coarse (2.5-10 μm; mean, 213 μg/m3) and fine (0.15-2.5 μm; mean, 238 μg/m3) concentrated ambient particles (CAPs), and filtered ambient and/or medical air. Twenty-five participants were exposed to ultrafine CAP (< 0.3 μm; mean, 136 μg/m3) and filtered medical air. Exposures lasted 130 min, separated by ≥ 2 weeks. Blood/urine samples were collected preexposure and 1 hr and 21 hr postexposure to determine blood interleukin-6 and C-reactive protein (inflammation), endothelin-1 and vascular endothelial growth factor (VEGF; vascular mediators), and malondialdehyde (lipid peroxidation); as well as urinary VEGF, 8-hydroxy-deoxy-guanosine (DNA oxidation), and malondialdehyde. Mixed-model regressions assessed pre- and postexposure differences.
RESULTS	One hour postexposure, for every 100- μg/m3 increase, coarse CAP was associated with increased blood VEGF (2.41 pg/mL; 95% CI: 0.41, 4.40) in models adjusted for O3, fine CAP with increased urinary malondialdehyde in single- (0.31 nmol/mg creatinine; 95% CI: 0.02, 0.60) and two-pollutant models, and ultrafine CAP with increased urinary 8-hydroxydeoxyguanosine in single- (0.69 ng/mg creatinine; 95% CI: 0.09, 1.29) and two-pollutant models, lasting < 21 hr. Endotoxin was significantly associated with biomarker changes similar to those found with CAPs.
CONCLUSIONS	Ambient particles with various sizes/constituents may influence systemic biomarkers differently. Endotoxin in ambient particles may contribute to vascular mediator changes and oxidative stress.

표-305. PubMed 논문번호 25616258의 내용 요약

구분	내용
PubMed ID	25616258
TITLE	MicroRNAs as potential signatures of environmental exposure or effect: a systematic review.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408459
AUTHORS	Vrijens Karen, Bollati Valentina, Nawrot Tim S
BACKGROUND	The exposome encompasses all life-course environmental exposures from the prenatal period onward that influence health. MicroRNAs (miRNAs) are interesting entities within this concept as markers and causation of disease. MicroRNAs are short oligonucleotide sequences that can interact with several mRNA targets.
OBJECTIVES	We reviewed the current state of the field on the potential of using miRNAs as biomarkers for environmental exposure. We investigated miRNA signatures in response to all types of environmental exposure to which a human can be exposed, including cigarette smoke, air pollution, nanoparticles, and diverse chemicals; and we examined the health conditions for which the identified miRNAs have been reported (i.e., cardiovascular disease, cancer, and diabetes).
METHODS	We searched the PubMed and ScienceDirect databases to identify relevant studies.
RESULTS	For all exposures incorporated in this review, 27 miRNAs were differentially expressed in at least two independent studies. miRNAs that had expression alterations associated with smoking observed in multiple studies are miR-21, miR-34b, miR-125b, miR-146a, miR-223, and miR-340; and those miRNAs that were observed in multiple air pollution studies are miR-9, miR-10b, miR-21, miR-128, miR-143, miR-155, miR-222, miR-223, and miR-338. We found little overlap among in vitro, in vivo, and human studies between miRNAs and exposure. Here, we report on disease associations for those miRNAs identified in multiple studies on exposure.
CONCLUSIONS	miRNA changes may be sensitive indicators of the effects of acute and chronic environmental exposure. Therefore, miRNAs are valuable novel biomarkers for exposure. Further studies should elucidate the role of the mediation effect of miRNA between exposures and effect through all stages of life to provide a more accurate assessment of the consequences of miRNA changes.

표-306. PubMed 논문번호 25625237의 내용 요약

구분	내용
PubMed ID	25625237
TITLE	A study of the combined effects of physical activity and air pollution on mortality in elderly urban residents: the Danish Diet, Cancer, and Health Cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408698
AUTHORS	Andersen Zorana Jovanovic, de Nazelle Audrey, Mendez Michelle Ann, Garcia-Aymerich Judith, Hertel Ole, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole, Nieuwenhuijsen Mark J
BACKGROUND	Physical activity reduces, whereas exposure to air pollution increases, the risk of premature mortality. Physical activity amplifies respiratory uptake and deposition of air pollutants in the lung, which may augment acute harmful effects of air pollution during exercise.
OBJECTIVES	We aimed to examine whether benefits of physical activity on mortality are moderated by long-term exposure to high air pollution levels in an urban setting.
METHODS	A total of 52,061 subjects (50-65 years of age) from the Danish Diet, Cancer, and Health cohort, living in Aarhus and Copenhagen, reported data on physical activity in 1993-1997 and were followed until 2010. High exposure to air pollution was defined as the upper 25th percentile of modeled nitrogen dioxide (NO2) levels at residential addresses. We associated participation in sports, cycling, gardening, and walking with total and cause-specific mortality by Cox regression, and introduced NO2 as an interaction term.
RESULTS	In total, 5,534 subjects died: 2,864 from cancer, 1,285 from cardiovascular disease, 354 from respiratory disease, and 122 from diabetes. Significant inverse associations of participation in sports, cycling, and gardening with total, cardiovascular, and diabetes mortality were not modified by NO2. Reductions in respiratory mortality associated with cycling and gardening were more pronounced among participants with moderate/low NO2 [hazard ratio (HR) = 0.55; 95% CI: 0.42, 0.72 and 0.55; 95% CI: 0.41, 0.73, respectively] than with high NO2 exposure (HR = 0.77; 95% CI: 0.54, 1.11 and HR = 0.81; 95% CI: 0.55, 1.18, p-interaction = 0.09 and 0.02, respectively).
CONCLUSIONS	In general, exposure to high levels of traffic-related air pollution did not modify associations, indicating beneficial effects of physical activity on mortality. These novel findings require replication in other study populations.

표-307. PubMed 논문번호 25625652의 내용 요약

구분	내용
PubMed ID	25625652
TITLE	Prenatal air pollution exposure and newborn blood pressure.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307419
AUTHORS	van Rossem Lenie, Rifas-Shiman Sheryl L, Melly Steven J, Kloog Itai, Luttmann-Gibson Heike, Zanobetti Antonella, Coull Brent A, Schwartz Joel D, Mittleman Murray A, Oken Emily, Gillman Matthew W, Koutrakis Petros, Gold Diane R
BACKGROUND	Air pollution exposure has been associated with increased blood pressure in adults.
OBJECTIVE	We examined associations of antenatal exposure to ambient air pollution with newborn systolic blood pressure (SBP).
METHODS	We studied 1,131 mother-infant pairs in a Boston, Massachusetts, area pre-birth cohort. We calculated average exposures by trimester and during the 2 to 90 days before birth for temporally resolved fine particulate matter (≤ 2.5 μm; PM2.5), black carbon (BC), nitrogen oxides, nitrogen dioxide, ozone (O3), and carbon monoxide measured at stationary monitoring sites, and for spatiotemporally resolved estimates of PM2.5 and BC at the residence level. We measured SBP at a mean age of 30 ± 18 hr with an automated device. We used mixed-effects models to examine associations between air pollutant exposures and SBP, taking into account measurement circumstances; child's birth weight; mother's age, race/ethnicity, socioeconomic position, and third-trimester BP; and time trend. Estimates represent differences in SBP associated with an interquartile range (IQR) increase in each pollutant.
RESULTS	Higher mean PM2.5 and BC exposures during the third trimester were associated with higher SBP (e.g., 1.0 mmHg; 95% CI: 0.1, 1.8 for a 0.32- μg/m3 increase in mean 90-day residential BC). In contrast, O3 was negatively associated with SBP (e.g., -2.3 mmHg; 95% CI: -4.4, -0.2 for a 13.5-ppb increase during the 90 days before birth).
CONCLUSIONS	Exposures to PM2.5 and BC in late pregnancy were positively associated with newborn SBP, whereas O3 was negatively associated with SBP. Longitudinal follow-up will enable us to assess the implications of these findings for health during later childhood and adulthood.

표-308. PubMed 논문번호 25625785의 내용 요약

구분	내용
PubMed ID	25625785
TITLE	Air pollution and atherosclerosis: a cross-sectional analysis of four European cohort studies in the ESCAPE study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1307711
AUTHORS	Perez Laura, Wolf Kathrin, Hennig Frauke, Penell Johanna, Basaga□a Xavier, Foraster Maria, Aguilera Inmaculada, Agis David, Beelen Rob, Brunekreef Bert, Cyrys Josef, Fuks Kateryna B, Adam Martin, Baldassarre Damiano, Cirach Marta, Elosua Roberto, Dratva Julia, Hampel Regina, Koenig Wolfgang, Marrugat Jaume, de Faire Ulf, Pershagen G□ran, Probst-Hensch Nicole M, de Nazelle Audrey, Nieuwenhuijsen Mark J, Rathmann Wolfgang, Rivera Marcela, Seissler Jochen, Schindler Christian, Thiery Joachim, Hoffmann Barbara, Peters Annette, K□nzli Nino
BACKGROUND	In four European cohorts, we investigated the cross-sectional association between long-term exposure to air pollution and intima-media thickness of the common carotid artery (CIMT), a preclinical marker of atherosclerosis.
METHODS	Individually assigned levels of nitrogen dioxide, nitrogen oxides, particulate matter ≤ 2.5 μm (PM2.5), absorbance of PM2.5 (PM2.5abs), PM10, PMcoarse, and two indicators of residential proximity to highly trafficked roads were obtained under a standard exposure protocol (European Study of Cohorts for Air Pollution Effects-ESCAPE study) in the Stockholm area (Sweden), the Ausburg and Ruhr area (Germany), and the Girona area (Spain). We used linear regression and meta-analyses to examine the association between long-term exposure to air pollution and CIMT.
RESULTS	The meta-analysis with 9,183 individuals resulted in an estimated increase in CIMT (geometric mean) of 0.72% (95% CI: -0.65%, 2.10%) per 5- μg/m3 increase in PM2.5 and 0.42% (95% CI: -0.46%, 1.30%) per 10-5/m increase in PM2.5abs. Living in proximity to high traffic was also positively but not significantly associated with CIMT. Meta-analytic estimates for other pollutants were inconsistent. Results were similar across different adjustment sets and sensitivity analyses. In an extended meta-analysis for PM2.5 with three other previously published studies, a 0.78% (95% CI: -0.18%, 1.75%) increase in CIMT was estimated for a 5- μg/m3 contrast in PM2.5.
CONCLUSIONS	Using a standardized exposure and analytical protocol in four European cohorts, we found that cross-sectional associations between CIMT and the eight ESCAPE markers of long-term residential air pollution exposure did not reach statistical significance. The additional meta-analysis of CIMT and PM2.5 across all published studies also was positive but not significant.

표-309. PubMed 논문번호 25628407의 내용 요약

구분	내용
PubMed ID	25628407
TITLE	Cardiac autonomic dysfunction: particulate air pollution effects are modulated by epigenetic immunoregulation of Toll-like receptor 2 and dietary flavonoid intake.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.114.001423
AUTHORS	Zhong Jia, Colicino Elena, Lin Xinyi, Mehta Amar, Kloog Itai, Zanobetti Antonella, Byun Hyang-Min, Bind Marie-Ab□le, Cantone Laura, Prada Diddier, Tarantini Letizia, Trevisi Letizia, Sparrow David, Vokonas Pantel, Schwartz Joel, Baccarelli Andrea A
BACKGROUND	Short-term fine particles (PM(2.5)) exposure is associated with reduced heart rate variability, a strong predictor of cardiac mortality among older people. Identifying modifiable factors that confer susceptibility is essential for intervention. We evaluated whether Toll-like receptor 2 (TLR2) methylation, a reversible immune-epigenetic process, and its dietary modulation by flavonoids and methyl nutrients, modify susceptibility to heart rate variability effects following PM(2.5) exposure.
METHODS AND RESULTS	We measured heart rate variability and PM(2.5) repeatedly over 11 years (1275 total observations) among 573 elderly men from the Normative Aging Study. Blood TLR2 methylation was analyzed using pyrosequencing. Daily flavonoid and methyl nutrients intakes were assessed through the Food Frequency Questionnaire (FFQ). Every 10 μg/m(3) increase in 48-hour PM(2.5) moving average was associated with 7.74% (95% CI: -1.21% to 15.90%; P=0.09), 7.46% (95% CI: 0.99% to 13.50%; P=0.02), 14.18% (95% CI: 1.14% to 25.49%; P=0.03), and 12.94% (95% CI: -2.36% to 25.96%; P=0.09) reductions in root mean square of successive differences, standard deviation of normal-to-normal intervals, low-frequency power, and high-frequency power, respectively. Higher TLR2 methylation exacerbated the root mean square of successive differences, standard deviation of normal-to-normal intervals, low-frequency, and high-frequency reductions associated with heightened PM2.5 (P(interaction)=0.006, 0.03, 0.05, 0.04, respectively). Every interquartile-range increase in flavonoid intake was associated with 5.09% reduction in mean TLR2 methylation (95% CI: 0.12% to 10.06%; P=0.05) and counteracted the effects of PM2.5 on low frequency (P(interaction)=0.05). No significant effect of methyl nutrients on TLR2 methylation was observed.
CONCLUSIONS	Higher TLR2 methylation may confer susceptibility to adverse cardiac autonomic effects of PM2.5 exposure in older individuals. Higher flavonoid intake may attenuate these effects, possibly by decreasing TLR2 methylation.

표-310. PubMed 논문번호 25633926의 내용 요약

구분	내용
PubMed ID	25633926
TITLE	Associations of mortality with long-term exposures to fine and ultrafine particles, species and sources: results from the California Teachers Study Cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408565
AUTHORS	Ostro Bart, Hu Jianlin, Goldberg Debbie, Reynolds Peggy, Hertz Andrew, Bernstein Leslie, Kleeman Michael J
BACKGROUND	Although several cohort studies report associations between chronic exposure to fine particles (PM2.5) and mortality, few have studied the effects of chronic exposure to ultrafine (UF) particles. In addition, few studies have estimated the effects of the constituents of either PM2.5 or UF particles.
METHODS	We used a statewide cohort of > 100,000 women from the California Teachers Study who were followed from 2001 through 2007. Exposure data at the residential level were provided by a chemical transport model that computed pollutant concentrations from > 900 sources in California. Besides particle mass, monthly concentrations of 11 species and 8 sources or primary particles were generated at 4-km grids. We used a Cox proportional hazards model to estimate the association between the pollutants and all-cause, cardiovascular, ischemic heart disease (IHD), and respiratory mortality.
RESULTS	We observed statistically significant ($p < 0.05$) associations of IHD with PM2.5 mass, nitrate, elemental carbon (EC), copper (Cu), and secondary organics and the sources gas- and diesel-fueled vehicles, meat cooking, and high-sulfur fuel combustion. The hazard ratio estimate of 1.19 (95% CI: 1.08, 1.31) for IHD in association with a 10-μg/m3 increase in PM2.5 is consistent with findings from the American Cancer Society cohort. We also observed significant positive associations between IHD and several UF components including EC, Cu, metals, and mobile sources.
CONCLUSIONS	Using an emissions-based model with a 4-km spatial scale, we observed significant positive associations between IHD mortality and both fine and ultrafine particle species and sources. Our results suggest that the exposure model effectively measured local exposures and facilitated the examination of the relative toxicity of particle species.

표-311. PubMed 논문번호 25663064의 내용 요약

구분	내용
PubMed ID	25663064
TITLE	Air Pollution and Emergency Department Visits for Hypertension in Edmonton and Calgary, Canada: A Case-Crossover Study.
JOURNAL	American journal of hypertension: 10.1093/ajh/hpu302
AUTHORS	Brook Robert D, Kousha Termeh
BACKGROUND	Ambient air pollutant exposures have been associated with a wide variety of cardiovascular events; however, few studies have evaluated their impact upon acute emergency department (ED) visits for hypertension.
METHODS	The purpose of this study was to examine the associations between ED visits for hypertension and ambient air pollution concentrations among 6,532 patients during the period of January 2010 to December 2011 in Edmonton and Calgary, Alberta, Canada. The associations were evaluated using a case-crossover design.
RESULTS	Odds ratios and their 95% confidence interval have been calculated for 1 unit increase in their interquartile range for lags (the time between air pollutant measurement and exposure-response) 0-8 days. During the cold season, statistically significant positive results were observed for SO2 among lag days 4-6 and 8 for females and lag days 5 and 6 for males. Moreover, statistically significant positive results were observed for NO2 on lag day 7 for females and for PM2.5 on lag days 5 and 7, for females and lag day 6 for males. During the warm season, statistically significant positive results were observed for O3 on lag days 3 and 4 and for SO2 on lag days 2 and 8 for females.
CONCLUSIONS	These findings support the hypothesis that recent exposures to ambient levels of several air pollutants can be capable of elevating blood pressure to a clinically significant extent such that it leads to ED visits for hypertension.

표-312. PubMed 논문번호 25691484의 내용 요약

구분	내용
PubMed ID	25691484
TITLE	Adult cardiopulmonary mortality and indoor air pollution: a 10-year retrospective cohort study in a low-income rural setting.
JOURNAL	Global heart: 10.1016/j.gheart.2012.06.008
AUTHORS	Alam Dewan S, Chowdhury Muhammad Ashique H, Siddiquee Ali Tanweer, Ahmed Shyfuddin, Hossain Mohammad Didar, Pervin Sonia, Streatfield Kim, Cravioto Alejandro, Niessen Louis W
BACKGROUND	Indoor air pollution (IAP) due to solid fuel use is a major risk factor of respiratory and cardiovascular mortality and morbidity. Rural Matlab in Bangladesh has been partly supplied with natural gas since the early 1990s, which offered a natural experiment to investigate the long-term impact of IAP on cardiopulmonary mortality.
OBJECTIVE	This study sought to compare adult cardiopulmonary mortality in relation to household fuel type as a surrogate for exposure to indoor air pollution.
STUDY DESIGN	This was a retrospective cohort study. We identified all households in 11 villages in Matlab, Bangladesh, and categorized them as either supplied with natural gas or using solid fuel for cooking or heating since January 1, 2001. Cause-specific mortality data including cardiopulmonary deaths were obtained through verbal autopsy as part of a permanent surveillance. Person-years (PYs) of exposure were computed from baseline until the event. Subjects with missing information on cause of death, outward migration, or on fuel type were excluded. Event rates for each fuel category were calculated as well as the relative risk of dying with 95% confidence intervals (CI).
SETTING	Rural Matlab, Bangladesh.
PATIENTS	Adults 18 years of age or older.
OUTCOME MEASURE	Death from cardiopulmonary diseases over a 10-year period.
FINDINGS	In total, 946 cardiopulmonary deaths occurred with 884 in the solid-fuel and 62 in the gas-supplied households (n=7,565 and n=508, respectively) over the 10-year period. Cardiopulmonary death rate was 6.2 per 1,000 PYs in the solid-fuel group and 5.3 per 1,000 PYs in people living in households using gas. Mortality due to cardiovascular event was 5.1 and 4.8 per 1,000 PY in people from the solid-fuel and gas-supplied households, respectively, and the incident rate ratio was 1.07 (95% CI: 0.82 to 1.41). Mortality due to respiratory disease was 1.2 and 0.5 per 1,000 PYs in the solid-fuel and gas-supplied groups, respectively, and the incident rate ratio was 2.26 (95% CI: 1.02 to 4.99).
INTERPRETATION	Household solid-fuel use is associated with increased respiratory mortality and nonsignificantly increased risk of cardiovascular mortality. Reduction of exposure to pollution due to in-household solid-fuel use is likely to improve survival in Bangladeshi and similar populations.

표-313. PubMed 논문번호 25701728의 내용 요약

구분	내용
PubMed ID	25701728
TITLE	Mortality and morbidity in a population exposed to multiple sources of air pollution: A retrospective cohort study using air dispersion models.
JOURNAL	Environmental research: 10.1016/j.envres.2014.10.036
AUTHORS	Ancona Carla, Badaloni Chiara, Mataloni Francesca, Bolignano Andrea, Bucci Simone, Cesaroni Giulia, Sozzi Roberto, Davoli Marina, Forastiere Francesco
BACKGROUND AND AIMS	A landfill, an incinerator, and a refinery plant have been operating since the early 1960s in a contaminated site located in the suburb of Rome (Italy). To evaluate their potential health effects, a population-based retrospective cohort study was conducted using dispersion modeling for exposure assessment.
METHODS	A fixed cohort was enrolled in the Rome Longitudinal Study in 2001, mortality and hospitalizations were followed-up until 2010. Exposure assessments to the landfill (H2S), the incinerator (PM10), and the refinery plant (SOX) were performed for each subject using a Lagrangian dispersion model. Individual and small-area variables were available (including exposures levels to NO2 from traffic and diesel trucks). Cox regression analysis was performed (hazard ratios, HRs, 95% CI) using linear terms for the exposures (5th-95th percentiles difference). Single and bi-pollutant models were run.
RESULTS	The cohort included 85,559 individuals. The estimated annual average exposures levels were correlated. H2S from the landfill was associated with cardiovascular hospital admissions in both genders (HR 1.04 95% CI 1.00-1.09 in women); PM10 from the incinerator was associated with pancreatic cancer mortality in both genders (HR 1.40 95% CI 1.03-1.90 in men, HR 1.47 95% CI 1.12-1.93 in women) and with breast morbidity in women (HR 1.13 95% CI 1.00-1.27). SOx from the refinery was associated with laryngeal cancer mortality in women (HR 4.99 95% CI 1.64-15.9) and respiratory hospital admissions (HR 1.13 95% CI 1.01-1.27).
CONCLUSIONS	We found an association of the pollution sources with some cancer forms and cardio-respiratory diseases. Although there was a high correlation between the estimated exposures, an indication of specific effects from the different sources emerged.

표-314. PubMed 논문번호 25710246의 내용 요약

구분	내용
PubMed ID	25710246
TITLE	Long-term exposure to air pollution and markers of inflammation, coagulation, and endothelial activation: a repeat-measures analysis in the Multi-Ethnic Study of Atherosclerosis (MESA).
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000267
AUTHORS	Hajat Anjum, Allison Matthew, Diez-Roux Ana V, Jenny Nancy Swords, Jorgensen Neal W, Szpiro Adam A, Vedal Sverre, Kaufman Joel D
BACKGROUND	Air pollution is associated with cardiovascular disease, and systemic inflammation may mediate this effect. We assessed associations between long- and short-term concentrations of air pollution and markers of inflammation, coagulation, and endothelial activation.
METHODS	We studied participants from the Multi-Ethnic Study of Atherosclerosis from 2000 to 2012 with repeat measures of serum C-reactive protein (CRP), interleukin-6 (IL-6), fibrinogen, D-dimer, soluble E-selectin, and soluble Intercellular Adhesion Molecule-1. Annual average concentrations of ambient fine particulate matter (PM2.5), individual-level ambient PM2.5 (integrating indoor concentrations and time-location data), oxides of nitrogen (NOx), nitrogen dioxide (NO2), and black carbon were evaluated. Short-term concentrations of PM2.5 reflected the day of blood draw, day prior, and averages of prior 2-, 3-, 4-, and 5-day periods. Random-effects models were used for long-term exposures and fixed effects for short-term exposures. The sample size was between 9,000 and 10,000 observations for CRP, IL-6, fibrinogen, and D-dimer; approximately 2,100 for E-selectin; and 3,300 for soluble Intercellular Adhesion Molecule-1.
RESULTS	After controlling for confounders, 5 □g/m increase in long-term ambient PM2.5 was associated with 6% higher IL-6 (95% confidence interval = 2%, 9%), and 40 parts per billion increase in long-term NOx was associated with 7% (95% confidence interval = 2%, 13%) higher level of D-dimer. PM2.5 measured at day of blood draw was associated with CRP, fibrinogen, and E-selectin. There were no other positive associations between blood markers and short- or long-term air pollution.
CONCLUSIONS	These data are consistent with the hypothesis that long-term exposure to air pollution is related to some markers of inflammation and fibrinolysis.

표-315. PubMed 논문번호 25748169의 내용 요약

구분	내용
PubMed ID	25748169
TITLE	Long-Term Air Pollution Exposure and Blood Pressure in the Sister Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408125
AUTHORS	Chan Stephanie H, Van Hee Victor C, Bergen Silas, Szpiro Adam A, DeRoo Lisa A, London Stephanie J, Marshall Julian D, Kaufman Joel D, Sandler Dale P
BACKGROUND	Exposure to air pollution has been consistently associated with cardiovascular morbidity and mortality, but mechanisms remain uncertain. Associations with blood pressure (BP) may help to explain the cardiovascular effects of air pollution.
OBJECTIVE	We examined the cross-sectional relationship between long-term (annual average) residential air pollution exposure and BP in the National Institute of Environmental Health Sciences' Sister Study, a large U.S. cohort study investigating risk factors for breast cancer and other outcomes.
METHODS	This analysis included 43,629 women 35-76 years of age, enrolled 2003-2009, who had a sister with breast cancer. Geographic information systems contributed to satellite-based nitrogen dioxide (NO2) and fine particulate matter (≤ 2.5 μm; PM2.5) predictions at participant residences at study entry. Generalized additive models were used to examine the relationship between pollutants and measured BP at study entry, adjusting for cardiovascular disease risk factors and including thin plate splines for potential spatial confounding.
RESULTS	A 10- μg/m(3) increase in PM2.5 was associated with 1.4-mmHg higher systolic BP (95% CI: 0.6, 2.3; p < 0.001), 1.0-mmHg higher pulse pressure (95% CI: 0.4, 1.7; p = 0.001), 0.8-mmHg higher mean arterial pressure (95% CI: 0.2, 1.4; p = 0.01), and no significant association with diastolic BP. A 10-ppb increase in NO2 was associated with a 0.4-mmHg (95% CI: 0.2, 0.6; p < 0.001) higher pulse pressure.
CONCLUSIONS	Long-term PM2.5 and NO2 exposures were associated with higher blood pressure. On a population scale, such air pollution-related increases in blood pressure could, in part, account for the increases in cardiovascular disease morbidity and mortality seen in prior studies.

표-316. PubMed 논문번호 25760672의 내용 요약

구분	내용
PubMed ID	25760672
TITLE	Air Pollution and Mortality in Seven Million Adults: The Dutch Environmental Longitudinal Study (DUELS).
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408254
AUTHORS	Fischer Paul H, Marra Marten, Ameling Caroline B, Hoek Gerard, Beelen Rob, de Hoogh Kees, Breugelmans Oscar, Kruize Hanneke, Janssen Nicole A H, Houthuijs Danny
BACKGROUND	Long-term exposure to air pollution has been associated with mortality in urban cohort studies. Few studies have investigated this association in large-scale population registries, including non-urban populations.
OBJECTIVES	The aim of the study was to evaluate the associations between long-term exposure to air pollution and nonaccidental and cause-specific mortality in the Netherlands based on existing national databases.
METHODS	We used existing Dutch national databases on mortality, individual characteristics, residence history, neighborhood characteristics, and national air pollution maps based on land use regression (LUR) techniques for particulates with an aerodynamic diameter ≤ 10 μm (PM10) and nitrogen dioxide (NO2). Using these databases, we established a cohort of 7.1 million individuals ≥ 30 years of age. We followed the cohort for 7 years (2004-2011). We applied Cox proportional hazard models adjusting for potential individual and area-specific confounders.
RESULTS	After adjustment for individual and area-specific confounders, for each 10- μg/m3 increase, PM10 and NO2 were associated with nonaccidental mortality [hazard ratio (HR) = 1.08; 95% CI: 1.07, 1.09 and HR = 1.03; 95% CI: 1.02, 1.03, respectively], respiratory mortality (HR = 1.13; 95% CI: 1.10, 1.17 and HR = 1.02; 95% CI: 1.01, 1.03, respectively), and lung cancer mortality (HR = 1.26; 95% CI: 1.21, 1.30 and HR = 1.10 95% CI: 1.09, 1.11, respectively). Furthermore, PM10 was associated with circulatory disease mortality (HR = 1.06; 95% CI: 1.04, 1.08), but NO2 was not (HR = 1.00; 95% CI: 0.99, 1.01). PM10 associations were robust to adjustment for NO2; NO2 associations remained for nonaccidental mortality and lung cancer mortality after adjustment for PM10.
CONCLUSIONS	Long-term exposure to PM10 and NO2 was associated with nonaccidental and cause-specific mortality in the Dutch population of ≥ 30 years of age.

표-317. PubMed 논문번호 25769126의 내용 요약

구분	내용
PubMed ID	25769126
TITLE	The relationship between transportation noise exposure and ischemic heart disease: a meta-analysis.
JOURNAL	Environmental research: 10.1016/j.envres.2015.02.023
AUTHORS	Vienneau Danielle, Schindler Christian, Perez Laura, Probst-Hensch Nicole, R□□sli Martin
BACKGROUND	There is a growing body of evidence that exposure to transportation related noise can adversely affect health and wellbeing. More recently, research on cardiovascular disease has specifically explored the hypothesis that exposure to transportation noise increases the risk for ischemic heart disease (IHD). Our objective was to review and conduct a meta-analysis to obtain an overall exposure-response association.
METHODS AND RESULTS	We conducted a systematic review and retained published studies on incident cases of IHD using sources of transportation noise as exposure. Study-specific results were transformed into risk estimates per 10dB increase in exposure. Subsequently we conducted a random effects meta-analysis to pool the estimates. We identified 10 studies on road and aircraft noise exposure conducted since the mid-1990s, providing a total of 12 risk estimates. Pooled relative risk for IHD was 1.06 (1.03-1.09) per 10dB increase in noise exposure with the linear exposure-response starting at 50dB. Based on a small number of studies, subgroup analyses were suggestive of higher risk for IHD for males compared to females (p=0.14), and for persons over 65 years of age compared to under (p=0.22). Air pollution adjustment, explored only in a subset of four studies, did not substantially attenuate the association between noise exposure and IHD.
CONCLUSIONS	The evidence for an effect of transportation noise with IHD necessitates further research into the threshold and the shape of the exposure-response association, potential sources of heterogeneity and effect modification. Research in different cultural contexts is also important to derive regional and local estimates for the contribution of transportation noise to the global burden of disease.

표-318. PubMed 논문번호 25793433의 내용 요약

구분	내용
PubMed ID	25793433
TITLE	Carotid Intima-Media Thickness and Long-Term Exposure to Traffic-Related Air Pollution in Middle-Aged Residents of Taiwan: A Cross-Sectional Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408553
AUTHORS	Su Ta-Chen, Hwang Juey-Jen, Shen Yu-Cheng, Chan Chang-Chuan
BACKGROUND	Associations between long-term exposure to air pollution and carotid intima-media thickness (CIMT) have inconsistent findings.
OBJECTIVES	In this study we aimed to evaluate association between 1-year average exposure to traffic-related air pollution and CIMT in middle-aged adults in Asia.
METHODS	CIMT was measured in Taipei, Taiwan, between 2009 and 2011 in 689 volunteers 35-65 years of age who were recruited as the control subjects of an acute coronary heart disease cohort study. We applied land-use regression models developed by the European Study of Cohorts for Air Pollution Effects (ESCAPE) to estimate each subject's 1-year average exposure to traffic-related air pollutants with particulate matter diameters ≤ 10 μm (PM10) and ≤ 2.5 μm (PM2.5) and the absorbance levels of PM2.5 (PM2.5abs), nitrogen dioxide (NO2), and nitrogen oxides (NOx) in the urban environment.
RESULTS	One-year average air pollution exposures were 44.21 ± 4.19 μg/m3 for PM10, 27.34 ± 5.12 μg/m3 for PM2.5, and (1.97 ± 0.36) × 10-5/m for PM2.5abs. Multivariate regression analyses showed average percentage increases in maximum left CIMT of 4.23% (95% CI: 0.32, 8.13) per 1.0 × 10-5/m increase in PM2.5abs; 3.72% (95% CI: 0.32, 7.11) per 10- μg/m3 increase in PM10; 2.81% (95% CI: 0.32, 5.31) per 20- μg/m3 increase in NO2; and 0.74% (95% CI: 0.08, 1.41) per 10- μg/m3 increase in NOx. The associations were not evident for right CIMT, and PM2.5 mass concentration was not associated with the outcomes.
CONCLUSIONS	Long-term exposures to traffic-related air pollution of PM2.5abs, PM10, NO2, and NOx were positively associated with subclinical atherosclerosis in middle-aged adults.

표-319. PubMed 논문번호 25797600의 내용 요약

구분	내용
PubMed ID	25797600
TITLE	The relationship between Asian dust events and out-of-hospital cardiac arrests in Japan.
JOURNAL	Journal of epidemiology: 10.2188/jea.JE20140179
AUTHORS	Nakamura Takahiro, Hashizume Masahiro, Ueda Kayo, Kubo Tatsuhiko, Shimizu Atsushi, Okamura Tomonori, Nishiwaki Yuji
BACKGROUND	Asian dust events are caused by dust storms that originate in the deserts of China and Mongolia and drift across East Asia. We hypothesized that the dust events would increase incidence of out-of-hospital cardiac arrests by triggering acute events or exacerbating chronic diseases.
METHODS	We analyzed the Utstein-Style data collected in 2005 to 2008 from seven prefectures covering almost the entire length of Japan to investigate the effect of Asian dust events on out-of-hospital cardiac arrests. Asian dust events were defined by the measurement of light detection and ranging. A time-stratified case-crossover analysis was performed. The strength of the association between Asian dust events and out-of-hospital cardiac arrests was shown by odds ratios and 95% confidence intervals in two conditional logistic models. A pooled estimate was obtained from area-specific results by random-effect meta-analysis.
RESULTS	The total number of cases of out-of-hospital cardiac arrest was 59 273, of which 35 460 were in men and 23 813 were in women. The total number of event days during the study period was smallest in Miyagi and Niigata and largest in Shimane and Nagasaki. There was no significant relationship between Asian dust events and out-of-hospital cardiac arrests by area in either of the models. In the pooled analysis, the highest odds ratios were observed at lag day 1 in both model 1 (OR 1.07; 95% CI, 0.97-1.19) and model 2 (OR 1.08; 95% CI, 0.97-1.20). However, these results were not statistically significant.
CONCLUSIONS	We found no evidence of an association between Asian dust events and out-of-hospital cardiac arrests.

표-320. PubMed 논문번호 25802269의 내용 요약

구분	내용
PubMed ID	25802269
TITLE	Exposure to parental smoking in childhood is associated with increased risk of carotid atherosclerotic plaque in adulthood: the Cardiovascular Risk in Young Finns Study.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.114.013485
AUTHORS	West Henry W, Juonala Markus, Gall Seana L, K□h□nen Mika, Laitinen Tomi, Taittonen Leena, Viikari Jorma S A, Raitakari Olli T, Magnussen Costan G
BACKGROUND	The association between passive smoking exposure in childhood and adverse cardiovascular health in adulthood is not well understood. Using a 26-year follow-up study, we examined whether childhood exposure to passive smoking was associated with carotid atherosclerotic plaque in young adults.
METHODS AND RESULTS	Participants were from the Cardiovascular Risk in Young Finns Study (n=2448). Information on childhood exposure to parental smoking was collected in 1980 and 1983. Carotid ultrasound data were collected in adulthood in 2001 or 2007. Childhood serum cotinine levels from 1980 were measured from frozen samples in 2014 (n=1578). The proportion of children with nondetectable cotinine levels was highest among households in which neither parent smoked (84%), was decreased in households in which 1 parent smoked (62%), and was lowest among households in which both parents smoked (43%). Regardless of adjustment for potential confounding and mediating variables, the relative risk of developing carotid plaque in adulthood increased among those children with 1 or both parents who smoked (relative risk, 1.7; 95% confidence interval, 1.0-2.8; P=0.04). Although children whose parents exercised good "smoking hygiene" (smoking parents whose children had nondetectable cotinine levels) had increased risk of carotid plaque compared with children with nonsmoking parents (relative risk, 1.6; 95% confidence interval, 0.6-4.0; P=0.34), children of smoking parents with poor smoking hygiene (smoking parents whose children had detectable serum cotinine levels) had substantially increased risk of plaque as adults (relative risk, 4.0; 95% confidence interval, 1.7-9.8; P=0.002).
CONCLUSIONS	Children of parents who smoke have increased risk of developing carotid atherosclerotic plaque in adulthood. However, parents who exercise good smoking hygiene can lessen their child's risk of developing plaque.

표-321. PubMed 논문번호 25803667의 내용 요약

구분	내용
PubMed ID	25803667
TITLE	Life course exposure to smoke and early menopause and menopausal transition.
JOURNAL	Menopause (New York, N.Y.): 10.1097/GME.0000000000000444
AUTHORS	Tawfik Hebatullah, Kline Jennie, Jacobson Judith, Tehranifar Parisa, Protacio Angeline, Flom Julie D, Cirillo Piera, Cohn Barbara A, Terry Mary Beth
OBJECTIVE	Early age at menopause is associated with increased risk of cardiovascular disease, stroke, osteoporosis, and all-cause mortality. Cigarette smoke exposure in adulthood is an established risk factor for earlier age at natural menopause and may be related to age at the menopausal transition. Using data from two US birth cohorts, we examined the association between smoke exposure at various stages of the life course (prenatal exposure, childhood exposure to parental smoking, and adult smoke exposure) and menopause status in 1,001 women aged 39 to 49 years at follow-up.
METHODS	We used logistic regression analysis (adjusting for age at follow-up) to estimate odds ratios (ORs) and 95% confidence intervals (CI) relating smoke exposure to natural menopause and the menopausal transition.
RESULTS	The magnitudes of the associations for natural menopause were similar but not statistically significant after adjustment for confounders among (i) women with prenatal smoke exposure who did not smoke on adult follow-up (OR, 2.7; 95% CI, 0.8-9.4) and (ii) current adult smokers who were not exposed prenatally (OR, 2.8; 95% CI, 0.9-9.0). Women who had been exposed to prenatal smoke and were current smokers had three times the risk of experiencing earlier natural menopause (adjusted OR, 3.4; 95% CI, 1.1-10.3) compared with women without smoke exposure in either period. Only current smoking of long duration (>26 y) was associated with the timing of the menopausal transition.
CONCLUSIONS	Our data suggest that exposure to smoke both prenatally and around the time of menopause accelerates ovarian aging.

표-322. PubMed 논문번호 25807578의 내용 요약

구분	내용
PubMed ID	25807578
TITLE	Association of Roadway Proximity with Fasting Plasma Glucose and Metabolic Risk Factors for Cardiovascular Disease in a Cross-Sectional Study of Cardiac Catheterization Patients.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1306980
AUTHORS	Ward-Caviness Cavin K, Kraus William E, Blach Colette, Haynes Carol S, Dowdy Elaine, Miranda Marie Lynn, Devlin Robert B, Diaz-Sanchez David, Cascio Wayne E, Mukerjee Shaibal, Stallings Casson, Smith Luther A, Gregory Simon G, Shah Svati H, Hauser Elizabeth R, Neas Lucas M
BACKGROUND	The relationship between traffic-related air pollution (TRAP) and risk factors for cardiovascular disease needs to be better understood in order to address the adverse impact of air pollution on human health.
OBJECTIVE	We examined associations between roadway proximity and traffic exposure zones, as markers of TRAP exposure, and metabolic biomarkers for cardiovascular disease risk in a cohort of patients undergoing cardiac catheterization.
METHODS	We performed a cross-sectional study of 2,124 individuals residing in North Carolina (USA). Roadway proximity was assessed via distance to primary and secondary roadways, and we used residence in traffic exposure zones (TEZs) as a proxy for TRAP. Two categories of metabolic outcomes were studied: measures associated with glucose control, and measures associated with lipid metabolism. Statistical models were adjusted for race, sex, smoking, body mass index, and socioeconomic status (SES).
RESULTS	An interquartile-range (990 m) decrease in distance to roadways was associated with higher fasting plasma glucose (β = 2.17 mg/dL; 95% CI: -0.24, 4.59), and the association appeared to be limited to women (β = 5.16 mg/dL; 95% CI: 1.48, 8.84 compared with β = 0.14 mg/dL; 95% CI: -3.04, 3.33 in men). Residence in TEZ 5 (high-speed traffic) and TEZ 6 (stop-and-go traffic), the two traffic zones assumed to have the highest levels of TRAP, was positively associated with high-density lipoprotein cholesterol (HDL-C; β = 8.36; 95% CI: -0.15, 16.9 and β = 5.98; 95% CI: -3.96, 15.9, for TEZ 5 and 6, respectively).
CONCLUSION	Proxy measures of TRAP exposure were associated with intermediate metabolic traits associated with cardiovascular disease, including fasting plasma glucose and possibly HDL-C.

표-323. PubMed 논문번호 25810496의 내용 요약

구분	내용
PubMed ID	25810496
TITLE	Short term exposure to air pollution and stroke: systematic review and meta-analysis.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.h1295
AUTHORS	Shah Anoop S V, Lee Kuan Ken, McAllister David A, Hunter Amanda, Nair Harish, Whiteley William, Langrish Jeremy P, Newby David E, Mills Nichola L
OBJECTIVE	To review the evidence for the short term association between air pollution and stroke.
DESIGN	Systematic review and meta-analysis of observational studies
DATA SOURCES	Medline, Embase, Global Health, Cumulative Index to Nursing and Allied Health Literature (CINAHL), and Web of Science searched to January 2014 with no language restrictions.
ELIGIBILITY CRITERIA	Studies investigating the short term associations (up to lag of seven days) between daily increases in gaseous pollutants (carbon monoxide, sulphur dioxide, nitrogen dioxide, ozone) and particulate matter (<2.5 □m or <10 □m diameter (PM2.5 and PM10)), and admission to hospital for stroke or mortality.
MAIN OUTCOME MEASURES	Admission to hospital and mortality from stroke.
RESULTS	From 2748 articles, 238 were reviewed in depth with 103 satisfying our inclusion criteria and 94 contributing to our meta-estimates. This provided a total of 6.2 million events across 28 countries. Admission to hospital for stroke or mortality from stroke was associated with an increase in concentrations of carbon monoxide (relative risk 1.015 per 1 ppm, 95% confidence interval 1.004 to 1.026), sulphur dioxide (1.019 per 10 ppb, 1.011 to 1.027), and nitrogen dioxide (1.014 per 10 ppb, 1.009 to 1.019). Increases in PM2.5 and PM10 concentration were also associated with admission and mortality (1.011 per 10 μg/m(3) (1.011 to 1.012) and 1.003 per 10 □g/m(3) (1.002 to 1.004), respectively). The weakest association was seen with ozone (1.001 per 10 ppb, 1.000 to 1.002). Strongest associations were observed on the day of exposure with more persistent effects observed for PM(2·5).
CONCLUSION	Gaseous and particulate air pollutants have a marked and close temporal association with admissions to hospital for stroke or mortality from stroke. Public and environmental health policies to reduce air pollution could reduce the burden of stroke.
SYSTEMATIC REVIEW REGISTRATION	PROSPERO-CRD42014009225.

표-324. PubMed 논문번호 25816055의 내용 요약

구분	내용
PubMed ID	25816055
TITLE	Air Pollution from Road Traffic and Systemic Inflammation in Adults: A Cross-Sectional Analysis in the European ESCAPE Project.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408224
AUTHORS	Lanki Timo, Hampel Regina, Tiittanen Pekka, Andrich Silke, Beelen Rob, Brunekreef Bert, Dratva Julia, De Faire Ulf, Fuks Kateryna B, Hoffmann Barbara, Imboden Medea, Jousilahti Pekka, Koenig Wolfgang, Mahabadi Amir A, K□nzli Nino, Pedersen Nancy L, Penell Johanna, Pershagen G□ran, Probst-Hensch Nicole M, Schaffner Emmanuel, Schindler Christian, Sugiri Dorothea, Swart Wim J R, Tsai Ming-Yi, Turunen Anu W, Weinmayr Gudrun, Wolf Kathrin, Yli-Tuomi Tarja, Peters Annette
BACKGROUND	Exposure to particulate matter air pollution (PM) has been associated with cardiovascular diseases.
OBJECTIVES	In this study we evaluated whether annual exposure to ambient air pollution is associated with systemic inflammation, which is hypothesized to be an intermediate step to cardiovascular disease.
METHODS	Six cohorts of adults from Central and Northern Europe were used in this cross-sectional study as part of the larger ESCAPE project (European Study of Cohorts for Air Pollution Effects). Data on levels of blood markers for systemic inflammation-high-sensitivity C-reactive protein (CRP) and fibrinogen-were available for 22,561 and 17,428 persons, respectively. Land use regression models were used to estimate cohort participants' long-term exposure to various size fractions of PM, soot, and nitrogen oxides (NOx). In addition, traffic intensity on the closest street and traffic load within 100 m from home were used as indicators of traffic air pollution exposure.
RESULTS	Particulate air pollution was not associated with systemic inflammation. However, cohort participants living on a busy (> 10,000 vehicles/day) road had elevated CRP values (10.2%; 95% CI: 2.4, 18.8%, compared with persons living on a quiet residential street with < 1,000 vehicles/day). Annual NOx concentration was also positively associated with levels of CRP (3.2%; 95% CI: 0.3, 6.1 per 20 μg/m3), but the effect estimate was more sensitive to model adjustments. For fibrinogen, no consistent associations were observed.
CONCLUSIONS	Living close to busy traffic was associated with increased CRP concentrations, a known risk factor for cardiovascular diseases. However, it remains unclear which specific air pollutants are responsible for the association.

표-325. PubMed 논문번호 25838021의 내용 요약

구분	내용
PubMed ID	25838021
TITLE	Air quality, stroke, and coronary events: results of the Heinz Nixdorf Recall Study from the Ruhr Region.
JOURNAL	Deutsches Arzteblatt international: 10.3238/arztebl.2015.0195
AUTHORS	Hoffmann Barbara, Weinmayr Gudrun, Hennig Frauke, Fuks Kateryna, Moebus Susanne, Weimar Christian, Dragano Nico, Hermann Dirk M, K□lsch Hagen, Mahabadi Amir A, Erbel Raimund, J□ckel Karl-Heinz
BACKGROUND	Studies have shown that air pollution is associated with cardiopulmonary mortality, but there has been less research of the possible effect of air pollution on stroke and non-fatal coronary events. The Heinz Nixdorf Recall (HNR) study addressed the question of the effect of long-term air pollution on stroke and coronary events. Ambient acoustic noise was also considered as a risk factor.
METHODS	The HNR study, initiated in 2000, is a prospective, population-based cohort study in the Ruhr region of Germany. Long-term exposure to fine-particle dust (PM10, PM2.5 and PM(2.5abs) [carbon black content]) and traffic noise at the subjects' home addresses were determined using land-use regression and dispersion models, respectively. Strokes and coronary events were ascertained from patient records by an independent end-point committee on the basis of predefined study criteria. The adjusted hazard ratio (HR) was calculated using Cox regression analysis for an increase in concentration from the 5th to the 95th percentile for each exposure.
RESULTS	Data from 4433 subjects were evaluated. The incidence of stroke was 2.03 per 1000 person-years (PY), and that of coronary events was 3.87 per 1000 PY. The highest hazard ratios for stroke were seen for PM10 (HR 2.61, 95% confidence interval [CI] 1.13-6.00) and PM2.5 (HR 3.20, 95% CI 1.26-8.09). The highest hazard ratios for coronary events were found for PM10 (HR 1.07, 95% CI 0.56-2.04) and for PM(2.5abs) (HR 1.37, 95% CI 0.80-2.36).
CONCLUSION	Long-term exposure to fine-particle dust is associated with a higher risk of stroke, regardless of the subject's exposure to noise at his or her home address. The results for coronary events are less clear, but still suggest increased risk.

표-326. PubMed 논문번호 25845694의 내용 요약

구분	내용
PubMed ID	25845694
TITLE	Sleep Disordered Breathing in Four Resource-Limited Settings in Peru: Prevalence, Risk Factors, and Association with Chronic Diseases.
JOURNAL	Sleep: 10.5665/sleep.4988
AUTHORS	Schwartz Noah G, Rattner Adi, Schwartz Alan R, Mokhlesi Babak, Gilman Robert H, Bernabe-Ortiz Antonio, Miranda J Jaime, Checkley William
STUDY OBJECTIVES	Sleep disordered breathing (SDB) is a highly prevalent condition in high-income countries, with major consequences for cardiopulmonary health, public safety, healthcare utilization, and mortality. However, its prevalence and effect in low- and middle-income countries are less well known. We sought to determine the prevalence, risk factors, and comorbidities of SDB symptoms in four resource-limited settings.
DESIGN	Cross-sectional analysis of the CRONICAS Cohort, a population-based age- and sex-stratified sample.
SETTING	Four resource-limited settings in Peru varying in altitude, urbanization, and air pollution.
PARTICIPANTS	There were 2,682 adults aged 35 to 92 y.
MEASUREMENTS AND RESULTS	Self-reported SDB symptoms (habitual snoring, observed apneas, Epworth Sleepiness Scale), sociodemographics, medical history, anthropometrics, spirometry, blood biomarkers were reported. We found a high prevalence of habitual snoring (30.2%, 95% confidence interval [CI] 28.5-32.0%), observed apneas (20.9%, 95% CI 19.4-22.5%) and excessive daytime sleepiness (18.6%, 95% CI 17.1-20.1%). SDB symptoms varied across sites; prevalence and adjusted odds for habitual snoring were greatest at sea level, whereas those for observed apneas were greatest at high altitude. In multivariable analysis, habitual snoring was associated with older age, male sex, body mass index (BMI), and higher socioeconomic status; observed apneas were associated with BMI; and excessive daytime sleepiness was associated with older age, female sex, and medium socioeconomic status. Adjusted odds of cardiovascular disease, depression, and hypertension and total chronic disease burden increased progressively with the number of SDB symptoms. A threefold increase in the odds of having an additional chronic comorbid disease (adjusted odds ratio 3.57, 95% CI 2.18-5.84) was observed in those with all three versus no SDB symptoms.
CONCLUSIONS	Sleep disordered breathing symptoms were highly prevalent, varied widely across four resource-limited settings in Peru, and exhibited strong independent associations with chronic diseases.

표-327. PubMed 논문번호 25863281의 내용 요약

구분	내용
PubMed ID	25863281
TITLE	Chronic disease prevalence in women and air pollution - A 30-year longitudinal cohort study.
JOURNAL	Environment international: 10.1016/j.envint.2015.03.017
AUTHORS	To Teresa, Zhu Jingqin, Villeneuve Paul J, Simatovic Jacqueline, Feldman Laura, Gao Chenwei, Williams Devon, Chen Hong, Weichenthal Scott, Wall Claus, Miller Anthony B
BACKGROUND	Air pollution, such as fine particulate matter (PM2.5), can increase risk of adverse health events among people with heart disease, diabetes, asthma and chronic obstructive pulmonary disease (COPD) by aggravating these conditions. Identifying the influence of PM2.5 on prevalence of these conditions may help target interventions to reduce disease morbidity among high-risk populations.
OBJECTIVES	The objective of this study is to measure the association of exposure of PM2.5 with prevalence risk of various chronic diseases among a longitudinal cohort of women.
METHODS	Women from Ontario who enrolled in the Canadian National Breast Screening Study (CNBSS) from 1980 to 1985 (n = 29,549) were linked to provincial health administrative data from April 1, 1992 to March 31, 2013 to determine the prevalence of major chronic disease and conditions (heart disease, diabetes, asthma, COPD, acute myocardial infarction, angina, stroke and cancers). Exposure to PM2.5 was measured using satellite data collected from January 1, 1998 to December 31, 2006 and assigned to resident postal-code at time of entry into study. Poisson regression models were used to describe the relationship between exposure to ambient PM2.5 and chronic disease prevalence. Prevalence rate ratios (PRs) were estimated while adjusting for potential confounders: baseline age, smoking, BMI, marital status, education and occupation. Separate models were run for each chronic disease and condition.
RESULTS	Congestive heart failure (PR = 1.31, 95% CI: 1.13, 1.51), diabetes (PR = 1.28, 95% CI: 1.16, 1.41), ischemic heart disease (PR = 1.22, 95% CI: 1.14, 1.30), and stroke (PR = 1.21, 95% CI: 1.09, 1.35) showed over a 20% increase in PRs per 10 μg/m(3) increase in PM2.5 after adjusting for risk factors. Risks were elevated in smokers and those with BMI greater than 30.
CONCLUSIONS	This study estimated significant elevated prevalent rate ratios per unit increase in PM2.5 in nine of the ten chronic diseases studied.

표-328. PubMed 논문번호 25872223의 내용 요약

구분	내용
PubMed ID	25872223
TITLE	Ambient Coarse Particulate Matter and Hospital Admissions in the Medicare Cohort Air Pollution Study, 1999-2010.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408720
AUTHORS	Powell Helen, Krall Jenna R, Wang Yun, Bell Michelle L, Peng Roger D
BACKGROUND	In recent years a number of studies have examined the short-term association between coarse particulate matter (PM(10-2.5)) and mortality and morbidity outcomes. These studies, however, have produced inconsistent conclusions.
OBJECTIVES	We estimated both the national- and regional-level associations between PM(10-2.5) and emergency hospitalizations for both cardiovascular and respiratory disease among Medicare enrollees ≥ 65 years of age during the 12-year period 1999 through 2010.
METHODS	Using air pollution data obtained from the U.S. Environmental Protection Agency air quality monitoring network and daily emergency hospitalizations for 110 large urban U.S. counties assembled from the Medicare Cohort Air Pollution Study (MCAPS), we estimated the association between short-term exposure to PM(10-2.5) and hospitalizations using a two-stage Bayesian hierarchical model and Poisson log-linear regression models.
RESULTS	A 10- μg/m3 increase in PM(10-2.5) was associated with a significant increase in same-day cardiovascular hospitalizations [0.69%; 95% posterior interval (PI): 0.45, 0.92]. After adjusting for PM2.5, this association remained significant (0.63%; 95% PI: 0.38, 0.88). A 10- μg/m3 increase in PM(10-2.5) was not associated with a significant increase in respiratory-related hospitalizations.
CONCLUSIONS	We found statistically significant evidence that daily variation in PM(10-2.5) is associated with emergency hospitalizations for cardiovascular diseases among Medicare enrollees ≥ 65 years of age. This association was robust to adjustment for concentrations of PM2.5.
CITATION	Powell H, Krall JR, Wang Y, Bell ML, Peng RD. 2015. Ambient coarse particulate matter and hospital admissions in the Medicare Cohort Air Pollution Study, 1999-2010. Environ Health Perspect 123:1152-1158; http://dx.doi.org/10.1289/ehp.1408720.

표-329. PubMed 논문번호 25875389의 내용 요약

구분	내용
PubMed ID	25875389
TITLE	Secondhand smoke exposure is associated with increased carotid artery intima-media thickness: the Bogalusa Heart Study.
JOURNAL	Atherosclerosis: 10.1016/j.atherosclerosis.2015.04.002
AUTHORS	Chen Wei, Yun Miaoying, Fernandez Camilo, Li Shengxu, Sun Dianjianyi, Lai Chin-Chih, Hua Yingxiao, Wang Fu, Zhang Tao, Srinivasan Sathanur R, Johnson Carolyn C, Berenson Gerald S
BACKGROUND	Secondhand smoke (SHS) exposure increases cardiovascular disease risk. The objective of this study was to examine the association of SHS exposure in childhood and adulthood with adult arterial thickness.
METHODS	The study cohort consisted of 415 nonsmoking adults (301 whites and 114 blacks; ages 26.2-48.0 years) enrolled in 2004-2010. The arterial wall thickness was measured as common, bulb and internal carotid artery intima-media thickness (IMT). SHS exposure data in childhood and adulthood were obtained by a questionnaire survey.
RESULTS	Increased adult composite carotid IMT was significantly associated with SHS exposure (regression coefficient, β = 53.1 μm, p < 0.001) after adjusting for race, age, gender, education, income, body mass index, systolic blood pressure, LDL cholesterol and triglycerides/HDL cholesterol ratio, with blacks (β = 81.2 μm, p = 0.005) and whites (β = 38.9 μm, p = 0.017) showing the same direction of the association. Furthermore, the SHS exposure in childhood showed a relatively stronger association with increased carotid IMT than the exposure in adulthood based on standardized βs (0.180 vs. 0.106); the same trend in the difference between childhood and adulthood exposure was noted for duration of SHS exposure (0.186 vs. 0.145). The covariates-adjusted composite carotid IMT showed a significant increasing trend by the order of exposure status of none, adulthood only, childhood only and both (p for trend<0.001).
CONCLUSIONS	If the relationship is causal, the associations observed in this study suggest that more awareness should be raised on the dangers of SHS exposure during childhood so that its effect may be mitigated and controlled early in the cardiovascular disease process.

표-330. PubMed 논문번호 25890359의 내용 요약

구분	내용
PubMed ID	25890359
TITLE	Controlled exposure to particulate matter from urban street air is associated with decreased vasodilation and heart rate variability in overweight and older adults.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-015-0081-9
AUTHORS	Hemmingsen Jette G, Rissler Jenny, Lykkesfeldt Jens, Sallsten Gerd, Kristiansen Jesper, M ø ller P Peter, Loft Steffen
BACKGROUND	Exposure to particulate matter (PM) is generally associated with elevated risk of cardiovascular morbidity and mortality. Elderly and obese subjects may be particularly susceptible, although short-term effects are poorly described.
METHODS	Sixty healthy subjects (25 males, 35 females, age 55 to 83 years, body mass index>25 kg/m2) were included in a cross-over study with 5 hours of exposure to particle- or sham-filtered air from a busy street using an exposure-chamber. The sham- versus particle-filtered air had average particle number concentrations of ~23.000 versus ~1800/cm3 and PM2.5 levels of 24 versus 3 μg/m3, respectively. The PM contained similar fractions of elemental and black carbon (~20-25%) in both exposure scenarios. Reactive hyperemia and nitroglycerin-induced vasodilation in finger arteries and heart rate variability (HRV) measured within 1 h after exposure were primary outcomes. Potential explanatory mechanistic variables included markers of oxidative stress (ascorbate/dehydroascorbate, nitric oxide-production cofactor tetrahydrobiopterin and its oxidation product dihydrobiopterin) and inflammation markers (C-reactive protein and leukocyte differential counts).
RESULTS	Nitroglycerin-induced vasodilation was reduced by 12% [95% confidence interval: -22%; -1.0%] following PM exposure, whereas hyperemia-induced vasodilation was reduced by 5% [95% confidence interval: -11.6%; 1.6%]. Moreover, HRV measurements showed that the high and low frequency domains were significantly decreased and increased, respectively. Redox and inflammatory status did not change significantly based on the above measures.
CONCLUSIONS	This study indicates that exposure to real-life levels of PM from urban street air impairs the vasomotor function and HRV in overweight middle-aged and elderly adults, although this could not be explained by changes in inflammation, oxidative stress or nitric oxide-cofactors.

표-331. PubMed 논문번호 25894856의 내용 요약

구분	내용
PubMed ID	25894856
TITLE	Long-term Exposure to Fine Particulate Matter Air Pollution and Mortality Among Canadian Women.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000294
AUTHORS	Villeneuve Paul J, Weichenthal Scott A, Crouse Daniel, Miller Anthony B, To Teresa, Martin Randall V, van Donkelaar Aaron, Wall Claus, Burnett Richard T
BACKGROUND	Long-term exposure to fine particulate matter (PM2.5) has been associated with increased mortality, especially from cardiovascular disease. There are, however, uncertainties about the nature of the exposure-response relation at lower concentrations. In Canada, where ambient air pollution levels are substantially lower than in most other countries, there have been few attempts to study associations between long-term exposure to PM2.5 and mortality.
METHODS	We present a prospective cohort analysis of 89,248 women who enrolled in the Canadian National Breast Screening Study between 1980 and 1985, and for whom residential measures of PM2.5 could be assigned. We derived individual-level estimates of long-term exposure to PM2.5 from satellite observations. We linked cohort records to national mortality data to ascertain mortality between 1980 and 2005. We used Cox proportional hazards models to characterize associations between PM2.5 and several causes of death. The hazard ratios (HRs) and 95% confidence intervals (CIs) computed from these models were adjusted for several individual and neighborhood-level characteristics.
RESULTS	The cohort was composed predominantly of Canadian-born (82%) and married (80%) women. The median residential concentration of PM2.5 was 9.1 μg/m(3) (standard deviation = 3.4). In fully adjusted models, a 10 μg/m(3) increase in PM2.5 exposure was associated with elevated risks of nonaccidental (HR: 1.12; 95% CI = 1.04, 1.19), and ischemic heart disease mortality (HR: 1.34; 95% CI = 1.09, 1.66).
CONCLUSIONS	The findings from this study provide additional support for the hypothesis that exposure to very low levels of ambient PM2.5 increases the risk of cardiovascular mortality.

표-332. PubMed 논문번호 25896330의 내용 요약

구분	내용
PubMed ID	25896330
TITLE	The impacts of traffic-related and woodsmoke particulate matter on measures of cardiovascular health: a HEPA filter intervention study.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2014-102696
AUTHORS	Kajbafzadeh Majid, Brauer Michael, Karlen Barbara, Carlsten Chris, van Eeden Stephan, Allen Ryan W
BACKGROUND	Combustion-generated fine particulate matter (PM2.5) is associated with cardiovascular morbidity. Both traffic-related air pollution and residential wood combustion may be important, but few studies have compared their impacts.
OBJECTIVES	To assess and compare effects of traffic-related and woodsmoke PM2.5 on endothelial function and systemic inflammation (C reactive protein, interleukin-6 and band cells) among healthy adults in Vancouver, British Columbia, Canada, using high efficiency particulate air (HEPA) filtration to introduce indoor PM2.5 exposure gradients.
METHODS	We recruited 83 healthy adults from 44 homes in traffic-impacted or woodsmoke-impacted areas to participate in this randomised, single-blind cross-over intervention study. PM2.5 concentrations were measured during two consecutive 7-day periods, one with filtration and the other with 'placebo filtration'. Endothelial function and biomarkers of systematic inflammation were measured at the end of each 7-day period.
RESULTS	HEPA filtration was associated with a 40% decrease in indoor PM2.5 concentrations. There was no relationship between PM2.5 exposure and endothelial function. There was evidence of an association between indoor PM2.5 and C reactive protein among those in traffic-impacted locations (42.1% increase in C reactive protein per IQR increase in indoor PM2.5, 95% CI 1.2% to 99.5%), but not among those in woodsmoke-impacted locations. There were no associations with interleukin-6 or band cells.
CONCLUSIONS	Evidence of an association between C reactive protein and indoor PM2.5 among healthy adults in traffic-impacted areas is consistent with the hypothesis that traffic-related particles, even at relatively low concentrations, play an important role in the cardiovascular effects of the urban PM mixture.
TRIAL REGISTRATION NUMBER	http://www.clinicaltrials.gov (NCT01570062).

표-333. PubMed 논문번호 25898780의 내용 요약

구분	내용
PubMed ID	25898780
TITLE	Controlled exposure to diesel exhaust and traffic noise-Effects on oxidative stress and activation in mononuclear blood cells.
JOURNAL	Mutation research: 10.1016/j.mrfmmm.2015.03.009
AUTHORS	Hemmingsen Jette Gjerke, M ø ller Peter, Jantzen Kim, J□nsson Bo A G, Albin Maria, Wierzbicka Aneta, Gudmundsson Anders, Loft Steffen, Rissler Jenny
UNLABELLED	Particulate air pollution increases risk of cancer and cardiopulmonary disease, partly through oxidative stress. Traffic-related noise increases risk of cardiovascular disease and may cause oxidative stress. In this controlled random sequence study, 18 healthy subjects were exposed for 3h to diesel exhaust (DE) at 276 μg/m(3) from a passenger car or filtered air, with co-exposure to traffic noise at 48 or 75 dB(A). Gene expression markers of inflammation, (interleukin-8 and tumor necrosis factor), oxidative stress (heme oxygenase (decycling-1)) and DNA repair (8-oxoguanine DNA glycosylase (OGG1)) were unaltered in peripheral blood mononuclear cells (PBMCs). No significant differences in DNA damage levels, measured by the comet assay, were observed after DE exposure, whereas exposure to high noise levels was associated with significantly increased levels of hOGG1-sensitive sites in PBMCs. Urinary levels of 8-oxo-7,8-dihydro-2'-deoxyguanosine were unaltered. In auxiliary ex vivo experiments whole blood was incubated with particles from the exposure chamber for 3h without effects on DNA damage in PBMCs or intracellular reactive oxygen species production and expression of CD11b and CD62L adhesion molecules in leukocyte subtypes.
CONCLUSION	3-h exposure to DE caused no genotoxicity, oxidative stress or inflammation in PBMCs, whereas exposure to noise might cause oxidatively damaged DNA.

표-334. PubMed 논문번호 25910279의 내용 요약

구분	내용
PubMed ID	25910279
TITLE	Satellite-Based Estimates of Long-Term Exposure to Fine Particles and Association with Mortality in Elderly Hong Kong Residents.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408264
AUTHORS	Wong Chit Ming, Lai Hak Kan, Tsang Hilda, Thach Thuan Quoc, Thomas G Neil, Lam Kin Bong Hubert, Chan King Pan, Yang Lin, Lau Alexis K H, Ayres Jon G, Lee Siu Yin, Chan Wai Man, Hedley Anthony J, Lam Tai Hing
BACKGROUND	A limited number of studies on long-term effects of particulate matter with aerodynamic diameter < 2.5 μm (PM2.5) on health suggest it can be an important cause of morbidity and mortality. In Asia where air quality is poor and deteriorating, local data on long-term effects of PM2.5 to support policy on air quality management are scarce.
OBJECTIVES	We assessed long-term effects of PM2.5 on the mortality in a single Asian city.
METHODS	For 10-13 years, we followed up a cohort of 66,820 participants ≥ 65 years of age who were enrolled and interviewed in all 18 Elderly Health Centres of the Department of Health, Hong Kong, in 1998-2001. Their residential addresses were geocoded into x- and y-coordinates, and their proxy exposures to PM2.5 at their addresses in 1 × 1 km grids were estimated from the U.S. National Aeronautics and Space Administration (NASA) satellite data. We used Cox regression models to calculate hazard ratios (HRs) of mortality associated with PM2.5.
RESULTS	Mortality HRs per 10- μg/m3 increase in PM2.5 were 1.14 (95% CI: 1.07, 1.22) for all natural causes, 1.22 (95% CI: 1.08, 1.39) for cardiovascular causes, 1.42 (95% CI: 1.16, 1.73) for ischemic heart disease, 1.24 (95% CI: 1.00, 1.53) for cerebrovascular disease, and 1.05 (95% CI: 0.90, 1.22) for respiratory causes.
CONCLUSIONS	Our methods in using NASA satellite data provide a readily accessible and affordable approach to estimation of a sufficient range of individual PM2.5 exposures in a single city. This approach can expand the capacity to conduct environmental accountability studies in areas with few measurements of fine particles.
CITATION	Wong CM, Lai HK, Tsang H, Thach TQ, Thomas GN, Lam KB, Chan KP, Yang L, Lau AK, Ayres JG, Lee SY, Chan WM, Hedley AJ, Lam TH. 2015. Satellite-based estimates of long-term exposure to fine particles and association with mortality in elderly Hong Kong residents. Environ Health Perspect 123:1167-1172; http://dx.doi.org/10.1289/ehp.1408264.

표-335. PubMed 논문번호 25944145의 내용 요약

구분	내용
PubMed ID	25944145
TITLE	Cardiac effects of seasonal ambient particulate matter and ozone co-exposure in rats.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-015-0087-3
AUTHORS	Farraj Aimen K, Walsh Leon, Haykal-Coates Najwa, Malik Fatiha, McGee John, Winsett Darrell, Duvall Rachelle, Kovalcik Kasey, Cascio Wayne E, Higuchi Mark, Hazari Mehdi S
BACKGROUND	The potential for seasonal differences in the physicochemical characteristics of ambient particulate matter (PM) to modify interactive effects with gaseous pollutants has not been thoroughly examined. The purpose of this study was to compare cardiac responses in conscious hypertensive rats co-exposed to concentrated ambient particulates (CAPs) and ozone (O3) in Durham, NC during the summer and winter, and to analyze responses based on particle mass and chemistry.
METHODS	Rats were exposed once for 4 hrs by whole-body inhalation to fine CAPs alone (target concentration: 150 μg/m3), O3 (0.2 ppm) alone, CAPs plus O3, or filtered air during summer 2011 and winter 2012. Telemetered electrocardiographic (ECG) data from implanted biosensors were analyzed for heart rate (HR), ECG parameters, heart rate variability (HRV), and spontaneous arrhythmia. The sensitivity to triggering of arrhythmia was measured in a separate cohort one day after exposure using intravenously administered aconitine. PM elemental composition and organic and elemental carbon fractions were analyzed by high-resolution inductively coupled plasma-mass spectrometry and thermo-optical pyrolytic vaporization, respectively. Particulate sources were inferred from elemental analysis using a chemical mass balance model.
RESULTS	Seasonal differences in CAPs composition were most evident in particle mass concentrations (summer, 171 μg/m3; winter, 85 μg/m3), size (summer, 324 nm; winter, 125 nm), organic:elemental carbon ratios (summer, 16.6; winter, 9.7), and sulfate levels (summer, 49.1 μg/m3; winter, 16.8 μg/m3). Enrichment of metals in winter PM resulted in equivalent summer and winter metal exposure concentrations. Source apportionment analysis showed enrichment for anthropogenic and marine salt sources during winter exposures compared to summer exposures, although only 4% of the total PM mass was attributed to marine salt sources. Single pollutant cardiovascular effects with CAPs and O3 were present during both summer and winter exposures, with evidence for unique effects of co-exposures and associated changes in autonomic tone.
CONCLUSIONS	These findings provide evidence for a pronounced effect of season on PM mass, size, composition, and contributing sources, and exposure-induced cardiovascular responses. Although there was inconsistency in biological responses, some cardiovascular responses were evident only in the co-exposure group during both seasons despite variability in PM physicochemical composition. These findings suggest that a single ambient PM metric alone is not sufficient to predict potential for interactive health effects with other air pollutants.

표-336. PubMed 논문번호 25956005의 내용 요약

구분	내용
PubMed ID	25956005
TITLE	Prospective Study of Ambient Particulate Matter Exposure and Risk of Pulmonary Embolism in the Nurses' Health Study Cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408927
AUTHORS	Pun Vivian C, Hart Jaime E, Kabrhel Christopher, Camargo Carlos A, Baccarelli Andrea A, Laden Francine
BACKGROUND	Pulmonary embolism (PE) is the most serious manifestation of venous thromboembolism and a leading cause of sudden death. Several studies have suggested associations of venous thromboembolism with short-term particulate matter (PM) exposure; evidence on long-term PM and traffic exposure is mixed.
OBJECTIVES	We examined the association of long-term exposure to PM2.5, PM2.5-10, and PM10 (PM with diameter of ≤ 2.5, 2.5-10, and ≤ 10 μm) and distance to roadways with overall incident PE and with PE subtypes in a cohort of U.S. women.
METHODS	The study included 115,745 women from the Nurses' Health Study, followed from 1992 through 2008. Incident PE cases were self-reported biennially. Nonidiopathic PE were cases for which the medical record revealed an underlying health condition related to PE (i.e., surgery, trauma, or malignancy); idiopathic PE were cases with no such history. We used spatiotemporal models combining spatial smoothing and geographic covariates to quantify exposure at residential addresses, and Cox proportional hazards models to calculate hazard ratios (HR) and 95% confidence intervals (CIs).
RESULTS	PM2.5 averaged over 1 month (HR = 1.22; 95% CI: 1.04, 1.44) or 12 months (HR = 1.17; 95% CI: 0.93, 1.48) was associated with incident PE, after adjusting for known risk factors and PM2.5-10. Equivalent analyses restricted to PE subtypes showed a positive association for PM2.5 with nonidiopathic PE, but not with idiopathic PE. We did not find evidence of an association between distance to roadways and PE risk.
CONCLUSIONS	We provide evidence that PM in the prior 1 and 12 months is associated with PE risk. Our results also suggest that women with underlying health conditions may be more susceptible to PE after PM exposure.

표-337. PubMed 논문번호 25967992의 내용 요약

구분	내용
PubMed ID	25967992
TITLE	Quantitative systematic review of the associations between short-term exposure to nitrogen dioxide and mortality and hospital admissions.
JOURNAL	BMJ open: 10.1136/bmjopen-2014-006946
AUTHORS	Mills I C, Atkinson R W, Kang S, Walton H, Anderson H R
BACKGROUND	Short-term exposure to NO_2 has been associated with adverse health effects and there is increasing concern that NO_2 is causally related to health effects, not merely a marker of traffic-generated pollution. No comprehensive meta-analysis of the time-series evidence on NO_2 has been published since 2007.
OBJECTIVE	To quantitatively assess the evidence from epidemiological time-series studies published worldwide to determine whether and to what extent short-term exposure to NO_2 is associated with increased numbers of daily deaths and hospital admissions.
DESIGN	We conducted a quantitative systematic review of 204 time-series studies of NO_2 and daily mortality and hospital admissions for several diagnoses and ages, which were indexed in three bibliographic databases up to May 2011. We calculated random-effects estimates by different geographic regions and globally, and also tested for heterogeneity and small study bias.
RESULTS	Sufficient estimates for meta-analysis were available for 43 cause-specific and age-specific combinations of mortality or hospital admissions (25 for 24 h NO_2 and 18 of the same combinations for 1 h measures). For the all-age group, a 10 □g/m(3) increase in 24 h NO_2 was associated with increases in all-cause, cardiovascular and respiratory mortality (0.71% (95% CI 0.43% to 1.00%), 0.88% (0.63% to 1.13%) and 1.09% (0.75% to 1.42%), respectively), and with hospital admissions for respiratory (0.57% (0.33% to 0.82%)) and cardiovascular (0.66% (0.32% to 1.01%)) diseases. Evidence of heterogeneity between geographical region-specific estimates was identified in more than half of the combinations analysed.
CONCLUSIONS	Our review provides clear evidence of health effects associated with short-term exposure to NO_2 although further work is required to understand reasons for the regional heterogeneity observed. The growing literature, incorporating large multicentre studies and new evidence from less well-studied regions of the world, supports further quantitative review to assess the independence of NO_2 health effects from other air pollutants.

표-338. PubMed 논문번호 25970426의 내용 요약

구분	내용
PubMed ID	25970426
TITLE	Carotid intima-media thickness, a marker of subclinical atherosclerosis, and particulate air pollution exposure: the meta-analytical evidence.
JOURNAL	PloS one: 10.1371/journal.pone.0127014
AUTHORS	Provost Eline B, Madhloum Narjes, Int Panis Luc, De Boever Patrick, Nawrot Tim S
INTRODUCTION	Studies on the association between atherosclerosis and long-term exposure to ambient air pollution suggest that carotid intima-media thickness (CIMT), a marker of subclinical atherosclerosis, is positively associated with particulate matter (PM) exposure. However, there is heterogeneity between the different studies concerning the magnitude of this association. We performed a meta-analysis to determine the strength of the association between CIMT and particulate air pollution.
METHODS	We queried PubMed citation database and Web of Knowledge up to March 2015 in order to identify studies on CIMT and particulate air pollution. Two investigators selected and computerized all relevant information, independently. Eight of the reviewed epidemiological publications provided sufficient details and met our inclusion criteria. Descriptive and quantitative information was extracted from each selected study. The meta-analysis included 18,349 participants from eight cohorts for the cross-sectional association between CIMT and PM and 7,268 participants from three cohorts for the longitudinal analysis on CIMT progression and PM exposure.
RESULTS	The average exposure to PM2.5 in the different study populations ranged from 4.1 to 20.8 □g/m3 and CIMT averaged (SD) 0.73 (0.14) mm. We computed a pooled estimate from a random-effects model. In the combined cross-sectional studies, an increase of 5 □g/m3 PM2.5 was associated with a 1.66% (95% CI: 0.86 to 2.46; P<0.0001) thicker CIMT, which corresponds to an average increase of 12.1 □m. None of the studies moved the combined estimate outside the confidence interval of the overall estimate. A funnel plot suggested absence of publication bias. The combined longitudinal estimate showed for each 5 □g/m3 higher PM2.5 exposure, a 1.04 □m per year (95% CI: 0.01 to 2.07; P=0.048) greater CIMT progression.
CONCLUSION	Our meta-analysis supports the evidence of a positive association between CIMT, a marker of subclinical atherosclerosis, and long-term exposure to particulate air pollution.

표-339. PubMed 논문번호 25978793의 내용 요약

구분	내용
PubMed ID	25978793
TITLE	Long-term Exposure to Particulate Matter Constituents and the Incidence of Coronary Events in 11 European Cohorts.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000300
AUTHORS	Wolf Kathrin, Stafoggia Massimo, Cesaroni Giulia, Andersen Zorana Jovanovic, Beelen Rob, Galassi Claudia, Hennig Frauke, Migliore Enrica, Penell Johanna, Ricceri Fulvio, S ø rensen Mette, Turunen Anu W, Hampel Regina, Hoffmann Barbara, K□lsch Hagen, Laatikainen Tiina, Pershagen G□ran, Raaschou-Nielsen Ole, Sacerdote Carlotta, Vineis Paolo, Badaloni Chiara, Cyrys Josef, de Hoogh Kees, Eriksen Kirsten T, Jedynska Aleksandra, Keuken Menno, Kooter Ingeborg, Lanki Timo, Ranzi Andrea, Sugiri Dorothea, Tsai Ming-Yi, Wang Meng, Hoek Gerard, Brunekreef Bert, Peters Annette, Forastiere Francesco
BACKGROUND	Long-term exposure to particulate matter (PM) has been associated with increased cardiovascular morbidity and mortality but little is known about the role of the chemical composition of PM. This study examined the association of residential long-term exposure to PM components with incident coronary events.
METHODS	Eleven cohorts from Finland, Sweden, Denmark, Germany, and Italy participated in this analysis. 5,157 incident coronary events were identified within 100,166 persons followed on average for 11.5 years. Long-term residential concentrations of PM < 10 μm (PM10), PM < 2.5 μm (PM2.5), and a priori selected constituents (copper, iron, nickel, potassium, silicon, sulfur, vanadium, and zinc) were estimated with land-use regression models. We used Cox proportional hazard models adjusted for a common set of confounders to estimate cohort-specific component effects with and without including PM mass, and random effects meta-analyses to pool cohort-specific results.
RESULTS	A 100 ng/m³ increase in PM10 K and a 50 ng/m³ increase in PM2.5 K were associated with a 6% (hazard ratio and 95% confidence interval: 1.06 [1.01, 1.12]) and 18% (1.18 [1.06, 1.32]) increase in coronary events. Estimates for PM10 Si and PM2.5 Fe were also elevated. All other PM constituents indicated a positive association with coronary events. When additionally adjusting for PM mass, the estimates decreased except for K.
CONCLUSIONS	This multicenter study of 11 European cohorts pointed to an association between long-term exposure to PM constituents and coronary events, especially for indicators of road dust.

표-340. PubMed 논문번호 26003939의 내용 요약

구분	내용
PubMed ID	26003939
TITLE	Years of life lost and morbidity cases attributable to transportation noise and air pollution: A comparative health risk assessment for Switzerland in 2010.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2015.05.003
AUTHORS	Vienneau Danielle, Perez Laura, Schindler Christian, Lieb Christoph, Sommer Heini, Probst-Hensch Nicole, K□nzli Nino, R□□sli Martin
BACKGROUND	There is growing evidence that chronic exposure to transportation related noise and air pollution affects human health. However, health burden to a country of these two pollutants have been rarely compared.
AIMS	As an input for external cost quantification, we estimated the cardiorespiratory health burden from transportation related noise and air pollution in Switzerland, incorporating the most recent findings related to the health effects of noise.
METHODS	Spatially resolved noise and air pollution models for the year 2010 were derived for road, rail and aircraft sources. Average day-evening-night sound level (Lden) and particulate matter (PM10) were selected as indicators, and population-weighted exposures derived by transportation source. Cause-specific exposure-response functions were derived from a meta-analysis for noise and literature review for PM10. Years of life lost (YLL) were calculated using life table methods; population attributable fraction was used for deriving attributable cases for hospitalisations, respiratory illnesses, visits to general practitioners and restricted activity days.
RESULTS	The mean population weighted exposure above a threshold of 48dB(A) was 8.74dB(A), 1.89dB(A) and 0.37dB(A) for road, rail and aircraft noise. Corresponding mean exposure contributions were 4.4, 0.54, 0.12 μg/m(3) for PM10. We estimated that in 2010 in Switzerland transportation caused 6000 and 14,000 YLL from noise and air pollution exposure, respectively. While there were a total of 8700 cardiorespiratory hospital days attributed to air pollution exposure, estimated burden due to noise alone amounted to 22,500 hospital days.
CONCLUSIONS	YLL due to transportation related pollution in Switzerland is dominated by air pollution from road traffic, whereas consequences for morbidity and indicators of quality of life are dominated by noise. In terms of total external costs the burden of noise equals that of air pollution.

표-341. PubMed 논문번호 26017256의 내용 요약

구분	내용
PubMed ID	26017256
TITLE	Smoking-related knowledge and health risk beliefs in a national sample of Aboriginal and Torres Strait Islander people.
JOURNAL	The Medical journal of Australia: 10.5694/mja14.00877
AUTHORS	Nicholson Anna K, Borland Ron, Couzos Sophia, Stevens Matthew, Thomas David P
OBJECTIVES	To describe general knowledge and perceived risk of the health consequences of smoking among Aboriginal and Torres Strait Islander people; and to asses whether knowledge varies among smokers and whether higher knowledge an perceived risk are associated with quitting.
DESIGN, SETTING AND PARTICIPANTS	The Talking About The Smokes project used quota sampling to recruit participants from communities served by 34 Aboriginal community-controlled health services and one community in the Torres Strai Baseline survey data were collected from 2522 Aboriginal and Torres Strait Islander adults from April 2012 to October 2013.
MAIN OUTCOME MEASURES	Knowledge of direct effects of smoking and harms of second-hand smoke (SHS), risk minimisation, health worry, and wanting and attempting to quit.
RESULTS	Most Aboriginal and Torres Strait Islander participants who were daily smokers demonstrated knowledge that smoking causes lung cancer (94%), heart disease (89%) and low birthweight (82%), but fewer were aware that it makes diabetes worse (68%). Similarly, almost all daily smokers knew of the harms of SHS: that it is dangerous to non-smokers (90%) and children (95% and that it causes asthma in children (91%). Levels of knowledge among dai smokers were lower than among non-daily smokers, ex-smokers and never-smokers. Among smokers, greater knowledge of SHS harms was associated with health worry, wanting to quit and having attempted to quit i the past year, but knowledge of direct harms of smoking was not.
CONCLUSION	Lack of basic knowledge about the health consequences of smoking is not an important barrier to trying to quit for Aboriginal and Torres Strait Islander smokers. Framing new messages about the negative health effects of smoking in ways that encompass the health of others is likely to contribute to goal setting and prioritising quitting among Aboriginal and Torres Strait Islander people.

표-342. PubMed 논문번호 26022815의 내용 요약

구분	내용
PubMed ID	26022815
TITLE	Cardiopulmonary benefits of reducing indoor particles of outdoor origin: a randomized, double-blind crossover trial of air purifiers.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2015.03.553
AUTHORS	Chen Renjie, Zhao Ang, Chen Honglei, Zhao Zhuohui, Cai Jing, Wang Cuicui, Yang Changyuan, Li Huichu, Xu Xiaohui, Ha Sandie, Li Tiantian, Kan Haidong
BACKGROUND	Indoor exposure to fine particulate matter (PM2.5) from outdoor sources is a major health concern, especially in highly polluted developing countries such as China. Few studies have evaluated the effectiveness of indoor air purification on the improvement of cardiopulmonary health in these areas.
OBJECTIVES	This study sought to evaluate whether a short-term indoor air purifier intervention improves cardiopulmonary health.
METHODS	We conducted a randomized, double-blind crossover trial among 35 healthy college students in Shanghai, China, in 2014. These students lived in dormitories that were randomized into 2 groups and alternated the use of true or sham air purifiers for 48 h with a 2-week washout interval. We measured 14 circulating biomarkers of inflammation, coagulation, and vasoconstriction; lung function; blood pressure (BP); and fractional exhaled nitric. We applied linear mixed-effect models to evaluate the effect of the intervention on health outcome variables.
RESULTS	On average, air purification resulted in a 57% reduction in PM2.5 concentration, from 96.2 to 41.3 μg/m3, within hours of operation. Air purification was significantly associated with decreases in geometric means of several circulating inflammatory and thrombogenic biomarkers, including 17.5% in monocyte chemoattractant protein-1, 68.1% in interleukin-1β, 32.8% in myeloperoxidase, and 64.9% in soluble CD40 ligand. Furthermore, systolic BP, diastolic BP, and fractional exhaled nitrous oxide were significantly decreased by 2.7%, 4.8%, and 17.0% in geometric mean, respectively. The impacts on lung function and vasoconstriction biomarkers were beneficial but not statistically significant.
CONCLUSIONS	This intervention study demonstrated clear cardiopulmonary benefits of indoor air purification among young, healthy adults in a Chinese city with severe ambient particulate air pollution. (Intervention Study on the Health Impact of Air Filters in Chinese Adults; NCT02239744).

표-343. PubMed 논문번호 26025448의 내용 요약

구분	내용
PubMed ID	26025448
TITLE	Association between fine particulate matter exposure and subclinical atherosclerosis: A meta-analysis.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487315588758
AUTHORS	Akintoye Emmanuel, Shi Liuhua, Obaitan Itegbemie, Olusunmade Mayowa, Wang Yan, Newman Jonathan D, Dodson John A, Akintoye Emmanuel, Shi Liuhua, Obaitan Itegbemie, Olusunmade Mayowa, Wang Yan, Newman Jonathan D, Dodson John A
BACKGROUND	Epidemiological studies in humans that have evaluated the association between fine particulate matter (PM2.5) and atherosclerosis have yielded mixed results.
DESIGN	In order to further investigate this relationship, we conducted a comprehensive search for studies published through May 2014 and performed a meta-analysis of all available observational studies that investigated the association between PM2.5 and three noninvasive measures of clinical and subclinical atherosclerosis: carotid intima media thickness, arterial calcification, and ankle-brachial index.
METHODS AND RESULTS	Five reviewers selected studies based on predefined inclusion criteria. Pooled mean change estimates and 95% confidence intervals were calculated using random-effects models. Assessment of between-study heterogeneity was performed where the number of studies was adequate. Our pooled sample included 11,947 subjects for carotid intima media thickness estimates, 10,750 for arterial calcification estimates, and 6497 for ankle-brachial index estimates. Per 10 □g/m(3) increase in PM2.5 exposure, carotid intima media thickness increased by 22.52 □m but this did not reach statistical significance (p = 0.06). We did not find similar associations for arterial calcification (p = 0.44) or ankle-brachial index (p = 0.85).
CONCLUSION	Our meta-analysis supports a relationship between PM2.5 and subclinical atherosclerosis measured by carotid intima media thickness. We did not find a similar relationship between PM2.5 and arterial calcification or ankle-brachial index, although the number of studies was small.

표-344. PubMed 논문번호 26035557의 내용 요약

구분	내용
PubMed ID	26035557
TITLE	Exposure to second-hand smoke and the risk of tuberculosis in children and adults: a systematic review and meta-analysis of 18 observational studies.
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1001835
AUTHORS	Patra Jayadeep, Bhatia Mehak, Suraweera Wilson, Morris Shaun K, Patra Cyril, Gupta Prakash C, Jha Prabhat
BACKGROUND	According to WHO Global Health Estimates, tuberculosis (TB) is among the top ten causes of global mortality and ranks second after cardiovascular disease in most high-burden regions. In this systematic review and meta-analysis, we investigated the role of second-hand smoke (SHS) exposure as a risk factor for TB among children and adults.
METHODS AND FINDINGS	We performed a systematic literature search of PubMed, Embase, Scopus, Web of Science, and Google Scholar up to August 31, 2014. Our a priori inclusion criteria encompassed only original studies where latent TB infection (LTBI) and active TB disease were diagnosed microbiologically, clinically, histologically, or radiologically. Effect estimates were pooled using fixed- and random-effects models. We identified 18 eligible studies, with 30,757 children and 44,432 adult non-smokers, containing SHS exposure and TB outcome data for inclusion in the meta-analysis. Twelve studies assessed children and eight studies assessed adult non-smokers; two studies assessed both populations. Summary relative risk (RR) of LTBI associated with SHS exposure in children was similar to the overall effect size, with high heterogeneity (pooled RR 1.64, 95% CI 1.00-2.83). Children showed a more than 3-fold increased risk of SHS-associated active TB (pooled RR 3.41, 95% CI 1.81-6.45), which was higher than the risk in adults exposed to SHS (summary RR 1.32, 95% CI 1.04-1.68). Positive and significant exposure-response relationships were observed among children under 5 y (RR 5.88, 95% CI 2.09-16.54), children exposed to SHS through any parent (RR 4.20, 95% CI 1.92-9.20), and children living under the most crowded household conditions (RR 5.53, 95% CI 2.36-12.98). Associations for LTBI and active TB disease remained significant after adjustment for age, biomass fuel (BMF) use, and presence of a TB patient in the household, although the meta-analysis was limited to a subset of studies that adjusted for these variables. There was a loss of association with increased risk of LTBI (but not active TB) after adjustment for socioeconomic status (SES) and study quality. The major limitation of this analysis is the high heterogeneity in outcomes among studies of pediatric cases of LTBI and TB disease.
CONCLUSIONS	We found that SHS exposure is associated with an increase in the relative risk of LTBI and active TB after controlling for age, BMF use, and contact with a TB patient, and there was no significant association of SHS exposure with LTBI after adjustment for SES and study quality. Given the high heterogeneity among the primary studies, our analysis may not show sufficient evidence to confirm an association. In addition, considering that the TB burden is highest in countries with increasing SHS exposure, it is important to confirm these results with higher quality studies. Research in this area may have important implications for TB and tobacco control programs, especially for children in settings with high SHS exposure and TB burden.

표-345. PubMed 논문번호 26035590의 내용 요약

구분	내용
PubMed ID	26035590
TITLE	Carotid Stiffness and Physical Activity in Elderly - A Short Report of the SAPALDIA 3 Cohort Study.
JOURNAL	PloS one: 10.1371/journal.pone.0128991
AUTHORS	Caviezel Seraina, Dratva Julia, Schaffner Emmanuel, Schindler Christian, Endes Simon, Autenrieth Christine S, Wanner Miriam, Martin Brian, de Groot Eric, Gaspoz Jean-Michel, K□nzli Nino, Probst-Hensch Nicole, Schmidt-Trucks□ss Arno
INTRODUCTION	Regular physical activity has been shown to reduce cardiovascular disease risk in the general population. While smaller studies in specified groups (highly trained versus untrained individuals) indicate a certain dose-dependent effect of physical activity on the reduction of carotid stiffness (an indicator of subclinical vascular disease), it is unclear whether this association is present in a representative sample. Thus, we investigated this question cross-sectionally in participants from the population-based Swiss Cohort Study on Air Pollution And Lung and Heart Diseases In Adults (SAPALDIA).
METHODS	Self-reported total, moderate and vigorous physical activity and distensibility as a measure of local arterial stiffness among 1636 participants aged 50 to 81 years without clinically manifest diseases were evaluated. Mixed regression models were used to examine associations of physical activity intensity with distensibility.
RESULTS	Vigorous physical activity, but not total nor moderate physical activity, was significantly associated with increased distensibility (= reduced carotid stiffness) in univariate analyses (percent change in the geometric mean and 95% confidence interval per 1 standard deviation increment in vigorous physical activity = 2.54 (0.69; 4.43), $p < 0.01$; in total physical activity = 1.62 (-0.22; 3.50), $p = 0.08$; in moderate physical activity = 0.70 (-1.12; 2.56), $p = 0.45$). These associations disappeared when we additionally adjusted for age.
CONCLUSION	After adjustment for the most important confounders and risk factors, we found no evidence for an association of physical activity with carotid stiffness in the general middle aged to elderly population.

표-346. PubMed 논문번호 26057255의 내용 요약

구분	내용
PubMed ID	26057255
TITLE	Long-term effects of elemental composition of particulate matter on inflammatory blood markers in European cohorts.
JOURNAL	Environment international: 10.1016/j.envint.2015.05.008
AUTHORS	Hampel Regina, Peters Annette, Beelen Rob, Brunekreef Bert, Cyrys Josef, de Faire Ulf, de Hoogh Kees, Fuks Kateryna, Hoffmann Barbara, H□ls Anke, Imboden Medea, Jedynska Aleksandra, Kooter Ingeborg, Koenig Wolfgang, K□nzli Nino, Leander Karin, Magnusson Patrik, M□nnist□ Satu, Penell Johanna, Pershagen G□ran, Phuleria Harish, Probst-Hensch Nicole, Pundt Noreen, Schaffner Emmanuel, Schikowski Tamara, Sugiri Dorothea, Tiittanen Pekka, Tsai Ming-Yi, Wang Meng, Wolf Kathrin, Lanki Timo
BACKGROUND	Epidemiological studies have associated long-term exposure to ambient particulate matter with increased mortality from cardiovascular and respiratory disorders. Systemic inflammation is a plausible biological mechanism behind this association. However, it is unclear how the chemical composition of PM affects inflammatory responses.
OBJECTIVES	To investigate the association between long-term exposure to elemental components of PM and the inflammatory blood markers high-sensitivity C-reactive protein (hsCRP) and fibrinogen as part of the European ESCAPE and TRANSPHORM multi-center projects.
METHODS	In total, 21,558 hsCRP measurements and 17,428 fibrinogen measurements from cross-sections of five and four cohort studies were available, respectively. Residential long-term concentrations of particulate matter <10 μm (PM10) and <2.5 μm (PM2.5) in diameter and selected elemental components (copper, iron, potassium, nickel, sulfur, silicon, vanadium, zinc) were estimated based on land-use regression models. Associations between components and inflammatory markers were estimated using linear regression models for each cohort separately. Cohort-specific results were combined using random effects meta-analysis. As a sensitivity analysis the models were additionally adjusted for PM mass.
RESULTS	A 5ng/m(3) increase in PM2.5 copper and a 500ng/m(3) increase in PM10 iron were associated with a 6.3% [0.7; 12.3%] and 3.6% [0.3; 7.1%] increase in hsCRP, respectively. These associations between components and fibrinogen were slightly weaker. A 10ng/m(3) increase in PM2.5 zinc was associated with a 1.2% [0.1; 2.4%] increase in fibrinogen; confidence intervals widened when additionally adjusting for PM2.5.
CONCLUSIONS	Long-term exposure to transition metals within ambient particulate matter, originating from traffic and industry, may be related to chronic systemic inflammation providing a link to long-term health effects of particulate matter.

표-347. PubMed 논문번호 26065915의 내용 요약

구분	내용
PubMed ID	26065915
TITLE	Lung Function in Rural Guatemalan Women Before and After a Chimney Stove Intervention to Reduce Wood Smoke Exposure: Results From the Randomized Exposure Study of Pollution Indoors and Respiratory Effects and Chronic Respiratory Effects of Early Childhood Exposure to Respirable Particulate Matter Study.
JOURNAL	Chest: 10.1378/chest.15-0261
AUTHORS	Guarnieri Michael, Diaz Esperanza, Pope Daniel, Eisen Ellen A, Mann Jennifer, Smith Kirk R, Smith-Sivertsen Tone, Bruce Nigel G, Balmes John R
BACKGROUND	COPD is the third most frequent cause of death globally, with much of this burden attributable to household biomass smoke exposure in developing countries. As biomass smoke exposure is also associated with cardiovascular disease, lower respiratory infection, lung cancer, and cataracts, it presents an important target for public health intervention.
METHODS	Lung function in Guatemalan women exposed to wood smoke from open fires was measured throughout the Randomized Exposure Study of Pollution Indoors and Respiratory Effects (RESPIRE) stove intervention trial and continued during the Chronic Respiratory Effects of Early Childhood Exposure to Respirable Particulate Matter (CRECER) cohort study. In RESPIRE, early stove households received a chimney woodstove at the beginning of the 18-month trial, and delayed stove households received a stove at trial completion. Personal exposure to wood smoke was assessed with exhaled breath carbon monoxide (CO) and personal CO tubes. Change in lung function between intervention groups and as a function of wood smoke exposure was assessed using random effects models.
RESULTS	Of 306 women participating in both studies, acceptable spirometry was collected in 129 early stove and 136 delayed stove households (n = 265), with a mean follow-up of 5.6 years. Despite reduced wood smoke exposures in early stove households, there were no significant differences in any of the measured spirometric variables during the study period (FEV1, FVC, FEV1/FVC ratio, and annual change) after adjustment for confounding.
CONCLUSIONS	In these young Guatemalan women, there was no association between lung function and early randomization to a chimney stove or personal wood smoke exposure. Future stove intervention trials should incorporate cleaner stoves, longer follow-up, or potentially susceptible groups to identify meaningful differences in lung function.

표-348. PubMed 논문번호 26068905의 내용 요약

구분	내용
PubMed ID	26068905
TITLE	A cross-sectional analysis of polycyclic aromatic hydrocarbons and diesel particulate matter exposures and hypertension among individuals of Mexican origin.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-015-0039-2
AUTHORS	Bangia Komal S, Symanski Elaine, Strom Sara S, Bondy Melissa
BACKGROUND	Epidemiological studies have found that particulate matter is associated with increases in blood pressure. Yet, less is known about the effects of specific sources or constituents of particulate matter, such as diesel particulate matter or polycyclic aromatic hydrocarbons (PAHs). We evaluated associations between self-reported hypertension and residential air levels of diesel particulate matter and PAHs among individuals of Mexican origin living in a large inner city.
METHODS	The Mano a Mano cohort (established in 2001 by the University of Texas MD Anderson Cancer Center) is comprised of individuals of Mexican origin residing in Houston, Texas. Using geographical information systems, we linked modeled annual estimates of PAHs and diesel particulate matter at the census tract level from the 2002 and 2005 U.S. Environmental Protection Agency's National-Scale Air Toxics Assessment to baseline residential addresses of cohort members who enrolled from 2001 to 2003 or 2004 to 2006, respectively. For each enrollment period, we applied mixed-effects logistic regression models to determine associations between diesel particulate matter and PAHs, separately, and self-reported hypertension while adjusting for confounders and the clustering of observations within census tracts and households.
RESULTS	The study population consisted of 11218 participants of which 77% were women. The mean participant age at baseline was 41 years. Following adjustment for age, there was a dose-dependent, positive association between PAHs and hypertension (medium exposure, adjusted odds ratio (OR) = 1.09, 95% CI: 0.88-1.36; high exposure, OR = 1.40, 95% CI: 1.01-1.94) for individuals enrolled during 2001-2003; associations were generally similar in magnitude, but less precise, following adjustment for age, gender, smoking, and BMI. No association was detected for the later period. There was no evidence of an association between residential levels of diesel particulate matter and hypertension.
CONCLUSIONS	This study builds on a limited number of prior investigations of the association between ambient air levels of PAHs or diesel particulate matter and hypertension by focusing on a relatively young cohort of predominantly adult women of Mexican origin. Future analyses are warranted to explore associations in the cohort using incident hypertension when sufficient data become available and to further examine associations between specific chemical constituents of particulate matter and hypertension in this and other populations.

표-349. PubMed 논문번호 26075655의 내용 요약

구분	내용
PubMed ID	26075655
TITLE	Ambient air pollution and neurotoxicity on brain structure: Evidence from women's health initiative memory study.
JOURNAL	Annals of neurology: 10.1002/ana.24460
AUTHORS	Chen Jiu-Chiuan, Wang Xinhui, Wellenius Gregory A, Serre Marc L, Driscoll Ira, Casanova Ramon, McArdle John J, Manson JoAnn E, Chui Helena C, Espeland Mark A
OBJECTIVE	The aim of this study was to examine the putative adverse effects of ambient fine particulate matter (PM2.5 : PM with aerodynamic diameters <2.5 μm) on brain volumes in older women.
METHODS	We conducted a prospective study of 1,403 community-dwelling older women without dementia enrolled in the Women's Health Initiative Memory Study, 1996-1998. Structural brain magnetic resonance imaging scans were performed at the age of 71-89 years in 2005-2006 to obtain volumetric measures of gray matter (GM) and normal-appearing white matter (WM). Given residential histories and air monitoring data, we used a spatiotemporal model to estimate cumulative PM2.5 exposure in 1999-2006. Multiple linear regression was employed to evaluate the associations between PM2.5 and brain volumes, adjusting for intracranial volumes and potential confounders.
RESULTS	Older women with greater PM2.5 exposures had significantly smaller WM, but not GM, volumes, independent of geographical region, demographics, socioeconomic status, lifestyles, and clinical characteristics, including cardiovascular risk factors. For each interquartile increment (3.49 μg/m(3)) of cumulative PM2.5 exposure, the average WM volume (WMV; 95% confidence interval) was 6.23cm(3) (3.72-8.74) smaller in the total brain and 4.47cm(3) (2.27-6.67) lower in the association areas, equivalent to 1 to 2 years of brain aging. The adverse PM2.5 effects on smaller WMVs were present in frontal and temporal lobes and corpus callosum (all p values <0.01). Hippocampal volumes did not differ by PM2.5 exposure.
INTERPRETATION	PM2.5 exposure may contribute to WM loss in older women. Future studies are needed to determine whether exposures result in myelination disturbance, disruption of axonal integrity, damages to oligodendrocytes, or other WM neuropathologies.

표-350. PubMed 논문번호 26079662의 내용 요약

구분	내용
PubMed ID	26079662
TITLE	Occupational Exposure to PM2.5 and Incidence of Ischemic Heart Disease: Longitudinal Targeted Minimum Loss-based Estimation.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000329
AUTHORS	Brown Daniel M, Petersen Maya, Costello Sadie, Noth Elizabeth M, Hammond Katherine, Cullen Mark, Laan Mark van der, Eisen Ellen
BACKGROUND	We investigated the incidence of ischemic heart disease (IHD) in relation to accumulated exposure to particulate matter (PM) in a cohort of aluminum workers. We adjusted for time varying confounding characteristic of the healthy worker survivor effect, using a recently introduced method for the estimation of causal target parameters.
METHODS	Applying longitudinal targeted minimum loss-based estimation, we estimated the difference in marginal cumulative risk of IHD in the cohort comparing counterfactual outcomes if always exposed above to always exposed below a PM2.5 exposure cut-off. Analyses were stratified by sub-cohort employed in either smelters or fabrication facilities. We selected two exposure cut-offs a priori, at the median and 10th percentile in each sub-cohort.
RESULTS	In smelters, the estimated IHD risk difference after 15 years of accumulating PM2.5 exposure during follow-up was 2.9% (0.6%, 5.1%) using the 10th percentile cut-off of 0.10 mg/m. For fabrication workers, the difference was 2.5% (0.8%, 4.1%) at the 10th percentile of 0.06 mg/m. Using the median exposure cut-off, results were similar in direction but smaller in size. We present marginal incidence curves describing the cumulative risk of IHD over the course of follow-up for each sub-cohort under each intervention regimen.
CONCLUSIONS	The accumulation of exposure to PM2.5 appears to result in higher risks of IHD in both aluminum smelter and fabrication workers. This represents the first longitudinal application of targeted minimum loss-based estimation, a method for generating doubly robust semi-parametric efficient substitution estimators of causal parameters, in the fields of occupational and environmental epidemiology.

표-351. PubMed 논문번호 26087770의 내용 요약

구분	내용
PubMed ID	26087770
TITLE	Long-term exposure to fine particulate matter and incidence of type 2 diabetes mellitus in a cohort study: effects of total and traffic-specific air pollution.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-015-0031-x
AUTHORS	Weinmayr Gudrun, Hennig Frauke, Fuks Kateryna, Nonnemacher Michael, Jakobs Hermann, M□hlenkamp Stefan, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara, Moebus Susanne
BACKGROUND	Studies investigating the link between long-term exposure to air pollution and incidence of diabetes are still scarce and results are inconsistent, possibly due to different compositions of the particle mixture. We investigate the long-term effect of traffic-specific and total particulate matter (PM) and road proximity on cumulative incidence of diabetes mellitus (mainly type 2) in a large German cohort.
METHODS	We followed prospectively 3607 individuals without diabetes at baseline (2000-2003) from the Heinz Nixdorf Recall Study in Germany (mean follow-up time 5.1 years). Mean annual exposures to total as well as traffic-specific PM10 and PM2.5 at residence were estimated using a chemistry transport model (EURAD, 1 km(2) resolution). Effect estimates for an increase of 1 μg/m(3) in PM were obtained with Poisson regression adjusting for sex, age, body mass index, lifestyle factors, area-level and individual-level socio-economic status, and city.
RESULTS	331 incident cases developed. Adjusted RRs for total PM10 and PM2.5 were 1.05 (95%-CI: 1.00;1.10) and 1.03 (95%-CI: 0.95;1.12), respectively. Markedly higher point estimates were found for local traffic-specific PM with RRs of 1.36 (95%-CI: 0.98;1.89) for PM10 and 1.36 (95%-CI: 0.97;1.89) for PM2.5. Individuals living closer than 100 m to a busy road had a more than 30% higher risk (1.37;95%-CI: 1.04;1.81) than those living further than 200 m away.
CONCLUSIONS	Long-term exposure to total PM increases type two diabetes risk in the general population, as does living close to a major road. Local traffic-specific PM was related to higher risks for type two diabetes than total PM.

표-352. PubMed 논문번호 26090776의 내용 요약

구분	내용
PubMed ID	26090776
TITLE	Use of the Adaptive LASSO Method to Identify PM2.5 Components Associated with Blood Pressure in Elderly Men: The Veterans Affairs Normative Aging Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1409021
AUTHORS	Dai Lingzhen, Koutrakis Petros, Coull Brent A, Sparrow David, Vokonas Pantel S, Schwartz Joel D, Dai Lingzhen, Koutrakis Petros, Coull Brent A, Sparrow David, Vokonas Pantel S, Schwartz Joel D
BACKGROUND	PM2.5 (particulate matter ≤ 2.5 μm) has been associated with adverse cardiovascular outcomes, but it is unclear whether specific PM2.5 components, particularly metals, may be responsible for cardiovascular effects.
OBJECTIVES	We aimed to determine which PM2.5 components are associated with blood pressure in a longitudinal cohort.
METHODS	We fit linear mixed-effects models with the adaptive LASSO penalty to longitudinal data from 718 elderly men in the Veterans Affairs Normative Aging Study, 1999-2010. We controlled for PM2.5 mass, age, body mass index, use of antihypertensive medication (ACE inhibitors, non-ophthalmic beta blockers, calcium channel blockers, diuretics, and angiotensin receptor antagonists), smoking status, alcohol intake, years of education, temperature, and season as fixed effects in the models, and additionally applied the adaptive LASSO method to select PM2.5 components associated with blood pressure. Final models were identified by the Bayesian Information Criterion (BIC).
RESULTS	For systolic blood pressure (SBP), nickel (Ni) and sodium (Na) were selected by the adaptive LASSO, whereas only Ni was selected for diastolic blood pressure (DBP). An interquartile range increase (2.5 ng/m3) in 7-day moving-average Ni was associated with 2.48-mmHg (95% CI: 1.45, 3.50 mmHg) increase in SBP and 2.22-mmHg (95% CI: 1.69, 2.75 mmHg) increase in DBP, respectively. Associations were comparable when the analysis was restricted to study visits with PM2.5 below the 75th percentile of the distribution (12 μg/m3).
CONCLUSIONS	Our study suggested that exposure to ambient Ni was associated with increased blood pressure independent of PM2.5 mass in our study population of elderly men. Further research is needed to confirm our findings, assess generalizability to other populations, and identify potential mechanisms for Ni effects.
CITATION	Dai L, Koutrakis P, Coull BA, Sparrow D, Vokonas PS, Schwartz JD. 2016. Use of the adaptive LASSO method to identify PM2.5 components associated with blood pressure in elderly men: the Veterans Affairs Normative Aging Study. Environ Health Perspect 124:120-125; http://dx.doi.org/10.1289/ehp.1409021.

표-353. PubMed 논문번호 26092807의 내용 요약

구분	내용
PubMed ID	26092807
TITLE	Association of air pollution with cognitive functions and its modification by APOE gene variants in elderly women.
JOURNAL	Environmental research: 10.1016/j.envres.2015.06.009
AUTHORS	Schikowski Tamara, Vossoughi Mohammad, Vierk□tter Andrea, Schulte Thomas, Teichert Tom, Sugiri Dorothee, Fehsel Karin, Tzivian Lilian, Bae Il-seok, Ranft Ulrich, Hoffmann Barbara, Probst-Hensch Nicole, Herder Christian, Kr□mer Ursula, Luckhaus Christian
BACKGROUND	Epidemiological studies have shown effects of long-term exposure to air pollution on cardiovascular and respiratory health. However, studies investigating the effects of air pollution on cognition and brain function are limited. We investigated if neurocognitive functions are associated with air pollution exposure and whether apolipoprotein E (ApoE) alleles modify the association of air pollution exposure with cognition.
METHODS	We investigated 789 women from the SALIA cohort during the 22-year follow-up examination (2008-2009). Exposure to particulate matter (PM) size fractions and nitrogen oxides (NOx) were assigned to home addresses. Traffic indicators were used to assess residential proximity to high traffic load. Level of cognitive performance was assessed using the CERAD-Plus test. Air pollution effects on cognitive functioning were estimated cross-sectionally using adjusted linear regression models.
RESULTS	Air pollution was negatively associated with cognitive function and cognitive performance in the subtests for semantic memory and visuo-construction. Significant associations could be observed for figure copying with an interquartile range increase of NO2 (β=-0.28 (95%CI:-0.44;-0.12)), NOx (β=-0.25 (95%CI:-0.40;-0.09)), PM10 (β=-0.14 (95%CI:-0.26;-0.02)) and PM2.5 (β=-0.19 (95%CI:-0.36;-0.02)). The association with traffic load was significant in carriers of one or two ApoE □4 risk alleles.
CONCLUSION	In this study of elderly women, markers of air pollution were associated with cognitive impairment in the visuospatial domain. The association of traffic exposure is significant in participants carrying the ApoE ε4 risk allele.

표-354. PubMed 논문번호 26105036의 내용 요약

구분	내용
PubMed ID	26105036
TITLE	Acute and recent air pollution exposure and cardiovascular events at labour and delivery.
JOURNAL	Heart (British Cardiac Society): 10.1136/heartjnl-2014-307366
AUTHORS	M□nnist□ Tuija, Mendola Pauline, Laughon Grantz Katherine, Leishear Kira, Sundaram Rajeshwari, Sherman Seth, Ying Qi, Liu Danping
OBJECTIVE	To study the relationship between acute air pollution exposure and cardiovascular events during labour/delivery.
METHODS	The Consortium on Safe Labor (2002-2008), an observational US cohort with 223,502 singleton deliveries provided electronic medical records. Air pollution exposure was estimated by modified Community Multiscale Air Quality models. Cardiovascular events (cardiac failure/arrest, stroke, myocardial infarcts and other events) were recorded in the hospital discharge records for 687 pregnancies (0.3%). Logistic regression with generalised estimating equations estimated the relationship between cardiovascular events and daily air pollutant levels for delivery day and the 7 days preceding delivery.
RESULTS	Increased odds of cardiovascular events were observed for each IQR increase in exposure to nitric oxides at 5 and 6 days prior to delivery (OR=1.17, 99% CI 1.04 to 1.30 and OR=1.15, 1.03 to 1.28, respectively). High exposure to toxic air pollution species such as ethylbenzene (OR=1.50, 1.08 to 2.09), m-xylene (OR=1.54, 1.11 to 2.13), o-xylene (OR=1.51, 1.09 to 2.09), p-xylene (OR=1.43, 1.03 to 1.99) and toluene (OR=1.42, 1.02 to 1.97) at 5 days prior to delivery were also associated with cardiovascular events. Decreased odds of events were observed with exposure to ozone.
CONCLUSIONS	Air pollution in the days prior to delivery, especially nitrogen oxides and some toxic air pollution species, was associated with increased risk of cardiovascular events during the labour/delivery admission.

표-355. PubMed 논문번호 26121293의 내용 요약

구분	내용
PubMed ID	26121293
TITLE	Zinc compound air releases from Toxics Release Inventory facilities and cardiovascular disease mortality rates.
JOURNAL	Environmental research: 10.1016/j.envres.2015.06.022
AUTHORS	Chen Bo-chiuan, Luo Juhua, Hendryx Michael
BACKGROUND	Inhaled zinc has been found in association with cardiopulmonary toxicity. However, limited human epidemiologic studies are available. This study analyzed the association between covariate-adjusted cardiovascular (CVD) mortality rates and zinc compound air releases in the United States.
METHODS	We conducted an ecological analysis on the association between zinc compound air releases for 1991-2000 using the Toxics Release Inventory database and average age-adjusted CVD mortality for 2006-2010, adjusting for race/ethnicity composition and several health and socioeconomic factors. Models were estimated for males and females and for metropolitan and nonmetropolitan counties.
RESULTS	Zinc compound air releases were positively associated with increased adjusted CVD mortality rates in all four models (β=0.0085, p<0.0001 for males in nonmetropolitan counties; β=0.0093, p<0.0001 for males in metropolitan counties; β=0.0145, p<0.0001 for females in nonmetropolitan counties; and β=0.0098, p<0.0001 for females in metropolitan counties). Results were largely robust to various sensitivity analyses.
CONCLUSION	This study provides epidemiological evidence for possible CVD health impacts of inhaled zinc in the United States. Although the strongest effect was found for females in nonmetropolitan counties, the associations were consistent in nonmetropolitan or metropolitan counties for both genders.

표-356. PubMed 논문번호 26142971의 내용 요약

구분	내용
PubMed ID	26142971
TITLE	Short-term exposure to ambient air pollution and coronary heart disease mortality in 8 Chinese cities.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2015.06.050
AUTHORS	Li Huichu, Chen Renjie, Meng Xia, Zhao Zhuohui, Cai Jing, Wang Cuicui, Yang Changyuan, Kan Haidong
BACKGROUND	Coronary heart disease (CHD) accounted for a large fraction of death globally. The association between air pollution and CHD has been reported, but evidence from highly-polluted regions was scarce. We aimed to estimate the acute effects of outdoor air pollution on daily CHD mortality in China.
METHODS	We collected daily CHD deaths in 8 large Chinese cities from 1996 to 2008. We firstly obtained the city-specific effect estimates of air pollution using generalized additive models with quasi-Poisson regression, controlling for time trends, meteorological indicators and day of the week. The random-effect model in meta-analysis was used to pool the exposure-response relationships.
RESULTS	We identified a total of 0.13 million CHD deaths. On average, an increase of 10 μg/m(3) in 2-day moving average concentrations of particulate matter$\leq$10 μm in aerodynamic diameter (PM10), sulfur dioxide (SO2) and nitrogen dioxide (NO2) was significantly associated with increases of 0.36% [95% confidence intervals (CIs): 0.12%, 0.61%], 0.86% (95% CIs: 0.30%,1.41%) and 1.30% (95% CIs: 0.45%, 2.14%) in daily CHD mortality over the 8 Chinese cities, respectively. The pooled exposure-response curves were almost linear and no apparent thresholds were identified. The effects were more pronounced in cities with lower levels of air pollution. The effects of PM10 and NO2 were more robust than SO2.
CONCLUSION	Our findings contributed to the very limited evidence regarding the hazardous effects of ambient air pollution on CHD mortality in highly-polluted regions such as China.

표-357. PubMed 논문번호 26163546의 내용 요약

구분	내용
PubMed ID	26163546
TITLE	Long-term residential exposure to urban air pollution, and repeated measures of systemic blood markers of inflammation and coagulation.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2014-102800
AUTHORS	Viehmann Anja, Hertel Sabine, Fuks Kateryna, Eisele Lewin, Moebus Susanne, M□hlenkamp Stefan, Nonnemacher Michael, Jakobs Hermann, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara
BACKGROUND	In several studies, exposure to fine particulate matter (PM) has been associated with inflammation, with inconsistent results. We used repeated measurements to examine the association of long-term fine and ultrafine particle exposure with several blood markers of inflammation and coagulation.
METHODS	We used baseline (2000-2003) and follow-up (2006-2008) data from the Heinz Nixdorf Recall Study, a German population-based prospective cohort of 4814 participants. A chemistry transport model was applied to model daily surface concentrations of PM air pollutants (PM10, PM2.5) and particle number on a grid of 1 km(2). Applying mixed regression models, we analysed associations of long-term (mean of 365 days prior to blood draw) particle exposure at each participant's residence with the level of high-sensitivity C reactive protein (hs-CRP), fibrinogen, platelet and white cell count (WCC), adjusting for short-term PM exposure (moving averages of 1-7 days), personal characteristics, season, ambient temperature (1-5 days), ozone and time trend.
RESULTS	We analysed 6488 observations: 3275 participants with baseline data and 3213 with follow-up data. An increase of 2.4 □g/m(3) in long-term PM2.5 was associated with an adjusted increase of 5.4% (95% CI 0.6% to 10.5%) in hs-CRP and of 2.3% (95% CI 1.4% to 3.3%) in the platelet count. Fibrinogen and WCC were not associated with long-term particle exposure.
CONCLUSIONS	In this population-based cohort, we found associations of long-term exposure to PM with markers of inflammation (hs-CRP) and coagulation (platelets). This finding supports the hypothesis that inflammatory processes might contribute to chronic effects of air pollution on cardiovascular disease.

표-358. PubMed 논문번호 26163761의 내용 요약

구분	내용
PubMed ID	26163761
TITLE	Long term effects of residential NO(x) exposure on total and cause-specific mortality and incidence of myocardial infarction in a Swedish cohort.
JOURNAL	Environmental research: 10.1016/j.envres.2015.06.045
AUTHORS	Stockfelt Leo, Andersson Eva M, Moln□r Peter, Rosengren Annika, Wilhelmsen Lars, Sallsten Gerd, Barregard Lars
BACKGROUND AND AIMS	Exposure to air pollution has been linked to total and cardiopulmonary mortality. However, few studies have examined the effects of exposure over decades, or which time windows of long term exposure are most relevant. We investigated the long term effects of residential air pollution on total and cause-specific mortality and incidence of myocardial infarction in a well-characterized cohort of men in Sweden.
METHODS	A cohort of 7494 men in Gothenburg was examined in 1970-1973 and followed subsequently to determine predictors of cardiovascular disease. We collected data on residential address and cause-specific mortality for the years 1973-2007. Each individual was assigned yearly nitrogen oxides (NOx) exposure based on dispersion models. Using multivariable Cox regression and generalized additive models with time-dependent exposure, we studied the association between three different time windows of residential NOx exposure, and selected outcomes.
RESULTS	In the years 1973-2007, a total of 5669 deaths, almost half of which were due to cardiovascular diseases, occurred in the cohort. Levels of NOx exposure decreased during the study period, from a median of 38 □g/m(3) in 1973 to 17 □g/m(3) in 2007. Total non-accidental mortality was associated with participants' NOx exposure in the last year (the year of outcome) (HR 1.03, 95% CI 1.01-1.05, per 10 □g/m(3)), with the mean NOx exposure during the last 5 years, and with the mean NOx exposure since enrolment (HR 1.02, 95% CI 1.01-1.04 for both). The associations were similar (HR 1.01-1.03), but generally not statistically significant, for cardiovascular, ischemic heart disease, and acute myocardial infarction mortality, and weaker for cerebrovascular and respiratory mortality. There was no association between NOx exposure and incident myocardial infarction.
DISCUSSION AND CONCLUSIONS	Long term residential exposure to NOx at these relatively low exposure levels in Gothenburg was associated with total non-accidental mortality. The association was as strong for NOx exposure in the last year as for longer exposure windows. The effect was near linear, and only marginally affected by confounders and effect modifiers. The improved air quality in Gothenburg has by these estimates led to a 6% decrease in excess non-accidental mortality during the study period.

표-359. PubMed 논문번호 26186643의 내용 요약

구분	내용
PubMed ID	26186643
TITLE	Associations between particulate matter composition and childhood blood pressure-The PIAMA study.
JOURNAL	Environment international: 10.1016/j.envint.2015.07.010
AUTHORS	Bilenko Natalya, Brunekreef Bert, Beelen Rob, Eeftens Marloes, de Hoogh Kees, Hoek Gerard, Koppelman Gerard H, Wang Meng, van Rossem Lenie, Gehring Ulrike
BACKGROUND	Childhood blood pressure is an important predictor of hypertension and cardiovascular disease in adulthood. Evidence for an association between ambient particulate matter (PM) exposure and blood pressure is increasing, but little is known about the relevance of different PM constituents.
OBJECTIVES	We investigated the association between particulate matter composition and blood pressure at age 12 years.
METHODS	Annual average concentrations of copper, iron, potassium, nickel, sulfur, silicon, vanadium, and zinc in particles with diameters of less than 2.5 μm (PM2.5) and 10 μm (PM10) were estimated by land-use regression modeling for the home addresses of the participants of the prospective PIAMA birth cohort study. Associations between element concentrations and blood pressure measurements performed at age 12 years were investigated by linear regression with and without adjustment for confounders.
RESULTS	After adjustment for potential confounders we found statistically significant positive associations of diastolic blood pressure with iron, silicon, and potassium in PM10 in children who lived at the same address since birth [mean difference (95% confidence interval) 0.67 (0.02;1.31) mmHg, 0.85 (0.18;1.52) mmHg, and 0.75 (0.09;1.41) mmHg, respectively, per interquartile range increase in exposure]. Also, we found marginally significant ($p<0.1$) positive associations between iron and silicon in PM2.5 and diastolic blood pressure. Part of the observed effects was found to be attributable to NO2, a marker of exhaust traffic emissions.
CONCLUSIONS	Exposure to particulate matter constituents, in particular iron may increase blood pressure in children. The possible association with iron may indicate the health relevance of non-exhaust emissions of traffic.

표-360. PubMed 논문번호 26188335의 내용 요약

구분	내용
PubMed ID	26188335
TITLE	Is air pollution associated with increased risk of cognitive decline? A systematic review.
JOURNAL	Age and ageing: 10.1093/ageing/afv087
AUTHORS	Peters Ruth, Peters Jean, Booth Andrew, Mudway Ian
INTRODUCTION	exposure to air pollution has been shown to increase risk of inflammatory processes and risk of cardiovascular mortality. Such exposure may therefore also be a risk factor for cognitive impairment/dementia.
METHOD	a systematic review of the literature was conducted with databases searched using keywords for air pollution, cognitive decline and dementia. All identified abstracts and potentially relevant articles were double read. For those papers meeting the inclusion criteria, summary tables were prepared and papers quality assessed.
RESULTS	from 1,551 abstracts identified, 10 articles were retrieved of which two were rejected. Of the eight remaining six reported prevalent cognitive assessment with historical pollution exposure and two incident cognitive decline, also with historical pollution exposure. In general, an association was reported between exposure and poorer prevalent measures of cognitive function. Data were mixed for incident cognitive decline with one study finding an association and the other not. Reports were limited by a lack of detailed reporting, use of proxy measures of pollution exposure and a lack of clarity regarding cognitive testing methodology and analysis.
CONCLUSION	this systematic review highlights that there is some evidence of a potential association between air pollution and subsequent cognitive decline. Further work is clearly required and longitudinal analysis of ongoing cohort studies or new research would add much needed clarity to this area.

표-361. PubMed 논문번호 26188829의 내용 요약

구분	내용
PubMed ID	26188829
TITLE	Risk of all-cause mortality and cardiovascular disease associated with secondhand smoke exposure: a systematic review and meta-analysis.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2015.07.011
AUTHORS	Lv Xiaofei, Sun Jichao, Bi Yufang, Xu Min, Lu Jieli, Zhao Liebin, Xu Yu
BACKGROUND	Emerging studies have assessed the association between secondhand smoke (SHS) exposure and cardiovascular disease (CVD) as well as all-cause mortality. However, findings were not consistent due to the heterogeneity of study characteristics.
METHODS	PubMed and Embase were searched through May 2014 for prospective cohort and case-control studies investigating the associations of SHS exposure in never smokers with all-cause mortality and the risk of CVD. The main analysis was performed in studies using self-reported SHS exposure and secondary analysis was performed in studies using objectively measured SHS exposure. Summary estimates were calculated using random-effects models.
RESULTS	Twenty-three prospective and 17 case-control studies were included. The pooled relative risks (RR) for never smokers exposed to SHS in comparison with those unexposed were 1.18 [95% confidence interval (CI): 1.10-1.27] for all-cause mortality (12 studies), and 1.23 (1.16-1.31) for CVD (38 studies). The association of SHS exposure with CVD was markedly stronger among studies conducted in China (RR=1.65, 95% CI 1.27-2.13) than that in the US (RR=1.09, 95% CI 1.03-1.16). Studies using objectively measured SHS exposure demonstrated a slightly higher risk for CVD compared with those using self-reported SHS exposure.
CONCLUSIONS	Exposure to SHS significantly increased the risk for all-cause mortality and CVD. The risk associated with SHS exposure was large in China while the risk was only modest in the US. Studies using objectively measured SHS exposure may yield a higher risk of CVD than those using self-reported SHS exposure.

표-362. PubMed 논문번호 26208764의 내용 요약

구분	내용
PubMed ID	26208764
TITLE	Influence of meteorological conditions on hospital admission in patients with acute coronary syndrome with and without ST-segment elevation: Results of the AIRACOS study.
JOURNAL	Medicina intensiva: 10.1016/j.medin.2015.04.007
AUTHORS	Dominguez-Rodriguez A, Juarez-Prera R A, Rodr□guez S, Abreu-Gonzalez P, Avanzas P, Dominguez-Rodriguez A, Juarez-Prera R A, Rodr□guez S, Abreu-Gonzalez P, Avanzas P
OBJECTIVE	Evaluate whether the meterological parameters affecting revenues in patients with ST-segment and non-ST-segment elevation ACS.
DESIGN	A prospective cohort study was carried out.
SETTING	Coronary Care Unit of Hospital Universitario de Canarias
PATIENTS	We studies a total of 307 consecutive patients with a diagnosis of ST-segment and non-ST-segment elevation ACS. We analyze the average concentrations of particulate smaller than 10 and 2.5 μm diameter, particulate black carbon, the concentrations of gaseous pollutants and meteorological parameters (wind speed, temperature, relative humidity and atmospheric pressure) that were exposed patients from one day up to 7 days prior to admission.
INTERVENTIONS	None.
VARIABLES OF INTEREST	Demographic, clinical, atmospheric particles, concentrations of gaseous pollutants and meterological parameters.
RESULTS	A total of 138 (45%) patients were classified as ST-segment and 169 (55%) as non-ST-segment elevation ACS. No statistically significant differences in exposure to atmospheric particles in both groups. Regarding meteorological data, we did not find statistically significant differences, except for higher atmospheric pressure in ST-segment elevation ACS (999.6±2.6 vs. 998.8±2.5 mbar, P=.008). Multivariate analysis showed that atmospheric pressure was significant predictor of ST-segment elevation ACS presentation (OR: 1.14, 95% CI: 1.04-1.24, P=.004).
CONCLUSIONS	In the patients who suffer ACS, the presence of higher number of atmospheric pressure during the week before the event increase the risk that the ST-segment elevation ACS.

표-363. PubMed 논문번호 26219103의 내용 요약

구분	내용
PubMed ID	26219103
TITLE	Desert Dust Outbreaks in Southern Europe: Contribution to Daily PM₁ □ Concentrations and Short-Term Associations with Mortality and Hospital Admissions.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1409164
AUTHORS	Stafoggia Massimo, Zauli-Sajani Stefano, Pey Jorge, Samoli Evangelia, Alessandrini Ester, Basaga□a Xavier, Cernigliaro Achille, Chiusolo Monica, Demaria Moreno, D□az Julio, Faustini Annunziata, Katsouyanni Klea, Kelessis Apostolos G, Linares Cristina, Marchesi Stefano, Medina Sylvia, Pandolfi Paolo, P□rez Noem□, Querol Xavier, Randi Giorgia, Ranzi Andrea, Tobias Aurelio, Forastiere Francesco, Stafoggia Massimo, Zauli-Sajani Stefano, Pey Jorge, Samoli Evangelia, Alessandrini Ester, Basaga□a Xavier, Cernigliaro Achille, Chiusolo Monica, Demaria Moreno, D□az Julio, Faustini Annunziata, Katsouyanni Klea, Kelessis Apostolos G, Linares Cristina, Marchesi Stefano, Medina Sylvia, Pandolfi Paolo, P□rez Noem□, Querol Xavier, Randi Giorgia, Ranzi Andrea, Tobias Aurelio, Forastiere Francesco
BACKGROUND	Evidence on the association between short-term exposure to desert dust and health outcomes is controversial.
OBJECTIVES	We aimed to estimate the short-term effects of particulate matter ≤ 10 μm (PM10) on mortality and hospital admissions in 13 Southern European cities, distinguishing between PM10 originating from the desert and from other sources.
METHODS	We identified desert dust advection days in multiple Mediterranean areas for 2001-2010 by combining modeling tools, back-trajectories, and satellite data. For each advection day, we estimated PM10 concentrations originating from desert, and computed PM10 from other sources by difference. We fitted city-specific Poisson regression models to estimate the association between PM from different sources (desert and non-desert) and daily mortality and emergency hospitalizations. Finally, we pooled city-specific results in a random-effects meta-analysis.
RESULTS	On average, 15% of days were affected by desert dust at ground level (desert PM10 > 0 μg/m3). Most episodes occurred in spring-summer, with increasing gradient of both frequency and intensity north-south and west-east of the Mediterranean basin. We found significant associations of both PM10 concentrations with mortality. Increases of 10 μg/m3 in non-desert and desert PM10 (lag 0-1 days) were associated with increases in natural mortality of 0.55% (95% CI: 0.24, 0.87%) and 0.65% (95% CI: 0.24, 1.06%), respectively. Similar associations were estimated for cardio-respiratory mortality and hospital admissions.
CONCLUSIONS	PM10 originating from the desert was positively associated with mortality and hospitalizations in Southern Europe. Policy measures should aim at reducing population exposure to anthropogenic airborne particles even in areas with large contribution from desert dust advections.
CITATION	Stafoggia M, Zauli-Sajani S, Pey J, Samoli E, Alessandrini E, Basaga□a X, Cernigliaro A, Chiusolo M, Demaria M, D□az J, Faustini A, Katsouyanni K, Kelessis AG, Linares C, Marchesi S, Medina S, Pandolfi P, P□rez N, Querol X, Randi G, Ranzi A, Tobias A, Forastiere F, MED-PARTICLES Study Group. 2016. Desert dust outbreaks in Southern Europe: contribution to daily PM10 concentrations and short-term associations with mortality and

표-364. PubMed 논문번호 26255248의 내용 요약

구분	내용
PubMed ID	26255248
TITLE	After 50 years and 200 papers, what can the Midspan cohort studies tell us about our mortality?
JOURNAL	Public health: 10.1016/j.puhe.2015.06.017
AUTHORS	Gruer L, Hart C L, Watt G C M, Gruer L, Hart C L, Watt G C M
OBJECTIVE	To distil the main findings from published papers on mortality in three cohorts involving over 27,000 adults, recruited in Scotland between 1965 and 1976 and followed up ever since.
METHOD	We read and summarized 48 peer-reviewed papers about all-cause and cause-specific mortality in these cohorts, published between 1978 and 2013.
RESULTS	Mortality rates were substantially higher among cigarette smokers in all social classes and both genders. Exposure to second-hand smoke was also damaging. Exposure to higher levels of black smoke pollution was associated with higher mortality. After smoking, diminished lung function was the risk factor most strongly related to higher mortality, even among never-smokers. On average, female mortality rates were much lower than male but the same risk factors were predictors of mortality. Mortality rates were highest among men whose paternal, own first and most recent jobs were manual. Specific causes of death were associated with different life stages. Upward and downward social mobility conferred intermediate mortality rates. Low childhood cognitive ability was strongly associated with low social class in adulthood and higher mortality before age 65 years. There was no evidence that daily stress contributed to higher mortality among people in lower social positions. Men in manual occupations with fathers in manual occupations, who smoked and drank >14 units of alcohol a week had cardiovascular disease mortality rates 4.5 times higher than non-manual men with non-manual fathers, who neither smoked nor drank >14 units. Men who were obese and drank >14 units of alcohol per day had a mortality rate due to liver disease 19 times that of normal or underweight non-drinkers. Among women who never smoked, mortality rates were highest in severely obese women in the lowest occupational classes.
CONCLUSION	These studies highlight the cumulative effect of adverse exposures throughout life, the complex interplay between social circumstances, culture and individual capabilities, and the damaging effects of smoking, air pollution, alcohol and obesity.

표-365. PubMed 논문번호 26275899의 내용 요약

구분	내용
PubMed ID	26275899
TITLE	Traffic pollution at the home address and pregnancy outcomes in Stockholm, Sweden.
JOURNAL	BMJ open: 10.1136/bmjopen-2014-007034
AUTHORS	Olsson David, Mogren Ingrid, Eneroth Kristina, Forsberg Bertil
BACKGROUND	For the past two decades, several studies have reported associations between elevated levels of ambient air pollution and adverse pregnancy outcomes, although with varying conclusions.
OBJECTIVES	To examine possible associations between the traffic pollution situation at the home address, for women who did not change address during pregnancy, and three types of pregnancy outcomes: spontaneous preterm delivery, children born small for gestational age (SGA) and pregnancy-induced hypertensive disorders.
METHODS	We used data for the Greater Stockholm Area from the Swedish Medical Birth Register to construct a cohort based on all pregnancies conceived between July 1997 and March 2006, n = 100 190. The pregnancy average nitrogen oxide, NOx, levels and annual mean daily vehicles at the home address were used as exposure variables. Mixed-model logistic regression was performed to assess any associations between exposure and outcome.
RESULTS	There was an association between elevated traffic pollution exposure during pregnancy and pregnancy-induced hypertensive disorders. A 10 □g/m(3) increase in the pregnancy average NOx level at the home address resulted in an OR of 1.17 (95% CI 1.10 to 1.26). The 2nd to 4th quartiles of NOx were all associated with an increased risk of SGA, but there was no difference in the risk estimate among the higher quartiles. There was a tendency of a higher risk of spontaneous preterm delivery in relation to higher levels of NOx. There was no evidence of an association between vehicle flow, the cruder indicator of traffic pollution, and the studied outcomes in this study.
CONCLUSIONS	In this large cohort, there was a fairly strong association between vehicle exhaust levels at the home address and pregnancy-induced hypertensive disorders, after adjustment for important risk factors.

표-366. PubMed 논문번호 26282452의 내용 요약

구분	내용
PubMed ID	26282452
TITLE	Carbon monoxide and stroke: A time series study of ambient air pollution and emergency hospitalizations.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2015.07.099
AUTHORS	Tian Linwei, Qiu Hong, Pun Vivian C, Ho Kin-Fai, Chan Chi Sing, Yu Ignatius T S
BACKGROUND	Recent experimental and clinical studies suggested that exogenous carbon monoxide (CO) at low concentrations may have beneficial neuroprotective effects under certain circumstances. However, population-based epidemiological studies of environmentally relevant CO exposure generated mixed findings. The present study aimed to examine the short-term association of ambient CO with emergency stroke hospitalizations.
METHODS	A time series study was conducted. Daily air pollution concentrations and emergency hospital admission data from January 2004 to December 2011 in Hong Kong were collected. Generalized additive Poisson models were used to estimate the associations between daily 24-hour mean concentrations of CO and emergency hospital admissions for stroke, while controlling for other traffic related co-pollutants: NO_2 and PM_2 .□. Sensitivity analyses were performed using daily 1-hour maximum concentration of CO as exposure indicator.
RESULTS	Negative associations were observed between ambient CO concentrations and emergency hospital admissions for stroke. The previous 1-3 day cumulative exposure to CO was associated with a -2.0% (95%CI, -3.3% to -0.7%) decrease in stroke admissions per interquartile range (IQR) increment in CO concentration (0.3 ppm). Similar results were obtained when using 1-hour maximum concentration of CO as exposure indicator. The negative association was robust to the co-pollutant adjustment for either NO_2 or PM_2 .□. Females and elders appeared to be more sensitive to ambient CO exposure. The negative association tended to be larger in cool season.
CONCLUSION	Short-term exposure to ambient CO was associated with decreased risk of emergency hospitalizations for stroke, suggesting some acute protective effects of CO exposure against stroke onsets.

표-367. PubMed 논문번호 26298834의 내용 요약

구분	내용
PubMed ID	26298834
TITLE	Association between long-term exposure to air pollution and mortality in France: A 25-year follow-up study.
JOURNAL	Environment international: 10.1016/j.envint.2015.08.006
AUTHORS	Bentayeb Malek, Wagner Verene, Stempfelet Morgane, Zins Marie, Goldberg Marcel, Pascal Mathilde, Larrieu Sophie, Beaudeau Pascal, Cassadou Sylvie, Eilstein Daniel, Filleul Laurent, Le Tertre Alain, Medina Sylvia, Pascal Laurence, Prouvost Helene, Qu□nel Philippe, Zeghnoun Abdelkrim, Lefranc Agnes
INTRODUCTION	Long-term exposure to air pollution (AP) has been shown to have an impact on mortality in numerous countries, but since 2005 no data exists for France.
OBJECTIVES	We analyzed the association between long-term exposure to air pollution and mortality at the individual level in a large French cohort followed from 1989 to 2013.
METHODS	The study sample consisted of 20,327 adults working at the French national electricity and gas company EDF-GDF. Annual exposure to PM10, PM10-2.5, PM2.5, NO2, O3, SO2, and benzene was assessed for the place of residence of participants using a chemistry-transport model and taking residential history into account. Hazard ratios were estimated using a Cox proportional-hazards regression model, adjusted for selected individual and contextual risk factors. Hazard ratios were computed for an interquartile range (IQR) increase in air pollutant concentrations.
RESULTS	The cohort recorded 1967 non-accidental deaths. Long-term exposures to b aseline PM2.5, PM10-25, NO2 and benzene were associated with an increase in non-accidental mortality (Hazard Ratio, HR = 1.09; 95% CI: 0.99, 1.20 per 5.9 μg/m3, PM10-25; HR=1.09; 95% CI: 1.04, 1.15 per 2.2 μg/m3, NO2: HR=1.14; 95% CI: 0.99, 1.31 per 19.3 μg/m3 and benzene: HR=1.10; 95% CI: 1.00, 1.22 per 1.7 μg/m3).The strongest association was found for PM10: HR = 1.14; 95% CI: 1.05, 1.25 per 7.8 μg/m3. PM10, PM10-25 and SO2 were associated with non-accidental mortality when using time varying exposure. No significant associations were observed between air pollution and cardiovascular and respiratory mortality.
CONCLUSION	Long-term exposure to fine particles, nitrogen dioxide, sulfur dioxide and benzene is associated with an increased risk of non-accidental mortality in France. Our results strengthen existing evidence that outdoor air pollution is a significant environmental risk factor for mortality. Due to the limited sample size and the nature of our study (occupational), further investigations are needed in France with a larger representative population sample.

표-368. PubMed 논문번호 26305859의 내용 요약

구분	내용
PubMed ID	26305859
TITLE	Traffic-Related Air Pollution and Dementia Incidence in Northern Sweden: A Longitudinal Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1408322
AUTHORS	Oudin Anna, Forsberg Bertil, Adolfsson Annelie Nordin, Lind Nina, Modig Lars, Nordin Maria, Nordin Steven, Adolfsson Rolf, Nilsson Lars-G□ran, Oudin Anna, Forsberg Bertil, Adolfsson Annelie Nordin, Lind Nina, Modig Lars, Nordin Maria, Nordin Steven, Adolfsson Rolf, Nilsson Lars-G□ran
BACKGROUND	Exposure to ambient air pollution is suspected to cause cognitive effects, but a prospective cohort is needed to study exposure to air pollution at the home address and the incidence of dementia.
OBJECTIVES	We aimed to assess the association between long-term exposure to traffic-related air pollution and dementia incidence in a major city in northern Sweden.
METHODS	Data on dementia incidence over a 15-year period were obtained from the longitudinal Betula study. Traffic air pollution exposure was assessed using a land-use regression model with a spatial resolution of 50 m × 50 m. Annual mean nitrogen oxide levels at the residential address of the participants at baseline (the start of follow-up) were used as markers for long-term exposure to air pollution.
RESULTS	Out of 1,806 participants at baseline, 191 were diagnosed with Alzheimer's disease during follow-up, and 111 were diagnosed with vascular dementia. Participants in the group with the highest exposure were more likely than those in the group with the lowest exposure to be diagnosed with dementia (Alzheimer's disease or vascular dementia), with a hazard ratio (HR) of 1.43 (95% CI: 0.998, 2.05 for the highest vs. the lowest quartile). The estimates were similar for Alzheimer's disease (HR 1.38) and vascular dementia (HR 1.47). The HR for dementia associated with the third quartile versus the lowest quartile was 1.48 (95% CI: 1.03, 2.11). A subanalysis that excluded a younger sample that had been retested after only 5 years of follow-up suggested stronger associations with exposure than were present in the full cohort (HR = 1.71; 95% CI: 1.08, 2.73 for the highest vs. the lowest quartile).
CONCLUSIONS	If the associations we observed are causal, then air pollution from traffic might be an important risk factor for vascular dementia and Alzheimer's disease.

표-369. PubMed 논문번호 26324253의 내용 요약

구분	내용
PubMed ID	26324253
TITLE	Pedigree-based Analysis of Inherited and Noninherited Risk Factors of Congenital Heart Defects.
JOURNAL	Early human development: 10.1016/j.earlhumdev.2015.08.001
AUTHORS	Yuan Yuan, Chen Weicheng, Ma Xiaojing, Wang Huijun, Yan Weili, Huang Guoying
BACKGROUND	Although congenital heart defect (CHD) pedigrees are rare, they are generally taken as evidence of the existence of a genetic etiologic mechanism or environmental factors common to family members, or a combination of both. Therefore, the analysis of CHD pedigrees is important for bridging the gap in our knowledge of its etiology.
AIMS	To assess the prevalence of CHD and evaluate the nongenetic factors in the CHD patients and healthy controls in the pedigrees.
STUDY DESIGN	Observational retrospective study.
SUBJECTS	Twenty-three CHD pedigrees were involved in the prevalence statistics; thirty-nine CHD cases and fifty-two healthy controls in the CHD pedigrees were included in the family-based noninherited factors analysis.
OUTCOME MEASURES	The three-degree relatives and overall CHD prevalence were calculated. Thirty-four noninherited risk factors were compared between the CHD and control groups, first by univariate analysis and later by multivariable logistic stepwise regression analysis.
RESULTS	The CHD prevalence of the probands' relatives in all pedigrees was 8.0%, and it was 10.9%, 2.9% and 11.9% in first-, second- and third-degree relatives, respectively. The three risk factors, including maternal febrile illnesses (OR=14.2, 95%CI: [1.5 - 133.7]), influenza (OR=6.9 [2.0 - 23.6]) and air pollution (OR=13.5 [2.6 - 70.5]), were strongly associated with a higher risk of CHD in our sample.
CONCLUSIONS	For the cluster and high prevalence of CHD in the collected pedigrees, our study confirms that genetic factors play a major role in the pathogenesis of CHD, while environmental factors, such as maternal febrile illnesses, influenza and air pollution, may also increase the burden of risk for CHD pathogenesis.

표-370. PubMed 논문번호 26340844의 내용 요약

구분	내용
PubMed ID	26340844
TITLE	Secondhand smoke and atrial fibrillation: Data from the Health eHeart Study.
JOURNAL	Heart rhythm: 10.1016/j.hrthm.2015.08.004
AUTHORS	Dixit Shalini, Pletcher Mark J, Vittinghoff Eric, Imburgia Kourtney, Maguire Carol, Whitman Isaac R, Glantz Stanton A, Olgin Jeffrey E, Marcus Gregory M, Dixit Shalini, Pletcher Mark J, Vittinghoff Eric, Imburgia Kourtney, Maguire Carol, Whitman Isaac R, Glantz Stanton A, Olgin Jeffrey E, Marcus Gregory M
BACKGROUND	Cigarette smoking is a risk factor for atrial fibrillation (AF), but whether secondhand smoke (SHS) impacts the risk of AF remains unknown.
OBJECTIVE	To determine if SHS exposure is associated with an increased risk of AF.
METHODS	We performed a cross-sectional analysis of data from participants enrolled in the Health eHeart Study, an internet-based, longitudinal cardiovascular cohort study, who completed baseline SHS exposure and medical conditions questionnaires. SHS was assessed through a validated 22-question survey, and prevalent AF was assessed by self-report, with validation of a subset (n = 42) by review of electronic medical records.
RESULTS	Of 4976 participants, 593 (11.9%) reported having AF. In unadjusted analyses, patients with AF were more likely to have been exposed to SHS in utero, as a child, as an adult, at home, and at work. After multivariable adjustment for potential confounders, having had a smoking parent during gestational development (OR 1.37, 95% CI 1.08-1.73, P = .009) and residing with a smoker during childhood (OR 1.40, 95% CI 1.10-1.79, P = .007) were each significantly associated with AF. Both positive associations were more pronounced among patients without risk factors for AF (P values for interaction <.05).
CONCLUSIONS	SHS exposure during gestational development and during childhood was associated with having AF later in life. This association was even stronger in the absence of established risk factors for AF. Our findings indicate that SHS in early life may be an important, potentially modifiable risk factor for the development of AF.

표-371. PubMed 논문번호 26364544의 내용 요약

구분	내용
PubMed ID	26364544
TITLE	Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(15)00128-2
AUTHORS	Forouzanfar Mohammad H, Alexander Lily, Anderson H Ross, Bachman Victoria F, Biryukov Stan, Brauer Michael, Burnett Richard, Casey Daniel, Coates Matthew M, Cohen Aaron, Delwiche Kristen, Estep Kara, Frostad Joseph J, Astha K C, Kyu Hmwe H, Moradi-Lakeh Maziar, Ng Marie, Slepak Erica Leigh, Thomas Bernadette A, Wagner Joseph, Aasvang Gunn Marit, Abbafati Cristiana, Abbasoglu Ozgoren Ayse, Abd-Allah Foad, Abera Semaw F, Aboyans Victor, Abraham Biju, Abraham Jerry Puthenpurakal, Abubakar Ibrahim, Abu-Rmeileh Niveen M E, Aburto Tania C, Achoki Tom, Adelekan Ademola, Adofo Koranteng, Adou Ars□ne K, Adsuar Jos□ C, Afshin Ashkan, Agardh Emilie E, Al Khabouri Mazin J, Al Lami Faris H, Alam Sayed Saidul, Alasfoor Deena, Albittar Mohammed I, Alegretti Miguel A, Aleman Alicia V, Alemu Zewdie A, Alfonso-Cristancho Rafael, Alhabib Samia, Ali Raghib, Ali Mohammed K, Alla Fran□ois, Allebeck Peter, Allen Peter J, Alsharif Ubai, Alvarez Elena, Alvis-Guzman Nelson, Amankwaa Adansi A, Amare Azmeraw T, Ameh Emmanuel A, Ameli Omid, Amini Heresh, Ammar Walid, Anderson Benjamin O, Antonio Carl Abelardo T, Anwari Palwasha, Argeseanu Cunningham Solveig, Arnl□v Johan, Arsenijevic Valentina S Arsic, Artaman Al, Asghar Rana J, Assadi Reza, Atkins Lydia S, Atkinson Charles, Avila Marco A, Awuah Baffour, Badawi Alaa, Bahit Maria C, Bakfalouni Talal, Balakrishnan Kalpana, Balalla Shivanthi, Balu Ravi Kumar, Banerjee Amitava, Barber Ryan M, Barker-Collo Suzanne L, Barquera Simon, Barregard Lars, Barrero Lope H, Barrientos-Gutierrez Tonatiuh, Basto-Abreu Ana C, Basu Arindam, Basu Sanjay, Basulaiman Mohammed O, Batis Ruvalcaba Carolina, Beardsley Justin, Bedi Neeraj, Bekele Tolesa, Bell Michelle L, Benjet Corina, Bennett Derrick A, Benzian Habib, Bernab□ Eduardo, Beyene Tariku J, Bhala Neeraj, Bhalla Ashish, Bhutta Zulfiqar A, Bikbov Boris, Bin Abdulhak Aref A, Blore Jed D, Blyth Fiona M, Bohensky Megan A, Bora Ba□ara Berrak, Borges Guilherme, Bornstein Natan M, Bose Dipan, Boufous Soufiane, Bourne Rupert R, Brainin Michael, Brazinova Alexandra, Breitborde Nicholas J, Brenner Hermann, Briggs Adam D M, Broday David M, Brooks Peter M, Bruce Nigel G, Brugha Traolach S, Brunekreef Bert, Buchbinder Rachelle, Bui Linh N, Bukhman Gene, Bulloch Andrew G, Burch Michael, Burney Peter G J, Campos-Nonato Ismael R, Campuzano Julio C, Cantoral Alejandra J, Caravanos Jack, C□rdenas Rosario, Cardis Elisabeth, Carpenter David O, Caso Valeria, Casta□eda-Orjuela Carlos A, Castro Ruben E, Catal□-L□pez Ferr□n, Cavalleri Fiorella, □avlin Alanur, Chadha Vineet K, Chang Jung-Chen, Charlson Fiona J, Chen Honglei, Chen Wanqing, Chen Zhengming, Chiang Peggy P, Chimed-Ochir Odgerel, Chowdhury Rajiv, Christophi Costas A, Chuang Ting-Wu, Chugh Sumeet S, Cirillo Massimo, Cla ß en Thomas K D, Colistro Valentina, Colomar Mercedes, Colquhoun Samantha M, Contreras Alejandra G, Cooper Cyrus, Cooperrider Kimberly, Cooper Leslie T, Coresh Josef, Courville Karen J, Criqui Michael H, Cuevas-Nasu Lucia, Damsere-Derry James, Danawi Hadi, Dandona Lalit, Dandona Rakhi, Dargan Paul I, Davis Adrian, Davitoiu Dragos V, Dayama Anand, de Castro E Filipa, De la Cruz-G□ngora Vanessa, De Leo Diego, de Lima Gra□a, Degenhardt Louisa, del Pozo-Cruz Borja, Dellavalle Robert P, Deribe Kebede, Derrett Sarah, Des Jarlais Don C, Dessalegn Muluken, deVeber Gabrielle A, Devries Karen M, Dharmaratne

표-372. PubMed 논문번호 26370657의 내용 요약

구분	내용
PubMed ID	26370657
TITLE	Ambient Particulate Matter Air Pollution Exposure and Mortality in the NIH-AARP Diet and Health Cohort.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1509676
AUTHORS	Thurston George D, Ahn Jiyoung, Cromar Kevin R, Shao Yongzhao, Reynolds Harmony R, Jerrett Michael, Lim Chris C, Shanley Ryan, Park Yikyung, Hayes Richard B, Thurston George D, Ahn Jiyoung, Cromar Kevin R, Shao Yongzhao, Reynolds Harmony R, Jerrett Michael, Lim Chris C, Shanley Ryan, Park Yikyung, Hayes Richard B
BACKGROUND	Outdoor fine particulate matter (≤ 2.5 μm; PM2.5) has been identified as a global health threat, but the number of large U.S. prospective cohort studies with individual participant data remains limited, especially at lower recent exposures.
OBJECTIVES	We aimed to test the relationship between long-term exposure PM2.5 and death risk from all nonaccidental causes, cardiovascular (CVD), and respiratory diseases in 517,041 men and women enrolled in the National Institutes of Health-AARP cohort.
METHODS	Individual participant data were linked with residence PM2.5 exposure estimates across the continental United States for a 2000-2009 follow-up period when matching census tract-level PM2.5 exposure data were available. Participants enrolled ranged from 50 to 71 years of age, residing in six U.S. states and two cities. Cox proportional hazard models yielded hazard ratio (HR) estimates per 10 μg/m3 of PM2.5 exposure.
RESULTS	PM2.5 exposure was significantly associated with total mortality (HR = 1.03; 95% CI: 1.00, 1.05) and CVD mortality (HR = 1.10; 95% CI: 1.05, 1.15), but the association with respiratory mortality was not statistically significant (HR = 1.05; 95% CI: 0.98, 1.13). A significant association was found with respiratory mortality only among never smokers (HR = 1.27; 95% CI: 1.03, 1.56). Associations with 10- μg/m3 PM2.5 exposures in yearly participant residential annual mean, or in metropolitan area-wide mean, were consistent with baseline exposure model results. Associations with PM2.5 were similar when adjusted for ozone exposures. Analyses of California residents alone also yielded statistically significant PM2.5 mortality HRs for total and CVD mortality.
CONCLUSIONS	Long-term exposure to PM2.5 air pollution was associated with an increased risk of total and CVD mortality, providing an independent test of the PM2.5-mortality relationship in a new large U.S. prospective cohort experiencing lower post-2000 PM2.5 exposure levels.
CITATION	Thurston GD, Ahn J, Cromar KR, Shao Y, Reynolds HR, Jerrett M, Lim CC, Shanley R, Park Y, Hayes RB. 2016. Ambient particulate matter air pollution exposure and mortality in the NIH-AARP Diet and Health cohort. Environ Health Perspect 124:484-490; http://dx.doi.org/10.1289/ehp.1509676.

표-373. PubMed 논문번호 26392179의 내용 요약

구분	내용
PubMed ID	26392179
TITLE	Chronic exposure to particulate matter and risk of cardiovascular mortality: cohort study from Taiwan.
JOURNAL	BMC public health: 10.1186/s12889-015-2272-6
AUTHORS	Tseng Eva, Ho Wen-Chao, Lin Meng-Hung, Cheng Tsun-Jen, Chen Pau-Chung, Lin Hsien-Ho
BACKGROUND	Evidence on the association between long-term exposure to air pollution and cardiovascular mortality is limited in Asian populations.
METHODS	We conducted a cohort study on the association between fine particulate matter (PM2.5) and cardiovascular mortality using 43,227 individuals in a civil servants health service in Taiwan. Each participant was assigned an exposure level of particulate matter based on their district of residence using air pollution data collected by the Taiwan Environmental Protection Agency and with modeling using geographic information systems. The participants were followed up from 1989 to 2008 and the vital status was ascertained from death records. Cox regression models were used to adjust for confounding factors.
RESULTS	The district-level average of PM2.5 ranged from 22.8 to 32.9 μg/m(3) in the study area. After a median follow-up of 18 years, 1992 deaths from all causes including 230 cardiovascular deaths occurred. After adjustment for potential confounders, PM2.5 levels were not significantly associated with mortality from cardiovascular disease [Hazard Ratio (HR) 0.80; 95 % Confidence Interval (CI), 0.43 to 1.50 per 10 μg/m(3) increase in PM2.5] or all causes (HR 0.92; 95 % CI, 0.72 to 1.17 per 10 μg/m(3) increase in PM2.5). The results were similar when the analysis was restricted to the urban areas and when the PM2.5 measurement was changed from the period average (2000-2008) to annual average.
DISCUSSION	Our findings are different from those in prior cohort studies conducted in Asia where ambient air pollutionwas associated with an increased risk of cardiovascular mortality. The high background level of air pollutionin our study area and the small number of event cases limited the power of this study.
CONCLUSIONS	In this population-based cohort study in Taiwan, we found no evidence of increased risk for all-cause or cardiovascular mortality with long-term exposure to PM2.5.

표-374. PubMed 논문번호 26421716의 내용 요약

구분	내용
PubMed ID	26421716
TITLE	Microbiological Evaluation of Household Drinking Water Treatment in Rural China Shows Benefits of Electric Kettles: A Cross-Sectional Study.
JOURNAL	PloS one: 10.1371/journal.pone.0138451
AUTHORS	Cohen Alasdair, Tao Yong, Luo Qing, Zhong Gemei, Romm Jeff, Colford John M, Ray Isha
BACKGROUND	In rural China ~607 million people drink boiled water, yet little is known about prevailing household water treatment (HWT) methods or their effectiveness. Boiling, the most common HWT method globally, is microbiologically effective, but household air pollution (HAP) from burning solid fuels causes cardiovascular and respiratory disease, and black carbon emissions exacerbate climate change. Boiled water is also easily re-contaminated. Our study was designed to identify the HWT methods used in rural China and to evaluate their effectiveness.
METHODS	We used a geographically stratified cross-sectional design in rural Guangxi Province to collect survey data from 450 households in the summer of 2013. Household drinking water samples were collected and assayed for Thermotolerant Coliforms (TTC), and physicochemical analyses were conducted for village drinking water sources. In the winter of 2013-2104, we surveyed 120 additional households and used remote sensors to corroborate self-reported boiling data.
FINDINGS	Our HWT prevalence estimates were: 27.1% boiling with electric kettles, 20.3% boiling with pots, 34.4% purchasing bottled water, and 18.2% drinking untreated water (for these analyses we treated bottled water as a HWT method). Households using electric kettles had the lowest concentrations of TTC (73% lower than households drinking untreated water). Multilevel mixed-effects regression analyses showed that electric kettles were associated with the largest Log10TTC reduction (-0.60, $p<0.001$), followed by bottled water (-0.45, $p<0.001$) and pots (-0.44, $p<0.01$). Compared to households drinking untreated water, electric kettle users also had the lowest risk of having TTC detected in their drinking water (risk ratio, RR = 0.49, 0.34-0.70, $p<0.001$), followed by bottled water users (RR = 0.70, 0.53-0.93, $p<0.05$) and households boiling with pots (RR = 0.74, 0.54-1.02, p = 0.06).
CONCLUSION	As far as we are aware, this is the first HWT-focused study in China, and the first to quantify the comparative advantage of boiling with electric kettles over pots. Our results suggest that electric kettles could be used to rapidly expand safe drinking water access and reduce HAP exposure in rural China.

표-375. PubMed 논문번호 26425807의 내용 요약

구분	내용
PubMed ID	26425807
TITLE	Exposure to long-term air pollution and road traffic noise in relation to cholesterol: A cross-sectional study.
JOURNAL	Environment international: 10.1016/j.envint.2015.09.021
AUTHORS	S ø rensen Mette, Hjortebjerg Dorrit, Eriksen Kirsten T, Ketzel Matthias, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
BACKGROUND	Exposure to traffic noise and air pollution have both been associated with cardiovascular disease, though the mechanisms behind are not yet clear.
OBJECTIVES	We aimed to investigate whether the two exposures were associated with levels of cholesterol in a cross-sectional design.
METHODS	In 1993 – 1997, 39,863 participants aged 50 – 64 year and living in the Greater Copenhagen area were enrolled in a population-based cohort study. For each participant, non-fasting total cholesterol was determined in whole blood samples on the day of enrolment. Residential addresses 5-years preceding enrolment were identified in a national register and road traffic noise (Lden) were modeled for all addresses. For air pollution, nitrogen dioxide (NO2) was modeled at all addresses using a dispersion model and PM2.5 was modeled at all enrolment addresses using a land-use regression model. Analyses were done using linear regression with adjustment for potential confounders as well as mutual adjustment for the three exposures.
RESULTS	Baseline residential exposure to the interquartile range of road traffic noise,NO2 and PM2.5 was associated with a 0.58 mg/dl (95% confidence interval: □0.09; 1.25), a 0.68 mg/dl (0.22; 1.16) and a 0.78 mg/dl (0.22; 1.34) higher level of total cholesterol in single pollutant models, respectively. In two pollutant models with adjustment for noise in air pollution models and vice versa, the association between air pollution and cholesterol remained for both air pollution variables (NO2: 0.72 (0.11; 1.34); PM2.5: 0.70 (0.12; 1.28) mg/dl), whereas there was no association for noise (□0.08mg/dl). In three-pollutant models (NO2, PM2.5 and road traffic noise), estimates for NO2 and PM2.5 were slightly diminished (NO2: 0.58 (□0.05; 1.22); PM2.5: 0.57 (□0.02; 1.17) mg/dl).
CONCLUSIONS	Air pollution and possibly also road traffic noise may be associated with slightly higher levels of cholesterol, though associations for the two exposures were difficult to separate.

표-376. PubMed 논문번호 26454658의 내용 요약

구분	내용
PubMed ID	26454658
TITLE	Long-term traffic air and noise pollution in relation to mortality and hospital readmission among myocardial infarction survivors.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2015.09.003
AUTHORS	Tonne Cathryn, Halonen Jaana I, Beevers Sean D, Dajnak David, Gulliver John, Kelly Frank J, Wilkinson Paul, Anderson H Ross, Tonne Cathryn, Halonen Jaana I, Beevers Sean D, Dajnak David, Gulliver John, Kelly Frank J, Wilkinson Paul, Anderson H Ross
BACKGROUND	There is relatively little evidence of health effects of long-term exposure to traffic-related pollution in susceptible populations. We investigated whether long-term exposure to traffic air and noise pollution was associated with all-cause mortality or hospital readmission for myocardial infarction (MI) among survivors of hospital admission for MI.
METHODS	Patients from the Myocardial Ischaemia National Audit Project database resident in Greater London (n = 1 8,138) were followed for death or readmission for MI. High spatially-resolved annual average air pollution (11 metrics of primary traffic, regional or urban background) derived from a dispersion model (resolution 20 m × 20 m) and road traffic noise for the years 2003-2010 were used to assign exposure at residence. Hazard ratios (HR, 95% confidence interval (CI)) were estimated using Cox proportional hazards models.
RESULTS	Most air pollutants were positively associated with all-cause mortality alone and in combination with hospital readmission. The largest associations with mortality per interquartile range (IQR) increase of pollutant were observed for non-exhaust particulate matter (PM(10)) (HR = 1.05 (95% CI 1.00, 1.10), IQR = 1.1 μg/m(3)); oxidant gases (HR = 1.05 (95% CI 1.00, 1.09), IQR = 3.2 μg/m(3)); and the coarse fraction of PM (HR = 1.05 (95% CI 1.00, 1.10), IQR = 0.9 μg/m(3)). Adjustment for traffic noise only slightly attenuated these associations. The association for a 5 dB increase in road-traffic noise with mortality was HR = 1.02 (95% CI 0.99, 1.06) independent of air pollution.
CONCLUSIONS	These data support a relationship of primary traffic and regional/urban background air pollution with poor prognosis among MI survivors. Although imprecise, traffic noise appeared to have a modest association with prognosis independent of air pollution.

표-377. PubMed 논문번호 26463695의 내용 요약

구분	내용
PubMed ID	26463695
TITLE	Long-Term Exposure to Particulate Matter Air Pollution Is a Risk Factor for Stroke: Meta-Analytical Evidence.
JOURNAL	Stroke: 10.1161/STROKEAHA.115.009913
AUTHORS	Scheers Hans, Jacobs Lotte, Casas Lidia, Nemery Benoit, Nawrot Tim S
BACKGROUND AND PURPOSE	Epidemiological studies suggest an association between stroke incidence and stroke mortality and long-term exposure to particulate matter (PM) air pollution. However, the magnitude of the association is still unclear.
METHODS	We searched the Pubmed citation database for epidemiological studies and reviews on stroke and PM exposure. Then, we carried out a meta-analysis to quantify the pooled association between stroke incidence and mortality and long-term exposure to PM. Meta-analyses were performed for stroke events and stroke mortality and for PM10 and PM2.5 separately and jointly.
RESULTS	We identified 20 studies, including a total of >10 million people, on long-term PM exposure and stroke event or stroke mortality. For exposure to PM10 (including estimated exposure to PM10 from studies using PM2.5), the pooled hazard ratio for each 10-μg/m3 increment in PM10 was 1.061 (95% confidence interval, 1.018-1.105) and 1.080 (0.992-1.177) for overall stroke events and stroke mortality, respectively. A stratified analysis by continent revealed that the association between stroke and long-term PM10 exposure was positive in North America (1.062 [1.015-1.110]) and Europe (1.057 [0.973-1.148]), but studies in Asia (1.010 [0.885-1.153]) showed a high degree of heterogeneity. Considering exposure to PM2.5 (Europe and North America combined), the hazard ratios for a 5-μg/m3 increment were 1.064 (1.021-1.109) and 1.125 (1.007-1.256) for stroke events and mortality, respectively.
CONCLUSIONS	The scientific evidence of the past decade identifies long-term exposure to PM, and PM2.5 in particular, as a risk factor for stroke. However, we found some currently unexplained geographical variability in this association.

표-378. PubMed 논문번호 26501300의 내용 요약

구분	내용
PubMed ID	26501300
TITLE	Association of Exposure to particular matter and Carotid Intima-Media Thickness: A Systematic Review and Meta-Analysis.
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph121012924
AUTHORS	Liu Xiaole, Lian Hui, Ruan Yanping, Liang Ruijuan, Zhao Xiaoyi, Routledge Michael, Fan Zhongjie
BACKGROUND	Long time exposure to particular matter has been linked to myocardial infarction, stroke and blood pressure, but its association with atherosclerosis is not clear. This meta-analysis was aimed at assessing whether PM_{2} .□ and PM_{1} □ have an effect on subclinical atherosclerosis measured by carotid intima-media thickness (CIMT).
METHODS	Pubmed, Ovid Medline, Embase and NICK between 1948 and 31 March 2015 were searched by combining the keywords about exposure to the outcome related words. The random-effects model was applied in computing the change of CIMT and their corresponding 95% confidence interval (95% CI). The effect of potential confounding factors was assessed by stratified analysis and the impact of traffic proximity was also estimated.
RESULTS	Among 56 identified studies, 11 articles satisfied the inclusion criteria. In overall analysis increments of 10 $\mu g/m^{3}$ in PM_{2} .□ and PM_{1} □ were associated with an increase of CIMT (16.79 μm; 95% CI, 4.95-28.63 μm and 4.13 μm; 95% CI, -5.79-14.04 μm, respectively). RESULTS shown in subgroup analysis had reference value for comparing with those of the overall analysis. The impact of traffic proximity on CIMT was uncertain.
CONCLUSIONS	Exposure to PM_{2} .□ had a significant association with CIMT and for women the effect may be more obvious.

표-379. PubMed 논문번호 26503387의 내용 요약

구분	내용
PubMed ID	26503387
TITLE	Long-term ambient air pollution exposure and self-reported morbidity in the Australian Longitudinal Study on Women's Health: a cross-sectional study.
JOURNAL	BMJ open: 10.1136/bmjopen-2015-008714
AUTHORS	Lazarevic Nina, Dobson Annette J, Barnett Adrian G, Knibbs Luke D
OBJECTIVE	We sought to assess the effect of long-term exposure to ambient air pollution on the prevalence of self-reported health outcomes in Australian women.
DESIGN	Cross-sectional study.
SETTING AND PARTICIPANTS	The geocoded residential addresses of 26,991 women across 3 age cohorts in the Australian Longitudinal Study on Women's Health between 2006 and 2011 were linked to nitrogen dioxide (NO2) exposure estimates from a land-use regression model. Annual average NO2 concentrations and residential proximity to roads were used as proxies of exposure to ambient air pollution.
OUTCOME MEASURES	Self-reported disease presence for diabetes mellitus, heart disease, hypertension, stroke, asthma, chronic obstructive pulmonary disease and self-reported symptoms of allergies, breathing difficulties, chest pain and palpitations.
METHODS	Disease prevalence was modelled by population-averaged Poisson regression models estimated by generalised estimating equations. Associations between symptoms and ambient air pollution were modelled by multilevel mixed logistic regression. Spatial clustering was accounted for at the postcode level.
RESULTS	No associations were observed between any of the outcome and exposure variables considered at the 1% significance level after adjusting for known risk factors and confounders.
CONCLUSIONS	Long-term exposure to ambient air pollution was not associated with self-reported disease prevalence in Australian women. The observed results may have been due to exposure and outcome misclassification, lack of power to detect weak associations or an actual absence of associations with self-reported outcomes at the relatively low annual average air pollution exposure levels across Australia.

표-380. PubMed 논문번호 26539762의 내용 요약

구분	내용
PubMed ID	26539762
TITLE	Environmental Tobacco Smoke and Atrial Fibrillation: The REasons for Geographic And Racial Differences in Stroke (REGARDS) Study.
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0000000000000565
AUTHORS	O'Neal Wesley T, Qureshi Waqas T, Judd Suzanne E, McClure Leslie A, Cushman Mary, Howard Virginia J, Howard George, Soliman Elsayed Z
OBJECTIVE	This study examines the association between environmental tobacco smoke (ETS) exposure and atrial fibrillation (AF).
METHODS	We examined the cross-sectional association between ETS exposure and AF in 12,021 participants (mean age: 65 ± 9.9 years; 60% women; 40% blacks) from the REasons for Geographic And Racial Differences in Stroke study who self-identified as never smokers between 2003 and 2007.
RESULTS	A total of 2503 (21%) participants reported ETS exposure. In a multivariate logistic regression model adjusted for sociodemographics and potential confounders, ETS exposure was significantly associated with AF (odds ratio=1.27, 95% confidence interval=1.08, 1.50).
CONCLUSIONS	Our findings suggest that the harmful effects of ETS exposure extend to sustained arrhythmias such as AF.

표-381. PubMed 논문번호 26546332의 내용 요약

구분	내용
PubMed ID	26546332
TITLE	Exposure to sub-chronic and long-term particulate air pollution and heart rate variability in an elderly cohort: the Normative Aging Study.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-015-0074-z
AUTHORS	Mordukhovich Irina, Coull Brent, Kloog Itai, Koutrakis Petros, Vokonas Pantel, Schwartz Joel
BACKGROUND	Short-term particulate air pollution exposure is associated with reduced heart rate variability (HRV), a risk factor for cardiovascular morbidity and mortality, in many studies. Associations with sub-chronic or long-term exposures, however, have been sparsely investigated. We evaluated the effect of fine particulate matter (PM2.5) and black carbon (BC) exposures on HRV in an elderly cohort: the Normative Aging Study.
METHODS	We measured power in high frequency (HF) and low frequency (LF), standard deviation of normal-to-normal intervals (SDNN), and the LF:HF ratio among participants from the Greater Boston area. Residential BC exposures for 540 men (1161 study visits, 2000-2011) were estimated using a spatio-temporal land use regression model, and residential PM2.5 exposures for 475 men (992 visits, 2003-2011) were modeled using a hybrid satellite based and land-use model. We evaluated associations between moving averages of sub-chronic (3-84 day) and long-term (1 year) pollutant exposure estimates and HRV parameters using linear mixed models.
RESULTS	One-standard deviation increases in sub-chronic, but not long-term, BC were associated with reduced HF, LF, and SDNN and an increased LF:HF ratio (e.g., 28 day BC: -2.3% HF [95% CI:-4.6, -0.02]). Sub-chronic and long-term PM2.5 showed evidence of relations to an increased LF and LF:HF ratio (e.g., 1 year PM: 21.0% LF:HF [8.6, 34.8]), but not to HF or SDNN, though the effect estimates were very imprecise and mostly spanned the null.
CONCLUSIONS	We observed some evidence of a relation between longer-term BC and PM2.5 exposures and changes in HRV in an elderly cohort. While previous studies focused on short-term air pollution exposures, our results suggest that longer-term exposures may influence cardiac autonomic function.

표-382. PubMed 논문번호 26548715의 내용 요약

구분	내용
PubMed ID	26548715
TITLE	The effect of smoking on carotid intima-media thickness progression rate and rate of lumen diameter reduction.
JOURNAL	European journal of internal medicine: 10.1016/j.ejim.2015.10.018
AUTHORS	Hansen Kristina, □stling Gerd, Persson Margaretha, Nilsson Peter M, Melander Olle, Engstr□m Gunnar, Hedblad Bo, Rosvall Maria, Hansen Kristina, □stling Gerd, Persson Margaretha, Nilsson Peter M, Melander Olle, Engstr□m Gunnar, Hedblad Bo, Rosvall Maria
OBJECTIVE	The purpose of the study was to investigate the long-term associations between smoking habits, environmental tobacco smoke exposure (ETS), carotid intima-media thickness (IMT) progression rate, and rate of lumen diameter reduction in the carotid artery during a 16-year follow-up. Another objective was to investigate if an effect of smoking on progression rate could be explained by increased low grade inflammation.
METHODS	The study population included 2992 middle-aged men and women in the 1991-1994 (baseline) and the 2007-2012 (re-examination) investigation of the Malm□ Diet and Cancer Study cardiovascular cohort. Associations between smoking, progression of carotid IMT and lumen diameter reduction due to plaque protrusion were assessed by linear regression.
RESULTS	IMT progression rates and rate of lumen diameter reduction increased from never smokers with no ETS through former, moderate and heavy smokers, even after adjustment for traditional risk factors (e.g., differences in yearly progression rates (mm/year) of maximal IMT in the carotid bifurcation compared to never smokers; former smokers 0.0074 (95% CI: 0.0018-0.0129), moderate smokers 0.0106 (95% CI: 0.0038-0.0175), and heavy smokers 0.0146 (95% CI: 0.0061-0.0230)). Former smokers showed distinct lowering of progression rates after more than five years since smoking cessation. Smoking and former smoking was associated with increased low grade inflammation, however, the effect of smoking on atherosclerotic progression rate remained fairly unchanged after such adjustment.
CONCLUSION	The effect of smoking and former smoking on carotid IMT progression rates and change in lumen reduction due to plaque protrusion could not be explained by differences in traditional risk factors or low grade inflammation.

표-383. PubMed 논문번호 26562062의 내용 요약

구분	내용
PubMed ID	26562062
TITLE	The Impact of Multipollutant Clusters on the Association Between Fine Particulate Air Pollution and Microvascular Function.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000415
AUTHORS	Ljungman Petter L, Wilker Elissa H, Rice Mary B, Austin Elena, Schwartz Joel, Gold Diane R, Koutrakis Petros, Benjamin Emelia J, Vita Joseph A, Mitchell Gary F, Vasan Ramachandran S, Hamburg Naomi M, Mittleman Murray A
BACKGROUND	Prior studies including the Framingham Heart Study have suggested associations between single components of air pollution and vascular function; however, underlying mixtures of air pollution may have distinct associations with vascular function.
METHODS	We used a k-means approach to construct five distinct pollution mixtures from elemental analyses of particle filters, air pollution monitoring data, and meteorology. Exposure was modeled as an interaction between fine particle mass (PM2.5), and concurrent pollution cluster. Outcome variables were two measures of microvascular function in the fingertip in the Framingham Offspring and Third Generation cohorts from 2003 to 2008.
RESULTS	In 1,720 participants, associations between PM2.5 and baseline pulse amplitude tonometry differed by air pollution cluster (interaction P value 0.009). Higher PM2.5 on days with low mass concentrations but high proportion of ultrafine particles from traffic was associated with 18% (95% confidence interval: 4.6%, 33%) higher baseline pulse amplitude per 5 μg/m and days with high contributions of oil and wood combustion with 16% (95% confidence interval: 0.2%, 34%) higher baseline pulse amplitude. We observed no variation in associations of PM2.5 with hyperemic response to ischemia observed across air pollution clusters.
CONCLUSIONS	PM2.5 exposure from air pollution mixtures with large contributions of local ultrafine particles from traffic, heating oil, and wood combustion was associated with higher baseline pulse amplitude but not hyperemic response. Our findings suggest little association between acute exposure to air pollution clusters reflective of select sources and hyperemic response to ischemia, but possible associations with excessive small artery pulsatility with potentially deleterious microvascular consequences.

표-384. PubMed 논문번호 26595125의 내용 요약

구분	내용
PubMed ID	26595125
TITLE	PM2.5 and Diabetes and Hypertension Incidence in the Black Women's Health Study.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000418
AUTHORS	Coogan Patricia F, White Laura F, Yu Jeffrey, Burnett Richard T, Seto Edmund, Brook Robert D, Palmer Julie R, Rosenberg Lynn, Jerrett Michael
BACKGROUND	Clinical studies have shown that exposure to fine particulate matter (PM2.5) can increase insulin resistance and blood pressure. The epidemiologic evidence for an association of PM2.5 exposure with the incidence of type 2 diabetes or hypertension is inconsistent. Even a modest association would have great public health importance given the ubiquity of exposure and high prevalence of the conditions.
METHODS	We used Cox proportional hazards models to calculate hazard ratios (HRs) and 95% confidence intervals (CIs) for incident type 2 diabetes and hypertension associated with exposure to PM2.5 in a large cohort of African American women living in 56 metropolitan areas across the US, using data from the Black Women's Health Study. Pollutant levels were estimated at all residential locations over follow-up with a hybrid model incorporating land use regression and Bayesian Maximum Entropy techniques.
RESULTS	During 1995 to 2011, 4,387 cases of diabetes and 9,570 cases of hypertension occurred. In models controlling for age, questionnaire cycle, and metro area, there were positive associations with diabetes (HR = 1.13, 95% CI = 1.04, 1.24) and hypertension (HR = 1.06, 95% CI = 1.00, 1.12) per interquartile range of PM2.5 (2.9 μg/m). Multivariable HRs, however, were 0.99 (95% CI = 0.90, 1.09) for diabetes and 0.99 (95% CI = 0.93, 1.06) for hypertension.
CONCLUSIONS	Our results provide little support for an association of PM2.5 with diabetes or hypertension incidence.

표-385. PubMed 논문번호 26599707의 내용 요약

구분	내용
PubMed ID	26599707
TITLE	Pretreatment with Antioxidants Augments the Acute Arterial Vasoconstrictio Caused by Diesel Exhaust Inhalation.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201506-1247OC
AUTHORS	Sack Cora S, Jansen Karen L, Cosselman Kristen E, Trenga Carol A, Stapleto Pat L, Allen Jason, Peretz Alon, Olives Casey, Kaufman Joel D
RATIONALE	Diesel exhaust inhalation, which is the model traffic-related air pollutant exposure, is associated with vascular dysfunction.
OBJECTIVES	To determine whether healthy subjects exposed to diesel exhaust exhibit acut vasoconstriction and whether this effect could be modified by the use of antioxidants or by common variants in the angiotensin II type 1 receptor (AGTR1) and other candidate genes.
METHODS	In a genotype-stratified, double-blind, four-way crossover study, 21 healthy adult subjects were exposed at rest in a randomized, balanced order to diesel exhaust (200 μg/m(3) particulate matter with an aerodynamic diameter $\leq$ 2.5 μm [PM2.5]) and filtered air, and to pretreatment with antioxidants (N-acetylcysteine and ascorbate) and placebo. Before and after each exposure, brachial artery diameter (BAd) was assessed using ultrasound. Changes in BAd were compared across pretreatment and exposure sessions. Gene-exposure interactions were evaluated in the AGTR1 A1166C polymorphism, on which recruitment was stratified, and other candidate genes, including TRPV1 and GSTM1.
MEASUREMENTS AND MAIN RESULTS	Compared with filtered air, exposure to diesel exhaust resulted in a significar reduction in BAd (mean, -0.09 mm, 95% confidence interval [CI], -0.01 to -0.17; P = 0.03). Pretreatment with antioxidants augmented diesel exhaust-related vasoconstriction with a mean change in BAd of -0.18 mm (95% CI, -0.28 to -0.07 mm; P = 0.001). Diesel exhaust-related vasoconstriction was primarily observed in the variant alleles of AGTR1 and TRPV1. No association was found between diesel exhaust inhalation and flow-mediated dilation.
CONCLUSIONS	We confirmed that short-term exposure to diesel exhaust in healthy subjects associated with acute vasoconstriction in a conductance artery and found suggestive evidence of involvement of nociception and renin-angiotensin systems in this effect. Pretreatment with an antioxidant regimen increased vasoconstriction.

표-386. PubMed 논문번호 26600256의 내용 요약

구분	내용
PubMed ID	26600256
TITLE	Long- and Short-term Exposure to Air Pollution and Inflammatory/Hemostatic Markers in Midlife Women.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000421
AUTHORS	Green Rochelle, Broadwin Rachel, Malig Brian, Basu Rupa, Gold Ellen B, Qi Lihong, Sternfeld Barbara, Bromberger Joyce T, Greendale Gail A, Kravitz Howard M, Tomey Kristin, Matthews Karen, Derby Carol A, Jackson Elizabeth A, Green Robin, Ostro Bart
BACKGROUND	Studies have reported associations between long-term air pollution exposures and cardiovascular mortality. The biological mechanisms connecting them remain uncertain.
METHODS	We examined associations of fine particles (PM2.5) and ozone with serum markers of cardiovascular disease risk in a cohort of midlife women. We obtained information from women enrolled at six sites in the multi-ethnic, longitudinal Study of Women's Health Across the Nation, including repeated measurements of high-sensitivity C-reactive protein, fibrinogen, tissue-type plasminogen activator antigen, plasminogen activator inhibitor type 1, and factor VIIc (factor VII coagulant activity). We obtained residence-proximate PM2.5 and ozone monitoring data for a maximum five annual visits, calculating prior year, 6-month, 1-month, and 1-day exposures and their relations to serum markers using longitudinal mixed models.
RESULTS	For the 2,086 women studied from 1999 to 2004, PM2.5 exposures were associated with all blood markers except factor VIIc after adjusting for age, race/ethnicity, education, site, body mass index, smoking, and recent alcohol use. Adjusted associations were strongest for prior year exposures for high-sensitivity C-reactive protein (21% increase per 10 $\mu g/m^3$ PM2.5, 95% confidence interval [CI]: 6.6, 37), tissue-type plasminogen activator antigen (8.6%, 95% CI: 1.8, 16), and plasminogen activator inhibitor (35%, 95% CI: 19, 53). An association was also observed between year prior ozone exposure and factor VIIc (5.7% increase per 10 ppb ozone, 95% CI: 2.9, 8.5).
CONCLUSIONS	Our findings suggest that prior year exposures to PM2.5 and ozone are associated with adverse effects on inflammatory and hemostatic pathways for cardiovascular outcomes in midlife women.

표-387. PubMed 논문번호 26605812의 내용 요약

구분	내용
PubMed ID	26605812
TITLE	Association Between Particulate Air Pollution and QT Interval Duration in an Elderly Cohort.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000424
AUTHORS	Mordukhovich Irina, Kloog Itai, Coull Brent, Koutrakis Petros, Vokonas Pantel, Schwartz Joel
BACKGROUND	Short-term fine particulate matter (PM2.5) exposure has been linked with increased QT interval duration, a marker of ventricular repolarization and a risk factor for cardiac arrhythmia and sudden death, in several studies. Only one previous study evaluated whether long-term PM exposure is related to the QT interval. We aim to evaluate whether subchronic and long-term exposure to PM2.5 at home is linked with QT duration in an elderly cohort.
METHODS	We measured heart-rate corrected QT interval duration among 404 participants from the Greater Boston area between 2003 and 2011. We modeled residential PM2.5 exposures using a hybrid satellite- and land use-based model. We evaluated associations between moving averages of short-term (1-2 days), subchronic (3-28 days), and long-term (1 year) pollutant exposures and corrected QT duration using linear mixed models. We also evaluated effect modification by oxidative stress genetic score using separated regression models and interaction terms.
RESULTS	We observed positive associations between subchronic and long-term PM2.5 exposure and corrected QT duration, with the strongest results for longer-term exposures. For example, a one standard deviation increase in 1-year PM2.5 was associated with a 6.3 ms increase in corrected QT (95% confidence interval: 1.8, 11). We observed somewhat greater effects among subjects with higher (8.5 ms) rather than lower (3.1 ms) oxidative stress allelic profiles (P interaction = 0.25).
CONCLUSIONS	PM2.5 was associated with increased corrected QT duration in an elderly cohort. While most previous studies focused on short-term air pollution exposures, our results suggest that longer-term exposures are associated with cardiac repolarization.

표-388. PubMed 논문번호 26607712의 내용 요약

구분	내용
PubMed ID	26607712
TITLE	Effect Modification of Long-Term Air Pollution Exposures and the Risk of Incident Cardiovascular Disease in US Women.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.115.002301
AUTHORS	Hart Jaime E, Puett Robin C, Rexrode Kathryn M, Albert Christine M, Laden Francine
BACKGROUND	Ambient air pollution exposures have been frequently linked to cardiovascular disease (CVD) morbidity and mortality. However, less is known about the populations most susceptible to these adverse effects.
METHODS AND RESULTS	We assessed the associations of long-term particulate matter (PM) exposures with incident CVD in a nationwide cohort of 114 537 women in the Nurses' Health Study, and performed analyses to identify subpopulations at the greatest risk. Residential address level time-varying monthly exposures to PM2.5, PM10, and PM2.5 to 10 microns in diameter were estimated from spatio-temporal prediction models. In multivariable models, increases in all size fractions of PM were associated with small, but not statistically significant, increased risks of total CVD, coronary heart disease, and stroke. PM-associated CVD risks were statistically significantly higher among women with diabetes as compared to those without (P-for-interaction <0.0001 for PM10 and PM2.5 and 0.007 for PM2.5 to 10). For each 10 μg/m(3) increase in 12-month average PM2.5, PM2.5 to 10, and PM10, the multivariable adjusted hazard ratios were 1.44 (95% CI: 1.23 to 1.68), 1.17 (95% CI: 1.05 to 1.30), and 1.19 (95% CI: 1.10 to 1.28) among women with diabetes. There were also suggestions of higher risks among older (≥70 years) women, the obese, and those living in the Northeast and South. Smoking status and family history did not consistently modify the association between PM and CVD, and risks were most elevated with exposures in the previous 12 months.
CONCLUSIONS	In this nationwide cohort, women with diabetes were identified as the subpopulation most sensitive to the adverse cardiovascular health effects of PM.

표-389. PubMed 논문번호 26613345의 내용 요약

구분	내용
PubMed ID	26613345
TITLE	Association between satellite-based estimates of long-term PM2.5 exposure and coronary artery disease.
JOURNAL	Environmental research: 10.1016/j.envres.2015.10.026
AUTHORS	McGuinn Laura A, Ward-Caviness Cavin K, Neas Lucas M, Schneider Alexandra, Diaz-Sanchez David, Cascio Wayne E, Kraus William E, Hauser Elizabeth, Dowdy Elaine, Haynes Carol, Chudnovsky Alexandra, Koutrakis Petros, Devlin Robert B, McGuinn Laura A, Ward-Caviness Cavin K, Neas Lucas M, Schneider Alexandra, Diaz-Sanchez David, Cascio Wayne E, Kraus William E, Hauser Elizabeth, Dowdy Elaine, Haynes Carol, Chudnovsky Alexandra, Koutrakis Petros, Devlin Robert B
BACKGROUND	Epidemiological studies have identified associations between long-term PM2.5 exposure and cardiovascular events, though most have relied on concentrations from central-site air quality monitors.
METHODS	We utilized a cohort of 5679 patients who had undergone cardiac catheterization at Duke University between 2002-2009 and resided in North Carolina. We used estimates of daily PM2.5 concentrations for North Carolina during the study period based on satellite derived Aerosol Optical Depth (AOD) measurements and PM2.5 concentrations from ground monitors, which were spatially resolved with a 10×10km resolution, matched to each patient's residential address and averaged for the year prior to catheterization. The Coronary Artery Disease (CAD) index was used to measure severity of CAD; scores >23 represent a hemodynamically significant coronary artery lesion in at least one major coronary vessel. Logistic regression modeled odds of having CAD or an MI with each 1 μg/m(3) increase in annual average PM2.5, adjusting for sex, race, smoking status and socioeconomic status.
RESULTS	In adjusted models, a 1 μg/m(3) increase in annual average PM2.5 was associated with an 11.1% relative increase in the odds of significant CAD (95% CI: 4.0-18.6%) and a 14.2% increase in the odds of having a myocardial infarction (MI) within a year prior (95% CI: 3.7-25.8%).
CONCLUSIONS	Satellite-based estimates of long-term PM2.5 exposure were associated with both coronary artery disease (CAD) and incidence of myocardial infarction (MI) in a cohort of cardiac catheterization patients.

표-390. PubMed 논문번호 26614250의 내용 요약

구분	내용
PubMed ID	26614250
TITLE	Association of Carbon Monoxide exposure with blood pressure among pregnant women in rural Ghana: Evidence from GRAPHS.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2015.10.004
AUTHORS	Quinn Ashlinn K, Ae-Ngibise Kenneth Ayuurebobi, Jack Darby W, Boamah Ellen Abrafi, Enuameh Yeetey, Mujtaba Mohammed Nuhu, Chillrud Steven N, Wylie Blair J, Owusu-Agyei Seth, Kinney Patrick L, Asante Kwaku Poku, Quinn Ashlinn K, Ae-Ngibise Kenneth Ayuurebobi, Jack Darby W, Boamah Ellen Abrafi, Enuameh Yeetey, Mujtaba Mohammed Nuhu, Chillrud Steven N, Wylie Blair J, Owusu-Agyei Seth, Kinney Patrick L, Asante Kwaku Poku
BACKGROUND AND OBJECTIVE	The Ghana Randomized Air Pollution and Health Study (GRAPHS) is a community-level randomized-controlled trial of cookstove interventions for pregnant women and their newborns in rural Ghana. Given that household air pollution from biomass burning may be implicated in adverse cardiovascular outcomes, we sought to determine whether exposure to carbon monoxide (CO) from woodsmoke was associated with blood pressure (BP) among 817 adult women.
METHODS	Multivariate linear regression models were used to evaluate the association between CO exposure, determined with 72 hour personal monitoring at study enrollment, and BP, also measured at study enrollment. At the time of these assessments, women were in the first or second trimester of pregnancy.
RESULTS	A significant positive association was found between CO exposure and diastolic blood pressure (DBP): on average, each 1 ppm increase in exposure to CO was associated with 0.43 mmHg higher DBP [0.01, 0.86]. A non-significant positive trend was also observed for systolic blood pressure (SBP).
CONCLUSION	This study is one of very few to have examined the relationship between household air pollution and blood pressure among pregnant women, who are at particular risk for hypertensive complications. The results of this cross-sectional study suggest that household air pollution from wood-burning fires is associated with higher blood pressure, particularly DBP, in pregnant women at early to mid-gestation. The clinical implications of the observed association toward the eventual development of chronic hypertension and/or hypertensive complications of pregnancy remain uncertain, as few of the women were overtly hypertensive at this point in their pregnancies.

표-391. PubMed 논문번호 26627181의 내용 요약

구분	내용
PubMed ID	26627181
TITLE	Meta-analysis of the association between second-hand smoke exposure and ischaemic heart diseases, COPD and stroke.
JOURNAL	BMC public health: 10.1186/s12889-015-2489-4
AUTHORS	Fischer Florian, Kraemer Alexander
BACKGROUND	Second-hand smoke (SHS) is the most important contaminant of indoor air in first world countries. The risks associated with SHS exposure are highly relevant, because many people are regularly, and usually involuntarily, exposed to SHS. This study aims to quantify the effects of SHS exposure. Therefore, its impact on ischaemic heart diseases (IHD), chronic obstructive pulmonary diseases (COPD) and stroke will be considered.
METHODS	A systematic literature review was conducted to identify articles dealing with the association between SHS and the three outcomes IHD, COPD and stroke. Overall, 24 articles were included in a meta-analysis using a random effects model. Effect sizes stratified for sex and for both sexes combined were calculated.
RESULTS	The synthesis of primary studies revealed significant effect sizes for the association between SHS exposure and all three outcomes. The highest RR for both sexes combined was found for COPD (RR = 1.66, 95 % CI: 1.38-2.00). The RR for both sexes combined was 1.35 (95 % CI: 1.22-1.50) for stroke and 1.27 (95 % CI: 1.10-1.48) for IHD. The risks were higher in women than in men for all three outcomes.
CONCLUSIONS	This is the first study to calculate effect sizes for the association between SHS exposure and the disease outcomes IHD, COPD, and stroke at once. Overall, the effect sizes are comparable with previous findings in meta-analyses and therefore assumed to be reliable. The results indicate the high relevance of public health campaigns and legislation to protect non-smokers from the adverse health effects attributable to SHS exposure.

표-392. PubMed 논문번호 26629599의 내용 요약

구분	내용
PubMed ID	26629599
TITLE	Ischemic Heart Disease Mortality and Long-Term Exposure to Source-Related Components of U.S. Fine Particle Air Pollution.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1509777
AUTHORS	Thurston George D, Burnett Richard T, Turner Michelle C, Shi Yuanli, Krewski Daniel, Lall Ramona, Ito Kazuhiko, Jerrett Michael, Gapstur Susan M, Diver W Ryan, Pope C Arden, Thurston George D, Burnett Richard T, Turner Michelle C, Shi Yuanli, Krewski Daniel, Lall Ramona, Ito Kazuhiko, Jerrett Michael, Gapstur Susan M, Diver W Ryan, Pope C Arden
BACKGROUND	Fine particulate matter (PM2.5) air pollution exposure has been identified as a global health threat. However, the types and sources of particles most responsible are not yet known.
OBJECTIVES	We sought to identify the causal characteristics and sources of air pollution underlying past associations between long-term PM2.5 exposure and ischemic heart disease (IHD) mortality, as established in the American Cancer Society's Cancer Prevention Study-II cohort.
METHODS	Individual risk factor data were evaluated for 445,860 adults in 100 U.S. metropolitan areas followed from 1982 through 2004 for vital status and cause of death. Using Cox proportional hazard models, we estimated IHD mortality hazard ratios (HRs) for PM2.5, trace constituents, and pollution source-associated PM2.5, as derived from air monitoring at central stations throughout the nation during 2000-2005.
RESULTS	Associations with IHD mortality varied by PM2.5 mass constituent and source. A coal combustion PM2.5 IHD HR = 1.05 (95% CI: 1.02, 1.08) per microgram/cubic meter, versus an IHD HR = 1.01 (95% CI: 1.00, 1.02) per microgram/cubic meter PM2.5 mass, indicated a risk roughly five times higher for coal combustion PM2.5 than for PM2.5 mass in general, on a per microgram/cubic meter PM2.5 basis. Diesel traffic-related elemental carbon (EC) soot was also associated with IHD mortality (HR = 1.03; 95% CI: 1.00, 1.06 per 0.26- μg/m3 EC increase). However, PM2.5 from both wind-blown soil and biomass combustion was not associated with IHD mortality.
CONCLUSIONS	Long-term PM2.5 exposures from fossil fuel combustion, especially coal burning but also from diesel traffic, were associated with increases in IHD mortality in this nationwide population. Results suggest that PM2.5-mortality associations can vary greatly by source, and that the largest IHD health benefits per microgram/cubic meter from PM2.5 air pollution control may be achieved via reductions of fossil fuel combustion exposures, especially from coal-burning sources.
CITATION	Thurston GD, Burnett RT, Turner MC, Shi Y, Krewski D, Lall R, Ito K, Jerrett M, Gapstur SM, Diver WR, Pope CA III. 2016. Ischemic heart disease mortality and long-term exposure to source-related components of U.S. fine particle air pollution. Environ Health Perspect 124:785-794; http://dx.doi.org/10.1289/ehp.1509777.

표-393. PubMed 논문번호 26644456의 내용 요약

구분	내용
PubMed ID	26644456
TITLE	Short-term metal particulate exposures decrease cardiac acceleration and deceleration capacities in welders: a repeated-measures panel study.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2015-103052
AUTHORS	Umukoro Peter E, Cavallari Jennifer M, Fang Shona C, Lu Chensheng, Lin Xihong, Mittleman Murray A, Christiani David C, Umukoro Peter E, Cavallari Jennifer M, Fang Shona C, Lu Chensheng, Lin Xihong, Mittleman Murray A, Christiani David C
OBJECTIVE	Acceleration (AC) and deceleration (DC) capacities measure heart rate variability during speeding up and slowing down of the heart, respectively. We investigated associations between AC and DC with occupational short-term metal PM2.5 exposures.
METHODS	A panel of 48 male welders had particulate matter less than 2.5 microns in diameter (PM2.5) exposure measurements over 4-6 h repeated over 5 sampling periods between January 2010 and June 2012. We simultaneously obtained continuous recordings of digital ECG using a Holter monitor. We analysed ECG data in the time domain to obtain hourly AC and DC. Linear mixed models were used to assess the associations between hourly PM2.5 exposure and each of hourly AC and DC, controlling for age, smoking status, active smoking, exposure to secondhand smoke, season/time of day when ECG reading was obtained and baseline AC or DC. We also ran lagged exposure response models for each successive hour up to 3 h after onset of exposure.
RESULTS	Mean (SD) shift PM2.5 exposure during welding was 0.47 (0.43) mg/m(3). Significant exposure-response associations were found for AC and DC with increased PM2.5 exposure. In our adjusted models without any lag between exposure and response, a 1 mg/m(3) increase of PM2.5 was associated with a decrease of 1.46 (95% CI 1.00 to 1.92) ms in AC and a decrease of 1.00 (95% CI 0.53 to 1.46) ms in DC. The effect of PM2.5 on AC and DC was maximal immediately postexposure and lasted 1 h following exposure.
CONCLUSIONS	There are short-term effects of metal particulates on AC and DC.

표-394. PubMed 논문번호 26660284의 내용 요약

구분	내용
PubMed ID	26660284
TITLE	Traffic-Related Air Pollution, Blood Pressure, and Adaptive Response of Mitochondrial Abundance.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.115.018802
AUTHORS	Zhong Jia, Cayir Akin, Trevisi Letizia, Sanchez-Guerra Marco, Lin Xinyi, Peng Cheng, Bind Marie-Ab□le, Prada Diddier, Laue Hannah, Brennan Kasey J M, Dereix Alexandra, Sparrow David, Vokonas Pantel, Schwartz Joel, Baccarelli Andrea A, Zhong Jia, Cayir Akin, Trevisi Letizia, Sanchez-Guerra Marco, Lin Xinyi, Peng Cheng, Bind Marie-Ab□le, Prada Diddier, Laue Hannah, Brennan Kasey J M, Dereix Alexandra, Sparrow David, Vokonas Pantel, Schwartz Joel, Baccarelli Andrea A
BACKGROUND	Exposure to black carbon (BC), a tracer of vehicular-traffic pollution, is associated with increased blood pressure (BP). Identifying biological factors that attenuate BC effects on BP can inform prevention. We evaluated the role of mitochondrial abundance, an adaptive mechanism compensating for cellular-redox imbalance, in the BC-BP relationship.
METHODS AND RESULTS	At ≥ 1 visits among 675 older men from the Normative Aging Study (observations=1252), we assessed daily BP and ambient BC levels from a stationary monitor. To determine blood mitochondrial abundance, we used whole blood to analyze mitochondrial-to-nuclear DNA ratio (mtDNA/nDNA) using quantitative polymerase chain reaction. Every standard deviation increase in the 28-day BC moving average was associated with 1.97 mm Hg (95% confidence interval [CI], 1.23-2.72; P<0.0001) and 3.46 mm Hg (95% CI, 2.06-4.87; P<0.0001) higher diastolic and systolic BP, respectively. Positive BC-BP associations existed throughout all time windows. BC moving averages (5-day to 28-day) were associated with increased mtDNA/nDNA; every standard deviation increase in 28-day BC moving average was associated with 0.12 standard deviation (95% CI, 0.03-0.20; P=0.007) higher mtDNA/nDNA. High mtDNA/nDNA significantly attenuated the BC-systolic BP association throughout all time windows. The estimated effect of 28-day BC moving average on systolic BP was 1.95-fold larger for individuals at the lowest mtDNA/nDNA quartile midpoint (4.68 mm Hg; 95% CI, 3.03-6.33; P<0.0001), in comparison with the top quartile midpoint (2.40 mm Hg; 95% CI, 0.81-3.99; P=0.003).
CONCLUSIONS	In older adults, short-term to moderate-term ambient BC levels were associated with increased BP and blood mitochondrial abundance. Our findings indicate that increased blood mitochondrial abundance is a compensatory response and attenuates the cardiac effects of BC.

표-395. PubMed 논문번호 26670352의 내용 요약

구분	내용
PubMed ID	26670352
TITLE	Health benefits related to the reduction of PM concentration in ambient air, Silesian Voivodeship, Poland.
JOURNAL	International journal of occupational medicine and environmental health: 10.13075/ijomeh.1896.00493
AUTHORS	Kowalski Michał, Kowalska Katarzyna, Kowalska Małgorzata
OBJECTIVES	Health Impact Assessments (HIA) approach can be executed by calculating the attributable burden of disease. The most common indicators used in the HIA methodology are: premature mortality, morbidity, life-expectancy, and Disability Adjusted Life-Year (DALY). The term Disability Adjusted Life-Years (DALYs) indicates months/years lost due to a premature death or disability. The aim of the study was to present health benefits, expressed in terms of lower total mortality and cardio-respiratory hospitalization rates, due to a decreased particulate matter (PM) concentration in ambient air, in Silesian voivodeship.
MATERIAL AND METHODS	In this paper, results obtained from the APHEKOM (Improving Knowledge and Communication for Decision Making on Air Pollution and Health in Europe) project, which provided specialized HIA tools, useful for assessing health benefits resulting from reducing air pollution, were used. Both short-term and long-term exposure HIA tools were applied with regard to the appropriate data for Silesian voivodeship. Exposure data were obtained from the Regional Environmental Inspectorate in Katowice, while population and health data were obtained from the Central Statistical Office of Poland or from the Silesian Voivodeship Office, respectively.
RESULTS	Health benefits that are related to an improvement of ambient air quality in Silesia region are similar to previous estimates obtained for Krak□w city. The reduction of short-term exposure to PM10 by 5 μg/m3 results in a lower number of yearly non-external deaths (2.6-2.75 per 100 000 inhabitants). This effect was also shown to be similar in the city of Zabrze, as well as in the whole Silesia region.
CONCLUSIONS	The Health Impact Assessments tools developed by the World Health Organization (WHO) can help public health experts make decisions in order to improve the health of populations living in particular regions of Europe.

표-396. PubMed 논문번호 26680605의 내용 요약

구분	내용
PubMed ID	26680605
TITLE	Long-Term Ozone Exposure and Mortality in a Large Prospective Study.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201508-1633OC
AUTHORS	Turner Michelle C, Jerrett Michael, Pope C Arden, Krewski Daniel, Gapstur Susan M, Diver W Ryan, Beckerman Bernardo S, Marshall Julian D, Su Jaso Crouse Daniel L, Burnett Richard T
RATIONALE	Tropospheric ozone (O3) is potentially associated with cardiovascular diseas risk and premature death. Results from long-term epidemiological studies o O3 are scarce and inconclusive.
OBJECTIVES	In this study, we examined associations between chronic ambient O3 exposu and all-cause and cause-specific mortality in a large cohort of U.S. adults.
METHODS	Cancer Prevention Study II participants were enrolled in 1982. A total of 669,046 participants were analyzed, among whom 237,201 deaths occurred through 2004. We obtained estimates of O3 concentrations at the participant residence from a hierarchical Bayesian space-time model. Estimates of fine particulate matter (particulate matter with an aerodynamic diameter of up to 2.5 μm [PM2.5]) and NO2 concentrations were obtained from land use regression. Cox proportional hazards regression models were used to examin mortality associations adjusted for individual- and ecological-level covariate
MEASUREMENTS AND MAIN RESULTS	In single-pollutant models, we observed significant positive associations between O3, PM2.5, and NO2 concentrations and all-cause and cause-specific mortality. In two-pollutant models adjusted for PM2.5, significant positive associations remained between O3 and all-cause (hazard ratio [HR] per 10 ppb, 1.02; 95% confidence interval [CI], 1.01-1.04), circulatory (HR, 1.03; 95% CI, 1.01-1.05), and respiratory mortality (HR, 1.12; 95% CI, 1.08-1.16) that were unchanged with further adjustment for NO2. We also observed positive mortality associations with both PM2.5 (both near source and regional) and NO2 in multipollutant models.
CONCLUSIONS	Findings derived from this large-scale prospective study suggest that long-term ambient O3 contributes to risk of respiratory and circulatory mortality. Substantial health and environmental benefits may be achieved by implementing further measures aimed at controlling O3 concentrations.

표-397. PubMed 논문번호 26717218의 내용 요약

구분	내용
PubMed ID	26717218
TITLE	Environmental triggers of acute myocardial infarction: results of a nationwide multiple-factorial population study.
JOURNAL	Acta cardiologica: 10.2143/AC.70.6.3120182
AUTHORS	Claeys Marc J, Coenen Sarah, Colpaert Charlotte, Bilcke Joke, Beutels Phillip, Wouters Kristien, Legrand Victor, Van Damme Pierre, Vrints Christiaan
OBJECTIVE	The objective of this study was to study the independent environmental triggers of ST-elevation myocardial infarction (STEMI) in a multifactorial environmental population model.
METHODS AND RESULTS	Daily counts of all STEMI patients who underwent urgent percutaneous coronary intervention over the period 2006-2009 in Belgium were associated with average daily meteorological data and influenza-like illness incidence data. The following meteorological measures were investigated: particulate matter less than 10 μM (PM10) and less than 2.5 μM (PM(2.5)), ozone, black smoke, temperature and relative humidity. During the study period a total of 15,964 STEMI patients (mean age 63, 75% male) were admitted with a daily average admission rate of 11 ± 4 patients. A multivariate Poisson regression analysis showed that only the temperature was significantly correlated with STEMI, with an 8% increase in the risk of STEMI for each 10° C decrease in temperature (adjusted incidence risk ratio (IRR) 0.92, 95% CI 0.89-0.96). The effects of temperature were consistent among several subpopulations but the strongest effect was seen in diabetic patients (IRR 0.85, 95% CI 0.78 -0.95). There was a trend for an incremental risk of STEMI for each 10 $\mu g/m^3$ PM(2.5) increase and during influenza epidemics with IRR of 1.02 (95% CI 1.00-1.04) and 1.07 (95% CI 0.98-1.16), respectively.
CONCLUSION	In a global environmental model, low temperature is the most important environmental trigger for STEMI, whereas air pollution and influenza epidemics only seem to have a modest effect.

표-398. PubMed 논문번호 26731791의 내용 요약

구분	내용
PubMed ID	26731791
TITLE	Genome-Wide Analysis of DNA Methylation and Fine Particulate Matter Air Pollution in Three Study Populations: KORA F3, KORA F4, and the Normative Aging Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1509966
AUTHORS	Panni Tommaso, Mehta Amar J, Schwartz Joel D, Baccarelli Andrea A, Just Allan C, Wolf Kathrin, Wahl Simone, Cyrys Josef, Kunze Sonja, Strauch Konstantin, Waldenberger Melanie, Peters Annette
BACKGROUND	Epidemiological studies have reported associations between particulate matter (PM) concentrations and cancer and respiratory and cardiovascular diseases. DNA methylation has been identified as a possible link but so far it has only been analyzed in candidate sites.
OBJECTIVES	We studied the association between DNA methylation and short- and mid-term air pollution exposure using genome-wide data and identified potential biological pathways for additional investigation.
METHODS	We collected whole blood samples from three independent studies-KORA F3 (2004-2005) and F4 (2006-2008) in Germany, and the Normative Aging Study (1999-2007) in the United States-and measured genome-wide DNA methylation proportions with the Illumina 450k BeadChip. PM concentration was measured daily at fixed monitoring stations and three different trailing averages were considered and regressed against DNA methylation: 2-day, 7-day and 28-day. Meta-analysis was performed to pool the study-specific results.
RESULTS	Random-effect meta-analysis revealed 12 CpG (cytosine-guanine dinucleotide) sites as associated with PM concentration (1 for 2-day average, 1 for 7-day, and 10 for 28-day) at a genome-wide Bonferroni significance level ($p \leq 7.5E-8$); 9 out of these 12 sites expressed increased methylation. Through estimation of I2 for homogeneity assessment across the studies, 4 of these sites (annotated in NSMAF, C1orf212, MSGN1, NXN) showed $p > 0.05$ and $I2 < 0.5$: the site from the 7-day average results and 3 for the 28-day average. Applying false discovery rate, p-value < 0.05 was observed in 8 and 1,819 additional CpGs at 7- and 28-day average PM2.5 exposure respectively.
CONCLUSION	The PM-related CpG sites found in our study suggest novel plausible systemic pathways linking ambient PM exposure to adverse health effect through variations in DNA methylation.
CITATION	Panni T, Mehta AJ, Schwartz JD, Baccarelli AA, Just AC, Wolf K, Wahl S, Cyrys J, Kunze S, Strauch K, Waldenberger M, Peters A. 2016. A genome-wide analysis of DNA methylation and fine particulate matter air pollution in three study populations: KORA F3, KORA F4, and the Normative Aging Study. Environ Health Perspect 124:983-990; http://dx.doi.org/10.1289/ehp.1509966.

표-399. PubMed 논문번호 26738952의 내용 요약

구분	내용
PubMed ID	26738952
TITLE	Respiratory Filter Reduces the Cardiovascular Effects Associated With Diesel Exhaust Exposure: A Randomized, Prospective, Double-Blind, Controlled Study of Heart Failure: The FILTER-HF Trial.
JOURNAL	JACC. Heart failure: 10.1016/j.jchf.2015.07.018
AUTHORS	Vieira Jefferson L, Guimaraes Guilherme V, de Andre Paulo A, Cruz F□tima D, Saldiva Paulo H Nascimento, Bocchi Edimar A
OBJECTIVES	The goal of this study was to test the effects of a respiratory filter intervention (filter) during controlled pollution exposure.
BACKGROUND	Air pollution is considered a risk factor for heart failure (HF) decompensation and mortality.
METHODS	This study was a double-blind, randomized to order, controlled, 3-way crossover, single-center clinical trial. It enrolled 26 patients with HF and 15 control volunteers. Participants were exposed in 3 separate sessions to clean air, unfiltered diesel exhaust exposure (DE), or filtered DE. Endpoints were endothelial function assessed by using the reactive hyperemia index (RHi), arterial stiffness, serum biomarkers, 6-min walking distance, and heart rate variability.
RESULTS	In patients with HF, DE was associated with a worsening in RHi from 2.17 (interquartile range [IQR]: 1.8 to 2.5) to 1.72 (IQR: 1.5 to 2.2; p = 0.002) and an increase in B-type natriuretic peptide (BNP) from 47.0 pg/ml (IQR: 17.3 to 118.0 pg/ml) to 66.5 pg/ml (IQR: 26.5 to 155.5 pg/ml; p = 0.004). Filtration reduced the particulate concentration (325 ± 31 μg/m(3) vs. 25 ± 6 μg/m(3); p < 0.001); in the group with HF, filter was associated with an improvement in RHi from 1.72 (IQR: 1.5 to 2.2) to 2.06 (IQR: 1.5 to 2.6; p = 0.019) and a decrease in BNP from 66.5 pg/ml (IQR: 26.5 to 155.5 pg/ml) to 44.0 pg/ml (IQR: 20.0 to 110.0 pg/ml; p = 0.015) compared with DE. In both groups, DE decreased the 6-min walking distance and arterial stiffness, although filter did not change these responses. DE had no effect on heart rate variability or exercise testing.
CONCLUSIONS	To our knowledge, this trial is the first to show that a filter can reduce both endothelial dysfunction and BNP increases in patients with HF during DE. Given these potential benefits, the widespread use of filters in patients with HF exposed to traffic-derived air pollution may have beneficial public health effects and reduce the burden of HF. (Effects of Air Pollution Exposure Reduction by Filter Mask on Heart Failure; NCT01960920).

표-400. PubMed 논문번호 26742457의 내용 요약

구분	내용
PubMed ID	26742457
TITLE	The effects of air pollution and weather conditions on the incidence of acute myocardial infarction.
JOURNAL	The American journal of emergency medicine: 10.1016/j.ajem.2015.11.068
AUTHORS	Sen Taner, Astarcioglu Mehmet Ali, Asarcikli Lale Dinc, Kilit Celal, Kafes Habibe, Parspur Afsin, Yaymaci Mehmet, Pinar Mesut, T□fekcioglu Omac, Amasyali Basri, Sen Taner, Astarcioglu Mehmet Ali, Asarcikli Lale Dinc, Kilit Celal, Kafes Habibe, Parspur Afsin, Yaymaci Mehmet, Pinar Mesut, T□fekcioglu Omac, Amasyali Basri
OBJECTIVE	In this retrospective study, we investigated the association between air pollution and weather conditions with the incidence of acute myocardial infarction (AMI) in the city of Kutahya.
METHODS	A total of 402 patients who were admitted with acute ST segment elevation MI and non-ST segment elevation MI were included in the study in 1 year. Daily maximum, minimum, and mean ambient temperature and mean barometric pressure data were obtained from the Kutahya Meteorology Department. Daily air pollution data were obtained from the Web site of National Air Quality Observation Network (http://www.havaizleme.gov.tr).
RESULTS	Increase in ambient air temperature in the day of MI and 2 days before the day of MI according to their control days was correlated with increase in number of MI cases. When we grouped the patients according to ages as 30-54, 55-65, and >65 years, we found that there was a relation between sulfur dioxide (SO2) and the occurrence of AMI for the age group of 30-54 for the same day (D0) (P<.017). The number of AMIs was the lowest in fall season, whereas the number of AMIs was the highest in winter season.
CONCLUSION	There was no statistically significant association between the particulates with diameter b=10 μm, SO2 concentrations, air pressure, and the risk of AMI, but there was statistically significant relation between occurrence of MI and SO2 for the patients under age of 55 years. The number of AMIs was the lowest in fall season, whereas the number of AMIs was the highest in winter season.

표-401. PubMed 논문번호 26745732의 내용 요약

구분	내용
PubMed ID	26745732
TITLE	Oxidative burden of fine particulate air pollution and risk of cause-specific mortality in the Canadian Census Health and Environment Cohort (CanCHEC).
JOURNAL	Environmental research: 10.1016/j.envres.2015.12.013
AUTHORS	Weichenthal Scott, Crouse Daniel L, Pinault Lauren, Godri-Pollitt Krystal, Lavigne Eric, Evans Greg, van Donkelaar Aaron, Martin Randall V, Burnett Rick T, Weichenthal Scott, Crouse Daniel L, Pinault Lauren, Godri-Pollitt Krystal, Lavigne Eric, Evans Greg, van Donkelaar Aaron, Martin Randall V, Burnett Rick T
BACKROUND	Fine particulate air pollution (PM2.5) is known to contribute to cardiorespiratory mortality but it is not clear how PM2.5 oxidative burden (i.e. the ability of PM2.5 to cause oxidative stress) may influence long-term mortality risk.
METHODS	We examined the relationship between PM2.5 oxidative burden and cause-specific mortality in Ontario, Canada. Integrated PM2.5 samples were collected from 30 provincial monitoring sites between 2012 and 2013. The oxidative potential (% depletion/□g) of regional PM2.5 was measured as the ability of filter extracts to deplete antioxidants (glutathione and ascorbate) in a synthetic respiratory tract lining fluid. PM2.5oxidative burden was calculated as the product of PM2.5 mass concentrations and regional estimates of oxidative potential. In total, this study included 193,300 people who completed the Canadian long-form census in 1991 and who lived within 5km of a site where oxidative potential was measured. Deaths occurring between 1991 and 2009 were identified through record linkages and Cox proportional hazard models were used to estimate hazard ratios (and 95% confidence intervals) for interquartile changes in exposure adjusting for individual-level covariates and indirect-adjustment for smoking and obesity.
RESULTS	Glutathione-related oxidative burden was associated with cause-specific mortality. For lung cancer specifically, this metric was associated with a 12% (95% CI: 5.0-19) increased risk of mortality whereas a 5.0% (95% CI: 0.1, 10) increase was observed for PM2.5. Indirect adjustment for smoking and obesity decreased the lung cancer hazard ratio for glutathione-related oxidative burden but it remained significantly elevated (HR=1.07, 95% CI: 1.005, 1.146). Ascorbate-related oxidative burden was not associated with mortality.
CONCLUSIONS	Our findings suggest that glutathione-related oxidative burden may be more strongly associated with lung cancer mortality than PM2.5 mass concentrations.

표-402. PubMed 논문번호 26764400의 내용 요약

구분	내용
PubMed ID	26764400
TITLE	Long-term physical activity is associated with reduced arterial stiffness in older adults: longitudinal results of the SAPALDIA cohort study.
JOURNAL	Age and ageing: 10.1093/ageing/afv172
AUTHORS	Endes Simon, Schaffner Emmanuel, Caviezel Seraina, Dratva Julia, Autenrieth Christine S, Wanner Miriam, Martin Brian, Stolz Daiana, Pons Marco, Turk Alexander, Bettschart Robert, Schindler Christian, K□nzli Nino, Probst-Hensch Nicole, Schmidt-Trucks□ss Arno
BACKGROUND	longitudinal analyses of physical activity (PA) and arterial stiffness in populations of older adults are scarce. We examined associations between long-term change of PA and arterial stiffness in the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases in Adults (SAPALDIA).
METHODS	we assessed PA in SAPALDIA 2 (2001-03) and SAPALDIA 3 (2010-11) using a short questionnaire with a cut-off of at least 150 min of moderate-to-vigorous PA per week for sufficient activity. Arterial stiffness was measured oscillometrically by means of the brachial-ankle pulse wave velocity (baPWV) in SAPALDIA 3. We used multivariable mixed linear regression models adjusted for several potential confounders in 2,605 persons aged 50-81.
RESULTS	adjusted means of baPWV were significantly lower in persons with sufficient moderate-to-vigorous PA (i) in SAPALDIA 2 but not in SAPALDIA 3 (P = 0.048) and (ii) in both surveys (P = 0.001) compared with persons with insufficient activity in both surveys. There was a significant interaction between sex and the level of change in PA concerning baPWV (P = 0.03). The triples of parameter estimates describing the association between level of PA change and baPWV were not significantly different between the two sex-specific models (P = 0.07).
CONCLUSIONS	keeping up or adopting a physically active lifestyle was associated with lower arterial stiffness in older adults after a follow-up of almost a decade. Increasing the proportion of older adults adhering to PA recommendations incorporating also vigorous PA may have a considerable impact on vascular health at older age and may contribute to healthy ageing in general.

표-403. PubMed 논문번호 26828511의 내용 요약

구분	내용
PubMed ID	26828511
TITLE	Association between Floods and Acute Cardiovascular Diseases: A Population-Based Cohort Study Using a Geographic Information System Approach.
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph13020168
AUTHORS	Vanasse Alain, Cohen Alan, Courteau Josiane, Bergeron Patrick, Dault Roxanne, Gosselin Pierre, Blais Claudia, B□langer Diane, Rochette Louis, Chebana Fateh
BACKGROUND	Floods represent a serious threat to human health beyond the immediate risk of drowning. There is few data on the potential link between floods and direct consequences on health such as on cardiovascular health. This study aimed to explore the impact of one of the worst floods in the history of Quebec, Canada on acute cardiovascular diseases (CVD).
METHODS	A cohort study with a time series design with multiple control groups was built with the adult population identified in the Quebec Integrated Chronic Disease Surveillance System. A geographic information system approach was used to define the study areas. Logistic regressions were performed to compare the occurrence of CVD between groups.
RESULTS	The results showed a 25%-27% increase in the odds in the flooded population in spring 2011 when compared with the population in the same area in springs 2010 and 2012. Besides, an increase up to 69% was observed in individuals with a medical history of CVD.
CONCLUSION	Despite interesting results, the association was not statistically significant. A possible explanation to this result can be that the population affected by the flood was probably too small to provide the statistical power to answer the question, and leaves open a substantial possibility for a real and large effect.

표-404. PubMed 논문번호 26842828의 내용 요약

구분	내용
PubMed ID	26842828
TITLE	Legislative smoking bans for reducing harms from secondhand smoke exposure, smoking prevalence and tobacco consumption.
JOURNAL	The Cochrane database of systematic reviews: 10.1002/14651858.CD005992.pub3
AUTHORS	Frazer Kate, Callinan Joanne E, McHugh Jack, van Baarsel Susan, Clarke Anna, Doherty Kirsten, Kelleher Cecily
BACKGROUND	Smoking bans have been implemented in a variety of settings, as well as being part of policy in many jurisdictions to protect the public and employees from the harmful effects of secondhand smoke (SHS). They also offer the potential to influence social norms and the smoking behaviour of those populations they affect. Since the first version of this review in 2010, more countries have introduced national smoking legislation banning indoor smoking.
OBJECTIVES	To assess the effects of legislative smoking bans on (1) morbidity and mortality from exposure to secondhand smoke, and (2) smoking prevalence and tobacco consumption.
SEARCH METHODS	We searched the Cochrane Tobacco Addiction Group Specialised Register, MEDLINE, EMBASE, PsycINFO, CINAHL and reference lists of included studies. We also checked websites of various organisations. Date of most recent search; February 2015.
SELECTION CRITERIA	We considered studies that reported legislative smoking bans affecting populations. The minimum standard was having an indoor smoking ban explicitly in the study and a minimum of six months follow-up for measures of smoking behaviour. Our search included a broad range of research designs including: randomized controlled trials, quasi-experimental studies (i.e. non-randomized controlled studies), controlled before-and-after studies, interrupted time series as defined by the Cochrane Effective Practice and Organisation of Care Group, and uncontrolled pre- and post-ban data.
DATA COLLECTION AND ANALYSIS	One author extracted characteristics and content of the interventions, participants, outcomes and methods of the included studies and a second author checked the details. We extracted health and smoking behaviour outcomes. We did not attempt a meta-analysis due to the heterogeneity in design and content of the studies included. We evaluated the studies using qualitative narrative synthesis.
MAIN RESULTS	There are 77 studies included in this updated review. We retained 12 studies from the original review and identified 65 new studies. Evidence from 21 countries is provided in this update, an increase of eight countries from the original review. The nature of the intervention precludes randomized controlled trials. Thirty-six studies used an interrupted time series study design, 23 studies use a controlled before-and-after design and 18 studies are before-and-after studies with no control group; six of these studies use a cohort design. Seventy-two studies reported health outcomes, including cardiovascular (44), respiratory (21), and perinatal outcomes (7). Eleven studies reported national mortality rates for smoking-related diseases. A number of the studies report multiple health outcomes. There is consistent evidence of a positive impact of national smoking bans on improving cardiovascular health outcomes, and reducing mortality for associated smoking-related illnesses. Effects on respiratory and perinatal health were less consistent. We found 24 studies evaluating the impact of national smoke-free

표-405. PubMed 논문번호 26856365의 내용 요약

구분	내용
PubMed ID	26856365
TITLE	Historic air pollution exposure and long-term mortality risks in England and Wales: prospective longitudinal cohort study.
JOURNAL	Thorax: 10.1136/thoraxjnl-2015-207111
AUTHORS	Hansell Anna, Ghosh Rebecca E, Blangiardo Marta, Perkins Chloe, Vienneau Danielle, Goffe Kayoung, Briggs David, Gulliver John
INTRODUCTION	Long-term air pollution exposure contributes to mortality but there are few studies examining effects of very long-term (>25 years) exposures.
METHODS	This study investigated modelled air pollution concentrations at residence for 1971, 1981, 1991 (black smoke (BS) and SO2) and 2001 (PM10) in relation to mortality up to 2009 in 367,658 members of the longitudinal survey, a 1% sample of the English Census. Outcomes were all-cause (excluding accidents), cardiovascular (CV) and respiratory mortality.
RESULTS	BS and SO2 exposures remained associated with mortality decades after exposure-BS exposure in 1971 was significantly associated with all-cause (OR 1.02 (95% CI 1.01 to 1.04)) and respiratory (OR 1.05 (95% CI 1.01 to 1.09)) mortality in 2002-2009 (ORs expressed per 10 μg/m(3)). Largest effect sizes were seen for more recent exposures and for respiratory disease. PM10 exposure in 2001 was associated with all outcomes in 2002-2009 with stronger associations for respiratory (OR 1.22 (95% CI 1.04 to 1.44)) than CV mortality (OR 1.12 (95% CI 1.01 to 1.25)). Adjusting PM10 for past BS and SO2 exposures in 1971, 1981 and 1991 reduced the all-cause OR to 1.16 (95% CI 1.07 to 1.26) while CV and respiratory associations lost significance, suggesting confounding by past air pollution exposure, but there was no evidence for effect modification. Limitations include limited information on confounding by smoking and exposure misclassification of historic exposures.
CONCLUSIONS	This large national study suggests that air pollution exposure has long-term effects on mortality that persist decades after exposure, and that historic air pollution exposures influence current estimates of associations between air pollution and mortality.

표-406. PubMed 논문번호 26859533의 내용 요약

구분	내용
PubMed ID	26859533
TITLE	Particulate Matter Exposure and Cardiopulmonary Differences in the Multi-Ethnic Study of Atherosclerosis.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1409451
AUTHORS	Aaron Carrie P, Chervona Yana, Kawut Steven M, Diez Roux Ana V, Shen Mingwu, Bluemke David A, Van Hee Victor C, Kaufman Joel D, Barr R Graham
BACKGROUND	Particulate matter (PM) exposure may directly affect the pulmonary vasculature. Although the pulmonary vasculature is not easily measurable, differential associations for right ventricular (RV) and left ventricular (LV) mass may provide an indirect assessment of pulmonary vascular damage.
OBJECTIVES	We tested whether long-term exposure to PM < 2.5 μm (PM2.5) is associated with greater RV mass and RV mass/end-diastolic volume ratio relative to the LV.
METHODS	The Multi-Ethnic Study of Atherosclerosis performed cardiac magnetic resonance (CMR) imaging among participants 45-84 years old without clinical cardiovascular disease in 2000-2002 in six U.S. cities. A fine-scale spatiotemporal model estimated ambient PM2.5 exposure in the year before CMR; individually weighted estimates accounted for indoor exposure to ambient PM2.5. Linear regression models were adjusted for demographics, anthropometrics, smoking status, cardiac risk factors, and LV parameters, with additional adjustment for city.
RESULTS	The 4,041 included participants had a mean age of 61.5 years, and 47% were never smokers. The mean ambient PM2.5 was 16.4 μg/m3 and individually weighted PM2.5 was 11.0 μg/m3. PM2.5 exposure was associated with greater RV mass [ambient: 0.11 g per 5 μg/m3 (95% CI: -0.05, 0.27); individually weighted: 0.20 g per 5 μg/m3 (95% CI: 0.04, 0.36)] and a greater RV mass/end-diastolic volume ratio conditional on LV parameters. City-adjusted results for RV mass were of greater magnitude and were statistically significant for both measures of PM2.5, whereas those for RV mass/end-diastolic volume ratio were attenuated.
CONCLUSIONS	Long-term PM2.5 exposures were associated with greater RV mass and RV mass/end-diastolic volume ratio conditional on the LV; however, additional adjustment for city attenuated the RV mass/end-diastolic volume findings. These findings suggest that PM2.5 exposure may be associated with subclinical cardiopulmonary differences in this general population sample.
CITATION	Aaron CP, Chervona Y, Kawut SM, Diez Roux AV, Shen M, Bluemke DA, Van Hee VC, Kaufman JD, Barr RG. 2016. Particulate matter exposure and cardiopulmonary differences in the Multi-Ethnic Study of Atherosclerosis. Environ Health Perspect 124:1166-1173; http://dx.doi.org/10.1289/ehp.1409451.

표-407. PubMed 논문번호 26864652의 내용 요약

구분	내용
PubMed ID	26864652
TITLE	Risk estimates of mortality attributed to low concentrations of ambient fine particulate matter in the Canadian community health survey cohort.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-016-0111-6
AUTHORS	Pinault Lauren, Tjepkema Michael, Crouse Daniel L, Weichenthal Scott, van Donkelaar Aaron, Martin Randall V, Brauer Michael, Chen Hong, Burnett Richard T
BACKGROUND	Understanding the shape of the relationship between long-term exposure to ambient fine particulate matter (PM2.5) concentrations and health risks is critical for health impact and risk assessment. Studies evaluating the health risks of exposure to low concentrations of PM2.5 are limited. Further, many existing studies lack individual-level information on potentially important behavioural confounding factors.
METHODS	A prospective cohort study was conducted among a subset of participants in a cohort that linked respondents of the Canadian Community Health Survey to mortality (n = 299,500) with satellite-derived ambient PM2.5 estimates. Participants enrolled between 2000 and 2008 were followed to date of death or December 31, 2011. Cox proportional hazards models were used to estimate hazard ratios (HRs) for mortality attributed to PM2.5 exposure, adjusted for individual-level and contextual covariates, including smoking behaviour and body mass index (BMI).
RESULTS	Approximately 26,300 non-accidental deaths, of which 32.5 % were due to circulatory disease and 9.1 % were due to respiratory disease, occurred during the follow-up period. Ambient PM2.5 exposures were relatively low (mean = 6.3 μg/m(3)), yet each 10 μg/m(3) increase in exposure was associated with increased risks of non-accidental (HR = 1.26; 95 % CI: 1.19-1.34), circulatory disease (HR = 1.19; 95 % CI: 1.07-1.31), and respiratory disease mortality (HR = 1.52; 95 % CI: 1.26-1.84) in fully adjusted models. Higher hazard ratios were observed for respiratory mortality among respondents who never smoked (HR = 1.97; 95 % CI: 1.24-3.13 vs. HR = 1.45; 95 % CI: 1.17-1.79 for ever smokers), and among obese (BMI ≥ 30) respondents (HR = 1.76; 95 % CI: 1.15-2.69 vs. HR = 1.41; 95 % CI: 1.04-1.91 for normal weight respondents), though differences between groups were not statistically significant. A threshold analysis for non-accidental mortality estimated a threshold concentration of 0 μg/m(3) (+95 % CI = 4.5 μg/m(3)).
CONCLUSIONS	Increased risks of non-accidental, circulatory, and respiratory mortality were observed even at very low concentrations of ambient PM2.5. HRs were generally greater than most literature values, and adjusting for behavioural covariates served to reduce HR estimates slightly.

표-408. PubMed 논문번호 26867595의 내용 요약

구분	내용
PubMed ID	26867595
TITLE	Road traffic noise, air pollution and myocardial infarction: a prospective cohort study.
JOURNAL	International archives of occupational and environmental health: 10.1007/s00420-016-1115-9
AUTHORS	Bodin Theo, Bj□rk Jonas, Mattisson Kristoffer, Bottai Matteo, Rittner Ralf, Gustavsson Per, Jakobsson Kristina, □stergren Per-Olof, Albin Maria
PURPOSE	Both road traffic noise and air pollution have been linked to cardiovascular disease. However, there are few prospective epidemiological studies available where both road traffic noise and air pollution have been analyzed simultaneously. The aim of this study was to investigate the relation between road traffic noise, air pollution and incident myocardial infarction in both current (1-year average) and medium-term (3-year average) perspective.
METHODS	This study was based on a stratified random sample of persons aged 18-80 years who answered a public health survey in Sk□ne, Sweden, in 2000 (n = 13,512). The same individuals received a repeated survey in 2005 and 2010. Diagnoses of myocardial infarction (MI) were obtained from medical records for both inpatient and outpatient specialized care. The endpoint was first MI during 2000-2010. Participants with prior myocardial infarction were excluded at baseline. Yearly average levels of noise (L DEN) and air pollution (NO x) were estimated using geographic information system for residential address every year until censoring.
RESULTS	The mean exposure levels for road traffic noise and air pollution in 2005 were L DEN 51 dB(A) and NO x 11 □g/m(3), respectively. After adjustment for individual confounders (age, sex, body mass index, smoking, education, alcohol consumption, civil status, year, country of birth and physical activity), a 10-dB(A) increase in current noise exposure did not increase the incidence rate ratio (IRR) for MI, 0.99 (95 % CI 0.86-1.14). Neither did a 10- μg/m(3) increase in current NO x increase the risk of MI, 1.02 (95 % CI 0.86-1.21). The IRR for MI associated with combined exposure to road traffic noise 〉55 dB(A) and NO x 〉20 □g/m(3) was 1.21 (95 % CI 0.90-1.64) compared to 〈55 dB(A) and 〈20 □g/m(3).
CONCLUSIONS	This study did not provide evidence for an increased risk of MI due to exposure to road traffic noise or air pollution at moderate average exposure levels.

표-409. PubMed 논문번호 26900394의 내용 요약

구분	내용
PubMed ID	26900394
TITLE	Health status of male steel workers at an electric arc furnace (EAF) in Trentino, Italy.
JOURNAL	Journal of occupational medicine and toxicology (London, England): 10.1186/s12995-016-0095-8
AUTHORS	Cappelletti Roberto, Ceppi Marcello, Claudatus Justina, Gennaro Valerio
BACKGROUND	The aim of this retrospective cohort study was to determine if the workers of an Electric Arc Furnace (EAF), which recycles scrap, had higher mortality and morbidity due to possible exposure to pollutants at work. EAFs do not run on coke ovens. In EAFs 40 % of the particulate matter (PM) is made up of PM 2.5. The foundry dust contained iron, aluminum, zinc, manganese, lead, chromium, nickel, cadmium, mercury, arsenic, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls and dioxins.
METHODS	Mortality study: a cohort of 331 exposed workers (6731 person-years) was studied from 19/03/1979 to 31/12/2009 (mean follow up 20.7 years). The group of exposed workers was compared to the general population and to a small control group of 32 workers from the same company. Morbidity study: rates of exemption from health fee for the seven major diseases of 235 exposed workers were compared to the rates of exemption in the Province of Trento.
RESULTS	Mortality study: an excess mortality was found in the exposed workers as compared to the general population (SMR 1.13; 95 % CI: 0.76-1.62; 29 deaths) and to the internal group (RR 2.34; 95 % CI: 0.39-95.7). The mortality rate was increased for all tumours (SMR 1.36; 95 % CI: 0.75-2.29; 14 cases), for lung cancer (SMR 3.35; 95 % CI 1.45-6.60; 8 cases), for ischemic heart disease (SMR 1.27; 95 % CI: 0.35-3.26; 4 cases), for chronic liver disease (SMR 1.16; 95 % CI: 0.14-4.20; 2 cases) and for injury and poisoning (SMR 1.32; 95 % CI: 0.48-2.88; 6 cases). Morbidity study: there was a statistically significant increase of diabetes, rheumatoid arthritis, hypertension and cardiovascular diseases in exposed workers.
CONCLUSIONS	With the limitations of this relatively small cohort, we found a statistically significant increase of diabetes, cardiovascular diseases and deaths due to lung cancer in exposed workers. These findings cannot be explained by PAH exposure alone; metal particulates are the most important pollutants in the working area of EAFs. A reliable method for measuring metal PM in tissues is urgently needed for exposure assessment. This study underlines the necessity to maximize the standards of security toward foundry dusts/diffuse emission. Further studies on EAF's are needed to confirm our findings and to increase statistical power.

표-410. PubMed 논문번호 26901215의 내용 요약

구분	내용
PubMed ID	26901215
TITLE	Exposure to Agrochemicals and Cardiovascular Disease: A Review.
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph13020229
AUTHORS	Sekhotha Matome M, Monyeki Kotsedi D, Sibuyi Masezi E
INTRODUCTION	In the agricultural world there is a continuous loss of food, fiber and other commodities due to pests, disease and weeds before harvesting time. These losses had create lots of financial burden to the farm owners that might lead to shutting down of their daily business. Worldwide, there is an overall very high loss of agricultural products due to weeds growth alone. To counteract this problem most farmers resort to the use of agrochemicals to increase their production but compromising the health of their farmworkers. The purpose of the study will be to assess the relationship between the agrochemical particles and cardiovascular diseases among farmworkers.
METHOD	Non-systematic review was used to collect data. The following database were use: Medline, EBSCO, and Science Direct to search for the existing journal articles.
RESULTS	This study addresses the relationship between agrochemicals particles and cardiovascular diseases in the farming industries using literature review.
DISCUSSION	Other researchers had already done an extensive research on the pathway of potential mechanisms linking the ultrafine particulate matter to cardiovascular diseases. The outcomes of those investigations were the clinical results of events that might lead to the development of myocardial infarction, congestive heart failure (CHF), stroke, arrhythmia and sudden death. Xenobiotic compounds that maybe implicated in the pathophysiology of human cardiovascular diseases, will be examined and included in this study. There is compelling evidence suggesting that toxic free radicals of pesticides play an important role in human health.
CONCLUSION	There is a close relationship between agrochemicals particle and cardiovascular diseases.

표-411. PubMed 논문번호 26908518의 내용 요약

구분	내용
PubMed ID	26908518
TITLE	Long-term exposure to ambient ozone and mortality: a quantitative systematic review and meta-analysis of evidence from cohort studies.
JOURNAL	BMJ open: 10.1136/bmjopen-2015-009493
AUTHORS	Atkinson R W, Butland B K, Dimitroulopoulou C, Heal M R, Stedman J R, Carslaw N, Jarvis D, Heaviside C, Vardoulakis S, Walton H, Anderson H R
OBJECTIVES	While there is good evidence for associations between short-term exposure to ozone and a range of adverse health outcomes, the evidence from narrative reviews for long-term exposure is suggestive of associations with respiratory mortality only. We conducted a systematic, quantitative evaluation of the evidence from cohort studies, reporting associations between long-term exposure to ozone and mortality.
METHODS	Cohort studies published in peer-reviewed journals indexed in EMBASE and MEDLINE to September 2015 and PubMed to October 2015 and cited in reviews/key publications were identified via search strings using terms relating to study design, pollutant and health outcome. Study details and estimate information were extracted and used to calculate standardised effect estimates expressed as HRs per 10 ppb increment in long-term ozone concentrations.
RESULTS	14 publications from 8 cohorts presented results for ozone and all-cause and cause-specific mortality. We found no evidence of associations between long-term annual O3 concentrations and the risk of death from all causes, cardiovascular or respiratory diseases, or lung cancer. 4 cohorts assessed ozone concentrations measured during the warm season. Summary HRs for cardiovascular and respiratory causes of death derived from 3 cohorts were 1.01 (95% CI 1.00 to 1.02) and 1.03 (95% CI 1.01 to 1.05) per 10 ppb, respectively.
CONCLUSIONS	Our quantitative review revealed a paucity of independent studies regarding the associations between long-term exposure to ozone and mortality. The potential impact of climate change and increasing anthropogenic emissions of ozone precursors on ozone levels worldwide suggests further studies of the long-term effects of exposure to high ozone levels are warranted.

표-412. PubMed 논문번호 26911134의 내용 요약

구분	내용
PubMed ID	26911134
TITLE	Health effects of smoke from planned burns: a study protocol.
JOURNAL	BMC public health: 10.1186/s12889-016-2862-y
AUTHORS	O'Keeffe David, Dennekamp Martine, Straney Lahn, Mazhar Mahjabeen, O'Dwyer Tom, Haikerwal Anjali, Reisen Fabienne, Abramson Michael J, Johnston Fay
BACKGROUND	Large populations are exposed to smoke from bushfires and planned burns. Studies investigating the association between bushfire smoke and health have typically used hospital or ambulance data and been done retrospectively on large populations. The present study is designed to prospectively assess the association between individual level health outcomes and exposure to smoke from planned burns.
METHODS/DESIGN	A prospective cohort study will be conducted during a planned burn season in three locations in Victoria (Australia) involving 50 adult participants who undergo three rounds of cardiorespiratory medical tests, including measurements for lung inflammation, endothelial function, heart rate variability and markers of inflammation. In addition daily symptoms and twice daily lung function are recorded. Outdoor particulate air pollution is continuously measured during the study period in these locations. The data will be analysed using mixed effect models adjusting for confounders.
DISCUSSION	Planned burns depend on weather conditions and dryness of 'fuels' (i.e. forest). It is potentially possible that no favourable conditions occur during the study period. To reduce the risk of this occurring, three separate locations have been identified as having a high likelihood of planned burn smoke exposure during the study period, with the full study being rolled out in two of these three locations. A limitation of this study is exposure misclassification as outdoor measurements will be conducted as a measure for personal exposures. However this misclassification will be reduced as participants are only eligible if they live in close proximity to the monitors.

표-413. PubMed 논문번호 26928412의 내용 요약

구분	내용
PubMed ID	26928412
TITLE	A panel study of the acute effects of personal exposure to household air pollution on ambulatory blood pressure in rural Indian women.
JOURNAL	Environmental research: 10.1016/j.envres.2016.02.024
AUTHORS	Norris Christina, Goldberg Mark S, Marshall Julian D, Valois Marie-France, Pradeep T, Narayanswamy M, Jain Grishma, Sethuraman Karthik, Baumgartner Jill
BACKGROUND	Almost half the world's population is exposed to household air pollution from biomass and coal combustion. The acute effects of household air pollution on the cardiovascular system are poorly characterized. We conducted a panel study of rural Indian women to assess whether personal exposures to black carbon during cooking were associated with acute changes in blood pressure.
METHODS	We enrolled 45 women (ages 25-66 years) who cooked with biomass fuels. During cooking sessions in winter and summer, we simultaneously measured their personal real-time exposure to black carbon and conducted ambulatory blood pressure measurements every 10min. We recorded ambient temperature and participants' activities while cooking. We assessed body mass index, socioeconomic status, and salt intake. Multivariate mixed effects regression models with random intercepts were used to estimate the associations between blood pressure and black carbon exposure, e.g., average exposure in the minutes preceding blood pressure measurement, and average exposure over an entire cooking session.
RESULTS	Women's geometric mean (GM) exposure to black carbon during cooking sessions was lower in winter (GM: 40 μg/m(3); 95% CI: 30, 53) than in summer (GM: 56 μg/m(3); 95% CI: 42, 76). Interquartile range increases in black carbon were associated with changes in systolic blood pressure from -0.4mm Hg (95% CI: -2.3, 1.5) to 1.9mm Hg (95% CI: -0.8, 4.7), with associations increasing in magnitude as black carbon values were assessed over greater time periods preceding blood pressure measurement. Interquartile range increases in black carbon were associated with small decreases in diastolic blood pressure from -0.9mm Hg (95% CI: -1.7, -0.1) to -0.4mm Hg (95% CI: -1.6, 0.8). Associations of a similar magnitude were estimated for cooking session-averaged values.
CONCLUSIONS	We found some evidence of an association between exposure to black carbon and acute increases in systolic blood pressure in Indian women cooking with biomass fuels, which may have implications for the development of cardiovascular diseases.

표-414. PubMed 논문번호 26945620의 내용 요약

구분	내용
PubMed ID	26945620
TITLE	Exposure to air pollution and cognitive functioning across the life course - A systematic literature review.
JOURNAL	Environmental research: 10.1016/j.envres.2016.01.018
AUTHORS	Clifford Angela, Lang Linda, Chen Ruoling, Anstey Kaarin J, Seaton Anthony
OBJECTIVES	Air pollution is associated with increased risk of respiratory, cardiovascular and cerebrovascular disease, but its association with cognitive functioning and impairment is unclear. The aim of this systematic review was to examine whether a relationship exists between these variables across the life course.
METHODS	We searched Web of Knowledge, Pubmed, SciVerse Scopus, CINAHL, PsychInfo and Science Direct up to October 2015 to identify studies that investigated the association between air pollution and performance on neurocognitive tests.
RESULTS	Variations in exposure assessment and outcome measures make meta-analysis impossible. Thirty one studies published between 2006 and 2015, from the Americas (n=15), Asia (n=5) and Europe (n=11), met the criteria for inclusion. Many showed weak but quantified relationships between various air pollutants and cognitive function. Pollution exposure in utero has been associated with increased risk of neuro-developmental delay. Exposure in childhood has been inversely associated with neuro-developmental outcomes in younger children and with academic achievement and neurocognitive performance in older children. In older adults, air pollution has been associated with accelerated cognitive decline.
CONCLUSIONS	The evidence to date is coherent in that exposure to a range of largely traffic-related pollutants has been associated with quantifiable impairment of brain development in the young and cognitive decline in the elderly. There is insufficient evidence at present to comment on consistency, in view of the different indices of pollution and end-points measured, the limited number of studies, and the probability at this stage of publication bias. However, plausible toxicological mechanisms have been demonstrated and the evidence as a whole suggests that vehicular pollution, at least, contributes to cognitive impairment, adding to pressure on governments and individuals to continue to reduce air pollution.

표-415. PubMed 논문번호 26949871의 내용 요약

구분	내용
PubMed ID	26949871
TITLE	Long-Term Metal PM2.5 Exposures Decrease Cardiac Acceleration and Deceleration Capacities in Welders.
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0000000000000661
AUTHORS	Umukoro Peter E, Fan Tianteng, Zhang Jinming, Cavallari Jennifer M, Fang Shona C, Lu Chensheng, Lin Xihong, Mittleman Murray A, Schmidt Georg, Christiani David C
OBJECTIVE	The aim of the study was to clarify whether long-term metal particulates affect cardiac acceleration capacity (AC), deceleration capacity (DC), or both.
METHODS	We calculated chronic exposure index (CEI) for PM2.5 over the work life of 50 boilermakers and obtained their resting AC and DC. Linear regression was used to assess the associations between CEI PM2.5 exposure and each of AC and DC, controlling for age, acute effects of welding exposure, and diurnal variation.
RESULTS	Mean (standard deviation) CEI for PM2.5 exposure was 1.6 (2.4)mg/m-work years and ranged from 0.001 to 14.6mg/m-work years. In our fully adjusted models, a 1mg/m-work year increase in CEI for PM2.5 was associated with a decrease of 1.03 (95% confidence interval: 0.10, 1.96)ms resting AC, and a decrease of 0.67 (95% confidence interval: -0.14, 1.49)ms resting DC.
CONCLUSIONS	Long-term metal particulate exposures decrease cardiac accelerations and decelerations.

표-416. PubMed 논문번호 26949872의 내용 요약

구분	내용
PubMed ID	26949872
TITLE	Are the Associations of Cardiac Acceleration and Deceleration Capacities With Fine Metal Particulate in Welders Mediated by Inflammation?
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0000000000000674
AUTHORS	Umukoro Peter E, Wong Jason Y Y, Cavallari Jennifer M, Fang Shona C, Lu Chensheng, Lin Xihong, Mittleman Murray A, Schmidt Georg, Christiani David C
OBJECTIVE	The aim of this study was to investigate whether associations of acceleration capacity (AC) and deceleration capacity (DC) with metal-PM2.5 are mediated by inflammation.
METHODS	We obtained PM2.5, C-reactive protein, interleukin (IL)-6, 8, and 10, and electrocardiograms to compute AC and DC, from 45 male welders. Mediation analyses were performed using linear mixed models to assess associations between PM2.5 exposure, inflammatory mediator, and AC or DC, controlling for covariates.
RESULTS	The proportion of total effect of PM2.5 on AC or DC (indirect effect) mediated through IL-6 on AC was 4% at most. Controlling for IL-6 (direct effect), a 1mg/m increase of PM2.5 was associated with a decrease of 2.16 (95% confidence interval -0.36 to 4.69) msec in AC and a decrease of 2.51 (95% confidence interval -0.90 to 5.93) msec in DC.
CONCLUSION	IL-6 may be mediating the effect of metal particulates on AC.

표-417. PubMed 논문번호 26955062의 내용 요약

구분	내용
PubMed ID	26955062
TITLE	Long-Term Exposure to Ambient Fine Particulate Matter and Renal Function in Older Men: The Veterans Administration Normative Aging Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1510269
AUTHORS	Mehta Amar J, Zanobetti Antonella, Bind Marie-Abele C, Kloog Itai, Koutrakis Petros, Sparrow David, Vokonas Pantel S, Schwartz Joel D
BACKGROUND	It is unknown if ambient fine particulate matter (PM2.5) is associated with lower renal function, a cardiovascular risk factor.
OBJECTIVE	We investigated whether long-term PM2.5 exposure was associated with estimated glomerular filtration rate (eGFR) in a cohort of older men living in the Boston Metropolitan area.
METHODS	This longitudinal analysis included 669 participants from the Veterans Administration Normative Aging Study with up to four visits between 2000 and 2011 (n = 1,715 visits). Serum creatinine was measured at each visit, and eGFR was calculated according to the Chronic Kidney Disease Epidemiology Collaboration equation. One-year exposure to PM2.5 prior to each visit was assessed using a validated spatiotemporal model that utilized satellite remote-sensing aerosol optical depth data. eGFR was modeled in a time-varying linear mixed-effects regression model as a continuous function of 1-year PM2.5, adjusting for important covariates.
RESULTS	One-year PM2.5 exposure was associated with lower eGFRs; a 2.1-μg/m3 interquartile range higher 1-year PM2.5 was associated with a 1.87 mL/min/1.73 m2 lower eGFR [95% confidence interval (CI): -2.99, -0.76]. A 2.1 μg/m3-higher 1-year PM2.5 was also associated with an additional annual decrease in eGFR of 0.60 mL/min/1.73 m2 per year (95% CI: -0.79, -0.40).
CONCLUSIONS	In this longitudinal sample of older men, the findings supported the hypothesis that long-term PM2.5 exposure negatively affects renal function and increases renal function decline.
CITATION	Mehta AJ, Zanobetti A, Bind MC, Kloog I, Koutrakis P, Sparrow D, Vokonas PS, Schwartz JD. 2016. Long-term exposure to ambient fine particulate matter and renal function in older men: the VA Normative Aging Study. Environ Health Perspect 124:1353-1360; http://dx.doi.org/10.1289/ehp.1510269.

표-418. PubMed 논문번호 26956024의 내용 요약

구분	내용
PubMed ID	26956024
TITLE	Ambient particulate matter and microRNAs in extracellular vesicles: a pilot study of older individuals.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-016-0121-0
AUTHORS	Rodosthenous Rodosthenis S, Coull Brent A, Lu Quan, Vokonas Pantel S, Schwartz Joel D, Baccarelli Andrea A
BACKGROUND	Air pollution from particulate matter (PM) has been linked to cardiovascular morbidity and mortality; however the underlying biological mechanisms remain to be uncovered. Gene regulation by microRNAs (miRNAs) that are transferred between cells by extracellular vesicles (EVs) may play an important role in PM-induced cardiovascular risk. This study sought to determine if ambient PM2.5 levels are associated with expression of EV-encapsulated miRNAs (evmiRNAs), and to investigate the participation of such evmiRNAs in pathways related to cardiovascular disease (CVD).
METHODS	We estimated the short- (1-day), intermediate- (1-week and 1-month) and long-term (3-month, 6-month, and 1-year) moving averages of ambient PM2.5 levels at participants' addresses using a validated hybrid spatio-temporal land-use regression model. We collected 42 serum samples from 22 randomly selected participants in the Normative Aging Study cohort and screened for 800 miRNAs using the NanoString nCounter® platform. Mixed effects regression models, adjusted for potential confounders were used to assess the association between ambient PM2.5 levels and evmiRNAs. All p-values were adjusted for multiple comparisons. In-silico Ingenuity Pathway Analysis (IPA) was performed to identify biological pathways that are regulated by PM-associated evmiRNAs.
RESULTS	We found a significant association between long-term ambient PM2.5 exposures and levels of multiple evmiRNAs circulating in serum. In the 6-month window, ambient PM2.5 exposures were associated with increased levels of miR-126-3p (0.74 ± 0.21; p = 0.02), miR-19b-3p (0.52 ± 0.15; p = 0.02), miR-93-5p (0.78 ± 0.22; p = 0.02), miR-223-3p (0.74 ± 0.22; p = 0.02), and miR-142-3p (0.81 ± 0.21; p = 0.03). Similarly, in the 1-year window, ambient PM2.5 levels were associated with increased levels of miR-23a-3p (0.83 ± 0.23; p = 0.02), miR-150-5p (0.90 ± 0.24; p = 0.02), miR-15a-5p (0.70 ± 0.21; p = 0.02), miR-191-5p (1.20 ± 0.35; p = 0.02), and let-7a-5p (1.42 ± 0.39; p = 0.02). In silico pathway analysis on PM2.5-associated evmiRNAs identified several key CVD-related pathways including oxidative stress, inflammation, and atherosclerosis.
CONCLUSIONS	We found an association between long-term ambient PM2.5 levels and increased levels of evmiRNAs circulating in serum. Further observational studies are warranted to confirm and extend these important findings in larger and more diverse populations, and experimental studies are needed to elucidate the exact roles of evmiRNAs in PM-induced CVD.

표-419. PubMed 논문번호 26960925의 내용 요약

구분	내용
PubMed ID	26960925
TITLE	Public health impacts of city policies to reduce climate change: findings from the URGENCHE EU-China project.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-016-0097-0
AUTHORS	Sabel Clive E, Hiscock Rosemary, Asikainen Arja, Bi Jun, Depledge Mike, van den Elshout Sef, Friedrich Rainer, Huang Ganlin, Hurley Fintan, Jantunen Matti, Karakitsios Spyros P, Keuken Menno, Kingham Simon, Kontoroupis Periklis, Kuenzli Nino, Liu Miaomiao, Martuzzi Marco, Morton Katie, Mudu Pierpaolo, Niittynen Marjo, Perez Laura, Sarigiannis Denis, Stahl-Timmins Will, Tobollik Myriam, Tuomisto Jouni, Willers Saskia
BACKGROUND	Climate change is a global threat to health and wellbeing. Here we provide findings of an international research project investigating the health and wellbeing impacts of policies to reduce greenhouse gas emissions in urban environments.
METHODS	Five European and two Chinese city authorities and partner academic organisations formed the project consortium. The methodology involved modelling the impact of adopted urban climate-change mitigation transport, buildings and energy policy scenarios, usually for the year 2020 and comparing them with business as usual (BAU) scenarios (where policies had not been adopted). Carbon dioxide emissions, health impacting exposures (air pollution, noise and physical activity), health (cardiovascular, respiratory, cancer and leukaemia) and wellbeing (including noise related wellbeing, overall wellbeing, economic wellbeing and inequalities) were modelled. The scenarios were developed from corresponding known levels in 2010 and pre-existing exposure response functions. Additionally there were literature reviews, three longitudinal observational studies and two cross sectional surveys.
RESULTS	There are four key findings. Firstly introduction of electric cars may confer some small health benefits but it would be unwise for a city to invest in electric vehicles unless their power generation fuel mix generates fewer emissions than petrol and diesel. Second, adopting policies to reduce private car use may have benefits for carbon dioxide reduction and positive health impacts through reduced noise and increased physical activity. Third, the benefits of carbon dioxide reduction from increasing housing efficiency are likely to be minor and co-benefits for health and wellbeing are dependent on good air exchange. Fourthly, although heating dwellings by in-home biomass burning may reduce carbon dioxide emissions, consequences for health and wellbeing were negative with the technology in use in the cities studied.
CONCLUSIONS	The climate-change reduction policies reduced CO2 emissions (the most common greenhouse gas) from cities but impact on global emissions of CO2 would be more limited due to some displacement of emissions. The health and wellbeing impacts varied and were often limited reflecting existing relatively high quality of life and environmental standards in most of the participating cities; the greatest potential for future health benefit occurs in less developed or developing countries.

표-420. PubMed 논문번호 26967543의 내용 요약

구분	내용
PubMed ID	26967543
TITLE	Quantile Regression Analysis of the Distributional Effects of Air Pollution on Blood Pressure, Heart Rate Variability, Blood Lipids, and Biomarkers of Inflammation in Elderly American Men: The Normative Aging Study.
JOURNAL	Environmental health perspectives: 10.1289/ehp.1510044
AUTHORS	Bind Marie-Abele, Peters Annette, Koutrakis Petros, Coull Brent, Vokonas Pantel, Schwartz Joel
BACKGROUND	Previous studies have observed associations between air pollution and heart disease. Susceptibility to air pollution effects has been examined mostly with a test of effect modification, but little evidence is available whether air pollution distorts cardiovascular risk factor distribution.
OBJECTIVES	This paper aims to examine distributional and heterogeneous effects of air pollution on known cardiovascular biomarkers.
METHODS	A total of 1,112 men from the Normative Aging Study and residents of the greater Boston, Massachusetts, area with mean age of 69 years at baseline were included in this study during the period 1995-2013. We used quantile regression and random slope models to investigate distributional effects and heterogeneity in the traffic-related responses on blood pressure, heart rate variability, repolarization, lipids, and inflammation. We considered 28-day averaged exposure to particle number, PM2.5 black carbon, and PM2.5 mass concentrations (measured at a single monitor near the site of the study visits).
RESULTS	We observed some evidence suggesting distributional effects of traffic-related pollutants on systolic blood pressure, heart rate variability, corrected QT interval, low density lipoprotein (LDL) cholesterol, triglyceride, and intercellular adhesion molecule-1 (ICAM-1). For example, among participants with LDL cholesterol below 80 mg/dL, an interquartile range increase in PM2.5 black carbon exposure was associated with a 7-mg/dL (95% CI: 5, 10) increase in LDL cholesterol, while among subjects with LDL cholesterol levels close to 160 mg/dL, the same exposure was related to a 16-mg/dL (95% CI: 13, 20) increase in LDL cholesterol. We observed similar heterogeneous associations across low versus high percentiles of the LDL distribution for PM2.5 mass and particle number.
CONCLUSIONS	These results suggest that air pollution distorts the distribution of cardiovascular risk factors, and that, for several outcomes, effects may be greatest among individuals who are already at high risk.
CITATION	Bind MA, Peters A, Koutrakis P, Coull B, Vokonas P, Schwartz J. 2016. Quantile regression analysis of the distributional effects of air pollution on blood pressure, heart rate variability, blood lipids, and biomarkers of inflammation in elderly American men: the Normative Aging Study. Environ Health Perspect 124:1189-1198; http://dx.doi.org/10.1289/ehp.1510044.

표-421. PubMed 논문번호 26969808의 내용 요약

구분	내용
PubMed ID	26969808
TITLE	Air pollution exposure, cause-specific deaths and hospitalizations in a highly polluted Italian region.
JOURNAL	Environmental research: 10.1016/j.envres.2016.03.003
AUTHORS	Carugno Michele, Consonni Dario, Randi Giorgia, Catelan Dolores, Grisotto Laura, Bertazzi Pier Alberto, Biggeri Annibale, Baccini Michela
BACKGROUND	The Lombardy region in northern Italy ranks among the most air polluted areas of Europe. Previous studies showed air pollution short-term effects on all-cause mortality. We examine here the effects of particulate matter with aerodynamic diameter ≤10□m (PM10) and nitrogen dioxide (NO2) exposure on deaths and hospitalizations from specific causes, including cardiac, cerebrovascular and respiratory diseases.
METHODS	We considered air pollution, mortality and hospitalization data for a non-opportunistic sample of 18 highly polluted and most densely populated areas of the region in the years 2003-2006. We obtained area-specific effect estimates for PM10 and NO2 from a Poisson regression model on the daily number of total deaths or cause-specific hospitalizations and then combined them in a Bayesian random-effects meta-analysis. For cause-specific mortality, we applied a case-crossover analysis. Age- and season-specific analyses were also performed. Effect estimates were expressed as percent variation in mortality or hospitalizations associated with a 10□g/m(3) increase in PM10 or NO2 concentration.
RESULTS	Natural mortality was positively associated with both pollutants (0.30%, 90% Credibility Interval [CrI]: -0.31; 0.78 for PM10; 0.70%, 90%CrI: 0.10; 1.27 for NO2). Cardiovascular deaths showed a higher percent variation in association with NO2 (1.12%, 90% Confidence Interval [CI]: 0.14; 2.11), while the percent variation for respiratory mortality was highest in association with PM10 (1.64%, 90%CI: 0.35; 2.93). The effect of both pollutants was more evident in the summer season. Air pollution was also associated to hospitalizations, the highest variations being 0.77% (90%CrI: 0.22; 1.43) for PM10 and respiratory diseases, and 1.70% (90%CrI: 0.39; 2.84) for NO2 and cerebrovascular diseases. The effect of PM10 on respiratory hospital admissions appeared to increase with age. For both pollutants, effects on cerebrovascular hospitalizations were more evident in subjects aged less than 75 years.
CONCLUSIONS	Our study provided a sound characterization of air pollution exposure and its potential effects on human health in the most polluted, and also most populated and productive, Italian region, further documenting the need for effective public health policies.

표-422. PubMed 논문번호 26986952의 내용 요약

구분	내용
PubMed ID	26986952
TITLE	Morning NO2 exposure sensitizes hypertensive rats to the cardiovascular effects of same day O3 exposure in the afternoon.
JOURNAL	Inhalation toxicology: 10.3109/08958378.2016.1148088
AUTHORS	Farraj Aimen K, Malik Fatiha, Haykal-Coates Najwa, Walsh Leon, Winsett Darrell, Terrell Dock, Thompson Leslie C, Cascio Wayne E, Hazari Mehdi S
CONTEXT	Within urban air sheds, specific ambient air pollutants typically peak at predictable times throughout the day. For example, in environments dominated by mobile sources, peak nitrogen dioxide (NO2) levels coincide with morning and afternoon rush hours, while peak levels of ozone (O3), occur in the afternoon.
OBJECTIVE	Given that exposure to a single pollutant might sensitize the cardiopulmonary system to the effects of a subsequent exposure to a second pollutant, we hypothesized that a morning exposure to NO2 will exaggerate the cardiovascular effects of an afternoon O3 exposure in rats.
MATERIALS AND METHODS	Rats were divided into four groups that were each exposed for 3 h in the morning (m) and 3 h in the afternoon (a) on the same day: (1) m-Air/a-Air, (2) m-Air/a-O3 (0.3 ppm), (3) m-NO2 (0.5 ppm)/a-Air and (4) m-NO2/a-O3. Implanted telemetry devices recorded blood pressure and electrocardiographic data. Sensitivity to the arrhythmogenic agent aconitine was measured in a separate cohort.
RESULTS	Only m-NO2/a-O3-exposed rats had significant changes in electrophysiological, mechanical and autonomic parameters. These included decreased heart rate and increased PR and QTc intervals and increased heart rate variability, suggesting increased parasympathetic tone. In addition, only m-NO2/a-O3 exposure decreased systolic and diastolic blood pressures and increased pulse pressure and QA interval, suggesting decreased cardiac contractility.
DISCUSSION AND CONCLUSION	The findings indicate that initial exposure to NO2 sensitized rats to the cardiovascular effects of O3 and may provide insight into the epidemiological data linking adverse cardiovascular outcomes with exposures to low concentrations of O3.

표-423. PubMed 논문번호 26994463의 내용 요약

구분	내용
PubMed ID	26994463
TITLE	The influence of pre-existing health conditions on short-term mortality risks of temperature: Evidence from a prospective Chinese elderly cohort in Hong Kong.
JOURNAL	Environmental research: 10.1016/j.envres.2016.03.012
AUTHORS	Sun Shengzhi, Tian Linwei, Qiu Hong, Chan King-Pan, Tsang Hilda, Tang Robert, Lee Ruby Siu-Yin, Thach Thuan-Quoc, Wong Chit-Ming
BACKGROUND	Both cold and hot temperatures are associated with adverse health outcomes. Less is known about the role of pre-existing medical conditions to confer individual's susceptibility to temperature extremes.
METHODS	We studied 66,820 subjects aged ≥65 who were enrolled and interviewed in all the 18 Elderly Health Centers of Department of Health, Hong Kong from 1998 to 2001, and followed up for 10-13 years. The distributed lag nonlinear model (DLNM) combined with a nested case-control study design was applied to estimate the nonlinear and delayed effects of cold or hot temperature on all natural mortality among subjects with different pre-existing diseases.
RESULTS	The relative risk of all natural mortality associated with a decrease of temperature from 25th percentile (19.5° C) to 1st percentile (11.3° C) over 0-21 lag days for participants who reported to have an active disease at the baseline was 2.21 (95% confidence interval (CI): 1.19, 4.10) for diabetes mellitus (DM), 1.59 (1.12, 2.26) for circulatory system diseases (CSD), and 1.23 (0.53, 2.84) for chronic obstructive pulmonary disease (COPD), whereas 1.04 (0.59, 1.85) for non-disease group (NDG). Compared with NDG, elders with COPD had excess risk of mortality associated with thermal stress attributable to hot temperature, while elders with DM and CSD were vulnerable to both hot and cold temperatures.
CONCLUSIONS	Elders with pre-existing health conditions were more vulnerable to excess mortality risk to hot and/or cold temperature. Preventative measures should target on elders with chronic health problems.

표-424. PubMed 논문번호 27012471의 내용 요약

구분	내용
PubMed ID	27012471
TITLE	Analysis of environmental risk factors for pulmonary embolism: A case-crossover study (2001-2013).
JOURNAL	European journal of internal medicine: 10.1016/j.ejim.2016.03.001
AUTHORS	de Miguel-D□ez Javier, Jim□nez-Garc□a Rodrigo, L□pez de Andr□s Ana, Hern□ndez-Barrera Valent□n, Carrasco-Garrido Pilar, Monreal Manuel, Jim□nez David, Jara-Palomares Luis, □lvaro-Meca Alejandro
BACKGROUND	The relationship between environmental factors and pulmonary embolism (PE) has received little attention. The aim of this study was to estimate the influence of climatological factors and air pollution levels on PE in Spain from 2001 to 2013.
METHODS	We carried out a retrospective study. Data were collected from the Minimum Basic Data Set (MBDS) and the State Meteorological Agency (AEMET) of Spain. A case-crossover analysis was applied to identify environmental risk factors related to hospitalizations and deaths. For each patient, climatic and pollutant factors were assigned using data from the meteorological station closest to his/her postal code.
RESULTS	A seasonal effect for PE hospital admission was observed, with more frequent admissions noted during Spain's colder seasons with peaks in autumn and winter. Lower temperatures as well as higher concentrations of NO2 and O3 at the time of admission (when 2weeks and 3weeks respectively were used as controls) were significant risk factors for hospital admissions with PE.
CONCLUSIONS	Pulmonary embolism epidemiology was adversely influenced by colder climatological factors (absolute temperature, and seasonality) and higher concentrations of ambient air pollution (NO2, O3).

표-425. PubMed 논문번호 27035690의 내용 요약

구분	내용
PubMed ID	27035690
TITLE	Long-term Coarse Particulate Matter Exposure and Heart Rate Variability in the Multi-ethnic Study of Atherosclerosis.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000455
AUTHORS	Adhikari Richa, D'Souza Jennifer, Soliman Elsayed Z, Burke Gregory L, Daviglus Martha L, Jacobs David R, Park Sung Kyun, Sheppard Lianne, Thorne Peter S, Kaufman Joel D, Larson Timothy V, Adar Sara Dubowsky
BACKGROUND	Reduced heart rate variability, a marker of impaired cardiac autonomic function, has been linked to short-term exposure to airborne particles. This research adds to the literature by examining associations with long-term exposures to coarse particles (PM10-2.5).
METHODS	Using electrocardiogram recordings from 2,780 participants (45-84 years) from three Multi-ethnic Study of Atherosclerosis sites, we assessed the standard deviation of normal to normal intervals and root-mean square differences of successive normal to normal intervals at a baseline (2000-2002) and follow-up (2010-2012) examination (mean visits/person = 1.5). Annual average concentrations of PM10-2.5 mass, copper, zinc, phosphorus, silicon, and endotoxin were estimated using site-specific spatial prediction models. We assessed associations for baseline heart rate variability and rate of change in heart rate variability over time using multivariable mixed models adjusted for time, sociodemographic, lifestyle, health, and neighborhood confounders, including copollutants.
RESULTS	In our primary models adjusted for demographic and lifestyle factors and site, PM10-2.5 mass was associated with 1.0% (95% confidence interval [CI]: -4.1, 2.1%) lower standard deviation of normal to normal interval levels per interquartile range of 2 μg/m. Stronger associations, however, were observed before site adjustment and with increasing residential stability. Similar patterns were found for root-mean square differences of successive normal to normal intervals. We found little evidence for associations with other chemical species and with the rate of change in heart rate variability, though endotoxin was associated with increasing heart rate variability over time.
CONCLUSION	We found only weak evidence that long-term PM10-2.5 exposures are associated with lowered heart rate variability. Stronger associations among residentially stable individuals suggest that confirmatory studies are needed.

표-426. PubMed 논문번호 27048714의 내용 요약

구분	내용
PubMed ID	27048714
TITLE	Predictors of blood pressure at 7-13 years: The "new millennium baby" study.
JOURNAL	Nutrition, metabolism, and cardiovascular diseases : NMCD: 10.1016/j.numecd.2015.11.005
AUTHORS	Brambilla P, Bedogni G, Pietrobelli A, Cianfarani S, Agostoni C, Brambilla P, Bedogni G, Pietrobelli A, Cianfarani S, Agostoni C
BACKGROUND AND AIMS	The purpose of this study is to evaluate the association between blood pressure (BP) at 7-13 years of age and body mass index (BMI), early feeding, lifestyle indicators, and parental characteristics.
METHODS AND RESULTS	Retrospective plus cross-sectional cohort study was started in 1294 children born in 2000-2004, right from their birth in primary care settings. Early feeding was estimated by measuring breast-feeding (BF) duration, complementary feeding (CF) introduction time, and lifestyle indicators such as daily screen time and weekly extracurricular sports activity time. Parental education, smoking, and obesity-related diseases were also considered. Multivariable linear regression and mediation analysis were used. CF introduction at 5-6 months of age was a negative predictor of systolic and diastolic BP (mean systolic BP-standard deviation score (SDS) -0.38 [95% CI: -0.47, -0.29] ($p < 0.001$); mean diastolic BP-SDS -0.32 [95% CI: -0.40, -0.24]) ($p < 0.001$); BMI was a positive predictor of systolic and diastolic BP ($p < 0.001$); and parental hypertension was a positive predictor of diastolic BP ($p < 0.05$). Predictors of mean BMI-SDS at 7-13 years of age were birth weight, screen time, and parental obesity and smoking ($p < 0.001$). BF had no effect on BP or BMI. Mediation analysis showed virtually no indication of the effect of CF on BP mediated by BMI.
CONCLUSIONS	CF introduction between 5 and 6 months of age could be associated with low BP at 7-13 years. The effect of CF on BP seems to be independent of BMI. Low screen time is associated with low BMI. CF time may play a role in the occurrence of surrogates of noncommunicable disorders in future.

표-427. PubMed 논문번호 27052768의 내용 요약

구분	내용
PubMed ID	27052768
TITLE	Environmental and occupational particulate matter exposures and ectopic heart beats in welders.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2015-103256
AUTHORS	Cavallari Jennifer M, Fang Shona C, Eisen Ellen A, Mittleman Murray A, Christiani David C
OBJECTIVES	Links between arrhythmias and particulate matter exposures have been found among sensitive populations. We examined the relationship between personal particulate matter ≤2.5 □m aerodynamic diameter (PM2.5) exposures and ectopy in a panel study of healthy welders.
METHODS	Simultaneous ambulatory ECG and personal PM2.5 exposure monitoring with DustTrak Aerosol Monitor was performed on 72 males during work and non-work periods for 5-90 h (median 40 h). ECGs were summarised hourly for supraventricular ectopy (SVE) and ventricular ectopy (VE). PM2.5 exposures both work and non-work periods were averaged hourly with lags from 0 to 7 h. Generalised linear mixed-effects models with a random participant intercept were used to examine the relationship between PM2.5 exposure and the odds of SVE or VE. Sensitivity analyses were performed to assess whether relationships differed by work period and among current smokers.
RESULTS	Participants had a mean (SD) age of 38 (11) years and were monitored over 2993 person-hours. The number of hourly ectopic events was highly skewed with mean (SD) of 14 (69) VE and 1 (4) SVE. We found marginally significant increases in VE with PM2.5 exposures in the sixth and seventh hour lags, yet no association with SVE. For every 100 μg/m(3) increase in sixth hour lagged PM2.5, the adjusted OR (95% CI) for VE was 1.03 (1.00 to 1.05). Results persisted in work or non-work exposure periods and non-smokers had increased odds of VE associated with PM2.5 as compared with smokers.
CONCLUSIONS	A small increase in the odds of VE with short-term PM2.5 exposure was observed among relatively healthy men with environmental and occupational exposures.

표-428. PubMed 논문번호 27083214의 내용 요약

구분	내용
PubMed ID	27083214
TITLE	Systematic Review and Meta-analysis of the Association Between Exposure to Environmental Tobacco Smoke and Periodontitis Endpoints Among Nonsmokers.
JOURNAL	Nicotine & tobacco research : official journal of the Society for Research on Nicotine and Tobacco: 10.1093/ntr/ntw105
AUTHORS	Akinkugbe Aderonke A, Slade Gary D, Divaris Kimon, Poole Charles
OBJECTIVE	A systematic review was conducted to summarize the epidemiological evidence on environmental tobacco smoke (ETS) exposure and prevalent periodontitis endpoints among nonsmokers.
METHODS	We searched PubMed, EMBASE, Web of Science, Pro-Quest dissertations, and conference proceedings of a dental research association. We included studies from which prevalence odds ratios (POR) could be extracted for periodontitis determined by examiner measurements of clinical attachment level (CAL) and/or probing pocket depth (PD) or self-report of missing teeth. Studies determined ETS exposure by self-report or biomarker (cotinine) levels.
RESULTS	For studies reporting CAL and/or PD (n = 6), associations were stronger with cotinine-measured exposure (n = 3; random effects POR [95% prediction interval] = 1.63 (0.90, 2.96)) than self-reported exposure (n = 3; random effects POR = 1.15 (0.68, 1.96)). There was no meaningful difference in summary estimate for studies reporting CAL and/or PD endpoint (n = 6; random effects POR = 1.34 (0.93, 1.94)) as opposed to tooth loss (n = 2; random effects POR = 1.33 (0.52, 3.40)).
CONCLUSIONS	There appears to be a positive association between exposure to ETS and prevalent periodontitis endpoints among nonsmokers, the magnitude of which depended mostly on the method of ETS assessment.
IMPLICATIONS	The notoriety of ETS is often discussed in terms of its associations with cancer, chronic conditions like cardiovascular diseases, and respiratory illnesses in children. However, very little attention is paid to its association with oral diseases, especially periodontitis. Periodontitis affects a large proportion of the population and is a major cause of tooth loss. This study summarized the epidemiologic association between exposure to ETS and periodontitis among nonsmokers. Although the findings are consistent with a positive association, methodological weaknesses relating to study design, assessment of ETS, periodontitis, and adjustment covariates were highlighted and recommendations for improvement in future studies provided.

표-429. PubMed 논문번호 27091488의 내용 요약

구분	내용
PubMed ID	27091488
TITLE	Cooking Coal Use and All-Cause and Cause-Specific Mortality in a Prospective Cohort Study of Women in Shanghai, China.
JOURNAL	Environmental health perspectives: 10.1289/EHP236
AUTHORS	Kim Christopher, Seow Wei Jie, Shu Xiao-Ou, Bassig Bryan A, Rothman Nathaniel, Chen Bingshu E, Xiang Yong-Bing, Hosgood H Dean, Ji Bu-Tian, Hu Wei, Wen Cuiju, Chow Wong-Ho, Cai Qiuyin, Yang Gong, Gao Yu-Tang, Zheng Wei, Lan Qing
BACKGROUND	Nearly 4.3 million deaths worldwide were attributable to exposure to household air pollution in 2012. However, household coal use remains widespread.
OBJECTIVES	We investigated the association of cooking coal and all-cause and cause-specific mortality in a prospective cohort of primarily never-smoking women in Shanghai, China.
METHODS	A cohort of 74,941 women were followed from 1996 through 2009 with annual linkage to the Shanghai vital statistics database. Cause-specific mortality was identified through 2009. Use of household coal for cooking was assessed through a residential history questionnaire. Cox proportional hazards models estimated the risk of mortality associated with household coal use.
RESULTS	In this cohort, 63% of the women ever used coal (n = 46,287). Compared with never coal use, ever use of coal was associated with mortality from all causes [hazard ratio (HR) = 1.12; 95% confidence interval (CI): 1.05, 1.21], cancer (HR = 1.14; 95% CI: 1.03, 1.27), and ischemic heart disease (overall HR = 1.61; 95% CI: 1.14, 2.27; HR for myocardial infarction specifically = 1.80; 95% CI: 1.16, 2.79). The risk of cardiovascular mortality increased with increasing duration of coal use, compared with the risk in never users. The association between coal use and ischemic heart disease mortality diminished with increasing years since cessation of coal use.
CONCLUSIONS	Evidence from this study suggests that past use of coal among women in Shanghai is associated with excess all-cause mortality, and from cardiovascular diseases in particular. The decreasing association with cardiovascular mortality as the time since last use of coal increased emphasizes the importance of reducing use of household coal where use is still widespread.
CITATION	Kim C, Seow WJ, Shu XO, Bassig BA, Rothman N, Chen BE, Xiang YB, Hosgood HD III, Ji BT, Hu W, Wen C, Chow WH, Cai Q, Yang G, Gao YT, Zheng W, Lan Q. 2016. Cooking coal use and all-cause and cause-specific mortality in a prospective cohort study of women in Shanghai, China. Environ Health Perspect 124:1384-1389; http://dx.doi.org/10.1289/EHP236.

표-430. PubMed 논문번호 27107129의 내용 요약

구분	내용
PubMed ID	27107129
TITLE	Effects of Air Pollution and Blood Mitochondrial DNA Methylation on Markers of Heart Rate Variability.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.116.003218
AUTHORS	Byun Hyang-Min, Colicino Elena, Trevisi Letizia, Fan Tianteng, Christiani David C, Baccarelli Andrea A
BACKGROUND	The mitochondrion is the primary target of oxidative stress in response to exogenous environments. Mitochondrial DNA (mtDNA) is independent from nuclear DNA and uses separate epigenetic machinery to regulate mtDNA methylation. The mtDNA damage induced by oxidative stress can cause mitochondrial dysfunction and is implicated in human diseases; however, mtDNA methylation has been largely overlooked in environmental studies relating to human disease. The purpose of this study was to examine the association between exposure to fine metal-rich particulates (particulate matter <2.5 □m in diameter [PM2.5]) from welding in a boilermaker union and blood mtDNA methylation in relation to heart rate variability.
METHODS AND RESULTS	Forty-eight healthy men were recruited on multiple sampling cycles at the Boilermaker Union Local 29, located in Quincy, Massachusetts. We measured personal PM2.5 in the background ambient environment. We measured blood mtDNA methylation in the mtDNA promoter (D-loop) and genes essential for ATP synthesis (MT-TF and MT-RNR1) by bisulfite pyrosequencing. All analyses were adjusted for demographics, type of job, season, welding-work day, and mtDNA methylation experimental batch effect. The participants' PM2.5 exposure was significantly higher after a welding-work day (mean 0.38 mg/m(3)) than the background personal level (mean 0.15 mg/m(3), P<0.001). Blood mtDNA methylation in the D-loop promoter was associated with PM2.5 levels (β=-0.99%, SE=0.41, P=0.02). MT-TF and MT-RNR1 methylation was not associated with PM2.5 exposure (β=0.10%, SE=0.45, P=0.82). Interaction of PM2.5 exposure levels and D-loop promoter methylation was significantly associated with markers of heart rate variability.
CONCLUSIONS	Blood mtDNA methylation levels were negatively associated with PM2.5 exposure and modified the adverse relationships between PM2.5 exposure and heart rate variability outcomes.

표-431. PubMed 논문번호 27107547의 내용 요약

구분	내용
PubMed ID	27107547
TITLE	Effects of reducing exposure to air pollution on submaximal cardiopulmonary test in patients with heart failure: Analysis of the randomized, double-blind and controlled FILTER-HF trial.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2016.04.071
AUTHORS	Vieira Jefferson L, Guimaraes Guilherme V, de Andre Paulo A, Saldiva Paulo H Nascimento, Bocchi Edimar A
BACKGROUND	Air pollution exposure could mitigate the health benefits of exercise in patients with heart failure (HF). We tested the effects of a respiratory filter on HF patients exposed to air pollution during exercise.
METHODS AND RESULTS	Ancillary analysis of the FILTER-HF trial, focused on the exercise outcomes. In a randomized, double-blind, 3-way crossover design, 26 HF patients and 15 control volunteers were exposed to clean air, unfiltered dilute diesel engine exhaust (DE), or filtered DE for 6min during a submaximal cardiopulmonary testing in a controlled-exposure facility. Prospectively collected data included six-minute walking test [6mwt], VO2, VE/VCO2 Slope, O2Pulse, pulmonary ventilation [VE], tidal volume, VD/Vt, oxyhemoglobin saturation and CO2-rebreathing. Compared to clean air, DE adversely affected VO2 (11.0±3.9 vs. 8.4±2.8ml/kg/min; p<0.001); 6mwt (243.3±13.0 vs. 220.8±13.7m; p=0.030); and O2Pulse (8.9±1.0 vs. 7.8±0.7ml/beat; p<0.001) in HF patients. Compared to DE, filtration reduced the particulate concentration from 325±31 to 25±6 μg/m(3), and was associated with an increase in VO2 (10.4±3.8ml/kg/min; p<0.001 vs. DE) and O2Pulse (9.7±1.1ml/beat; p<0.001 vs. DE) in patients with HF. Filtration was associated with higher VE and CO2-rebreathing in both groups. VE/VCO2 Slope was higher among patients with HF.
CONCLUSION	DE adversely affects exercise capacity in patients with HF. A simple respiratory filter can reduce the adverse effects of pollution on VO2 and O2Pulse. Given the worldwide prevalence of exposure to traffic-related air pollution, these findings are relevant for public health especially in this highly susceptible population. The filter intervention holds great promise that needs to be tested in future studies.

표-432. PubMed 논문번호 27126478의 내용 요약

구분	내용
PubMed ID	27126478
TITLE	Short-Term Exposure to Air Pollution and Biomarkers of Oxidative Stress: The Framingham Heart Study.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.115.002742
AUTHORS	Li Wenyuan, Wilker Elissa H, Dorans Kirsten S, Rice Mary B, Schwartz Joel, Coull Brent A, Koutrakis Petros, Gold Diane R, Keaney John F, Lin Honghuang, Vasan Ramachandran S, Benjamin Emelia J, Mittleman Murray A
BACKGROUND	Short-term exposure to elevated air pollution has been associated with higher risk of acute cardiovascular diseases, with systemic oxidative stress induced by air pollution hypothesized as an important underlying mechanism. However, few community-based studies have assessed this association.
METHODS AND RESULTS	Two thousand thirty-five Framingham Offspring Cohort participants living within 50 km of the Harvard Boston Supersite who were not current smokers were included. We assessed circulating biomarkers of oxidative stress including blood myeloperoxidase at the seventh examination (1998-2001) and urinary creatinine-indexed 8-epi-prostaglandin F2α (8-epi-PGF2α) at the seventh and eighth (2005-2008) examinations. We measured fine particulate matter (PM2.5), black carbon, sulfate, nitrogen oxides, and ozone at the Supersite and calculated 1-, 2-, 3-, 5-, and 7-day moving averages of each pollutant. Measured myeloperoxidase and 8-epi-PGF2α were loge transformed. We used linear regression models and linear mixed-effects models with random intercepts for myeloperoxidase and indexed 8-epi-PGF2α, respectively. Models were adjusted for demographic variables, individual- and area-level measures of socioeconomic position, clinical and lifestyle factors, weather, and temporal trend. We found positive associations of PM2.5 and black carbon with myeloperoxidase across multiple moving averages. Additionally, 2- to 7-day moving averages of PM2.5 and sulfate were consistently positively associated with 8-epi-PGF2α. Stronger positive associations of black carbon and sulfate with myeloperoxidase were observed among participants with diabetes than in those without.
CONCLUSIONS	Our community-based investigation supports an association of select markers of ambient air pollution with circulating biomarkers of oxidative stress.

표-433. PubMed 논문번호 27129131의 내용 요약

구분	내용
PubMed ID	27129131
TITLE	Long-Term Fine Particulate Matter Exposure and Major Depressive Disorder in a Community-Based Urban Cohort.
JOURNAL	Environmental health perspectives: 10.1289/EHP192
AUTHORS	Kim Kyoung-Nam, Lim Youn-Hee, Bae Hyun Joo, Kim Myounghee, Jung Kweon, Hong Yun-Chul
BACKGROUND	Previous studies have associated short-term air pollution exposure with depression. Although an animal study showed an association between long-term exposure to particulate matter ≤ 2.5 μm (PM2.5) and depression, epidemiological studies assessing the long-term association are scarce.
OBJECTIVE	We aimed to determine the association between long-term PM2.5 exposure and major depressive disorder (MDD).
METHODS	A total of 27,270 participants 15-79 years of age who maintained an address within the same districts in Seoul, Republic of Korea, throughout the entire study period (between 2002 and 2010) and without a previous MDD diagnosis were analyzed. We used three district-specific exposure indices as measures of long-term PM2.5 exposure. Cox proportional hazards models adjusted for potential confounding factors and measured at district and individual levels were constructed. We further conducted stratified analyses according to underlying chronic diseases such as diabetes mellitus, cardiovascular disease, and chronic obstructive pulmonary disease.
RESULTS	The risk of MDD during the follow-up period (2008-2010) increased with an increase of 10 μg/m3 in PM2.5 in 2007 [hazard ratio (HR) = 1.44; 95% CI: 1.17, 1.78], PM2.5 between 2007 and 2010 (HR = 1.59; 95% CI: 1.02, 2.49), and 12-month moving average of PM2.5 until an event or censor (HR = 1.47; 95% CI: 1.14, 1.90). The association between long-term PM2.5 exposure and MDD was greater in participants with underlying chronic diseases than in participants without these diseases.
CONCLUSION	Long-term PM2.5 exposure increased the risk of MDD among the general population. Individuals with underlying chronic diseases are more vulnerable to long-term PM2.5 exposure.
CITATION	Kim KN, Lim YH, Bae HJ, Kim M, Jung K, Hong YC. 2016. Long-term fine particulate matter exposure and major depressive disorder in a community-based urban cohort. Environ Health Perspect 124:1547-1553; http://dx.doi.org/10.1289/EHP192.

표-434. PubMed 논문번호 27151956의 내용 요약

구분	내용
PubMed ID	27151956
TITLE	Coronary heart disease and household air pollution from use of solic systematic review.
JOURNAL	British medical bulletin: 10.1093/bmb/ldw015
AUTHORS	Fatmi Zafar, Coggon David
BACKGROUND	Evidence is emerging that indoor air pollution (IAP) from use of soli cooking and heating may be an important risk factor for coronary h disease (CHD).
SOURCES OF DATA	We searched the Ovid Medline, Embase Classic, Embase and Web o databases from inception through to June 12, 2015, to identify repor primary epidemiological research concerning the relationship of CH from solid fuel, the likely magnitude of any increase in risk, and pote pathogenic mechanisms.
AREAS OF AGREEMENT	The current balance of epidemiological evidence points to an increas CHD from IAP as a consequence of using solid, and especially biom for cooking and heating. Relative risks from long-term exposure co to 4-fold.
AREAS OF CONTROVERSY	The evidence base is still limited, and although an association of CH such IAP from solid fuel is consistent with the known hazards from environmental tobacco smoke and ambient air pollution, and suppo evidence of effects on inflammatory processes, atherosclerosis and bl pressure, it requires confirmation by larger and more robust studies.
GROWING POINTS	The completion of two relatively small case-control studies on CHD from use of biomass fuel demonstrates the feasibility of such researc an encouragement to further, larger studies using similar methods.
AREAS TIMELY FOR DEVELOPING RESEARCH	The need for such research is particularly pressing because the incide CHD in developing countries is rising, and IAP may interact synergis with the risk factors that are driving that increase. Furthermore, rela cheap methods are available to reduce IAP from use of solid fuels, a are indications from intervention studies that these may impact bene CHD as well as other diseases caused by such pollution.

표-435. PubMed 논문번호 27152932의 내용 요약

구분	내용
PubMed ID	27152932
TITLE	Ambient Fine Particulate Matter and Mortality among Survivors of Myocardial Infarction: Population-Based Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP185
AUTHORS	Chen Hong, Burnett Richard T, Copes Ray, Kwong Jeffrey C, Villeneuve Paul J, Goldberg Mark S, Brook Robert D, van Donkelaar Aaron, Jerrett Michael, Martin Randall V, Brook Jeffrey R, Kopp Alexander, Tu Jack V
BACKGROUND	Survivors of acute myocardial infarction (AMI) are at increased risk of dying within several hours to days following exposure to elevated levels of ambient air pollution. Little is known, however, about the influence of long-term (months to years) air pollution exposure on survival after AMI.
OBJECTIVE	We conducted a population-based cohort study to determine the impact of long-term exposure to fine particulate matter ≤ 2.5 μm in diameter (PM2.5) on post-AMI survival.
METHODS	We assembled a cohort of 8,873 AMI patients who were admitted to 1 of 86 hospital corporations across Ontario, Canada in 1999-2001. Mortality follow-up for this cohort extended through 2011. Cumulative time-weighted exposures to PM2.5 were derived from satellite observations based on participants' annual residences during follow-up. We used standard and multilevel spatial random-effects Cox proportional hazards models and adjusted for potential confounders.
RESULTS	Between 1999 and 2011, we identified 4,016 nonaccidental deaths, of which 2,147 were from any cardiovascular disease, 1,650 from ischemic heart disease, and 675 from AMI. For each 10-μg/m3 increase in PM2.5, the adjusted hazard ratio (HR10) of nonaccidental mortality was 1.22 [95% confidence interval (CI): 1.03, 1.45]. The association with PM2.5 was robust to sensitivity analyses and appeared stronger for cardiovascular-related mortality: ischemic heart (HR10 = 1.43; 95% CI: 1.12, 1.83) and AMI (HR10 = 1.64; 95% CI: 1.13, 2.40). We estimated that 12.4% of nonaccidental deaths (or 497 deaths) could have been averted if the lowest measured concentration in an urban area (4 μg/m3) had been achieved at all locations over the course of the study.
CONCLUSIONS	Long-term air pollution exposure adversely affects the survival of AMI patients.
CITATION	Chen H, Burnett RT, Copes R, Kwong JC, Villeneuve PJ, Goldberg MS, Brook RD, van Donkelaar A, Jerrett M, Martin RV, Brook JR, Kopp A, Tu JV. 2016. Ambient fine particulate matter and mortality among survivors of myocardial infarction: population-based cohort study. Environ Health Perspect 124:1421-1428; http://dx.doi.org/10.1289/EHP185.

표-436. PubMed 논문번호 27164554의 내용 요약

구분	내용
PubMed ID	27164554
TITLE	Residential exposure to traffic noise and risk of incident atrial fibrillation: A cohort study.
JOURNAL	Environment international: 10.1016/j.envint.2016.04.039
AUTHORS	Monrad Maria, Sajadieh Ahmad, Christensen Jeppe Schultz, Ketzel Matthias, Raaschou-Nielsen Ole, Tj ø nneland Anne, Overvad Kim, Loft Steffen, S ø rensen Mette
BACKGROUND	Studies have found long-term exposure to traffic noise to be associated with higher risk for hypertension, ischemic heart disease and stroke. We aimed to investigate the novel hypothesis that traffic noise increases the risk of atrial fibrillation (A-fib).
METHODS	In a population-based cohort of 57,053 people aged 50-64years at enrolment in 1993-1997, we identified 2692 cases of first-ever hospital admission of A-fib from enrolment to end of follow-up in 2011 using a nationwide registry. The mean follow-up time was 14.7years. Present and historical residential addresses were identified for all cohort members from 1987 to 2011. For all addresses, exposure to road traffic and railway noise was estimated using the Nordic prediction method and exposure to air pollution was estimated using a validated dispersion model. We used Cox proportional hazard model for the analyses with adjustment for lifestyle, socioeconomic position and air pollution.
RESULTS	A 10dB higher 5-year time-weighted mean exposure to road traffic noise was associated with a 6% higher risk of A-fib (incidence rate ratio (IRR): 1.06; 95% confidence interval (95% CI): 1.00-1.12) in models adjusted for factors related to lifestyle and socioeconomic position. The association followed a monotonic exposure-response relationship. In analyses with adjustment for air pollution, NOx or NO2, there were no statistically significant associations between exposure to road traffic noise and risk of A-fib; IRR: 1.04; (95% CI: 0.96-1.11) and IRR: 1.01; (95% CI: 0.94-1.09), respectively. Exposure to railway noise was not associated with A-fib.
CONCLUSION	Exposure to residential road traffic noise may be associated with higher risk of A-fib, though associations were difficult to separate from exposure to air pollution.

표-437. PubMed 논문번호 27177127의 내용 요약

구분	내용
PubMed ID	27177127
TITLE	Long-Term Exposure to Particulate Matter and Self-Reported Hypertension: A Prospective Analysis in the Nurses' Health Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP163
AUTHORS	Zhang Zhenyu, Laden Francine, Forman John P, Hart Jaime E
BACKGROUND	Studies have suggested associations between elevated blood pressure and short-term air pollution exposures, but the evidence is mixed regarding long-term exposures on incidence of hypertension.
OBJECTIVES	We examined the association of hypertension incidence with long-term residential exposures to ambient particulate matter (PM) and residential distance to roadway.
METHODS	We estimated 24-month and cumulative average exposures to PM10, PM2.5, and PM2.5-10 and residential distance to road for women participating in the prospective nationwide Nurses' Health Study. Hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated for incident hypertension from 1988 to 2008 using Cox proportional hazards models adjusted for potential confounders. We considered effect modification by age, diet, diabetes, obesity, region, and latitude.
RESULTS	Among 74,880 participants, 36,812 incident cases of hypertension were observed during 960,041 person-years. In multivariable models, 10-μg/m3 increases in 24-month average PM10, PM2.5, and PM2.5-10 were associated with small increases in the incidence of hypertension (HR: 1.02, 95% CI: 1.00, 1.04; HR: 1.04, 95% CI: 1.00, 1.07; and HR: 1.03, 95% CI: 1.00, 1.07, respectively). Associations were stronger among women < 65 years of age (HR: 1.04, 95% CI: 1.01, 1.06; HR: 1.07, 95% CI: 1.02, 1.12; and HR: 1.05, 95% CI: 1.01, 1.09, respectively) and the obese (HR: 1.07, 95% CI: 1.04, 1.12; HR: 1.15, 95% CI: 1.07, 1.23; and HR: 1.13, 95% CI: 1.07, 1.19, respectively), with p-values for interaction < 0.05 for all models except age and PM2.5-10. There was no association with roadway proximity.
CONCLUSIONS	Long-term exposure to particulate matter was associated with small increases in risk of incident hypertension, particularly among younger women and the obese.
CITATION	Zhang Z, Laden F, Forman JP, Hart JE. 2016. Long-term exposure to particulate matter and self-reported hypertension: a prospective analysis in the Nurses' Health Study. Environ Health Perspect 124:1414-1420; http://dx.doi.org/10.1289/EHP163.

표-438. PubMed 논문번호 27193463의 내용 요약

구분	내용
PubMed ID	27193463
TITLE	Disease burden of ischaemic heart disease from short-term outdoor air pollution exposure in Tianjin, 2002-2006.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487316651352
AUTHORS	Li Guoxing, Zeng Qiang, Pan Xiaochuan
AIMS	To obtain a reliable estimation of the health impact of air pollution, we explored the effects of air pollutants on years of life lost from ischaemic heart disease in Tianjin, China.
METHODS AND RESULTS	We conducted a retrospective ecological analysis using daily time series set in six urban districts in Tianjin. Our data included 28,365 ischaemic heart disease deaths registered in 2002-2006. The main outcome measures were years of life lost and deaths from ischaemic heart disease. We found mean concentrations of daily particulate matter with an aerodynamic diameter of less than 10 □m in size (PM10), sulfur dioxide and nitrogen dioxide of 125.6 □g/m3, 78.7 □g/m3, 55.1 □g/m3, respectively. Interquartile range increases of PM10, sulfur dioxide and nitrogen dioxide were associated with increases in years of life lost from ischaemic heart disease of 13.8, 4.8 and 22.7 years in single pollutant models, respectively. The effects associated with an interquartile range increase in PM10 were greater in women than men. During the 5 years, there were 27,485 years and 1252 deaths advanced by PM10 pollution over expected rates when daily levels did exceed the World Health Organization air quality guidelines. That is to say, on average, 63.12 hours per person were lost by air pollution in the 5-year period in Tianjin.
CONCLUSIONS	Short-term exposure to air pollution has a significant impact on ischaemic heart disease years of life lost and mortality in Tianjin, particularly among women. Air quality improvement has become necessary to protect public health.

표-439. PubMed 논문번호 27218271의 내용 요약

구분	내용
PubMed ID	27218271
TITLE	The Association Between Air Pollution Exposure and Glucose and Lipids Levels.
JOURNAL	The Journal of clinical endocrinology and metabolism: 10.1210/jc.2016-1378
AUTHORS	Yitshak Sade Maayan, Kloog Itai, Liberty Idit F, Schwartz Joel, Novack Victor
CONTEXT	Evidence from recent decades supports a causal association between air pollution (particulate matter <10 μm in diameter [PM10] and PM <2.5 μm in diameter [PM2.5]) and oxidative stress, possibly involving impaired metabolism of glucose and lipids.
OBJECTIVE	Using a satellite based model to assess PM exposure at 1-km spatial resolution, we examined the associations between PM and glucose, hemoglobin A1c (HbA1c), and lipids.
DESIGN	Population-based retrospective cohort study of a 10-year period.
SETTING	Members of the largest health care provider in Southern Israel.
PARTICIPANTS	We included all serum glucose, HbA1c, and lipids tests of subjects with known cardiovascular diseases and risk factors. Subjects' glycemic status was defined as normal or diabetes.
MAIN OUTCOME	Log-transformed glucose, HbA1c, and lipid values were explored by mixed models, with adjustment for personal and seasonal confounders.
RESULTS	We assessed 73 117 subjects with over 600 000 samples. Three-month average concentration of PM10, but not 1- to 7-d exposure, was associated with increases of serum glucose, HbA1c, low-density lipoprotein and triglycerides, and decrease of high-density lipoprotein. The strongest associations were observed among subjects with diabetes (percent increase [95% confidence interval], for interquartile range increase of PM10 and PM2.5): 3.58% (1.03%; 6.20%) and 2.93% (0.35%; 5.59%) increase in HbA1c and 2.37% (2.11%; 2.63%) and 1.54% (1.26%; 1.83%) increase in low-density lipoprotein. Antidiabetic medications (other than insulin) attenuated the air pollution effect on serum glucose.
CONCLUSIONS	Intermediate-term, but not short term, exposure to PM is associated with alterations in glucose, HbA1c, and lipids, especially among people with diabetes.

표-440. PubMed 논문번호 27221567의 내용 요약

구분	내용
PubMed ID	27221567
TITLE	Effects of urban fine particulate matter and ozone on HDL functionality.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-016-0139-3
AUTHORS	Ramanathan Gajalakshmi, Yin Fen, Speck Mary, Tseng Chi-Hong, Brook Jeffrey R, Silverman Frances, Urch Bruce, Brook Robert D, Araujo Jesus A
BACKGROUND	Exposures to ambient particulate matter (PM) are associated with increased morbidity and mortality. PM2.5 (<2.5 μm) and ozone exposures have been shown to associate with carotid intima media thickness in humans. Animal studies support a causal relationship between air pollution and atherosclerosis and identified adverse PM effects on HDL functionality. We aimed to determine whether brief exposures to PM2.5 and/or ozone could induce effects on HDL anti-oxidant and anti-inflammatory capacity in humans.
METHODS	Subjects were exposed to fine concentrated ambient fine particles (CAP) with PM2.5 targeted at 150 μg/m(3), ozone targeted at 240 μg/m(3) (120 ppb), PM2.5 plus ozone targeted at similar concentrations, and filtered air (FA) for 2 h, on 4 different occasions, at least two weeks apart, in a randomized, crossover study. Blood was obtained before exposures (baseline), 1 h after and 20 h after exposures. Plasma HDL anti-oxidant/anti-inflammatory capacity and paraoxonase activity were determined. HDL anti-oxidant/anti-inflammatory capacity was assessed by a cell-free fluorescent assay and expressed in units of a HDL oxidant index (HOI). Changes in HOI (ΔHOI) were calculated as the difference in HOI from baseline to 1 h after or 20 h after exposures.
RESULTS	There was a trend towards bigger ΔHOI between PM2.5 and FA 1 h after exposures ($p = 0.18$) but not 20 h after. This trend became significant ($p < 0.05$) when baseline HOI was lower (<1.5 or <2.0), indicating decreased HDL anti-oxidant/anti-inflammatory capacity shortly after the exposures. There were no significant effects of ozone alone or in combination with PM2.5 on the change in HOI at both time points. The change in HOI due to PM2.5 showed a positive trend with particle mass concentration ($p = 0.078$) and significantly associated with the slope of systolic blood pressure during exposures ($p = 0.005$).
CONCLUSIONS	Brief exposures to concentrated PM2.5 elicited swift effects on HDL anti-oxidant/anti-inflammatory functionality, which could indicate a potential mechanism for how particulate air pollution induces harmful cardiovascular effects.

표-441. PubMed 논문번호 27233746의 내용 요약

구분	내용
PubMed ID	27233746
TITLE	Association between air pollution and coronary artery calcification within six metropolitan areas in the USA (the Multi-Ethnic Study of Atherosclerosis and Air Pollution): a longitudinal cohort study.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(16)00378-0
AUTHORS	Kaufman Joel D, Adar Sara D, Barr R Graham, Budoff Matthew, Burke Gregory L, Curl Cynthia L, Daviglus Martha L, Diez Roux Ana V, Gassett Amanda J, Jacobs David R, Kronmal Richard, Larson Timothy V, Navas-Acien Ana, Olives Casey, Sampson Paul D, Sheppard Lianne, Siscovick David S, Stein James H, Szpiro Adam A, Watson Karol E
BACKGROUND	Long-term exposure to fine particulate matter less than 2.5 μm in diameter (PM2.5) and traffic-related air pollutant concentrations are associated with cardiovascular risk. The disease process underlying these associations remains uncertain. We aim to assess association between long-term exposure to ambient air pollution and progression of coronary artery calcium and common carotid artery intima-media thickness.
METHODS	In this prospective 10-year cohort study, we repeatedly measured coronary artery calcium by CT in 6795 participants aged 45-84 years enrolled in the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air) in six metropolitan areas in the USA. Repeated scans were done for nearly all participants between 2002 and 2005, for a subset of participants between 2005 and 2007, and for half of all participants between 2010 and 2012. Common carotid artery intima-media thickness was measured by ultrasound in all participants at baseline and in 2010-12 for 3459 participants. Residence-specific spatio-temporal pollution concentration models, incorporating community-specific measurements, agency monitoring data, and geographical predictors, estimated concentrations of PM2.5 and nitrogen oxides (NOX) between 1999 and 2012. The primary aim was to examine the association between both progression of coronary artery calcium and mean carotid artery intima-media thickness and long-term exposure to ambient air pollutant concentrations (PM2.5, NOX, and black carbon) between examinations and within the six metropolitan areas, adjusting for baseline age, sex, ethnicity, socioeconomic characteristics, cardiovascular risk factors, site, and CT scanner technology.
FINDINGS	In this population, coronary calcium increased on average by 24 Agatston units per year (SD 58), and intima-media thickness by 12 μm per year (10), before adjusting for risk factors or air pollutant exposures. Participant-specific pollutant concentrations averaged over the years 2000-10 ranged from 9.2-22.6 μg PM2.5/m(3) and 7.2-139.2 parts per billion (ppb) NOX. For each 5 μg PM2.5/m(3) increase, coronary calcium progressed by 4.1 Agatston units per year (95% CI 1.4-6.8) and for each 40 ppb NOX coronary calcium progressed by 4.8 Agatston units per year (0.9-8.7). Pollutant exposures were not associated with intima-media thickness change. The estimate for the effect of a 5 μg/m(3) higher long-term exposure to PM2.5 in intima-media thickness was -0.9 μm per year (95% CI -3.0 to 1.3). For 40 ppb higher NOX, the estimate was 0.2 μm per year (-1.9 to 2.4).
INTERPRETATION	Increased concentrations of PM2.5 and traffic-related air pollution within metropolitan areas, in ranges commonly encountered worldwide, are associated with progression in coronary calcification, consistent with acceleration of atherosclerosis. This study supports the case for global efforts of pollution reduction in prevention of cardiovascular diseases.

표-442. PubMed 논문번호 27258294의 내용 요약

구분	내용
PubMed ID	27258294
TITLE	Tracking Restoration of Park and Urban Street Settings in Coronary Artery Disease Patients.
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph13060550
AUTHORS	Grazuleviciene Regina, Vencloviene Jone, Kubilius Raimondas, Grizas Vytautas, Danileviciute Asta, Dedele Audrius, Andrusaityte Sandra, Vitkauskiene Astra, Steponaviciute Rasa, Nieuwenhuijsen Mark J
UNLABELLED	The physiological effects of natural and urban environments on the cardiovascular system of coronary artery disease (CAD) patients are not fully understood. This controlled field study examines the effects of restorative walking in a park vs. in an urban street environment on CAD patients' stress parameters and cardiac function.
METHODS	Twenty stable CAD patients were randomly allocated to 7 days controlled walking in a city park or in an urban street environment group. The relationship between different environmental exposures and health effects was analyzed using Wilcoxon signed-rank test and exact Mann-Whitney U test.
RESULTS	The mean reduction in cortisol levels and negative effects after the walk on the first day was greater in the city park than in the urban street exposed group, while a reduction in negative effects in the urban group were greater after seven days. The reduction in diastolic blood pressure (DBP) in the park group was evident on the seventh day before the walk (-4 mm Hg, $p = 0.031$) and 60 min after the walk (-6.00 mm Hg, $p = 0.002$). The cortisol slope was negatively associated with the DBP changes ($r = -0.514$, $p < 0.05$).
CONCLUSIONS	Physical activity in a green environment with noise and air pollution levels lower than in an urban environment has a greater positive effect on CAD patients' stress level and hemodynamic parameters. Mitigating green environmental influences may allow urban residents to maintain health and reduce disability.

표-443. PubMed 논문번호 27258721의 내용 요약

구분	내용
PubMed ID	27258721
TITLE	Particulate Matter and Subclinical Atherosclerosis: Associations between Different Particle Sizes and Sources with Carotid Intima-Media Thickness in the SAPALDIA Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP161
AUTHORS	Aguilera Inmaculada, Dratva Julia, Caviezel Seraina, Burdet Luc, de Groot Eric, Ducret-Stich Regina E, Eeftens Marloes, Keidel Dirk, Meier Reto, Perez Laura, Rothe Thomas, Schaffner Emmanuel, Schmit-Trucks□ss Arno, Tsai Ming-Yi, Schindler Christian, K□nzli Nino, Probst-Hensch Nicole
BACKGROUND	Subclinical atherosclerosis has been associated with long-term exposure to particulate matter (PM), but the relevance of particle size and sources of exposure remains unclear.
OBJECTIVES	We investigated the association of long-term exposure to PM10 (≤ 10 μm), PM2.5 (≤ 2.5 μm: total mass, vehicular, and crustal sources), and ultrafine particles [UFP < 0.1 μm: particle number concentration (PNC) and lung-deposited surface area (LDSA)] with carotid intima-media thickness (CIMT).
METHODS	We used data from 1,503 participants ≥ 50 years old who participated in the third examination of the Swiss SAPALDIA cohort. Exposures were obtained from dispersion models and land-use regression models. Covariate information, including previous cardiovascular risk factors, was obtained from the second and third SAPALDIA examinations.
RESULTS	The adjusted percent difference in CIMT associated with an exposure contrast between the 10th and 90th percentile was 1.58% (95% CI: -0.30, 3.47%) for PM10, 2.10% (95% CI: 0.04, 4.16%) for PM2.5, 1.67% (95% CI: -0.13, 3.48%) for the vehicular source of PM2.5, -0.58% (95% CI: -3.95, 2.79%) for the crustal source of PM2.5, 2.06% (95% CI: 0.03, 4.10%) for PNC, and 2.32% (95% CI: 0.23, 4.40%) for LDSA. Stronger associations were observed among diabetics, subjects with low-educational level, and those at higher cardiovascular risk.
CONCLUSIONS	CIMT was associated with exposure to PM10, PM2.5, and UFP. The PM2.5 source-specific analysis showed a positive association for the vehicular source but not for the crustal source. Although the effects of PNC and LDSA were similar in magnitude, two-pollutant and residual-based models suggested that LDSA may be a better marker for the health relevance of UFP. Citation: Aguilera I, Dratva J, Caviezel S, Burdet L, de Groot E, Ducret-Stich RE, Eeftens M, Keidel D, Meier R, Perez L, Rothe T, Schaffner E, Schmit-Trucks□ss A, Tsai MY, Schindler C, K□nzli N, Probst-Hensch N. 2016. Particulate matter and subclinical atherosclerosis: associations between different particle sizes and sources with carotid intima-media thickness in the SAPALDIA study. Environ Health Perspect 124:1700-1706; http://dx.doi.org/10.1289/EHP161.

표-444. PubMed 논문번호 27291521의 내용 요약

구분	내용
PubMed ID	27291521
TITLE	Global burden of stroke and risk factors in 188 countries, during 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013.
JOURNAL	The Lancet. Neurology: 10.1016/S1474-4422(16)30073-4
AUTHORS	Feigin Valery L, Roth Gregory A, Naghavi Mohsen, Parmar Priya, Krishnamurthi Rita, Chugh Sumeet, Mensah George A, Norrving Bo, Shiue Ivy, Ng Marie, Estep Kara, Cercy Kelly, Murray Christopher J L, Forouzanfar Mohammad H
BACKGROUND	The contribution of modifiable risk factors to the increasing global and regional burden of stroke is unclear, but knowledge about this contribution is crucial for informing stroke prevention strategies. We used data from the Global Burden of Disease Study 2013 (GBD 2013) to estimate the population-attributable fraction (PAF) of stroke-related disability-adjusted life-years (DALYs) associated with potentially modifiable environmental, occupational, behavioural, physiological, and metabolic risk factors in different age and sex groups worldwide and in high-income countries and low-income and middle-income countries, from 1990 to 2013.
METHODS	We used data on stroke-related DALYs, risk factors, and PAF from the GBD 2013 Study to estimate the burden of stroke by age and sex (with corresponding 95% uncertainty intervals [UI]) in 188 countries, as measured with stroke-related DALYs in 1990 and 2013. We evaluated attributable DALYs for 17 risk factors (air pollution and environmental, dietary, physical activity, tobacco smoke, and physiological) and six clusters of risk factors by use of three inputs: risk factor exposure, relative risks, and the theoretical minimum risk exposure level. For most risk factors, we synthesised data for exposure with a Bayesian meta-regression method (DisMod-MR) or spatial-temporal Gaussian process regression. We based relative risks on meta-regressions of published cohort and intervention studies. Attributable burden for clusters of risks and all risks combined took into account evidence on the mediation of some risks, such as high body-mass index (BMI), through other risks, such as high systolic blood pressure (SBP) and high total cholesterol.
FINDINGS	Globally, 90·5% (95% UI 88·5-92·2) of the stroke burden (as measured in DALYs) was attributable to the modifiable risk factors analysed, including 74·2% (95% UI 70·7-76·7) due to behavioural factors (smoking, poor diet, and low physical activity). Clusters of metabolic factors (high SBP, high BMI, high fasting plasma glucose, high total cholesterol, and low glomerular filtration rate; 72·4%, 95% UI 70·2-73·5) and environmental factors (air pollution and lead exposure; 33·4%, 95% UI 32·4-34·3) were the second and third largest contributors to DALYs. Globally, 29·2% (95% UI 28·2-29·6) of the burden of stroke was attributed to air pollution. Although globally there were no significant differences between sexes in the proportion of stroke burden due to behavioural, environmental, and metabolic risk clusters, in the low-income and middle-income countries, the PAF of behavioural risk clusters in males was greater than in females. The PAF of all risk factors increased from 1990 to 2013 (except for second-hand smoking and household air pollution from solid fuels) and varied significantly between countries.
INTERPRETATION	Our results suggest that more than 90% of the stroke burden is attributable to modifiable risk factors, and achieving control of behavioural and metabolic risk factors could avert more than three-quarters of the global stroke burden. Air pollution has emerged as a significant contributor to global stroke burden,

표-445. PubMed 논문번호 27297340의 내용 요약

구분	내용
PubMed ID	27297340
TITLE	Household Fuel Use and Cardiovascular Disease Mortality: Golestan Cohort Study.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.115.020288
AUTHORS	Mitter Sumeet S, Vedanthan Rajesh, Islami Farhad, Pourshams Akram, Khademi Hooman, Kamangar Farin, Abnet Christian C, Dawsey Sanford M, Pharoah Paul D, Brennan Paul, Fuster Valentin, Boffetta Paolo, Malekzadeh Reza
BACKGROUND	Household air pollution is the third largest risk factor for global disease burden, but direct links with cardiovascular disease mortality are limited. This study aimed to evaluate the relationship between household fuel use and cardiovascular disease mortality.
METHODS AND RESULTS	The Golestan Cohort Study in northeastern Iran enrolled 50 045 individuals 40 to 75 years of age between 2004 and 2008 and collected data on lifetime household fuel use and other baseline exposures. Participants were followed up through 2012 with a 99% successful follow-up rate. Cox proportional hazards models were fitted to calculate hazard ratios for associations between pehen (local dung), wood, kerosene/diesel, or natural gas burning for cooking and heating and all-cause and cause-specific mortality, with adjustment for lifetime exposure to each of these fuels and potential confounders. A total of 3073 participants (6%) died during follow-up; 78% of these deaths were attributable to noncommunicable diseases, including cardiovascular, oncological, and respiratory illnesses. Adjusted 10-year hazard ratios from kerosene/diesel burning were 1.06 (95% confidence interval, 1.02-1.10) and 1.11 (95% confidence interval, 1.06-1.17) for all-cause and cardiovascular mortality, respectively. Subtype-specific analyses revealed a significant increase in ischemic heart disease (10-year hazard ratio, 1.14; 95% confidence interval, 1.06-1.21) and a trend toward cerebrovascular accident (10-year hazard ratio, 1.08; 95% confidence interval, 0.99-1.17) mortality. Stratification by sex revealed a potential signal for increased risk for all-cause and cardiovascular disease mortality among women compared with men, with similar risk for ischemic heart disease mortality.
CONCLUSIONS	Household exposure to high-pollution fuels was associated with increased risk for all-cause and cardiovascular disease mortality. Replicating these results worldwide would support efforts to reduce such exposures.

표-446. PubMed 논문번호 27312220의 내용 요약

구분	내용
PubMed ID	27312220
TITLE	Residential Proximity to Major Roads, Exposure to Fine Particulate Matter, and Coronary Artery Calcium: The Framingham Heart Study.
JOURNAL	Arteriosclerosis, thrombosis, and vascular biology: 10.1161/ATVBAHA.116.307141
AUTHORS	Dorans Kirsten S, Wilker Elissa H, Li Wenyuan, Rice Mary B, Ljungman Petter L, Schwartz Joel, Coull Brent A, Kloog Itai, Koutrakis Petros, D'Agostino Ralph B, Massaro Joseph M, Hoffmann Udo, O'Donnell Christopher J, Mittleman Murray A
OBJECTIVE	Long-term exposure to traffic and particulate matter air pollution is associated with a higher risk of cardiovascular disease, potentially via atherosclerosis promotion. Prior research on associations of traffic and particulate matter with coronary artery calcium Agatston score (CAC), an atherosclerosis correlate, has yielded inconsistent findings. Given this background, we assessed whether residential proximity to major roadway or fine particulate matter were associated with CAC in a Northeastern US study.
APPROACH AND RESULTS	We measured CAC ≤2 times from 2002 to 2005 and 2008 to 2011 among Framingham Offspring or Third-Generation Cohort participants. We assessed associations of residential distance to major roadway and residential fine particulate matter (2003 average; spatiotemporal model) with detectable CAC, using generalized estimating equation regression. We used linear mixed effects models to assess associations with loge(CAC). We also assessed associations with CAC progression. Models were adjusted for demographic variables, socioeconomic position markers, and time. Among 3399 participants, 51% had CAC measured twice. CAC was detectable in 47% of observations. At first scan, mean age was 52.2 years (standard deviation 11.7); 51% male. There were no consistent associations with detectable CAC, continuous CAC, or CAC progression. We observed heterogeneous associations of distance to major roadway with odds of detectable CAC by hypertensive status; interpretation of these findings is questionable.
CONCLUSIONS	Our findings add to prior work and support evidence against strong associations of traffic or fine particulate matter with the presence, extent, or progression of CAC in a region with relatively low levels of and little variation in fine particulate matter.

표-447. PubMed 논문번호 27318724의 내용 요약

구분	내용
PubMed ID	27318724
TITLE	Association of long-term exposure to local industry- and traffic-specific particulate matter with arterial blood pressure and incident hypertension.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2016.05.008
AUTHORS	Fuks Kateryna B, Weinmayr Gudrun, Hennig Frauke, Tzivian Lilian, Moebus Susanne, Jakobs Hermann, Memmesheimer Michael, K□lsch Hagen, Andrich Silke, Nonnemacher Michael, Erbel Raimund, J□ckel Karl-Heinz, Hoffmann Barbara
BACKGROUND	Long-term exposure to fine particulate matter (PM2.5) may lead to increased blood pressure (BP). The role of industry- and traffic-specific PM2.5 remains unclear.
OBJECTIVE	We investigated the associations of residential long-term source-specific PM2.5 exposure with arterial BP and incident hypertension in the population-based Heinz Nixdorf Recall cohort study.
METHODS	We defined hypertension as systolic BP≥140mmHg, or diastolic BP≥90mmHg, or current use of BP lowering medication. Long-term concentrations of PM2.5 from all local sources (PM2.5ALL), local industry (PM2.5IND) and traffic (PM2.5TRA) were modeled with a dispersion and chemistry transport model (EURAD-CTM) with a 1km(2) resolution. We performed a cross-sectional analysis with BP and prevalent hypertension at baseline, using linear and logistic regression, respectively, and a longitudinal analysis with incident hypertension at 5-year follow-up, using Poisson regression with robust variance estimation. We adjusted for age, sex, body mass index, lifestyle, education, and major road proximity. Change in BP (mmHg), odds ratio (OR) and relative risk (RR) for hypertension were calculated per 1 μg/m(3) of exposure concentration.
RESULTS	PM2.5ALL was highly correlated with PM2.5IND (Spearman's ρ=0.92) and moderately with PM2.5TRA (ρ=0.42). In adjusted cross-sectional analysis with 4539 participants, we found positive associations of PM2.5ALL with systolic (0.42 [95%-CI: 0.03, 0.80]) and diastolic (0.25 [0.04, 0.46]) BP. Higher, but less precise estimates were found for PM2.5IND (systolic: 0.55 [-0.05, 1.14]; diastolic: 0.35 [0.03, 0.67]) and PM2.5TRA (systolic: 0.88 [-1.55, 3.31]; diastolic: 0.41 [-0.91, 1.73]). We found crude positive association of PM2.5TRA with prevalence (OR 1.41 [1.10, 1.80]) and incidence of hypertension (RR 1.38 [1.03, 1.85]), attenuating after adjustment (OR 1.19 [0.90, 1.58] and RR 1.28 [0.94, 1.72]). We found no association of PM2.5ALL and PM2.5IND with hypertension.
CONCLUSIONS	Long-term exposures to all-source and industry-specific PM2.5 were positively related to BP. We could not separate the effects of industry-specific PM2.5 from all-source PM2.5. Estimates with traffic-specific PM2.5 were generally higher but inconclusive.

표-448. PubMed 논문번호 27343184의 내용 요약

구분	내용
PubMed ID	27343184
TITLE	Traffic pollution and the incidence of cardiorespiratory outcomes in an adult cohort in London.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2015-103531
AUTHORS	Carey I M, Anderson H R, Atkinson R W, Beevers S, Cook D G, Dajnak D, Gulliver J, Kelly F J
OBJECTIVES	The epidemiological evidence for adverse health effects of long-term exposure to air and noise pollution from traffic is not coherent. Further, the relative roles of background versus near traffic pollution concentrations in this process are unclear. We investigated relationships between modelled concentrations of air and noise pollution from traffic and incident cardiorespiratory disease in London.
METHODS	Among 211 016 adults aged 40-79 years registered in 75 Greater London practices between 2005 and 2011, the first diagnosis for a range of cardiovascular and respiratory outcomes were identified from primary care and hospital records. Annual baseline concentrations for nitrogen oxide (NOx), particulate matter with a median aerodynamic diameter <2.5 μm (PM2.5) attributable to exhaust and non-exhaust sources, traffic intensity and noise were estimated at 20 m2 resolution from dispersion models, linked to clinical data via residential postcode. HRs were adjusted for confounders including smoking and area deprivation.
RESULTS	The largest observed associations were between traffic-related air pollution and heart failure (HR=1.10 for 20 μg/m3 change in NOx, 95% CI 1.01 to 1.21). However, no other outcomes were consistently associated with any of the pollution indicators, including noise. The greater variations in modelled air pollution from traffic between practices, versus within, hampered meaningful fine spatial scale analyses.
CONCLUSIONS	The associations observed with heart failure may suggest exacerbatory effects rather than underlying chronic disease. However, the overall failure to observe wider associations with traffic pollution may reflect that exposure estimates based on residence inadequately represent the relevant pattern of personal exposure, and future studies must address this issue.

표-449. PubMed 논문번호 27348520의 내용 요약

구분	내용
PubMed ID	27348520
TITLE	Pathways from parental educational attainment to adolescent blood pressure.
JOURNAL	Journal of hypertension: 10.1097/HJH.0000000000001003
AUTHORS	Kwok Man Ki, Schooling Catherine Mary, Subramanian Subu V, Leung Gabriel M, Kawachi Ichiro
OBJECTIVES	Lower parental education is associated with higher adolescent blood pressure (BP). We examined the contribution of modifiable risk factors from infancy to adolescence that could potentially explain the link between parental education and SBP and DBP in the offspring.
METHODS	In a prospective Chinese birth cohort, 'Children of 1997' of 5604 adolescents (68% follow-up), we analyzed the relation between parental educational attainment and sex-specific, age-specific and height-specific BP z-scores at ~13 years. Using mediation analysis, we examined the contribution of household income at birth (both absolute income and relative income deprivation), exposures during infancy (breastfeeding and early life second-hand smoking), lifestyles during childhood (diet, physical activity and screen-time), weight or BMI status during fetal, infancy, childhood and puberty, pubertal stage as well as parental BMI.
RESULTS	We found that adolescent BMI, but not birth weight or infant growth or childhood BMI, mediated the inverse association of parental education with adolescent SBP (proportion mediated: 24%), followed by maternal BMI (proportion mediated: 18%). Factors explaining the link between parental education and DBP were less clear. Absolute income, breastfeeding, childhood diet and physical activity, pubertal stage and paternal BMI did not mediate the association between parental education and adolescent BP.
CONCLUSION	Low parental education is a risk factor for high SBP and, to a lesser extent, DBP in adolescents. Important mediators of this relation include adolescent and maternal body weight.

표-450. PubMed 논문번호 27372639의 내용 요약

구분	내용
PubMed ID	27372639
TITLE	Fine Particulate Matter, Residential Proximity to Major Roads, and Markers of Small Vessel Disease in a Memory Study Population.
JOURNAL	Journal of Alzheimer's disease : JAD: 10.3233/JAD-151143
AUTHORS	Wilker Elissa H, Martinez-Ramirez Sergi, Kloog Itai, Schwartz Joel, Mostofsky Elizabeth, Koutrakis Petros, Mittleman Murray A, Viswanathan Anand
BACKGROUND	Long-term exposure to ambient air pollution has been associated with impaired cognitive function and vascular disease in older adults, but little is known about these associations among people with concerns about memory loss.
OBJECTIVE	To examine associations between exposures to fine particulate matter and residential proximity to major roads and markers of small vessel disease.
METHODS	From 2004-2010, 236 participants in the Massachusetts Alzheimer's Disease Research Center Longitudinal Cohort participated in neuroimaging studies. Residential proximity to major roads and estimated 2003 residential annual average of fine particulate air pollution (PM2.5) were linked to measures of brain parenchymal fraction (BPF), white matter hyperintensities (WMH), and cerebral microbleeds. Associations were modeled using linear and logistic regression and adjusted for clinical and lifestyle factors.
RESULTS	In this population (median age [interquartile range]=74 [12], 57% female) living in a region with median 2003 PM2.5 annual average below the current Environmental Protection Agency (EPA) standard, there were no associations between living closer to a major roadway or for a 2 μg/m3 increment in PM2.5 and smaller BPF, greater WMH volume, or a higher odds of microbleeds. However, a 2 μg/m3 increment in PM2.5 was associated with -0.19 (95% Confidence Interval (CI): -0.37, -0.005) lower natural log-transformed WMH volume. Other associations had wide confidence intervals.
CONCLUSIONS	In this population, where median 2003 estimated PM2.5 levels were below the current EPA standard, we observed no pattern of association between residential proximity to major roads or 2003 average PM2.5 and greater burden of small vessel disease or neurodegeneration.

표-451. PubMed 논문번호 27397927의 내용 요약

구분	내용
PubMed ID	27397927
TITLE	DNA hypomethylation and its mediation in the effects of fine particulate air pollution on cardiovascular biomarkers: A randomized crossover trial.
JOURNAL	Environment international: 10.1016/j.envint.2016.06.026
AUTHORS	Chen Renjie, Meng Xia, Zhao Ang, Wang Cuicui, Yang Changyuan, Li Huichu, Cai Jing, Zhao Zhuohui, Kan Haidong
BACKGROUND	Short-term exposure to fine particulate matter (PM2.5) air pollution has been associated with altered DNA methylation in observational studies, but it remains unclear whether this change mediates the effects on cardiovascular biomarkers.
OBJECTIVE	To examine the impact of ambient PM2.5 on gene-specific DNA methylation and its potential mediation in the acute effects of PM2.5 on cardiovascular biomarkers.
METHODS	We designed a randomized, double-blind crossover trial using true or sham air purifiers for 48h among 35 healthy college students in Shanghai, China, in 2014. We measured blood global methylation estimated in long interspersed nucleotide element-1 (LINE□1) and Alu repetitive elements, methylation in ten specific genes, and ten cardiovascular biomarkers. We used linear mixed-effect models to examine the associations between PM2.5 and methylation. We also performed causal mediation analyses to evaluate the potential mediation of methylation in the associations between PM2.5 and biomarkers.
RESULTS	Air purification increased DNA methylation in repetitive elements and all candidate genes. An IQR increase (64 μg/m(3)) in PM2.5 was significantly associated with reduction of methylation in LINE-1 (1.44%), one pro-inflammatory gene (CD40LG, 9.13%), two pro-coagulant genes (F3, 15.20%; SERPINE1, 3.69%), and two pro-vasoconstriction genes (ACE, 4.64%; EDN1, 9.74%). There was a significant mediated effect (17.82%, P=0.03) of PM2.5 on sCD40L protein through CD40LG hypomethylation. Hypomethylation in other candidate genes generally showed positive but non-significant mediation.
CONCLUSIONS	This intervention study provided robust human evidence that ambient PM2.5 could induce rapid decreases in DNA methylation and consequently partly mediate its effects on cardiovascular biomarkers.

표-452. PubMed 논문번호 27397929의 내용 요약

구분	내용
PubMed ID	27397929
TITLE	Personal exposure to fine particulate matter and blood pressure: A role of angiotensin converting enzyme and its DNA methylation.
JOURNAL	Environment international: 10.1016/j.envint.2016.07.001
AUTHORS	Wang Cuicui, Chen Renjie, Cai Jing, Shi Jingjin, Yang Changyuan, Tse Lap Ah, Li Huichu, Lin Zhijing, Meng Xia, Liu Cong, Niu Yue, Xia Yongjie, Zhao Zhuohui, Kan Haidong
BACKGROUND	The underlying intermediate mechanisms about the association between fine particulate matter (PM2.5) air pollution and blood pressure (BP) were unclear. Few epidemiological studies have explored the potential mediation effects of angiotensin-converting enzyme (ACE) and its DNA methylation.
METHODS	We designed a longitudinal panel study with 4 follow-ups among 36 healthy college students in Shanghai, China from December 17, 2014 to July 11, 2015. We measured personal real-time exposure to PM2.5, serum ACE level, and blood methylation of ACE gene and the repetitive elements. We applied linear mixed-effects models to examine the effects of PM2.5 on ACE protein, DNA methylation and BP markers. Furthermore, we conducted mediation analyses to evaluate the potential pathways.
RESULTS	An interquartile range increase (26.78 μg/m(3)) in 24-h average exposure to PM2.5 was significantly associated with 1.12 decreases in ACE average methylation (%5mC), 13.27% increase in ACE protein, and increments of 1.13mmHg in systolic BP, 0.66mmHg in diastolic BP and 0.82mmHg in mean arterial pressure. ACE hypomethylation mediated 11.78% (P=0.03) of the elevated ACE protein by PM2.5. Increased ACE protein accounted for 3.90~13.44% (P=0.35~0.68) of the elevated BP by PM2.5. Repetitive-element methylation was also decreased but did not significantly mediate the association between PM2.5 and BP.
CONCLUSIONS	This investigation provided strong evidence that short-term exposure to PM2.5 was significantly associated with BP, ACE protein and ACE methylation. Our findings highlighted a possible involvement of ACE and ACE methylation in the effects of PM2.5 on elevating BP.

표-453. PubMed 논문번호 27399797의 내용 요약

구분	내용
PubMed ID	27399797
TITLE	High blood pressure: prevalence and adherence to guidelines in a population-based cohort.
JOURNAL	Swiss medical weekly: 10.4414/smw.2016.14323
AUTHORS	Walther Diana, Curjuric Ivan, Dratva Julia, Schaffner Emmanuel, Quinto Carlos, Rochat Thierry, Gaspoz Jean-Michel, Burdet Luc, Bridevaux Pierre-Olivier, Pons Marco, Gerbase Margaret W, Schindler Christian, Probst-Hensch Nicole
QUESTIONS UNDER STUDY	High blood pressure, the single leading health risk factor worldwide, contributes greatly to morbidity and mortality. This study aimed to add to the understanding of diagnosed and undiagnosed high blood pressure in Switzerland and to evaluate adherence to hypertension guidelines.
METHODS	Included were 3962 participants from the first (2001-2003) and second (2010-2011) follow-ups of the population-based Swiss Cohort Study on Air Pollution and Lung and Heart Disease in Adults. High blood pressure was defined as blood pressure ≥140/90 mm Hg and the prevalence of doctor-diagnosed hypertension was based on questionnaire information.
RESULTS	High blood pressure was found in 34.9% of subjects, 49.1% of whom were unaware of this condition; 30.0% had doctor-diagnosed hypertension and, although 82.1% of these received drug treatments, in only 40.8% was blood pressure controlled (<140/90 mm Hg). Substantial first-line beta-blocker use and nonadherence to comorbidity-specific prescription guidelines were observed and remained mostly unexplained. Age-adjusted rates of unawareness and uncontrolled hypertension were more than 20% higher than in the USA.
CONCLUSIONS	There is room for improvement in managing hypertension in Switzerland. Population-based observational studies are essential for identifying and evaluating unmet needs in healthcare; however, to pinpoint the underlying causes it is imperative to facilitate linkage of cohort data to medical records.

표-454. PubMed 논문번호 27443553의 내용 요약

구분	내용
PubMed ID	27443553
TITLE	Distinguishing the associations between daily mortality and hospital admissions and nitrogen dioxide from those of particulate matter: a systematic review and meta-analysis.
JOURNAL	BMJ open: 10.1136/bmjopen-2015-010751
AUTHORS	Mills I C, Atkinson R W, Anderson H R, Maynard R L, Strachan D P
OBJECTIVES	To quantitatively assess time-series studies of daily nitrogen dioxide (NO2) and mortality and hospital admissions which also controlled for particulate matter (PM) to determine whether or to what extent the NO2 associations are independent of PM.
DESIGN	A systematic review and meta-analysis.
METHODS	Time-series studies-published in peer-reviewed journals worldwide, up to May 2011-that reported both single-pollutant and two-pollutant model estimates for NO2 and PM were ascertained from bibliographic databases (PubMed, EMBASE and Web of Science) and reviews. Random-effects summary estimates were calculated globally and stratified by different geographical regions, and effect modification was investigated.
OUTCOME MEASURES	Mortality and hospital admissions for various cardiovascular or respiratory diseases in different age groups in the general population.
RESULTS	60 eligible studies were identified, and meta-analysis was conducted on 23 outcomes. Two-pollutant model study estimates generally showed that the NO2 associations were independent of PM mass. For all-cause mortality, a 10 □g/m(3) increase in 24-hour NO2 was associated with a 0.78% (95% CI 0.47% to 1.09%) increase in the risk of death, which reduced to 0.60% (0.33% to 0.87%) after control for PM. Heterogeneity between geographical region-specific estimates was removed by control for PM (I(2) from 66.9% to 0%). Estimates of PM and daily mortality assembled from the same studies were greatly attenuated after control for NO2: from 0.51% (0.29% to 0.74%) to 0.18% (-0.11% to 0.47%) per 10 □g/m(3) PM10 and 0.74% (0.34% to 1.14%) to 0.54% (-0.25% to 1.34%) for PM2.5.
CONCLUSIONS	The association between short-term exposure to NO2 and adverse health outcomes is largely independent of PM mass. Further studies should attempt to investigate whether this is a generic PM effect or whether it is modified by the source and physicochemical characteristics of PM. This finding strengthens the argument for NO2 having a causal role in health effects.

표-455. PubMed 논문번호 27460097의 내용 요약

구분	내용
PubMed ID	27460097
TITLE	Associations between microvascular function and short-term exposure to traffic-related air pollution and particulate matter oxidative potential.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-016-0157-5
AUTHORS	Zhang Xian, Staimer Norbert, Tjoa Tomas, Gillen Daniel L, Schauer James J, Shafer Martin M, Hasheminassab Sina, Pakbin Payam, Longhurst John, Sioutas Constantinos, Delfino Ralph J
BACKGROUND	Short-term exposure to ambient air pollution has been associated with acute increases in cardiovascular hospitalization and mortality. However, causative chemical components and underlying pathophysiological mechanisms remain to be clarified. We hypothesized that endothelial dysfunction would be associated with mobile-source (traffic) air pollution and that pollutant components with higher oxidative potential to generate reactive oxygen species (ROS) would have stronger associations.
METHODS	We carried out a cohort panel study in 93 elderly non-smoking adults living in the Los Angeles metropolitan area, during July 2012-February 2014. Microvascular function, represented by reactive hyperemia index (RHI), was measured weekly for up to 12 weeks (N = 845). Air pollutant data included daily data from regional air-monitoring stations, five-day average PM chemical components and oxidative potential in three PM size-fractions, and weekly personal nitrogen oxides (NOx). Linear mixed-effect models estimated adjusted changes in microvascular function with exposure.
RESULTS	RHI was inversely associated with traffic-related pollutants such as ambient PM2.5 black carbon (BC), NOx, and carbon monoxide (CO). An interquartile range change increase (1.06 μg/m(3)) in 5-day average BC was associated with decreased RHI, -0.093 (95 % CI: -0.151, -0.035). RHI was inversely associated with other mobile-source components/tracers (polycyclic aromatic hydrocarbons, elemental carbon, and hopanes), and PM oxidative potential as quantified in two independent assays (dithiothreitol and in vitro macrophage ROS) in accumulation and ultrafine PM, and transition metals.
CONCLUSIONS	Our findings suggest that short-term exposures to traffic-related air pollutants with high oxidative potential are major components contributing to microvascular dysfunction.

표-456. PubMed 논문번호 27468926의 내용 요약

구분	내용
PubMed ID	27468926
TITLE	Ambient Air Pollution Is Associated With the Severity of Coronary Atherosclerosis and Incident Myocardial Infarction in Patients Undergoing Elective Cardiac Evaluation.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.116.003947
AUTHORS	Hartiala Jaana, Breton Carrie V, Tang W H Wilson, Lurmann Frederick, Hazen Stanley L, Gilliland Frank D, Allayee Hooman
BACKGROUND	The effect of air pollution exposure on atherosclerosis severity or incident clinical events in patients with coronary artery disease is not known.
METHODS AND RESULTS	We conducted a prospective longitudinal cohort study of 6575 Ohio residents undergoing elective diagnostic coronary angiography. Multinomial regression and Cox proportional hazards models were used to assess the relationship between exposure to fine particulate matter <2.5 μm in diameter (PM2.5) and nitrogen dioxide on coronary artery disease severity at baseline and risk of myocardial infarction, stroke, or all-cause mortality over 3 years of follow-up. Among participants with coronary artery disease, exposure to PM2.5 levels was associated with increased likelihood of having coronary atherosclerosis that was mild (odds ratio 1.43, 95% CI 1.11-1.83, P=0.005) and severe (odds ratio 1.63, 95% CI 1.26-2.11, P<0.0001), with the effect on severe coronary artery disease being significantly increased compared with mild disease (Ptrend=0.03). Exposure to higher PM2.5 levels was also significantly associated with increased risk of incident myocardial infarction (hazard ratio 1.33, 95% CI 1.02-1.73, P=0.03) but not stroke or all-cause mortality. The association of PM2.5 with incident myocardial infarction was not affected after adjustment for Framingham Adult Treatment Panel III (ATP III) risk score or statin therapy. In comparison, there were no significant associations between nitrogen dioxide levels and all-cause mortality or risk of stroke after adjustment for Framingham ATP III risk score.
CONCLUSIONS	Exposure to PM2.5 increased the likelihood of having severe coronary artery disease and the risk of incident myocardial infarction among patients undergoing elective cardiac evaluation. These results suggest that ambient air pollution exposure may be a modifiable risk factor for risk of myocardial infarction in a highly susceptible patient population.

표-457. PubMed 논문번호 27472911의 내용 요약

구분	내용
PubMed ID	27472911
TITLE	Long-Term Exposure to Traffic-Related Air Pollution and Risk of Incident Atrial Fibrillation: A Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP392
AUTHORS	Monrad Maria, Sajadieh Ahmad, Christensen Jeppe Schultz, Ketzel Matthias, Raaschou-Nielsen Ole, Tjønneland Anne, Overvad Kim, Loft Steffen, Sørensen Mette, Monrad Maria, Sajadieh Ahmad, Christensen Jeppe Schultz, Ketzel Matthias, Raaschou-Nielsen Ole, Tjønneland Anne, Overvad Kim, Loft Steffen, Sørensen Mette
BACKGROUND	Atrial fibrillation is the most common sustained arrhythmia and is associated with cardiovascular morbidity and mortality. The few studies conducted on short-term effects of air pollution on episodes of atrial fibrillation indicate a positive association, though not consistently.
OBJECTIVES	The aim of this study was to evaluate the long-term impact of traffic-related air pollution on incidence of atrial fibrillation in the general population.
METHODS	In the Danish Diet, Cancer, and Health cohort of 57,053 people 50-64 years old at enrollment in 1993-1997, we identified 2,700 cases of first-ever hospital admission for atrial fibrillation from enrollment to end of follow-up in 2011. For all cohort members, exposure to traffic-related air pollution assessed as nitrogen dioxide (NO2) and nitrogen oxides (NOx) was estimated at all present and past residential addresses from 1984 to 2011 using a validated dispersion model. We used Cox proportional hazard model to estimate associations between long-term residential exposure to NO2 and NOx and risk of atrial fibrillation, after adjusting for lifestyle and socioeconomic position.
RESULTS	A 10 μg/m3 higher 10-year time-weighted mean exposure to NO2 preceding diagnosis was associated with an 8% higher risk of atrial fibrillation [incidence rate ratio: 1.08; 95% confidence interval (CI): 1.01, 1.14] in adjusted analysis. Though weaker, similar results were obtained for long-term residential exposure to NOx. We found no clear tendencies regarding effect modification of the association between NO2 and atrial fibrillation by sex, smoking, hypertension or myocardial infarction.
CONCLUSION	We found long-term residential traffic-related air pollution to be associated with higher risk of atrial fibrillation. Accordingly, the present findings lend further support to the demand for abatement of air pollution. Citation: Monrad M, Sajadieh A, Christensen JS, Ketzel M, Raaschou-Nielsen O, Tjønneland A, Overvad K, Loft S, Sørensen M. 2017. Long-term exposure to traffic-related air pollution and risk of incident atrial fibrillation: a cohort study. Environ Health Perspect 125:422-427; http://dx.doi.org/10.1289/EHP392.

표-458. PubMed 논문번호 27500855의 내용 요약

구분	내용
PubMed ID	27500855
TITLE	Short-term effects of air temperature on plasma metabolite concentrations in patients undergoing cardiac catheterization.
JOURNAL	Environmental research: 10.1016/j.envres.2016.07.010
AUTHORS	Hampel Regina, Breitner Susanne, Kraus William E, Hauser Elizabeth, Shah Svati, Ward-Caviness Cavin K, Devlin Robert, Diaz-Sanchez David, Neas Lucas, Cascio Wayne, Peters Annette, Schneider Alexandra
BACKGROUND	Epidemiological studies have shown associations between air temperature and cardiovascular health outcomes. Metabolic dysregulation might also play a role in the development of cardiovascular disease.
OBJECTIVES	To investigate short-term temperature effects on metabolites related to cardiovascular disease.
METHODS	Concentrations of 45 acylcarnitines, 15 amino acids, ketone bodies and total free fatty acids were available in 2869 participants from the CATHeterization GENetics cohort recruited at the Duke University Cardiac Catheterization Clinic (Durham, NC) between 2001 and 2007. Ten metabolites were selected based on quality criteria and cluster analysis. Daily averages of meteorological variables were obtained from the North American Regional Reanalysis project. Immediate, lagged, and cumulative temperature effects on metabolite concentrations were analyzed using (piecewise) linear regression models.
RESULTS	Linear temperature effects were found for glycine, C16-OH:C14:1-DC, and aspartic acid/asparagine. A 5° C increase in temperature was associated with a 1.8% [95%-confidence interval: 0.3%; 3.3%] increase in glycine (5-day average), a 3.2% [0.1%; 6.3%] increase in C16-OH:C14:1-DC (lag of four days), and a -1.4% [-2.4%; -0.3%] decrease in aspartic acid/asparagine (lag of two days). Non-linear temperature effects were observed for alanine and total ketone bodies with breakpoint of 4° C and 20° C, respectively. Both a 5° C decrease in temperature on colder days (<4° C)and a 5° C increase in temperature on warmer days (≥4° C) were associated with a four day delayed increase in alanine by 6.6% [11.7; 1.8%] and 1.9% [0.3%; 3.4%], respectively. For ketone bodies we found immediate (0-day lag) increases of 4.2% [-0.5%; 9.1%] and 12.3% [0.1%; 26.0%] associated with 5° C decreases on colder (<20° C) days and 5° C increases on warmer days (≥20° C), respectively.
CONCLUSIONS	We observed multiple effects of air temperature on metabolites several of which are reported to be involved in cardiovascular disease. Our findings might help to understand the link between air temperature and cardiovascular disease.

표-459. PubMed 논문번호 27541680의 내용 요약

구분	내용
PubMed ID	27541680
TITLE	Air pollution and ST-elevation myocardial infarction: A case-crossover study of the Belgian STEMI registry 2009-2013.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2016.07.191
AUTHORS	Argacha J F, Collart P, Wauters A, Kayaert P, Lochy S, Schoors D, Sonck J, de Vos T, Forton M, Brasseur O, Beauloye C, Gevaert S, Evrard P, Coppieters Y, Sinnaeve P, Claeys M J
BACKGROUND	Previous studies have shown that air pollution particulate matter (PM) is associated with an increased risk for myocardial infarction. The effects of air pollution on the risk of ST-elevation myocardial infarction (STEMI), in particular the role of gaseous air pollutants such as NO2 and O3 and the susceptibility of specific populations, are still under debate.
METHODS	All patients entered in the Belgian prospective STEMI registry between 2009 and 2013 were included. Based on a validated spatial interpolation model from the Belgian Environment Agency, a national index was used to address the background level of air pollution exposure of Belgian population. A time-stratified and temperature-matched case-crossover analysis of the risk of STEMI was performed.
RESULTS	A total of 11,428 STEMI patients were included in the study. Each 10 μg/m3 increase in PM10, PM2.5 and NO2 was associated with an increased odds ratio (ORs) of STEMI of 1.026 (CI 95%: 1.005-1.048), 1.028 (CI 95%: 1.003-1.054) and 1.051 (CI 95%: 1.018-1.084), respectively. No effect of O3 was found. STEMI was associated with PM10 exposure in patients ≥75y.o. (OR: 1.046, CI 95%: 1.002-1.092) and with NO2 in patients ≤54y.o. (OR: 1.071, CI 95%: 1.010-1.136). No effect of air pollution on cardiac arrest or in-hospital STEMI mortality was found.
CONCLUSION	PM2.5 and NO2 exposures incrementally increase the risk of STEMI. The risk related to PM appears to be greater in the elderly, while younger patients appear to be more susceptible to NO2 exposure.

표-460. PubMed 논문번호 27562361의 내용 요약

구분	내용
PubMed ID	27562361
TITLE	Cardiovascular Benefits of Wearing Particulate-Filtering Respirators: A Randomized Crossover Trial.
JOURNAL	Environmental health perspectives: 10.1289/EHP73
AUTHORS	Shi Jingjin, Lin Zhijing, Chen Renjie, Wang Cuicui, Yang Changyuan, Cai Jing, Lin Jingyu, Xu Xiaohui, Ross Jennifer A, Zhao Zhuohui, Kan Haidong, Shi Jingjin, Lin Zhijing, Chen Renjie, Wang Cuicui, Yang Changyuan, Cai Jing, Lin Jingyu, Xu Xiaohui, Ross Jennifer A, Zhao Zhuohui, Kan Haidong
BACKGROUND	Practical approaches to protect individuals from ambient particulate matter (PM) are urgently needed in developing countries. Evidence on the health benefits of wearing particulate-filtering respirators is limited.
OBJECTIVES	We evaluated the short-term cardiovascular health effects of wearing respirators in China.
METHODS	A randomized crossover trial was performed in 24 healthy young adults in Shanghai, China in 2014. The subjects were randomized into two groups and wore particulate-filtering respirators for 48 hr alternating with a 3-week washout interval. Heart rate variability (HRV) and ambulatory blood pressure (BP) were continuously monitored during the 2nd 24 hr in each intervention. Circulating biomarkers were measured at the end of each intervention. Linear mixed-effect models were applied to evaluate the effects of wearing respirators on health outcomes.
RESULTS	During the intervention periods, the mean daily average concentration of PM with an aerodynamic diameter < 2.5 μm (PM2.5) was 74.2 μg/m3. Compared with the absence of respirators, wearing respirators was associated with a decrease of 2.7 mmHg [95% confidence interval (CI): 0.1, 5.2 mmHg] in systolic BP and increases of HRV parameters, including 12.5% (95% CI: 3.8%, 21.2%) in high frequency (HF) power, 10.9% (95% CI: 1.8%, 20.0%) in the root mean square of the successive differences, and 22.1% (95% CI: 3.6%, 40.7%) in the percentage of normal RR intervals with duration > 50 msec different from the previous normal RR interval (pNN50). The presence of respirators was also associated with a decrease of 7.8% (95% CI: 3.5%, 12.1%) in the ratio of low frequency (LF)/HF power.
CONCLUSIONS	Short-term wearing of particulate-filtering respirators may produce cardiovascular benefits by improving autonomic nervous function and reducing BP. Citation: Shi J, Lin Z, Chen R, Wang C, Yang C, Cai J, Lin J, Xu X, Ross JA, Zhao Z, Kan H. 2017. Cardiovascular benefits of wearing particulate-filtering respirators: a randomized crossover trial. Environ Health Perspect 125:175-180; http://dx.doi.org/10.1289/EHP73.

표-461. PubMed 논문번호 27590407의 내용 요약

구분	내용
PubMed ID	27590407
TITLE	Association between air pollution and development of atrial fibrillation: A meta-analysis of observational studies.
JOURNAL	Heart & lung : the journal of critical care: 10.1016/j.hrtlng.2016.08.001
AUTHORS	Shao Qingmiao, Liu Tong, Korantzopoulos Panagiotis, Zhang Zhiwei, Zhao Jianping, Li Guangping
BACKGROUND	Current evidence suggests that gaseous or particulate pollutants may increase the risk of atrial fibrillation (AF), although this association is still uncertain.
METHODS	We conducted a systematic review of literature using PubMed, Ovid, Embase and Web of Science to identify studies reporting on the association between gaseous (ozone, carbon monoxide, sulfur dioxide, and nitrogen dioxide) or particulate matter 2.5 (PM2.5) and AF risk published until March 2015. The overall effect estimate was presented as the population-attributable risks with 95% CI. We used both fix-effects and random effects models to calculate the overall effect estimate.
RESULTS	We retrieved 4 studies, involving 461,441 participants. There was a statistically significant association between AF development and all gaseous pollutant as well as PM2.5 [NO: 1.19% (0.70-1.67%), CO: 0.60 (0.20-1.09), SO2: 0.90 (0.60-1.28), O3: 1.09 (0.20-1.86), PM2.5: 0.89 (0.20-1.57)].
CONCLUSIONS	Our comprehensive meta-analysis suggests that gaseous or particulate pollutants are associated with the increased risk of AF.

표-462. PubMed 논문번호 27625155의 내용 요약

구분	내용
PubMed ID	27625155
TITLE	Long-term exposure to traffic-related air pollution and cancer among survivors of myocardial infarction: A 20-year follow-up study.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487316669415
AUTHORS	Cohen Gali, Levy Ilan, Yuval Technion Center of Excellence in Exposure Science and Environmental Health, Technion Israel Institute of Technology, Israel, Kark Jeremy D, Levin Noam, Broday David M, Steinberg David M, Gerber Yariv, Cohen Gali, Levy Ilan, Yuval Technion Center of Excellence in Exposure Science and Environmental Health, Technion Israel Institute of Technology, Israel, Kark Jeremy D, Levin Noam, Broday David M, Steinberg David M, Gerber Yariv
BACKGROUND	Previous studies suggested a carcinogenic effect of exposure to traffic-related air pollution. Recently, higher rates of cancer incidence were observed among myocardial infarction survivors compared with the general population. We examined the association between chronic exposure to nitrogen oxides, a proxy measure for traffic-related air pollution, and cancer incidence and mortality in a cohort of myocardial infarction patients.
METHODS	Patients aged ≤65 years admitted to hospital in central Israel with a first myocardial infarction in 1992-1993 were followed to 2013 for cancer incidence and cause-specific mortality. Data on sociodemographic and cancer risk factors were obtained, including time-varying information on smoking. Using land use regression models, annual averages of nitrogen oxides during follow-up were estimated individually according to home addresses. Cox proportional hazards models were constructed to study the relationships with cancer outcomes.
RESULTS	During a mean follow-up of 16 (SD 7) years, 262 incident cancers and 105 cancer deaths were identified among 1393 cancer-free patients at baseline (mean age 54 years; 81% men). In adjusted models, a 10 ppb increase in mean nitrogen oxide exposure was associated with a hazard ratio (HR) of 1.06 (95% confidence interval (CI) 0.96-1.18) for cancer incidence and HR of 1.08 (95% CI 0.93-1.26) for cancer mortality. The association with lung, bladder, kidney or prostate cancer (previously linked to air pollution) was stronger (HR 1.16; 95% CI 1.00-1.33).
CONCLUSIONS	Chronic exposure to traffic-related air pollution may constitute an environmental risk factor for cancer post-myocardial infarction. Variation in the strength of association between specific cancers needs to be explored further.

표-463. PubMed 논문번호 27639206의 내용 요약

구분	내용
PubMed ID	27639206
TITLE	Mortality burden of ambient fine particulate air pollution in six Chinese cities: Results from the Pearl River Delta study.
JOURNAL	Environment international: 10.1016/j.envint.2016.09.007
AUTHORS	Lin Hualiang, Liu Tao, Xiao Jianpeng, Zeng Weilin, Li Xing, Guo Lingchuan, Zhang Yonghui, Xu Yanjun, Tao Jun, Xian Hong, Syberg Kevin M, Qian Zhengmin Min, Ma Wenjun
BACKGROUND	Epidemiological studies have reported significant association between ambient fine particulate matter air pollution (PM2.5) and mortality, however, few studies have investigated the relationship of mortality with PM2.5 and associated mortality burden in China, especially in a multicity setting.
METHODS	We investigated the PM2.5-mortality association in six cities of the Pearl River Delta region from 2013 to 2015. We used generalized additive Poisson models incorporating penalized smoothing splines to control for temporal trend, temperature, and relative humidity. We applied meta-analyses using random-effects models to pool the effect estimates in the six cities. We also examined these associations in stratified analyses by sex, age group, education level and location of death. We further estimated the mortality burden (attributable fraction and attributable mortality) due to ambient PM2.5 exposures.
RESULTS	During the study period, a total of 316,305 deaths were recorded in the study area. The analysis revealed a significant association between PM2.5 and mortality. Specifically, a 10 μg/m3 increase in 4-day averaged (lag03) PM2.5 concentration corresponded to a 1.76% (95% confidence interval (CI): 1.47%, 2.06%) increase in total mortality, 2.19% (95% CI: 1.80%, 2.59%) in cardiovascular mortality, and 1.68% (95% CI: 1.00%, 2.37%) in respiratory mortality. The results were generally robust to model specifications and adjustment of gaseous air pollutants. We estimated that 0.56% (95% CI: 0.47%, 0.66%) and 3.79% (95% CI: 3.14%, 4.45%) of all-cause mortalities were attributable to PM2.5 using China's and WHO's air quality standards as the reference, corresponding to 1661 (95% CI: 1379, 1946) and 11,176 (95% CI: 9261, 13,120) attributable premature mortalities, respectively.
CONCLUSION	This analysis adds to the growing body of evidence linking PM2.5 with daily mortality, and mortality burdens, particularly in one Chinese region with high levels of air pollution.

표-464. PubMed 논문번호 27644030의 내용 요약

구분	내용
PubMed ID	27644030
TITLE	Spatial variation in nitrogen dioxide concentrations and cardiopulmonary hospital admissions.
JOURNAL	Environmental research: 10.1016/j.envres.2016.09.008
AUTHORS	Dijkema Marieke B A, van Strien Robert T, van der Zee Saskia C, Mallant Sanne F, Fischer Paul, Hoek Gerard, Brunekreef Bert, Gehring Ulrike
BACKGROUND	Air pollution episodes are associated with increased cardiopulmonary hospital admissions. Cohort studies showed associations of spatial variation in traffic-related air pollution with respiratory and cardiovascular mortality. Much less is known in particular about associations with cardiovascular morbidity. We explored the relation between spatial variation in nitrogen dioxide (NO2) concentrations and cardiopulmonary hospital admissions.
METHODS	This ecological study was based on hospital admissions data (2001-2004) from the National Medical Registration and general population data for the West of the Netherlands (population 4.04 million). At the 4-digit postcode area level (n=683) associations between modeled annual average outdoor NO2 concentrations and hospital admissions for respiratory and cardiovascular causes were evaluated by linear regression with the log of the postcode-specific percentage of subjects that have been admitted at least once during the study period as the dependent variable. All analyses were adjusted for differences in composition of the population of the postcode areas (age, sex, income).
RESULTS	At the postcode level, positive associations were found between outdoor NO2 concentrations and hospital admission rates for asthma, chronic obstructive pulmonary disease (COPD), all cardiovascular causes, ischemic heart disease and stroke (e.g. adjusted relative risk (95% confidence interval) for the second to fourth quartile relative to the first quartile of exposure were 1.87 (1.46-2.40), 2.34 (1.83-3.01) and 2.81 (2.16-3.65) for asthma; 1.44 (1.19-1.74), 1.50 (1.24-1.82) and 1.60 (1.31-1.96) for COPD). Associations remained after additional (indirect) adjustment for smoking (COPD admission rate) and degree of urbanization.
CONCLUSIONS	Our study suggests an increased risk of hospitalization for respiratory and cardiovascular causes in areas with higher levels of NO2. Our findings add to the currently limited evidence of a long-term effect of air pollution on hospitalization. The ecological design of our study is a limitation and more studies with individual data are needed to confirm our findings.

표-465. PubMed 논문번호 27648591의 내용 요약

구분	내용
PubMed ID	27648591
TITLE	Impact of Road Traffic Pollution on Pre-eclampsia and Pregnancy-induced Hypertensive Disorders.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000555
AUTHORS	Pedersen Marie, Halldorsson Thorhallur I, Olsen Sjurdur F, Hjortebjerg Dorrit, Ketzel Matthias, Grandstr□m Charlotta, Raaschou-Nielsen Ole, S ø rensen Mette
BACKGROUND	Road traffic is a major source of air pollution and noise. Both exposures have been associated with hypertension in adults, but pregnant women have been less studied.
METHODS	We examined single and joint effects of ambient air pollution and road traffic noise on pre-eclampsia and pregnancy-induced hypertensive disorders among 72,745 singleton pregnancies (1997-2002) from the Danish National Birth Cohort with complete covariate data and residential address history from conception until live born birth. Nitrogen dioxide (NO2) and noise from road traffic (Lden) were modeled at all addresses. Outcome and covariate data were derived from registries, hospital records, and questionnaires.
RESULTS	A 10-□g/m increase in NO2 exposure during first trimester was associated with increased risk of pre-eclampsia (n = 1,880, adjusted odds ratio = 1.07 [95% confidence interval = 1.01, 1.14]) and pregnancy-induced hypertensive disorders (n = 2,430, adjusted odds ratio = 1.07 [1.01, 1.13]). A 10 dB higher road traffic noise was also associated with increased risk of pre-eclampsia (1.10 [1.02, 1.18]) and pregnancy-induced hypertensive disorders (1.08 [1.02, 1.15]). For both exposures, the associations were strongest for mild pre-eclampsia (n = 1,393) and early-onset pre-eclampsia (n = 671), whereas higher risk for severe pre-eclampsia (n = 487) was not evident. In mutually adjusted models, estimates for both exposures decreased and only the association between NO2 and mild pre-eclampsia remained.
CONCLUSIONS	Road traffic may increase the risk of pre-eclampsia and hypertensive disorders in pregnancy through exposure to both ambient air pollution and noise, although associations with the two exposures were generally not found to be independent of one another. See video abstract, http://links.lww.com/EDE/B112.

표-466. PubMed 논문번호 27685325의 내용 요약

구분	내용
PubMed ID	27685325
TITLE	Associations of Novel and Traditional Vascular Biomarkers of Arterial Stiffness: Results of the SAPALDIA 3 Cohort Study.
JOURNAL	PloS one: 10.1371/journal.pone.0163844
AUTHORS	Endes Simon, Caviezel Seraina, Schaffner Emmanuel, Dratva Julia, Schindler Christian, K□nzli Nino, Bachler Martin, Wassertheurer Siegfried, Probst-Hensch Nicole, Schmidt-Trucks□ss Arno
BACKGROUND AND OBJECTIVES	There is a lack of evidence concerning associations between novel parameters of arterial stiffness as cardiovascular risk markers and traditional structural and functional vascular biomarkers in a population-based Caucasian cohort. We examined these associations in the second follow-up of the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases in Adults (SAPALDIA 3).
METHODS	Arterial stiffness was measured oscillometrically by pulse wave analysis to derive the cardio-ankle vascular index (CAVI), brachial-ankle (baPWV) and aortic pulse wave velocity (aPWV), and amplitude of the forward and backward wave. Carotid ultrasonography was used to measure carotid intima-media thickness (cIMT) and carotid lumen diameter (LD), and to derive a distensibility coefficient (DC). We used multivariable linear regression models adjusted for several potential confounders for 2,733 people aged 50-81 years.
RESULTS	CAVI, aPWV and the amplitude of the forward and backward wave were significant predictors of cIMT ($p < 0.001$). All parameters were significantly associated with LD ($p < 0.001$), with aPWV and the amplitude of the forward wave explaining the highest proportion of variance (2%). Only CAVI and baPWV were significant predictors of DC ($p < 0.001$), explaining more than 0.3% of the DC variance.
CONCLUSION	We demonstrated that novel non-invasive oscillometric arterial stiffness parameters are differentially associated with specific established structural and functional local stiffness parameters. Longitudinal studies are needed to follow-up on these cross-sectional findings and to evaluate their relevance for clinical phenotypes.

표-467. PubMed 논문번호 27688985의 내용 요약

구분	내용
PubMed ID	27688985
TITLE	Cardiovascular Disease Death Before Age 65 in 168 Countries Correlated Statistically with Biometrics, Socioeconomic Status, Tobacco, Gender, Exercise, Macronutrients, and Vitamin K.
JOURNAL	Cureus: 10.7759/cureus.748
AUTHORS	Cundiff David K, Agutter Paul S
BACKGROUND	Nutrition researchers recently recognized that deficiency of vitamin K2 (menaquinone: MK-4-MK-13) is widespread and contributes to cardiovascular disease (CVD). The deficiency of vitamin K2 or vitamin K inhibition with warfarin leads to calcium deposition in the arterial blood vessels.
METHODS	Using publicly available sources, we collected food commodity availability data and derived nutrient profiles including vitamin K2 for people from 168 countries. We also collected female and male cohort data on early death from CVD (ages 15-64 years), insufficient physical activity, tobacco, biometric CVD risk markers, socioeconomic risk factors for CVD, and gender. The outcome measures included (1) univariate correlations of early death from CVD with each risk factor, (2) a multiple regression-derived formula relating early death from CVD (dependent variable) to macronutrient profile, vitamin K1 and K2 and other risk factors (independent variables), (3) for each risk factor appearing in the multiple regression formula, the portion of CVD risk attributable to that factor, and (4) similar univariate and multivariate analyses of body mass index (BMI), fasting blood sugar (FBS) (simulated from diabetes prevalence), systolic blood pressure (SBP), and cholesterol/ HDL-C ratio (simulated from serum cholesterol) (dependent variables) and dietary and other risk factors (independent variables).
RESULTS	Female and male cohorts in countries that have vitamin K2 〈 5□g per 2000 kcal/day per capita (n = 70) had about 2.2 times the rate of early CVD deaths as people in countries with 〉 24 □g/day of vitamin K2 per 2000 kcal/day (n = 72). A multiple regression-derived formula relating early death from CVD to dietary nutrients and other risk factors accounted for about 50% of the variance between cohorts in early CVD death. The attributable risks of the variables in the CVD early death formula were: too much alcohol (0.38%), too little vitamin K2 (6.95%), tobacco (6.87%), high blood pressure (9.01%), air pollution (9.15%), early childhood death (3.64%), poverty (7.66%), and male gender (6.13%).
CONCLUSIONS	Worldwide dietary vitamin K2 data derived from food commodities add much understanding to the analysis of CVD risk factors and the etiology of CVD. Vitamin K2 in food products should be systematically quantified. Public health programs should be considered to increase the intake of vitamin K2-containing fermented plant foods such as sauerkraut, miso, and natto.

표-468. PubMed 논문번호 27692672의 내용 요약

구분	내용
PubMed ID	27692672
TITLE	Road traffic noise, blood pressure and heart rate: Pooled analyses of harmonized data from 88,336 participants.
JOURNAL	Environmental research: 10.1016/j.envres.2016.09.014
AUTHORS	Zijlema Wilma, Cai Yutong, Doiron Dany, Mbatchou St□phane, Fortier Isabel, Gulliver John, de Hoogh Kees, Morley David, Hodgson Susan, Elliott Paul, Key Timothy, Kongsgard Havard, Hveem Kristian, Gaye Amadou, Burton Paul, Hansell Anna, Stolk Ronald, Rosmalen Judith
INTRODUCTION	Exposure to road traffic noise may increase blood pressure and heart rate. It is unclear to what extent exposure to air pollution may influence this relationship. We investigated associations between noise, blood pressure and heart rate, with harmonized data from three European cohorts, while taking into account exposure to air pollution.
METHODS	Road traffic noise exposure was assessed using a European noise model based on the Common Noise Assessment Methods in Europe framework (CNOSSOS-EU). Exposure to air pollution was estimated using a European-wide land use regression model. Blood pressure and heart rate were obtained by trained clinical professionals. Pooled cross-sectional analyses of harmonized data were conducted at the individual level and with random-effects meta-analyses.
RESULTS	We analyzed data from 88,336 participants, across the three participating cohorts (mean age 47.0 (±13.9) years). Each 10dB(A) increase in noise was associated with a 0.93 (95% CI 0.76;1.11) bpm increase in heart rate, but with a decrease in blood pressure of 0.01 (95% CI -0.24;0.23) mmHg for systolic and 0.38 (95% CI -0.53; -0.24) mmHg for diastolic blood pressure. Adjustments for PM10 or NO2 attenuated the associations, but remained significant for DBP and HR. Results for BP differed by cohort, with negative associations with noise in LifeLines, no significant associations in EPIC-Oxford, and positive associations with noise >60dB(A) in HUNT3.
CONCLUSIONS	Our study suggests that road traffic noise may be related to increased heart rate. No consistent evidence for a relation between noise and blood pressure was found.

표-469. PubMed 논문번호 27712935의 내용 요약

구분	내용
PubMed ID	27712935
TITLE	Associations of night-time road traffic noise with carotid intima-media thickness and blood pressure: The Whitehall II and SABRE study cohorts.
JOURNAL	Environment international: 10.1016/j.envint.2016.09.023
AUTHORS	Halonen Jaana I, Dehbi Hakim-Moulay, Hansell Anna L, Gulliver John, Fecht Daniela, Blangiardo Marta, Kelly Frank J, Chaturvedi Nish, Kivim□ki Mika, Tonne Cathryn, Halonen Jaana I, Dehbi Hakim-Moulay, Hansell Anna L, Gulliver John, Fecht Daniela, Blangiardo Marta, Kelly Frank J, Chaturvedi Nish, Kivim□ki Mika, Tonne Cathryn
BACKGROUND	Road traffic noise has been linked to increased risk of stroke, for which hypertension and carotid intima-media thickness (cIMT) are risk factors. A link between traffic noise and hypertension has been established, but there are few studies on blood pressure and no studies on cIMT.
OBJECTIVES	To examine cross-sectional associations for long-term exposure to night-time noise with cIMT, systolic blood pressure (SBP), diastolic blood pressure (DBP) and hypertension.
METHODS	The study population consisted of 2592 adults from the Whitehall II and SABRE cohort studies living within Greater London who had cIMT, SBP and DBP measured. Exposure to night-time road traffic noise (A-weighted dB, referred to as dBA) was estimated at each participant's residential postcode centroid.
RESULTS	Mean night-time road noise levels were 52dBA (SD=4). In the pooled analysis adjusted for cohort, sex, age, ethnicity, marital status, smoking, area-level deprivation and NOx there was a 9.1 μm (95% CI: -7.1, 25.2) increase in cIMT in association with 10dBA increase in night-time noise. Analyses by noise categories of 55-60dBA (16.2 μm, 95% CI: -8.7, 41.2), and >60dBA (21.2 μm, 95% CI: -2.5, 44.9) vs. <55dBA were also positive but non-significant, expect among those not using antihypertensive medication and exposed to >60dBA vs. <55dBA (32.6 μm, 95% CI: 6.2, 59.0). Associations for SBP, DPB and hypertension were close to null.
CONCLUSIONS	After adjustments, including for air pollution, the association between night-time road traffic noise and cIMT was only observed among non-medication users but associations with blood pressure and hypertension were largely null.

표-470. PubMed 논문번호 27713110의 내용 요약

구분	내용
PubMed ID	27713110
TITLE	Associations between Environmental Quality and Mortality in the Contiguous United States, 2000-2005.
JOURNAL	Environmental health perspectives: 10.1289/EHP119
AUTHORS	Jian Yun, Messer Lynne C, Jagai Jyotsna S, Rappazzo Kristen M, Gray Christine L, Grabich Shannon C, Lobdell Danelle T, Jian Yun, Messer Lynne C, Jagai Jyotsna S, Rappazzo Kristen M, Gray Christine L, Grabich Shannon C, Lobdell Danelle T
BACKGROUND	Assessing cumulative effects of the multiple environmental factors influencing mortality remains a challenging task.
OBJECTIVES	This study aimed to examine the associations between cumulative environmental quality and all-cause and leading cause-specific (heart disease, cancer, and stroke) mortality rates.
METHODS	We used the overall Environmental Quality Index (EQI) and its five domain indices (air, water, land, built, and sociodemographic) to represent environmental exposure. Associations between the EQI and mortality rates (CDC WONDER) for counties in the contiguous United States (n = 3,109) were investigated using multiple linear regression models and random intercept and random slope hierarchical models. Urbanicity, climate, and a combination of the two were used to explore the spatial patterns in the associations.
RESULTS	We found 1 standard deviation increase in the overall EQI (worse environment) was associated with a mean 3.22% (95% CI: 2.80%, 3.64%) increase in all-cause mortality, a 0.54% (95% CI: -0.17%, 1.25%) increase in heart disease mortality, a 2.71% (95% CI: 2.21%, 3.22%) increase in cancer mortality, and a 2.25% (95% CI: 1.11%, 3.39%) increase in stroke mortality. Among the environmental domains, the associations ranged from -1.27% (95% CI: -1.70%, -0.84%) to 3.37% (95% CI: 2.90%, 3.84%) for all-cause mortality, -2.62% (95% CI: -3.52%, -1.73%) to 4.50% (95% CI: 3.73%, 5.27%) for heart disease mortality, -0.88% (95% CI: -2.12%, 0.36%) to 3.72% (95% CI: 2.38%, 5.06%) for stroke mortality, and -0.68% (95% CI: -1.19%, -0.18%) to 3.01% (95% CI: 2.46%, 3.56%) for cancer mortality. Air had the largest associations with all-cause, heart disease, and cancer mortality, whereas the sociodemographic index had the largest association with stroke mortality. Across the urbanicity gradient, no consistent trend was found. Across climate regions, the associations ranged from 2.29% (95% CI: 1.87%, 2.72%) to 5.30% (95% CI: 4.30%, 6.30%) for overall EQI, and larger associations were generally found in dry areas for both overall EQI and domain indices.
CONCLUSIONS	These results suggest that poor environmental quality, particularly poor air quality, was associated with increased mortality and that associations vary by urbanicity and climate region. Citation: Jian Y, Messer LC, Jagai JS, Rappazzo KM, Gray CL, Grabich SC, Lobdell DT. 2017. Associations between environmental quality and mortality in the contiguous United States, 2000-2005. Environ Health Perspect 125:355-362; http://dx.doi.org/10.1289/EHP119.

표-471. PubMed 논문번호 The Global Burden of Diseases, Injuries, and Risk Factors Study 2015 provides an up-to-date synthesis of the evidence for risk factor exposure and the attributable burden of disease. By providing national and subnational assessments spanning the past 25 years, this study can inform debates on the importance of addressing risks in context.의 내용 요약

구분	내용
BACKGROUND	The Global Burden of Diseases, Injuries, and Risk Factors Study 2015 provides an up-to-date synthesis of the evidence for risk factor exposure and the attributable burden of disease. By providing national and subnational assessments spanning the past 25 years, this study can inform debates on the importance of addressing risks in context.
METHODS	We used the comparative risk assessment framework developed for previous iterations of the Global Burden of Disease Study to estimate attributable deaths, disability-adjusted life-years (DALYs), and trends in exposure by age group, sex, year, and geography for 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks from 1990 to 2015. This study included 388 risk-outcome pairs that met World Cancer Research Fund-defined criteria for convincing or probable evidence. We extracted relative risk and exposure estimates from randomised controlled trials, cohorts, pooled cohorts, household surveys, census data, satellite data, and other sources. We used statistical models to pool data, adjust for bias, and incorporate covariates. We developed a metric that allows comparisons of exposure across risk factors-the summary exposure value. Using the counterfactual scenario of theoretical minimum risk level, we estimated the portion of deaths and DALYs that could be attributed to a given risk. We decomposed trends in attributable burden into contributions from population growth, population age structure, risk exposure, and risk-deleted cause-specific DALY rates. We characterised risk exposure in relation to a Socio-demographic Index (SDI).
FINDINGS	Between 1990 and 2015, global exposure to unsafe sanitation, household air pollution, childhood underweight, childhood stunting, and smoking each decreased by more than 25%. Global exposure for several occupational risks, high body-mass index (BMI), and drug use increased by more than 25% over the same period. All risks jointly evaluated in 2015 accounted for 57·8% (95% CI 56·6-58·8) of global deaths and 41·2% (39·8-42·8) of DALYs. In 2015, the ten largest contributors to global DALYs among Level 3 risks were high systolic blood pressure (211·8 million [192·7 million to 231·1 million] global DALYs), smoking (148·6 million [134·2 million to 163·1 million]), high fasting plasma glucose (143·1 million [125·1 million to 163·5 million]), high BMI (120·1 million [83·8 million to 158·4 million]), childhood undernutrition (113·3 million [103·9 million to 123·4 million]), ambient particulate matter (103·1 million [90·8 million to 115·1 million]), high total cholesterol (88·7 million [74·6 million to 105·7 million]), household air pollution (85·6 million [66·7 million to 106·1 million]), alcohol use (85·0 million [77·2 million to 93·0 million]), and diets high in sodium (83·0 million [49·3 million to 127·5 million]). From 1990 to 2015, attributable DALYs declined for micronutrient deficiencies, childhood undernutrition, unsafe sanitation and water, and household air pollution; reductions in risk-deleted DALY rates rather than reductions in exposure drove these declines. Rising exposure contributed to notable increases in attributable DALYs from high BMI, high fasting plasma glucose, occupational carcinogens, and drug use. Environmental risks and childhood undernutrition declined steadily with SDI; low physical activity, high BMI, and high fasting plasma glucose increased with SDI. In 119 countries, metabolic risks, such as high BMI and fasting plasma glucose, contributed the most attributable DALYs in 2015. Regionally, smoking still ranked among the leading five risk factors for attributable DALYs in 109 countries; childhood underweight and unsafe sex remained primary drivers of early death and disability in much of sub-Saharan Africa.

표-472. PubMed 논문번호 27740511의 내용 요약

구분	내용
PubMed ID	27740511
TITLE	Sex-Specific Associations between Particulate Matter Exposure and Gene Expression in Independent Discovery and Validation Cohorts of Middle-Aged Men and Women.
JOURNAL	Environmental health perspectives: 10.1289/EHP370
AUTHORS	Vrijens Karen, Winckelmans Ellen, Tsamou Maria, Baeyens Willy, De Boever Patrick, Jennen Danyel, de Kok Theo M, Den Hond Elly, Lefebvre Wouter, Plusquin Michelle, Reynders Hans, Schoeters Greet, Van Larebeke Nicolas, Vanpoucke Charlotte, Kleinjans Jos, Nawrot Tim S, Vrijens Karen, Winckelmans Ellen, Tsamou Maria, Baeyens Willy, De Boever Patrick, Jennen Danyel, de Kok Theo M, Den Hond Elly, Lefebvre Wouter, Plusquin Michelle, Reynders Hans, Schoeters Greet, Van Larebeke Nicolas, Vanpoucke Charlotte, Kleinjans Jos, Nawrot Tim S
BACKGROUND	Particulate matter (PM) exposure leads to premature death, mainly due to respiratory and cardiovascular diseases.
OBJECTIVES	Identification of transcriptomic biomarkers of air pollution exposure and effect in a healthy adult population.
METHODS	Microarray analyses were performed in 98 healthy volunteers (48 men, 50 women). The expression of eight sex-specific candidate biomarker genes (significantly associated with PM10 in the discovery cohort and with a reported link to air pollution-related disease) was measured with qPCR in an independent validation cohort (75 men, 94 women). Pathway analysis was performed using Gene Set Enrichment Analysis. Average daily PM2.5 and PM10 exposures over 2-years were estimated for each participant's residential address using spatiotemporal interpolation in combination with a dispersion model.
RESULTS	Average long-term PM10 was 25.9 (± 5.4) and 23.7 (± 2.3) μg/m3 in the discovery and validation cohorts, respectively. In discovery analysis, associations between PM10 and the expression of individual genes differed by sex. In the validation cohort, long-term PM10 was associated with the expression of DNAJB5 and EAPP in men and ARHGAP4 (p = 0.053) in women. AKAP6 and LIMK1 were significantly associated with PM10 in women, although associations differed in direction between the discovery and validation cohorts. Expression of the eight candidate genes in the discovery cohort differentiated between validation cohort participants with high versus low PM10 exposure (area under the receiver operating curve = 0.92; 95% CI: 0.85, 1.00; p = 0.0002 in men, 0.86; 95% CI: 0.76, 0.96; p = 0.004 in women).
CONCLUSIONS	Expression of the sex-specific candidate genes identified in the discovery population predicted PM10 exposure in an independent cohort of adults from the same area. Confirmation in other populations may further support this as a new approach for exposure assessment, and may contribute to the discovery of molecular mechanisms for PM-induced health effects.

표-473. PubMed 논문번호 27745901의 내용 요약

구분	내용
PubMed ID	27745901
TITLE	Ambient Air Pollution and the Risk of Central Retinal Artery Occlusion.
JOURNAL	Ophthalmology: 10.1016/j.ophtha.2016.08.046
AUTHORS	Cheng Hui-Chen, Pan Ren-Hao, Yeh Huan-Jui, Lai K Robert, Yen May-Yung, Chan Chien-Lung, Wang An-Guor
PURPOSE	To investigate whether daily changes in ambient air pollution were associated with an increased risk of central retinal artery occlusion (CRAO).
DESIGN	Retrospective population-based cohort study.
PARTICIPANTS	We identified patients newly diagnosed with CRAO between 2001 and 2013 in a representative database of 1 000 000 patients that were randomly selected from all registered beneficiaries of the National Health Insurance program in Taiwan. We identified air pollutant monitoring stations located near these patients' residences in different administrative areas in Taiwan to determine the recorded concentrations of particulate matter ≤2.5 μm (PM2.5), particulate matter ≤10 μm (PM10), nitrogen dioxide (NO2), sulfur dioxide (SO2), and ozone (O3). Patients without corresponding monitoring stations were excluded.
METHODS	We used a time-stratified case-crossover study design and conditional logistic regression analysis to assess associations between the risk of CRAO and the air pollutant levels in the days preceding each event.
MAIN OUTCOME MEASURES	Odds ratios (ORs) and 95% confidence intervals (CIs).
RESULTS	We enrolled 96 patients with CRAO in this study. The mean age was 65.6 years (standard deviation, 12.7 years) and 67.7% of patients were male. The risk of CRAO onset was significantly increased (OR, 1.09; 95% CI, 1.01-1.17; P = 0.03) during a 5-day period following a 1 part per billion increase in NO2 levels. After multipollutant adjustment, the increase in risk was most prominent after 4 days (OR, 1.40; 95% CI, 1.05-1.87; P = 0.02) to 5 days (OR, 2.16; 95% CI, 1.10-4.23; P = 0.03) of elevated NO2 levels in diabetic patients. The risk of CRAO onset also significantly increased in patients with hypertension and in patients ≥65 years old, after 1 day of elevated SO2 levels (OR, 1.88; 95% CI, 1.07-3.29; P = 0.03 and OR, 1.90; 95% CI, 1.13-3.21; P = 0.02, respectively). The transient concentration of the other air pollutants, including PM2.5, PM10, and O3, did not significantly affect the occurrence of CRAO in this study.
CONCLUSIONS	These results demonstrated a positive association between air pollution and CRAO onset, particularly in patients with diabetes or hypertension and those older than 65 years.

표-474. PubMed 논문번호 27763780의 내용 요약

구분	내용
PubMed ID	27763780
TITLE	Systemic effects of controlled exposure to diesel exhaust: a meta-analysis from randomized controlled trials.
JOURNAL	Annals of medicine: 10.1080/07853890.2016.1252054
AUTHORS	Vieira Jefferson Luis, Macedo Francisco Yuri, Benjo Alexandre Miguel, Guimar□es Guilherme V, Contreras Johanna Paola, Bocchi Edimar A, Vieira Jefferson Luis, Macedo Francisco Yuri, Benjo Alexandre Miguel, Guimar□es Guilherme V, Contreras Johanna Paola, Bocchi Edimar A
INTRODUCTION	Ambient air pollution is associated with adverse cardiovascular events. This meta-analysis aimed to investigate the short-term association between air pollution and cardiovascular effects on healthy volunteers.
METHODS	We searched databases to identify randomized trials with controlled human exposures to either of two models for studying ambient particulate matter: diesel-exhaust or concentrated ambient particles. Estimates of size effect were performed using standardized mean difference (SMD). Heterogeneity was assessed with I2 statistics. Outcomes were vascular function estimated by forearm blood flow (FBF), blood pressure, heart rate, and blood analysis.
RESULTS	Database searches yielded 17 articles (n = 342) with sufficient information for meta-analyses. High levels of heterogeneity for the some outcomes were analyzed using random-effects model. The pooled effect estimate showed that short-term exposure to air pollution impaired FBF response from 2.7 to 2.5 mL/100 mL tissue/min (SMD 0.404; p = .006). There was an increase in 5000 platelet/mm3 following pollution exposure (SMD 0.390; p = .050) but no significant differences for other outcomes.
CONCLUSION	Controlled human exposures to air pollution are associated with the surrogates of vascular dysfunction and increase in platelet count, which might be related to adverse cardiovascular events. Given the worldwide prevalence of exposure to air pollution, these findings are relevant for public health. KEY MESSAGES Controlled exposure to air pollution impairs vasomotor response, which is a surrogate for adverse cardiovascular events. This is the first meta-analysis from randomized clinical trials showing short-term association between air pollution and cardiovascular effects on healthy volunteers. Given the worldwide prevalence of exposure to air pollution, this finding is important for public health.

표-475. PubMed 논문번호 27765554의 내용 요약

구분	내용
PubMed ID	27765554
TITLE	Environmental Tobacco Smoke Exposure and Risk of Stroke in Never Smokers: An Updated Review with Meta-Analysis.
JOURNAL	Journal of stroke and cerebrovascular diseases : the official journal of National Stroke Association: 10.1016/j.jstrokecerebrovasdis.2016.09.011
AUTHORS	Lee Peter N, Thornton Alison J, Forey Barbara A, Hamling Jan S, Lee Peter N, Thornton Alison J, Forey Barbara A, Hamling Jan S
OBJECTIVES	The study aimed to review the epidemiological evidence relating environmental tobacco smoke exposure to stroke in never smokers.
METHODS	The study is similar to our review in 2006, with searches extended to March 2016.
RESULTS	Twelve further studies were identified. A total of 28 studies varied considerably in design, exposure indices used, and disease definition. Based on 39 sex-specific estimates and the exposure index current spousal exposure (or nearest equivalent), the meta-analysis gave an overall fixed-effect relative risk estimate of 1.23 (95% confidence interval: 1.16-1.31), with significant (P < .05) heterogeneity. There was no significant heterogeneity by sex, continent, fatality, disease end point, or degree of adjustment for potential confounding factors. Relative risks were less elevated in prospective studies (1.15, 1.06-1.24) than in case-control studies (1.44, 1.22-1.60) or cross-sectional studies (1.40, 1.21-1.61). They also varied by publication year, but with no trend. A significant increase was not seen in studies that excluded smokers of any tobacco (1.07, .97-1.17), but was seen for studies that included pipe- or cigar-only smokers, occasional smokers, or long-term former smokers. No elevation was seen for hemorrhagic stroke. Relative risk estimates were similar using ever rather than current exposure, or total rather than spousal exposure. Eleven studies provided dose-response estimates, the combined relative risk for the highest exposure level being 1.56 (1.37-1.79). Many studies have evident weaknesses, recall bias, and particularly publication bias being major concerns.
CONCLUSIONS	Although other reviewers inferred a causal relationship, we consider the evidence does not conclusively demonstrate this. We repeat our call for publication of data from existing large prospective studies.

표-476. PubMed 논문번호 27780829의 내용 요약

구분	내용
PubMed ID	27780829
TITLE	Exposure to Fine Particulate Air Pollution Is Associated With Endothelial Injury and Systemic Inflammation.
JOURNAL	Circulation research: 10.1161/CIRCRESAHA.116.309279
AUTHORS	Pope C Arden, Bhatnagar Aruni, McCracken James P, Abplanalp Wesley, Conklin Daniel J, O'Toole Timothy
RATIONALE	Epidemiological evidence indicates that exposures to fine particulate matter air pollution (PM2.5) contribute to global burden of disease, primarily as a result of increased risk of cardiovascular morbidity and mortality. However, mechanisms by which PM2.5 exposure induces cardiovascular injury remain unclear. PM2.5-induced endothelial dysfunction and systemic inflammation have been implicated, but direct evidence is lacking.
OBJECTIVE	To examine whether acute exposure to PM2.5 is associated with endothelial injury and systemic inflammation.
METHODS AND RESULTS	Blood was collected from healthy, nonsmoking, young adults during 3 study periods that included episodes of elevated PM2.5 levels. Microparticles and immune cells in blood were measured by flow cytometry, and plasma cytokine/growth factors were measured using multiplexing laser beads. PM2.5 exposure was associated with the elevated levels of endothelial microparticles (annexin V+/CD41-/CD31+), including subtypes expressing arterial-, venous-, and lung-specific markers, but not microparticles expressing CD62+. These changes were accompanied by suppressed circulating levels of proangiogenic growth factors (EGF [epidermal growth factor], sCD40L [soluble CD40 ligand], PDGF [platelet-derived growth factor], RANTES [regulated on activation, normal T-cell-expressed and secreted], GROα [growth-regulated protein α], and VEGF [vascular endothelial growth factor]), and an increase in the levels of antiangiogenic (TNFα [tumor necrosis factor α], IP-10 [interferon γ-induced protein 10]), and proinflammatory cytokines (MCP-1 [monocyte chemoattractant protein 1], MIP-1α/β [macrophage inflammatory protein 1α/β], IL-6 [interleukin 6], and IL-1β [interleukin 1β]), and markers of endothelial adhesion (sICAM-1 [soluble intercellular adhesion molecule 1] and sVCAM-1 [soluble vascular cellular adhesion molecule 1]). PM2.5 exposure was also associated with an inflammatory response characterized by elevated levels of circulating CD14+, CD16+, CD4+, and CD8+, but not CD19+ cells.
CONCLUSIONS	Episodic PM2.5 exposures are associated with increased endothelial cell apoptosis, an antiangiogenic plasma profile, and elevated levels of circulating monocytes and T, but not B, lymphocytes. These changes could contribute to the pathogenic sequelae of atherogenesis and acute coronary events.

표-477. PubMed 논문번호 27792908의 내용 요약

구분	내용
PubMed ID	27792908
TITLE	Associations among plasma metabolite levels and short-term exposure to PM〈sub〉2.5〈/sub〉 and ozone in a cardiac catheterization cohort.
JOURNAL	Environment international: 10.1016/j.envint.2016.10.012
AUTHORS	Breitner Susanne, Schneider Alexandra, Devlin Robert B, Ward-Caviness Cavin K, Diaz-Sanchez David, Neas Lucas M, Cascio Wayne E, Peters Annette, Hauser Elizabeth R, Shah Svati H, Kraus William E
RATIONALE	Exposure to ambient particulate matter (PM) and ozone has been associated with cardiovascular disease (CVD). However, the mechanisms linking PM and ozone exposure to CVD remain poorly understood.
OBJECTIVE	This study explored associations between short-term exposures to PM with a diameter 〈2.5 μm (PM2.5) and ozone with plasma metabolite concentrations.
METHODS AND RESULTS	We used cross-sectional data from a cardiac catheterization cohort at Duke University, North Carolina (NC), USA, accumulated between 2001 and 2007. Amino acids, acylcarnitines, ketones and total non-esterified fatty acid plasma concentrations were determined in fasting samples. Daily concentrations of PM2.5 and ozone were obtained from a Bayesian space-time hierarchical model, matched to each patient's residential address. Ten metabolites were selected for the analysis based on quality criteria and cluster analysis. Associations between metabolites and PM2.5 or ozone were analyzed using linear regression models adjusting for long-term trend and seasonality, calendar effects, meteorological parameters, and participant characteristics. We found delayed associations between PM2.5 or ozone and changes in metabolite levels of the glycine-ornithine-arginine metabolic axis and incomplete fatty acid oxidation associated with mitochondrial dysfunction. The strongest association was seen for an increase of 8.1 μg/m3 in PM2.5 with a lag of one day and decreased mean glycine concentrations (-2.5% [95% confidence interval: -3.8%; -1.2%]).
CONCLUSIONS	Short-term exposures to ambient PM2.5 and ozone is associated with changes in plasma concentrations of metabolites in a cohort of cardiac catheterization patients. Our findings might help to understand the link between air pollution and cardiovascular disease.

표-478. PubMed 논문번호 27806308의 내용 요약

구분	내용
PubMed ID	27806308
TITLE	The impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest: A systematic review and meta-analysis.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2016.10.053
AUTHORS	Zhao Rongxian, Chen Shi, Wang Weiye, Huang Jiao, Wang Ke, Liu Li, Wei Sheng, Zhao Rongxian, Chen Shi, Wang Weiye, Huang Jiao, Wang Ke, Liu Li, Wei Sheng
BACKGROUND	Acute exposure to outdoor air pollution was considered to be associated with the incidence of out-of-hospital cardiac arrest (OHCA). But the relation between specific air pollutants and OHCA remains controversial. We conducted a systematic review and meta-analysis to quantitatively assess the acute effects of air pollutants, including particulate matter (PM10 and PM2.5), sulfur dioxide (SO2), nitrogen dioxide (NO2), carbon monoxide (CO) and ozone (O3) on OHCA onset.
METHODS	Six databases were searched to identify studies analyzing the association between OHCA and the main air pollutants. We summarized the pooled estimates using random-effect models. Heterogeneity within studies was assessed using Cochran's Q and I2 statistics. Funnel plots, Egger's regression test and Begg's rank correlation method were constructed to evaluate publication bias. Subgroup analyses and sensitivity analyses were also conducted to evaluate the potential sources of heterogeneity.
RESULTS	A total of 15 studies met the inclusion criteria. PM10, PM2.5, NO2 and O3 were found to be significantly associated with increase in OHCA risk (PM10 1.021, 95%CI: 1.006-1.037; PM2.5 1.041, 95%CI: 1.012-1.071; NO2 1.015, 95%CI: 1.001-1.030 and O3 1.016, 95%CI: 1.008-1.024). The acute exposure to SO2 and CO was not associated with the incidence of OHCA. Additional analyses verified the findings in the overall analyses except SO2 and NO2. Population attributable fractions for PM10, PM2.5, and O3 were 2.1%, 3.9% and 1.6%, respectively.
CONCLUSION	The current evidence confirmed the associations between short-term exposure to PM2.5, PM10 and O3 and a high risk of OHCA, with the strongest association being observed for PM2.5.

표-479. PubMed 논문번호 27839815의 내용 요약

구분	내용
PubMed ID	27839815
TITLE	Mortality due to acute myocardial infarction in China from 1987 to 2014: Secular trends and age-period-cohort effects.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2016.11.130
AUTHORS	Chang Jie, Liu Xin, Sun Yang, Chang Jie, Liu Xin, Sun Yang
BACKGROUND	In contrast with most developed countries, mortality from acute myocardial infarction (AMI) continues to rise in China. We examined secular trends and age, period, and cohort effects in mortality from AMI in China from 1987 to 2014.
METHODS	Data on deaths from AMI between 1987 and 2014 were obtained from the Chinese Health Statistics Annual Report (1987-2001) and the Chinese Health Statistics Yearbook (2003-2015). We then conducted an age-period-cohort analysis using the intrinsic estimator approach.
RESULTS	There was an upward trend in AMI mortality in both urban and rural populations that accelerated from 2004 onwards. AMI mortality increased 5.6-fold from 1987 to 2014. The net age effect on AMI mortality increased exponentially. AMI mortality risk in the 80-84years of age bracket was 220.15 and 190.70 times higher than in the 15-19years of age bracket in urban and rural populations, respectively. There was a V-shaped trend in the net period effect between 1999 and 2009. Meanwhile, although there was a global reduction in the cohort effect in urban and rural AMI mortality, we found a modest increase among urban populations born between 1975 and 1989 and rural populations born between 1990 and 1999.
CONCLUSION	The changes in exposure to lifestyle-related risk factors and triggers such as air pollution have contributed to the increase in AMI mortality in China over recent decades. The sharp increase in AMI mortality since 2004 is primarily attributable to population aging and the rise in AMI deaths among younger generations.

표-480. PubMed 논문번호 27839852의 내용 요약

구분	내용
PubMed ID	27839852
TITLE	Effect of a clean stove intervention on inflammatory biomarkers in pregnant women in Ibadan, Nigeria: A randomized controlled study.
JOURNAL	Environment international: 10.1016/j.envint.2016.11.004
AUTHORS	Olopade Christopher O, Frank Elizabeth, Bartlett Emily, Alexander Donee, Dutta Anindita, Ibigbami Tope, Adu Damilola, Olamijulo John, Arinola Ganiyu, Karrison Theodore, Ojengbede Oladosu, Olopade Christopher O, Frank Elizabeth, Bartlett Emily, Alexander Donee, Dutta Anindita, Ibigbami Tope, Adu Damilola, Olamijulo John, Arinola Ganiyu, Karrison Theodore, Ojengbede Oladosu
BACKGROUND	Exposure to household air pollution (HAP) has been linked to systemic inflammation. We determined the impact of transition from traditional firewood/kerosene stove to bioethanol-burning stove on inflammatory biomarkers in pregnant Nigerian women.
METHODS	Women (n=324), cooking with kerosene/firewood, were recruited during their first trimester of pregnancy from June 2013-October 2015 and were randomly allocated to either control (n=162) or intervention (n=162) group using web-based randomization. Controls continued to use their own firewood/kerosene stove, while intervention participants received bioethanol CleanCook stoves. Serum concentrations of retinol-binding protein (RBP), malondialdehyde (MDA), tumor necrosis factor alpha (TNF)-α, interleukin (IL)-6, and IL-8 were measured by ELISA.
RESULTS	After excluding 53 women (loss of follow-up, untimely biomarker assessments, incorrect dates of enrollment), data from 271 women were included in analysis. Mean (SD) change in RBP, MDA, TNF-α, IL-6, and IL-8 between baseline and third trimester was -2.16 (4.47), -19.6 (46.4), 3.72 (37.2), 0.51 (14.4), and 13.2 (197), respectively, in intervention and -2.25 (4.30), -24.6 (43.6), 7.17 (32.6), -1.79, (11.4), and 31.3 (296) in control groups. None of these changes differed significantly between the two treatment arms. However, changes from baseline in TNF-α levels were significantly different between intervention and control groups in subset of women (n=99) using firewood before trial (-7.03 [32.9] vs. +12.4 [33.6]; 95% CI for group difference: -35.4 to -3.4, p=0.018).
CONCLUSIONS	Decrease in TNF-α concentration from baseline to third trimesters in intervention group women could indicate reduced cardiovascular stress and prothrombotic effects from decreased HAP. Our findings suggest that ethanol-burning stoves may mitigate cardiovascular health risks.

표-481. PubMed 논문번호 27939045의 내용 요약

구분	내용
PubMed ID	27939045
TITLE	Air pollution and cardiovascular mortality with over 25years follow-up: A combined analysis of two British cohorts.
JOURNAL	Environment international: 10.1016/j.envint.2016.12.004
AUTHORS	Dehbi Hakim-Moulay, Blangiardo Marta, Gulliver John, Fecht Daniela, de Hoogh Kees, Al-Kanaani Zaina, Tillin Therese, Hardy Rebecca, Chaturvedi Nish, Hansell Anna L, Dehbi Hakim-Moulay, Blangiardo Marta, Gulliver John, Fecht Daniela, de Hoogh Kees, Al-Kanaani Zaina, Tillin Therese, Hardy Rebecca, Chaturvedi Nish, Hansell Anna L
BACKGROUND	Adverse effects of air pollution on cardiovascular disease (CVD) mortality are well established. There are comparatively fewer studies in Europe, and in the UK particularly, than in North America. We examined associations in two British cohorts with >25years of follow-up.
METHODS	Annual average NO2, SO2 and black smoke (BS) air pollution exposure estimates for 1991 were obtained from land use regression models using contemporaneous monitoring data. From the European Study of Cohorts and Air Pollution (ESCAPE), air pollution estimates in 2010-11 were obtained for NO2, NOx, PM10, PMcoarse and PM2.5. The exposure estimates were assigned to place of residence 1989 for participants in a national birth cohort born in 1946, the MRC National Study of Health and Development (NSHD), and an adult multi-ethnic London cohort, Southall and Brent Revisited (SABRE) recruited 1988-91. The combined median follow-up was 26years. Single-pollutant competing risk models were employed, adjusting for individual risk factors.
RESULTS	Elevated non-significant hazard ratios for CVD mortality were seen with 1991 BS and SO2 and with ESCAPE PM10 and PM2.5 in fully adjusted linear models. Per 10 μg/m3 increase HRs were 1.11 [95% CI: 0.76-1.61] for BS, 1.05 [95% CI: 0.91-1.22] for SO2, 1.16 [95% CI: 0.70-1.92] for PM10 and 1.30 [95% CI: 0.39-4.34] for PM2.5, with largest effects seen in the fourth quartile of BS and PM2.5 compared to the first with HR 1.24 [95% CI: 0.91-1.61] and 1.21 [95% CI: 0.88-1.66] respectively. There were no consistent associations with other ESCAPE pollutants, or with 1991 NO2. Modelling using Cox regression led to similar results.
CONCLUSION	Our results support a detrimental long-term effect for air pollutants on cardiovascular mortality.

표-482. PubMed 논문번호 27940104의 내용 요약

구분	내용
PubMed ID	27940104
TITLE	Ozone and hypertensive disorders of pregnancy in Florida: Identifying critical windows of exposure.
JOURNAL	Environmental research: 10.1016/j.envres.2016.12.002
AUTHORS	Hu Hui, Ha Sandie, Xu Xiaohui, Hu Hui, Ha Sandie, Xu Xiaohui
INTRODUCTION	Ozone (O3) has been linked to hypertensive disorders of pregnancy (HDP). However, inconsistent results have been reported, and no study has examined the critical exposure windows during pregnancy.
MATERIALS AND METHODS	We used Florida birth vital statistics records to investigate the association between HDP and O3 exposure among 655,529 pregnancies with conception dates between 2005 and 2007. Individual O3 exposure was assessed at mothers' home address at the time of delivery using the Hierarchical Bayesian space-time statistical model. We examined the association during three predefined exposure windows including trimester 1, trimester 2, and trimesters 1&2, as well as in each week of the first two trimesters using distributed lag models.
RESULTS	Pregnancies with HDP had a higher mean exposure to O3 (39.07 in trimester 1, 39.02 in trimester 2, and 39.06 in trimesters 1&2, unit: ppb) than those without HDP (38.65 in trimester 1, 38.57 in trimester 2, and 38.61 in trimesters 1&2, unit: ppb). In the adjusted logistic regression model, increased odds of HDP were observed for each 5 ppb increase in O3 (ORTrimester1=1.04, 95% CI: 1.03, 1.06; ORTrimester2=1.03, 95% CI: 1.02, 1.04; ORTrimester1&2=1.07, 95% CI: 1.05, 1.08). In the distributed lag models, elevated odds of HDP were observed with increased O3 exposure during the 1st to 24th weeks of gestation, with higher odds during early pregnancy.
CONCLUSIONS	O3 exposure during pregnancy is related to increased odds of HDP, and early pregnancy appears to be a potentially critical window of exposure.

표-483. PubMed 논문번호 27977410의 내용 요약

구분	내용
PubMed ID	27977410
TITLE	How serious are health impacts in one of the most polluted regions of Central Europe?
JOURNAL	Reviews on environmental health: 10.1515/reveh-2016-0031
AUTHORS	Ji□□k V□t□zslav, Daleck□ Andrea, Va□endov□ Veronika, Janoutov□ Jana, Janout Vladim□r
BACKGROUND	The long-term exposure to pollutants in ambient air is associated with higher mortality and occurrence of respiratory and cardiopulmonary diseases. The longitudinal cross-section study focuses on the associations between long-term exposures to carcinogenic and non-carcinogenic pollutants and the prevalence and incidence of such specific diseases including immunodeficiencies.
METHODS	The data on health status from industrial and non-industrial regions were obtained from health documentation for a 5-year period from 2007 to 2011 and represent the whole population living in polluted (1,249,323 inhabitants) and unpolluted (631,387 inhabitants) regions. The data on concentrations of PM10, PM2,5, NO2, SO2, benzene and benzo[a]pyrene were collected. The concentrations of pollutants were estimated from measured data by using dispersion models. The average population-weighted concentration of pollutants, which is representative for a defined geographic area and time period from 2007 to 2011, was calculated from the obtained data. The logistic regression and the Mantel-Haenszel $\chi 2$ test were used to determine the odds ratios (OR) and p-values for a linear trend. Moreover, the relative risks of mortality and morbidity to specific diseases were calculated according to theoretical dose-response association published by World Health Organization (WHO).
RESULTS	The probability of incidence of chronic obstructive pulmonary disease and bronchial asthma is statistically significantly higher in the population living in the polluted region compared to the population living in the unpolluted region. The association between long-term exposure to pollutants and the prevalence of immunodeficiency with predominantly antibody defects (D80) was confirmed. The strongest association was found for exposures to particulate matter (PM2,5). The prevalence of immunodeficiency with predominantly antibody defects was also observed in both regions depending on the age of the population and statistically significant difference was only found in the group of adults (20 and over).
CONCLUSION	These associations encourage the hypothesis, that the long-term exposure to PM2.5 might cause the activation of cellular immune response. Further research is needed to explore the correlative immunoregulatory mechanism linking PM2.5 (or other pollutants - SO2) and immune cells. Nowadays, it is also believed that these associations are important in the increase of incidence of immune inflammatory response which is proven risk factor for cardiovascular disease (atherosclerotic disease, coronary heart disease and sudden cardiac death). Positive association between long-term exposure and prevalence of bronchial asthma and chronic obstructive pulmonary disease might be skewed due to important socio-economic factors (especially smoking).

표-484. PubMed 논문번호 27984424의 내용 요약

구분	내용
PubMed ID	27984424
TITLE	Estimating Acute Cardiorespiratory Effects of Ambient Volatile Organic Compounds.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000607
AUTHORS	Ye Dongni, Klein Mitchel, Chang Howard H, Sarnat Jeremy A, Mulholland James A, Edgerton Eric S, Winquist Andrea, Tolbert Paige E, Sarnat Stefanie Ebelt
BACKGROUND	The health effects of ambient volatile organic compounds (VOCs) have received less attention in epidemiologic studies than other commonly measured ambient pollutants. In this study, we estimated acute cardiorespiratory effects of ambient VOCs in an urban population.
METHODS	Daily concentrations of 89 VOCs were measured at a centrally-located ambient monitoring site in Atlanta and daily counts of emergency department visits for cardiovascular diseases and asthma in the five-county Atlanta area were obtained for the 1998-2008 period. To understand the health effects of the large number of species, we grouped these VOCs a priori by chemical structure and estimated the associations between VOC groups and daily counts of emergency department visits in a time-series framework using Poisson regression. We applied three analytic approaches to estimate the VOC group effects: an indicator pollutant approach, a joint effect analysis, and a random effect meta-analysis, each with different assumptions. We performed sensitivity analyses to evaluate copollutant confounding.
RESULTS	Hydrocarbon groups, particularly alkenes and alkynes, were associated with emergency department visits for cardiovascular diseases, while the ketone group was associated with emergency department visits for asthma.
CONCLUSIONS	The associations observed between emergency department visits for cardiovascular diseases and alkenes and alkynes may reflect the role of traffic exhaust, while the association between asthma visits and ketones may reflect the role of secondary organic compounds. The different patterns of associations we observed for cardiovascular diseases and asthma suggest different modes of action of these pollutants or the mixtures they represent.

표-485. PubMed 논문번호 27993830의 내용 요약

구분	내용
PubMed ID	27993830
TITLE	Secondhand Smoke Exposure and Subclinical Cardiovascular Disease: The Multi-Ethnic Study of Atherosclerosis.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.115.002965
AUTHORS	Jones Miranda R, Magid Hoda S, Al-Rifai Mahmoud, McEvoy John W, Kaufman Joel D, Hinckley Stukovsky Karen D, Szklo Moyses, Polak Joseph, Burke Gregory L, Post Wendy S, Blaha Michael J, Navas-Acien Ana
BACKGROUND	Few studies have evaluated the association between secondhand smoke (SHS) and subclinical cardiovascular disease among ethnically diverse populations. This study assesses the impact of SHS on inflammation and atherosclerosis (carotid intima-media thickness, coronary artery calcification, and peripheral arterial disease).
METHODS AND RESULTS	We examined 5032 nonsmoking adults aged 45 to 84 years without prior cardiovascular disease participating in the Multi-Ethnic Study of Atherosclerosis (MESA) from 2000 to 2002. SHS exposure was determined by self-report, and urinary cotinine was measured in a representative subset (n=2893). The multi-adjusted geometric mean ratios (95% CIs) for high-sensitivity C-reactive protein and interleukin-6 comparing 407 participants with SHS ≥12 h/wk versus 3035 unexposed participants were 1.13 (1.02-1.26) and 1.04 (0.98-1.11), respectively. The multi-adjusted geometric mean ratio for carotid intima-media thickness was 1.02 (0.97-1.07). Fibrinogen and coronary artery calcification were not associated with SHS. The prevalence of peripheral arterial disease (ankle-brachial index ≤0.9 or ≥1.4) was associated with detectable urinary cotinine (odds ratio, 2.10; 95% CI, 1.09-4.04) but not with self-reported SHS. Urinary cotinine was not associated with inflammation or carotid intima-media thickness.
CONCLUSIONS	Despite limited exposure assessment, this study supports the association of SHS exposure with inflammation and peripheral arterial disease.

표-486. PubMed 논문번호 28001123의 내용 요약

구분	내용
PubMed ID	28001123
TITLE	Fine-scale spatial and temporal variation in temperature and arrhythmia episodes in the VA Normative Aging Study.
JOURNAL	Journal of the Air & Waste Management Association (1995): 10.1080/10962247.2016.1252808
AUTHORS	Zanobetti Antonella, Coull Brent A, Kloog Itai, Sparrow David, Vokonas Pantel S, Gold Diane R, Schwartz Joel
IMPLICATIONS	This is the first study to provide evidence that residence-specific temperature exposure is associated with increased risk of ventricular arrhythmias in cohort of elderly subjects without known chronic medical conditions; that the delayed effect of temperature has a nonlinear relationship; and therefore that both warm and cold temperature increase the risk of having ventricular arrhythmias. Moreover, we show that the use of residence-specific temperature data reduces downward bias due to exposure error, by comparing the estimated health effect based on our spatiotemporal exposure prediction model to those based on a single local weather monitor.

표-487. PubMed 논문번호 28011231의 내용 요약

구분	내용
PubMed ID	28011231
TITLE	Metabolic syndrome and occupation: Any association? Prevalence among auto technicians and school teachers in South West Nigeria.
JOURNAL	Diabetes & metabolic syndrome: 10.1016/j.dsx.2016.12.035
AUTHORS	Akintunde Adeseye A, Oloyede Taiwo W, Akintunde Adeseye A, Oloyede Taiwo W
AIMS	To determine occupational association with metabolic syndrome among auto technicians and school teachers.
METHODS	One hundred and sixty six subjects were selected for this study. Clinical data was obtained while laboratory investigations including plasma glucose and lipid profile were performed. Statistical analysis was done with SPSS 20.0. P<0.05 was taken as statistically significant.
RESULTS	School teachers had significantly higher mean total cholesterol and LDL-cholesterol compared to auto technicians. Mean serum triglycerides was significantly higher among auto-technicians compared to school teachers in this study. The prevalence of some conventional cardiovascular risk factors was significantly higher among auto-technicians compared to school teachers: Impaired blood glucose (9.6% vs. 1.2%, p<0.05), hypertriglyceridaemia (18.1% vs. 7.2%, p<0.05) and low HDL-Cholesterol (47.0% vs. 37.3%, p<0.05) respectively. However elevated LDL-cholesterol (56.6% vs. 32.5%, p<0.05) was significantly more common among school teachers compared to auto-technicians. Based on the WHO and the Harmonized criteria, metabolic syndrome was more frequent among auto-technicians compared to school teachers. (8.4% vs. 1.2% and 19.3% vs. 7.2% respectively, p<0.05) CONCLUSION: The prevalence of metabolic syndrome appears to be significantly higher among auto technicians despite their high level of physical activity, exertion and education compared to teachers. This may be related to the increased occupational exposure to organic and inorganic materials from dust particles, oil and oil related matter and particulate matter among auto technicians. Prompt definitive evaluation of this concept and appropriate health education to encourage safety mechanism can reduce this burden of metabolic syndrome among auto technicians in Nigeria.

표-488. PubMed 논문번호 28039198의 내용 요약

구분	내용
PubMed ID	28039198
TITLE	Built environment and health: a systematic review of studies in Germany.
JOURNAL	Journal of public health (Oxford, England): 10.1093/pubmed/fdw141
AUTHORS	Schulz Maike, Romppel Matthias, Grande Gesine
Objectives	Empirical research on the relationship between the built environment and health is increasing at a tremendous pace. However, findings from Anglo-American countries may not apply to the European context. Therefore, we systematically reviewed the existing evidence on the role of the built environment for leading risk factors and health behavior in Germany.
Methods	Through an extensive search via PubMed and Web of Science, using predefined selection criteria, two independent reviewers identified 25 empirical studies. We described the main study variables (year of publication, study design, data source, sample characteristics), classified the studies according to their respective operationalization of environmental features and health outcomes, and qualitatively summarized the main results.
Results	The majority of the studies focused on anthropometric measures and physical activity. Access to respective destinations was associated with sports-related physical activity but not with body composition. Also, people living in urban structures tended to show more health-adverse behaviors such as smoking and drinking. Some evidence indicated that higher levels of noise and air pollution were related to higher blood pressure levels. No association was observed between green spaces or street design and health.
Conclusions	Future research should investigate relationships using a sound theoretical basis and research designs that better account for the complex relationship between the built environment and health.

표-489. PubMed 논문번호 28061370의 내용 요약

구분	내용
PubMed ID	28061370
TITLE	Fine particulate air pollution and premature ventricular contractions: The REasons for Geographic And Racial Differences in Stroke (REGARDS) Study.
JOURNAL	Environmental research: 10.1016/j.envres.2016.12.031
AUTHORS	O'Neal Wesley T, Soliman Elsayed Z, Efird Jimmy T, Howard Virginia J, Howard George, McClure Leslie A
BACKGROUND	It is unknown if higher levels of ambient particulate matter (PM) exposure increase the risk for premature ventricular contractions (PVC) in a population-based study of men and women, and if this relationship varies by race or sex.
METHODS	We examined the association of PM 〈2.5□m in diameter (PM2.5) concentration with PVCs in 26,121 (mean age=64±9.3 years; 55% female; 41% black) participants from the REasons for Geographic And Racial Differences in Stroke (REGARDS) study. Estimates of short- (2-week) and long-term (1-year) PM2.5 exposures were computed prior to the baseline visit using geographic information system data on the individual level at the coordinates of study participants' residences. PVCs were identified from baseline electrocardiograms.
RESULTS	PVCs were detected in 1719 (6.6%) study participants. Short- (OR=1.08, 95%CI=1.03, 1.14) and long- (OR=1.06, 95%CI=1.01, 1.12) term PM2.5 exposures were associated with PVCs. Interactions were not detected by race or sex. An interaction between short-term PM2.5 exposure and PVCs was detected for those with cardiovascular disease (OR=1.16, 95%CI=1.06, 1.27) compared with those without cardiovascular disease (OR=1.05, 95%CI=0.99, 1.12; p-interaction=0.027).
CONCLUSION	Our findings suggest that PM2.5 exposure is associated with an increased risk for PVCs in a biracial population-based study of men and women. We also have identified persons with cardiovascular disease as an at-risk population for PVCs when increases in short-term PM2.5 concentration occur.

표-490. PubMed 논문번호 28081369의 내용 요약

구분	내용
PubMed ID	28081369
TITLE	Randomized Controlled Ethanol Cookstove Intervention and Blood Pressure in Pregnant Nigerian Women.
JOURNAL	American journal of respiratory and critical care medicine: 10.1164/rccm.201606-1177OC
AUTHORS	Alexander Donee, Northcross Amanda, Wilson Nathaniel, Dutta Anindita, Pandya Rishi, Ibigbami Tope, Adu Damilola, Olamijulo John, Morhason-Bello Oludare, Karrison Theodore, Ojengbede Oladosu, Olopade Christopher O
RATIONALE	Hypertension during pregnancy is a leading cause of maternal mortality. Exposure to household air pollution elevates blood pressure (BP).
OBJECTIVES	To investigate the ability of a clean cookstove intervention to lower BP durin pregnancy.
METHODS	We conducted a randomized controlled trial in Nigeria. Pregnant women cooking with kerosene or firewood were randomly assigned to an ethanol ar (n = 162) or a control arm (n = 162). BP measurements were taken during six antenatal visits. In the primary analysis, we compared ethanol users with control subjects. In subgroup analyses, we compared baseline kerosene users assigned to the intervention with kerosene control subjects and compared baseline firewood users assigned to ethanol with firewood control subjects.
MEASUREMENTS AND MAIN RESULTS	The change in diastolic blood pressure (DBP) over time was significantly different between ethanol users and control subjects (P = 0.040); systolic blood pressure (SBP) did not differ (P = 0.86). In subgroup analyses, there wa no significant intervention effect for SBP; a significant difference for DBP (P = 0.031) existed among preintervention kerosene users. At the last visit, mean DBP was 2.8 mm Hg higher in control subjects than in ethanol users (3.6 mm Hg greater in control subjects than in ethanol users among preintervention kerosene users), and 6.4% of control subjects were hypertensive (SBP ≥140 and/or DBP ≥90 mm Hg) versus 1.9% of ethanol users (P = 0.051). Among preintervention kerosene users, 8.8% of control subjects were hypertensive compared with 1.8% of ethanol users (P = 0.029).
CONCLUSIONS	To our knowledge, this is the first cookstove randomized controlled trial examining prenatal BP. Ethanol cookstoves have potential to reduce DBP an hypertension during pregnancy. Accordingly, clean cooking fuels may reduce adverse health impacts associated with household air pollution. Clinical trial registered with www.clinicaltrials.gov (NCT02394574).

표-491. PubMed 논문번호 28096146의 내용 요약

구분	내용
PubMed ID	28096146
TITLE	Long-Term Exposure to NO2 and Ozone and Hypertension Incidence in the Black Women's Health Study.
JOURNAL	American journal of hypertension: 10.1093/ajh/hpw168
AUTHORS	Coogan Patricia F, White Laura F, Yu Jeffrey, Brook Robert D, Burnett Richard T, Marshall Julian D, Bethea Traci N, Rosenberg Lynn, Jerrett Michael
BACKGROUND	Evidence shows that exposure to air pollutants can increase blood pressure in the short and long term. Some studies show higher levels of hypertension prevalence in areas of high pollution. Few data exist on the association of air pollution with hypertension incidence. The purpose of the present study was to prospectively assess the associations of the traffic-related nitrogen dioxide (NO2) and of ozone with the incidence of hypertension in the Black Women's Health Study (BWHS), a large cohort study of African American women.
METHODS	We used Cox proportional hazards models to calculate hazard ratios (HRs) and 95% confidence intervals (CI) for hypertension associated with exposure to NO2 and ozone among 33,771 BWHS participants. NO2 and ozone levels at participant residential locations were estimated with validated models.
RESULTS	From 1995 to 2011, 9,570 incident cases of hypertension occurred in a total of 348,154 person-years (median follow-up time, 11 years). The multivariable HRs per interquartile range of NO2 (9.7 ppb) and ozone (6.7 ppb) were 0.92 (95% CI = 0.86, 0.98) and 1.09 (95% CI = 1.00, 1.18).
CONCLUSIONS	In this large cohort of African American women, higher ozone levels were associated with an increase in hypertension incidence. Higher NO2 levels were not associated with greater hypertension incidence; indeed, incidence was lower at higher NO2 levels.

표-492. PubMed 논문번호 28102874의 내용 요약

구분	내용
PubMed ID	28102874
TITLE	Cardiorespiratory hospitalisation and mortality reductions after smoking bans in Switzerland.
JOURNAL	Swiss medical weekly: 10.4414/smw.2016.14381
AUTHORS	Vicedo-Cabrera Ana M, R□□sli Martin, Radovanovic Dragana, Grize Leticia, Witassek Fabienne, Schindler Christian, Perez Laura
INTRODUCTION	Smoking bans are considered one of the most effective policies to reduce population exposure to tobacco smoke and prevent adverse health outcomes. However, evidence on the effect of contextual variables on the effectiveness of smoking bans is still lacking.
AIMS	The patchwork of cantonal smoke-free laws in Switzerland was used as a quasi-experimental setting to assess changes after their introduction in: hospitalisations and mortality due to cardiorespiratory diseases in adults; total hospitalisations and hospitalisations due to respiratory disorders in children; and the modifying effects of contextual factors and the effectiveness of the laws.
METHODS	Using hospital and mortality registry data for residents in Switzerland (2005-2012), we conducted canton-specific interrupted time-series analyses followed by random effects meta-analyses to obtain nationwide smoking ban estimates by subgroups of age, sex and causes of hospitalisation or death. Heterogeneity of the impact caused by strictness of the ban and other smoking-related characteristics of the cantons was explored through meta-regression.
RESULTS	Total hospitalisation rates due to cardiovascular and respiratory diseases did not significantly change after the introduction of the ban. Post-ban changes were detected in ischaemic heart disease hospitalisations, with a 2.5% reduction (95% confidence interval [CI)] -6.2 to 1.3%) for all ages and 5.5% (95% CI -10.8 to -0.2%) in adults 35-64 years old. Total mortality due to respiratory diseases decreased by 8.2% (95% CI -15.2 to -0.6%) over all ages, and chronic obstructive pulmonary disease mortality decreased by 14.0% (95% CI -22.3 to -4.5%) in adults ≥65 years old. Cardiovascular mortality did not change after the introduction of the ban, but there was an indication of post-ban reductions in mortality due to hypertensive disorders (-5.4%, 95% CI -12.6 to 2.3%), and congestive heart failure (-6.0%, 95% CI -14.5 to 3.4%). No benefits were observed for hospitalisations due to respiratory diseases in children or for infant mortality. The type of smoking ban implemented explained the heterogeneity of benefits across cantons for some outcomes.
CONCLUSION	Smoking bans in Switzerland were associated with overall reductions in cardiovascular and respiratory hospitalisation and mortality in adults.

표-493. PubMed 논문번호 28103841의 내용 요약

구분	내용
PubMed ID	28103841
TITLE	A two-site, two-arm, 34-week, double-blind, parallel-group, randomized controlled trial of reduced nicotine cigarettes in smokers with mood and/or anxiety disorders: trial design and protocol.
JOURNAL	BMC public health: 10.1186/s12889-016-3946-4
AUTHORS	Allen Sophia I, Foulds Jonathan, Pachas Gladys N, Veldheer Susan, Cather Corinne, Azzouz Nour, Hrabovsky Shari, Hameed Ahmad, Yingst Jessica, Hammett Erin, Modesto Jennifer, Krebs Nicolle M, Zhu Junjia, Liao Jason, Muscat Joshua E, Richie John, Evins A Eden
BACKGROUND	The U.S. Food and Drug Administration can set standards for cigarettes that could include reducing their nicotine content. Such a standard should improve public health without causing unintended serious consequences for sub-populations. This study evaluates the effect of progressive nicotine reduction in cigarettes on smoking behavior, toxicant exposure, and psychiatric symptoms in smokers with comorbid mood and/or anxiety disorders using a two-site, two-arm, double-blind, parallel group, randomized controlled trial (RCT) in four phases over 34 weeks.
METHODS	Adult smokers (N = 200) of 5 or more cigarettes per day will be randomized across two sites (Penn State and Massachusetts General). Participants must have not had a quit attempt in the prior month, nor be planning to quit in the next 6 months, meet criteria for a current or lifetime unipolar mood and/or anxiety disorder based on the structured Mini-International Neuropsychiatric Interview, and must not have an unstable medical or psychiatric condition. After a week of smoking their own cigarettes, participants receive two weeks of Spectrum research cigarettes with usual nicotine content (11.6 mg). After this baseline period, participants will be randomly assigned to continue smoking Spectrum research cigarettes that contain either (a) Usual Nicotine Content (11.6 mg); or (b) Reduced Nicotine Content: the nicotine content per cigarette is progressively reduced from approximately 11.6 mg to 0.2 mg in five steps over 18 weeks. At the end of the randomization phase, participants will be offered the choice to either (a) quit smoking with assistance, (b) continue smoking free research cigarettes, or (c) return to purchasing their own cigarettes, for the final 12 weeks of the study. The primary outcome measure is blood cotinine; key secondary outcomes are: exhaled carbon monoxide, urinary total NNAL-4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol and 1-hydroxypyrene, oxidative stress biomarkers including 8-isoprostanes, measures of psychiatric symptoms (e.g., depression, anxiety), smoking behavior and dependence (e.g., cigarette consumption, quit attempts), and health effects (e.g., blood pressure, respiratory symptoms).
DISCUSSION	Results from this study will inform FDA on the potential effects of regulating the nicotine content of cigarettes and help determine whether smokers with mood and/or anxiety disorders can safely transition to significantly reduced nicotine content cigarettes.
TRIAL REGISTRATION	TRN: NCT01928758 , registered August 21, 2013.

표-494. PubMed 논문번호 28236545의 내용 요약

구분	내용
PubMed ID	28236545
TITLE	Air pollution and ST-elevation myocardial infarction treated with primary percutaneous coronary angioplasty: A direct correlation.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2017.02.050
AUTHORS	Zuin Marco, Rigatelli Gianluca, dell'Avvocata Fabio, Picariello Claudio, Conte Luca, Marcantoni Lina, Cardaioli Paolo, Zuliani Giovanni, Roncon Loris
PURPOSE	The relationships between air pollutant concentration levels and admission for primary percutaneous coronary intervention (PCI) in patients with ST-elevation myocardial infarction (STEMI) have never been assessed.
METHODS	We retrospectively reviewed 4 consecutive years of medical and instrumental data (1st January 2012 to 1st March 2016) to identify patients admitted with STEMI and subsequently treated with primary PCI in our third referral center. Daily atmospheric pressure data (in hectopascal [hPa]) and air pollutant concentration levels were obtained from the regional meteorological service which had a monitoring site in our city (Rovigo, Italy). Pollutants investigated were nitrogen dioxide (NO2), particulate matter ≤10 μm (PM10), ozone (O3), sulfur dioxide (SO2) and carbon monoxide (CO). Safety air concentration levels for the air pollutants were also considered.
RESULTS	PCI in STEMI patients was more frequent when AP was higher than 1013.15hPa (61.8% vs 38.2%, p<0.001). The incidences of STEMI patients when NO2, PM10 and O3 levels overcame the safe threshold were 83.1%, 52% and 8.5%, respectively. A positive correlation was found between the daily number of STEMI subsequently treated with primary PCI and the air pollutant levels of the same day for NO2 (r=0.205, p=0.001), PM10 (r=0.349, p<0.0001) and O3 (r=0.191, p=0.002).
CONCLUSIONS	A direct and significant correlation exists between the number of daily STEMI patients and the NO2, PM10 and O3 air concentration levels of the same day.

표-495. PubMed 논문번호 28248974의 내용 요약

구분	내용
PubMed ID	28248974
TITLE	The effect of randomised exposure to different types of natural outdoor environments compared to exposure to an urban environment on people with indications of psychological distress in Catalonia.
JOURNAL	PloS one: 10.1371/journal.pone.0172200
AUTHORS	Triguero-Mas Margarita, Gidlow Christopher J, Mart□nez David, de Bont Jeroen, Carrasco-Turigas Gl□ria, Mart□nez-□□iguez Tania, Hurst Gemma, Masterson Daniel, Donaire-Gonzalez David, Seto Edmund, Jones Marc V, Nieuwenhuijsen Mark J
INTRODUCTION	Experimental studies have reported associations between short-term exposure to natural outdoor environments (NOE) and health benefits. However, they lack insight into mechanisms, often have low external and ecological validity, and have rarely focused on people with some psycho-physiological affection. The aim of this study was to use a randomized, case-crossover design to investigate: (i) the effects of unconstrained exposure to real natural and urban environments on psycho-physiological indicators of people with indications of psychological distress, (ii) the possible differential effects of 30 and 30+180 minutes exposures, and (iii) the possible mechanisms explaining these effects.
MATERIAL AND METHODS	People (n = 26) with indications of psychological distress were exposed to green (Collserola Natural Park), blue (Castelldefels beach) and urban (Eixample neighbourhood) environments in Catalonia. They were exposed to all environments in groups for a period of 30+180 minutes between October 2013 and January 2014. During the exposure period, participants were instructed to do what they would usually do in that environment. Before, during (at 30 and 30+180 minutes) and after each exposure, several psycho-physiological measures were taken: mood (measured as Total Mood Disturbance, TMD), attention capacity (measured as backwards digit-span task), stress levels (measures as salivary cortisol), systolic and diastolic blood pressure, heart rate, autonomous nervous system (assessed as heart rate variability and the indicators: low frequency power (LF), high frequency power (HF), ratio between LF and HF (LF:HF), and coefficients of component variance of LF, HF, and LF:HF). We also measured several potential mediators: air pollution, noise, physical activity, social interactions, and self-perceived restoration experience.
RESULTS	When compared with responses to urban environment, we found statistically significantly lower TMD [-4.78 (-7.77, -1.79) points difference], and salivary cortisol [-0.21 (-0.34, -0.08) log nmol/L] in the green exposure environment, and statistically significantly lower TMD [-4.53 (-7.57, -1.49) points difference], and statistically significant favourable changes in heart rate variability indicators (specifically LF:HF and CCV-LF:HF with around -0.20 points of difference of the indicators) in the blue exposure environment. Physical activity and self-perceived restoration experience partially mediated the associations between NOE and TMD. Physical activity and air pollution partially mediated the associations between NOE and heart rate variability.
DISCUSSION AND CONCLUSIONS	This study extends the existing evidence on the benefits of NOE for people's health. It also suggests NOE potential as a preventive medicine, specifically focusing on people with indications of psychological distress.
TRIAL REGISTRATION	Clinicaltrials.gov NCT02624921.

표-496. PubMed 논문번호 28255648의 내용 요약

구분	내용
PubMed ID	28255648
TITLE	Short-term effects of fine particulate matter pollution on daily health events in Latin America: a systematic review and meta-analysis.
JOURNAL	International journal of public health: 10.1007/s00038-017-0960-y
AUTHORS	Fajersztajn La□s, Saldiva Paulo, Pereira Luiz Alberto Amador, Leite Victor Figueiredo, Buehler Anna Maria
OBJECTIVES	Ambient air pollution is among the leading risks for health worldwide and by 2050 will largely overcome deaths due to unsafe sanitation and malaria, but local evidence from Latin America (LA) is scarce. We aimed to summarize the effect of short-term exposure to fine particulate air pollution (PM2.5) on morbidity and mortality in Latin America and evaluate evidence coverage and quality, using systematic review and meta-analysis.
METHODS	The comprehensive search (six online databases and hand-searching) identified studies investigating the short-term associations between PM2.5 and daily health events in LA. Two reviewers independently accessed the internal validity of the studies and used random-effect models in the meta-analysis.
RESULTS	We retrieved 1628 studies. Nine were elected for the qualitative analysis and seven for the quantitative analyses. Each 10 □g/m3 increments in daily PM2.5 concentrations was significantly associated with increased risk for respiratory and cardiovascular mortality in all-ages (polled RR = 1.02, 95% CI, 1.02-1.02 and RR = 1.01, 95% CI , 1.01-1.02, respectively).
CONCLUSIONS	Short-term exposure to PM2.5 in LA is significantly associated with increased risk for respiratory and cardiovascular mortality. Evidence is concentrated in few cities and some presented high risk of bias.

표-497. PubMed 논문번호 28270143의 내용 요약

구분	내용
PubMed ID	28270143
TITLE	Residential distance to major roadways and cardiac structure in African Americans: cross-sectional results from the Jackson Heart Study.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-017-0226-4
AUTHORS	Weaver Anne M, Wellenius Gregory A, Wu Wen-Chih, Hickson DeMarc A, Kamalesh Masoor, Wang Yi
BACKGROUND	Heart failure (HF) is a significant source of morbidity and mortality among African Americans. Ambient air pollution, including from traffic, is associated with HF, but the mechanisms remain unknown. The objectives of this study were to estimate the cross-sectional associations between residential distance to major roadways with markers of cardiac structure: left ventricular (LV) mass index, LV end-diastolic diameter, LV end-systolic diameter, and LV hypertrophy among African Americans.
METHODS	We studied baseline participants of the Jackson Heart Study (recruited 2000-2004), a prospective cohort of cardiovascular disease (CVD) among African Americans living in Jackson, Mississippi, USA. All cardiac measures were assessed from echocardiograms. We assessed the associations between residential distance to roads and cardiac structure indicators using multivariable linear regression or multivariable logistic regression, adjusting for potential confounders.
RESULTS	Among 4826 participants, residential distance to road was <150 m for 103 participants, 150-299 m for 158, 300-999 for 1156, and ≥1000 m for 3409. Those who lived <150 m from a major road had mean 1.2 mm (95% CI 0.2, 2.1) greater LV diameter at end-systole compared to those who lived ≥1000 m. We did not observe statistically significant associations between distance to roads and LV mass index, LV end-diastolic diameter, or LV hypertrophy. Results did not materially change after additional adjustment for hypertension and diabetes or exclusion of those with CVD at baseline; results strengthened when modeling distance to A1 roads (such as interstate highways) as the exposure of interest.
CONCLUSIONS	We found that residential distance to roads may be associated with LV end-systolic diameter, a marker of systolic dysfunction, in this cohort of African Americans, suggesting a potential mechanism by which exposure to traffic pollution increases the risk of HF.

표-498. PubMed 논문번호 28302634의 내용 요약

구분	내용
PubMed ID	28302634
TITLE	Residential proximity to major roads, exposure to fine particulate matte aortic calcium: the Framingham Heart Study, a cohort study.
JOURNAL	BMJ open: 10.1136/bmjopen-2016-013455
AUTHORS	Dorans Kirsten S, Wilker Elissa H, Li Wenyuan, Rice Mary B, Ljungma Petter L, Schwartz Joel, Coull Brent A, Kloog Itai, Koutrakis Petros, D'Agostino Ralph B, Massaro Joseph M, Hoffmann Udo, O'Donnell Christopher J, Mittleman Murray A
OBJECTIVES	Traffic and ambient air pollution exposure are positively associated wit cardiovascular disease, potentially through atherosclerosis promotion. F studies have assessed associations of these exposures with thoracic aorti calcium Agatston score (TAC) or abdominal aortic calcium Agatston sc (AAC), systemic atherosclerosis correlates. We assessed whether living c a major road and residential fine particulate matter (PM2.5) exposure w associated with TAC and AAC in a Northeastern US cohort.
DESIGN	Cohort study.
SETTING	Framingham Offspring and Third Generation participants residing in th Northeastern USA.
PARTICIPANTS AND OUTCOME MEASURES	Among 3506 participants, mean age was 55.8 years; 50% female. TAC measured from 2002 to 2005 and AAC up to two times (2002-2005; 2008-2011) among participants from the Framingham Offspring or Th Generation cohorts. We first assessed associations with detectable TAC (logistic regression) and AAC (generalised estimating equation regressio link). As aortic calcium scores were right skewed, we used linear regres models and mixed-effects models to assess associations with natural log-transformed TAC and AAC, respectively, among participants with detectable aortic calcium. We also assessed associations with AAC progression. Models were adjusted for demographic variables, socioeco position indicators and time.
RESULTS	There were no consistent associations of major roadway proximity or F with the presence or extent of TAC or AAC, or with AAC progression. estimates were in the opposite direction than expected.
CONCLUSIONS	In this cohort from a region with relatively low levels of and variation i PM2.5, there were no strong associations of proximity to a major road PM2.5 with the presence or extent of aortic calcification, or with AAC progression.

표-499. PubMed 논문번호 28332341의 내용 요약

구분	내용
PubMed ID	28332341
TITLE	Risk factors for acute exacerbation of idiopathic pulmonary fibrosis: A systematic review and meta-analysis.
JOURNAL	The clinical respiratory journal: 10.1111/crj.12631
AUTHORS	Qiu Meihua, Chen Yuqing, Ye Qiao, Qiu Meihua, Chen Yuqing, Ye Qiao
BACKGROUND	Idiopathic pulmonary fibrosis (IPF) is a chronic and progressive fibrotic disease limited to the lungs. The course of disease varies widely, with some patients experiencing acute respiratory deterioration, a condition called acute exacerbations of IPF (AE-IPF). The risk factors contributing to AE-IPF are unclear. This systematic review and meta-analysis investigated the risk factors for AE-IPF.
METHODS	Studies of risk factors for AE-IPF were identified in Medline, EMBASE and Cochrane databases. Fixed effects models were used to calculate pooled relative risks and weighted mean differences (WMD).The meta-analysis included seven articles involving 14 risk factors for AE-IPF.
RESULTS	Risk factors for AE included reductions in vital capacity (VC; WMD - 10.58, 95% confidence interval (CI) -17.17 to - 3.99), forced vital capacity (FVC; WMD -6.02, 95%CI - 8.58 to - 3.47), total lung capacity (TLC; WMD -4.88, 95%CI -7.59 to - 2.17), and PaO2 (WMD -4.19, 95%CI -7.66 to -0.71) and a higher alveolar-arterial oxygen difference (AaDO2 ; WMD 4.4, 95%CI 0.24 to 8.57). Mechanical procedures, higher serum KL-6 concentration and secondary pulmonary hypertension, might be risk factors for AE-IPF. In contrast, age, sex, body mass index (BMI), differences in diffusing lung capacity for carbon monoxide (DLCO), exposure to seasonal variations and air pollution, and virus infection might be unrelated to AE-IPF.
CONCLUSIONS	Poor pulmonary function, mechanical procedures, higher serum KL-6 and secondary pulmonary hypertension were associated with increased risks of AE-IPF.

표-500. PubMed 논문번호 28334645의 내용 요약

구분	내용
PubMed ID	28334645
TITLE	Long-term residential road traffic noise and NO₂ exposure in relation to risk of incident myocardial infarction - A Danish cohort study.
JOURNAL	Environmental research: 10.1016/j.envres.2017.03.019
AUTHORS	Roswall Nina, Raaschou-Nielsen Ole, Ketzel Matthias, Gammelmark Anders, Overvad Kim, Olsen Anja, S ø rensen Mette
BACKGROUND	Road traffic is a source of both air pollution and noise; two environmental hazards both found to increase the risk of ischemic heart disease. Given the high correlation between these pollutants, it is important to investigate combined effects, in relation to myocardial infarction (MI).
METHODS	Among 50,744 middle-aged Danes enrolled into the Diet, Cancer and Health cohort from 1993 to 97, we identified 2403 cases of incident MI during a median follow-up of 14.5 years. Present and historical residential addresses from 1987 to 2011 were found in national registries, and traffic noise (Lden) and air pollution (NO2) were modelled for all addresses. Analyses were performed using Cox proportional hazard models.
RESULTS	Road traffic noise and NO2 were both individually associated with a higher risk of MI, with hazard ratios of 1.14 (1.07-1.21) and 1.08 (1.03-1.12) per inter-quartile range higher 10-year mean of road traffic noise and NO2, respectively. Mutual exposure adjustment reduced the association with 10-year NO2 exposure (1.02 (0.96-1.08)), whereas the association with road traffic noise remained: 1.12 (1.03-1.21). For fatal incident MI, the pattern was similar, but the associations for both pollutants were stronger. In analyses of tertiles across both pollutants, the strongest effects were seen for combined medium/high exposure, especially for fatal MI's.
CONCLUSION	Both road traffic noise and NO2 were associated with a higher risk of MI in single-pollutant models. In two-pollutant models, mainly noise was associated with MI. Combined exposure to both pollutants was associated with the highest risk.

표-501. PubMed 논문번호 28363975의 내용 요약

구분	내용
PubMed ID	28363975
TITLE	Fine particulate matter exposure and incidence of stroke: A cohort study in Hong Kong.
JOURNAL	Neurology: 10.1212/WNL.0000000000003903
AUTHORS	Qiu Hong, Sun Shengzhi, Tsang Hilda, Wong Chit-Ming, Lee Ruby Siu-Yin, Schooling C Mary, Tian Linwei
OBJECTIVE	We aimed to assess the association of long-term residential exposure to fine particulate matter (PM) with aerodynamic diameter less than 2.5 μm (PM2.5) with the incidence of stroke and its major subtypes.
METHODS	We ascertained the first occurrence of emergency hospital admission for stroke in a Hong Kong Chinese cohort of 66,820 older people (65+ years) who enrolled during 1998-2001 (baseline) and were followed up to December 31, 2010. High-resolution (1 × 1 km) yearly mean concentrations of PM2.5 were predicted from local monitoring data and US National Aeronautics and Space Administration satellite data using linear regression. Baseline residential PM2.5 exposure was used as a proxy for long-term exposure. We used Cox proportional hazards to evaluate the risk of incident stroke associated with PM2.5 exposure adjusted for potential confounders, including individual and neighborhood factors.
RESULTS	Over a mean follow-up of 9.4 years, we ascertained 6,733 cases of incident stroke, of which 3,526 (52.4%) were ischemic and 1,175 (17.5%) were hemorrhagic. The hazard ratio for every 10 μg/m3 higher PM2.5 concentration was statistically significant at 1.21 (95% confidence interval [CI] 1.04-1.41) for ischemic and non-statistically significant at 0.90 (95% CI 0.70-1.17) for hemorrhagic stroke in fully adjusted model 3. The estimates for ischemic stroke were higher in older participants (>70 years), less educated participants, and in men for current smokers.
CONCLUSION	Long-term PM2.5 exposure was associated with higher risk of incident ischemic stroke, but the association with incident hemorrhagic stroke was less clear.

표-502. PubMed 논문번호 28376798의 내용 요약

구분	내용
PubMed ID	28376798
TITLE	Effects of extreme temperatures on cardiovascular emergency hospitalizations in a Mediterranean region: a self-controlled case series study.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-017-0238-0
AUTHORS	Ponjoan Anna, Blanch Jordi, Alves-Cabratosa Lia, Mart□-Lluch Ruth, Comas-Cuf□ Marc, Parramon D□dac, Del Mar Garcia-Gil Mar□a, Ramos Rafel, Petersen Irene
BACKGROUND	Cold spells and heatwaves increase mortality. However little is known about the effect of heatwaves or cold spells on cardiovascular morbidity. This study aims to assess the effect of cold spells and heatwaves on cardiovascular diseases in a Mediterranean region (Catalonia, Southern Europe).
METHODS	We conducted a population-based retrospective study. Data were obtained from the System for the Development of Research in Primary Care and from the Catalan Meteorological Service. The outcome was first emergency hospitalizations due to coronary heart disease, stroke, or heart failure. Exposures were: cold spells; cold spells and 3 or 7 subsequent days; and heatwaves. Incidence rate ratios (IRR) and 95% confidence intervals were calculated using the self-controlled case series method. We accounted for age, time trends, and air pollutants; results were shown by age groups, gender or cardiovascular event type.
RESULTS	There were 22,611 cardiovascular hospitalizations in winter and 17,017 in summer between 2006 and 2013. The overall incidence of cardiovascular hospitalizations significantly increased during cold spells (IRR = 1.120; CI 95%: 1.10-1.30) and the effect was even stronger in the 7 days subsequent to the cold spell (IRR = 1.29; CI 95%: 1.22-1.36). Conversely, cardiovascular hospitalizations did not increase during heatwaves, neither in the overall nor in the stratified analysis.
CONCLUSIONS	Cold spells but not heatwaves, increased the incidence of emergency cardiovascular hospitalizations in Catalonia. The effect of cold spells was greater when including the 7 subsequent days. Such knowledge might be useful to develop strategies to reduce the impact of extreme temperature episodes on human health.

표-503. PubMed 논문번호 28386038의 내용 요약

구분	내용
PubMed ID	28386038
TITLE	Ambient PM〈sub〉2.5〈/sub〉 and Stroke: Effect Modifiers and Population Attributable Risk in Six Low- and Middle-Income Countries.
JOURNAL	Stroke: 10.1161/STROKEAHA.116.015739
AUTHORS	Lin Hualiang, Guo Yanfei, Di Qian, Zheng Yang, Kowal Paul, Xiao Jianpeng, Liu Tao, Li Xing, Zeng Weilin, Howard Steven W, Nelson Erik J, Qian Zhengmin, Ma Wenjun, Wu Fan
BACKGROUND AND PURPOSE	Short-term exposure to ambient fine particulate pollution (PM2.5) has been linked to increased stroke. Few studies, however, have examined the effects of long-term exposure.
METHODS	A total of 45 625 participants were interviewed and included in this study, the participants came from the Study on Global Ageing and Adult Health, a prospective cohort in 6 low- and middle-income countries. Ambient PM2.5 levels were estimated for participants' communities using satellite data. A multilevel logistic regression model was used to examine the association between long-term PM2.5 exposure and stroke. Potential effect modification by physical activity and consumption of fruit and vegetables was assessed.
RESULTS	The odds of stroke were 1.13 (95% confidence interval, 1.04-1.22) for each 10 μg/m3 increase in PM2.5. This effect remained after adjustment for confounding factors including age, sex, smoking, and indoor air pollution (adjusted odds ratio=1.12; 95% confidence interval, 1.04-1.21). Further stratified analyses suggested that participants with higher levels of physical activity had greater odds of stroke, whereas those with higher consumption of fruit and vegetables had lower odds of stroke. These effects remained robust in sensitivity analyses. We further estimated that 6.55% (95% confidence interval, 1.97%-12.01%) of the stroke cases could be attributable to ambient PM2.5 in the study population.
CONCLUSIONS	This study suggests that ambient PM2.5 may increase the risk of stroke and may be responsible for the astounding stroke burden in low- and middle-income countries. In addition, greater physical activity may enhance, whereas greater consumption of fruit and vegetables may mitigate the effect.

표-504. PubMed 논문번호 28392067의 내용 요약

구분	내용
PubMed ID	28392067
TITLE	Cognitive impacts of ambient air pollution in the National Social Health and Aging Project (NSHAP) cohort.
JOURNAL	Environment international: 10.1016/j.envint.2017.03.019
AUTHORS	Tallon Lindsay A, Manjourides Justin, Pun Vivian C, Salhi Carmel, Suh Helen
BACKGROUND	Pathways through which air pollution may impact cognitive function are poorly understood, particularly with regard to whether and how air pollution interacts with social and emotional factors to influence cognitive health.
OBJECTIVE	To examine the association between air pollutant exposures and cognitive outcomes among older adults participating in the National Social Life, Health, and Aging Project (NSHAP) cohort study.
METHODS	Measures of cognitive function, social connectedness, and physical and mental health were obtained for each NSHAP participant starting with Wave 1 of the study in 2005. Cognitive function was assessed using the Chicago Cognitive Function Measure (CCFM) for 3377 participants. Exposures to fine particles (PM2.5) were estimated for each participant using GIS-based spatio-temporal models, and exposures to nitrogen dioxide (NO2) were obtained from the nearest EPA monitors.
RESULTS	In adjusted linear regression models, IQR increases in 1 to 7year PM2.5 exposures were associated with a 0.22 (95% CI: -0.44, -0.01) to a 0.25 (95% CI: -0.43, -0.06) point decrease in CCFM scores, equivalent to aging 1.6years, while exposures to NO2 were equivalent to aging 1.9years. The impacts of PM2.5 on cognition were modified by stroke, anxiety, and stress, and were mediated by depression. The impacts of NO2 were mediated by stress and effect modification by impaired activities of daily living for NO2 was found.
CONCLUSIONS	Exposures to long-term PM2.5 and NO2 were associated with decreased cognitive function in our cohort of older Americans, and individuals who experienced a stroke or elevated anxiety were more susceptible to the effects of PM2.5 on cognition. Additionally, mediation results suggest that PM2.5 may impact cognition through pathways related to mood disorders.

표-505. PubMed 논문번호 28392078의 내용 요약

구분	내용
PubMed ID	28392078
TITLE	Association of air pollution with increased incidence of ventricular tachyarrhythmias recorded by implantable cardioverter defibrillators: Vulnerable patients to air pollution.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2017.03.122
AUTHORS	Kim In-Soo, Sohn Jungwoo, Lee Seung-Jun, Park Jin-Kyu, Uhm Jae-Sun, Pak Hui-Nam, Lee Moon-Hyoung, Kim Changsoo, Joung Boyoung
BACKGROUND	This study investigated the acute effects of exposure to air pollution on ventricular tachyarrhythmias (VTAs) in an East Asian population. The association between air pollution and VTA has not yet been studied in an East Asian country affected by the Asian dust phenomenon, which worsens air quality.
METHODS	The study cohort consisted of 160patients with implantable cardioverter defibrillator (ICD) devices in the Seoul metropolitan area who were followed for 5.5±3.8years. We used ICD records of VTAs and matched these with hourly measurements of air pollutant concentrations and meteorological data. Fine particle mass and gaseous air pollution plus temperature and relative humidity were measured hourly during the study period.
RESULTS	During the study period, 1064 VTA events including 204 instances of ventricular fibrillation (VF) were observed. We found a statistically significant association between overall VTA events and SO2 (lag 24h; OR 1.49, 95%CI 1.16-1.92, p=0.002), PM10 (lag 2h; OR 2.56, 95%CI 2.03-3.23, p<0.001), NO2 (lag 24h; OR 1.25, 95%CI 1.19-1.31, p<0.001) and CO (lag 24h; OR 1.05, 95%CI 1.02-1.08, p=0.003). Sustained ventricular tachycardia or VF was also independently associated with SO2, PM10, NO2 and CO (all p<0.01). Exposures to SO2, PM10, NO2, and CO (all p<0.01) were significantly related to overall VTAs, especially in patients with structural heart disease (SHD).
CONCLUSIONS	Associations between air pollution and VTA were observed in a metropolitan area of an East Asian country. Exposures to SO2, PM10, NO2, and CO were significantly associated with VTAs in ICD patients with SHD.

표-506. PubMed 논문번호 28395240의 내용 요약

구분	내용
PubMed ID	28395240
TITLE	Associations between lifestyle and air pollution exposure: Potential for confounding in large administrative data cohorts.
JOURNAL	Environmental research: 10.1016/j.envres.2017.03.050
AUTHORS	Strak Maciej, Janssen Nicole, Beelen Rob, Schmitz Oliver, Karssenberg Derek, Houthuijs Danny, van den Brink Carolien, Dijst Martin, Brunekreef Bert, Hoek Gerard
BACKGROUND	Cohorts based on administrative data have size advantages over individual cohorts in investigating air pollution risks, but often lack in-depth information on individual risk factors related to lifestyle. If there is a correlation between lifestyle and air pollution, omitted lifestyle variables may result in biased air pollution risk estimates. Correlations between lifestyle and air pollution can be induced by socio-economic status affecting both lifestyle and air pollution exposure.
OBJECTIVES	Our overall aim was to assess potential confounding by missing lifestyle factors on air pollution mortality risk estimates. The first aim was to assess associations between long-term exposure to several air pollutants and lifestyle factors. The second aim was to assess whether these associations were sensitive to adjustment for individual and area-level socioeconomic status (SES), and whether they differed between subgroups of the population. Using the obtained air pollution-lifestyle associations and indirect adjustment methods, our third aim was to investigate the potential bias due to missing lifestyle information on air pollution mortality risk estimates in administrative cohorts.
METHODS	We used a recent Dutch national health survey of 387,195 adults to investigate the associations of PM10, PM2.5, PM2.5-10, PM2.5 absorbance, OPDTT, OPESR and NO2 annual average concentrations at the residential address from land use regression models with individual smoking habits, alcohol consumption, physical activity and body mass index. We assessed the associations with and without adjustment for neighborhood and individual SES characteristics typically available in administrative data cohorts. We illustrated the effect of including lifestyle information on the air pollution mortality risk estimates in administrative cohort studies using a published indirect adjustment method.
RESULTS	Current smoking and alcohol consumption were generally positively associated with air pollution. Physical activity and overweight were negatively associated with air pollution. The effect estimates were small (mostly <5% of the air pollutant standard deviations). Direction and magnitude of the associations depended on the pollutant, use of continuous vs. categorical scale of the lifestyle variable, and level of adjustment for individual and area-level SES. Associations further differed between subgroups (age, sex) in the population. Despite the small associations between air pollution and smoking intensity, indirect adjustment resulted in considerable changes of air pollution risk estimates for cardiovascular and especially lung cancer mortality.
CONCLUSIONS	Individual lifestyle-related risk factors were weakly associated with long-term exposure to air pollution in the Netherlands. Indirect adjustment for missing lifestyle factors in administrative data cohort studies may substantially affect air pollution mortality risk estimates.

표-507. PubMed 논문번호 28408373의 내용 요약

구분	내용
PubMed ID	28408373
TITLE	Association of Air Pollution Exposures With High-Density Lipoprotein Cholesterol and Particle Number: The Multi-Ethnic Study of Atherosclerosis.
JOURNAL	Arteriosclerosis, thrombosis, and vascular biology: 10.1161/ATVBAHA.116.308193
AUTHORS	Bell Griffith, Mora Samia, Greenland Philip, Tsai Michael, Gill Ed, Kaufman Joel D
OBJECTIVE	The relationship between air pollution and cardiovascular disease may be explained by changes in high-density lipoprotein (HDL).
APPROACH AND RESULTS	We examined the cross-sectional relationship between air pollution and both HDL cholesterol and HDL particle number in the MESA Air study (Multi-Ethnic Study of Atherosclerosis Air Pollution). Study participants were 6654 white, black, Hispanic, and Chinese men and women aged 45 to 84 years. We estimated individual residential ambient fine particulate pollution exposure (PM2.5) and black carbon concentrations using a fine-scale likelihood-based spatiotemporal model and cohort-specific monitoring. Exposure periods were averaged to 12 months, 3 months, and 2 weeks prior to examination. HDL cholesterol and HDL particle number were measured in the year 2000 using the cholesterol oxidase method and nuclear magnetic resonance spectroscopy, respectively. We used multivariable linear regression to examine the relationship between air pollution exposure and HDL measures. A 0.7×10-6 m-1 higher exposure to black carbon (a marker of traffic-related pollution) averaged over a 1-year period was significantly associated with a lower HDL cholesterol (-1.68 mg/dL; 95% confidence interval, -2.86 to -0.50) and approached significance with HDL particle number (-0.55 mg/dL; 95% confidence interval, -1.13 to 0.03). In the 3-month averaging time period, a 5 μg/m3 higher PM2.5 was associated with lower HDL particle number (-0.64 μmol/L; 95% confidence interval, -1.01 to -0.26), but not HDL cholesterol (-0.05 mg/dL; 95% confidence interval, -0.82 to 0.71).
CONCLUSIONS	These data are consistent with the hypothesis that exposure to air pollution is adversely associated with measures of HDL.

표-508. PubMed 논문번호 28412860의 내용 요약

구분	내용
PubMed ID	28412860
TITLE	Modeling vascular inflammation and atherogenicity after inhalation of ambient levels of ozone: exploratory lessons from transcriptomics.
JOURNAL	Inhalation toxicology: 10.1080/08958378.2017.1310333
AUTHORS	Tham Andrea, Lullo Dominic, Dalton Sarah, Zeng Siyang, van Koeverden Ian, Arjomandi Mehrdad
BACKGROUND	Epidemiologic studies have linked inhalation of air pollutants such as ozone to cardiovascular mortality. Human exposure studies have shown that inhalation of ambient levels of ozone causes airway and systemic inflammation and an imbalance in sympathetic/parasympathetic tone.
METHODS	To explore molecular mechanisms through which ozone inhalation contributes to cardiovascular mortality, we compared transcriptomics data previously obtained from bronchoalveolar lavage (BAL) cells obtained from healthy subjects after inhalational exposure to ozone (200 ppb for 4 h) to those of various cell samples from 11 published studies of patients with atherosclerotic disease using the Nextbio genomic data platform. Overlapping gene ontologies that may be involved in the transition from pulmonary to systemic vascular inflammation after ozone inhalation were explored. Local and systemic enzymatic activity of an overlapping upregulated gene, matrix metalloproteinase-9 (MMP-9), was measured by zymography after ozone exposure.
RESULTS	A set of differentially expressed genes involved in response to stimulus, stress, and wounding were in common between the ozone and most of the atherosclerosis studies. Many of these genes contribute to biological processes such as cholesterol metabolism dysfunction, increased monocyte adherence, endothelial cell lesions, and matrix remodeling, and to diseases such as heart failure, ischemia, and atherosclerotic occlusive disease. Inhalation of ozone increased MMP-9 enzymatic activity in both BAL fluid and serum.
CONCLUSIONS	Comparison of transcriptomics between BAL cells after ozone exposure and various cell types from patients with atherosclerotic disease reveals commonly regulated processes and potential mechanisms by which ozone inhalation may contribute to progression of pre-existent atherosclerotic lesions.

표-509. PubMed 논문번호 28417138의 내용 요약

구분	내용
PubMed ID	28417138
TITLE	Long-term exposure to ambient air pollution and traffic noise and incident hypertension in seven cohorts of the European study of cohorts for air pollution effects (ESCAPE).
JOURNAL	European heart journal: 10.1093/eurheartj/ehw413
AUTHORS	Fuks Kateryna B, Weinmayr Gudrun, Basaga□a Xavier, Gruzieva Olena, Hampel Regina, Oftedal Bente, S ø rensen Mette, Wolf Kathrin, Aamodt Geir, Aasvang Gunn Marit, Aguilera Inmaculada, Becker Thomas, Beelen Rob, Brunekreef Bert, Caracciolo Barbara, Cyrys Josef, Elosua Roberto, Eriksen Kirsten Thorup, Foraster Maria, Fratiglioni Laura, Hilding Agneta, Houthuijs Danny, Korek Michal, K□nzli Nino, Marrugat Jaume, Nieuwenhuijsen Mark, □stenson Claes-G□ran, Penell Johanna, Pershagen G□ran, Raaschou-Nielsen Ole, Swart Wim J R, Peters Annette, Hoffmann Barbara
Aims	We investigated whether traffic-related air pollution and noise are associated with incident hypertension in European cohorts.
Methods and results	We included seven cohorts of the European study of cohorts for air pollution effects (ESCAPE). We modelled concentrations of particulate matter with aerodynamic diameter ≤2.5 □m (PM2.5), ≤10 □m (PM10), 〉2.5, and ≤10 □m (PMcoarse), soot (PM2.5 absorbance), and nitrogen oxides at the addresses of participants with land use regression. Residential exposure to traffic noise was modelled at the facade according to the EU Directive 2002/49/EC. We assessed hypertension as (i) self-reported and (ii) measured (systolic BP ≥ 140 mmHg or diastolic BP ≥ 90 mmHg or intake of BP lowering medication (BPLM). We used Poisson regression with robust variance estimation to analyse associations of traffic-related exposures with incidence of hypertension, controlling for relevant confounders, and combined the results from individual studies with random-effects meta-analysis. Among 41 072 participants free of self-reported hypertension at baseline, 6207 (15.1%) incident cases occurred within 5-9 years of follow-up. Incidence of self-reported hypertension was positively associated with PM2.5 (relative risk (RR) 1.22 [95%-confidence interval (CI):1.08; 1.37] per 5 □g/m^3) and PM2.5 absorbance (RR 1.13 [95% CI:1.02; 1.24] per 10 − 5m − 1). These estimates decreased slightly upon adjustment for road traffic noise. Road traffic noise was weakly positively associated with the incidence of self-reported hypertension. Among 10 896 participants at risk, 3549 new cases of measured hypertension occurred. We found no clear associations with measured hypertension.
Conclusion	Long-term residential exposures to air pollution and noise are associated with increased incidence of self-reported hypertension.

표-510. PubMed 논문번호 28425896의 내용 요약

구분	내용
PubMed ID	28425896
TITLE	A longitudinal cohort study examining determinants of overweight and obesity in adulthood.
JOURNAL	Canadian journal of public health = Revue canadienne de sante publique: 10.17269/cjph.108.5772
AUTHORS	Barakat-Haddad Caroline, Saeed Usman, Elliott Susan
OBJECTIVES	Adulthood overweight and obesity are multifaceted conditions influenced by a combination of biological, environmental and socio-cultural factors across the lifespan. Using a longitudinal study design, we aimed to identify determinants of adulthood overweight and obesity, in relation to: 1) childhood and life course factors, 2) geographical differences in air quality, and 3) gender-specific factors, in a cohort followed from childhood into adulthood.
METHODS	Childhood data were acquired (1978-1986) from children residing in four distinct Hamilton neighbourhoods (Ontario, Canada), including air-quality assessments. Adulthood data were obtained (2006-2007) from successfully retraced participants (n = 315) using comprehensive self-administered questionnaires. Multivariate logistic regressions were used to evaluate determinants of adulthood overweight (BMI: 25-29.9 kg/m2) and obesity (BMI: ≥30).
RESULTS	The prevalence of normal weight decreased drastically at follow-up in adulthood, while that of overweight and obesity increased. Both overweight and obesity in adulthood were associated with male gender and occupational exposures to contaminants. Childhood residence in Hamilton neighbourhoods with better air quality was associated with lesser odds of adulthood overweight, whereas adulthood obesity was strongly linked to childhood weight gain (overweight or obesity). Among females, childhood weight status predicted overweight and obesity in adulthood, with always living in Hamilton, lack of additional health insurance, negative self-appraisal and high blood pressure during adulthood identified as other significant predictors. Among males, prolonged occupational exposures to contaminants emerged as a unique determinant of adulthood weight gain.
CONCLUSION	Adulthood overweight and obesity are associated with childhood and life course determinants, including childhood weight status, residential air quality and occupational contaminant exposures, in a gender-specific manner.

표-511. PubMed 논문번호 28455324의 내용 요약

구분	내용
PubMed ID	28455324
TITLE	Long-Term Exposure to Ambient Air Pollution and Subclinical Cerebrovascular Disease in NOMAS (the Northern Manhattan Study).
JOURNAL	Stroke: 10.1161/STROKEAHA.117.016672
AUTHORS	Kulick Erin R, Wellenius Gregory A, Kaufman Joel D, DeRosa Janet T, Kinney Patrick L, Cheung Ying Kuen, Wright Clinton B, Sacco Ralph L, Elkind Mitchell S
BACKGROUND AND PURPOSE	Long-term exposure to ambient air pollution is associated with higher risk of cardiovascular disease and stroke. We hypothesized that long-term exposure to air pollution would be associated with magnetic resonance imaging markers of subclinical cerebrovascular disease.
METHODS	Participants were 1075 stroke-free individuals aged ≥50 years drawn from the magnetic resonance imaging subcohort of the Northern Manhattan Study who had lived at the same residence for at least 2 years before magnetic resonance imaging. Cross-sectional associations between ambient air pollution and subclinical cerebrovascular disease were analyzed.
RESULTS	We found an association between distance to roadway, a proxy for residential exposure to traffic pollution, and white matter hyperintensity volume; however, after adjusting for risk factors, this relationship was no longer present. All other associations between pollutant measures and white matter hyperintensity volume were null. There was no clear association between exposure to air pollutants and subclinical brain infarcts or total cerebral brain volume.
CONCLUSIONS	We found no evidence that long-term exposure to ambient air pollution is independently associated with subclinical cerebrovascular disease in an urban population-based cohort.

표-512. PubMed 논문번호 28456465의 내용 요약

구분	내용
PubMed ID	28456465
TITLE	Fine Particulate Matter (PM_{2.5}) and the Risk of Stroke in the REGARDS Cohort.
JOURNAL	Journal of stroke and cerebrovascular diseases : the official journal of National Stroke Association: 10.1016/j.jstrokecerebrovasdis.2017.03.041
AUTHORS	McClure Leslie A, Loop Matthew S, Crosson William, Kleindorfer Dawn, Kissela Brett, Al-Hamdan Mohammad
BACKGROUND	Ambient particulate matter has been shown to be associated with declining human health, although the association between fine particulate matter (PM2.5) and stroke is uncertain.
METHODS	We utilized satellite-derived measures of PM2.5 to examine the association between exposure and stroke in the REasons for Geographic And Racial Differences in Stroke (REGARDS) study. We used a time-stratified case-crossover design, with exposure lags of 1 day, 2 days, and 3 days. We examined all strokes, as well as ischemic and hemorrhagic strokes separately.
RESULTS	Among 30,239 participants in the REGARDS study, 746 incident events were observed: 72 hemorrhagic, 617 ischemic, and 57 of unknown type. Participants exposed to higher levels of PM2.5 more often resided in urban areas compared to rural, and in the southeastern United States. After adjustment for temperature and relative humidity, no association was observed between PM2.5 exposure and stroke, regardless of the lag (1-day lag OR = .99, 95% CI: .83-1.19; 2-day lag OR = .95, 95% CI: .80-1.14; 3-day lag OR = .95, 95% CI = .79-1.13). Similar results were observed for the stroke subtypes.
CONCLUSIONS	In this large cohort of African-Americans and whites, no association was observed between PM2.5 and stroke. The ability to examine this association with a large number of outcomes and by stroke subtype helps fill a gap in the literature examining the association between PM2.5 and stroke.

표-513. PubMed 논문번호 28478397의 내용 요약

구분	내용
PubMed ID	28478397
TITLE	Copenhagen Airport Cohort: air pollution, manual baggage handling and health.
JOURNAL	BMJ open: 10.1136/bmjopen-2016-012651
AUTHORS	M ø ller Karina Lauenborg, Brauer Charlotte, Mikkelsen Sigurd, Loft Steffen, Simonsen Erik B, Koblauch Henrik, Bern Stine Hvid, Alkjær Tine, Hertel Ole, Becker Thomas, Larsen Karin Helweg, Bonde Jens Peter, Thygesen Lau Caspar
PURPOSE	Copenhagen Airport Cohort 1990-2012 presents a unique data source for studies of health effects of occupational exposure to air pollution (ultrafine particles) and manual baggage handling among airport employees. We describe the extent of information in the cohort and in the follow-up based on data linkage to the comprehensive Danish nationwide health registers. In the cohort, all information is linked to the personal identification number that also is used in Denmark Statistics demographic and socioeconomic databases and in the nationwide health registers.
PARTICIPANTS	The cohort covers 69 175 men in unskilled positions. The exposed cohort includes men in unskilled jobs employed at Copenhagen Airport in the period 1990-2012 either as baggage handlers or in other outdoor work. The reference cohort includes men in unskilled jobs working in the greater Copenhagen area.
FINDINGS TO DATE	The cohort includes environmental Global Positioning System (GPS) measurements in Copenhagen Airport, information on job function/task for each calendar year of employment between 1990 and 2012, exposure to air pollution at residence, average weight of baggage lifted per day and lifestyle. By linkage to registers, we retrieved socioeconomic and demographic data and data on healthcare contacts, drug subscriptions, incident cancer and mortality.
FUTURE PLANS	The size of the cohort and the completeness of the register-based follow-up allow a more accurate assessment of the possible health risks of occupational exposure to ultrafine particles and manual baggage handling at airports than in previous studies. We plan to follow the cohort for the incidence of ischaemic heart diseases, cerebrovascular disease, lung and bladder cancer, asthma and chronic obstructive pulmonary disease, and further for associations between heavy manual baggage handling and musculoskeletal disorders.
TRIAL REGISTRATION	number 2012-41-0199.

표-514. PubMed 논문번호 28479961의 내용 요약

구분	내용
PubMed ID	28479961
TITLE	A Systematic Review on the Effects of Polycyclic Aromatic Hydrocarbons on Cardiometabolic Impairment.
JOURNAL	International journal of preventive medicine: 10.4103/ijpvm.IJPVM_144_17
AUTHORS	Poursafa Parinaz, Moosazadeh Mahmood, Abedini Ehsan, Hajizadeh Yaghoub, Mansourian Marjan, Pourzamani Hamidreza, Amin Mohammad-Mehdi
BACKGROUND	Various epidemiological studies have shown that exposure to environmental pollutants including polycyclic aromatic hydrocarbons (PAHs) might increase the risk of cardiovascular diseases (CVDs) and their risk factors. This study aims to systematically review the association of PAH exposure with metabolic impairment.
METHODS	Data were collected by searching for relevant studies in international databases using the following keywords: "polycyclic aromatic hydrocarbon" + "cardiovascular disease," PAH + CVD, polycyclic aromatic hydrocarbon and "air pollutant" + "CVD," and the desired data were extracted and included in the study according to the systematic review process.
RESULTS	From the 14 articles included in the present systematic review, eight articles were conducted on the relationship between PAH and CVDs, four articles were conducted to examine the association of PAH exposure with blood pressure (BP), and two articles investigated the link between PAH and obesity.
CONCLUSIONS	Most studies included in this systematic review reported a significant positive association of PAH exposure with increased risk of CVDs and its major risk factors including elevated BP and obesity. These findings should be confirmed by longitudinal studies with long-term follow-up.

표-515. PubMed 논문번호 28521192의 내용 요약

구분	내용
PubMed ID	28521192
TITLE	Long-term exposure to residential ambient fine and coarse particulate matter and incident hypertension in post-menopausal women.
JOURNAL	Environment international: 10.1016/j.envint.2017.05.009
AUTHORS	Honda Trenton, Eliot Melissa N, Eaton Charles B, Whitsel Eric, Stewart James D, Mu Lina, Suh Helen, Szpiro Adam, Kaufman Joel D, Vedal Sverre, Wellenius Gregory A
BACKGROUND	Long-term exposure to ambient particulate matter (PM) has been previously linked with higher risk of cardiovascular events. This association may be mediated, at least partly, by increasing the risk of incident hypertension, a key determinant of cardiovascular risk. However, whether long-term exposure to PM is associated with incident hypertension remains unclear.
METHODS	Using national geostatistical models incorporating geographic covariates and spatial smoothing, we estimated annual average concentrations of residential fine (PM2.5), respirable (PM10), and course (PM10-2.5) fractions of particulate matter among 44,255 post-menopausal women free of hypertension enrolled in the Women's Health Initiative (WHI) clinical trials. We used time-varying Cox proportional hazards models to evaluate the association between long-term average residential pollutant concentrations and incident hypertension, adjusting for potential confounding by sociodemographic factors, medical history, neighborhood socioeconomic measures, WHI study clinical site, clinical trial, and randomization arm.
RESULTS	During 298,383 person-years of follow-up, 14,511 participants developed incident hypertension. The adjusted hazard ratios per interquartile range (IQR) increase in PM2.5, PM10, and PM10-2.5 were 1.13 (95% CI: 1.08, 1.17), 1.06 (1.03, 1.10), and 1.01 (95% CI: 0.97, 1.04), respectively. Statistically significant concentration-response relationships were identified for PM2.5 and PM10 fractions. The association between PM2.5 and hypertension was more pronounced among non-white participants and those residing in the Northeastern United States.
CONCLUSIONS	In this cohort of post-menopausal women, ambient fine and respirable particulate matter exposures were associated with higher incidence rates of hypertension. These results suggest that particulate matter may be an important modifiable risk factor for hypertension.

표-516. PubMed 논문번호 28541501의 내용 요약

구분	내용
PubMed ID	28541501
TITLE	Satellite-based estimates of long-term exposure to fine particulate matter are associated with C-reactive protein in 30 034 Taiwanese adults.
JOURNAL	International journal of epidemiology: 10.1093/ije/dyx069
AUTHORS	Zhang Zilong, Chang Ly-Yun, Lau Alexis K H, Chan Ta-Chien, Chieh Chuang Yuan, Chan Jimmy, Lin Changqing, Kai Jiang Wun, Dear Keith, Zee Benny C Y, Yeoh Eng-Kiong, Hoek Gerard, Tam Tony, Qian Lao Xiang
Background	Particulate matter (PM) air pollution is associated with the risk of cardiovascular morbidity and mortality. However, the biological mechanism underlying the associations remains unclear. Atherosclerosis, the underlying pathology of cardiovascular disease, is a chronic inflammatory process. We therefore investigated the association of long-term exposure to fine PM (PM2.5) with C-reactive protein (CRP), a sensitive marker of systemic inflammation, in a large Taiwanese population.
Methods	Participants were from a large cohort who participated in a standard medical examination programme with measurements of high-sensitivity CRP between 2007 and 2014. We used a spatiotemporal model to estimate 2-year average PM2.5 exposure at each participant's address, based on satellite-derived aerosol optical depth data. General regression models were used for baseline data analysis and mixed-effects linear regression models were used for repeated data analysis to investigate the associations between PM2.5 exposure and CRP, adjusting for a wide range of potential confounders.
Results	In this population of 30 034 participants with 39 096 measurements, every 5 μg/m3 PM2.5 increment was associated with a 1.31% increase in CRP [95% confidence interval (CI): 1.00%, 1.63%) after adjusting for confounders. For those participants with repeated CRP measurements, no significant changes were observed between the first and last measurements (0.88 mg/l vs 0.89 mg/l, P = 0.337). The PM2.5 concentrations remained stable over time between 2007 and 2014.
Conclusions	Long-term exposure to PM2.5 is associated with increased level of systemic inflammation, supporting the biological link between PM2.5 air pollution and deteriorating cardiovascular health. Air pollution reduction should be an important strategy to prevent cardiovascular disease.

표-517. PubMed 논문번호 28557713의 내용 요약

구분	내용
PubMed ID	28557713
TITLE	Effects of Prenatal PM〈sub〉10〈/sub〉 Exposure on Fetal Cardiovascular Malformations in Fuzhou, China: A Retrospective Case-Control Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP289
AUTHORS	Liu Chao-Bin, Hong Xin-Ru, Shi Miao, Chen Xiao-Qiu, Huang Hui-Juan, Chen Jin-Hua, Yang Kai, Chen Su-Qing, Chen Han-Qiang, Kan Hai-Dong, Sun Qing-Hua
BACKGROUND	Maternal exposure to ambient air pollution has been associated with an increased risk of congenital heart defects in offspring; however, the results are inconsistent.
OBJECTIVES	We investigated whether there is an association between prenatal exposure to particulate matter with diameter $\leq 10\,\mu$m (PM10) during early pregnancy and fetal cardiovascular malformations.
METHODS	The gravidae from a hospital-based case-control study in Fuzhou, China, during 2007-2013 were assigned 10-d or 1-mo averages of daily PM10 using an air monitor-based inverse distance weighting method during early pregnancy. A total of 662 live-birth or selectively terminated cases and 3,972 live-birth controls were enrolled. The exposure was considered as a categorical variable. A multivariable logistic regression model was constructed to quantify the adjusted odds ratios (aORs) of the exposure to PM10 and the risks of fetal cardiovascular malformations.
RESULTS	PM10 levels were positively associated with the risks of atrial septal defect (aORs ranging from 1.29 to 2.17), patent ductus arteriosus [aORs = 1.54, 1.63; 95% confidence intervals (CIs): 1.17, 2.23; 1.06, 3.24], overall fetal cardiovascular malformations (aOR = 1.28; 95% CI: 1.03, 1.61), ventricular septal defect (aOR = 1.19; 95% CI: 1.00, 1.43), and tetralogy of Fallot (aOR = 1.44; 95% CI: 1.01, 2.19) in the various observed periods scaled by 10 d or 1 mo in the first and second gestation months. The strongest associations were observed for exposure to PM10 in the second quartile, whereas the associations were attenuated when higher concentrations of PM10 in the third and fourth quartiles of the exposure were evaluated. No correlations of PM10 levels with these cardiovascular malformations in the other time periods of gestation were observed.
CONCLUSIONS	Our findings suggest some positive associations between maternal exposure to ambient PM10 during the first two months of pregnancy and fetal cardiovascular malformations. https://doi.org/10.1289/EHP289.

표-518. PubMed 논문번호 28558275의 내용 요약

구분	내용
PubMed ID	28558275
TITLE	Heat and mortality for ischemic and hemorrhagic stroke in 12 cities of Jiangsu Province, China.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2017.05.169
AUTHORS	Zhou Lian, Chen Kai, Chen Xiaodong, Jing Yuanshu, Ma Zongwei, Bi Jun, Kinney Patrick L
BACKGROUND	Little evidence exists on the relationship between heat and subtypes of stroke mortality, especially in China. Moreover, few studies have reported the effect modification by individual characteristics on heat-related stroke mortality. In this study, we aimed to evaluate the effect of heat exposure on total, ischemic, and hemorrhagic stroke mortality and its individual modifiers in 12 cities in Jiangsu Province, China during 2009 to 2013.
METHODS	We first used a distributed lag non-linear model with quasi-Poisson regression to examine the city-specific heat-related total, ischemic, and hemorrhagic stroke mortality risks at 99th percentile vs. 75th percentile of daily mean temperature in the whole year for each city, while adjusting for long-term trend, season, relative humidity, and day of the week. Then, we used a random-effects meta-analysis to pool the city-specific risk estimates. We also considered confounding by air pollution and effect modification by gender, age, education level, and death location.
RESULTS	Overall, the heat-related mortality risk in 12 Jiangsu cities was 1.54 (95%CI: 1.44 to 1.65) for total stroke, 1.63 (95%CI: 1.48 to 1.80) for ischemic stroke, and 1.36 (95%CI: 1.26 to 1.48) for hemorrhagic stroke, respectively. Estimated total, ischemic, and hemorrhagic stroke mortality risks were higher for women versus men, older people versus younger people, those with low education levels versus high education levels, and deaths that occurred outside of hospital. Air pollutants did not significantly influence the heat-related stroke mortality risk.
CONCLUSIONS	Heat exposure significantly increased both ischemic and hemorrhagic stroke mortality risks in Jiangsu Province, China. Females, the elderly, and those with low education levels are particularly vulnerable to this effect.

표-519. PubMed 논문번호 28575405의 내용 요약

구분	내용
PubMed ID	28575405
TITLE	Long-term exposure to road traffic noise, ambient air pollution, and cardiovascular risk factors in the HUNT and lifelines cohorts.
JOURNAL	European heart journal: 10.1093/eurheartj/ehx263
AUTHORS	Cai Yutong, Hansell Anna L, Blangiardo Marta, Burton Paul R, de Hoogh Kees, Doiron Dany, Fortier Isabel, Gulliver John, Hveem Kristian, Mbatchou St□phane, Morley David W, Stolk Ronald P, Zijlema Wilma L, Elliott Paul, Hodgson Susan
Aims	Blood biochemistry may provide information on associations between road traffic noise, air pollution, and cardiovascular disease risk. We evaluated this in two large European cohorts (HUNT3, Lifelines).
Methods and results	Road traffic noise exposure was modelled for 2009 using a simplified version of the Common Noise Assessment Methods in Europe (CNOSSOS-EU). Annual ambient air pollution (PM10, NO2) at residence was estimated for 2007 using a Land Use Regression model. The statistical platform DataSHIELD was used to pool data from 144 082 participants aged ≥20 years to enable individual-level analysis. Generalized linear models were fitted to assess cross-sectional associations between pollutants and high-sensitivity C-reactive protein (hsCRP), blood lipids and for (Lifelines only) fasting blood glucose, for samples taken during recruitment in 2006-2013. Pooling both cohorts, an inter-quartile range (IQR) higher day-time noise (5.1 dB(A)) was associated with 1.1% [95% confidence interval (95% CI: 0.02-2.2%)] higher hsCRP, 0.7% (95% CI: 0.3-1.1%) higher triglycerides, and 0.5% (95% CI: 0.3-0.7%) higher high-density lipoprotein (HDL); only the association with HDL was robust to adjustment for air pollution. An IQR higher PM10 (2.0 □g/m3) or NO2 (7.4 □g/m3) was associated with higher triglycerides (1.9%, 95% CI: 1.5-2.4% and 2.2%, 95% CI: 1.6-2.7%), independent of adjustment for noise. Additionally for NO2, a significant association with hsCRP (1.9%, 95% CI: 0.5-3.3%) was seen. In Lifelines, an IQR higher noise (4.2 dB(A)) and PM10 (2.4 □g/m3) was associated with 0.2% (95% CI: 0.1-0.3%) and 0.6% (95% CI: 0.4-0.7%) higher fasting glucose respectively, with both remaining robust to adjustment for air/noise pollution.
Conclusion	Long-term exposures to road traffic noise and ambient air pollution were associated with blood biochemistry, providing a possible link between road traffic noise/air pollution and cardio-metabolic disease risk.

표-520. PubMed 논문번호 28575495의 내용 요약

구분	내용
PubMed ID	28575495
TITLE	Maternal Smoking: A Life Course Blood Pressure Determinant?
JOURNAL	Nicotine & tobacco research : official journal of the Society for Research on Nicotine and Tobacco: 10.1093/ntr/ntx117
AUTHORS	Cabral Maria, Fonseca Maria J, Gonz□lez-Beiras Camila, Santos Ana C, Correia-Costa Liane, Barros Henrique
Introduction	Exposure to maternal smoking early in life may affect blood pressure (BP) control mechanisms. We examined the association between maternal smoking (before conception, during pregnancy, and 4 years after delivery) and BP in preschool children.
Methods	We evaluated 4295 of Generation XXI children, recruited at birth in 2005-2006 and reevaluated at the age of 4. At birth, information was collected by face-to-face interview and additionally abstracted from clinical records. At 4-year follow-up, interviews were performed and children's BP measured. Linear regression models were fitted to estimate the association between maternal smoking and children's BP.
Results	Children of smoking mothers presented significantly higher BP levels. After adjustment for maternal education, gestational hypertensive disorders, and child's body mass index, children exposed during pregnancy to maternal smoking presented a higher systolic BP (SBP) z-score (β = 0.08, 95% confidence interval [CI] 0.04 to 0.14). In crude models, maternal smoking was associated with higher SBP z-score at every assessed period. However, after adjustment, an attenuation of the association estimates occurred (β = 0.08, 95% CI 0.03 to 0.13 before conception; β = 0.07, 95%CI 0.02 to 0.12; β = 0.04, 95%CI -0.02 to 0.10; and β = 0.06, 95%CI 0.00 to 0.13 for the first, second, and third pregnancy trimesters, respectively; and β = 0.07, 95%CI 0.02 to 0.12 for current maternal smoking). No significant association was observed for diastolic BP z-score levels.
Conclusion	Maternal smoking before, during, and after pregnancy was independently associated with systolic BP z-score in preschool children. This study provides additional evidence to the public health relevance of maternal smoking cessation programs if early cardiovascular health of children is envisaged.
Implications	Using observational longitudinal data from the birth cohort Generation XXI, this study showed that exposure to maternal smoking-before pregnancy, during pregnancy, and 4 years after delivery-was associated with a systolic BP-raising effect in children at the age of 4. The findings of this study add an important insight into the need to support maternal smoke-free environments in order to provide long-term cardiovascular benefit, starting as early as possible in life.

표-521. PubMed 논문번호 28600978의 내용 요약

구분	내용
PubMed ID	28600978
TITLE	Long-term effects of total and source-specific particulate air pollution on incident cardiovascular disease in Gothenburg, Sweden.
JOURNAL	Environmental research: 10.1016/j.envres.2017.05.036
AUTHORS	Stockfelt Leo, Andersson Eva M, Moln□r Peter, Gidhagen Lars, Segersson David, Rosengren Annika, Barregard Lars, Sallsten Gerd
BACKGROUND AND AIMS	Long-term exposure to air pollution increases cardiopulmonary morbidity and mortality, but it is not clear which components of air pollution are the most harmful, nor which time window of exposure is most relevant. Further studies at low exposure levels have also been called for. We analyzed two Swedish cohorts to investigate the effects of total and source-specific particulate matter (PM) on incident cardiovascular disease for different time windows of exposure.
METHODS	Two cohorts initially recruited to study predictors of cardiovascular disease (the PPS cohort and the GOT-MONICA cohort) were followed from 1990 to 2011. We collected data on residential addresses and assigned each individual yearly total and source-specific PM and Nitrogen Oxides (NOx) exposures based on dispersion models. Using multivariable Cox regression models with time-dependent exposure, we studied the association between three different time windows (lag 0, lag 1-5, and exposure at study start) of residential PM and NOx exposure, and incidence of ischemic heart disease, stroke, heart failure and atrial fibrillation.
RESULTS AND DISCUSSION	During the study period, there were 2266 new-onset cases of ischemic heart disease, 1391 of stroke, 925 of heart failure and 1712 of atrial fibrillation. The majority of cases were in the PPS cohort, where participants were older. Exposure levels during the study period were moderate (median: 13□g/m3 for PM10 and 9□g/m3 for PM2.5), and similar in both cohorts. Road traffic and residential heating were the largest local sources of PM air pollution, and long distance transportation the largest PM source in total. In the PPS cohort, there were positive associations between PM in the last five years and both ischemic heart disease (HR: 1.24 [95% CI: 0.98-1.59] per 10□g/m3 of PM10, and HR: 1.38 [95% CI: 1.08-1.77] per 5□g/m3 of PM2.5) and heart failure. In the GOT-MONICA cohort, there were positive but generally non-significant associations between PM and stroke (HR: 1.48 [95% CI: 0.88-2.49] per 10□g/m3 of PM10, and HR: 1.50 [95% CI: 0.90-2.51] per 5□g/m3 of PM2.5, in the last five years). Effect estimates were stronger for women, non-smokers, and higher socioeconomic classes. Exposure in the last five years seemed to be more strongly associated with outcomes than other exposure time windows. Associations between source-specific PM air pollution and outcomes were mixed and generally weak. High correlations between the main pollutants limited the use of multi-pollutant models.
CONCLUSIONS	The main PM air pollutants were associated with ischemic heart disease and stroke (in women) at the relatively low exposure levels in Gothenburg, Sweden. The associations tended to be stronger for women than for men, for non-smokers than for smokers, and for higher socioeconomic classes than for lower. The associations could not be attributed to a specific PM source or type, and differed somewhat between the two cohorts. The results of this study confirm that further efforts to reduce air pollution exposure should be undertaken in Sweden to reduce the negative health effects in the general population.

표-522. PubMed 논문번호 28615066의 내용 요약

구분	내용
PubMed ID	28615066
TITLE	Air pollution, cardiovascular endpoints and susceptibility by stress and material resources: a systematic review of the evidence.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-017-0270-0
AUTHORS	Fuller Christina H, Feeser Karla R, Sarnat Jeremy A, O'Neill Marie S
BACKGROUND AND METHODS	Evidence shows that both the physical and social environments play a role in the development of cardiovascular disease. The purpose of this systematic review is two-fold: First, we summarize research from the past 12 years from the growing number of studies focused on effect modification of the relationships between air pollution and cardiovascular disease (CVD) outcomes by socioeconomic position (SEP) and; second, we identify research gaps throughout the published literature on this topic and opportunities for addressing these gaps in future study designs.
RESULTS	We identified 30 articles that examined the modifying effects of either material resources or psychosocial stress (both related to SEP) on associations between short and long-term air pollution exposure and CVD endpoints. Although 18 articles identified at least one interaction between an air pollutant and material resource indicator, 11 others did not. Support for susceptibility to air pollution by psychosocial stress was weaker; however, only three articles tested this hypothesis. Further studies are warranted to investigate how air pollution and SEP together may influence CVD.
CONCLUSIONS	We recommend that such research include thorough assessment of air pollution and SEP correlations, including spatial correlation; investigate air pollution indices or multi-pollutant models; use standardized metrics of SEP to enhance comparability across studies; and evaluate potentially susceptible populations.

표-523. PubMed 논문번호 28629640의 내용 요약

구분	내용
PubMed ID	28629640
TITLE	Is physical activity a modifier of the association between air pollution and arterial stiffness in older adults: The SAPALDIA cohort study.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2017.06.001
AUTHORS	Endes Simon, Schaffner Emmanuel, Caviezel Seraina, Dratva Julia, Stolz Daiana, Schindler Christian, K□nzli Nino, Schmidt-Trucks□ss Arno, Probst-Hensch Nicole
INTRODUCTION AND OBJECTIVES	Air pollution and insufficient physical activity have been associated with inflammation and oxidative stress, molecular mechanisms linked to arterial stiffness and cardiovascular disease. There are no studies on how physical activity modifies the association between air pollution and arterial stiffness. We examined whether the adverse cardiovascular effects of air pollution were modified by individual physical activity levels in 2823 adults aged 50-81 years from the well-characterized Swiss Cohort Study on Air Pollution and Lung and Heart Diseases (SAPALDIA).
METHODS	We assessed arterial stiffness as the brachial-ankle pulse wave velocity (baPWV [m/s]) with an oscillometric device. We administered a self-reported physical activity questionnaire to classify each subject's physical activity level. Air pollution exposure was estimated by the annual average individual home outdoor PM10 and PM2.5 (particulate matter $<10\,\mu$m and $<2.5\,\mu$m in diameter, respectively) and NO2 (nitrogen dioxide) exposure estimated for the year preceding the survey. Exposure estimates for ultrafine particles calculated as particle number concentration (PNC) and lung deposited surface area (LDSA) were available for a subsample (N=1353). We used mixed effects logistic regression models to regress increased arterial stiffness (baPWV≥14.4m/s) on air pollution exposure and physical activity while adjusting for relevant confounders.
RESULTS	We found evidence that the association of air pollution exposure with baPWV was different between inactive and active participants. The probability of having increased baPWV was significantly higher with higher PM10, PM2.5, NO2, PNC and LDSA exposure in inactive, but not in physically active participants. We found some evidence of an interaction between physical activity and ambient air pollution exposure for PM10, PM2.5 and NO2 (pinteraction=0.06, 0.09, and 0.04, respectively), but not PNC and LDSA (pinteraction=0.32 and 0.35).
CONCLUSIONS	Our study provides some indication that physical activity may protect against the adverse vascular effects of air pollution in low pollution settings. Additional research in large prospective cohorts is needed to assess whether the observed effect modification translates to high pollution settings in mega-cities of middle and low-income countries.

표-524. PubMed 논문번호 28632785의 내용 요약

구분	내용
PubMed ID	28632785
TITLE	The association between various smoking behaviors, cotinine biomarkers and skin autofluorescence, a marker for advanced glycation end product accumulation.
JOURNAL	PloS one: 10.1371/journal.pone.0179330
AUTHORS	van Waateringe Robert P, Mook-Kanamori Marjonneke J, Slagter Sandra N, van der Klauw Melanie M, van Vliet-Ostaptchouk Jana V, Graaff Reindert, Lutgers Helen L, Suhre Karsten, El-Din Selim Mohammed M, Mook-Kanamori Dennis O, Wolffenbuttel Bruce H R
BACKGROUND	Skin autofluorescence, a biomarker for advanced glycation end products (AGEs) accumulation, has been shown to predict diabetes-related cardiovascular complications and is associated with several environmental and lifestyle factors. In the present study, we examined the association between various smoking behaviors and skin autofluorescence, as well as the association between several cotinine biomarkers and skin autofluorescence, using both epidemiological and metabolomics data.
METHODS	In a cross-sectional study, we evaluated participants from the LifeLines Cohort Study and the Qatar Metabolomics Study on Diabetes (QMDiab). In the LifeLines Cohort Study smoking behavior and secondhand smoking were assessed in 8,905 individuals including 309 individuals (3.5%) with type 2 diabetes. In QMDiab, cotinine biomarkers were measured in saliva, plasma and urine in 364 individuals of whom 188 (51%) had type 2 diabetes. Skin autofluorescence was measured non-invasively in all participants using the AGE Reader.
RESULTS	Skin autofluorescence levels increased with a higher number of hours being exposed to secondhand smoking. Skin autofluorescence levels of former smokers approached levels of never smokers after around 15 years of smoking cessation. Urinary cotinine N-oxide, a biomarker of nicotine exposure, was found to be positively associated with skin autofluorescence in the QMDiab study ($p = 0.03$).
CONCLUSIONS	In the present study, we have demonstrated that secondhand smoking is associated with higher skin autofluorescence levels whereas smoking cessation has a beneficial effect on skin autofluorescence. Finally, urinary cotinine N-oxide might be used as an alternative way for questionnaires to examine the effect of (environmental) tobacco smoking on skin autofluorescence.

표-525. PubMed 논문번호 28645070의 내용 요약

구분	내용
PubMed ID	28645070
TITLE	Passive smoking and mortality from aortic dissection or aneurysm.
JOURNAL	Atherosclerosis: 10.1016/j.atherosclerosis.2017.06.022
AUTHORS	Kihara Tomomi, Yamagishi Kazumasa, Iso Hiroyasu, Tamakoshi Akiko
BACKGROUND AND AIMS	Evidence on the association between passive smoking and risk of aortic dissection or aneurysm is limited. This study aimed to investigate whether passive smoking increases risk of mortality from aortic dissection or aneurysm.
METHODS	The Japan Collaborative Cohort (JACC) Study is a prospective community-based cohort study begun in 1988-90 and followed up to the end of 2009. We examined 48,677 individuals (mean age, 56 years; women, 46%) without history of stroke, coronary heart disease, or cancer, who provided valid responses to a lifestyle questionnaire including questions on active and passive smoking. We used 3 categories (passive smoking out of home, passive smoking at home, and passive smoking out of or at home combined) to divide never-smokers into 3 exposure groups: low, intermediate, and high exposures, respectively. The endpoint was underlying cause of death from aortic dissection or aneurysm.
RESULTS	During the median 19-year follow-up of 48,677 study participants, 66 died of aortic dissection, and 75 of aortic aneurysm. Multivariable hazard ratios (95% confidence intervals) for the high passive-smoking group as compared with the low passive-smoking group were 2.45 (1.02-5.88) out of home, 1.82 (0.84-3.96) at home, and 2.35 (1.09-5.09) out of or at home combined. The corresponding hazard ratios for current smokers as compared with the low passive-smoking group were 3.97 (2.14-7.39), 3.41 (1.84-6.32) and 4.09 (1.99-8.39), respectively.
CONCLUSIONS	Out-of-home passive smoking and out-of- or at-home combined passive smoking were associated with increased mortality from aortic dissection or aneurysm.

표-526. PubMed 논문번호 28662448의 내용 요약

구분	내용
PubMed ID	28662448
TITLE	Arterial blood pressure responses to short-term exposure to fine and ultrafine particles from indoor sources - A randomized sham-controlled exposure study of healthy volunteers.
JOURNAL	Environmental research: 10.1016/j.envres.2017.06.006
AUTHORS	Soppa Vanessa J, Schins Roel P F, Hennig Frauke, Nieuwenhuijsen Mark J, Hellack Bryan, Quass Ulrich, Kaminski Heinz, Sasse Birgitta, Shinnawi Samir, Kuhlbusch Thomas A J, Hoffmann Barbara
OBJECTIVES	Particulate air pollution is linked to adverse cardiovascular effects. The aim of the study was to investigate the effect of short-term exposure to indoor particles on blood pressure (BP).
METHODS	We analyzed the association of particle emissions from indoor sources (candle burning, toasting bread, frying sausages) with BP changes in 54 healthy volunteers in a randomized cross-over controlled exposure study. Particle mass concentration (PMC), size-specific particle number concentration (PNC) and lung-deposited particle surface area concentration (PSC) were measured during the 2h exposure. Systolic and diastolic blood pressure were measured before, during, directly, 2, 4 and 24h after exposure. We performed multiple mixed linear regression analyses of different particle metrics and BP.
RESULTS	BP significantly increased with increasing PMC, PSC and PNC resulting from toasting bread. For example, an increase per 10□g/m3 PM10 and PM2.5, systolic BP increased at all time points with largest changes 1h after exposure initiation of 1.5mmHg (95%-CI: 1.1; 1.9) and of 2.2mmHg (95%-CI: 1.3; 3.1), respectively.
CONCLUSIONS	Our study suggests an association of short-term exposure to fine and ultrafine particles emitted from toasting bread with increases in BP. Particles emitted from frying sausages and candle burning did not consistently affect BP.

표-527. PubMed 논문번호 28689210의 내용 요약

구분	내용
PubMed ID	28689210
TITLE	Residential Air Pollution, Road Traffic, Greenness and Maternal Hypertension: Results from GINIplus and LISAplus.
JOURNAL	The international journal of occupational and environmental medicine: 10.15171/ijoem.2017.1073
AUTHORS	Jendrossek Mario, Standl Marie, Koletzko Sibylle, Lehmann Irina, Bauer Carl-Peter, Schikowski Tamara, von Berg Andrea, Berdel Dietrich, Heinrich Joachim, Markevych Iana
BACKGROUND	The public health burden of hypertension is high, but its relationship with long-term residential air pollution, road traffic, and greenness remains unclear.
OBJECTIVE	To investigate associations between residential air pollution, traffic, greenness, and hypertension among mothers.
METHODS	Information on doctor-diagnosed maternal hypertension was collected at the 15-year follow-up of two large population-based multicenter German birth cohorts-GINIplus and LISAplus (n=3063). Residential air pollution was modelled by land use regression models within the ESCAPE and universal kriging within the APMoSPHERE projects. Road traffic was defined as traffic load on major roads within a 100-m buffer around residences. Vegetation level (ie, greenness) was defined as the mean Normalized Difference Vegetation Index in a 500-m buffer around residences and was assessed from Landsat 5 TM satellite images. All the exposure variables were averaged over three residential addresses during the last 10 years and categorized into tertiles or dichotomized. The individual associations between each of the exposure variables and hypertension were assessed using logistic regression analysis.
RESULTS	No significant and consistent associations across different levels of adjustment were observed between the exposures of interest and hypertension. The only significant estimate was found with coarse particulate matter concentrations (OR 1.66, 95% CI 1.01 to 2.74; 3rdvs 1st tertile) among mothers residing in the Wesel area. No significant associations were observed with traffic load or greenness.
CONCLUSION	This study does not provide evidence on detrimental effects of air pollution and road traffic or beneficial effects of greenness on hypertension among German adults.

표-528. PubMed 논문번호 28709636의 내용 요약

구분	내용
PubMed ID	28709636
TITLE	Effects of climate and fine particulate matter on hospitalizations and deaths for heart failure in elderly: A population-based cohort study.
JOURNAL	Environment international: 10.1016/j.envint.2017.06.001
AUTHORS	Vanasse Alain, Talbot Denis, Chebana Fateh, B□langer Diane, Blais Claudia, Gamache Philippe, Giroux Jean-Xavier, Dault Roxanne, Gosselin Pierre
BACKGROUND	There are limited data on the effects of climate and air pollutant exposure on heart failure (HF) within taking into account individual and contextual variables.
OBJECTIVES	We measured the lag effects of temperature, relative humidity, atmospheric pressure and fine particulate matter (PM2.5) on hospitalizations and deaths for HF in elderly diagnosed with this disease on a 10-year period in the province of Quebec, Canada.
METHODS	Our population-based cohort study included 112,793 elderly diagnosed with HF between 2001 and 2011. Time dependent Cox regression models approximated with pooled logistic regressions were used to evaluate the 3- and 7-day lag effects of daily temperature, relative humidity, atmospheric pressure and PM2.5 exposure on HF morbidity and mortality controlling for several individual and contextual covariates.
RESULTS	Overall, 18,309 elderly were hospitalized and 4297 died for the main cause of HF. We observed an increased risk of hospitalizations and deaths for HF with a decrease in the average temperature of the 3 and 7days before the event. An increase in atmospheric pressure in the previous 7days was also associated with a higher risk of having a HF negative outcome, but no effect was observed in the 3-day lag model. No association was found with relative humidity and with PM2.5 regardless of the lag period.
CONCLUSIONS	Lag effects of temperature and other meteorological parameters on HF events were limited but present. Nonetheless, preventive measures should be issued for elderly diagnosed with HF considering the burden and the expensive costs associated with the management of this disease.

표-529. PubMed 논문번호 28711846의 내용 요약

구분	내용
PubMed ID	28711846
TITLE	Vascular and cardiac autonomic function and PM〈sub〉2.5〈/sub〉 constituents among the elderly: A longitudinal study.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2017.07.077
AUTHORS	Lim Youn-Hee, Bae Hyun-Joo, Yi Seung-Muk, Park EunHa, Lee Bo-Eun, Hong Yun-Chul
BACKGROUND	Although epidemiologic studies have shown an association between the total mass of particulate matter 〈2.5 μm in aerodynamic diameter (PM2.5) and cardiovascular disease, few studies have examined PM2.5 constituents associated with vascular and cardiac autonomic dysfunction.
METHODS	In this longitudinal study, we investigated the relationship between PM2.5 constituents and blood pressure (BP), and markers of the autonomic nervous system. In 466 elderly subjects residing in communities in Seoul, Korea, we examined 16 constituents, seven sources, and total mass concentrations of PM2.5. We measured the BP, heart rate (HR), and indices of heart rate variability (HRV), such as the standard deviation of normal-to-normal intervals (SDNN), square root of the mean squared differences of successive NN intervals (rMSSD), and two frequency-domain variables (low frequency [LF] and high frequency [HF]). We used linear mixed effects models to assess the association of PM2.5 constituents and sources with cardiovascular markers.
RESULTS	BP, HR, and rMSSD were associated with concentration of total mass of PM2.5. For each increase of the interquartile range in PM2.5 constituents, systolic and diastolic BP, and HR increased by 2.1-3.3mmHg, 1.2-2.3mmHg, and 1.2-1.9bpm, respectively, while the rMSSD, LF, and HF decreased by 8.1-9.3%, 16.6%, and 20.4%, respectively. Particularly, elemental carbon, sulfate, ammonium, lead, and strontium in the PM2.5 constituents and emissions from oil combustion and incineration were associated with increased BP, HR, and decreased HRV.
CONCLUSIONS	Our results suggest an association between specific PM2.5 constituents and vascular and cardiac autonomic functions. These findings may provide supportive evidence for developing a pollution reduction plan to prevent cardiovascular diseases.

표-530. PubMed 논문번호 28724840의 내용 요약

구분	내용
PubMed ID	28724840
TITLE	Exposure to Parental Smoking in Childhood is Associated with High C-Reactive Protein in Adulthood: The Cardiovascular Risk in Young Finns Study.
JOURNAL	Journal of atherosclerosis and thrombosis: 10.5551/jat.40568
AUTHORS	Wang Di, Juonala Markus, Viikari Jorma S A, Wu Feitong, Hutri-K□h□nen Nina, Raitakari Olli T, Magnussen Costan G
AIM	Children exposed to parental smoking are at increased long-term risk of subclinical atherosclerosis in adulthood. However, it has not been quantified if exposure to parental smoking in childhood is associated with adult systemic inflammation. This study aimed to determine if childhood exposure to parental smoking was associated with high-sensitivity C-reactive protein (hsCRP) in adulthood.
METHODS	This longitudinal analysis of 2,511 participants used data from the Cardiovascular Risk in Young Finns Study, a prospective cohort of Finnish children. In 1980 or 1983, parents self-reported their smoking status and serum hsCRP was collected up to 31 years later in adulthood.
RESULTS	Compared with children with non-smoking parents, the relative risk of developing high hsCRP (>3 mg/L) in adulthood increased among those with 1 or both parents who smoked [relative risk (RR), 1.3; 95%confidence interval (CI), 1.0-1.8] after adjustment for socioeconomic status, cardiovascular risk factors, and smoking status in childhood and adulthood. Moreover, children exposed to mother smoking [RR, 2.4; 95% CI, 1.3-4.2] had highest risk of developing high hsCRP in adulthood compared with those exposed to father smoking [RR, 1.6; 95% CI, 1.2-2.3] and both parents smoking [RR, 1.4; 95% CI, 0.9-2.0].
CONCLUSION	Our findings suggest that children exposed to parental smoking are at increased risk of having high hsCRP in adulthood. Limiting children's exposure to passive smoking may have long-term benefits on general low-grade inflammation.

표-531. PubMed 논문번호 28732305의 내용 요약

구분	내용
PubMed ID	28732305
TITLE	Association between gaseous air pollutants and inflammatory, hemostatic and lipid markers in a cohort of midlife women.
JOURNAL	Environment international: 10.1016/j.envint.2017.07.004
AUTHORS	Wu Xiangmei May, Basu Rupa, Malig Brian, Broadwin Rachel, Ebisu Keita, Gold Ellen B, Qi Lihong, Derby Carol, Green Rochelle S
BACKGROUND	Exposures to ambient gaseous pollutants have been linked to cardiovascular diseases (CVDs), but the biological mechanisms remain uncertain.
OBJECTIVES	This study examined the changes in CVD marker levels resulting from elevated exposure to ambient gaseous pollutants in midlife women.
METHODS	Annual repeated measurements of several inflammatory, hemostatic and lipid makers were obtained from 2306 midlife women enrolled in the longitudinal Study of Women's Health Across the Nation (SWAN) between 1999 and 2004. Ambient carbon monoxide (CO), nitrogen dioxide (NO2), and sulfur dioxide (SO2) data were assigned to each woman based on proximity of the monitoring station to her residential address. Short- and long-term exposures were calculated, and their associations with markers were examined using linear mixed-effects regression models, adjusted for demographic, health and other factors.
RESULTS	Short-term CO exposure was associated with increased fibrinogen, i.e., every interquartile increase of average prior one-week exposure to CO was associated with 1.3% (95% CI: 0.6%, 2.0%) increase in fibrinogen. Long-term exposures to NO2 and SO2 were associated with reduced high-density lipoproteins and apolipoprotein A1, e.g., 4.0% (1.7%, 6.3%) and 4.7% (2.8%, 6.6%) decrease per interquartile increment in prior one-year average NO2 concentration, respectively. Fine particle (PM2.5) exposure confounded associations between CO/NO2 and inflammatory/hemostatic markers, while associations with lipoproteins were generally robust to PM2.5 adjustment.
CONCLUSIONS	Exposures to these gas pollutants at current ambient levels may increase thrombotic potential and disrupt cholesterol metabolism, contributing to greater risk of CVDs in midlife women. Caution should be exercised in evaluating the confounding by PM2.5 exposure.

표-532. PubMed 논문번호 28732501의 내용 요약

구분	내용
PubMed ID	28732501
TITLE	Ambulatory monitoring demonstrates an acute association between cookstove-related carbon monoxide and blood pressure in a Ghanaian cohort.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-017-0282-9
AUTHORS	Quinn Ashlinn K, Ae-Ngibise Kenneth Ayuurebobi, Kinney Patrick L, Kaali Seyram, Wylie Blair J, Boamah Ellen, Shimbo Daichi, Agyei Oscar, Chillrud Steven N, Mujtaba Mohammed, Schwartz Joseph E, Abdalla Marwah, Owusu-Agyei Seth, Jack Darby W, Asante Kwaku Poku
BACKGROUND	Repeated exposure to household air pollution may intermittently raise blood pressure (BP) and affect cardiovascular outcomes. We investigated whether hourly carbon monoxide (CO) exposures were associated with acute increases in ambulatory blood pressure (ABP); and secondarily, if switching to an improved cookstove was associated with BP changes. We also evaluated the feasibility of using 24-h ambulatory blood pressure monitoring (ABPM) in a cohort of pregnant women in Ghana.
METHODS	Participants were 44 women enrolled in the Ghana Randomized Air Pollution and Health Study (GRAPHS). For 27 of the women, BP was measured using 24-h ABPM; home blood pressure monitoring (HBPM) was used to measure BP in the remaining 17 women. Personal CO exposure monitoring was conducted alongside the BP monitoring.
RESULTS	ABPM revealed that peak CO exposure (defined as ≥4.1 ppm) in the 2 hours prior to BP measurement was associated with elevations in hourly systolic BP (4.3 mmHg [95% CI: 1.1, 7.4]) and diastolic BP (4.5 mmHg [95% CI: 1.9, 7.2]), as compared to BP following lower CO exposures. Women receiving improved cookstoves had lower post-intervention SBP (within-subject change in SBP of −2.1 mmHg [95% CI: −6.6, 2.4] as compared to control), though this result did not reach statistical significance. 98.1% of expected 24-h ABPM sessions were successfully completed, with 92.5% of them valid according to internationally defined criteria.
CONCLUSIONS	We demonstrate an association between acute exposure to carbon monoxide and transient increases in BP in a West African setting. ABPM shows promise as an outcome measure for assessing cardiovascular health benefits of cookstove interventions.
TRIAL REGISTRATION	The GRAPHS trial was registered with clinicaltrials.gov on 13 April 2011 with the identifier NCT01335490 .

표-533. PubMed 논문번호 28735151의 내용 요약

구분	내용
PubMed ID	28735151
TITLE	Do air pollution and neighborhood greenness exposures improve the predicted cardiovascular risk?
JOURNAL	Environment international: 10.1016/j.envint.2017.07.011
AUTHORS	Yitshak-Sade Maayan, Kloog Itai, Novack Victor
BACKGROUND	Numerous studies show associations between exposure to Particulate Matter and Cardiovascular disease (CVD). Current cardiovascular equations incorporate the major risk factors for CVD. The patients' environment, however, is not incorporated in these equations.
METHODS	In a retrospective analysis, we assessed the contribution of neighborhood greenness and particulate matter (coarse-PM and PM<2.5 μm-PM2.5) to the development of CVD by analyzing the change in prediction abilities. We included members of the largest health-care provider in Southern-Israel, who had at least one cardiovascular risk factor (dyslipidemia, diabetes, hypertension or smokers). PM exposure and neighborhood greenness (Normalized Difference Vegetation Index-NDVI) were assessed by satellite-based models. We used pooled logistic mixed regressions to obtain the CVD risks including conventional risk factors (i.e. age, gender, blood-pressure, etc.) and measured the model performance with and without PM and NDVI.
RESULTS	We included 23,110 subjects, of whom 12% had CVD. Coarse-PM exposure was associated with stroke and Myocardial-Infarction (MI) (OR 1.02,p<0.01 for both). NDVI was associated with MI: OR 0.72(p<0.01) for NDVI 0.1-0.2; and OR 0.52(p=0.270) for NDVI >0.2. The c-statistics slightly improved from 77.30%-77.40% for the prediction of MI (p=0.004) and from 75.60%-75.76% for the prediction of stroke (p=0.027). Calibration was fair in all models. The associations were partially mediated through the patients' comorbidities.
CONCLUSION	The negligible improvement in the prediction performance, despite significant associations with PM and NDVI, may be due to partial mediation of these associations through the conventional cardiovascular risk factors, suggesting the importance in assessing the environmental effects on more basic physiological pathways when addressing the contribution to the cardiovascular risk.

표-534. PubMed 논문번호 28735152의 내용 요약

구분	내용
PubMed ID	28735152
TITLE	A systematic review of cardiovascular emergency departm admissions and mortality associated with ambient black c
JOURNAL	Environment international: 10.1016/j.envint.2017.07.005
AUTHORS	Luben Thomas J, Nichols Jennifer L, Dutton Steven J, Kir Elizabeth O, Datko-Williams Laura, Madden Meagan, Sa
BACKGROUND	Black carbon (BC) is a ubiquitous component of particula emitted from combustion-related sources and is associate health outcomes.
OBJECTIVES	We conducted a systematic review to evaluate the potentia morbidity and mortality following exposure to ambient B component elemental carbon (EC), in the context of what about the associations between exposure to fine particulat and cardiovascular health outcomes.
DATA SOURCES	We conducted a stepwise systematic literature search of th and employed Preferred Reporting Items for Systematic R Meta-Analyses (PRISMA) guidelines for reporting our res
STUDY ELIGIBILITY CRITERIA	Studies meeting inclusion criteria (i.e., include a quantitati BC or EC used to characterize exposure and an effect esti association of the exposure metric with ED visits, hospital mortality due to cardiovascular disease) were evaluated fo study design and results.
STUDY APPRAISAL AND SYNTHESIS METHODS	Risk of bias evaluations assess some aspects of internal va findings based on study design, conduct, and reporting an issues related to confounding or other biases.
RESULTS	The results of our systematic review demonstrate similar r and PM2.5; that is, a generally modest, positive associatio measurement with cardiovascular emergency department admissions, and mortality. There is no clear evidence that greater for either BC or EC when compared to one anothe compared to PM2.5.
LIMITATIONS	We were unable to adequately evaluate the role of copollu differential spatial heterogeneity for BC or EC compared t
CONCLUSIONS AND IMPLICATIONS OF KEY FINDINGS	Overall, the evidence at present indicates that BC or EC is associated with cardiovascular morbidity and mortality bu conclude that BC or EC is independently associated with t than being an indicator for PM2.5 mass.
SYSTEMATIC REVIEW REGISTRATION NUMBER	Not available.

표-535. PubMed 논문번호 28763730의 내용 요약

구분	내용
PubMed ID	28763730
TITLE	Fine particulate matter and cardiovascular disease: Comparison of assessment methods for long-term exposure.
JOURNAL	Environmental research: 10.1016/j.envres.2017.07.041
AUTHORS	McGuinn Laura A, Ward-Caviness Cavin, Neas Lucas M, Schneider Alexandra, Di Qian, Chudnovsky Alexandra, Schwartz Joel, Koutrakis Petros, Russell Armistead G, Garcia Val, Kraus William E, Hauser Elizabeth R, Cascio Wayne, Diaz-Sanchez David, Devlin Robert B
BACKGROUND	Adverse cardiovascular events have been linked with PM2.5 exposure obtained primarily from air quality monitors, which rarely co-locate with participant residences. Modeled PM2.5 predictions at finer resolution may more accurately predict residential exposure; however few studies have compared results across different exposure assessment methods.
METHODS	We utilized a cohort of 5679 patients who had undergone a cardiac catheterization between 2002-2009 and resided in NC. Exposure to PM2.5 for the year prior to catheterization was estimated using data from air quality monitors (AQS), Community Multiscale Air Quality (CMAQ) fused models at the census tract and 12km spatial resolutions, and satellite-based models at 10km and 1km resolutions. Case status was either a coronary artery disease (CAD) index >23 or a recent myocardial infarction (MI). Logistic regression was used to model odds of having CAD or an MI with each 1-unit (μg/m3) increase in PM2.5, adjusting for sex, race, smoking status, socioeconomic status, and urban/rural status.
RESULTS	We found that the elevated odds for CAD>23 and MI were nearly equivalent for all exposure assessment methods. One difference was that data from AQS and the census tract CMAQ showed a rural/urban difference in relative risk, which was not apparent with the satellite or 12km-CMAQ models.
CONCLUSIONS	Long-term air pollution exposure was associated with coronary artery disease for both modeled and monitored data.

표-536. PubMed 논문번호 28765026의 내용 요약

구분	내용
PubMed ID	28765026
TITLE	Passive Smoking Is Not Associated with Risk of Intracranial Aneurysm Rupture in Nonsmoking Women.
JOURNAL	World neurosurgery: 10.1016/j.wneu.2017.07.120
AUTHORS	Feng Xin, Wang Luyao, Guo Erkang, Zhang Baorui, Qian Zenghui, Wen Xiaolong, Xu Wenjuan, Li Youxiang, Jiang Chuhan, Wu Zhongxue, Liu Aihua
BACKGROUND	Active smoking is a major risk factor for intracranial aneurysm rupture (IAR); however, little is known about the effects of passive smoking on IAR. In China, female passive smoking is widespread and severe. This study aimed to assess whether passive smoking is associated with increased risk of IAR among nonsmoking women.
METHODS	We enrolled and retrospectively analyzed 385 consecutive female patients with intracranial aneurysms (IAs; 87 ruptured, 298 unruptured) who were admitted to our center between June 2015 and January 2017. Data on female active smoking, passive smoking, and other factors potentially influencing IAR were precisely compared between ruptured and unruptured IAs.
RESULTS	For all aneurysms, when adjusting for potential confounders, current smoking was significantly associated with IAR (odds ratio [OR], 3.31; 95% confidence interval [CI], 1.08-10.20; P = 0.037). Furthermore, bifurcation location (OR, 5.73; 95% CI, 3.27-10.03; P < 0.001) and educational level (OR, 1.90; 95% CI, 1.10-3.28; P = 0.022) significantly increased the risk of IAR. However, for nonsmoking female patients, approximately one fifth of those with IAs were affected by passive smoking; however, passive smoking was not significantly associated with IAR. The results also showed that bifurcation location (OR, 6.21; 95% CI, 3.46-11.15; P < 0.001) and the location of posterior circulation (OR, 3.23; 95% CI, 1.31-7.93; P = 0.011) significantly increased the risk of IAR.
CONCLUSIONS	Although active current smoking was strongly associated with aneurysm rupture in female patients, passive smoking was not an independent risk factor for aneurysm rupture in nonsmoking women.

표-537. PubMed 논문번호 28768298의 내용 요약

구분	내용
PubMed ID	28768298
TITLE	Estimating the Causal Effect of Low Levels of Fine Particulate Matter on Hospitalization.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000690
AUTHORS	Makar Maggie, Antonelli Joseph, Di Qian, Cutler David, Schwartz Joel, Dominici Francesca
BACKGROUND	In 2012, the EPA enacted more stringent National Ambient Air Quality Standards (NAAQS) for fine particulate matter (PM2.5). Few studies have characterized the health effects of air pollution levels lower than the most recent NAAQS for long-term exposure to PM2.5 (now 12 μg/m).
METHODS	We constructed a cohort of 32,119 Medicare beneficiaries residing in 5138 US ZIP codes who were interviewed as part of the Medicare Current Beneficiary Survey (MCBS) between 2002 and 2010 and had 1 year of follow-up. We considered four outcomes: all-cause hospitalizations, hospitalizations for circulatory diseases and respiratory diseases, and death.
RESULTS	We found that increasing exposure to PM2.5 from levels lower than 12 μg/m to levels higher than 12 μg/m is associated with increases in all-cause admission rates of 7% (95% CI = 3%, 10%) and in circulatory admission hazard rates of 6% (95% CI = 2%, 9%). When we restricted analysis to enrollees with exposure always lower than 12 μg/m, we found that increasing exposure from levels lower than 8 μg/m to levels higher than 8 μg/m increased all-cause admission hazard rates by 15% (95% CI = 8%, 23%), circulatory by 18% (95% CI = 10%, 27%), and respiratory by 21% (95% CI = 9%, 34%).
CONCLUSIONS	In a nationally representative sample of Medicare enrollees, changes in exposure to PM2.5, even at levels consistently below standards, are associated with increases in hospital admissions for all causes and cardiovascular and respiratory diseases. The robustness of our results to inclusion of many additional individual level potential confounders adds validity to studies of air pollution that rely entirely on administrative data.

표-538. PubMed 논문번호 28792710의 내용 요약

구분	내용
PubMed ID	28792710
TITLE	Effects of Passive Smoking on Glycemic Parameters and Lipid Profiles in a Chinese Female Population.
JOURNAL	Clinical laboratory: 10.7754/Clin.Lab.2017.170102
AUTHORS	Gu Lijuan, Li Jing, Pan Gui, Zhou Xiaojun, Zhang Jianmin, Lai Xiaoyang, Liu Jianping
BACKGROUND	The adverse effects of exposure to second-hand cigarette smoke on glucose and lipid parameters in women and the underlying mechanisms are not well understood. In this prospective community-based study, we examined the effects of passive smoking on glycemic parameters and lipid profiles in a Chinese female population.
METHODS	Of 3197 healthy women enrolled (30 - 75 years), 2082 self-reported passive exposure to smoke (≥ 15 minutes/day, 3 days/week, > 1 year) and 1115 had no smoke exposure (control group). Data was collected via questionnaire, body measurements, and laboratory assays for glycemic parameters and lipid profiles.
RESULTS	(1) Women exposed to second-hand smoke had significantly higher fasting plasma glucose, hemoglobin A1c, total cholesterol, triglycerides, low-density lipoprotein cholesterol, and waist-to-hip ratio compared with the control group ($p < 0.05$), while 2-h plasma glucose, high-density lipoprotein cholesterol levels, and body mass indexes were similar compared with the control group ($p > 0.05$). (2) Logistic regression analysis showed that after adjusting for potential confounders, passive smoking women increased the risks of hemoglobin A1c and total cholesterol ($p < 0.05$).
CONCLUSIONS	In this population of Chinese women, exposure to second-hand cigarette smoke was associated with adverse effects on glucose and lipid profiles, suggesting an increased risk of diabetes and cardiovascular disease. These findings support the benefit of stopping smoking in the home and implementing no-smoking regulations in public areas in China and other developing countries to prevent diabetes and other chronic diseases.

표-539. PubMed 논문번호 28806967의 내용 요약

구분	내용
PubMed ID	28806967
TITLE	MicroRNA-181c inhibits cigarette smoke-induced chronic obstructive pulmonary disease by regulating CCN1 expression.
JOURNAL	Respiratory research: 10.1186/s12931-017-0639-1
AUTHORS	Du Yong, Ding Yi, Chen Xuru, Mei Zhoufang, Ding Heyuan, Wu Yi, Jie Zhijun
BACKGROUND	Chronic obstructive pulmonary disease (COPD) is an obstinate pulmonary disease, causing irreversible alveoli collapse and increasing the risk for cardiovascular disease. Accumulating evidence has shown that the dysregulation of miRNAs is crucially involved in the pathogenesis and development of COPD. However, the effects and role of microRNA-181c (miR-181c) have not been investigated in a murine model of COPD.
METHODS	miR-181c expression was detected in human lung tissue samples of 34 patients, an in vivo murine model of CS exposure, and primary human bronchial epithelial cells (HBECs) by qRT-PCR. Degeneration of lung tissue, necrosis, infiltration and neutrophil cells were assessed with H&E and flow cytometry. Interleukin (IL)-6 and IL-8 levels were determined by an enzyme-linked immunosorbent assay and qRT-PCR. Luciferase reporter assay and correlation analyses were used to confirm and measure the levels between miR-181c and its target CCN1.
RESULTS	We showed that miR-181c was significantly down-regulated in lung tissues from patients with COPD compared to individuals who had never smoked ($p < 0.01$). We also observed a down-regulation of miR-181c in HBECs and a mouse model after cigarette smoke (CS) exposure. Functional assays demonstrated that miR-181c over-expression decreased the inflammatory response, neutrophil infiltration, reactive oxygen species (ROS) generation, and inflammatory cytokines induced by CS, while its down-regulation produced the opposite effects. Subsequent investigation found that CCN1 was a direct target of miR-181c. CCN1 expression was increased in lung tissues of COPD patients, and was negatively correlated with miR-181c expression in human COPD samples ($p < 0.01$).
CONCLUSIONS	Taken together, our data suggest the critical roles of miR-181c and its target CCN1 in COPD development, and provide potential therapeutic targets for COPD treatment.

표-540. PubMed 논문번호 28808144의 내용 요약

구분	내용
PubMed ID	28808144
TITLE	Particulate Matter Exposure and Stress Hormone Levels: A Randomized, Double-Blind, Crossover Trial of Air Purification.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.116.026796
AUTHORS	Li Huichu, Cai Jing, Chen Renjie, Zhao Zhuohui, Ying Zhekang, Wang Lin, Chen Jianmin, Hao Ke, Kinney Patrick L, Chen Honglei, Kan Haidong
BACKGROUND	Exposure to ambient particulate matter (PM) is associated with a number of adverse health outcomes, but potential mechanisms are largely unknown. Metabolomics represents a powerful approach to study global metabolic changes in response to environmental exposures. We therefore conducted this study to investigate changes in serum metabolites in response to the reduction of PM exposure among healthy college students.
METHODS	We conducted a randomized, double-blind crossover trial in 55 healthy college students in Shanghai, China. Real and sham air purifiers were placed in participants' dormitories in random order for 9 days with a 12-day washout period. Serum metabolites were quantified by using gas chromatography-mass spectrometry and ultrahigh performance liquid chromatography-mass spectrometry. Between-treatment differences in metabolites were examined using orthogonal partial least square-discriminant analysis and mixed-effect models. Secondary outcomes include blood pressure, corticotropin-releasing hormone, adrenocorticotropic hormone, insulin resistance, and biomarkers of oxidative stress and inflammation.
RESULTS	The average personal exposure to PMs with aerodynamic diameters ≤2.5 μm was 24.3 μg/m3 during the real purification and 53.1 μg/m3 during the sham purification. Metabolomics analysis showed that higher exposure to PMs with aerodynamic diameters ≤2.5 μm led to significant increases in cortisol, cortisone, epinephrine, and norepinephrine. Between-treatment differences were also observed for glucose, amino acids, fatty acids, and lipids. We found significantly higher blood pressure, hormones, insulin resistance, and biomarkers of oxidative stress and inflammation among individuals exposed to higher PMs with aerodynamic diameters ≤2.5 μm.
CONCLUSIONS	This study suggests that higher PM may induce metabolic alterations that are consistent with activations of the hypothalamus-pituitary-adrenal and sympathetic-adrenal-medullary axes, adding potential mechanistic insights into the adverse health outcomes associated with PM. Furthermore, our study demonstrated short-term reductions in stress hormone following indoor air purification.
CLINICAL TRIAL REGISTRATION	URL: http://www.clinicaltrials.gov. Unique identifier: NCT02712333.

표-541. PubMed 논문번호 28825983의 내용 요약

구분	내용
PubMed ID	28825983
TITLE	Fine particulate matter constituents and blood pressure in patients with chronic obstructive pulmonary disease: A panel study in Shanghai, China.
JOURNAL	Environmental research: 10.1016/j.envres.2017.08.024
AUTHORS	Lin Zhijing, Niu Yue, Chen Renjie, Xu Wenxi, Li Huichu, Liu Cong, Cai Jing, Zhao Zhuohui, Kan Haidong, Qiao Liping
OBJECTIVE	The evidence is limited about the potentially different health effects of various chemical constituents of fine particulate matter (PM2.5). We thus assessed the acute effects of various chemical constituents of PM2.5 on blood pressure (BP).
METHODS	We performed a longitudinal panel study with six repeated visits in 28 urban residents with chronic obstructive pulmonary disease in Shanghai, China from May to July, 2014. Twelve (43%) of them took antihypertensive medications. We measured resting BP by using a mercury sphygmomanometer and monitored real-time concentrations of PM2.5 constituents at a nearby site. Based on the linear mixed-effects model, we evaluated the effects of 10 major constituents in PM2.5 on BP, using a single-constituent model and a constituent-residual model after accounting for the multicollinearity.
RESULTS	We obtained a total of 168 pairs of effective BP measurements during the study period. There are moderate or high correlations among various PM2.5 constituents. An interquartile range increase of PM2.5 (19.1 μg/m3) was associated with increments of 1.90mmHg [95% confidence interval (CI): 0.66, 3.13] in systolic BP, 0.68mmHg (95%CI: -0.02, 1.37) in diastolic BP and 1.23mmHg (95%CI: 0.19, 2.29) in pulse pressure. Some constituents of PM2.5, including organic carbon, elemental carbon, nitrate and ammonium, were robustly associated with elevated BP after controlling for total PM2.5 mass and accounting for multi-collinearity. Two constituents (magnesium and calcium) were associated with decreased BP.
CONCLUSIONS	Organic carbon, elemental carbon, nitrate and ammonium may be mainly responsible for elevated BP from a short-term exposure to PM2.5.

표-542. PubMed 논문번호 28850858의 내용 요약

구분	내용
PubMed ID	28850858
TITLE	Associations between fine particulate matter and mortality in the 2001 Canadian Census Health and Environment Cohort.
JOURNAL	Environmental research: 10.1016/j.envres.2017.08.037
AUTHORS	Pinault Lauren L, Weichenthal Scott, Crouse Daniel L, Brauer Michael, Erickson Anders, Donkelaar Aaron van, Martin Randall V, Hystad Perry, Chen Hong, Fin□s Philippe, Brook Jeffrey R, Tjepkema Michael, Burnett Richard T
BACKGROUND	Large cohort studies have been used to characterise the association between long-term exposure to fine particulate matter (PM2.5) air pollution with non-accidental, and cause-specific mortality. However, there has been no consensus as to the shape of the association between concentration and response.
METHODS	To examine the shape of this association, we developed a new cohort based on respondents to the 2001 Canadian census long-form. We applied new annual PM2.5 concentration estimates based on remote sensing and ground measurements for Canada at a 1km spatial scale from 1998 to 2011. We followed 2.4 million respondents who were non-immigrants aged 25-90 years and did not reside in an institution over a 10 year period for mortality. Exposures were assigned as a 3-year mean prior to the follow-up year. Income tax files were used to account for residential mobility among respondents using postal codes, with probabilistic imputation used for missing postal codes in the tax data. We used Cox survival models to determine hazard ratios (HRs) for cause-specific mortality. We also estimated Shape Constrained Health Impact Functions (a concentration-response function) for selected causes of death.
RESULTS	In models stratified by age, sex, airshed, and population centre size, and adjusted for individual and neighbourhood socioeconomic variables, HR estimates for non-accidental mortality were HR = 1.18 (95% CI: 1.15-1.21) per 10 μg/m3 increase in concentration. We observed higher HRs for cardiovascular disease (HR=1.25; 95% CI: 1.19-1.31), cardio-metabolic disease (HR = 1.27; 95% CI: 1.21-1.33), ischemic heart disease (HR = 1.36; 95% CI: 1.28-1.44) and chronic obstructive pulmonary disease (COPD) mortality (HR = 1.24; 95% CI: 1.11-1.39) compared to HR for all non-accidental causes of death. For non-accidental, cardio-metabolic, ischemic heart disease, respiratory and COPD mortality, the shape of the concentration-response curve was supra-linear, with larger differences in relative risk for lower concentrations. For both pneumonia and lung cancer, there was some suggestion that the curves were sub-linear.
CONCLUSIONS	Associations between ambient concentrations of fine particulate matter and several causes of death were non-linear for each cause of death examined.

표-543. PubMed 논문번호 28871018의 내용 요약

구분	내용
PubMed ID	28871018
TITLE	Early-life exposure to air pollutants and adverse pregnancy outcomes: protocol for a prospective cohort study in Beijing.
JOURNAL	BMJ open: 10.1136/bmjopen-2017-015895
AUTHORS	Song Jing, Chen Yi, Wei Ling, Ma Ying, Tian Ning, Huang Shi Yun, Dai Yin Mei, Zhao Li Hong, Kong Yuan Yuan
INTRODUCTION	The association between early exposure to ambient air pollution and adverse pregnancy outcomes in China is unclear. This study will assess the risk of early-life exposure to air pollutants in Beijing and explore the viability of 8-hydroxydeoxyguanosine (8-OHdG) as a biological indicator to assess oxidative stress induced by early-life exposure to air pollution.
METHODS AND ANALYSIS	Here, 2500 women with singleton pregnancies and their infants will be recruited from the Beijing Obstetrics and Gynecology Hospital. We will collect nine types of biological samples, including maternal serum, urine, placental tissue, umbilical cord tissue and umbilical cord blood during all three trimesters. The air pollution data (particulate matter (PM)2.5, PM10 and similar factors) will be recorded at official fixed-site monitoring stations closest to where the pregnant women live. We plan to assess the effect of air pollutants on adverse pregnancy outcomes and infant respiratory and circulatory disease using Cox regression and competitive risk analysis and explore possible critical windows of exposure during pregnancy using daily pollutant concentrations averaged over various periods of pregnancy combined with individual activity and physiological parameters. Maternal and umbilical cord blood samples (1000 samples) will be randomly selected for 8-OHdG assays to assess the correlation between exposures to air pollutants and oxidative stress. We will determine whether air pollutant exposure or 8-OHdG levels are associated with adverse pregnancy outcomes. SPSS and SAS statistical software will be used for data analysis. Cox regression and competing risk analysis will be used to compute the HR and population attributable risk.
ETHICS AND DISSEMINATION	This research protocol has already been approved by the Medical Ethics Committee of Beijing Obstetrics and Gynecology Hospital. Written informed consent will be obtained from all study participants prior to enrolment. The results will be published in peer-reviewed journals or disseminated through conference presentations.
TRIAL REGISTRATION NUMBER	This study has been registered in WHO International Clinical Trial Register-Chinese Clinical Trial Registry under registrationnumber ChiCTR-ROC-16010181 (http :// www.chictr.org.cn / showproj.aspx ?proj=17328).

표-544. PubMed 논문번호 28883037의 내용 요약

구분	내용
PubMed ID	28883037
TITLE	Ischaemic heart disease in the former Soviet Union 1990-2015 according to the Global Burden of Disease 2015 Study.
JOURNAL	Heart (British Cardiac Society): 10.1136/heartjnl-2016-311142
AUTHORS	Murphy Adrianna, Johnson Catherine O, Roth Gregory A, Forouzanfar Mohammad H, Naghavi Mohsen, Ng Marie, Pogosova Nana, Vos Theo, Murray Christopher J L, Moran Andrew E, Murphy Adrianna, Johnson Catherine O, Roth Gregory A, Forouzanfar Mohammad H, Naghavi Mohsen, Ng Marie, Pogosova Nana, Vos Theo, Murray Christopher J L, Moran Andrew E
OBJECTIVE	The objective of this study was to compare ischaemic heart disease (IHD) mortality and risk factor burden across former Soviet Union (fSU) and satellite countries and regions in 1990 and 2015.
METHODS	The fSU and satellite countries were grouped into Central Asian, Central European and Eastern European regions. IHD mortality data for men and women of any age were gathered from national vital registration, and age, sex, country, year-specific IHD mortality rates were estimated in an ensemble model. IHD morbidity and mortality burden attributable to risk factors was estimated by comparative risk assessment using population attributable fractions.
RESULTS	In 2015, age-standardised IHD death rates in Eastern European and Central Asian fSU countries were almost two times that of satellite states of Central Europe. Between 1990 and 2015, rates decreased substantially in Central Europe (men -43.5% (95% uncertainty interval -45.0%, -42.0%); women -42.9% (-44.0%, -41.0%)) but less in Eastern Europe (men -5.6% (-9.0, -3.0); women -12.2% (-15.5%, -9.0%)). Age-standardised IHD death rates also varied within regions: within Eastern Europe, rates decreased -51.7% in Estonian men (-54.0, -47.0) but increased +19.4% in Belarusian men (+12.0, +27.0). High blood pressure and cholesterol were leading risk factors for IHD burden, with smoking, body mass index, dietary factors and ambient air pollution also ranking high.
CONCLUSIONS	Some fSU countries continue to experience a high IHD burden, while others have achieved remarkable reductions in IHD mortality. Control of blood pressure, cholesterol and smoking are IHD prevention priorities.

표-545. PubMed 논문번호 28890218의 내용 요약

구분	내용
PubMed ID	28890218
TITLE	Serum perfluoroalkyl substances and cardiometabolic consequences in adolescents exposed to the World Trade Center disaster and a matched comparison group.
JOURNAL	Environment international: 10.1016/j.envint.2017.08.003
AUTHORS	Koshy Tony T, Attina Teresa M, Ghassabian Akhgar, Gilbert Joseph, Burdine Lauren K, Marmor Michael, Honda Masato, Chu Dinh Binh, Han Xiaoxia, Shao Yongzhao, Kannan Kurunthachalam, Urbina Elaine M, Trasande Leonardo
BACKGROUND	Large amounts of various chemical contaminants, including perfluoroalkyl substances (PFASs), were released at the time of the World Trade Center (WTC) disaster. Thousands of children who lived and/or attended school near the disaster site were exposed to these substances but few studies have examined the possible consequences related to these exposures.
OBJECTIVES	To examine the relationship of PFASs serum levels with cardiometabolic profile in children and adolescents enrolled in the World Trade Center Health Registry (WTCHR) and a matched comparison group.
METHODS	We evaluated WTCHR enrollees who resided in New York City and were born between September 11, 1993 and September 10, 2001, and a matched comparison group consisting of individuals who were ineligible for WTCHR participation upon distance of their home, school or work from the WTC and lack of participation in rescue and recovery activities. Matching was based on date of birth, sex, race, ethnicity, and income. We assessed exposure to PFASs, as measured by serum levels and association with cardiometabolic profile as measured by arterial wall stiffness, body mass index, insulin resistance, fasting total cholesterol, HDL, LDL and triglycerides.
RESULTS	A total of 402 participants completed the study and serum samples were analyzed from 308 participants, 123 in the WTCHR group and 185 in the comparison group. In multivariable regression analysis, after adjusting for relevant confounders, we observed a significant, positive association of perfluorooctanoic acid (PFOA) with triglycerides (beta coefficient=0.14, 95% CI: 0.02, 0.27, 15.1% change), total cholesterol (beta coefficient=0.09, 95% CI: 0.04, 0.14, 9.2% change), and LDL cholesterol (beta coefficient=0.11, 95% CI: 0.03, 0.19, 11.5% change). Perfluorohexanesulfonic acid levels were associated with decreased insulin resistance (beta coefficient=−0.09, 95% CI: −0.18, −0.003, −8.6% change); PFOA and perfluorononanoic acid were associated with increased brachial artery distensibility.
CONCLUSIONS	This research adds to our knowledge of the physical health impacts in a large group of children exposed to the WTC disaster. Abnormal lipid levels in young adults might be an early marker of atherosclerosis and cardiovascular diseases and our findings highlight the importance of conducting longitudinal studies in this population.

표-546. PubMed 논문번호 28902692의 내용 요약

구분	내용
PubMed ID	28902692
TITLE	The association between maternal exposure to pollutant particulate matter 2.5 and neonatal congenital heart defects: a systematic review protocol.
JOURNAL	JBI database of systematic reviews and implementation reports: 10.11124/JBISRIR-2016-003284
AUTHORS	Hall Katie C, Robinson Jennifer C
REVIEW QUESTION	The objective of this review is to identify if there is an association between maternal exposure to pollutant particulate matter 2.5 during the first trimester of pregnancy and neonatal congenital heart defects within the first year of life.

표-547. PubMed 논문번호 The Global Burden of Diseases, Injuries, and Risk Factors Study 2016 (GBD 2016) provides a comprehensive assessment of risk factor exposure and attributable burden of disease. By providing estimates over a long time series, this study can monitor risk exposure trends critical to health surveillance and inform policy debates on the importance of addressing risks in context.의 내용 요약

구분	내용
BACKGROUND	The Global Burden of Diseases, Injuries, and Risk Factors Study 2016 (GBD 2016) provides a comprehensive assessment of risk factor exposure and attributable burden of disease. By providing estimates over a long time series, this study can monitor risk exposure trends critical to health surveillance and inform policy debates on the importance of addressing risks in context.
METHODS	We used the comparative risk assessment framework developed for previous iterations of GBD to estimate levels and trends in exposure, attributable deaths, and attributable disability-adjusted life-years (DALYs), by age group, sex, year, and location for 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks from 1990 to 2016. This study included 481 risk-outcome pairs that met the GBD study criteria for convincing or probable evidence of causation. We extracted relative risk (RR) and exposure estimates from 22 717 randomised controlled trials, cohorts, pooled cohorts, household surveys, census data, satellite data, and other sources, according to the GBD 2016 source counting methods. Using the counterfactual scenario of theoretical minimum risk exposure level (TMREL), we estimated the portion of deaths and DALYs that could be attributed to a given risk. Finally, we explored four drivers of trends in attributable burden: population growth, population ageing, trends in risk exposure, and all other factors combined.
FINDINGS	Since 1990, exposure increased significantly for 30 risks, did not change significantly for four risks, and decreased significantly for 31 risks. Among risks that are leading causes of burden of disease, child growth failure and household air pollution showed the most significant declines, while metabolic risks, such as body-mass index and high fasting plasma glucose, showed significant increases. In 2016, at Level 3 of the hierarchy, the three leading risk factors in terms of attributable DALYs at the global level for men were smoking (124·1 million DALYs [95% UI 111·2 million to 137·0 million]), high systolic blood pressure (122·2 million DALYs [110·3 million to 133·3 million], and low birthweight and short gestation (83·0 million DALYs [78·3 million to 87·7 million]), and for women, were high systolic blood pressure (89·9 million DALYs [80·9 million to 98·2 million]), high body-mass index (64·8 million DALYs [44·4 million to 87·6 million]), and high fasting plasma glucose (63·8 million DALYs [53·2 million to 76·3 million]). In 2016 in 113 countries, the leading risk factor in terms of attributable DALYs was a metabolic risk factor. Smoking remained among the leading five risk factors for DALYs for 109 countries, while low birthweight and short gestation was the leading risk factor for DALYs in 38 countries, particularly in sub-Saharan Africa and South Asia. In terms of important drivers of change in trends of burden attributable to risk factors, between 2006 and 2016 exposure to risks explains an 9·3% (6·9-11·6) decline in deaths and a 10·8% (8·3-13·1) decrease in DALYs at the global level, while population ageing accounts for 14·9% (12·7-17·5) of deaths and 6·2% (3·9-8·7) of DALYs, and population growth for 12·4% (10·1-14·9) of deaths and 12·4% (10·1-14·9) of DALYs. The largest contribution of trends in risk exposure to disease burden is seen between ages 1 year and 4 years, where a decline of 27·3% (24·9-29·7) of the change in DALYs between 2006 and 2016 can be attributed to declines in exposure to risks.
INTERPRETATION	Increasingly detailed understanding of the trends in risk exposure and the RRs for each risk-outcome pair provide insights into both the magnitude of health loss attributable to risks and how modification of risk exposure has contributed to health trends. Metabolic risks warrant particular policy

표-548. PubMed 논문번호 28923332의 내용 요약

구분	내용
PubMed ID	28923332
TITLE	Association Between PM〈sub〉2.5〈/sub〉 Exposure and the Prognosis of Patients with Acute Myocardial Infraction.
JOURNAL	Archives of medical research: 10.1016/j.arcmed.2017.06.006
AUTHORS	Zang Xiwen, Qi Xiangqian
BACKGROUND	Numerous case-crossover and time series studies of PM2.5 exposure and cardiovascular health effects have been conducted, fever studies have examined the effects of PM2.5 on the outcomes of CHD, especially on acute myocardial infarction (AMI).
OBJECTIVE	The objective of this research is to investigate the association between PM2.5 exposure in Tianjin City and the outcome of AMI.
METHODS	We conducted a retrospective analysis of a total of 598 patients with AMI in TEDA International Cardiovascular Hospital, from Oct 28th, 2013-Apr 30th 2014. All patients were divided into five groups according to the National Air Quality Classification Standard. Major adverse cardiovascular events (MACEs) including all-cause death, heart failure, myocardial infarction and target lesion revascularization (TLR) during one year follow-up were defined as endpoint. Furthermore, we divided the patients into two groups according to better and worse air quality, then examined the incidence of MACEs in the two groups. The prognostic was assessed by using multivariate Cox regression analysis.
RESULTS	With the increase of the concentration of PM2.5, the incidence of MACEs were higher in patients after 1 year of AMI (In the five groups, Hazard Ratio [HR] 1.622, 95% CI 1.352-1.947; p = 0.000. In the two groups, HR 3.255, 95% CI 2.008-5.276; p = 0.000).
CONCLUSION	PM2.5 exposure was associated with the outcome of patients with AMI, especially, the poorer air quality it is, the worse prognosis of patients will be.

표-549. PubMed 논문번호 28934719의 내용 요약

구분	내용
PubMed ID	28934719
TITLE	Exposure to Road, Railway, and Aircraft Noise and Arterial Stiffness in the SAPALDIA Study: Annual Average Noise Levels and Temporal Noise Characteristics.
JOURNAL	Environmental health perspectives: 10.1289/EHP1136
AUTHORS	Foraster Maria, Eze Ikenna C, Schaffner Emmanuel, Vienneau Danielle, H□ritier Harris, Endes Simon, Rudzik Franziska, Thiesse Laurie, Pieren Reto, Schindler Christian, Schmidt-Trucks□ss Arno, Brink Mark, Cajochen Christian, Marc Wunderli Jean, R□□sli Martin, Probst-Hensch Nicole
BACKGROUND	The impact of different transportation noise sources and noise environments on arterial stiffness remains unknown.
OBJECTIVES	We evaluated the association between residential outdoor exposure to annual average road, railway, and aircraft noise levels, total noise intermittency (IR), and total number of noise events (NE) and brachial-ankle pulse wave velocity (baPWV) following a cross-sectional design.
METHODS	We measured baPWV (meters/second) in 2,775 participants (49-81 y old) at the second follow-up (2010-2011) of the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases in Adults (SAPALDIA). We assigned annual average road, railway, and aircraft noise levels (Ldensource), total day- and nighttime NEtime and IRtime (percent fluctuation=0%, none or constant noise; percent fluctuation=100%, high fluctuation) at the most exposed fa□ade using 2011 Swiss noise models. We applied multivariable linear mixed regression models to analyze associations.
RESULTS	Medians [interquartile ranges (IQRs)] were baPWV=13.4 (3.1) m/s; Ldenair (57.6% exposed)=32.8 (8.0) dB; Ldenrail (44.6% exposed)=30.0 (8.1) dB; Ldenroad (99.7% exposed): 54.2 (10.6) dB; NEnight=123 (179); NEday=433 (870); IRnight=73% (27); and IRday=63.8% (40.3). We observed a 0.87% (95% CI: 0.31, 1.43%) increase in baPWV per IQR of Ldenrail, which was greater with IRnight>80% or with daytime sleepiness. We observed a nonsignificant positive association between Ldenroad and baPWV in urban areas and a negative tendency in rural areas. NEnight, but not NEday, was associated with baPWV. Associations were independent of the other noise sources and air pollution.
CONCLUSIONS	Long-term exposure to railway noise, particularly in an intermittent nighttime noise environment, and to nighttime noise events, mainly related to road noise, may affect arterial stiffness, a major determinant of cardiovascular disease. Ascertaining noise exposure characteristics beyond average noise levels may be relevant to better understand noise-related health effects. https://doi.org/10.1289/EHP1136.

표-550. PubMed 논문번호 28934721의 내용 요약

구분	내용
PubMed ID	28934721
TITLE	Association of Long-Term Exposure to Transportation Noise and Traffic-Related Air Pollution with the Incidence of Diabetes: A Prospective Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP1279
AUTHORS	Clark Charlotte, Sbihi Hind, Tamburic Lillian, Brauer Michael, Frank Lawrence D, Davies Hugh W
BACKGROUND	Evidence for an association between transportation noise and cardiovascular disease has increased; however, few studies have examined metabolic outcomes such as diabetes or accounted for environmental coexposures such as air pollution, greenness, or walkability.
OBJECTIVES	Because diabetes prevalence is increasing and may be on the causal pathway between noise and cardiovascular disease, we examined the influence of long-term residential transportation noise exposure and traffic-related air pollution on the incidence of diabetes using a population-based cohort in British Columbia, Canada.
METHODS	We examined the influence of transportation noise exposure over a 5-y period (1994-1998) on incident diabetes cases in a population-based prospective cohort study (n=380,738) of metropolitan Vancouver (BC) residents who were 45-85 y old, with 4-y of follow-up (1999-2002). Annual average transportation noise (Lden), air pollution [black carbon, particulate matter with aerodynamic diameter <2.5 μm (PM2.5), nitrogen oxides], greenness [Normalized Difference Vegetation Index (NDVI)], and neighborhood walkability at each participant's residence were modeled. Incident diabetes cases were identified using administrative health records.
RESULTS	Transportation noise was associated with the incidence of diabetes [interquartile range (IQR) increase, 6.8 A-weighted decibels (dBA); OR=1.08 (95% CI: 1.05, 1.10)]. This association remained after adjustment for environmental coexposures including traffic-related air pollutants, greenness, and neighborhood walkability. After adjustment for coexposure to noise, traffic-related air pollutants were not associated with the incidence of diabetes, whereas greenness was protective.
CONCLUSION	We found a positive association between residential transportation noise and diabetes, adding to the growing body of evidence that noise pollution exposure may be independently linked to metabolic health and should be considered when developing public health interventions. https://doi.org/10.1289/EHP1279.

표-551. PubMed 논문번호 28953453의 내용 요약

구분	내용
PubMed ID	28953453
TITLE	Long-Term Exposure to Road Traffic Noise and Nitrogen Dioxide and Risk of Heart Failure: A Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP1272
AUTHORS	S ø rensen Mette, Wendelboe Nielsen Olav, Sajadieh Ahmad, Ketzel Matthias, Tj ø nneland Anne, Overvad Kim, Raaschou-Nielsen Ole
BACKGROUND	Although air pollution and road traffic noise have been associated with higher risk of cardiovascular diseases, associations with heart failure have received only little attention.
OBJECTIVES	We aimed to investigate whether long-term exposure to road traffic noise and nitrogen dioxide (NO2) were associated with incident heart failure.
METHODS	In a cohort of 57,053 people 50-64 y of age at enrollment in the period 1993-1997, we identified 2,550 cases of first-ever hospital admission for heart failure during a mean follow-up time of 13.4 y. Present and historical residential addresses from 1987 to 2011 were found in national registers, and road traffic noise (Lden) and NO2 were modeled for all addresses. Analyses were done using Cox proportional hazard model.
RESULTS	An interquartile range higher 10-y time-weighted mean exposure for Lden and NO2 was associated with incidence rate ratios (IRR) for heart failure of 1.14 (1.08-1.21) and 1.11 (1.07-1.16), respectively, in models adjusted for gender, lifestyle, and socioeconomic status. In models with mutual exposure adjustment, IRRs were 1.08 (1.00-1.16) for Lden and 1.07 (1.01-1.14) for NO2. We found statistically significant modification of the NO2-heart failure association by gender (strongest association among men), baseline hypertension (strongest association among hypertensive), and diabetes (strongest association among diabetics). The same tendencies were seen for noise, but interactions were not statistically significant.
CONCLUSIONS	Long-term exposure to NO2 and road traffic noise was associated with higher risk of heart failure, mainly among men, in both single- and two-pollutant models. High exposure to both pollutants was associated with highest risk. https://doi.org/10.1289/EHP1272.

표-552. PubMed 논문번호 28954921의 내용 요약

구분	내용
PubMed ID	28954921
TITLE	Synergism of Short-Term Air Pollution Exposures and Neighborhood Disadvantage on Initial Stroke Severity.
JOURNAL	Stroke: 10.1161/STROKEAHA.117.018816
AUTHORS	Wing Jeffrey J, S□nchez Brisa N, Adar Sara D, Meurer William J, Morgenstern Lewis B, Smith Melinda A, Lisabeth Lynda D
BACKGROUND AND PURPOSE	Little is known about the relation between environment and stroke severity. We investigated associations between environmental exposures, including neighborhood socioeconomic disadvantage and short-term exposure to airborne particulate matter <2.5 μm and ozone, and their interactions with initial stroke severity.
METHODS	First-ever ischemic stroke cases were identified from the Brain Attack Surveillance in Corpus Christi project (2000-2012). Associations between pollutants, disadvantage, and National Institutes of Health Stroke Scale were modeled using linear and logistic regression with adjustment for demographics and risk factors. Pollutants and disadvantage were modeled individually, jointly, and with interactions.
RESULTS	Higher disadvantage scores and previous-day ozone concentrations were associated with higher odds of severe stroke. Higher levels of particulate matter <2.5 μm were associated with higher odds of severe stroke among those in higher disadvantage areas (odds ratio, 1.24; 95% confidence interval, 1.00-1.55) but not in lower disadvantage areas (odds ratio, 0.82; 95% confidence interval, 0.56-1.22; P interaction =0.097).
CONCLUSIONS	Air pollution exposures and neighborhood socioeconomic status may be important in understanding stroke severity. Future work should consider the multiple levels of influence on this important stroke outcome.

표-553. PubMed 논문번호 28974306의 내용 요약

구분	내용
PubMed ID	28974306
TITLE	Effects of long-term exposure to particulate matter and metal components on mortality in the Rome longitudinal study.
JOURNAL	Environment international: 10.1016/j.envint.2017.09.005
AUTHORS	Badaloni Chiara, Cesaroni Giulia, Cerza Francesco, Davoli Marina, Brunekreef Bert, Forastiere Francesco
BACKGROUND	The effect of long-term exposure to metal components in particulate matter on mortality are still controversial.
OBJECTIVES	To study the association between long-term exposure to PM10, PM2.5, PM2.5 absorbance, particulate matter components (copper, iron, zinc, sulfur, silicon, potassium, nickel, and vanadium) and non-accidental, cardiovascular (CVD), and ischemic heart disease (IHD) mortality.
METHODS	All 30+ year olds from the Rome Longitudinal Study were followed for vital status from October 2001 until December 2010. We used land use regression models to estimate annual average concentrations at residences and Cox models to estimate the associations between pollutants and cause-specific mortality, adjusting for individual and contextual characteristics. Hazard ratios (HRs) were expressed per increments equal to the 5th-95th percentile range of each pollutant distribution.
RESULTS	We analyzed 1,249,108 residents and found strong associations between all exposure indicators and mortality. We observed higher mortality risk with increasing exposure to PM2.5 absorbance (HR=1.05; 95% CI: 1.03-1.06) and to tracers of non-tailpipe traffic emissions such as tire and brake wear (Cu, Fe, and Zn); for PM2.5Zn, we found HR=1.06 (95% CI: 1.04-1.08) for non-accidental mortality, HR=1.07 (95% CI: 1.04-1.10) for CVD, and HR=1.11 (95% CI: 1.06-1.16) for IHD mortality. With increasing levels of nickel in PM10, we found HR=1.07 (95% CI: 1.05-1.09) for non-accidental mortality, HR=1.08 (95% CI: 1.05-1.11) for CVD, and HR=1.13 (95% CI: 1.08-1.18) for IHD mortality. Results were robust when we adjusted for PM mass and for cardiovascular mortality when we adjusted for NO2.
CONCLUSIONS	In addition to vehicular exhaust pollutants, PM related to non-tailpipe emissions and mixed oil burning/industry plays an important role in mortality.

표-554. PubMed 논문번호 28976899의 내용 요약

구분	내용
PubMed ID	28976899
TITLE	Community Noise Exposure and its Effect on Blood Pressure and Renal Function in Patients with Hypertension and Cardiovascular Disease.
JOURNAL	Folia medica: 10.1515/folmed-2017-0045
AUTHORS	Dzhambov Angel M, Tokmakova Mariya P, Gatseva Penka D, Zdravkov Nikolai G, Gencheva Dolina G, Ivanova Nevena G, Karastanev Krasimir I, Vladeva Stefka V, Donchev Aleksandar T, Dermendzhiev Svetlan M
BACKGROUND	Road traffic noise (RTN) is a risk factor for cardiovascular disease (CVD) and hypertension; however, few studies have looked into its association with blood pressure (BP) and renal function in patients with prior CVD.
AIM	This study aimed to explore the effect of residential RTN exposure on BP and renal function in patients with CVD from Plovdiv Province.
MATERIALS AND METHODS	We included 217 patients with ischemic heart disease and/or hypertension from three tertiary hospitals in the city of Plovdiv (March - May 2016). Patients' medical history, medical documentation, and medication regimen were reviewed, and blood pressure and anthropometric measurements were taken. Blood samples were analyzed for creatinine, total cholesterol, and blood glucose. Participants also filled a questionnaire. Glomerular filtration rate was estimated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. All participants were asked about their annoyance by different noise sources at home, and those living in the city of Plovdiv (n = 132) were assigned noise map Lden and Lnight exposure. The effects of noise exposure on systolic blood pressure (SBP), diastolic blood pressure (DBP), and estimated glomerular filtration rate (eGFR) were explored using mixed linear models.
RESULTS	Traffic noise annoyance was associated with higher SBP in the total sample. The other noise indicators were associated with non-significant elevation in SBP and reduction in eGFR. The effect of Lden was more pronounced in patients with prior ischemic heart disease/stroke, diabetes, obesity, not taking Ca-channel blockers, and using solid fuel/gas at home. Lnight had stronger effect among those not taking statins, sleeping in a bedroom with noisy fa□ade, having a living room with quiet fa□ade, and spending more time at home. The increase in Lden was associated with a significant decrease in eGFR among men, patients with ischemic heart disease/stroke, and those exposed to lower air pollution. Regarding Lnight, there was significant effect modification by gender, diabetes, obesity, and time spent at home. In some subgroups, the effect of RTN was statistically significant.
CONCLUSIONS	Given that generic risk factors for poor progression of cardiovascular diseases cannot be controlled sufficiently at individual level, environmental interventions to reduce residential noise exposure might result in some improvement in the management of blood pressure and kidney function in patients with CVD.

표-555. PubMed 논문번호 28988023의 내용 요약

구분	내용
PubMed ID	28988023
TITLE	Acute effects of fine particulate matter constituents on mortality: A systemati review and meta-regression analysis.
JOURNAL	Environment international: 10.1016/j.envint.2017.09.010
AUTHORS	Achilleos Souzana, Kioumourtzoglou Marianthi-Anna, Wu Chih-Da, Schwartz Joel D, Koutrakis Petros, Papatheodorou Stefania I
BACKGROUND	The link between PM2.5 exposure and adverse health outcomes is well documented from studies across the world. However, the reported effect estimates vary across studies, locations and constituents. We aimed to conduc a meta-analysis on associations between short-term exposure to PM2.5 constituents and mortality using city-specific estimates, and explore factors that may explain some of the observed heterogeneity.
METHODS	We systematically reviewed epidemiological studies on particle constituents and mortality using PubMed and Web of Science databases up to July 2015.We included studies that examined the association between short-term exposure to PM2.5 constituents and all-cause, cardiovascular, and respirator mortality, in the general adult population. Each study was summarized based on pre-specified study key parameters (e.g., location, time period, population diagnostic classification standard), and we evaluated the risk of bias using the Office of Health Assessment and Translation (OHAT) Method for each included study. We extracted city-specific mortality risk estimates for each constituent and cause of mortality. For multi-city studies, we requested the city-specific risk estimates from the authors unless reported in the article. We performed random effects meta-analyses using city-specific estimates, and examined whether the effects vary across regions and city characteristics (PM2.5 concentration levels, air temperature, elevation, vegetation, size of elderly population, population density, and baseline mortality).
RESULTS	We found a 0.89% (95% CI: 0.68, 1.10%) increase in all-cause, a 0.80% (95% CI: 0.41, 1.20%) increase in cardiovascular, and a 1.10% (95% CI: 0.59, 1.62%) increase in respiratory mortality per 10 μg/m3 increase in PM2.5. Accounting for the downward bias induced by studies of single days, the all-cause mortality estimate increased to 1.01% (95% CI: 0.81, 1.20%). We found significant associations between mortality and several PM2.5 constituents. The most consistent and stronger associations were observed for elemental carbon (EC) and potassium (K). For most of the constituents, we observed high variability of effect estimates across cities.
CONCLUSIONS	Our meta-analysis suggests that (a) combustion elements such as EC and K have a stronger association with mortality, (b) single lag studies underestimat effects, and (c) estimates of PM2.5 and constituents differ across regions. Accounting for PM mass in constituent's health models may lead to more stable and comparable effect estimates across different studies.
SYSTEMATIC REVIEW REGISTRATION	PROSPERO: CRD42017055765.

표-556. PubMed 논문번호 29056239의 내용 요약

구분	내용
PubMed ID	29056239
TITLE	Short-term exposure to air pollutants increases the risk of ST elevation myocardial infarction and of infarct-related ventricular arrhythmias and mortality.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2017.10.004
AUTHORS	Ba□eras Jordi, Ferreira-Gonz□lez Ignacio, Marsal Josep Ramon, Barrab□s Jos□ A, Ribera Aida, Lid□n Rosa Maria, Domingo Enric, Mart□ Gerard, Garc□a-Dorado David, Ba□eras Jordi, Ferreira-Gonz□lez Ignacio, Marsal Josep Ramon, Barrab□s Jos□ A, Ribera Aida, Lid□n Rosa Maria, Domingo Enric, Mart□ Gerard, Garc□a-Dorado David
BACKGROUND	The relation between STEMI and air pollution (AP) is scant. We aimed to investigate the short term association between AP and the incidence of STEMI, and STEMI-related ventricular arrhythmias (VA) and mortality.
METHODS	The study was carried out in the area of Barcelona from January 2010 to December 2011. Daily STEMI rates and incidence of STEMI-related VA and mortality were obtained prospectively. The corresponding daily levels of the main pollutants were recorded as well as the atmospheric variables. Three cohorts were defined in order to minimize exposure bias. The magnitude of association was estimated using a time-series design and was adjusted according to atmospheric variables.
RESULTS	The daily rate of hospital admissions for STEMI was associated with increases in PM 2.5, PM 10, lead and NO2 concentrations. VA incidence and mortality were associated with increases in PM 2.5 and PM 10 concentrations. In the most specific cohort, BCN (Barcelona) Attended & Resident, STEMI incidence was associated with increases in PM 2.5 (1.009% per 10 μg/m3) and PM 10 concentrations (1.005% per 10 μg/m3). VA was associated with increases in PM 2.5 (1.021%) and PM 10 (1.015%) and mortality was associated with increases in PM 2.5 (1.083%) and PM 10 (1.045%).
CONCLUSIONS	Short-term exposure to high levels of PM 2.5 and PM 10 is associated with increased daily STEMI admissions and STEMI-related VA and mortality. Exposure to high levels of lead and NO2 is associated with increased daily STEMI admissions, and NO2 with higher mortality in STEMI patients.

표-557. PubMed 논문번호 29072932의 내용 요약

구분	내용
PubMed ID	29072932
TITLE	Global Association of Air Pollution and Cardiorespiratory Diseases: A Systematic Review, Meta-Analysis, and Investigation of Modifier Variables.
JOURNAL	American journal of public health: 10.2105/AJPH.2017.303839
AUTHORS	Requia Weeberb J, Adams Matthew D, Arain Altaf, Papatheodorou Stefania, Koutrakis Petros, Mahmoud Moataz, Requia Weeberb J, Adams Matthew D, Arain Altaf, Papatheodorou Stefania, Koutrakis Petros, Mahmoud Moataz
BACKGROUND	Little is known about the health risks of air pollution and cardiorespiratory diseases, globally, across regions and populations, which may differ because of external factors.
OBJECTIVES	We systematically reviewed the evidence on the association between air pollution and cardiorespiratory diseases (hospital admissions and mortality), including variability by energy, transportation, socioeconomic status, and air quality.
SEARCH METHODS	We conducted a literature search (PubMed and Web of Science) for studies published between 2006 and May 11, 2016.
SELECTION CRITERIA	We included studies if they met all of the following criteria: (1) considered at least 1 of these air pollutants: carbon monoxide, sulfur dioxide, nitrogen dioxide, ozone, or particulate matter (PM2.5 or PM10); (2) reported risk for hospital admissions, mortality, or both; (3) presented individual results for respiratory diseases, cardiovascular diseases, or both; (4) considered the age groups younger than 5 years, older than 65 years, or all ages; and (5) did not segregate the analysis by gender.
DATA COLLECTION AND ANALYSIS	We extracted data from each study, including location, health outcome, and risk estimates. We performed a meta-analysis to estimate the overall effect and to account for both within- and between-study heterogeneity. Then, we applied a model selection (least absolute shrinkage and selection operator) to assess the modifier variables, and, lastly, we performed meta-regression analyses to evaluate the modifier variables contributing to heterogeneity among studies.
MAIN RESULTS	We assessed 2183 studies, of which we selected 529 for in-depth review, and 70 articles fulfilled our study inclusion criteria. The 70 studies selected for meta-analysis encompass more than 30 million events across 28 countries. We found positive associations between cardiorespiratory diseases and different air pollutants. For example, when we considered only the association between PM2.5 and respiratory diseases (Figure 1 , we observed a risk equal to 2.7% (95% confidence interval = 0.9%, 7.7%). Our results showed statistical significance in the test of moderators for all pollutants, suggesting that the modifier variables influence the average cardiorespiratory disease risk and may explain the varying effects of air pollution.
CONCLUSIONS	Variables related to aspects of energy, transportation, and socioeconomic status may explain the varying effect size of the association between air pollution and cardiorespiratory diseases. Public Health Implications. Our study provides a transferable model to estimate the health effects of air pollutants to support the creation of environmental health public policies for national and international intervention.

표-558. PubMed 논문번호 29100550의 내용 요약

구분	내용
PubMed ID	29100550
TITLE	Effects of a liquefied petroleum gas stove intervention on pollutant exposure and adult cardiopulmonary outcomes (CHAP): study protocol for a randomized controlled trial.
JOURNAL	Trials: 10.1186/s13063-017-2179-x
AUTHORS	Fandi□o-Del-Rio Magdalena, Goodman Dina, Kephart Josiah L, Miele Catherine H, Williams Kendra N, Moazzami Mitra, Fung Elizabeth C, Koehler Kirsten, Davila-Roman Victor G, Lee Kathryn A, Nangia Saachi, Harvey Steven A, Steenland Kyle, Gonzales Gustavo F, Checkley William
BACKGROUND	Biomass fuel smoke is a leading risk factor for the burden of disease worldwide. International campaigns are promoting the widespread adoption of liquefied petroleum gas (LPG) in resource-limited settings. However, it is unclear if the introduction and use of LPG stoves, in settings where biomass fuels are used daily, reduces pollution concentration exposure, improves health outcomes, or how cultural and social barriers influence the exclusive adoption of LPG stoves.
METHODS	We will conduct a randomized controlled, field intervention trial of LPG stoves and fuel distribution in rural Puno, Peru, in which we will enroll 180 female participants aged 25-64 years and follow them for 2 years. After enrollment, we will collect information on sociodemographic characteristics, household characteristics, and cooking practices. During the first year of the study, LPG stoves and fuel tanks will be delivered to the homes of 90 intervention participants. During the second year, participants in the intervention arm will keep their LPG stoves, but the gas supply will stop. Control participants will receive LPG stoves and vouchers to obtain free fuel from distributors at the beginning of the second year, but gas will not be delivered. Starting at baseline, we will collect longitudinal measurements of respiratory symptoms, pulmonary function, blood pressure, endothelial function, carotid artery intima-media thickness, 24-h dietary recalls, exhaled carbon monoxide, quality-of-life indicators, and stove-use behaviors. Environmental exposure assessments will occur six times over the 2-year follow-up period, consisting of 48-h personal exposure and kitchen concentration measurements of fine particulate matter and carbon monoxide, and 48-h kitchen concentrations of nitrogen dioxide for a subset of 100 participants.
DISCUSSION	Findings from this study will allow us to better understand behavioral patterns, environmental exposures, and cardiovascular and pulmonary outcomes resulting from the adoption of LPG stoves. If this trial indicates that LPG stoves are a feasible and effective way to reduce household air pollution and improve health, it will provide important information to support widespread adoption of LPG fuel as a strategy to reduce the global burden of disease.
TRIAL REGISTRATION	ClinicalTrials.gov, ID: NCT02994680 , Cardiopulmonary Outcomes and Household Air Pollution (CHAP) Trial. Registered on 28 November 2016.

표-559. PubMed 논문번호 29102669의 내용 요약

구분	내용
PubMed ID	29102669
TITLE	Acute particulate matter affects cardiovascular autonomic modulation and IFN-γ methylation in healthy volunteers.
JOURNAL	Environmental research: 10.1016/j.envres.2017.10.036
AUTHORS	Tobaldini Eleonora, Bollati Valentina, Prado Marta, Fiorelli Elisa M, Pecis Marica, Bissolotti Giorgio, Albetti Benedetta, Cantone Laura, Favero Chiara, Cogliati Chiara, Carrer Paolo, Baccarelli Andrea, Bertazzi Pier Alberto, Montano Nicola
AIMS	Air particulate matter (PM) is associated with increased cardiovascular morbidity and mortality. Altered autonomic functions play a key role in PM-induced cardiovascular disease. However, previous studies have not address the impact of PM on sympathetic and parasympathetic control of heart function, independently, and using controlled conditions, i.e., increasing titration of PM of known composition, in absence of other potential confounding factors. To fill this gap, here we used symbolic analysis that is capable of detecting non-mutual changes of the two autonomic branches, thus considering them as independent, and concentrations of PM as they could be measured at peak levels in Milan during a polluted winter day.
METHODS AND RESULTS	In this randomized, cross-over study, we enrolled 12 healthy subjects who underwent two random sessions: inhalation of filtered air mixture or inhalation of filtered air containing particulate mixture (PM 10, PM 2.5, PM 1.0 and PM 0.5□m). ECG and respiration for autonomic analysis and blood sample for DNA Methylation were collected at baseline (T1), after air exposure (T2) and after 2h (T3). Spectral and symbolic analysis of heart rate variability (HRV) were performed for autonomic control of cardiac function, while alterations in DNA methylation of candidate genes were used to index pro-inflammatory modifications. In the PM expose group, autonomic analysis revealed a significant decrease of 2UV%, index of parasympathetic modulation (14% vs 9%, p = 0.0309), while DNA analysis showed a significant increase of interferon γ (IFN- γ) methylation, from T1 to T3. In a mixed model using T1, T2 and T3, fine and ultrafine PM fractions showed significant associations with IFN- γ methylation and parasympathetic modulation.
CONCLUSIONS	Our study shows, for the first time, that in healthy subjects, acute exposure to PM affects parasympathetic control of heart function and it increases methylation of a pro-inflammatory gene (i.e. methylation of interferon γ). Thus, our study suggests that, even in absence of other co-factors and in otherwise healthy individuals, PM per se is sufficient to trigger parasympathetic dysautonomia, independently from changes in sympathetic control, and inflammation, in a dose-dependent manner.

표-560. PubMed 논문번호 29116930의 내용 요약

구분	내용
PubMed ID	29116930
TITLE	Long-term Fine Particulate Matter Exposure and Nonaccidental and Cause-specific Mortality in a Large National Cohort of Chinese Men.
JOURNAL	Environmental health perspectives: 10.1289/EHP1673
AUTHORS	Yin Peng, Brauer Michael, Cohen Aaron, Burnett Richard T, Liu Jiangmei, Liu Yunning, Liang Ruiming, Wang Weihua, Qi Jinlei, Wang Lijun, Zhou Maigeng
BACKGROUND	Cohort studies in North America and western Europe have reported increased risk of mortality associated with long-term exposure to fine particles (PM2.5), but to date, no such studies have been reported in China, where higher levels of exposure are experienced.
OBJECTIVES	We estimated the association between long-term exposure to PM2.5 with nonaccidental and cause-specific mortality in a cohort of Chinese men.
METHODS	We conducted a prospective cohort study of 189,793 men 40 y old or older during 1990-91 from 45 areas in China. Annual average PM2.5 levels for the years 1990, 1995, 2000, and 2005 were estimated for each cohort location using a combination of satellite-based estimates, chemical transport model simulations, and ground-level measurements developed for the Global Burden of Disease (GBD) 2013 study. A Cox proportional hazards regression model was used to estimate hazard ratios (HR) for nonaccidental cardiovascular disease (CVD), chronic obstructive pulmonary disease (COPD), and lung-cancer mortality. We also assessed the shape of the concentration-response relationship and compared the risk estimates with those predicted by Integrated Exposure-Response (IER) function, which incorporated estimates of mortality risk from previous cohort studies in western Europe and North America.
RESULTS	The mean level of PM2.5 exposure during 2000-2005 was 43.7 μg/m3 (ranging from 4.2 to 83.8 μg/m3). Mortality HRs (95% CI) per 10- μg/m3 increase in PM2.5 were 1.09 (1.08, 1.09) for nonaccidental causes; 1.09 (1.08, 1.10) for CVD, 1.12 (1.10, 1.13) for COPD; and 1.12 (1.07, 1.14) for lung cancer. The HR estimate from our cohort was consistently higher than IER predictions.
CONCLUSIONS	Long-term exposure to PM2.5 was associated with nonaccidental, CVD, lung cancer, and COPD mortality in China. The IER estimator may underestimate the excess relative risk of cause-specific mortality due to long-term exposure to PM2.5 over the exposure range experienced in China and other low- and middle-income countries. https://doi.org/10.1289/EHP1673.

표-561. PubMed 논문번호 29118034의 내용 요약

구분	내용
PubMed ID	29118034
TITLE	Cardiovascular Effects of Long-Term Exposure to Air Pollution: A Population-Based Study With 900 845 Person-Years of Follow-up.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.117.007170
AUTHORS	Kim Hyeanji, Kim Joonghee, Kim Sunhwa, Kang Si-Hyuck, Kim Hee-Jun, Kim Ho, Heo Jongbae, Yi Seung-Muk, Kim Kyuseok, Youn Tae-Jin, Chae In-Ho
BACKGROUND	Studies have shown that long-term exposure to air pollution such as fine particulate matter (≤2.5 μm in aerodynamic diameter [PM2.5]) increases the risk of all-cause and cardiovascular mortality. To date, however, there are limited data on the impact of air pollution on specific cardiovascular diseases. This study aimed to evaluate cardiovascular effects of long-term exposure to air pollution among residents of Seoul, Korea.
METHODS AND RESULTS	Healthy participants with no previous history of cardiovascular disease were evaluated between 2007 and 2013. Exposure to air pollutants was estimated by linking the location of outdoor monitors to the ZIP code of each participant's residence. Crude and adjusted analyses were performed using Cox regression models to evaluate the risk for composite cardiovascular events including cardiovascular mortality, acute myocardial infarction, congestive heart failure, and stroke. A total of 136 094 participants were followed for a median of 7.0 years (900 845 person-years). The risk of major cardiovascular events increased with higher mean concentrations of PM2.5 in a linear relationship, with a hazard ratio of 1.36 (95% confidence interval, 1.29-1.43) per 1 μg/m3 PM2.5. Other pollutants including PM2.5-10 of CO, SO2, and NO2, but not O3, were significantly associated with increased risk of cardiovascular events. The burden from air pollution was comparable to that from hypertension and diabetes mellitus.
CONCLUSIONS	This large-scale population-based study demonstrated that long-term exposure to air pollution including PM2.5 increases the risk of major cardiovascular disease and mortality. Air pollution should be considered an important modifiable environmental cardiovascular risk factor.

표-562. PubMed 논문번호 29154226의 내용 요약

구분	내용
PubMed ID	29154226
TITLE	Short-term exposures to PM〈sub〉2.5〈/sub〉 and cause-specific mortality of cardiovascular health in China.
JOURNAL	Environmental research: 10.1016/j.envres.2017.10.046
AUTHORS	Chen Chen, Zhu Pengfei, Lan Li, Zhou Lian, Liu Ruicong, Sun Qinghua, Ban Jie, Wang Wentao, Xu Dandan, Li Tiantian, Chen Chen, Zhu Pengfei, Lan Li, Zhou Lian, Liu Ruicong, Sun Qinghua, Ban Jie, Wang Wentao, Xu Dandan, Li Tiantian
BACKGROUND	Many multi-center epidemiological studies have robustly examined the acute health effects of exposure to low concentrations of fine particulate matter (PM2.5) on cardiovascular mortality in developed counties. However, data limitations have resulted in few related studies being conducted in developing counties with high levels of PM2.5 exposure. In recent years, people in China with a heavy cardiovascular disease burden have been exposed to particularly high levels of PM2.5.
OBJECTIVE	We conducted a multi-county time series study investigating the acute effects of PM2.5 on the increased risk of cardiovascular death across China, and explored subpopulations susceptible to PM2.5 exposure.
METHODS	Appling a county-specific Poisson regression in 30 Chinese counties, we estimated PM2.5 effects on all-cause mortality and cause-specific mortality of cardiovascular health for 2013-2015. We also considered PM2.5 effects on several subpopulations, including males, females, and three age groups (〈 65, 65-74 and 〉 74 years old). We pooled the county-specific results across China using a random effects meta-analysis by cause and by subpopulation.
RESULTS	We found a 0.13% (95% confidence interval (CI), 0.04-0.22) increase in all-cause mortality, a 0.12% increase (95% CI, 0.001-0.25) increase in cardiovascular disease (CVD), a 0.42% (95% CI, 0.03-0.81) increase in AMI, a 0.17% (95% CI, -0.04-0.40) increase in coronary heart disease, and a 0.13% (95% CI, -0.12-0.33) increase in stroke in association with a 10- μg/m3 increase in PM2.5 concentrations on the same day. The magnitudes of the associations were less than those reported in developed counties with lower PM2.5 levels. A vulnerable effect on all-cause mortality was observed in the elderly population (older than 65 years) and on CVD in males.
CONCLUSIONS	This study showed the positive magnitude of PM2.5 effects with high exposure on all natural, CVD, and cause-specific mortality and on the susceptible populations in China. The findings complemented evidence related to exposure-mortality relationships at the higher end of short-term exposure to PM2.5 on a global scale.

표-563. PubMed 논문번호 29191927의 내용 요약

구분	내용
PubMed ID	29191927
TITLE	Associations Between Residential Proximity to Traffic and Vascular Disease in a Cardiac Catheterization Cohort.
JOURNAL	Arteriosclerosis, thrombosis, and vascular biology: 10.1161/ATVBAHA.117.310003
AUTHORS	Ward-Caviness Cavin K, Kraus William E, Blach Colette, Haynes Carol S, Dowdy Elaine, Miranda Marie Lynn, Devlin Robert, Diaz-Sanchez David, Cascio Wayne E, Mukerjee Shaibal, Stallings Casson, Smith Luther A, Gregory Simon G, Shah Svati H, Neas Lucas M, Hauser Elizabeth R, Ward-Caviness Cavin K, Kraus William E, Blach Colette, Haynes Carol S, Dowdy Elaine, Miranda Marie Lynn, Devlin Robert, Diaz-Sanchez David, Cascio Wayne E, Mukerjee Shaibal, Stallings Casson, Smith Luther A, Gregory Simon G, Shah Svati H, Neas Lucas M, Hauser Elizabeth R
OBJECTIVE	Exposure to mobile source emissions is nearly ubiquitous in developed nations and is associated with multiple adverse health outcomes. There is an ongoing need to understand the specificity of traffic exposure associations with vascular outcomes, particularly in individuals with cardiovascular disease.
APPROACH AND RESULTS	We performed a cross-sectional study using 2124 individuals residing in North Carolina, United States, who received a cardiac catheterization at the Duke University Medical Center. Traffic-related exposure was assessed via 2 metrics: (1) the distance between the primary residence and the nearest major roadway; and (2) location of the primary residence in regions defined based on local traffic patterns. We examined 4 cardiovascular disease outcomes: hypertension, peripheral arterial disease, the number of diseased coronary vessels, and recent myocardial infarction. Statistical models were adjusted for race, sex, smoking, type 2 diabetes mellitus, body mass index, hyperlipidemia, and home value. Results are expressed in terms of the odds ratio (OR). A 23% decrease in residential distance to major roadways was associated with higher prevalence of peripheral arterial disease (OR=1.29; 95% confidence interval, 1.08-1.55) and hypertension (OR=1.15; 95% confidence interval, 1.01-1.31). Associations with peripheral arterial disease were strongest in men (OR=1.42; 95% confidence interval, 1.17-1.74) while associations with hypertension were strongest in women (OR=1.21; 95% confidence interval, 0.99-1.49). Neither myocardial infarction nor the number of diseased coronary vessels were associated with traffic exposure.
CONCLUSIONS	Traffic-related exposure is associated with peripheral arterial disease and hypertension while no associations are observed for 2 coronary-specific vascular outcomes.

표-564. PubMed 논문번호 29212623의 내용 요약

구분	내용
PubMed ID	29212623
TITLE	Intrauterine and Early Postnatal Exposure to Particulate Air Pollution and Kawasaki Disease: A Nationwide Longitudinal Survey in Japan.
JOURNAL	The Journal of pediatrics: 10.1016/j.jpeds.2017.10.012
AUTHORS	Yorifuji Takashi, Tsukahara Hirokazu, Kashima Saori, Doi Hiroyuki, Yorifuji Takashi, Tsukahara Hirokazu, Kashima Saori, Doi Hiroyuki
OBJECTIVES	To examine the effects of prenatal and postnatal exposure to particulate matter on Kawasaki disease (KD) occurrence, using data from a nationwide population-based longitudinal survey in Japan that began in 2010.
STUDY DESIGN	Prenatal and postnatal suspended particulate matter concentrations were obtained at municipality level and assigned to participants based on their municipality of birth. We analyzed data from 30 367 participants with data on either exposure period. We used hospital admission for KD from 6 to 30 months of age as the main outcome of interest. We conducted a multilevel logistic regression analysis, adjusting for individual and municipality-level variables.
RESULTS	Children who were exposed to higher levels of suspended particulate matter, in particular during pregnancy, were more likely to be hospitalized for KD. The ORs for ≥25 □g/m3 exposure compared with 〈20 □g/m3 exposure were 1.59 (95% CI 1.06, 2.38) for prenatal exposure and 1.41 (0.82, 2.41) for postnatal exposure. Prenatal exposure during mid-to-late gestation seemed to be more relevant for the increased risk.
CONCLUSIONS	Early life exposure to particulate air pollution, in particular during pregnancy, is associated with an increased risk of KD hospital admission in early childhood in a nationally representative sample in Japan.

표-565. PubMed 논문번호 29221643의 내용 요약

구분	내용
PubMed ID	29221643
TITLE	Respiratory and cardiovascular responses to walking down a traffic-polluted road compared with walking in a traffic-free area in participants aged 60 years and older with chronic lung or heart disease and age-matched healthy controls: a randomised, crossover study.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(17)32643-0
AUTHORS	Sinharay Rudy, Gong Jicheng, Barratt Benjamin, Ohman-Strickland Pamela, Ernst Sabine, Kelly Frank J, Zhang Junfeng Jim, Collins Peter, Cullinan Paul, Chung Kian Fan, Sinharay Rudy, Gong Jicheng, Barratt Benjamin, Ohman-Strickland Pamela, Ernst Sabine, Kelly Frank J, Zhang Junfeng Jim, Collins Peter, Cullinan Paul, Chung Kian Fan
BACKGROUND	Long-term exposure to pollution can lead to an increase in the rate of decline of lung function, especially in older individuals and in those with chronic obstructive pulmonary disease (COPD), whereas shorter-term exposure at higher pollution levels has been implicated in causing excess deaths from ischaemic heart disease and exacerbations of COPD. We aimed to assess the effects on respiratory and cardiovascular responses of walking down a busy street with high levels of pollution compared with walking in a traffic-free area with lower pollution levels in older adults.
METHODS	In this randomised, crossover study, we recruited men and women aged 60 years and older with angiographically proven stable ischaemic heart disease or stage 2 Global initiative for Obstructive Lung Disease (GOLD) COPD who had been clinically stable for 6 months, and age-matched healthy volunteers. Individuals with ischaemic heart disease or COPD were recruited from existing databases or outpatient respiratory and cardiology clinics at the Royal Brompton & Harefield NHS Foundation Trust and age-matched healthy volunteers using advertising and existing databases. All participants had abstained from smoking for at least 12 months and medications were taken as recommended by participants' doctors during the study. Participants were randomly assigned by drawing numbered disks at random from a bag to do a 2 h walk either along a commercial street in London (Oxford Street) or in an urban park (Hyde Park). Baseline measurements of participants were taken before the walk in the hospital laboratory. During each walk session, black carbon, particulate matter (PM) concentrations, ultrafine particles, and nitrogen dioxide (NO2) concentrations were measured.
FINDINGS	Between October, 2012, and June, 2014, we screened 135 participants, of whom 40 healthy volunteers, 40 individuals with COPD, and 39 with ischaemic heart disease were recruited. Concentrations of black carbon, NO2, PM10, PM2.5, and ultrafine particles were higher on Oxford Street than in Hyde Park. Participants with COPD reported more cough (odds ratio [OR] 1·95, 95% CI 0·96-3·95; p<0·1), sputum (3·15, 1·39-7·13; p<0·05), shortness of breath (1·86, 0·97-3·57; p<0·1), and wheeze (4·00, 1·52-10·50; p<0·05) after walking down Oxford Street compared with Hyde Park. In all participants, irrespective of their disease status, walking in Hyde Park led to an increase in lung function (forced expiratory volume in the first second [FEV1] and forced vital capacity [FVC]) and a decrease in pulse wave velocity (PWV) and augmentation index up to 26 h after the walk. By contrast, these beneficial responses were attenuated after walking on Oxford Street. In participants with COPD, a reduction in FEV1 and FVC, and an increase in R5-20 were associated with an increase in during-walk exposure to NO2, ultrafine particles and PM2.5, and an increase in PWV and augmentation index with NO2 and ultrafine particles. In healthy volunteers, PWV and

표-566. PubMed 논문번호 29247470의 내용 요약

구분	내용
PubMed ID	29247470
TITLE	Acute effect of multiple ozone metrics on mortality by season in 34 Chinese counties in 2013-2015.
JOURNAL	Journal of internal medicine: 10.1111/joim.12724
AUTHORS	Sun Q, Wang W, Chen C, Ban J, Xu D, Zhu P, He M Z, Li T
BACKGROUND	Although numerous multicentre studies have estimated the association between ozone exposure and mortality, there are currently no nationally representative multicentre studies of the ozone-mortality relationship in China.
OBJECTIVE	To investigate the effect on total (nonaccidental) and cause-specific mortality of short-term exposure to ambient ozone, and examine different exposure metrics.
METHODS	The effects of short-term exposure to ozone were analysed using various metrics (daily 1-h maximum, daily 8-h maximum and daily average) on total (nonaccidental) and cause-specific (circulatory and respiratory) mortality from 2013 to 2015 in 34 counties in 10 cities across China. We used distributed lag nonlinear models for estimating county-specific relative risk of mortality and combined the county-specific relative rates by conducting a random-effects meta-analysis.
RESULTS	In all-year analyses, a 10 μg m-3 increase in daily average, daily 1-h maximum and daily 8-h maximum ozone at lag02 corresponded to an increase of 0.6% (95% CI: 0.33, 0.88), 0.26% (95% CI: 0.12, 0.39) and 0.37% (95% CI: 0.2, 0.55) in total (nonaccidental) mortality, 0.66% (95% CI: 0.28, 1.04), 0.31% (95% CI: 0.11, 0.51) and 0.39% (95% CI: 0.16, 0.62) in circulatory mortality, and 0.57% (95% CI: -0.09, 1.23), 0.11% (95% CI: -0.22, 0.44) and 0.22% (95% CI: -0.28, 0.72) in respiratory mortality, respectively. These estimates had a different seasonal pattern by cause of death. In general, the seasonal patterns were consistent with the times of year when ozone concentrations are highest.
CONCLUSIONS	Our findings suggest that in China, the acute effects of ozone are more closely related to daily average exposure than any other metric.

표-567. PubMed 논문번호 29268751의 내용 요약

구분	내용
PubMed ID	29268751
TITLE	Exposure to ambient air pollution and calcification of the mitral annulus and aortic valve: the multi-ethnic study of atherosclerosis (MESA).
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-017-0346-x
AUTHORS	Tibuakuu Martin, Jones Miranda R, Navas-Acien Ana, Zhao Di, Guallar Eliseo, Gassett Amanda J, Sheppard Lianne, Budoff Matthew J, Kaufman Joel D, Michos Erin D
BACKGROUND	Long-term exposure to high ambient air pollution has been associated with coronary artery calcium (CAC), a marker of cardiovascular disease (CVD). Calcifications of left-sided heart valves are also markers of CVD risk. We investigated whether air pollution was associated with valvular calcification and its progression.
METHODS	We studied 6253 MESA participants aged 45-84 years who underwent two cardiac CT scans 2.5 years apart to quantify aortic valve calcium (AVC) and mitral annular calcium (MAC). CAC was included for the same timeframe for comparison with AVC/MAC. Ambient particulate matter <2.5 μm (PM2.5) and oxides of nitrogen (NOx) concentrations were predicted from residence-specific spatio-temporal models.
RESULTS	The mean age (SD) of the study sample was 62 (10) years, 39% were white, 27% black, 22% Hispanic, and 12% Chinese. The prevalence of AVC and MAC at baseline were 13% and 9% respectively, compared to 50% prevalence of CAC. The adjusted prevalence ratios of AVC and MAC for each 5 μg/m3 higher PM2.5 was 1.19 (95% CI 0.87, 1.62) and 1.20 (0.81, 1.77) respectively, and for CAC was 1.14 (1.01, 1.27). Over 2.5 years, the mean change in Agatston units/year for each 5 μg/m3 higher PM2.5 concentration was 0.29 (-5.05, 5.63) for AVC and 4.38 (-9.13, 17.88) for MAC, compared to 8.66 (0.61, 16.71) for CAC. We found no significant associations of NOx with AVC and MAC.
CONCLUSION	Our findings suggest a trend towards increased 2.5-year progression of MAC with exposure to outdoor PM2.5, although this association could not be confirmed. Additional well-powered studies with longer periods of follow-up are needed to further study associations of air pollution with valvular calcium.
TRIAL REGISTRATION	Although MESA is not a clinical trial, this cohort is registered at ClinicalTrials.gov Identifier: NCT00005487; Date of registration May 25, 2000.

표-568. PubMed 논문번호 29306794의 내용 요약

구분	내용
PubMed ID	29306794
TITLE	The short-term effects of indoor size-fractioned particulate matter and black carbon on cardiac autonomic function in COPD patients.
JOURNAL	Environment international: 10.1016/j.envint.2017.12.037
AUTHORS	Pan Lu, Wu Shaowei, Li Hongyu, Xu Junhui, Dong Wei, Shan Jiao, Yang Xuan, Chen Yahong, Shima Masayuki, Deng Furong, Guo Xinbiao
BACKGROUND	Previous studies have reported adverse health effects of indoor air pollutants especially particulate matter (PM) and black carbon (BC). Patients with chronic obstructive pulmonary disease (COPD) have been shown to be more likely with cardiovascular comorbidities in which cardiac autonomic dysfunction plays an important role. However, there is little evidence for the effect of indoor PM and BC exposures on cardiac autonomic function in COPD patients.
OBJECTIVES	To evaluate the association between exposure to indoor size-fractioned PM and BC and changes in HRV and HR in COPD patients.
METHODS	Forty-three doctor diagnosed, stable COPD patients were recruited and measured for 24-h HRV and HR. Real-time indoor size-fractioned PM and BC were monitored on the day before and the day of performing health measurements. Mixed-effects models were used to estimate the associations between indoor PM and BC and HRV indices and HR after controlling for potential confounders.
RESULTS	Increasing levels of size-fractioned PM and BC were associated with decreased HRV indices and increased HR. An IQR (3.14 μg/m3) increase in 8-h BC moving average and an IQR (20.72 μg/m3) increase in 5-min PM0.5 moving average concentrations were associated with declines of 7.45% (95% CI: -10.89%, -3.88%) and 16.40% (95% CI: -21.06%, -11.41%) in LF, respectively. The smaller the particles size, the greater effects on HRV indices and HR. Patients' BMI modified the associations between size-fractioned PM and BC and their HRV and HR. For an IQR increase in PM0.5, there was decline in HF of 34.85% (95% CI: -39.08%, -30.33%) in overweight patients, compared to a 2.01% (95% CI: -6.44%, 11.19%) increase in normal-weight patients.
CONCLUSIONS	Exposures to indoor PM and BC were associated with altered cardiac autonomic function in COPD patients, and the associations for HRV measures of parasympathetic activity (e.g., HF) were more apparent in overweight patients.

표-569. PubMed 논문번호 29310044의 내용 요약

구분	내용
PubMed ID	29310044
TITLE	Short-term exposure to traffic-related air pollution and ischemic stroke onset in Barcelona, Spain.
JOURNAL	Environmental research: 10.1016/j.envres.2017.12.024
AUTHORS	Vivanco-Hidalgo Rosa Maria, Wellenius Gregory A, Basaga□a Xavier, Cirach Marta, Gonz□lez Alejandra G□mez, Ceballos Pablo de, Zabalza Ana, Jim□nez-Conde Jordi, Soriano-Tarraga Carolina, Giralt-Steinhauer Eva, Alastuey Andr□s, Querol Xavier, Sunyer Jordi, Roquer Jaume
OBJECTIVE	To assess the relationship between short-term exposure to outdoor ambient air pollutants (fine particulate matter [PM2.5] and black carbon [BC]), ischemic stroke (IS) and its different subtypes, and the potential modifying effect of neighborhood greenspace and noise.
METHODS	This time-stratified case-crossover study was based on IS and transient ischemic attacks (TIA) recorded in a hospital-based prospective stroke register (BASICMAR 2005-2014) in Barcelona (Catalonia, Spain). Daily and hourly pollutant concentrations and meteorological data were obtained from monitoring stations in the city. Time-lags (from previous 72h to acute stroke onset) were analyzed. Greenness and noise were determined from the Normalized Difference Vegetation Index (NDVI) and daily average noise level at the street nearest to residential address, respectively.
RESULTS	The 2742 cases with known onset date and time, living in the study area, were analyzed. After adjusting for temperature, no statistically significant association between pollutants exposure and overall stroke risk was found. In subtype analysis, an association was detected between BC exposure at 24-47h (odds ratio, 1.251; 95% confidence interval [CI], 1.001-1.552; P = 0.042) and 48-72h (1.211; 95% CI, 0.988-1.484; P = 0.065) time-lag prior to stroke onset and large-artery atherosclerosis subtype. No clear modifying effect of greenness or noise was observed.
CONCLUSIONS	Overall, no association was found between PM2.5 and BC exposure and acute IS risk. By stroke subtype, large-artery atherosclerotic stroke could be triggered by daily increases in BC, a diesel fuel-related pollutant in the study area.

표-570. PubMed 논문번호 29319630의 내용 요약

구분	내용
PubMed ID	29319630
TITLE	Exposure to Ambient Ultrafine Particles and Nitrogen Dioxide and Incident Hypertension and Diabetes.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000798
AUTHORS	Bai Li, Chen Hong, Hatzopoulou Marianne, Jerrett Michael, Kwong Jeffrey C, Burnett Richard T, van Donkelaar Aaron, Copes Ray, Martin Randall V, Van Ryswyk Keith, Lu Hong, Kopp Alexander, Weichenthal Scott
BACKGROUND	Previous studies reported that long-term exposure to traffic-related air pollution may increase the incidence of hypertension and diabetes. However, little is known about the associations of ultrafine particles (≤0.1 μm in diameter) with these two conditions.
METHODS	We conducted a population-based cohort study to investigate the associations between exposures to ultrafine particles and nitrogen dioxide (NO2) and the incidence of diabetes and hypertension. Our study population included all Canadian-born residents aged 30 to 100 years who lived in the City of Toronto, Canada, from 1996 to 2012. Outcomes were ascertained using validated province-wide databases. We estimated annual concentrations of ultrafine particles and NO2 using land-use regression models and assigned these estimates to participants' annual postal code addresses during the follow-up period. Using random-effects Cox proportional hazards models, we calculated hazard ratios (HRs) and 95% confidence intervals (CIs) for ultrafine particles and NO2, adjusted for individual- and neighborhood-level covariates. We considered both single- and multipollutant models.
RESULTS	Each interquartile change in exposure to ultrafine particles was associated with increased risk of incident hypertension (HR = 1.03; 95% CI = 1.02, 1.04) and diabetes (HR = 1.06; 95% CI = 1.05, 1.08) after adjusting for all covariates. These results remained unaltered with further control for fine particulate matter (≤2.5 μm; PM2.5) and NO2. Similarly, NO2 was positively associated with incident diabetes (HR = 1.06; 95% CI = 1.05, 1.07) after controlling for ultrafine particles and PM2.5.
CONCLUSIONS	Exposure to traffic-related air pollution including ultrafine particles and NO2 may increase the risk for incident hypertension and diabetes. See video abstract at, http://links.lww.com/EDE/B337.

표-571. PubMed 논문번호 29342453의 내용 요약

구분	내용
PubMed ID	29342453
TITLE	Fine Particulate Air Pollution and the Expression of microRNAs and Circulating Cytokines Relevant to Inflammation, Coagulation, and Vasoconstriction.
JOURNAL	Environmental health perspectives: 10.1289/EHP1447
AUTHORS	Chen Renjie, Li Huichu, Cai Jing, Wang Cuicui, Lin Zhijing, Liu Cong, Niu Yue, Zhao Zhuohui, Li Weihua, Kan Haidong
BACKGROUND	MicroRNAs (miRNAs) are a key factor in epigenetic regulation of gene expression, but miRNA responses to fine particulate matter (PM2.5) air pollution and their potential contribution to cardiovascular effects of PM2.5 are unknown.
OBJECTIVE	We explored the potential influence of PM2.5 on the expression of selected cytokines relevant to systemic inflammation, coagulation, and vasoconstriction, and on miRNAs that may regulate their expression.
METHODS	We designed a double-blind, randomized crossover study in which true and sham air purifiers were used to expose 55 healthy young adult students in Shanghai, China, to reduced or ambient levels of indoor PM2.5 during two-week periods, and we measured the expression (mRNA and protein) of 10 serum cytokines, and miRNAs that target them, after each intervention period. We used linear mixed-effect models to estimate associations of the intervention, and time-weighted personal PM2.5 exposures, with the cytokines, mRNA, and miRNAs; we also explored potential mediation by miRNAs.
RESULTS	The findings were generally consistent for associations with the intervention and for associations with an interquartile range increase in time-weighted PM2.5. Specifically, higher PM2.5 exposure was positively associated with the expression (mRNA, protein, or both) of interleukin-1 (encoded by IL1), IL6, tumor necrosis factor (encoded by TNF), toll-like receptor 2 (encoded by TLR2), coagulation factor 3 (encoded by F3), and endothelin 1 (encoded by EDN1), and was negatively associated with miRNAs (miR-21-5p, miR-187-3p, miR-146a-5p, miR-1-3p, and miR-199a-5p) predicted to target mRNAs of IL1, TNF, TLR2, and EDN1.
CONCLUSIONS	Our findings require confirmation but suggest that effects of PM2.5 on cardiovascular diseases may be related to acute effects on cytokine expression, which may be partly mediated through effects of PM2.5 on miRNAs that regulate cytokine expression. https://doi.org/10.1289/EHP1447.

표-572. PubMed 논문번호 29351544의 내용 요약

구분	내용
PubMed ID	29351544
TITLE	Long-Term Exposure to Fine Particulate Matter, Blood Pressure, and Incident Hypertension in Taiwanese Adults.
JOURNAL	Environmental health perspectives: 10.1289/EHP2466
AUTHORS	Zhang Zilong, Guo Cui, Lau Alexis K H, Chan Ta-Chien, Chuang Yuan Chieh, Lin Changqing, Jiang Wun Kai, Yeoh Eng-Kiong, Tam Tony, Woo Kam S, Yan Bryan P, Chang Ly-Yun, Wong Martin C S, Lao Xiang Qian
BACKGROUND	Long-term exposure to particulate matter (PM) air pollution may increase blood pressure and the risk of hypertension. However, epidemiological evidence is scarce and inconsistent.
OBJECTIVES	We investigated the associations between long-term exposure to PM with an aerodynamic diameter <2.5 μm (PM2.5), blood pressure, and incident hypertension in a large Taiwanese cohort.
METHODS	We studied 361,560 adults ≥18y old from a large cohort who participated in a standard medical examination program during 2001 to 2014. Among this group, 125,913 nonhypertensive participants were followed up. A satellite-based spatiotemporal model was used to estimate the 2-y average PM2.5 concentrations at each participant's address. Multivariable linear regression was used in the cross-sectional data analysis with the 361,560 participants to investigate the associations between PM2.5 and systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse pressure (PP), and Cox proportional hazard regression was used in the cohort data analysis with the 125,913 participants to investigate the associations between PM2.5 and incident hypertension.
RESULTS	Each 10- μg/m3 increment in the 2-y average PM2.5 concentration was associated with increases of 0.45 mmHg [95% confidence interval (CI): 0.40, 0.50], 0.07 mmHg (95% CI: 0.04, 0.11), and 0.38 mmHg (95% CI: 0.33, 0.42) in SBP, DBP, and PP, respectively, after adjusting for a wide range of covariates and possible confounders. Each 10- μg/m3 increment in the 2-y average PM2.5 concentration was associated with an increase of 3% in the risk of developing hypertension [hazard ratio=1.03 (95% CI: 1.01, 1.05)]. Stratified and sensitivity analyses yielded similar results.
CONCLUSIONS	Long-term exposure to PM2.5 air pollution is associated with higher blood pressure and an increased risk of hypertension. These findings reinforce the importance of air pollution mitigation strategies to reduce the risk of cardiovascular disease. https://doi.org/10.1289/EHP2466.

표-573. PubMed 논문번호 29354807의 내용 요약

구분	내용
PubMed ID	29354807
TITLE	Main air pollutants and ventricular arrhythmias in patients with implantable cardioverter-defibrillators: A systematic review and meta-analysis.
JOURNAL	Chronic diseases and translational medicine: 10.1016/j.cdtm.2017.09.001
AUTHORS	Yang Hong-Jie, Liu Xin, Qu Chuan, Shi Shao-Bo, Liang Jin-Jun, Yang Bo
Objective	Previous studies of ambient air pollutants and ventricular arrhythmias in patients with implantable cardioverter-defibrillator (ICD) have yielded mixed results, and the association between air pollution and ventricular arrhythmias in these patients remains unclear. This study aimed to assess and quantify the association between exposure to major air pollutants [CO, inhalable particles (PM10), SO2, fine particulate matter (PM2.5), O3, and NO2] and the presence of ventricular arrhythmia in patients with ICD.
Methods	The Medline, PubMed, Web of Science, Global Health Library, Virtual Health Library, Population Information Online (POPLINE), and New York Academy of Medicine Grey Literature Report databases were searched to identify studies analyzing the association between ventricular arrhythmias in patients with ICD and the abovementioned main air pollutants. Pooled estimates were generated using a random-effects model or fixed-effects model, according to the value of heterogeneity. Heterogeneity within studies was assessed using Cochran's Q and I2 statistics. Funnel plots, Egger's regression test, and Begg's rank correlation method were used to evaluate publication bias. Sensitivity analyses were also conducted to evaluate the potential sources of heterogeneity.
Results	After a detailed screening of 167 studies, seven separate studies were identified. Ventricular arrhythmias in patients with ICD were found to be positively, but not significantly, associated with CO, PM10, SO2, PM2.5, and NO2, with a pooled estimate [odds ratio (OR) associated with each 10 μg/m3 increase in pollutant concentration, except for CO, which was associated with each 1 mg/m3 increase in concentration] of 1.03 [95% confidence interval (CI): 0.92-1.17, P = 0.59] for CO, 1.01 (95%CI: 0.97-1.05, P = 0.55) for PM10, 1.09 (95%CI: 0.95-1.24, P = 0.22) for SO2, 1.07 (95%CI: 0.95-1.21, P = 0.25) for PM2.5, and 1.06 (95%CI: 0.98-1.14, P = 0.16) for NO2. No increased risk of ventricular arrhythmias in patients with ICD was found to be associated with O3 (OR = 1.00; 95%CI: 0.98-1.01, P = 0.56).
Conclusions	The results of this study provide little evidence that ambient air pollutants affect the risk of ICD discharges for treating ventricular arrhythmias.

표-574. PubMed 논문번호 29361317의 내용 요약

구분	내용
PubMed ID	29361317
TITLE	Associations between ambient air pollution and daily mortality in a cohort of congestive heart failure: Case-crossover and nested case-control analyses using a distributed lag nonlinear model.
JOURNAL	Environment international: 10.1016/j.envint.2018.01.003
AUTHORS	Buteau Stephane, Goldberg Mark S, Burnett Richard T, Gasparrini Antonio, Valois Marie-France, Brophy James M, Crouse Dan L, Hatzopoulou Marianne
BACKGROUND	Persons with congestive heart failure may be at higher risk of the acute effects related to daily fluctuations in ambient air pollution. To meet some of the limitations of previous studies using grouped-analysis, we developed a cohort study of persons with congestive heart failure to estimate whether daily non-accidental mortality were associated with spatially-resolved, daily exposures to ambient nitrogen dioxide (NO2) and ozone (O3), and whether these associations were modified according to a series of indicators potentially reflecting complications or worsening of health.
METHODS	We constructed the cohort from the linkage of administrative health databases. Daily exposure was assigned from different methods we developed previously to predict spatially-resolved, time-dependent concentrations of ambient NO2 (all year) and O3 (warm season) at participants' residences. We performed two distinct types of analyses: a case-crossover that contrasts the same person at different times, and a nested case-control that contrasts different persons at similar times. We modelled the effects of air pollution and weather (case-crossover only) on mortality using distributed lag nonlinear models over lags 0 to 3 days. We developed from administrative health data a series of indicators that may reflect the underlying construct of "declining health", and used interactions between these indicators and the cross-basis function for air pollutant to assess potential effect modification.
RESULTS	The magnitude of the cumulative as well as the lag-specific estimates of association differed in many instances according to the metric of exposure. Using the back-extrapolation method, which is our preferred exposure model, we found for the case-crossover design a cumulative mean percentage changes (MPC) in daily mortality per interquartile increment in NO2 (8.8 ppb) of 3.0% (95% CI: -0.4, 6.6%) and for O3 (16.5 ppb) 3.5% (95% CI: -4.5, 12.1). For O3 there was strong confounding by weather (unadjusted MPC = 7.1%; 95% CI: 1.7, 12.7%). For the nested case-control approach the cumulative MPC for NO2 in daily mortality was 2.9% (95% CI: -0.9, 6.9%) and for O3 7.3% (95% CI: 3.0, 11.9%). We found evidence of effect modification between daily mortality and cumulative NO2 and O3 according to the prescribed dose of furosemide in the nested case-control analysis, but not in the case-crossover analysis.
CONCLUSIONS	Mortality in congestive heart failure was associated with exposure to daily ambient NO2 and O3 predicted from a back-extrapolation method using a land use regression model from dense sampling surveys. The methods used to assess exposure can have considerable influence on the estimated acute health effects of the two air pollutants.

표-575. PubMed 논문번호 29395277의 내용 요약

구분	내용
PubMed ID	29395277
TITLE	Shorter telomere length in cord blood associated with prenatal air pollution exposure: Benefits of intervention.
JOURNAL	Environment international: 10.1016/j.envint.2018.01.005
AUTHORS	Perera Frederica, Lin Chia-Jung, Qu Lirong, Tang Deliang
BACKGROUND	To examine the molecular benefits of the government action to close the local coal burning power plant in Tongliang County, Chongqing Municipality, we compared biologic markers and health outcomes in two successive birth cohorts enrolled before and after the plant was shut down. In this city, polycyclic aromatic hydrocarbons (PAH) were primarily emitted by the coal burning facility. We previously reported that cord blood levels of PAH-DNA adducts (a biomarker of exposure) and various adverse health outcomes were reduced in the second cohort, whereas levels of brain-derived neurotrophic factor/BDNF (a protein involved in neuronal growth) were increased. Here we assessed telomere length (TL), which has been associated with risk of certain chronic diseases, early mortality, aging and cognitive decline in adults.
OBJECTIVES	The goals of the present study were to determine whether TL differed between the two cohorts and whether prenatal PAH exposure, estimated by PAH-DNA adducts in cord white blood cells of newborns in China, were predictive of shorter TL in cord blood, suggesting the potential accrual of risk of certain chronic diseases during the prenatal period. We explored relationships of TL with BDNF and neurodevelopmental outcomes, each previously associated with PAH-DNA adducts in these cohorts, as well as the potential mediating role of TL in the associations between adducts and neurodevelopmental outcomes.
METHODS	We analyzed TL in cord blood of 255 newborns who also had data on PAH-DNA adducts, BDNF, and relevant covariates. Multiple regression analysis was carried out to test associations between adducts and TL and between TL and BDNF, adjusting for relevant covariates. In the subset with developmental quotient (DQ) scores from Gesell testing at age 2 (N = 210), we explored whether TL was a mediator of the relationship between PAH-DNA adducts and DQ scores by first examining the associations between cord adducts and DQ, cord adducts and TL, and TL and DQ, adjusting for the same covariates.
RESULTS	As hypothesized, the mean TL was significantly higher in the second cohort compared to the first cohort. Overall, PAH-DNA cord adducts were significantly and inversely correlated with TL. Multiple regression analysis showed a significant association between adducts and TL, after adjusting for key covariates: β (effect size per standard deviation adducts) = −0.019, p = .003. The regression coefficient of TL on (Ln) BDNF was also significant (β = 0.167, p < .001). Exploratory analysis, regressing TL on Gesell developmental scores, showed generally inverse, but not significant associations. TL was not, therefore, deemed to be a potential mediator of the association between adducts and developmental scores at age two.
CONCLUSION	This study provides the first evidence that prenatal PAH exposure from coal burning may adversely affect TL, with potential implications for future risk of chronic diseases including cardiovascular disease. The improvement in TL in the second cohort and the observed correlation between increased TL and higher levels of BDNF indicate direct benefits to the health and development of children resulting from the government's closure of the power plant.

표-576. PubMed 논문번호 29398771의 내용 요약

구분	내용
PubMed ID	29398771
TITLE	Lessons from in-home air filtration intervention trials to reduce urban ultrafine particle number concentrations.
JOURNAL	Building and environment: 10.1016/j.buildenv.2017.10.007
AUTHORS	Brugge Doug, Simon Matthew C, Hudda Neelakshi, Zellmer Marisa, Corlin Laura, Cleland Stephanie, Lu Eda Yiqi, Rivera Sonja, Byrne Megan, Chung Mei, Durant John L
Background	Exposure to airborne ultrafine particle (UFP; <100 nm in aerodynamic diameter) is an emerging public health problem. Nevertheless, the benefit of using high efficiency particulate arrestance (HEPA) filtration to reduce UFP concentrations in homes is not yet clear.
Methods	We conducted a randomized crossover study of HEPA filtration without a washout period in 23 homes of low-income Puerto Ricans in Boston and Chelsea, MA (USA). Most participants were female, older adults who were overweight or obese. Particle number concentrations (PNC, a proxy for UFP) were measured indoors and outdoors at each home continuously for six weeks. Homes received both HEPA filtration and sham filtration for three weeks each in random order.
Results	Median PNC under HEPA filtration was 50-85% lower compared to sham filtration in most homes, but we found no benefit in terms of reduced inflammation; associations between hsCRP, IL-6, or TNFRII in blood samples and indoor PNC were inverse and not statistically significant.
Conclusions	Limitations to our study design likely contributed to our findings. Limitations included carry-over effects, a population that may have been relatively unresponsive to UFP, reduction in PNC even during sham filtration that limited differences between HEPA and sham filtration, window opening by participants, and lack of fine-grained (room-specific) participant time-activity information. Our approach was similar to other recent HEPA intervention studies of particulate matter exposure and cardiovascular risk, suggesting that there is a need for better study designs.

표-577. PubMed 논문번호 29440184의 내용 요약

구분	내용
PubMed ID	29440184
TITLE	Household air pollution and measures of blood pressure, arterial stiffness and central haemodynamics.
JOURNAL	Heart (British Cardiac Society): 10.1136/heartjnl-2017-312595
AUTHORS	Baumgartner Jill, Carter Ellison, Schauer James J, Ezzati Majid, Daskalopoulou Stella S, Valois Marie-France, Shan Ming, Yang Xudong
OBJECTIVE	We evaluated the exposure-response associations between personal exposure to air pollution from biomass stoves and multiple vascular and haemodynamic parameters in rural Chinese women.
METHODS	We analysed the baseline information from a longitudinal study in southwestern China. Women's brachial and central blood pressure and pulse pressure, carotid-femoral pulse wave velocity and augmentation index, and their 48-hour personal exposures to fine particulate matter (PM2.5) and black carbon were measured in summer and winter. We evaluated the associations between exposure to air pollution and haemodynamic parameters using mixed-effects regression models adjusted for known cardiovascular risk factors.
RESULTS	Women's (n=205, ages 27-86 years) exposures to PM2.5 and black carbon ranged from 14 □g/m3 to 1405 □g/m3 and 0.1-121.8 □g/m3, respectively. Among women aged ≥50 years, increased PM2.5 exposure was associated with higher systolic (brachial: 3.5 mm Hg (P=0.05); central: 4.4 mm Hg (P=0.005)) and diastolic blood pressure (central: 1.3 mm Hg (P=0.10)), higher pulse pressure (peripheral: 2.5 mm Hg (P=0.05); central: 2.9 mm Hg (P=0.008)) and lower peripheral-central pulse pressure amplification (-0.007 (P=0.04)). Among younger women, the associations were inconsistent in the direction of effect and not statistically significant. Increased PM2.5 exposure was associated with no difference in pulse wave velocity and modestly higher augmentation index though the CI included zero (1.1%; 95% CI -0.2% to 2.4%). Similar associations were found for black carbon exposure.
CONCLUSIONS	Exposure to household air pollution was associated with higher blood pressure and central haemodynamics in older Chinese women, with no associations observed with pulse wave velocity.

표-578. PubMed 논문번호 29447790의 내용 요약

구분	내용
PubMed ID	29447790
TITLE	Fine particulate matter and incident coronary heart disease in the REGARDS cohort.
JOURNAL	American heart journal: 10.1016/j.ahj.2017.11.007
AUTHORS	Loop Matthew Shane, McClure Leslie A, Levitan Emily B, Al-Hamdan Mohammad Z, Crosson William L, Safford Monika M, Loop Matthew Shane, McClure Leslie A, Levitan Emily B, Al-Hamdan Mohammad Z, Crosson William L, Safford Monika M
METHODS	Therefore, we estimated the relationship between chronic exposure to PM2.5 and risk for CHD in among participants in the REasons for Geographic And Racial Differences in Stroke (REGARDS) cohort who were free from CHD at baseline (n=17,126). REGARDS is a sample of whites and blacks of both genders living across the continental United States. We fit Cox proportional hazards models for time to CHD to estimate the hazard ratio for baseline 1-year mean PM2.5 exposure, adjusting for environmental variables, demographics, and other risk factors for CHD including the Framingham Risk Score.
RESULTS	The hazard ratio (95% CI) for a 2.7- μg/m3 increase (interquartile range) 1-year mean concentration of PM2.5 was 0.94 (0.83-1.06) for combined CHD death and nonfatal MI, 1.13 (0.92-1.40) for CHD death, and 0.85 (0.73-0.99) for nonfatal MI. We also did not find evidence that these associations depended upon overall CHD risk factor burden.
CONCLUSIONS	Our results do not provide strong evidence for an association between PM2.5 and incident CHD in a heterogeneous cohort, and we conclude that the effects of chronic exposure to fine particulate matter on CHD require further evaluation.

표-579. PubMed 논문번호 29459230의 내용 요약

구분	내용
PubMed ID	29459230
TITLE	Associations of long-term fine particulate matter exposure with prevalent hypertension and increased blood pressure in older Americans.
JOURNAL	Environmental research: 10.1016/j.envres.2018.02.008
AUTHORS	Honda Trenton, Pun Vivian C, Manjourides Justin, Suh Helen
BACKGROUND	Hypertension is a highly prevalent cardiovascular risk factor. It is possible that air pollution, also an established cardiovascular risk factor, may contribute to cardiovascular disease through increasing blood pressure. Previous studies evaluating associations between air pollution and blood pressure have had mixed results.
METHODS	We examined the association between long-term (one-year moving average) air pollutant exposures, prevalent hypertension and blood pressure in 4121 older Americans (57+ years) enrolled in the National Social Life, Health, and Aging Project. We estimated exposures to PM2.5 using spatio-temporal models and used logistic regression accounting for repeated measures to evaluate the association between long-term average PM2.5 and prevalence odds of hypertension. We additionally used linear regression to evaluate the associations between air pollutants and systolic, diastolic, mean arterial, and pulse pressures. Health effect models were adjusted for a number of demographic, health and socioeconomic covariates.
RESULTS	An inter-quartile range (3.91 μg/m3) increase in the one-year moving average of PM2.5 was associated with increased: Odds of prevalent hypertension (POR 1.24, 95% CI: 1.11, 1.38), systolic blood pressure (0.93 mm Hg, 95% CI: 0.05, 1.80) and pulse pressure (0.89 mm Hg, 95% CI: 0.21, 1.58). Dose-response relationships were also observed.
CONCLUSIONS	PM2.5 was associated with increased odds of prevalent hypertension, and increased systolic pressure and pulse pressure in a cohort of older Americans. These findings add to the growing evidence that air pollution may be an important risk factor for hypertension and perturbations in blood pressure.

표-580. PubMed 논문번호 29467108의 내용 요약

구분	내용
PubMed ID	29467108
TITLE	The Association of Long-Term Exposure to Particulate Matter Air Pollution with Brain MRI Findings: The ARIC Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP2152
AUTHORS	Power Melinda C, Lamichhane Archana P, Liao Duanping, Xu Xiaohui, Jack Clifford R, Gottesman Rebecca F, Mosley Thomas, Stewart James D, Yanosky Jeff D, Whitsel Eric A
BACKGROUND	Increasing evidence links higher particulate matter (PM) air pollution exposure to late-life cognitive impairment. However, few studies have considered associations between direct estimates of long-term past exposures and brain MRI findings indicative of neurodegeneration or cerebrovascular disease.
OBJECTIVE	Our objective was to quantify the association between brain MRI findings and PM exposures approximately 5 to 20 y prior to MRI in the Atherosclerosis Risk in Communities (ARIC) study.
METHODS	ARIC is based in four U.S. sites: Washington County, Maryland; Minneapolis suburbs, Minnesota; Forsyth County, North Carolina; and Jackson, Mississippi. A subset of ARIC participants underwent 3T brain MRI in 2011-2013 (n=1,753). We estimated mean exposures to PM with an aerodynamic diameter less than 10 or 2.5 μm (PM10 and PM2.5) in 1990-1998, 1999-2007, and 1990-2007 at the residential addresses of eligible participants with MRI data. We estimated site-specific associations between PM and brain MRI findings and used random-effect, inverse variance-weighted meta-analysis to combine them.
RESULTS	In pooled analyses, higher mean PM2.5 and PM10 exposure in all time periods were associated with smaller deep-gray brain volumes, but not other MRI markers. Higher PM2.5 exposures were consistently associated with smaller total and regional brain volumes in Minnesota, but not elsewhere.
CONCLUSIONS	Long-term past PM exposure in was not associated with markers of cerebrovascular disease. Higher long-term past PM exposures were associated with smaller deep-gray volumes overall, and higher PM2.5 exposures were associated with smaller brain volumes in the Minnesota site. Further work is needed to understand the sources of heterogeneity across sites. https://doi.org/10.1289/EHP2152.

표-581. PubMed 논문번호 29477875의 내용 요약

구분	내용
PubMed ID	29477875
TITLE	Mortality among rescue and recovery workers and community members exposed to the September 11, 2001 World Trade Center terrorist attacks, 2003-2014.
JOURNAL	Environmental research: 10.1016/j.envres.2018.01.004
AUTHORS	Jordan Hannah T, Stein Cheryl R, Li Jiehui, Cone James E, Stayner Leslie, Hadler James L, Brackbill Robert M, Farfel Mark R
BACKGROUND	Multiple chronic health conditions have been associated with exposure to the September 11, 2001 World Trade Center (WTC) terrorist attacks (9/11). We assessed whether excess deaths occurred during 2003-2014 among persons directly exposed to 9/11, and examined associations of 9/11-related exposures with mortality risk.
MATERIALS AND METHODS	Deaths occurring in 2003-2014 among members of the World Trade Center Health Registry, a cohort of rescue/recovery workers and lower Manhattan community members who were exposed to 9/11, were identified via linkage to the National Death Index. Participants' overall levels of 9/11-related exposure were categorized as high, intermediate, or low. We calculated standardized mortality ratios (SMR) using New York City reference rates from 2003 to 2012. Proportional hazards were used to assess associations of 9/11-related exposures with mortality, accounting for age, sex, race/ethnicity and other potential confounders.
RESULTS	We identified 877 deaths among 29,280 rescue/recovery workers (3.0%) and 1694 deaths among 39,643 community members (4.3%) during 308,340 and 416,448 person-years of observation, respectively. The SMR for all causes of death was 0.69 [95% confidence interval (CI) 0.65-0.74] for rescue/recovery workers and 0.86 (95% CI 0.82-0.90) for community members. SMRs for diseases of the cardiovascular and respiratory systems were significantly lower than expected in both groups. SMRs for several other causes of death were significantly elevated, including suicide among rescue recovery workers (SMR 1.82, 95% CI 1.35-2.39), and brain malignancies (SMR 2.25, 95% CI 1.48-3.28) and non-Hodgkin's lymphoma (SMR 1.79, 95% CI 1.24-2.50) among community members. Compared to low exposure, both intermediate [adjusted hazard ratio (AHR) 1.36, 95% CI 1.10-1.67] and high (AHR 1.41, 95% CI 1.06-1.88) levels of 9/11-related exposure were significantly associated with all-cause mortality among rescue/recovery workers (p-value for trend 0.01). For community members, intermediate (AHR 1.13, 95% CI 1.01-1.27), but not high (AHR 1.14, 95% CI 0.94-1.39) exposure was significantly associated with all-cause mortality (p-value for trend 0.03). AHRs for associations of overall 9/11-related exposure with heart disease- and cancer-related mortality were similar in magnitude to those for all-cause mortality, but with 95% CIs crossing the null value.
CONCLUSIONS	Overall mortality was not elevated. Among specific causes of death that were significantly elevated, suicide among rescue/recovery workers is a plausible long-term consequence of 9/11 exposure, and is potentially preventable. Elevated mortality due to other causes, including non-Hodgkin's lymphoma and brain cancer, and small but statistically significant associations of 9/11-related exposures with all-cause mortality hazard warrant additional surveillance.

표-582. PubMed 논문번호 29523649의 내용 요약

구분	내용
PubMed ID	29523649
TITLE	Projected Temperature-Related Years of Life Lost From Stroke Due To Global Warming in a Temperate Climate City, Asia: Disease Burden Caused by Future Climate Change.
JOURNAL	Stroke: 10.1161/STROKEAHA.117.020042
AUTHORS	Li Guoxing, Guo Qun, Liu Yang, Li Yixue, Pan Xiaochuan
BACKGROUND AND PURPOSE	Global warming has attracted worldwide attention. Numerous studies have indicated that stroke is associated with temperature; however, few studies are available on the projections of the burden of stroke attributable to future climate change. We aimed to investigate the future trends of stroke years of life lost (YLL) associated with global warming.
METHODS	We collected death records to examine YLL in Tianjin, China, from 2006 to 2011. We fitted a standard time-series Poisson regression model after controlling for trends, day of the week, relative humidity, and air pollution. We estimated temperature-YLL associations with a distributed lag nonlinear model. These models were then applied to the local climate projections to estimate temperature-related YLL in the 2050s and 2070s. We projected temperature-related YLL from stroke in Tianjin under 19 global-scale climate models and 3 different greenhouse gas emission scenarios.
RESULTS	The results showed a slight decrease in YLL with percent decreases of 0.85%, 0.97%, and 1.02% in the 2050s and 0.94%, 1.02%, and 0.91% in the 2070s for the 3 scenarios, respectively. The increases in heat-related annual YLL and the decreases in cold-related YLL under the high emission scenario were the strongest. The monthly analysis showed that the most significant increase occurred in the summer months, particularly in August, with percent changes >150% in the 2050s and up to 300% in the 2070s.
CONCLUSIONS	Future changes in climate are likely to lead to an increase in heat-related YLL, and this increase will not be offset by adaptation under both medium emission and high emission scenarios. Health protections from hot weather will become increasingly necessary, and measures to reduce cold effects will also remain important.

표-583. PubMed 논문번호 29540609의 내용 요약

구분	내용
PubMed ID	29540609
TITLE	Residential Proximity to Major Roadways and Risk of Incident Ischemic Stroke in NOMAS (The Northern Manhattan Study).
JOURNAL	Stroke: 10.1161/STROKEAHA.117.019580
AUTHORS	Kulick Erin R, Wellenius Gregory A, Boehme Amelia K, Sacco Ralph L, Elkind Mitchell S
BACKGROUND AND PURPOSE	The evidence supporting the deleterious cardiovascular health effects of living near a major roadway is growing, although this association is not universal. In primary analyses, we hypothesized that residential proximity to a major roadway would be associated with incident ischemic stroke and that cardiovascular risk factors would modify that association.
METHODS	NOMAS (The Northern Manhattan Study) is an ongoing, population-based cohort study designed to measure cardiovascular risk factors, stroke incidence, and other outcomes in a multiethnic urban population. Recruitment occurred from 1993 to 2001 and participants are followed-up annually by telephone. Residential addresses at baseline were geocoded and Euclidean distance to nearest major roadway was estimated and categorized as in prior studies. We used Cox proportional hazard models to calculate hazard ratios and 95% confidence intervals for the association of this distance to incidence of stroke and other outcomes, adjusting for sociodemographic and cardiovascular risk factors, year at baseline, and neighborhood socioeconomic status. We assessed whether these associations varied by age, sex, smoking status, diabetes mellitus, and hypertension.
RESULTS	During a median follow-up period of 15 years (n=3287), 11% of participants were diagnosed with ischemic stroke. Participants living <100 m from a roadway had a 42% (95% confidence interval, 1.01-2.02) higher rate of ischemic stroke versus those living >400 m away. This association was more pronounced among noncurrent smokers (hazard ratio, 1.54; 95% confidence interval, 1.05-2.26) and not evident among smokers (hazard ratio, 0.69; 95% confidence interval, 0.23-2.06). There was no clear pattern of association between proximity to major roadways and other cardiovascular events including myocardial infarction, all-cause death, or vascular death.
CONCLUSIONS	In this urban multiethnic cohort, we found evidence supporting that within-city variation in residential proximity to major roadway is associated with higher risk of ischemic stroke. An individual's smoking history modified this association, with the association remaining only among participants not currently smokers.

표-584. PubMed 논문번호 29545240의 내용 요약

구분	내용
PubMed ID	29545240
TITLE	Particulate Matter Air Pollution and Racial Differences in Cardiovascular Disease Risk.
JOURNAL	Arteriosclerosis, thrombosis, and vascular biology: 10.1161/ATVBAHA.117.310305
AUTHORS	Erqou Sebhat, Clougherty Jane E, Olafiranye Oladipupo, Magnani Jared W, Aiyer Aryan, Tripathy Sheila, Kinnee Ellen, Kip Kevin E, Reis Steven E
OBJECTIVE	We aimed to assess racial differences in air pollution exposures to ambient fine particulate matter (particles with median aerodynamic diameter 〈2.5 □m [PM2.5]) and black carbon (BC) and their association with cardiovascular disease (CVD) risk factors, arterial endothelial function, incident CVD events, and all-cause mortality.
APPROACH AND RESULTS	Data from the HeartSCORE study (Heart Strategies Concentrating on Risk Evaluation) were used to estimate 1-year average air pollution exposure to PM2.5 and BC using land use regression models. Correlates of PM2.5 and BC were assessed using linear regression models. Associations with clinical outcomes were determined using Cox proportional hazards models, adjusting for traditional CVD risk factors. Data were available on 1717 participants (66% women; 45% blacks; 59±8 years). Blacks had significantly higher exposure to PM2.5 (mean 16.1±0.75 versus 15.7±0.73□g/m3; P=0.001) and BC (1.19±0.11 versus 1.16±0.13abs; P=0.001) compared with whites. Exposure to PM2.5, but not BC, was independently associated with higher blood glucose and worse arterial endothelial function. PM2.5 was associated with a higher risk of incident CVD events and all-cause mortality combined for median follow-up of 8.3 years. Blacks had 1.45 (95% CI, 1.00-2.09) higher risk of combined CVD events and all-cause mortality than whites in models adjusted for relevant covariates. This association was modestly attenuated with adjustment for PM2.5.
CONCLUSIONS	PM2.5 exposure was associated with elevated blood glucose, worse endothelial function, and incident CVD events and all-cause mortality. Blacks had a higher rate of incident CVD events and all-cause mortality than whites that was only partly explained by higher exposure to PM2.5.

표-585. PubMed 논문번호 29545261의 내용 요약

구분	내용
PubMed ID	29545261
TITLE	Associations Between Ambient Particle Radioactivity and Blood Pressure: The NAS (Normative Aging Study).
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.117.008245
AUTHORS	Nyhan Marguerite M, Coull Brent A, Blomberg Annelise J, Vieira Carol L Z, Garshick Eric, Aba Abdulaziz, Vokonas Pantel, Gold Diane R, Schwartz Joel, Koutrakis Petros
BACKGROUND	The cardiovascular effects of low-level environmental radiation exposures are poorly understood. Although particulate matter (PM) has been linked to cardiovascular morbidity and mortality, and elevated blood pressure (BP), the properties promoting its toxicity remain uncertain. Addressing a knowledge gap, we evaluated whether BP increased with higher exposures to radioactive components of ambient PM, herein referred to as particle radioactivity (PR).
METHODS AND RESULTS	We performed a repeated-measures analysis of 852 men to examine associations between PR exposure and BP using mixed-effects regression models. As a surrogate for PR, we used gross β activity, measured by the US Environmental Protection Agency's radiation monitoring network. Higher PR exposure was associated with increases in both diastolic BP and systolic BP, for exposures from 1 to 28 days. An interquartile range increase in 28-day PR exposure was associated with a 2.95-mm Hg increase in diastolic BP (95% confidence interval, 2.25-3.66; P<0.001) and a 3.94-mm Hg increase in systolic BP (95% confidence interval, 2.62-5.27; P<0.001). For models including both PR and PM ≤2.5 □m, the PR-BP associations remained stable and significant. For models including PR and black carbon or PR and particle number, the PR-BP associations were attenuated; however, they remained significant for many exposure durations.
CONCLUSIONS	This is the first study to demonstrate the potential adverse effects of PR on both systolic and diastolic BPs. These were independent and similar in magnitude to those of PM ≤2.5 □m, black carbon, and particle number. Understanding the effects of particle-bound radionuclide exposures on BP may have important implications for environmental and public health policy.

표-586. PubMed 논문번호 29551089의 내용 요약

구분	내용
PubMed ID	29551089
TITLE	Secondhand smoke exposure and risk of incident peripheral arterial disease and mortality: a Scotland-wide retrospective cohort study of 4045 non-smokers with cotinine measurement.
JOURNAL	BMC public health: 10.1186/s12889-018-5227-x
AUTHORS	Lu Liya, Mackay Daniel F, Pell Jill P
BACKGROUND	Active smoking is an important risk factor for all-cause mortality and peripheral arterial disease (PAD). In contrast, published studies on the associations with secondhand smoke (SHS) are limited. The aim of this study was to examine the associations between SHS exposure and incident PAD, as well as mortality, among middle-aged non-smokers.
METHODS	We undertook a retrospective, cohort study using record linkage of the Scottish Health Surveys between 1998 and 2010 to hospital admissions and death certificates. Inclusion was restricted to participants aged > 45 years. Cox proportional hazard models were used to examine the association between SHS exposure and incident PAD (hospital admission or death) and all-cause mortality, with adjustment for potential confounders.
RESULTS	Of the 4045 confirmed non-smokers (self-reported non-smokers with salivary cotinine concentrations < 15 ng/mL), 1163 (28.8%) had either moderate or high exposure to SHS at baseline. In men, high exposure to SHS (cotinine ≥2.7 ng/mL) was associated with increased risk of all-cause mortality (fully adjusted hazard ratio [HR] 1.54, 95% CI 1.07-2.22, p = 0.020) with evidence of a dose-relationship (p for trend = 0.004). In men, high exposure to SHS was associated with increased risk of incident PAD over the first five years of follow-up (fully adjusted HR 4.29, 95% CI 1.14-16.10, p = 0.031) but the association became non-significant over longer term follow-up.
CONCLUSIONS	SHS exposure was independently associated with all-cause mortality and may be associated with PAD, but larger studies, or meta-analyses, are required to confirm the latter.

표-587. PubMed 논문번호 29558518의 내용 요약

구분	내용
PubMed ID	29558518
TITLE	Risk factors for type 2 diabetes mellitus: An exposure-wide umbrella review of meta-analyses.
JOURNAL	PloS one: 10.1371/journal.pone.0194127
AUTHORS	Bellou Vanesa, Belbasis Lazaros, Tzoulaki Ioanna, Evangelou Evangelos
BACKGROUND	Type 2 diabetes mellitus (T2DM) is a global epidemic associated with increased health expenditure, and low quality of life. Many non-genetic risk factors have been suggested, but their overall epidemiological credibility has not been assessed.
METHODS	We searched PubMed to capture all meta-analyses and Mendelian randomization studies for risk factors of T2DM. For each association, we estimated the summary effect size, its 95% confidence and prediction interval, and the I2 metric. We examined the presence of small-study effects and excess significance bias. We assessed the epidemiological credibility through a set of predefined criteria.
RESULTS	We captured 86 eligible papers (142 associations) covering a wide range of biomarkers, medical conditions, and dietary, lifestyle, environmental and psychosocial factors. Adiposity, low hip circumference, serum biomarkers (increased level of alanine aminotransferase, gamma-glutamyl transferase, uric acid and C-reactive protein, and decreased level of adiponectin and vitamin D), an unhealthy dietary pattern (increased consumption of processed meat and sugar-sweetened beverages, decreased intake of whole grains, coffee and heme iron, and low adherence to a healthy dietary pattern), low level of education and conscientiousness, decreased physical activity, high sedentary time and duration of television watching, low alcohol drinking, smoking, air pollution, and some medical conditions (high systolic blood pressure, late menarche age, gestational diabetes, metabolic syndrome, preterm birth) presented robust evidence for increased risk of T2DM.
CONCLUSIONS	A healthy lifestyle pattern could lead to decreased risk for T2DM. Future randomized clinical trials should focus on identifying efficient strategies to modify harmful daily habits and predisposing dietary patterns.

표-588. PubMed 논문번호 29567495의 내용 요약

구분	내용
PubMed ID	29567495
TITLE	Characterizing exposure to household air pollution within the Prospective Urban Rural Epidemiology (PURE) study.
JOURNAL	Environment international: 10.1016/j.envint.2018.02.033
AUTHORS	Arku Raphael E, Birch Aaron, Shupler Matthew, Yusuf Salim, Hystad Perry, Brauer Michael
BACKGROUND	Household air pollution (HAP) from combustion of solid fuels is an important contributor to disease burden in low- and middle-income countries (LIC, and MIC). However, current HAP disease burden estimates are based on integrated exposure response curves that are not currently informed by quantitative HAP studies in LIC and MIC. While there is adequate evidence supporting causal relationships between HAP and respiratory disease, large cohort studies specifically examining relationships between quantitative measures of HAP exposure with cardiovascular disease are lacking.
OBJECTIVE	We aim to improve upon exposure proxies based on fuel type, and to reduce exposure misclassification by quantitatively measuring exposure across varying cooking fuel types and conditions in diverse geographies and socioeconomic settings. We leverage technology advancements to estimate household and personal PM2.5 (particles below 2.5 μm in aerodynamic diameter) exposure within the large (N~250,000) multi-country (N~26) Prospective Urban and Rural Epidemiological (PURE) cohort study. Here, we detail the study protocol and the innovative methodologies being used to characterize HAP exposures, and their application in epidemiologic analyses.
METHODS/DESIGN	This study characterizes HAP PM2.5 exposures for participants in rural communities in ten PURE countries with >10% solid fuel use at baseline (Bangladesh, Brazil, Chile, China, Colombia, India, Pakistan, South Africa, Tanzania, and Zimbabwe). PM2.5 monitoring includes 48-h cooking area measurements in 4500 households and simultaneous personal monitoring of male and female pairs from 20% of the selected households. Repeat measurements occur in 20% of households to assess impacts of seasonality. Monitoring began in 2017, and will continue through 2019. The Ultrasonic Personal Aerosol Sampler (UPAS), a novel, robust, and inexpensive filter based monitor that is programmable through a dedicated mobile phone application is used for sampling. Pilot study field evaluation of cooking area measurements indicated high correlation between the UPAS and reference Harvard Impactors (r = 0.91; 95% CI: 0.84, 0.95; slope = 0.95). To facilitate tracking and to minimize contamination and analytical error, the samplers utilize barcoded filters and filter cartridges that are weighed pre- and post-sampling using a fully automated weighing system. Pump flow and pressure measurements, temperature and RH, GPS coordinates and semi-quantitative continuous particle mass concentrations based on filter differential pressure are uploaded to a central server automatically whenever the mobile phone is connected to the internet, with sampled data automatically screened for quality control parameters. A short survey is administered during the 48-h monitoring period. Post-weighed filters are further analyzed to estimate black carbon concentrations through a semi-automated, rapid, cost-effective image analysis approach. The measured PM2.5 data will then be combined with PURE survey information on household characteristics and behaviours collected at baseline and during follow-up to develop quantitative HAP models for PM2.5 exposures for all rural PURE participants (~50,000) and across different cooking fuel types within the 10 index countries. Both the measured (in the subset) and the modelled exposures will be used in separate

표-589. PubMed 논문번호 29569250의 내용 요약

구분	내용
PubMed ID	29569250
TITLE	Impact of upper respiratory tract infections on perioperative outcomes of children undergoing therapeutic cardiac catheterisation.
JOURNAL	Acta anaesthesiologica Scandinavica: 10.1111/aas.13113
AUTHORS	Zhang S, Ding S, Cai M, Bai J, Zhang M, Huang Y, Zheng J
BACKGROUND	Recent upper respiratory tract infection (URI) is associated with increased incidence of perioperative complications in children undergoing open heart surgery. As a result, surgery is often postponed. However, the effect of recent URI on the incidence of perioperative complications in children undergoing therapeutic cardiac catheterisation is unknown. We investigated the perioperative outcomes of congenital heart disease (CHD) children with recent URI who underwent elective therapeutic catheterisation.
METHODS	We prospectively included children treated for CHD. Before surgery, parents or legal guardians were interviewed to complete a questionnaire on the child's demographics, history of asthma and passive smoking, and URI symptoms. Recorded perioperative respiratory adverse events (PRAEs) included laryngospasm, bronchospasm, breath holding, oxygen desaturation, and severe cough. Information on postoperative dysphoria, fever, copious sputum, and vomiting was obtained by telephone 24 h after surgery.
RESULTS	Of 363 included children, 169 had recently (within 2 weeks) had a URI. The URI did not affect the incidence of laryngospasm, bronchospasm, breath holding, fever, or vomiting. The incidence of desaturation, severe cough, dysphoria, and copious sputum were significantly increased. Independent risk factors for PRAEs in children with a recent URI included age, passive smoking, and presence of rhinorrhoea or moist cough. The lengths of stay in the hospital and intensive care unit were not significantly different between groups.
CONCLUSION	Although recent URI increased the incidence of PRAEs in children undergoing therapeutic cardiac catheterisation, most CHD patients with recent URI can undergo elective therapeutic cardiac catheterisation without serious adverse events or prolonged hospitalisation.

표-590. PubMed 논문번호 29575857의 내용 요약

구분	내용
PubMed ID	29575857
TITLE	Ischemic cardiovascular disease in workers occupationally exposed to urban air pollution - A systematic review.
JOURNAL	Annals of agricultural and environmental medicine : AAEM: 10.26444/aaem/79922
AUTHORS	De Marchis Paola, Verso Maria Gabriella, Tramuto Fabio, Amodio Emanuele, Picciotto Diego, De Marchis Paola, Verso Maria Gabriella, Tramuto Fabio, Amodio Emanuele, Picciotto Diego
INTRODUCTION	Cardiovascular disease is the first cause of morbidity and mortality worldwide. Among several known risk factors, researchers also focus their attention on the chronic exposure to air pollution. There is much evidence that exposure to air pollution, especially to ultrafine particles, can damage the endothelium and can favour cardiovascular diseases in the general population. Occupational exposition could be an additive risk factor for the cardiovascular system. This article presents a scientific review of the linkage between occupational exposure to air pollution and ischemic heart disease.
MATERIAL AND METHODS	A scientific review was undertaken, followed by PRISMA Statements. Observational studies were selected from several scientific databases, likesuch as Pubmed, Google Scholar, Nioshtic-2 and Reserchgate, searching for selected key words: police workers, professional drivers, mail carriers, filling station attendants, road cleaners, garage workers, motor vehicles and engine maintenance. All the key words were combined with "Boolean Operators" with the following words: cardiovascular (or cardiac) disease, cardiovascular function, cardiovascular system, ischemic heart disease, coronary disease, myocardial infarction. During the systematic research, the focus was on retrospective and prospective studies from January 1990 - December 2014.
RESULTS	Both the retrospective and prospective studies showed an increased risk of ischemic heart disease in occupationally occupied people exposed to air pollution. Only one study presented a ly minor risk.
CONCLUSIONS	The findings of this systematic review suggest a possible linkage between occupational exposure to urban air pollution, especially to motor exhaust and particulate, and ischemic heart disease.

표-591. PubMed 논문번호 29614179의 내용 요약

구분	내용
PubMed ID	29614179
TITLE	Association of Solid Fuel Use With Risk of Cardiovascular and All-Cause Mortality in Rural China.
JOURNAL	JAMA: 10.1001/jama.2018.2151
AUTHORS	Yu Kuai, Qiu Gaokun, Chan Ka-Hung, Lam Kin-Bong Hubert, Kurmi Om P, Bennett Derrick A, Yu Canqing, Pan An, Lv Jun, Guo Yu, Bian Zheng, Yang Ling, Chen Yiping, Hu Frank B, Chen Zhengming, Li Liming, Wu Tangchun
Importance	When combusted indoors, solid fuels generate a large amount of pollutants such as fine particulate matter.
Objective	To assess the associations of solid fuel use for cooking and heating with cardiovascular and all-cause mortality.
Design, Setting, and Participants	This nationwide prospective cohort study recruited participants from 5 rural areas across China between June 2004 and July 2008; mortality follow-up was until January 1, 2014. A total of 271 217 adults without a self-reported history of physician-diagnosed cardiovascular disease at baseline were included, with a random subset (n = 10 892) participating in a resurvey after a mean interval of 2.7 years.
Exposures	Self-reported primary cooking and heating fuels (solid: coal, wood, or charcoal; clean: gas, electricity, or central heating), switching of fuel type before baseline, and use of ventilated cookstoves.
Main Outcomes and Measures	Death from cardiovascular and all causes, collected through established death registries.
Results	Among the 271 217 participants, the mean (SD) age was 51.0 (10.2) years, and 59% (n = 158 914) were women. A total of 66% (n = 179 952) of the participants reported regular cooking (at least weekly) and 60% (n = 163 882) reported winter heating, of whom 84% (n = 150 992) and 90% (n = 147 272) used solid fuels, respectively. There were 15 468 deaths, including 5519 from cardiovascular causes, documented during a mean (SD) of 7.2 (1.4) years of follow-up. Use of solid fuels for cooking was associated with greater risk of cardiovascular mortality (absolute rate difference [ARD] per 100 000 person-years, 135 [95% CI, 77-193]; hazard ratio [HR], 1.20 [95% CI, 1.02-1.41]) and all-cause mortality (ARD, 338 [95% CI, 249-427]; HR, 1.11 [95% CI, 1.03-1.20]). Use of solid fuels for heating was also associated with greater risk of cardiovascular mortality (ARD, 175 [95% CI, 118-231]; HR, 1.29 [95% CI, 1.06-1.55]) and all-cause mortality (ARD, 392 [95% CI, 297-487]; HR, 1.14 [95% CI, 1.03-1.26]). Compared with persistent solid fuel users, participants who reported having previously switched from solid to clean fuels for cooking had a lower risk of cardiovascular mortality (ARD, 138 [95% CI, 71-205]; HR, 0.83 [95% CI, 0.69-0.99]) and all-cause mortality (ARD, 407 [95% CI, 317-497]; HR, 0.87 [95% CI, 0.79-0.95]), while for heating, the ARDs were 193 (95% CI, 128-258) and 492 (95% CI, 383-601), and the HRs were 0.57 (95% CI, 0.42-0.77) and 0.67 (95% CI, 0.57-0.79), respectively. Among solid fuel users, use of ventilated cookstoves was also associated with lower risk of cardiovascular mortality (ARD, 33 [95% CI, -9 to 75]; HR, 0.89 [95% CI, 0.80-0.99]) and all-cause mortality (ARD, 87 [95% CI, 20-153]; HR, 0.91 [95% CI, 0.85-0.96]).
Conclusions and Relevance	In rural China, solid fuel use for cooking and heating was associated with higher risks of cardiovascular and all-cause mortality. These risks may be

표-592. PubMed 논문번호 29615204의 내용 요약

구분	내용
PubMed ID	29615204
TITLE	Effect of air quality alerts on human health: a regression discontinuity analysis in Toronto, Canada.
JOURNAL	The Lancet. Planetary health: 10.1016/S2542-5196(17)30185-7
AUTHORS	Chen Hong, Li Qiongsi, Kaufman Jay S, Wang Jun, Copes Ray, Su Yushan, Benmarhnia Tarik
BACKGROUND	Ambient air pollution is a major health risk globally. To reduce adverse health effects on days when air pollution is high, government agencies worldwide have implemented air quality alert programmes. Despite their widespread use, little is known about whether these programmes produce any observable public-health benefits. We assessed the effectiveness of such programmes using a quasi-experimental approach.
METHODS	We assembled a population-based cohort comprising all individuals who resided in the city of Toronto (Ontario, Canada) from 2003 to 2012 (about 2·6 million people). We ascertained seven health outcomes known to be affected by short-term elevation of air pollution, using provincial health administrative databases. These health outcomes were cardiovascular-related mortality, respiratory-related mortality, and hospital admissions or emergency-department visits for acute myocardial infarction, heart failure, stroke, asthma, and chronic obstructive pulmonary disease (COPD). We applied a regression discontinuity design to assess the effectiveness of an intervention (ie, the air quality alert programme). To quantify the effect of the air quality alert programme, we estimated for each outcome both the absolute rate difference and the rate ratio attributable to programme eligibility (by intention-to-treat analysis) and the alerts themselves (by two-stage regression approach), respectively.
FINDINGS	Between Jan 1, 2003, and Dec 31, 2012, on average between three and 27 daily cardiovascular or respiratory events were reported in Toronto (depending on the outcome). Alert announcements reduced asthma-related emergency-department visits by 4·73 cases per 1 000 000 people per day (95% CI 0·55-9·38), or in relative terms by 25% (95% CI 1-47). Programme eligibility also led to 2·05 (95% CI 0·07-4·00) fewer daily emergency-department visits for asthma. We did not detect a significant reduction in any other health outcome as a result of alert announcements or programme eligibility. However, a non-significant trend was noted towards decreased asthma-related and COPD-related admissions.
INTERPRETATION	In this population-based cohort, the air quality alert programme was related to some reductions in respiratory morbidity, but not any other health outcome examined. This finding suggests that issuing air quality alerts alone has a limited effect on public health and that implementing enforced public actions to reduce air pollution on high pollution days could be warranted. Together with accumulating evidence of substantial burden from long-term air pollution exposure, this study underscores the need for further strengthening of global efforts that can lead to long-term improvement of overall air quality.
FUNDING	Public Health Ontario, Canadian Institutes for Health Research.

표-593. PubMed 논문번호 29622024의 내용 요약

구분	내용
PubMed ID	29622024
TITLE	Longitudinal associations of long-term exposure to ultrafine particles with blood pressure and systemic inflammation in Puerto Rican adults.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-018-0379-9
AUTHORS	Corlin Laura, Woodin Mark, Hart Jaime E, Simon Matthew C, Gute David M, Stowell Joanna, Tucker Katherine L, Durant John L, Brugge Doug
BACKGROUND	Few longitudinal studies have examined the association between ultrafine particulate matter (UFP, particles < 0.1 μm aerodynamic diameter) exposure and cardiovascular disease (CVD) risk factors. We used data from 791 adults participating in the longitudinal Boston Puerto Rican Health Study (Massachusetts, USA) between 2004 and 2015 to assess whether UFP exposure was associated with blood pressure and high sensitivity C-reactive protein (hsCRP, a biomarker of systemic inflammation).
METHODS	Residential annual average UFP exposure (measured as particle number concentration, PNC) was assigned using a model accounting for spatial and temporal trends. We also adjusted PNC values for participants' inhalation rate to obtain the particle inhalation rate (PIR) as a secondary exposure measure. Multilevel linear models with a random intercept for each participant were used to examine the association of UFP with blood pressure and hsCRP.
RESULTS	Overall, in adjusted models, an inter-quartile range increase in PNC was associated with increased hsCRP (β = 6.8; 95% CI = - 0.3, 14.0%) but not with increased systolic blood pressure (β = 0.96; 95% CI = - 0.33, 2.25 mmHg), pulse pressure (β = 0.70; 95% CI = - 0.27, 1.67 mmHg), or diastolic blood pressure (β = 0.55; 95% CI = - 0.20, 1.30 mmHg). There were generally stronger positive associations among women and never smokers. Among men, there were inverse associations of PNC with systolic blood pressure and pulse pressure. In contrast to the primary findings, an inter-quartile range increase in the PIR was positively associated with systolic blood pressure (β = 1.03; 95% CI = 0.00, 2.06 mmHg) and diastolic blood pressure (β = 1.01; 95% CI = 0.36, 1.66 mmHg), but not with pulse pressure or hsCRP.
CONCLUSIONS	We observed that exposure to PNC was associated with increases in measures of CVD risk markers, especially among certain sub-populations. The exploratory PIR exposure metric should be further developed.

표-594. PubMed 논문번호 29643111의 내용 요약

구분	내용
PubMed ID	29643111
TITLE	Cardiovascular and Cerebrovascular Emergency Department Visits Associated With Wildfire Smoke Exposure in California in 2015.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.117.007492
AUTHORS	Wettstein Zachary S, Hoshiko Sumi, Fahimi Jahan, Harrison Robert J, Cascio Wayne E, Rappold Ana G
BACKGROUND	Wildfire smoke is known to exacerbate respiratory conditions; however, evidence for cardiovascular and cerebrovascular events has been inconsistent, despite biological plausibility.
METHODS AND RESULTS	A population-based epidemiologic analysis was conducted for daily cardiovascular and cerebrovascular emergency department (ED) visits and wildfire smoke exposure in 2015 among adults in 8 California air basins. A quasi-Poisson regression model was used for zip code-level counts of ED visits, adjusting for heat index, day of week, seasonality, and population. Satellite-imaged smoke plumes were classified as light, medium, or dense based on model-estimated concentrations of fine particulate matter. Relative risk was determined for smoky days for lag days 0 to 4. Rates of ED visits by age- and sex-stratified groups were also examined. Rates of all-cause cardiovascular ED visits were elevated across all lags, with the greatest increase on dense smoke days and among those aged ≥65 years at lag 0 (relative risk 1.15, 95% confidence interval [1.09, 1.22]). All-cause cerebrovascular visits were associated with smoke, especially among those 65 years and older, (1.22 [1.00, 1.49], dense smoke, lag 1). Respiratory conditions were also increased, as anticipated (1.18 [1.08, 1.28], adults >65 years, dense smoke, lag 1). No association was found for the control condition, acute appendicitis. Elevated risks for individual diagnoses included myocardial infarction, ischemic heart disease, heart failure, dysrhythmia, pulmonary embolism, ischemic stroke, and transient ischemic attack.
CONCLUSIONS	Analysis of an extensive wildfire season found smoke exposure to be associated with cardiovascular and cerebrovascular ED visits for all adults, particularly for those over aged 65 years.

표-595. PubMed 논문번호 29669550의 내용 요약

구분	내용
PubMed ID	29669550
TITLE	Cardiorespiratory health effects of gaseous ambient air pollution exposure in low and middle income countries: a systematic review and meta-analysis.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-018-0380-3
AUTHORS	Newell Katherine, Kartsonaki Christiana, Lam Kin Bong Hubert, Kurmi Om
BACKGROUND	Lack of research on the effects of gaseous pollutants (nitrogen oxides [NOx], sulfur dioxide [SO2], carbon monoxide [CO] and ozone [O3]) in the ambient environment on health outcomes from within low and middle income countries (LMICs) is leading to reliance on results from studies performed within high income countries (HICs). This systematic review and meta-analysis examines the cardiorespiratory health effects of gaseous pollutants in LMICs exclusively.
METHODS	Systematic searching was carried out and estimates pooled by pollutant, lag and outcome, and presented as excess relative risk per 10 μg/m3 (NOx, SO2, O3) or 1 ppm (CO) increase pollutant. Sub-group analysis was performed examining estimates by specific outcomes, city and co-pollutant adjustment.
RESULTS	Sixty studies met the inclusion criteria, most (44) from the East Asia and Pacific region. A 10 μg/m3 increase in same day NOx was associated with 0.92% (95% CI: 0.44, 1.39), and 0.70% (0.01, 1.40) increases in cardiovascular and respiratory mortality respectively, same day NOx was not associated with morbidity. Same day sulfur dioxide was associated with 0.73% (0.04, 1.42) and 0.50% (0.01, 1.00) increases in respiratory morbidity and in cardiovascular mortality respectively.
CONCLUSIONS	Acute exposure to gaseous ambient air pollution (AAP) is associated with increases in morbidity and mortality in LMICs, with greatest associations observed for cardiorespiratory mortality.

표-596. PubMed 논문번호 29673904의 내용 요약

구분	내용
PubMed ID	29673904
TITLE	Impact of 2 Successive Smoking Bans on Hospital Admissions for Cardiovascular Diseases in Spain.
JOURNAL	Revista espanola de cardiologia (English ed.): 10.1016/j.rec.2017.10.055
AUTHORS	Gal□n I□aki, Sim□n Lorena, Boldo Elena, Ortiz Cristina, Medrano Mar□a Jos□, Fern□ndez-Cuenca Rafael, Linares Cristina, Pastor-Barriuso Roberto
INTRODUCTION AND OBJECTIVES	To evaluate the impact of 2 smoking bans enacted in 2006 (partial ban) and 2011 (comprehensive ban) on hospitalizations for cardiovascular disease in the Spanish adult population.
METHODS	The study was performed in 14 provinces in Spain. Hospital admission records were collected for acute myocardial infarction (AMI), ischemic heart disease (IHD), and cerebrovascular disease (CVD) in patients aged ≥ 18 years from 2003 through 2012. We estimated immediate and 1-year effects with segmented-linear models. The coefficients for each province were combined using random-effects multivariate meta-analysis models.
RESULTS	Overall, changes in admission rates immediately following the implementation of the partial ban and 1 year later were -1.8% and +1.2% for AMI, +0.1 and +0.4% for IHD, and +1.0% and +2.8% for CVD (P>.05). After the comprehensive ban, immediate changes were -2.3% for AMI, -2.6% for IHD, and -0.8% for CVD (P>.05), only to return to precomprehensive ban values 1 year later. For patients aged ≥ 65 years of age, immediate changes associated with the comprehensive ban were -5.0%, -3.9%, and -2.3% for AMI, IHD, and CVD, respectively (P<.05). Again, the 1-year changes were not statistically significant.
CONCLUSIONS	In Spain, smoking bans failed to significantly reduce hospitalizations for AMI, IHD, or CVD among patients ≥ 18 years of age. In the population aged ≥ 65 years, hospital admissions due to these diseases showed significant decreases immediately after the implementation of the comprehensive ban, but these reductions disappeared at the 1-year evaluation.

표-597. PubMed 논문번호 29679333의 내용 요약

구분	내용
PubMed ID	29679333
TITLE	A Mixed-Methods Study to Examine the Role of Psychosocial Stress and Air Pollution on Hypertension in Mexican-Origin Hispanics.
JOURNAL	Journal of racial and ethnic health disparities: 10.1007/s40615-018-0490-1
AUTHORS	Rammah Amal, Walker Whitworth Kristina, Han Inkyu, Chan Wenyaw, Jimenez Maria D, Strom Sara S, Bondy Melissa L, Symanski Elaine, Rammah Amal, Walker Whitworth Kristina, Han Inkyu, Chan Wenyaw, Jimenez Maria D, Strom Sara S, Bondy Melissa L, Symanski Elaine
PURPOSE	Independent and combined effects of air pollution and psychosocial stressors on hypertension, a risk factor for cardiovascular disease, among Hispanics are not well studied.
METHODS	We administered a pilot-tested questionnaire on individual- and neighborhood-level psychosocial stressors, developed with community input, to nearly 2500 individuals from the MD Anderson Cancer Center cohort of Mexican-Americans. We used data from local air quality monitors to estimate individual exposures to ozone (O3) and fine particulate matter (PM2.5) for the 12-month period preceding enrollment using inverse distance interpolation. We applied logistic regression models to examine relationships between exposures to psychosocial stressors and air pollution with prevalent hypertension and used stratified analyses to examine the interacting effects of these two exposures on hypertension. RESULTS: There was a positive association between prevalent hypertension and a high frequency of feeling anxious or depressed (prevalence odds ratio (POR) = 1.36, 95% CI [1.06-1.75]) and experiencing aches and pains (POR = 1.29, 95% CI [1.01-1.64]). The odds of having hypertension were also elevated among those worrying about their own health (POR = 1.65, 95% CI [1.30-2.06]) or about not having enough money (POR = 1.27, 95% CI [1.01-1.6]). We observed an inverse association between O3 and hypertension. There was no interaction between psychosocial stressors and O3 on hypertension.
CONCLUSION	Our findings add to the evidence of a positive association between individual and family stressors on hypertension among Hispanics and other racial/ethnic groups. Contrary to previous studies reporting positive associations, our results suggest that long-term exposure to O3 may be inversely related to prevalent hypertension.

표-598. PubMed 논문번호 29689465의 내용 요약

구분	내용
PubMed ID	29689465
TITLE	Two-way effect modifications of air pollution and air temperature on total natural and cardiovascular mortality in eight European urban areas.
JOURNAL	Environment international: 10.1016/j.envint.2018.04.021
AUTHORS	Chen Kai, Wolf Kathrin, Breitner Susanne, Gasparrini Antonio, Stafoggia Massimo, Samoli Evangelia, Andersen Zorana Jovanovic, Bero-Bedada Getahun, Bellander Tom, Hennig Frauke, Jacquemin B□n□dicte, Pekkanen Juha, Hampel Regina, Cyrys Josef, Peters Annette, Schneider Alexandra
BACKGROUND	Although epidemiological studies have reported associations between mortality and both ambient air pollution and air temperature, it remains uncertain whether the mortality effects of air pollution are modified by temperature and vice versa. Moreover, little is known on the interactions between ultrafine particles (diameter ≤ 100 nm, UFP) and temperature.
OBJECTIVE	We investigated whether the short-term associations of particle number concentration (PNC in the ultrafine range (≤ 100 nm) or total PNC ≤ 3000 nm, as a proxy for UFP), particulate matter ≤ 2.5 μm (PM2.5) and ≤ 10 μm (PM10), and ozone with daily total natural and cardiovascular mortality were modified by air temperature and whether air pollution levels affected the temperature-mortality associations in eight European urban areas during 1999-2013.
METHODS	We first analyzed air temperature-stratified associations between air pollution and total natural (nonaccidental) and cardiovascular mortality as well as air pollution-stratified temperature-mortality associations using city-specific over-dispersed Poisson additive models with a distributed lag nonlinear temperature term in each city. All models were adjusted for long-term and seasonal trend, day of the week, influenza epidemics, and population dynamics due to summer vacation and holidays. City-specific effect estimates were then pooled using random-effects meta-analysis.
RESULTS	Pooled associations between air pollutants and total and cardiovascular mortality were overall positive and generally stronger at high relatively compared to low air temperatures. For example, on days with high air temperatures (〉75th percentile), an increase of 10,000 particles/cm3 in PNC corresponded to a 2.51% (95% CI: 0.39%, 4.67%) increase in cardiovascular mortality, which was significantly higher than that on days with low air temperatures (〈25th percentile) [-0.18% (95% CI: -0.97%, 0.62%)]. On days with high air pollution (〉50th percentile), both heat- and cold-related mortality risks increased.
CONCLUSION	Our findings showed that high temperature could modify the effects of air pollution on daily mortality and high air pollution might enhance the air temperature effects.

표-599. PubMed 논문번호 29698900의 내용 요약

구분	내용
PubMed ID	29698900
TITLE	Outdoor air pollution and mosaic loss of chromosome Y in older men from the Cardiovascular Health Study.
JOURNAL	Environment international: 10.1016/j.envint.2018.04.030
AUTHORS	Wong Jason Y Y, Margolis Helene G, Machiela Mitchell, Zhou Weiyin, Odden Michelle C, Psaty Bruce M, Robbins John, Jones Rena R, Rotter Jerome I, Chanock Stephen J, Rothman Nathaniel, Lan Qing, Lee Jennifer S
BACKGROUND	Mosaic loss of chromosome Y (mLOY) can occur in a fraction of cells as men age, which is potentially linked to increased mortality risk. Smoking is related to mLOY; however, the contribution of air pollution is unclear.
OBJECTIVE	We investigated whether exposure to outdoor air pollution, age, and smoking were associated with mLOY.
METHODS	We analyzed baseline (1989-1993) blood samples from 933 men ≥65 years of age from the prospective Cardiovascular Health Study. Particulate matter ≤10 μm (PM10), carbon monoxide, nitrogen dioxide, sulfur dioxide, and ozone data were obtained from the U.S. EPA Aerometric Information Retrieval System for the year prior to baseline. Inverse-distance weighted air monitor data were used to estimate each participants' monthly residential exposure. mLOY was detected with standard methods using signal intensity (median log-R ratio (mLRR)) of the male-specific chromosome Y regions from Illumina array data. Linear regression models were used to evaluate relations between mean exposure in the prior year, age, smoking and continuous mLRR.
RESULTS	Increased PM10 was associated with mLOY, namely decreased mLRR (p-trend = 0.03). Compared with the lowest tertile (≤28.5 μg/m3), the middle (28.5-31.0 μg/m3; β = -0.0044, p = 0.09) and highest (≥31 μg/m3; β = -0.0054, p = 0.04) tertiles had decreased mLRR, adjusted for age, clinic, race/cohort, smoking status and pack-years. Additionally, increasing age (β = -0.00035, p = 0.06) and smoking pack-years (β = -0.00011, p = 1.4E-3) were associated with decreased mLRR, adjusted for each other and race/cohort. No significant associations were found for other pollutants.
CONCLUSIONS	PM10 may increase leukocyte mLOY, a marker of genomic instability. The sample size was modest and replication is warranted.

표-600. PubMed 논문번호 29704672의 내용 요약

구분	내용
PubMed ID	29704672
TITLE	Environmental noise pollution and risk of preeclampsia.
JOURNAL	Environmental pollution (Barking, Essex : 1987): 10.1016/j.envpol.2018.04.060
AUTHORS	Auger Nathalie, Duplaix Mathilde, Bilodeau-Bertrand Marianne, Lo Ernest, Smargiassi Audrey
BACKGROUND	Environmental noise exposure is associated with a greater risk of hypertension, but the link with preeclampsia, a hypertensive disorder of pregnancy, is unclear.
OBJECTIVES	We sought to determine the relationship between environmental noise pollution and risk of preeclampsia during pregnancy.
METHODS	We analyzed a population-based cohort comprising 269,263 deliveries on the island of Montreal, Canada between 2000 and 2013. We obtained total environmental noise pollution measurements (LAeq24, Lden, Lnight) from land use regression models, and assigned noise levels to each woman based on the residential postal code. We computed odds ratios (OR) and 95% confidence intervals (CI) for the association of noise with preeclampsia in mixed logistic regression models with participants as a random effect, and adjusted for air pollution, neighbourhood walkability, maternal age, parity, multiple pregnancy, comorbidity, socioeconomic deprivation, and year of delivery. We assessed whether noise exposure was more strongly associated with severe or early onset preeclampsia than mild or late onset preeclampsia.
RESULTS	Prevalence of preeclampsia was higher for women exposed to elevated environmental noise pollution levels (LAeq24h ≥ 65 dB(A) = 37.9 per 1000 vs. <50 dB(A) = 27.9 per 1000). Compared with 50 dB(A), an LAeq24h of 65.0 dB(A) was not significantly associated the risk of preeclampsia (OR 1.09, 95% CI 0.99-1.20). Associations were however present with severe (OR 1.29, 95% CI 1.09-1.54) and early onset (OR 1.71, 95% CI 1.20-2.43) preeclampsia, with results consistent across all noise indicators. The associations were much weaker or absent for mild and late preeclampsia.
CONCLUSIONS	Environmental noise pollution may be a novel risk factor for pregnancy-related hypertension, particularly more severe variants of preeclampsia.

표-601. PubMed 논문번호 29730535의 내용 요약

구분	내용
PubMed ID	29730535
TITLE	Long term exposure to air pollution and mortality in an elderly cohort in Hong Kong.
JOURNAL	Environment international: 10.1016/j.envint.2018.04.034
AUTHORS	Yang Yang, Tang Robert, Qiu Hong, Lai Poh-Chin, Wong Paulina, Thach Thuan-Quoc, Allen Ryan, Brauer Michael, Tian Linwei, Barratt Benjamin
BACKGROUND	Several studies have reported associations between long term exposure to air pollutants and cause-specific mortality. However, since the concentrations of air pollutants in Asia are much higher compared to those reported in North American and European cohort studies, cohort studies on long term effects of air pollutants in Asia are needed for disease burden assessment and to inform policy.
OBJECTIVES	To assess the effects of long-term exposure to particulate matter with aerodynamic diameter <2.5 μm (PM2.5), black carbon (BC) and nitrogen dioxide (NO2) on cause-specific mortality in an elderly cohort in Hong Kong.
METHODS	In a cohort of 66,820 participants who were older than or equal to 65 years old in Hong Kong from 1998 to 2011, air pollutant concentrations were estimated by land use regression and assigned to the residential addresses of all participants at baseline and for each year during a 11 year follow up period. Hazard ratios (HRs) of cause-specific mortality (including all natural cause, cardiovascular and respiratory mortality) associated with air pollutants were estimated with Cox models, including a number of personal and area-level socioeconomic, demographic, and lifestyle factors.
RESULTS	The median concentration of PM2.5 during the baseline period was 42.2 μg/m3 with an IQR of 5.5 μg/m3, 12.1 (9.6) μg/m3 for BC and 104 (25.6) μg/m3 for NO2. For PM2.5, adjusted HR per IQR increase and per 10 μg/m3 for natural cause mortality was 1.03 (95%CI: 1.01, 1.06) and 1.06 (95%CI: 1.02, 1.11) respectively. The corresponding HR were 1.06 (95%CI: 1.02, 1.10) and 1.01 (95%CI: 0.96, 1.06) for cardiovascular disease and respiratory disease mortality, respectively. For BC, the HR of an interquartile range increase for all natural cause mortality was 1.03 (95%CI: 1.00, 1.05). The corresponding HR was 1.07 (95%CI: 1.03, 1.11) and 0.99 (95%CI: 0.94, 1.04) for cardiovascular disease and respiratory disease mortality. For NO2, almost all HRs were approximately 1.0, except for IHD (ischemic heart disease) mortality.
CONCLUSION	Long-term exposure to ambient PM2.5 and BC was associated with an elevated risk of cardiovascular mortality. Despite far higher air pollution exposure concentrations, HRs per unit increase in PM2.5 were similar to those from recent comparable studies in North America.

표-602. PubMed 논문번호 29743082의 내용 요약

구분	내용
PubMed ID	29743082
TITLE	Combined effect of silica dust exposure and cigarette smoking on total and cause-specific mortality in iron miners: a cohort study.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-018-0391-0
AUTHORS	Lai Hanpeng, Liu Yuewei, Zhou Min, Shi Tingming, Zhou Yun, Weng Shaofan, Chen Weihong
BACKGROUND	Both cigarette smoking and long-term exposure to crystalline silica dust were reported to be associated with increased mortality. However, the combined effect of both factors has not been well evaluated.
METHODS	We investigated a retro-prospective cohort of 7,665 workers from one Chinese iron mine with a median follow-up of 42.8 years. Cumulative silica exposure was estimated for each worker by linking work histories with a job-exposure matrix. Cigarette smoking information was collected through face-to-face questionnaires. Hazard ratios (HRs) for total and cause-specific mortality due to silica exposure and smoking were estimated using Cox proportional hazards models.
RESULTS	A total of 2,814 deaths occurred during 315,772.9 person-years of follow-up. Significantly elevated mortality from all causes, cardiovascular disease, non-malignant respiratory disease and lung cancer was observed among silica-exposed workers, while elevated mortality from non-malignant respiratory disease and lung cancer was observed among smokers. Combined exposure to silica dust and cigarette smoking elevated the proportion of mortality and accounted for 21.2, 76.0, 35.7 and 81.4% of all causes, non-malignant respiratory disease, cardiovascular disease, and lung cancer, respectively. Significant additive joint effects of silica exposure and cigarette smoking on mortality from lung cancer (HR 1.893, 95% CI 0.628 to 3.441) and pneumoconiosis (6.457, 0.725 to 39.114), together with a significant multiplicative joint effect from all causes (1.002, 1.000 to 1.004) were observed.
CONCLUSIONS	The present findings indicated that silica exposure in combination with cigarette smoking accounted for a fraction of extra deaths in our cohort. Our research showed the urgent need for smoking cessation and silica control among iron miners.

표-603. PubMed 논문번호 29746370의 내용 요약

구분	내용
PubMed ID	29746370
TITLE	Long-term Concentrations of Nitrogen Dioxide and Mortality: A Meta-analysis of Cohort Studies.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000847
AUTHORS	Atkinson Richard W, Butland Barbara K, Anderson H Ross, Maynard Robert L
BACKGROUND	Concentrations of outdoor nitrogen dioxide (NO2) have been associated with increased mortality. Hazard ratios (HRs) from cohort studies are used to assess population health impact and burden. We undertook meta-analyses to derive concentration-response functions suitable for such evaluations and assessed their sensitivity to study selection based upon cohort characteristics.
METHODS	We searched online databases and existing reviews for cohort studies published to October 2016 that reported HRs for NO2 and mortality. We calculated meta-analytic summary estimates using fixed/random-effects models.
RESULTS	We identified 48 articles analyzing 28 cohorts. Meta-analysis of HRs found positive associations between NO2 and all cause (1.02 [95% confidence interval (CI): 1.01, 1.03]; prediction interval [PI]: [0.99, 1.06] per 10 □g/m increment in NO2), cardiovascular (1.03 [95% CI: 1.02, 1.05]; PI: [0.98, 1.08]), respiratory (1.03 [95% CI: 1.01, 1.05]; PI: [0.97, 1.10]), and lung cancer mortality (1.05 [95% CI: 1.02, 1.08]; PI: [0.94, 1.17]) with evidence of substantial heterogeneity between studies. In subgroup analysis, summary HRs varied by age at cohort entry, spatial resolution of pollution estimates, and adjustment for smoking and body mass index at the individual level; for some subgroups, the HR was close to unity, with lower confidence limits below 1.
CONCLUSIONS	Given the many uncertainties inherent in the assessment of this evidence base and the sensitivity of health impact calculations to small changes in the magnitude of the HRs, calculation of the impact on health of policies to reduce long-term exposure to NO2 should use prediction intervals and report ranges of impact rather than focusing upon point estimates.

표-604. PubMed 논문번호 29748348의 내용 요약

구분	내용
PubMed ID	29748348
TITLE	Association of ambient particulate matter with heart failure incidence and all-cause readmissions in Tasmania: an observational study.
JOURNAL	BMJ open: 10.1136/bmjopen-2018-021798
AUTHORS	Huynh Quan L, Blizzard Christopher Leigh, Marwick Thomas H, Negishi Kazuaki
OBJECTIVES	We sought to investigate the relationship between air quality and heart failure (HF) incidence and rehospitalisation to elucidate whether there is a threshold in this relationship and whether this relationship differs for HF incidence and rehospitalisation.
METHODS	This retrospective observational study was performed in an Australian state-wide setting, where air pollution is mainly associated with wood-burning for winter heating. Data included all 1246 patients with a first-ever HF hospitalisation and their 3011 subsequent all-cause readmissions during 2009-2012. Daily particulate matter <2.5 □m (PM2.5), temperature, relative humidity and influenza infection were recorded. Poisson regression was used, with adjustment for time trend, public and school holiday and day of week.
RESULTS	Tasmania has excellent air quality (median PM2.5=2.9 □g/m3 (IQR: 1.8-6.0)). Greater HF incidences and readmissions occurred in winter than in other seasons (p<0.001). PM2.5 was detrimentally associated with HF incidence (risk ratio (RR)=1.29 (1.15-1.42)) and weakly so with readmission (RR=1.07 (1.02-1.17)), with 1 day time lag. In multivariable analyses, PM2.5 significantly predicted HF incidence (RR=1.12 (1.01-1.24)) but not readmission (RR=0.96 (0.89-1.04)). HF incidence was similarly low when PM <4 □g/m3 and only started to rise when PM2.5≥4 □g/m3. Stratified analyses showed that PM2.5 was associated with readmissions among patients not taking beta-blockers but not among those taking beta-blockers (pinteraction=0.011).
CONCLUSIONS	PM2.5 predicted HF incidence, independent of other environmental factors. A possible threshold of PM2.5=4 □g/m3 is far below the daily Australian national standard of 25 □g/m3. Our data suggest that beta-blockers might play a role in preventing adverse association between air pollution and patients with HF.

표-605. PubMed 논문번호 29753251의 내용 요약

구분	내용
PubMed ID	29753251
TITLE	Long-term exposure to ambient particulate matter (PM_{2.5}) is associated with platelet counts in adults.
JOURNAL	Environmental pollution (Barking, Essex : 1987): 10.1016/j.envpol.2018.04.123
AUTHORS	Zhang Zilong, Chan Ta-Chien, Guo Cui, Chang Ly-Yun, Lin Changqing, Chuang Yuan Chieh, Jiang Wun Kai, Ho Kin Fai, Tam Tony, Woo Kam S, Lau Alexis K H, Lao Xiang Qian
BACKGROUND	The prothrombotic effects of particulate matter (PM) may underlie the association of air pollution with increased risks of cardiovascular disease. This study aimed to investigate the association between long-term exposure to PM with an aerodynamic diameter ≤2.5 μm (PM2.5) and platelet counts, a marker of coagulation profiles.
METHODS	The study participants were from a cohort consisting of 362,396 Taiwanese adults who participated in a standard medical examination program between 2001 and 2014. Platelet counts were measured through Complete Blood Count tests. A satellite-based spatio-temporal model was used to estimate 2-year average ambient PM2.5 concentration at each participant's address. Mixed-effects linear regression models were used to investigate the association between PM2.5 exposure and platelet counts.
RESULTS	This analysis included 175,959 men with 396,248 observations and 186,437 women with 397,877 observations. Every 10- μg/m3 increment in the 2-year average PM2.5 was associated with increases of 0.42% (95% CI: 0.38%, 0.47%) and 0.49% (95% CI: 0.44%, 0.54%) in platelet counts in men and women, respectively. A series of sensitivity analyses, including an analysis in participants free of cardiometabolic disorders, confirmed the robustness of the observed associations. Baseline data analyses showed that every 10- μg/m3 increment in PM2.5 was associated with higher risk of 17% and 14% of having elevated platelet counts (≥90th percentile) in men and women, respectively.
CONCLUSIONS	Long-term exposure to PM2.5 appears to be associated with increased platelet counts, indicating potential adverse effects on blood coagulability.

표-606. PubMed 논문번호 29759065의 내용 요약

구분	내용
PubMed ID	29759065
TITLE	High-resolution mapping of traffic related air pollution with Google street view cars and incidence of cardiovascular events within neighborhoods in Oakland, CA.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-018-0382-1
AUTHORS	Alexeeff Stacey E, Roy Ananya, Shan Jun, Liu Xi, Messier Kyle, Apte Joshua S, Portier Christopher, Sidney Stephen, Van Den Eeden Stephen K
BACKGROUND	Some studies have linked long-term exposure to traffic related air pollutants (TRAP) with adverse cardiovascular health outcomes; however, previous studies have not linked highly variable concentrations of TRAP measured at street-level within neighborhoods to cardiovascular health outcomes.
METHODS	Long-term pollutant concentrations for nitrogen dioxide [NO2], nitric oxide [NO], and black carbon [BC] were obtained by street-level mobile monitoring on 30 m road segments and linked to residential addresses of 41,869 adults living in Oakland during 2010 to 2015. We fit Cox proportional hazard models to estimate the relationship between air pollution exposures and time to first cardiovascular event. Secondary analyses examined effect modification by diabetes and age.
RESULTS	Long-term pollutant concentrations [mean, (standard deviation; SD)] for NO2, NO and BC were 9.9 ppb (SD 3.8), 4.9 ppb (SD 3.8), and 0.36 μg/m3 (0.17) respectively. A one SD increase in NO2, NO and BC, was associated with a change in risk of a cardiovascular event of 3% (95% confidence interval [CI] -6% to 12%), 3% (95% CI -5% to 12%), and - 1% (95% CI -8% to 7%), respectively. Among the elderly (≥65 yrs), we found an increased risk of a cardiovascular event of 12% for NO2 (95% CI: 2%, 24%), 12% for NO (95% CI: 3%, 22%), and 7% for BC (95% CI: -3%, 17%) per one SD increase. We found no effect modification by diabetes.
CONCLUSIONS	Street-level differences in long-term exposure to TRAP were associated with higher risk of cardiovascular events among the elderly, indicating that within-neighborhood differences in TRAP are important to cardiovascular health. Associations among the general population were consistent with results found in previous studies, though not statistically significant.

표-607. PubMed 논문번호 29803602의 내용 요약

구분	내용
PubMed ID	29803602
TITLE	Association of Short- and Medium-Term Particulate Matter Exposure with Risk of Mortality after Spontaneous Intracerebral Hemorrhage.
JOURNAL	Journal of stroke and cerebrovascular diseases : the official journal of National Stroke Association: 10.1016/j.jstrokecerebrovasdis.2018.05.007
AUTHORS	Forlivesi Stefano, Turcato Gianni, Zivelonghi Cecilia, Zannoni Massimo, Ricci Giorgio, Cervellin Gianfranco, Lippi Giuseppe, Bovi Paolo, Bonetti Bruno, Cappellari Manuel
OBJECTIVE	We investigated the association of short- and medium-term particulate matter (PM) exposure with risk of mortality in patients with spontaneous intracerebral hemorrhage (ICH) identified according to strict etiologic criteria.
METHODS	We conducted a retrospective analysis of prospectively collected data from consecutive patients with spontaneous ICH admitted to the emergency department of the University Hospital of Verona from March 2011 to December 2014. Outcome measures were mortality within 1 month after ICH and significant hematoma expansion (HE) defined as an absolute growth of more than 12.5 mL or a relative increase of more than 50% from baseline to follow-up computed tomography scan.
RESULTS	A final number of 308 patients were included. In the adjusted model, higher PM2.5 and PM10 values in the last 3 days (odds ratio [OR] 1.827, 95% confidence interval [CI] 1.057-3.159, P = .031 and OR 1.949, 95% CI 1.025-3.704, P = .042, respectively) and in the last 4 weeks (OR 4.975, 95% CI 2.174-11.381, P < .001 and OR 9.781, 95% CI 3.425-27.932, P < .001, respectively) before ICH were associated with higher mortality rate. No association was found between PM exposure and significant HE.
CONCLUSIONS	PM exposure in the short- and medium-term before spontaneous ICH was associated with risk of 1-month mortality, independent of predictors such as age, sex, stroke severity, intraventricular hemorrhage, ICH volume, ICH location, ICH etiologic subtype, significant HE, antithrombotic therapy, atrial fibrillation, and blood glucose levels.

표-608. PubMed 논문번호 29851627의 내용 요약

구분	내용
PubMed ID	29851627
TITLE	Urban greenness and mortality in Canada's largest cities: a national cohort study.
JOURNAL	The Lancet. Planetary health: 10.1016/S2542-5196(17)30118-3
AUTHORS	Crouse Dan L, Pinault Lauren, Balram Adele, Hystad Perry, Peters Paul A, Chen Hong, van Donkelaar Aaron, Martin Randall V, M□nard Richard, Robichaud Alain, Villeneuve Paul J
BACKGROUND	Findings from published studies suggest that exposure to and interactions with green spaces are associated with improved psychological wellbeing and have cognitive, physiological, and social benefits, but few studies have examined their potential effect on the risk of mortality. We therefore undertook a national study in Canada to examine associations between urban greenness and cause-specific mortality.
METHODS	We used data from a large cohort study (the 2001 Canadian Census Health and Environment Cohort [2001 CanCHEC]), which consisted of approximately 1·3 million adult (aged ≥19 years), non-immigrant, urban Canadians in 30 cities who responded to the mandatory 2001 Statistics Canada long-form census. The cohort has been linked by Statistics Canada to the Canadian mortality database and to annual income tax filings through 2011. We measured greenness with images from the moderate-resolution imaging spectroradiometer from NASA's Aqua satellite. We assigned estimates of exposure to greenness derived from remotely sensed Normalized Difference Vegetation Index (NDVI) within both 250 m and 500 m of participants' residences for each year during 11 years of follow-up (between 2001 and 2011). We used Cox proportional hazards models to estimate associations between residential greenness (as a continuous variable) and mortality. We estimated hazard ratios (HRs) and corresponding 95% CIs per IQR (0·15) increase in NDVI adjusted for personal (eg, education and income) and contextual covariates, including exposures to fine particulate matter, ozone, and nitrogen dioxide. We also considered effect modification by selected personal covariates (age, sex, household income adequacy quintiles, highest level of education, and marital status).
FINDINGS	Our cohort consisted of approximately 1 265 000 individuals at baseline who contributed 11 523 770 person-years. We showed significant decreased risks of mortality in the range of 8-12% from all causes of death examined with increased greenness around participants' residence. In the fully adjusted analyses, the risk was significantly decreased for all causes of death (non-accidental HR 0·915, 95% CI 0·905-0·924; cardiovascular plus diabetes 0·911, 0·895-0·928; cardiovascular 0·911, 0·894-0·928; ischaemic heart disease 0·904, 0·882-0·927; cerebrovascular 0·942, 0·902-0·983; and respiratory 0·899, 0·869-0·930). Greenness associations were more protective among men than women (HR 0·880, 95% CI 0·868-0·893 vs 0·955, 0·941-0·969), and among individuals with higher incomes (highest quintile 0·812, 0·791-0·834 vs lowest quintile 0·991, 0·972-1·011) and more education (degree or more 0·816, 0·791-0·842 vs did not complete high school 0·964, 0·950-0·978).
INTERPRETATION	Increased amounts of residential greenness were associated with reduced risks of dying from several common causes of death among urban Canadians. We identified evidence of inequalities, both in terms of exposures to greenness and mortality risks, by personal socioeconomic status among individuals living in generally similar environments, and with reasonably similar access to health

표-609. PubMed 논문번호 29851649의 내용 요약

구분	내용
PubMed ID	29851649
TITLE	Cardiorespiratory health effects of particulate ambient air pollution exposure in low-income and middle-income countries: a systematic review and meta-analysis.
JOURNAL	The Lancet. Planetary health: 10.1016/S2542-5196(17)30166-3
AUTHORS	Newell Katherine, Kartsonaki Christiana, Lam Kin Bong Hubert, Kurmi Om P
BACKGROUND	Most prospective studies on the health effects of particulate ambient air pollution exposure have focused on high-income countries, which have much lower pollutant concentrations than low-income and middle-income countries (LMICs) and different sources of pollution. We aimed to investigate the cardiorespiratory health effects of particulate ambient air pollution exposure in LMICs exclusively.
METHODS	For this systematic review and meta-analysis, we searched PubMed, Web of Science, Embase, LILACS, Global Health, and Proquest for studies published between database inception and Nov 28, 2016, investigating the cardiorespiratory health effects of particulate ambient air pollution exposure in LMICs. Data were extracted from published studies by one author, and then checked and verified by all authors independently. We pooled estimates by pollutant type (particulate matter with a diameter of <2·5 μm [PM2·5] or 2·5-10 μm [PM10]), lag, and outcome, and presented them as excess relative risk per 10 μg/m3 increase in particulate ambient air pollution. We used a random-effects model to derive overall excess risk. The study protocol is registered with PROSPERO, number CRD42016051733.
FINDINGS	Of 1553 studies identified, 91 met the full eligibility criteria. Only four long-term exposure studies from China were identified and not included in the meta-analysis. A 10 μg/m3 increase in same-day PM2·5 was associated with a 0·47% (95% CI 0·34-0·61) increase in cardiovascular mortality and a 0·57% (0·28-0·86) increase in respiratory mortality. A 10 μg/m3 increase in same-day PM10 was associated with a 0·27% (0·11-0·44) increase in cardiovascular mortality and a 0·56% (0·24-0·87) increase in respiratory mortality.
INTERPRETATION	Short-term exposure to particulate ambient air pollution is associated with increases in cardiorespiratory morbidity and mortality in LMIC's, with apparent regional-specific variations.
FUNDING	None.

표-610. PubMed 논문번호 29851739의 내용 요약

구분	내용
PubMed ID	29851739
TITLE	Risk of Stroke Among Survivors of the September 11, 2001, World Trade Center Disaster.
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0000000000001361
AUTHORS	Yu Shengchao, Alper Howard E, Nguyen Angela-Maithy, Brackbill Robert M
OBJECTIVE	The aim of this study was to investigate the association between 9/11-related posttraumatic stress disorder (PTSD), dust cloud exposure, and subsequent development of stroke among 42,527 enrollees in the World Trade Center (WTC) Health Registry.
METHODS	Using four waves of longitudinal data from the WTC Health Registry surveys, we employed Cox proportional hazards regression models to assess the associations.
RESULTS	Incidence of stroke was higher among those with PTSD or intense dust cloud exposure than those without, and it was even higher for those who had experienced both. In fully adjusted models, participants with PTSD had an increased risk of developing stroke [adjusted hazards ratio (AHR) 1.69, 95% confidence interval (95% CI) 1.42 to 2.02], as did those with intense dust exposure (AHR 1.29, 95% CI 1.09 to 1.53).
CONCLUSION	We found that individuals with 9/11-related PTSD and/or intense dust exposure may have an increased risk of developing stroke.

표-611. PubMed 논문번호 29884205의 내용 요약

구분	내용
PubMed ID	29884205
TITLE	Ambient air quality and spatio-temporal patterns of cardiovascular emergency department visits.
JOURNAL	International journal of health geographics: 10.1186/s12942-018-0138-8
AUTHORS	Yoo Eun-Hye, Brown Patrick, Eum Youngseob
BACKGROUND	Air pollutants have been associated with various adverse health effects, including increased rates of hospital admissions and emergency room visits. Although numerous time-series studies and case-crossover studies have estimated associations between day-to-day variation in pollutant levels and mortality/morbidity records, studies on geographic variations in emergency department use and the spatial effects in their associations with air pollution exposure are rare.
METHODS	We focused on the elderly who visited emergency room for cardiovascular related disease (CVD) in 2011. Using spatially and temporally resolved multi-pollutant exposures, we investigated the effect of short-term exposures to ambient air pollution on emergency department utilization. We developed two statistical models with and without spatial random effects within a hierarchical Bayesian framework to capture the spatial heterogeneity and spatial autocorrelation remaining in emergency department utilization.
RESULTS	Although the cardiovascular effect of spatially homogeneous pollutants, such as PM2.5 and ozone, was unchanged, we found the cardiovascular effect of NO[Formula: see text] was pronounced after accounting for the spatially correlated structure in emergency department utilization. We also identified areas with high ED utilization for CVD among the elderly and assessed the uncertainty associated with risk estimates.
CONCLUSIONS	We assessed the short-term effect of multi-pollutants on cardiovascular risk of the elderly and demonstrated the use of community multiscale air quality model-derived spatially and temporally resolved multi-pollutant exposures to an epidemiological study. Our results indicate that NO[Formula: see text] was significantly associated with the elevated ED utilization for CVD among the elderly.

표-612. PubMed 논문번호 29889687의 내용 요약

구분	내용
PubMed ID	29889687
TITLE	Associations of Source-apportioned Fine Particles with Cause-specific Mortality in California.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000873
AUTHORS	Berger Kimberly, Malig Brian J, Hasheminassab Sina, Pearson Dharshani L, Sioutas Constantinos, Ostro Bart, Basu Rupa
BACKGROUND	Exposure to ambient fine particulate matter (PM2.5) has been linked with premature mortality, but sources of PM2.5 have been less studied.
METHODS	We evaluated associations between source-specific PM2.5 exposures and cause-specific short-term mortality in eight California locations from 2002 to 2011. Speciated PM2.5 measurements were source-apportioned using Positive Matrix Factorization into eight sources and combined with death certificate data. We used time-stratified case-crossover analysis with conditional logistic regression by location and meta-analysis to calculate pooled estimates.
RESULTS	Biomass burning was associated with all-cause mortality lagged 2 days after exposure (lag2) (% changelag2 in odds per interquartile range width increase in biomass burning PM2.5 = 0.8, 95% confidence interval [CI] = 0.2, 1.4), cardiovascular (% changelag2 = 1.3, 95% CI = 0.3, 2.4), and ischemic heart disease (% changelag2 = 2.0, 95% CI = 0.6, 3.5). Vehicular emissions were associated with increases in cardiovascular mortality (% changelag0 = 1.4, 95% CI = 0.0, 2.9). Several other sources exhibited positive associations as well. Many findings persisted during the cool season. Warm season biomass burning was associated with respiratory/thoracic cancer mortality (% changelag1 = 5.9, 95% CI = 0.7, 11.3), and warm season traffic was associated with all-cause (% changelag0 = 1.9, 95% CI = 0.1, 3.6) and cardiovascular (% changelag0 = 2.9, 95% CI = 0.1, 5.7) mortality.
CONCLUSIONS	Our results suggest that acute exposures to biomass burning and vehicular emissions are linked with cardiovascular mortality, with additional sources (i.e., soil, secondary nitrate, secondary sulfate, aged sea salt, and chlorine sources) showing associations with other specific mortality types.

표-613. PubMed 논문번호 29929325의 내용 요약

구분	내용
PubMed ID	29929325
TITLE	The association between short and long-term exposure to PM〈sub〉2.5〈/sub〉 and temperature and hospital admissions in New England and the synergistic effect of the short-term exposures.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2018.05.181
AUTHORS	Yitshak-Sade Maayan, Bobb Jennifer F, Schwartz Joel D, Kloog Itai, Zanobetti Antonella
BACKGROUND	Particulate matter 〈2.5 μm in diameter (PM2.5) and heat are strong predictors of morbidity, yet few studies have examined the effects of long-term exposures on non-fatal events, or assessed the short and long-term effect on health simultaneously.
OBJECTIVE	We jointly investigated the association of short and long-term exposures to PM2.5 and temperature with hospital admissions, and explored the modification of the associations with the short-term exposures by one another and by temperature variability.
METHODS	Daily ZIP code counts of respiratory, cardiac and stroke admissions of adults ≥65 (N = 2,015,660) were constructed across New-England (2001-2011). Daily PM2.5 and temperature exposure estimates were obtained from satellite-based spatio-temporally resolved models. For each admission cause, a Poisson regression was fit on short and long-term exposures, with a random intercept for ZIP code. Modifications of the short-term effects were tested by adding interaction terms with temperature, PM2.5 and temperature variability.
RESULTS	Associations between short and long-term exposures were observed for all of the outcomes, with stronger effects of long-term exposures to PM2.5. For respiratory admissions, the short-term PM2.5 effect (percent increase per IQR) was larger on warmer days (1.12% versus -0.53%) and in months of higher temperature variability (1.63% versus -0.45%). The short-term temperature effect was higher in months of higher temperature variability as well. For cardiac admissions, the PM2.5 effect was larger on colder days (0.56% versus -0.30%) and in months of higher temperature variability (0.99% versus -0.56%).
CONCLUSIONS	We observed synergistic effects of short-term exposures to PM2.5, temperature and temperature variability. Long-term exposures to PM2.5 were associated with larger effects compared to short-term exposures.

표-614. PubMed 논문번호 29945580의 내용 요약

구분	내용
PubMed ID	29945580
TITLE	Neighborhood deprivation and biomarkers of health in Britain: the mediating role of the physical environment.
JOURNAL	BMC public health: 10.1186/s12889-018-5667-3
AUTHORS	Chaparro M Pia, Benzeval Michaela, Richardson Elizabeth, Mitchell Richard
BACKGROUND	Neighborhood deprivation has been consistently linked to poor individual health outcomes; however, studies exploring the mechanisms involved in this association are scarce. The objective of this study was to investigate whether objective measures of the physical environment mediate the association between neighborhood socioeconomic deprivation and biomarkers of health in Britain.
METHODS	We linked individual-level biomarker data from Understanding Society: The UK Household Longitudinal Survey (2010-2012) to neighborhood-level data from different governmental sources. Our outcome variables were forced expiratory volume in 1 s (FEV1%; n=16,347), systolic blood pressure (SBP; n=16,846), body mass index (BMI; n=19,417), and levels of C-reactive protein (CRP; n=11,825). Our measure of neighborhood socioeconomic deprivation was the Carstairs index, and the neighborhood-level mediators were levels of air pollutants (sulphur dioxide [SO2], particulate matter [PM10], nitrogen dioxide [NO2], and carbon monoxide [CO]), green space, and proximity to waste and industrial facilities. We fitted a multilevel mediation model following a multilevel structural equation framework in MPlus v7.4, adjusting for age, gender, and income.
RESULTS	Residents of poor neighborhoods and those exposed to higher pollution and less green space had worse health outcomes. However, only SO2 exposure significantly and partially mediated the association between neighborhood socioeconomic deprivation and SBP, BMI, and CRP.
CONCLUSION	Reducing air pollution exposure and increasing access to green space may improve population health but may not decrease health inequalities in Britain.

표-615. PubMed 논문번호 29946352의 내용 요약

구분	내용
PubMed ID	29946352
TITLE	Does chronic disease influence susceptibility to the effects of air pollution on depressive symptoms in China?
JOURNAL	International journal of mental health systems: 10.1186/s13033-018-0212-4
AUTHORS	Wang Qing, Yang Zhiming
Background	Exogenous stressors resulting from air pollution can lead to depression and chronic disease. Chinese levels of air pollution are among the highest in the world, and although associated adverse health effects are gradually emerging, research determining individual vulnerability is limited. This study estimated the association between air pollution and depressive symptoms and identified whether chronic disease influences an individual's susceptibility to depressive symptoms relating to air pollution.
Methods	Individual sample data from the China Health and Retirement Longitudinal Study and a group of city-level variables in 2011 and 2013 were used with the random effects model and Tobit model. Adjustments were made for demographic, socioeconomic status, health behavior, and city-level climate variables with respect to living areas. Analysis was also stratified using chronic disease characteristics.
Results	The total Center for Epidemiological Studies Depression scale evaluating depressive symptoms ranged between 7 and 28 [average 11.623 (SD = 4.664)]. An 1% increase in sulfur dioxide and total suspended particulate emission intensities was associated with depressive symptoms scores that were 1.266 (SE = 0.107, P < 0.001, 95% CI 1.057-1.475) and 1.318 (SE = 0.082, P < 0.001, 95% CI 1.157-1.480) higher, respectively. Compared to respondents without chronic disease, those with chronic diseases such as hypertension, dyslipidemia, diabetes or high blood sugar, cardiovascular diseases, cancer or malignant tumor, liver disease, chronic lung diseases, kidney disease, stomach or other digestive disease, arthritis or rheumatism, and asthma had scores that were higher for depressive symptoms.
Conclusions	Results confirm that the adverse health effects of air pollution should be considered when developing air pollution policies. Findings also provide justification for mental health interventions targeting air pollution exposure, especially for people with chronic diseases.

표-616. PubMed 논문번호 29991645의 내용 요약

구분	내용
PubMed ID	29991645
TITLE	Post-Traumatic Stress Disorder and Cardiovascular Diseases: A Cohort Study of Men and Women Involved in Cleaning the Debris of the World Trade Center Complex.
JOURNAL	Circulation. Cardiovascular quality and outcomes: 10.1161/CIRCOUTCOMES.117.004572
AUTHORS	Remch Molly, Laskaris Zoey, Flory Janine, Mora-McLaughlin Consuelo, Morabia Alfredo
BACKGROUND	We sought to determine whether post-traumatic stress disorder (PTSD) is a risk factor for myocardial infarction (MI) and stroke, beyond the expected effects from recognized cardiovascular risk factors and depression.
METHODS AND RESULTS	World Trade Center-Heart is an observational prospective cohort study of 6481 blue-collar first responders nested within the World Trade Center Health Program in New York City. Baseline measures in 2012 and 2013 included blood pressure, weight and height, and blood lipids. PTSD, depression, smoking, and dust exposure during the 2001 cleanup were self-reported. During the 4-year follow-up, outcomes were assessed through (1) interview-based incident, nonfatal MI, and stroke, validated in medical charts (n=118); and (2) hospitalizations for MI and stroke for New York city and state residents (n=180). Prevalence of PTSD was 19.9% in men and 25.9% in women, that is, at least twice that of the general population. Cumulative incidence of MI or stroke was consistently larger for men or women with PTSD across follow-up. Adjusted hazard ratios (HRs) were 2.22 (95% confidence interval [CI], 1.30-3.82) for MI and 2.51 (95% CI, 1.39-4.57) for stroke. For pooled MI and stroke, adjusted HRs were 2.35 (95% CI, 1.57-3.52) in all and 1.88 (95% CI, 1.01-3.49) in men free of depression. Using hospitalization registry data, adjusted HRs were 2.17 (95% CI, 1.41-3.32) for MI; 3.01 (95% CI, 1.84-4.93) for stroke; and for pooled MI and stroke, the adjusted HR was 2.40 (95% CI, 1.73-3.34) in all, HR was 2.44 (95% CI, 1.05-5.55) in women, and adjusted HR was 2.27 (95% CI, 1.41-3.67) in men free of depression. World Trade Center dust exposure had no effect.
CONCLUSIONS	This cohort study confirms that PTSD is a risk factor for MI and stroke of similar magnitude in men and women, independent of depression.

표-617. PubMed 논문번호 30005252의 내용 요약

구분	내용
PubMed ID	30005252
TITLE	Household biomass fuel use, blood pressure and carotid intima media thickness; a cross sectional study of rural dwelling women in Southern Nigeria.
JOURNAL	Environmental pollution (Barking, Essex : 1987): 10.1016/j.envpol.2018.06.102
AUTHORS	Ofori Sandra N, Fobil Julius N, Odia Osaretin J
BACKGROUND	Rising prevalence of cardiovascular disease requires in-depth understanding of predisposing factors. Studies show an association between air pollution and CVD but this association is not well documented in southern Nigeria where the use of biomass fuels (BMF) for domestic purposes is prevalent.
PURPOSE	This study aimed to explore the association between household BMF use and blood pressure (BP) and carotid intima media thickness (CIMT) among rural-dwelling women.
METHODS	A cross-sectional study of 389 women aged 18 years and older. Questionnaires were used to obtain data on predominant fuel used and a brief medical history. Wood, charcoal and agricultural waste were classified as BMF while kerosene, bottled gas and electricity were classified as non-BMF. Blood pressure and CIMT were measured using standard protocols. Regression analysis was used to assess the relationship between fuel type and BP, CIMT, pre-hypertension and hypertension after adjusting for confounders.
RESULTS	There was a significant difference in the mean (standard deviation) systolic BP (135.3, 26.7 mmHg vs 123.8, 22.6 mmHg; $p < 0.01$), diastolic BP (83.7, 18.5 mmHg vs 80.1, 13.8 mmHg; $p = 0.043$) and CIMT (0.63, 0.16 mm vs 0.56, 0.14 mm; $p = 0.004$) among BMF users compared to non-BMF users. In regression analysis, the use of BMF was significantly associated with 2.7 mmHg higher systolic BP ($p = 0.040$), 0.04 mm higher CIMT ($p = 0.048$) in addition to increased odds of pre-hypertension (OR 1.67 95% CI 1.56, 4.99, $P = 0.035$) but not hypertension (OR 1.23 95% CI 0.73, 2.07, $P = 0.440$).
CONCLUSION	In this population, there was a significant association between BMF use and increased SBP, CIMT and pre-hypertension. This requires further exploration with a large-scale longitudinal study design because there are policy implications for countries like Nigeria where a large proportion of the population still rely on BMF for domestic energy.

표-618. PubMed 논문번호 30015250의 내용 요약

구분	내용
PubMed ID	30015250
TITLE	Effect on blood pressure and eye health symptoms in a climate-financed randomized cookstove intervention study in rural India.
JOURNAL	Environmental research: 10.1016/j.envres.2018.06.044
AUTHORS	Aung Ther W, Baumgartner Jill, Jain Grishma, Sethuraman Karthik, Reynolds Conor, Marshall Julian D, Brauer Michael
BACKGROUND	Air pollution from cooking with solid fuels is a potentially modifiable risk factor for increased blood pressure and may lead to eye irritation.
OBJECTIVES	To evaluate whether a climate motivated cookstove intervention reduced blood pressure and eye irritation symptoms in Indian women.
METHODS	Households using traditional stoves were randomized to receive a rocket stove or continue using traditional stoves. Systolic (SBP) and diastolic blood pressure (DBP), and self-reported eye symptoms were measured twice, pre-intervention and at least 124 days post-intervention in women > 25 years old in control (N = 111) and intervention (N = 111) groups in rural Karnataka, India. Daily (24-h) fine particle (PM2.5) mass and absorbance (Abs) were measured in cooking areas at each visit. Mixed-effect models were used to estimate before-and-after differences in SBP, DBP and eye symptoms.
RESULTS	We observed a lower SBP (-2.0 (-4.5, 0.5) mmHg) and DBP (-1.1 (-2.9, 0.6) mmHg) among exclusive users of intervention stove, although confidence intervals included zero. Stacking or mixed use of intervention and traditional stoves contributed to a small increase in SBP 2.6 (-0.4, 5.7) mmHg) and DBP (1.2 (-0.9, 3.3) mmHg). Exclusive and mixed stove users experienced higher post-intervention reductions, on average, in self-reported eye irritation symptoms for burning sensation in eyes, and eyes look red often compared to control. Median air pollutant concentrations increased post-intervention in all stove groups, with the lowest median PM2.5 increase in the exclusive intervention stove group.
CONCLUSIONS	Health benefits were limited due to stacking and lower-than-predicted efficiency of the intervention stove in the field. Stove adoption and use behavior, in addition to stove technology, affects achievement of health co-benefits. Carbon-financing schemes need to align with international guidelines that have been set based on health outcomes to maximize health co-benefits from cookstove interventions.

표-619. PubMed 논문번호 30016318의 내용 요약

구분	내용
PubMed ID	30016318
TITLE	Short-term association between ambient temperature and acute myocardial infarction hospitalizations for diabetes mellitus patients: A time series study.
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1002612
AUTHORS	Lam Holly Ching Yu, Chan Juliana Chung Ngor, Luk Andrea On Yan, Chan Emily Ying Yang, Goggins William Bernard
BACKGROUND	Acute myocardial infarction (AMI) is the leading cause of death among people with diabetes mellitus (DM) and has been found to occur more frequently with extreme temperatures. With the increasing prevalence of DM and the rising global mean temperature, the number of heat-related AMI cases among DM patients may increase. This study compares excess risk of AMI during periods of extreme temperatures between patients with DM and without DM.
METHODS	Distributed lag nonlinear models (DLNMs) were used to estimate the short-term association between daily mean temperature and AMI admissions (International Classification of Diseases 9th revision [ICD-9] code: 410.00-410.99), stratified by DM status (ICD-9: 250.00-250.99), to all public hospitals in Hong Kong from 2002 to 2011, adjusting for other meteorological variables and air pollutants. Analyses were also stratified by season, age group, gender, and admission type (first admissions and readmissions). The admissions data and meteorological data were obtained from the Hong Kong Hospital Authority (HA) and the Hong Kong Observatory (HKO).
FINDINGS	A total of 53,769 AMI admissions were included in the study. AMI admissions among DM patients were linearly and negatively associated with temperature in the cold season (cumulative relative risk [cumRR] [95% confidence interval] in lag 0-22 days (12 ° C versus 24 ° C) = 2.10 [1.62-2.72]), while those among patients without DM only started increasing when temperatures dropped below 22 ° C with a weaker association (cumRR = 1.43 [1.21-1.69]). In the hot season, AMI hospitalizations among DM patients started increasing when the temperature dropped below or rose above 28.8 ° C (cumRR in lag 0-4 days [30.4 versus 28.8 ° C] = 1.14 [1.00-1.31]), while those among patients without DM showed no association with temperature. The differences in sensitivity to temperature between patients with DM and without DM were most apparent in the group <75 years old and among first-admission cases in the cold season. The main limitation of this study was the unavailability of data on individual exposure to ambient temperature.
CONCLUSIONS	DM patients had a higher increased risk of AMI admissions than non-DM patients during extreme temperatures. AMI admissions risks among DM patients rise sharply in both high and low temperatures, with a stronger effect in low temperatures, while AMI risk among non-DM patients only increased mildly in low temperatures. Targeted health protection guidelines should be provided to warn DM patients and physicians about the dangers of extreme temperatures. Further studies to project the impacts of AMI risks on DM patients by climate change are warranted.

표-620. PubMed 논문번호 30021805의 내용 요약

구분	내용
PubMed ID	30021805
TITLE	Effects of Leisure-Time and Transport-Related Physical Activities on the Risk of Incident and Recurrent Myocardial Infarction and Interaction With Traffic-Related Air Pollution: A Cohort Study.
JOURNAL	Journal of the American Heart Association: 10.1161/JAHA.118.009554
AUTHORS	Kubesch Nadine J, Therming J ø rgensen Jeanette, Hoffmann Barbara, Loft Steffen, Nieuwenhuijsen Mark J, Raaschou-Nielsen Ole, Pedersen Marie, Hertel Ole, Overvad Kim, Tj ø nneland Anne, Prescot Eva, Andersen Zorana J
BACKGROUND	Physical activity enhances the uptake of air pollutants, possibly reducing its beneficial effects. We examined the effects of leisure-time and transport-related physical activities on the risk of myocardial infarction (MI), and whether potential benefits on MI are reduced by exposure to traffic-related air pollution.
METHODS AND RESULTS	A group of 57 053 participants (50-65 years of age) from the Danish Diet, Cancer, and Health cohort reported physical activity at baseline (1993-1997) and were linked to registry data on hospital contacts and out-of-hospital deaths caused by MI, until December 2015. Nitrogen dioxide levels were estimated at participants' baseline residences. We used Cox regressions to associate participation in sports, cycling, walking, and gardening with incident and recurrent MI, and tested for interaction by nitrogen dioxide. Of 50 635 participants without MI at baseline, 2936 developed incident MI, and of 1233 participants with MI before baseline, 324 had recurring MI during follow-up. Mean nitrogen dioxide concentration was 18.7 μg/m3 at baseline (1993-1997). We found inverse statistically significant associations between participation in sports (hazard ratio; 95% confidence interval: 0.85; 0.79-0.92), cycling (0.91; 0.84-0.98), gardening (0.87; 0.80-0.95), and incident MI, while the association with walking was statistically nonsignificant (0.95; 0.83-1.08). Recurrent MI was statistically nonsignificantly inversely associated with cycling (0.80; 0.63-1.02), walking (0.82, 0.57-1.16), and gardening (0.91; 0.71-1.18), and positively with sports (1.06; 0.83-1.35). There was no effect modification of the associations between physical activity and MI by nitrogen dioxide.
CONCLUSIONS	Benefits of physical activity on both the incidence and the recurrence of MI are not reduced by exposure to high levels of air pollution.

표-621. PubMed 논문번호 30032102의 내용 요약

구분	내용
PubMed ID	30032102
TITLE	Association between benzene and congenital anomalies in Oklahoma, 1997-2009.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2018-105054
AUTHORS	Janitz Amanda E, Dao Hanh Dung, Campbell Janis E, Stoner Julie A, Peck Jennifer D
OBJECTIVES	Although the most common cause of death in infants, little is known about the aetiology of congenital anomalies. Recent studies have increasingly focused on environmental exposures, including benzene. While benzene is known to affect the central nervous system, the effects on the developing fetus are unclear.
METHODS	We conducted a retrospective cohort study to evaluate the association between ambient benzene exposure and the prevalence of congenital anomalies among 628 121 singleton births in Oklahoma from 1997 to 2009. We obtained benzene from the Environmental Protection Agency's 2005 National-Scale Air Toxics Assessment for the census tract of the birth residence. We used modified Poisson regression with robust SEs to calculate prevalence proportion ratios (PPRs) and 95% CIs between quartiles of benzene exposure and critical congenital heart defects (CCHDs), neural tube defects (NTDs) and oral clefts adjusted for maternal education and tobacco use.
RESULTS	Median benzene exposure concentration in Oklahoma was 0.57 □g/m3. We observed no association between benzene exposure and oral clefts, CCHDs or NTDs. When specific anomalies were examined, we observed an increased prevalence of cleft lip among those exposed to the second quartile of benzene compared with the first (PPR 1.50, 95% CI 1.05 to 2.13), though no association with higher levels of exposure.
CONCLUSIONS	Our findings do not provide support for an increased prevalence of anomalies in areas more highly exposed to benzene. Future studies would benefit from pooling data from multiple states to increase statistical power and precision in studies of air pollutants and specific anomalies.

표-622. PubMed 논문번호 30044232의 내용 요약

구분	내용
PubMed ID	30044232
TITLE	Associations between Living Near Water and Risk of Mortality among Urban Canadians.
JOURNAL	Environmental health perspectives: 10.1289/EHP3397
AUTHORS	Crouse Dan L, Balram Adele, Hystad Perry, Pinault Lauren, van den Bosch Matilda, Chen Hong, Rainham Daniel, Thomson Errol M, Close Christopher H, van Donkelaar Aaron, Martin Randall V, M□nard Richard, Robichaud Alain, Villeneuve Paul J
BACKGROUND	Increasing evidence suggests that residential exposures to natural environments, such as green spaces, are associated with many health benefits. Only a single study has examined the potential link between living near water and mortality.
OBJECTIVE	We sought to examine whether residential proximity to large, natural water features (e.g., lakes, rivers, coasts, "blue space") was associated with cause-specific mortality.
METHODS	Our study is based on a population-based cohort of nonimmigrant adults living in the 30 largest Canadian cities [i.e., the 2001 Canadian Census Health and Environment Cohort) (CanCHEC)]. Subjects were drawn from the mandatory 2001 Statistics Canada long-form census, who were linked to the Canadian mortality database and to annual income-tax filings, through 2011. We estimated associations between living within of blue space and deaths from several common causes of death. We adjusted models for many personal and contextual covariates, as well as for exposures to residential greenness and ambient air pollution.
RESULTS	Our cohort included approximately 1.3 million subjects at baseline, 106,180 of whom died from nonaccidental causes during follow-up. We found significant, reduced risks of mortality in the range of 12-17% associated with living within of water in comparison with living farther away, among all causes of death examined, except with external/accidental causes. Protective effects were found to be higher among women and all older adults than among other subjects, and protective effects were found to be highest against deaths from stroke and respiratory-related causes.
CONCLUSIONS	Our findings suggest that living near blue spaces in urban areas has important benefits to health, but further work is needed to better understand the drivers of this association. https://doi.org/10.1289/EHP3397.

표-623. PubMed 논문번호 30074537의 내용 요약

구분	내용
PubMed ID	30074537
TITLE	Diabetes Status and Susceptibility to the Effects of PM2.5 Exposure on Cardiovascular Mortality in a National Canadian Cohort.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000908
AUTHORS	Pinault Lauren, Brauer Michael, Crouse Daniel L, Weichenthal Scott, Erickson Anders, van Donkelaar Aaron, Martin Randall V, Charbonneau Shannon, Hystad Perry, Brook Jeffrey R, Tjepkema Michael, Christidis Tanya, M□nard Richard, Robichaud Alain, Burnett Richard T
BACKGROUND	Diabetes is infrequently coded as the primary cause of death but may contribute to cardiovascular disease (CVD) mortality in response to fine particulate matter (PM2.5) exposure. We analyzed all contributing causes of death to examine susceptibility of diabetics to CVD mortality from long-term exposure.
METHODS	We linked a subset of the 2001 Canadian Census Health and Environment Cohort (CanCHEC) with 10 years of follow-up to all causes of death listed on death certificates. We used survival models to examine the association between CVD deaths (n = 123,500) and exposure to PM2.5 among deaths that co-occurred with diabetes (n = 20,600) on the death certificate. More detailed information on behavioral covariates and diabetes status at baseline available in the Canadian Community Health Survey (CCHS)-mortality cohort (n = 12,400 CVD deaths, with 2,800 diabetes deaths) complemented the CanCHEC analysis.
RESULTS	Among CanCHEC subjects, comention of diabetes on the death certificate increased the magnitude of association between CVD mortality and PM2.5 (HR = 1.51 [1.39-1.65] per 10 μg/m) versus all CVD deaths (HR = 1.25 [1.21-1.29]) or CVD deaths without diabetes (HR = 1.20 [1.16-1.25]). Among CCHS subjects, diabetics who used insulin or medication (included as proxies for severity) had higher HR estimates for CVD deaths from PM2.5 (HR = 1.51 [1.08-2.12]) relative to the CVD death estimate for all respondents (HR = 1.31 [1.16-1.47]).
CONCLUSIONS	Mention of diabetes on the death certificate resulted in higher magnitude associations between PM2.5 and CVD mortality, specifically among those who manage their diabetes with insulin or medication. Analyses restricted to the primary cause of death likely underestimate the role of diabetes in air pollution-related mortality. See video abstract at, http://links.lww.com/EDE/B408.

표-624. PubMed 논문번호 30093188의 내용 요약

구분	내용
PubMed ID	30093188
TITLE	The Effects of Secondhand Smoke Exposure on Postoperative Pain and Ventilation Values During One-Lung Ventilation: A Prospective Clinical Tria
JOURNAL	Journal of cardiothoracic and vascular anesthesia: 10.1053/j.jvca.2018.06.02
AUTHORS	Ozkan Ahmet Selim, Ucar Muharrem, Akbas Sedat, Ozkan Ahmet Selim, Ucar Muharrem, Akbas Sedat
OBJECTIVES	To investigate the relationships between secondhand smoke (SHS) exposure and oxygenation during one-lung ventilation (OLV) in lobectomy surgery an between SHS exposure and postoperative analgesic consumption.
DESIGN	Prospective study.
SETTING	University, Faculty of Medicine, operating room.
PARTICIPANTS	Sixty adult patients with American Society of Anesthesiologists score II to III aged 18 to 65 years, with a body mass index (BMI) <35 kg/m2 scheduled for lobectomy surgery by open thoracotomy.
INTERVENTIONS	Patients were divided into 2 groups: the SHS group (n = 30) (urine cotinine level ≥6.0 ng/mL) and the NS (nonsmoker) group (n = 30) (urine cotinine level <6.0 ng/mL and no smoking history). SHS exposure was defined according to a previously published algorithm.
MEASUREMENTS AND MAIN RESULTS	Noninvasive blood pressure, electrocardiography, capnography, and peripheral oxygen saturation were monitored, and intra- and postoperative arterial oxygen tension (PaO2), arterial carbon dioxide tension (PaCO2), and intraoperative peak airway pressure were compared between the 2 groups. Postoperative analgesic consumption was calculated. No significant differences in demographics or preoperative data were noted between the 2 groups. PaO2 values 10 minutes after OLV onset and 10 minutes after the en of OLV were increased significantly in the NS group compared with those in the SHS group ($p < 0.05$). PaO2 values after 10 minutes of OLV in the NS an SHS groups were 285.5 ± 90 mmHg and 186.7 ± 66 mmHg, respectively. PaO2 values after OLV termination in the NS and SHS groups were 365.8 ± 58 mmHg and 283.6 ± 64 mmHg ($p < 0.05$), respectively. PaCO2 values 10 minutes after OLV onset, 10 minutes after the end of OLV, at the end of surgery, and upon arrival in the intermediate care unit were significantly different between the 2 groups ($p < 0.05$).
CONCLUSION	The present study demonstrated that during OLV, patients exposed to SHS exhibited significantly lower arterial oxygen pressure compared with nonsmokers. Arterial carbon dioxide values were increased significantly in SHS-exposed patients. Morphine consumption for postoperative analgesia also was increased in patients exposed to SHS compared with that in nonsmokers.

표-625. PubMed 논문번호 30097052의 내용 요약

구분	내용
PubMed ID	30097052
TITLE	Cardiovascular and inflammatory mechanisms in healthy humans exposed to air pollution in the vicinity of a steel mill.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-018-0270-4
AUTHORS	Kumarathasan Premkumari, Vincent Renaud, Blais Erica, Bielecki Agnieszka, Gu□nette Jos□e, Filiatreault Alain, Brion Orly, Cakmak Sabit, Thomson Errol M, Shutt Robin, Kauri Lisa Marie, Mahmud Mamun, Liu Ling, Dales Robert
BACKGROUND	There is a paucity of mechanistic information that is central to the understanding of the adverse health effects of source emission exposures. To identify source emission-related effects, blood and saliva samples from healthy volunteers who spent five days near a steel plant (Bayview site, with and without a mask that filtered many criteria pollutants) and at a well-removed College site were tested for oxidative stress, inflammation and endothelial dysfunction markers.
METHODS	Biomarker analyses were done using multiplexed protein-array, HPLC-Fluorescence, EIA and ELISA methods. Mixed effects models were used to test for associations between exposure, biological markers and physiological outcomes. Heat map with hierarchical clustering and Ingenuity Pathway Analysis (IPA) were used for mechanistic analyses.
RESULTS	Mean CO, SO2 and ultrafine particles (UFP) levels on the day of biological sampling were higher at the Bayview site compared to College site. Bayview site exposures "without" mask were associated with increased ($p < 0.05$) pro-inflammatory cytokines (e.g IL-4, IL-6) and endothelins (ETs) compared to College site. Plasma IL-1β, IL-2 were increased ($p < 0.05$) after Bayview site "without" compared to "with" mask exposures. Interquartile range (IQR) increases in CO, UFP and SO2 were associated with increased ($p < 0.05$) plasma pro-inflammatory cytokines (e.g. IL-6, IL-8) and ET-1(1-21) levels. Plasma/saliva BET-1 levels were positively associated ($p < 0.05$) with increased systolic BP. C-reactive protein (CRP) was positively associated ($p < 0.05$) with increased heart rate. Protein network analyses exhibited activation of distinct inflammatory mechanisms after "with" and "without" mask exposures at the Bayview site relative to College site exposures.
CONCLUSIONS	These findings suggest that air pollutants in the proximity of steel mill site can influence inflammatory and vascular mechanisms. Use of mask and multiple biomarker data can be valuable in gaining insight into source emission-related health impacts.

표-626. PubMed 논문번호 30113549의 내용 요약

구분	내용
PubMed ID	30113549
TITLE	The association between prenatal exposure to environmental tobacco smoke and childhood obesity: a systematic review.
JOURNAL	JBI database of systematic reviews and implementation reports: 10.11124/JBISRIR-2017-003558
AUTHORS	Qureshi Rubab, Jadotte Yuri, Zha Peijia, Porter Sallie Ann, Holly Cheryl, Salmond Susan, Watkins Elizabeth Ann
OBJECTIVE	The objective of the review was to determine if prenatal exposure to environmental tobacco smoke (ETS) increases the risk of obesity and overweight in children.
INTRODUCTION	Childhood obesity has reached epidemic proportions in many developed countries. This is of great concern as childhood obesity is associated with early onset of chronic diseases such as coronary artery disease, type II diabetes and hypertension in adulthood. Extensive research suggests a multifactorial etiology. These factors include genetic markers, individual lifestyle, social and environmental factors, particularly the interaction between these factors. Among environmental factors, prenatal exposure to ETS has been linked to increased rates of obesity and overweight in childhood.
INCLUSION CRITERIA	This review considered studies on children of women who were non-smokers and who reported exposure to ETS during pregnancy. The exposure of interest was exposure to ETS or second hand smoke during pregnancy, determined by either: i) self-reported maternal exposure; and/or ii) serum cotinine levels. Observational studies such as cohort studies, case control studies, retrospective studies and analytical cross-sectional studies were included. Outcomes of interest were weight, height and body mass index of children from birth up to 18 years.
METHODS	A three-step search strategy was used to search for published and unpublished studies in the English language. No search range (years) was set. Two reviewers assessed the studies for inclusion and methodological quality using the Joanna Briggs Institute System for the Unified Management, Assessment and Review of Information (JBI SUMARI) standardized appraisal instruments. Data was extracted by two people independently and entered into the JBI extraction tool. Extracted data was pooled in a statistical meta-analysis based on a random effects model.
RESULTS	Nineteen studies were included in the review. Eight of the studies were included in the final meta-analysis. Findings suggest that there was an association between prenatal exposure to ETS and childhood obesity (odds ratio [OR]: 1.905, CI: 1.23-2.94), and no association between ETS exposure and overweight (OR: 1.51, CI: 0.49-4.59). The high rates of heterogeneity between studies in both of the meta-analyses determined by the I statistic (97% and 99%, respectively) sanction caution in the interpretation and use of these findings.
CONCLUSIONS	Based on the evidence, childhood obesity is associated with exposure to prenatal ETS, however overweight does not appear to be associated with this type of exposure.

표-627. PubMed 논문번호 30122215의 내용 요약

구분	내용
PubMed ID	30122215
TITLE	Secondhand Smoke Exposure in Childhood and Adulthood in Relation to Adult Mortality Among Never Smokers.
JOURNAL	American journal of preventive medicine: 10.1016/j.amepre.2018.05.005
AUTHORS	Diver W Ryan, Jacobs Eric J, Gapstur Susan M
INTRODUCTION	Secondhand smoke is known to have adverse effects on the lung and vascular systems in both children and adults. It is unknown if childhood exposure to secondhand smoke is associated with adult mortality.
METHODS	The authors examined associations of childhood and adult secondhand smoke exposure with death from all causes, ischemic heart disease, stroke, and chronic obstructive pulmonary disease among 70,900 never smoking men and women, predominantly aged ≥50 years, from the Cancer Prevention Study-II Nutrition Cohort in 1992-1993. There were 25,899 participant deaths during follow-up through 2014. During 2016-2017, Cox proportional hazards regression models were used to calculate multivariable-adjusted hazard ratios and 95% CIs.
RESULTS	Childhood secondhand smoke exposure was not associated with all-cause mortality. However, childhood secondhand smoke (living with a smoker for 16-18 years during childhood) was associated with higher mortality from chronic obstructive pulmonary disease (hazard ratio=1.31, 95% CI=1.05, 1.65). Adult secondhand smoke exposure of ≥10 hours/week at enrollment was associated with a higher risk of all-cause (hazard ratio=1.09, 95% CI=1.04, 1.14); ischemic heart disease (hazard ratio=1.27, 95% CI=1.14, 1.42); stroke (hazard ratio=1.23, 95% CI=1.04, 1.45); and chronic obstructive pulmonary disease (hazard ratio=1.42, 95% CI=0.97, 2.09) mortality.
CONCLUSIONS	These results suggest that childhood secondhand smoke exposure, as well as adult secondhand smoke exposure, increase the risk of chronic obstructive pulmonary disease death in adulthood. Consistent with previous studies, the results also show that adult secondhand smoke is meaningfully associated with higher mortality from vascular disease and all causes. Overall, these findings provide further evidence for reducing secondhand smoke exposure throughout life.

표-628. PubMed 논문번호 30134853의 내용 요약

구분	내용
PubMed ID	30134853
TITLE	Exposure to air pollution during preconceptional and prenatal periods and risk of hypertensive disorders of pregnancy: a retrospective cohort study in Seoul, Korea.
JOURNAL	BMC pregnancy and childbirth: 10.1186/s12884-018-1982-z
AUTHORS	Choe Seung-Ah, Jun Yoon-Bae, Kim Sun-Young
BACKGROUND	Previous studies suggested associations between prenatal exposure to air pollution and hypertensive disorders of pregnancy. We explored the associations between ambient concentrations of five major air pollutants during preconceptional and prenatal periods and three hypertensive disorders of pregnancy in Seoul, Korea, using a population-representative cohort.
METHODS	We obtained heath and demographic data of pregnant women residing in Seoul for 2002-2013 from the Korean National Health Insurance Service-National Sample Cohort. For mother's individual exposures to air pollution, we computed concentrations of particulate matter ≤10 μm in diameter (PM10), nitrogen dioxide (NO2), carbon monoxide (CO), sulfur dioxide (SO2), and ozone (O3) during 1, 3, 6, and 12 months to birth using regulatory monitoring data in Seoul. The associations between air pollution and hypertensive disorders were explored by using logistic regression models after adjusting for individual confounders.
RESULTS	Among 18,835 pregnant women in Seoul, 0.6, 0.5, and 0.4% of women developed gestational hypertension, preeclampsia, and preeclampsia requiring magnesium sulfate (Mg-preeclampsia), respectively. Although most odds ratios (ORs) were not statistically significant, we found increasing risk gradients with disease severity depending on the pollutant. There was the association between PM10 during 6 months to birth and gestational hypertension (OR for an interquartile range increase = 1.68 [95% confidence interval = 1.09-2.58]). NO2 and ozone during 12 and 1 month, respectively, before birth were associated with Mg-preeclampsia (1.43 [1.01-2.03], 1.53 [1.03-2.27]).
CONCLUSIONS	We observed positive associations of exposure to some air pollutants before and during pregnancy with hypertensive disorders of pregnancy among the Korean general population. Future studies with refined exposure metrics should confirm our findings.

표-629. PubMed 논문번호 30172196의 내용 요약

구분	내용
PubMed ID	30172196
TITLE	Cardiovascular benefits of short-term indoor air filtration intervention in elderly living in Beijing: An extended analysis of BIAPSY study.
JOURNAL	Environmental research: 10.1016/j.envres.2018.08.026
AUTHORS	Liu Shuo, Chen Jie, Zhao Qian, Song Xiaoming, Shao Danqing, Meliefste Kees, Du Yipeng, Wang Juan, Wang Meng, Wang Tong, Feng Baihuan, Wu Rongshan, Xu Hongbing, Bei He, Brunekreef Bert, Huang Wei
BACKGROUND	Adverse cardiovascular effects associated with air pollution exposure have been widely demonstrated. However, inconsistent cardiovascular responses were observed from reducing indoor air pollution exposure. We aimed to assess whether short-term air filtration intervention could benefit cardiovascular health in elderly living in high pollution area.
METHODS	A randomized crossover intervention study of short-term indoor air filtration intervention on cardiovascular health was conducted among 35 non-smoking elderly participants living in Beijing in the winter of 2013, as part of Beijing Indoor Air Purifier StudY (BIAPSY). Portable air filtration units were randomly allocated to active filtration for 2 weeks and sham filtration for 2 weeks in the households. Twelve-hour daytime ambulatory heart rate variability (HRV) and blood pressure (ABP) were measured during active and sham filtration. Concurrently, real-time indoor and outdoor particulate matter with diameter less than 2.5 □m (PM2.5) and indoor black carbon (BC) concentrations were measured. We applied generalized additive mixed models to evaluate the associations of 1- to 10-h moving average (MA) exposures of indoor PM2.5 and BC with HRV and ABP indices, and to explore whether these associations could be modified by air filtration.
RESULTS	We observed decreases of 34.8% in indoor PM2.5 and 35.3% in indoor BC concentrations during active filtration. Indoor PM2.5 and BC exposures were significantly associated with reduced HRV and increased ABP indices, and greater changes were observed during sham filtration. In specific, each 10 μg/m3 increase in indoor PM2.5 at MA8-h was associated with a significant reduction of 1.34% (95% CI: -2.42, -0.26) in SDNN during sham filtration, compared with a non-significant reduction of 0.81% (95% CI: -6.00, 4.68) during active filtration (Pinter$<$0.001). Each 1 μg/m3 increase in indoor BC at MA8-h was associated with a significant increase of 2.41% (95% CI: 0.38, 4.47) in SBP during sham filtration, compared with a non-significant increase of -1.09% (95% CI: -4.06, 1.96) during active filtration (Pinter = 0.135). Nonlinear inverse exposure-response relationships of indoor air pollution exposures with predicted HRV and ABP indices also confirmed some cardiovascular benefits of short-term air filtration intervention.
CONCLUSIONS	Our results suggested that short-term indoor air filtration intervention can be of some cardiovascular benefits in elderly living with high pollution episodes.

표-630. PubMed 논문번호 30172425의 내용 요약

구분	내용
PubMed ID	30172425
TITLE	Passive Smoking Exacerbates Nicotinamide-Adenine Dinucleotide Phosphate Oxidase Isoform 2-Induced Oxidative Stress and Arterial Dysfunction in Children with Persistent Allergic Rhinitis.
JOURNAL	The Journal of pediatrics: 10.1016/j.jpeds.2018.06.053
AUTHORS	Loffredo Lorenzo, Zicari Anna Maria, Occasi Francesca, Perri Ludovica, Carnevale Roberto, Battaglia Simona, Angelico Francesco, Del Ben Maria, Martino Francesco, Nocella Cristina, Farcomeni Alessio, De Castro Giovanna, Duse Marzia, Violi Francesco
OBJECTIVE	To characterize nicotinamide-adenine dinucleotide phosphate oxidase isoform 2 (NOX2), oxidative stress, and endothelial function in children with and without allergic rhinitis and to ascertain the effect of passive smoke exposure on these factors, because there is an established association between allergic rhinitis and increased cardiovascular risk in adults.
METHODS	We recruited 130 children-65 with persistent allergic rhinitis and 65 healthy controls. A cross-sectional study was performed to compare endothelial function by flow-mediated dilation, blood levels of isoprostanes, serum activity of soluble NOX2-dp (sNOX2-dp), and nitric oxide bioavailability, in these 2 groups of children. Serum cotinine levels were assessed to measure exposure to passive smoking.
RESULTS	Compared with healthy controls, children with persistent allergic rhinitis had significantly higher sNOX2-dp and isoprostanes levels, lower flow-mediated dilation, and reduced nitric oxide bioavailability. Multivariable linear regression analysis showed that flow-mediated dilation, isoprostanes, and cotinine were independently associated with sNOX2-dp levels. Of note, sNOX2-dp serum levels were significantly higher in children with allergic rhinitis exposed to smoke, as compared with unexposed children with allergic rhinitis.
CONCLUSION	NOX2 is activated in children with persistent allergic rhinitis and passive smoke exposure exacerbates this effect. We further demonstrate an association between higher sNOX2-dp and oxidative stress and endothelial dysfunction.

표-631. PubMed 논문번호 30172448의 내용 요약

구분	내용
PubMed ID	30172448
TITLE	The Relationship Between Ambient Air Pollution and Acute Ischemic Stroke: A Time-Stratified Case-Crossover Study in a City-State With Seasonal Exposure to the Southeast Asian Haze Problem.
JOURNAL	Annals of emergency medicine: 10.1016/j.annemergmed.2018.06.037
AUTHORS	Ho Andrew F W, Zheng Huili, De Silva Deidre A, Wah Win, Earnest Arul, Pang Yee H, Xie Zhenjia, Pek Pin P, Liu Nan, Ng Yih Y, Wong Ting H, Foo Ling L, Ong Marcus E H
STUDY OBJECTIVE	Studies are divided on the short-term association of air pollution with stroke. Singapore is exposed to seasonal transboundary haze. We aim to investigate the association between air pollution and stroke incidence in Singapore.
METHODS	We performed a time-stratified case-crossover analysis on all ischemic stroke cases reported to the Singapore Stroke Registry from 2010 to 2015. Exposure on days was compared with control days on which exposure did not occur. Control days were chosen on the same day of the week earlier and later in the same month in the same year. We fitted a conditional Poisson regression model to daily stroke incidence that included Pollutant Standards Index and environmental confounders. The index was categorized according to established classification (0 to 50=good, 51 to 100=moderate, and ≥101=unhealthy). We assessed the relationship between stroke incidence and Pollutant Standards Index in the entire cohort and in predetermined subgroups of individual-level characteristics.
RESULTS	There were 29,384 ischemic stroke cases. Moderate and unhealthy Pollutant Standards Index levels showed association with stroke occurrence, with incidence risk ratio 1.10 (95% confidence interval 1.06 to 1.13) and 1.14 (95% confidence interval 1.03 to 1.25), respectively. Subgroup analyses showed generally significant association, except in Indians and nonhypertensive patients. The association was significant in subgroups aged 65 years or older, women, Chinese, nonsmokers and those with history of diabetes, hypertension, and hyperlipidemia. Stratified by age and smoking, the risk diminished in smokers of all ages. Risk remained elevated for 5 days after exposure.
CONCLUSION	We found a short-term elevated risk of ischemic stroke after exposure to air pollution. These findings have public health implications for stroke prevention and emergency health services delivery.

표-632. PubMed 논문번호 30190659의 내용 요약

구분	내용
PubMed ID	30190659
TITLE	Premature Deaths Attributable to Long-term Exposure to Ambient Fine Particulate Matter in the Republic of Korea.
JOURNAL	Journal of Korean medical science: 10.3346/jkms.2018.33.e251
AUTHORS	Kim Jong-Hun, Oh In-Hwan, Park Jae-Hyun, Cheong Hae-Kwan
Background	Ambient fine particulate matter (PM2.5) is the major environmental health risk factor in Korea. Exposure to PM2.5 has been a growing public concern nationwide. With the rapid aging of the Korean population, the health effects attributable to long-term exposure to PM2.5 were expected to increase further in the future. We aimed to estimate premature deaths attributable to long-term exposure to ambient PM2.5 in Korea.
Methods	A modelled estimation of long-term exposure to PM2.5 was used to calculate the nationwide exposure level. Hazard ratios of long-term exposure to PM2.5 were obtained from a large prospective cohort study in North America. Modified cause of death (CoD) data, which applied the garbage code reclassification algorithm, were used to calculate premature deaths attributable to long-term exposure to PM2.5.
Results	From 1990 to 2013, the average population-weighted PM2.5 concentration in Korea was 30.2 μg/m3. The estimated number of premature deaths was 17,203 (95% confidence interval [CI], 11,056-22,772). The most common CoD was ischemic stroke (5,382; 3,101-7,403), followed by cancer of trachea, bronchus, and lung (4,958; 2,857-6,820), hemorrhagic stroke (3,452; 1,989-4,748), and ischemic heart disease (3,432; 1,383-5,358).
Conclusion	Premature deaths due to long-term exposure to PM2.5 accounted for 6.4% of all deaths in Korea. However, individual efforts alone cannot prevent the effects of air pollution. This disease burden study can serve as a basis for the establishment of government policies and budgets and can be used to assess the effectiveness of environmental health policies.

표-633. PubMed 논문번호 30193931의 내용 요약

구분	내용
PubMed ID	30193931
TITLE	Role of Apparent Temperature and Air Pollutants in Hospital Admissions for Acute Myocardial Infarction in the North of Spain.
JOURNAL	Revista espanola de cardiologia (English ed.): 10.1016/j.rec.2018.07.009
AUTHORS	Roy□ Dominic, Zarrabeitia Mar□a Teresa, Fdez-Arroyabe Pablo, □lvarez Guti□rrez Alberto, Santurt□n Ana, Roy□ Dominic, Zarrabeitia Mar□a Teresa, Fdez-Arroyabe Pablo, □lvarez Guti□rrez Alberto, Santurt□n Ana
INTRODUCTION AND OBJECTIVES	The role of the environment on cardiovascular health is becoming more prominent in the context of global change. The aim of this study was to analyze the relationship between apparent temperature (AT) and air pollutants and acute myocardial infarction (AMI) and to study the temporal pattern of this disease and its associated mortality.
METHODS	We performed a time-series study of admissions for AMI in Cantabria between 2001 and 2015. The association between environmental variables (including a biometeorological index, AT) and AMI was analyzed using a quasi-Poisson regression model. To assess potential delayed and non-linear effects of these variables on AMI, a lag non-linear model was fitted in a generalized additive model.
RESULTS	The incidence rate and the mortality followed a downward trend during the study period (CC=-0.714; P=.0002). An annual pattern was found in hospital admissions (P=.005), with the highest values being registered in winter; a weekly trend was also identified, reaching a minimum during the weekends (P=.000005). There was an inverse association between AT and the number of hospital admissions due to AMI and a direct association with particulate matter with a diameter smaller than 10 μm.
CONCLUSIONS	Hospital admissions for AMI followed a downward trend between 2007 and 2015. Mortality associated with admissions due to this diagnosis has decreased. Predictive factors for this disease were AT and particulate matter with a diameter smaller than 10 μm.

표-634. PubMed 논문번호 30206085의 내용 요약

구분	내용
PubMed ID	30206085
TITLE	Are noise and air pollution related to the incidence of dement study in London, England.
JOURNAL	BMJ open: 10.1136/bmjopen-2018-022404
AUTHORS	Carey Iain M, Anderson H Ross, Atkinson Richard W, Beeve Derek G, Strachan David P, Dajnak David, Gulliver John, Ke
OBJECTIVE	To investigate whether the incidence of dementia is related to of air and noise pollution in London.
DESIGN	Retrospective cohort study using primary care data.
SETTING	75 Greater London practices.
PARTICIPANTS	130 978 adults aged 50-79 years registered with their general January 2005, with no recorded history of dementia or care h
PRIMARY AND SECONDARY OUTCOME MEASURES	A first recorded diagnosis of dementia and, where specified, s Alzheimer's disease and vascular dementia during 2005-2013 annual concentrations during 2004 of nitrogen dioxide (NO2) matter with a median aerodynamic diameter ≤2.5 □m (PM2 (O3) were estimated at 20×20 m resolution from dispersion m intensity, distance from major road and night-time noise leve estimated at the postcode level. All exposure measures were li anonymously to clinical data via residential postcode. HRs fr were adjusted for age, sex, ethnicity, smoking and body mass further adjustments explored for area deprivation and comorb
RESULTS	2181 subjects (1.7%) received an incident diagnosis of dement mentioning Alzheimer's disease, 29% vascular dementia). The exposure response relationship between dementia and all mea pollution except O3, which was not readily explained by furth Adults living in areas with the highest fifth of NO2 concentra (〉41.5 □g/m3) versus the lowest fifth (〈31.9 □g/m3) were at dementia (HR=1.40, 95% CI 1.12 to 1.74). Increases in deme also observed with PM2.5, PM2.5 specifically from primary t only and Lnight, but only NO2 and PM2.5 remained statistic multipollutant models. Associations were more consistent for disease than vascular dementia.
CONCLUSIONS	We have found evidence of a positive association between resi air pollution across London and being diagnosed with dement unexplained by known confounding factors.

표-635. PubMed 논문번호 30208394의 내용 요약

구분	내용
PubMed ID	30208394
TITLE	Effect of Portable Air Filtration Systems on Personal Exposure to Fine Particulate Matter and Blood Pressure Among Residents in a Low-Income Senior Facility: A Randomized Clinical Trial.
JOURNAL	JAMA internal medicine: 10.1001/jamainternmed.2018.3308
AUTHORS	Morishita Masako, Adar Sara D, D'Souza Jennifer, Ziemba Rosemary A, Bard Robert L, Spino Catherine, Brook Robert D
Importance	Fine particulate matter (smaller than 2.5 μm) (PM2.5) air pollution is a major global risk factor for cardiovascular (CV) morbidity and mortality. Few studies have tested the benefits of portable air filtration systems in urban settings in the United States.
Objective	To investigate the effectiveness of air filtration at reducing personal exposures to PM2.5 and mitigating related CV health effects among older adults in a typical US urban location.
Design, Setting, and Participants	This randomized, double-blind crossover intervention study was conducted from October 21, 2014, through November 4, 2016, in a low-income senior residential building in Detroit, Michigan. Forty nonsmoking older adults were enrolled, with daily CV health outcome and PM2.5 exposure measurements.
Interventions	Participants were exposed to the following three 3-day scenarios separated by 1-week washout periods: unfiltered air (sham filtration), low-efficiency (LE) high-efficiency particulate arrestance (HEPA)-type filtered air, and high-efficiency (HE) true-HEPA filtered air using filtration systems in their bedroom and living room.
Main Outcomes and Measures	The primary outcome was brachial blood pressure (BP). Secondary outcomes included aortic hemodynamics, pulse-wave velocity, and heart rate variability. Exposures to PM2.5 were measured in the participants' residences and by personal monitoring.
Results	The 40 participants had a mean (SD) age of 67 (8) years (62% men). Personal PM2.5 exposures were significantly reduced by air filtration from a mean (SD) of 15.5 (10.9) μg/m3 with sham filtration to 10.9 (7.4) μg/m3 with LE fitration and 7.4 (3.3) μg/m3 with HE filtration. Compared with sham filtration, any filtration for 3 days decreased brachial systolic and diastolic BP by 3.2 mm Hg (95% CI, −6.1 to −0.2 mm Hg) and 1.5 mm Hg (95% CI, −3.3 to 0.2 mm Hg), respectively. A continuous decrease occurred in systolic and diastolic BP during the 3-day period of LE filtration, with a mean of 3.4 mm Hg (95% CI, −6.8 to −0.1 mm Hg) and 2.2 mm Hg (95% CI, −4.2 to −0.3 mm Hg), respectively. For HE filtration, systolic and diastolic BP decreased by 2.9 mm Hg (95% CI, −6.2 to 0.5 mm Hg) and 0.8 mm Hg (95% CI, −2.8 to 1.2 mm Hg), respectively. Most secondary outcomes were not significantly improved.
Conclusions and Relevance	Results of this study showed that short-term use of portable air filtration systems reduced personal PM2.5 exposures and systolic BP among older adults living in a typical US urban location. The use of these relatively inexpensive systems is potentially cardioprotective against PM2.5 exposures and warrants further research.
Trial Registration	ClinicalTrials.gov identifier: NCT03334565.

표-636. PubMed 논문번호 30214863의 내용 요약

구분	내용
PubMed ID	30214863
TITLE	Air pollution and telomere length: a systematic review of 12,058 subjects.
JOURNAL	Cardiovascular diagnosis and therapy: 10.21037/cdt.2018.06.05
AUTHORS	Zhao Bing, Vo Ha Q, Johnston Fay H, Negishi Kazuaki
Background	Over recent decades, adverse effects of ambient air pollution on the cardiovascular system have been clearly demonstrated. However, the underlying mechanisms are not fully elucidated. Air pollution may accelerates biological aging and thereby the susceptibility to cardiovascular diseases (CVDs). Telomeres are tandem repetitive DNA complexes that play a critical role in maintaining chromosome stability. There are, however, heterogeneities among the reported effects of air pollution on telomere. This study sought to evaluate the existing literature on the association between air pollution and telomere length (TL).
Methods	Two reviewers independently searched on electronic databases including PUBMED, EMBASE, SCOPUS, WEB OF SCIENCE and Ovid. The key terms were "air pollution" and "telomere" without language restriction. Articles relating to tobacco smoke were excluded.
Results	A total of 12,058 subjects from 25 articles remained for final review. All were observational studies: 14 cross-sectional, 6 cohort and 5 case-control studies. Nineteen (76%) assessed leukocyte telomere length (LTL) of which 15 found associations between air pollution and shorter TL, 2 with longer TL, 1 had mixed results, and a study of patients with type 2 diabetes found non-significant associations with TL. One found longer TL from saliva. The remaining studies were of placental cells, buccal cells or sperm and all reported shorter TL associated with air pollution. Particulate matter (PM) was investigated in 8 articles, and the remainder assessed black carbon (BC), benzene, lead, cadmium and polycyclic aromatic hydrocarbon (PAH). Geographically, 11 studies were conducted in Europe, with 10 in Asia and 4 in North America. While all followed Cawthon's protocol for TL assessment, discordance in the reporting formats did not allow us to perform a quantitative meta-analysis.
Conclusions	Most of the studies support the association of shorter TL with air pollution. Uniform reporting format would be warranted for future studies to estimate true effect size of air pollution on TL.

표-637. PubMed 논문번호 30216846의 내용 요약

구분	내용
PubMed ID	30216846
TITLE	Extracellular vesicle-enriched microRNAs interact in the association between long-term particulate matter and blood pressure in elderly men.
JOURNAL	Environmental research: 10.1016/j.envres.2018.09.002
AUTHORS	Rodosthenous Rodosthenis S, Kloog Itai, Colicino Elena, Zhong Jia, Herrera Luis A, Vokonas Pantel, Schwartz Joel, Baccarelli Andrea A, Prada Diddier
BACKGROUND	Several studies have shown that exposure to particulate matter (PM) may lead to increased systemic blood pressure, but the underlying biological mechanisms remain unknown. Emerging evidence shows that extracellular vesicle-enriched miRNAs (evmiRNAs) are associated with PM exposure and cardiovascular risk. In this study, we investigated the role of evmiRNAs in the association between PM and blood pressure, as well as their epigenetic regulation by DNA methylation.
METHODS	Participants (n = 22, men) were randomly selected from the Veterans Affairs Normative Aging Study (NAS). Long-term (1-year and 6-month average) PM2.5 exposure was estimated at 1 × 1-km resolution using spatio-temporal prediction models and BC was estimated using validated time varying land use regression models. We analyzed 31 evmiRNAs detected in ≥ 90% of all individuals and for statistical analysis, we used mixed effects models with random intercept adjusted for age, body mass index, smoking, C-reactive protein, platelets, and white blood cells.
RESULTS	We found that per each 2-standard deviations increase in 6-month PM2.5 ambient levels, there was an increase in 0.19 mm Hg (95% Confidence Interval [95%CI]: 0.11, 0.28 mmHg; $p < 0.001$) in systolic blood pressure (SBP). Per each 2-standard deviations increase in 1-year PM2.5 levels, there was an increase in 0.11 mm Hg (95% Confidence Interval [95% CI]: 0.03, 0.19 mmHg; $p = 0.012$) in SBP in older male individuals. We also found that both miR-199a/b ($\beta = 6.13$ mmHg; 95% CI: 0.87, 11.39; pinteraction = 0.07) and miR-223-3p ($\beta = 30.17$ mmHg; 95% CI: 11.96, 48.39 mmHg; pinteraction = 0.01) modified the association between 1-year PM2.5 and SBP. When exploring DNA methylation as a potential mechanism that could epigenetically regulate expression of evmiRNAs, we found that PM2.5 ambient levels were negatively associated with DNA methylation levels at CpG (cg23972892) near the enhancer region of miR-199a/b ($\beta = -13.11$; 95% CI: -17.70, -8.52; pBonferroni < 0.01), but not miR-223-3p.
CONCLUSIONS	Our findings suggest that expression of evmiRNAs may be regulated by DNA methylation in response to long-term PM2.5 ambient levels and modify the magnitude of association between PM2.5 and systolic blood pressure in older individuals.

표-638. PubMed 논문번호 30217927의 내용 요약

구분	내용
PubMed ID	30217927
TITLE	Long-term exposure to air pollutants from multiple sources and mortality in an industrial area: a cohort study.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2018-105059
AUTHORS	Bauleo Lisa, Bucci Simone, Antonucci Chiara, Sozzi Roberto, Davoli Marina, Forastiere Francesco, Ancona Carla, Bauleo Lisa, Bucci Simone, Antonucci Chiara, Sozzi Roberto, Davoli Marina, Forastiere Francesco, Ancona Carla
BACKGROUND AND AIMS	Residents near industrial areas are exposed to several toxins from various sources and the assessment of the health effects is difficult. The area of Civitavecchia (Italy) has several sources of environmental contamination with potential health effects. We evaluated the association between exposure to pollutants from multiple sources and mortality in a cohort of people living in the area.
METHODS	All residents of the area in 1996 were enrolled (from municipal registers) and followed until 2013. Long-term exposures to emissions from industrial sources (PM10) and traffic (NOx) at the residential addresses were assessed using a dispersion model. Residence close to the harbour was also considered. Cox survival analysis was conducted including a linear term for industrial PM10 and NOx exposure and a dichotomous variable to indicate residence within 500 m of the harbour. Age, sex, calendar period, occupation and area-based socioeconomic position (SEP) were considered (HRs, 95% CI).
RESULTS	71 362 people were enrolled (52% female, 43% low SEP) and 14 844 died during the follow-up. We found an association between industrial PM10 and mortality from non-accidental causes (HR=1.06, 95% CI 1.01 to 1.12), all cancers (HR=1.11, 95% CI 1.01 to 1.21) and cardiac diseases (HR=1.12, 95% CI 1.01 to 1.23). We also found an association between NOx exposure from traffic and mortality from all cancers (HR=1.13, 95% CI 1.01 to 1.26) and neurological diseases (HR=1.50, 95% CI 1.01 to 2.20). Living near the harbour was associated with higher mortality from lung cancer (HR=1.31, 95% CI 1.04 to 1.66) and neurological diseases (HR=1.51, 95% CI 1.05 to 2.18).
CONCLUSIONS	Estimated exposures to different pollution sources in this area were independently associated with several mortality outcomes while adjusting for occupation and socioeconomic status.

표-639. PubMed 논문번호 30223374의 내용 요약

구분	내용
PubMed ID	30223374
TITLE	Health impacts of the Southeast Asian haze problem - A time-stratified case crossover study of the relationship between ambient air pollution and sudden cardiac deaths in Singapore.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2018.04.070
AUTHORS	Ho Andrew Fu Wah, Wah Win, Earnest Arul, Ng Yih Yng, Xie Zhenjia, Shahidah Nur, Yap Susan, Pek Pin Pin, Liu Nan, Lam Sean Shao Wei, Ong Marcus Eng Hock
OBJECTIVES	To investigate the association between air pollution and out-of-hospital cardiac arrest (OHCA) incidence in Singapore.
DESIGN	A time-stratified case-crossover design study.
SETTING	OHCA incidences of all etiology in Singapore.
PARTICIPANTS	8589 OHCA incidences reported to Pan-Asian Resuscitation Outcomes Study (PAROS) registry in Singapore between 2010 and 2015.
MAIN OUTCOME MEASURES	A conditional Poisson regression model was applied to daily OHCA incidence that included potential confounders such as daily temperature, rainfall, wind speed, Pollutant Standards Index (PSI) and age. All models were adjusted for over-dispersion, autocorrelation and population at risk. We assessed the relationship with OHCA incidence and PSI in the entire cohort and in predetermined subgroups of demographic and clinical characteristics.
RESULTS	334 out of 8589 (3.89%) cases survived. Moderate (Risk ratio/RR = 1.1, 95% CI = 1.07-1.15) and unhealthy (RR =1.37, 95% CI = 1.2-1.56) levels of PSI showed significant association with increased OHCA occurrence. Sub-group analysis based on individual demographic and clinical features showed generally significant association between OHCA incidence and moderate/unhealthy PSI, except in age < 65, Malay and other ethnicity, traumatic arrests and history of heart disease and diabetes. The association was most pronounced among cases age > 65, male, Indian and non-traumatic. Each increment of 30 unit in PSI on the same day and previous 1-5 days was significantly associated with 5.8-8.1% increased risk of OHCA ($p < 0.001$).
CONCLUSIONS	We found a transient effect of short-term air pollution on OHCA incidence after adjusting for meteorological indicators and individual characteristics. These finding have public health implications for prevention of OHCA and emergency health services during haze.

표-640. PubMed 논문번호 30284325의 내용 요약

구분	내용
PubMed ID	30284325
TITLE	Congenital malformations are associated with secondhand smoke among nonsmoking women: A meta-analysis.
JOURNAL	Birth (Berkeley, Calif.): 10.1111/birt.12401
AUTHORS	Zheng Zan, Xie GuoHong, Yang Tubao, Qin Jiabi, Zheng Zan, Xie GuoHong, Yang Tubao, Qin Jiabi
BACKGROUND	The association between active maternal smoking and congenital malformations is well established, but little is known about the association between secondhand smoke and congenital malformations. Moreover, studies regarding the association between congenital malformations and secondhand smoke have not yielded consistent results.
METHODS	In July 2018, we searched PubMed, EMBASE, and China Biology Medicine databases for observational studies characterizing the relationship between secondhand smoke and congenital malformations of offspring in nonsmoking women. Two reviewers independently decided on whether a study should be included, did data extraction, and assessed study quality. Pooled risks with 95% confidence intervals were calculated using either the fixed-effects models or random-effects models. Further subgroup analyses and sensitivity analyses were performed to explore the potential source of heterogeneity and to examine the robustness of risk estimates.
RESULTS	Thirty-three studies with a total of 31 944 cases and 32 335 controls were included. Secondhand smoke exposure was associated with an increased risk of congenital malformations (odds ratio = 1.92; 95% confidence interval 1.61-2.30). Secondhand smoke was correlated with significantly increased risk for digestive system (1.17 [1.05-1.32]), nervous system (1.74 [1.33-2.29]), and cardiovascular system (2.10 [1.32-3.35]) malformations and for oral clefts (1.87 [1.47-2.39]).
CONCLUSIONS	Secondhand smoke exposure increases the risk for overall and several organ-system malformations. These findings highlight the necessity of improving community awareness to prevent secondhand smoke exposure during the preconception and conception periods.

표-641. PubMed 논문번호 30286080의 내용 요약

구분	내용
PubMed ID	30286080
TITLE	Association between ambient air pollution and daily hospital admissions for ischemic stroke: A nationwide time-series analysis.
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1002668
AUTHORS	Tian Yaohua, Liu Hui, Zhao Zuolin, Xiang Xiao, Li Man, Juan Juan, Song Jing, Cao Yaying, Wang Xiaowen, Chen Libo, Wei Chen, Hu Yonghua, Gao Pei
BACKGROUND	Evidence of the short-term effects of ambient air pollution on the risk of ischemic stroke in low- and middle-income countries is limited and inconsistent. We aimed to examine the associations between air pollution and daily hospital admissions for ischemic stroke in China.
METHODS AND FINDINGS	We identified hospital admissions for ischemic stroke in 2014-2016 from the national database covering up to 0.28 billion people who received Urban Employee Basic Medical Insurance (UEBMI) in China. We examined the associations between air pollution and daily ischemic stroke admission using a two-stage method. Poisson time-series regression models were firstly fitted to estimate the effects of air pollution in each city. Random-effects meta-analyses were then conducted to combine the estimates. Meta-regression models were applied to explore potential effect modifiers. More than 2 million hospital admissions for ischemic stroke were identified in 172 cities in China. In single-pollutant models, increases of 10 μg/m3 in particulate matter with aerodynamic diameter <2.5 μm (PM2.5), sulfur dioxide (SO2), nitrogen dioxide (NO2), and ozone (O3) and 1 mg/m3 in carbon monoxide (CO) concentrations were associated with 0.34% (95% confidence interval [CI], 0.20%-0.48%), 1.37% (1.05%-1.70%), 1.82% (1.45%-2.19%), 0.01% (-0.14%-0.16%), and 3.24% (2.05%-4.43%) increases in hospital admissions for ischemic stroke on the same day, respectively. SO2 and NO2 associations remained significant in two-pollutant models, but not PM2.5 and CO associations. The effect estimates were greater in cities with lower air pollutant levels and higher air temperatures, as well as in elderly subgroups. The main limitation of the present study was the unavailability of data on individual exposure to ambient air pollution.
CONCLUSIONS	As the first national study in China to systematically examine the associations between short-term exposure to ambient air pollution and ischemic stroke, our findings indicate that transient increase in air pollution levels may increase the risk of ischemic stroke, which may have significant public health implications for the reduction of ischemic stroke burden in China.

표-642. PubMed 논문번호 30292144의 내용 요약

구분	내용
PubMed ID	30292144
TITLE	Mortality burden attributable to PM〈sub〉1〈/sub〉 in Zhejiang province, China.
JOURNAL	Environment international: 10.1016/j.envint.2018.09.033
AUTHORS	Hu Kejia, Guo Yuming, Hu Deyun, Du Rongguang, Yang Xuchao, Zhong Jieming, Fei Fangrong, Chen Feng, Chen Gongbo, Zhao Qi, Yang Jun, Zhang Yunquan, Chen Qian, Ye Tingting, Li Shanshan, Qi Jiaguo
BACKGROUND	Limited evidence is available on the health effects of particulate matter with an aerodynamic diameter of 〈1 μm (PM1), mainly due to the lack of its ground measurement worldwide.
OBJECTIVES	To identify and examine the mortality risks and mortality burdens associated with PM1, PM2.5, and PM10 in Zhejiang province, China.
METHODS	We collected daily data regarding all-cause (stratified by age and gender), cardiovascular, stroke, respiratory, and chronic obstructive pulmonary disease (COPD) mortality, and PM1, PM2.5, and PM10, from 11 cities in Zhejiang province, China during 2013 and 2017. We used a quasi-Poisson regression model to estimate city-specific associations between mortality and PM concentrations. Then we used a random-effect meta-analysis to pool the provincial estimates. To show the mortality burdens of PM1, PM2.5, and PM10, we calculated the mortality fractions and deaths attributable to these PMs.
RESULTS	Daily concentrations of PM1, PM2.5, and PM10 ranged between 0-199 μg/m3, 0-218 μg/m3, and 0-254 μg/m3, respectively; Mortality effects were significant in lag 0-2 days. The relative risks for all-cause mortality were 1.0064 (95% CI: 1.0034, 1.0094), 1.0061 (95% CI: 1.0034, 1.0089), and 1.0060 (95% CI: 1.0038, 1.0083) associated with a 10 μg/m3 increase in PM1, PM2.5, and PM10, respectively. Age- and gender-stratified analysis shows that elderly people (aged 65+) and females are more sensitive to PMs. The mortality fractions of all-cause mortality were estimated to be 2.39% (95% CI: 1.28, 3.48) attributable to PM1, 2.53% (95% CI: 1.42, 3.63) attributable to PM2.5, and 3.08% (95% CI: 1.95, 4.19) attributable to PM10. The ratios of attributable cause-specific deaths for PM1/PM2.5, PM1/PM10, and PM2.5/PM10 were higher than the ratios of their respective concentrations.
CONCLUSIONS	PM1, PM2.5 and PM10 are risk factors of all-cause, cardiovascular, stroke, respiratory, and COPD mortality. PM1 accounts for the vast majority of short-term PM2.5- and PM10-induced mortality. Our analyses support the notion that smaller size fractions of PM have a more toxic mortality impacts, which suggests to develop strategies to prevent and control PM1 in China, such as to foster strict regulations for automobile and industrial emissions.

표-643. PubMed 논문번호 30312757의 내용 요약

구분	내용
PubMed ID	30312757
TITLE	Impact of dense "smoke" detected on transesophageal echocardiography on stroke risk in patients with atrial fibrillation undergoing catheter ablation.
JOURNAL	Heart rhythm: 10.1016/j.hrthm.2018.10.004
AUTHORS	Gedikli □mer, Mohanty Sanghamitra, Trivedi Chintan, Gianni Carola, Chen Qiong, Della Rocca Domenico Giovanni, Burkhardt J David, Sanchez Javier E, Hranitzky Patrick, Gallinghouse G Joseph, Al-Ahmad Amin, Horton Rodney, Di Biase Luigi, Natale Andrea, Gedikli □mer, Mohanty Sanghamitra, Trivedi Chintan, Gianni Carola, Chen Qiong, Della Rocca Domenico Giovanni, Burkhardt J David, Sanchez Javier E, Hranitzky Patrick, Gallinghouse G Joseph, Al-Ahmad Amin, Horton Rodney, Di Biase Luigi, Natale Andrea
BACKGROUND	Spontaneous echocardiographic contrast ("smoke") within the left atrial cavity on transesophageal echocardiography (TEE) suggests low blood flow velocities in the heart that may lead to thromboembolic (TE) events.
OBJECTIVE	The purpose of this study was to evaluate the risk of TE events in the periprocedural period and at long-term follow-up in atrial fibrillation (AF) patients having dense smoke on preprocedural TEE.
METHODS	A total of 2511 patients undergoing AF ablation were included in this analysis. They were classified as group 1 (dense smoke detected on TEE at baseline; n = 234) and group 2 (no smoke on baseline TEE; n = 2277). Patients were followed up for TE events, which included both stroke and transient ischemic attacks (TIAs). In order to attenuate the observed imbalance in baseline covariates between the study groups, a propensity score matching technique was used (covariates were age, sex, AF type, diabetes, and CHADS2VASc score).
RESULTS	In the periprocedural period, no TE events were reported in group 1 and 3 events (0.13%) were reported in group 2. At follow-up of 6.62 ± 2.01 years, 6 (2.6%) TE complications (2 TIA, 4 stroke) occurred in group 1 and 16 (0.70%) TE complications (6 TIA, 10 stroke) in group 2 (P = .004). In the propensity-matched population, 6 (2.56%) TE complications occurred in group 1 and 1 (0.2%) in group 2 (P = .007).
CONCLUSION	In our study population, the presence of dense left atrial smoke did not show any correlation with periprocedural TE events in patients undergoing catheter ablation with uninterrupted anticoagulation. However, significant association was observed with late stroke/TIA, irrespective of CHA2DS2-VASc score.

표-644. PubMed 논문번호 30340847의 내용 요약

구분	내용
PubMed ID	30340847
TITLE	Forecasting life expectancy, years of life lost, and all-cause and cause-specific mortality for 250 causes of death: reference and alternative scenarios for 2016-40 for 195 countries and territories.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(18)31694-5
AUTHORS	Foreman Kyle J, Marquez Neal, Dolgert Andrew, Fukutaki Kai, Fullman Nancy, McGaughey Madeline, Pletcher Martin A, Smith Amanda E, Tang Kendrick, Yuan Chun-Wei, Brown Jonathan C, Friedman Joseph, He Jiawei, Heuton Kyle R, Holmberg Mollie, Patel Disha J, Reidy Patrick, Carter Austin, Cercy Kelly, Chapin Abigail, Douwes-Schultz Dirk, Frank Tahvi, Goettsch Falko, Liu Patrick Y, Nandakumar Vishnu, Reitsma Marissa B, Reuter Vince, Sadat Nafis, Sorensen Reed J D, Srinivasan Vinay, Updike Rachel L, York Hunter, Lopez Alan D, Lozano Rafael, Lim Stephen S, Mokdad Ali H, Vollset Stein Emil, Murray Christopher J L
BACKGROUND	Understanding potential trajectories in health and drivers of health is crucial to guiding long-term investments and policy implementation. Past work on forecasting has provided an incomplete landscape of future health scenarios, highlighting a need for a more robust modelling platform from which policy options and potential health trajectories can be assessed. This study provides a novel approach to modelling life expectancy, all-cause mortality and cause of death forecasts -and alternative future scenarios-for 250 causes of death from 2016 to 2040 in 195 countries and territories.
METHODS	We modelled 250 causes and cause groups organised by the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) hierarchical cause structure, using GBD 2016 estimates from 1990-2016, to generate predictions for 2017-40. Our modelling framework used data from the GBD 2016 study to systematically account for the relationships between risk factors and health outcomes for 79 independent drivers of health. We developed a three-component model of cause-specific mortality: a component due to changes in risk factors and select interventions; the underlying mortality rate for each cause that is a function of income per capita, educational attainment, and total fertility rate under 25 years and time; and an autoregressive integrated moving average model for unexplained changes correlated with time. We assessed the performance by fitting models with data from 1990-2006 and using these to forecast for 2007-16. Our final model used for generating forecasts and alternative scenarios was fitted to data from 1990-2016. We used this model for 195 countries and territories to generate a reference scenario or forecast through 2040 for each measure by location. Additionally, we generated better health and worse health scenarios based on the 85th and 15th percentiles, respectively, of annualised rates of change across location-years for all the GBD risk factors, income per person, educational attainment, select intervention coverage, and total fertility rate under 25 years in the past. We used the model to generate all-cause age-sex specific mortality, life expectancy, and years of life lost (YLLs) for 250 causes. Scenarios for fertility were also generated and used in a cohort component model to generate population scenarios. For each reference forecast, better health, and worse health scenarios, we generated estimates of mortality and YLLs attributable to each risk factor in the future.
FINDINGS	Globally, most independent drivers of health were forecast to improve by 2040, but 36 were forecast to worsen. As shown by the better health scenarios, greater progress might be possible, yet for some drivers such as high body-mass index (BMI), their toll will rise in the absence of intervention. We

표-645. PubMed 논문번호 30357335의 내용 요약

구분	내용
PubMed ID	30357335
TITLE	A systematic analysis of mutual effects of transportation noise and air pollution exposure on myocardial infarction mortality: a nationwide cohort study in Switzerland.
JOURNAL	European heart journal: 10.1093/eurheartj/ehy650
AUTHORS	H□ritier Harris, Vienneau Danielle, Foraster Maria, Eze Ikenna C, Schaffner Emmanuel, de Hoogh Kees, Thiesse Laurie, Rudzik Franziska, Habermacher Manuel, K□pfli Micha, Pieren Reto, Brink Mark, Cajochen Christian, Wunderli Jean Marc, Probst-Hensch Nicole, R□□sli Martin
AIMS	The present study aimed to disentangle the risk of the three major transportation noise sources-road, railway, and aircraft traffic-and the air pollutants NO2 and PM2.5 on myocardial infarction (MI) mortality in Switzerland based on high quality/fine resolution exposure modelling.
METHODS AND RESULTS	We modelled long-term exposure to outdoor road traffic, railway, and aircraft noise levels, as well as NO2 and PM2.5 concentration for each address of the 4.40 million adults (>30 years) in the Swiss National Cohort (SNC). We investigated the association between transportation noise/air pollution exposure and death due to MI during the follow-up period 2000-08, by adjusting noise [Lden(Road), Lden(Railway), and Lden(Air)] estimates for NO2 and/or PM2.5 and vice versa by multipollutant Cox regression models considering potential confounders. Adjusting noise risk estimates of MI for NO2 and/or PM2.5 did not change the hazard ratios (HRs) per 10 dB increase in road traffic (without air pollution: 1.032, 95% CI: 1.014-1.051, adjusted for NO2 and PM2.5: 1.034, 95% CI: 1.014-1.055), railway traffic (1.020, 95% CI: 1.007-1.033 vs. 1.020, 95% CI: 1.007-1.033), and aircraft traffic noise (1.025, 95% CI: 1.006-1.045 vs. 1.025, 95% CI: 1.005-1.046). Conversely, noise adjusted HRs for air pollutants were lower than corresponding estimates without noise adjustment. Hazard ratio per 10 $\mu g/m^3$ increase with and without noise adjustment were 1.024 (1.005-1.043) vs. 0.990 (0.965-1.016) for NO2 and 1.054 (1.013-1.093) vs. 1.019 (0.971-1.071) for PM2.5.
CONCLUSION	Our study suggests that transportation noise is associated with MI mortality, independent from air pollution. Air pollution studies not adequately adjusting for transportation noise exposure may overestimate the cardiovascular disease burden of air pollution.

표-646. PubMed 논문번호 30369511의 내용 요약

구분	내용
PubMed ID	30369511
TITLE	Japanese Nationwide Study on the Association Between Short-term Exposure to Particulate Matter and Mortality.
JOURNAL	Journal of epidemiology: 10.2188/jea.JE20180122
AUTHORS	Michikawa Takehiro, Ueda Kayo, Takami Akinori, Sugata Seiji, Yoshino Ayako, Nitta Hiroshi, Yamazaki Shin, Michikawa Takehiro, Ueda Kayo, Takami Akinori, Sugata Seiji, Yoshino Ayako, Nitta Hiroshi, Yamazaki Shin
BACKGROUND	From around 2012, the use of automated equipment for fine particulate matter (PM2.5) measurement with equivalence to a reference method has become popular nationwide in Japan. This enabled us to perform a national health effect assessment employing PM2.5 concentrations based on the standardized measurement method. We evaluated the association between non-accidental mortality and short-term exposure to PM2.5 and coarse particulate matter (PM), with the latter estimated as the difference between suspended particulate matter and PM2.5, for the fiscal years 2012-2014.
METHODS	This was a time-stratified case-crossover study in 100 highly-populated Japanese cities. Mortality data was obtained from the Ministry of Health, Labour and Welfare. City-specific estimates of PM-mortality association were calculated by applying a conditional logistic regression analysis, and combined with a random-effects meta-analysis.
RESULTS	The respective averages of daily mean concentration were 14.6 □g/m3 for PM2.5 and 6.4 □g/m3 for coarse PM. A 10 □g/m3 increase in PM2.5 concentrations for the average of the day of death and the previous day was associated with an increase of 1.3% (95% confidence interval (CI), 0.9-1.6%) in total non-accidental mortality. For cause-specific mortality, PM2.5 was positively associated with cardiovascular and respiratory mortality. After adjustment for PM2.5, we observed a 1.4% (95% CI, 0.2-2.6%) increase in total mortality with a 10 □g/m3 increase in coarse PM.
CONCLUSION	The study revealed that short-term exposure to PM2.5 had adverse effects on total non-accidental, cardiovascular, and respiratory mortality in Japan. Coarse PM exposure also increased the risk of total mortality.

표-647. PubMed 논문번호 30392394의 내용 요약

구분	내용
PubMed ID	30392394
TITLE	Long-Term Exposure to Ambient Fine Particulate Matter and Chronic Kidney Disease: A Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP3304
AUTHORS	Chan Ta-Chien, Zhang Zilong, Lin Bo-Cheng, Lin Changqing, Deng Han-Bing, Chuang Yuan Chieh, Chan Jimmy W M, Jiang Wun Kai, Tam Tony, Chang Ly-Yun, Hoek Gerard, Lau Alexis K H, Lao Xiang Qian
BACKGROUND	Chronic kidney disease (CKD) is a serious global public health challenge, but there is limited information on the connection between air pollution and risk of CKD.
OBJECTIVE	The aim of this study was to investigate the association between long-term exposure to particulate matter (PM) with an aerodynamic diameter of less than [Formula: see text] ([Formula: see text]) and the development of CKD in a large cohort.
METHODS	A total of 100,629 nonCKD Taiwanese residents age 20 y or above were included in this study between 2001 and 2014. Ambient [Formula: see text] concentration was estimated at each participant's address using a satellite-based spatiotemporal model. Incident CKD cases were identified by an estimated glomerular filtration rate (eGFR) of less than [Formula: see text]. We collected information on a wide range of potential confounders/modifiers during the medical examinations. Cox proportional hazard regression was applied to calculate hazard ratios (HRs).
RESULTS	During the follow-up, 4,046 incident CKD cases were identified, and the incidence rate was 6.24 per 1,000 person-years. In contrast with participants with the first quintile exposure of [Formula: see text], participants with the fourth and fifth quintiles exposure of [Formula: see text] had increased risk of CKD development, adjusting for age, sex, educational level, smoking, drinking, body mass index, systolic blood pressure, fasting glucose, total cholesterol, and self-reported heart disease or stroke, with an HR [95% confidence interval (CI)] of 1.11 (1.02, 1.22) and 1.15 (1.05, 1.26), respectively. A significant concentration-response trend was observed ([Formula: see text]). Every [Formula: see text] increment in the [Formula: see text] concentration was associated with a 6% higher risk of developing CKD (HR: 1.06, 95% CI: 1.02, 1.10). Sensitivity and stratified analyses yielded similar results.
CONCLUSIONS	Long-term exposure to ambient [Formula: see text] was associated with an increased risk of CKD development. Our findings reinforce the urgency to develop global strategies of air pollution reduction to prevent CKD. https://doi.org/10.1289/EHP3304.

표-648. PubMed 논문번호 30392401의 내용 요약

구분	내용
PubMed ID	30392401
TITLE	Longitudinal Analysis of Long-Term Air Pollution Levels and Blood Pressure: A Cautionary Tale from the Multi-Ethnic Study of Atherosclerosis.
JOURNAL	Environmental health perspectives: 10.1289/EHP2966
AUTHORS	Adar Sara D, Chen Yeh-Hsin, D'Souza Jennifer C, O'Neill Marie S, Szpiro Adam A, Auchincloss Amy H, Park Sung Kyun, Daviglus Martha L, Diez Roux Ana V, Kaufman Joel D
BACKGROUND	Air pollution exposures are hypothesized to impact blood pressure, yet few longitudinal studies exist, their findings are inconsistent, and different adjustments have been made for potentially distinct confounding by calendar time and age.
OBJECTIVE	We aimed to investigate the associations of long- and short-term [Formula: see text] and [Formula: see text] concentrations with systolic and diastolic blood pressures and incident hypertension while also accounting for potential confounding by age and time.
METHODS	Between 2000 and 2012, Multi-Ethnic Study of Atherosclerosis participants were measured for systolic and diastolic blood pressure at five exams. We estimated annual average and daily [Formula: see text] and [Formula: see text] concentrations for 6,569 participants using spatiotemporal models and measurements, respectively. Associations of exposures with blood pressure corrected for medication were studied using mixed-effects models. Incident hypertension was examined with Cox regression. We adjusted all models for sex, race/ethnicity, socioeconomic status, smoking, physical activity, diet, season, and site. We compared associations from models adjusting for time-varying age with those that adjusted for both time-varying age and calendar time.
RESULTS	We observed decreases in pollution and blood pressures (adjusted for age and medication) over time. Strong, positive associations of long- and short-term exposures with blood pressure were found only in models with adjustment for time-varying age but not adjustment for both time-varying age and calendar time. For example, [Formula: see text] higher annual average [Formula: see text] concentrations were associated with 2.7 (95% CI: 1.5, 4.0) and [Formula: see text] (95% CI: [Formula: see text] 1.0) mmHg in systolic blood pressure with and without additional adjustment for time, respectively. Associations with incident hypertension were similarly weakened by additional adjustment for time. Sensitivity analyses indicated that air pollution did not likely cause the temporal trends in blood pressure.
CONCLUSIONS	In contrast to experimental evidence, we found no associations between long- or short-term exposures to air pollution and blood pressure after accounting for both time-varying age and calendar time. This research suggests that careful consideration of both age and time is needed in longitudinal studies with trending exposures. https://doi.org/10.1289/EHP2966.

표-649. PubMed 논문번호 30408890의 내용 요약

구분	내용
PubMed ID	30408890
TITLE	Assessment of impact of traffic-related air pollution on morbidity and mortality in Copenhagen Municipality and the health gain of reduced exposure.
JOURNAL	Environment international: 10.1016/j.envint.2018.09.050
AUTHORS	Brønnum-Hansen Henrik, Bender Anne Mette, Andersen Zorana Jovanovic, Sørensen Jan, Bønløkke Jakob Hjort, Boshuizen Hendriek, Becker Thomas, Diderichsen Finn, Loft Steffen
BACKGROUND	Health impact assessment (HIA) of exposure to air pollution is commonly based on city level (fine) particle concentration and may underestimate health consequences of changing local traffic. Exposure to traffic-related air pollution can be assessed at a high resolution by modelling levels of nitrogen dioxide (NO2), which together with ultrafine particles mainly originate from diesel-powered vehicles in urban areas. The purpose of this study was to estimate the health benefits of reduced exposure to vehicle emissions assessed as NO2 at the residence among the citizens of Copenhagen Municipality, Denmark.
METHODS	We utilized residential NO2 concentrations modelled by use of chemistry transport models to calculate contributions from emission sources to air pollution. The DYNAMO-HIA model was applied to the population of Copenhagen Municipality by using NO2 concentration estimates combined with demographic data and data from nationwide registers on incidence and prevalence of selected diseases, cause specific mortality, and total mortality of the population of Copenhagen. We used exposure-response functions linking NO2 concentration estimates at the residential address with the risk of diabetes, cardiovascular diseases, and respiratory diseases derived from a large Danish cohort study with the majority of subjects residing in Copenhagen between 1971 and 2010. Different scenarios were modelled to estimate the dynamic impact of NO2 exposure on related diseases and the potential health benefits of lowering the NO2 level in the Copenhagen Municipality.
RESULTS	The annual mean NO2 concentration was 19.6 μg/m3 and for 70% of the population the range of exposure was between 15 and 21 μg/m3. If NO2 exposure was reduced to the annual mean rural level of 6 μg/m3, life expectancy in 2040 would increase by one year. The greatest gain in disease-free life expectancy would be lifetime without ischemic heart disease (1.4 years), chronic obstructive pulmonary disease (1.5 years for men and 1.6 years for women), and asthma (1.3 years for men and 1.5 years for women). Lowering NO2 exposure by 20% would increase disease-free life expectancy for the different diseases by 0.3-0.5 years. Using gender specific relative risks affected the results.
CONCLUSIONS	Reducing the NO2 exposure by controlling traffic-related air pollution reduces the occurrence of some of the most prevalent chronic diseases and increases life expectancy. Such health benefits can be quantified by DYNAMO-HIA in a high resolution exposure modelling. This paper demonstrates how traffic planners can assess health benefits from reduced levels of traffic-related air pollution.

표-650. PubMed 논문번호 30412999의 내용 요약

구분	내용
PubMed ID	30412999
TITLE	Long-Term Effect of Outdoor Air Pollution on Mortality and Morbidity: A 12-Year Follow-Up Study for Metropolitan France.
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph15112487
AUTHORS	Sanyal Shreosi, Rochereau Thierry, Maesano Cara Nichole, Com-Ruelle Laure, Annesi-Maesano Isabella
BACKGROUND	Short-term effects of air pollution are documented more than long-term effects.
OBJECTIVE	We investigated 12-year impacts of ambient air pollutants on cardiovascular and respiratory morbidity and mortality at the departmental level in metropolitan France.
METHODS	Daily air pollution data at 2-km resolution, including concentrations of particulate matter of 10 □m or 2.5 □m in diameter or less (PM10 and PM2.5), nitrogen dioxide (NO_2), and ozone (O_3), were accrued from the CHIMERE database for 1999 and 2000. Simultaneously, morbidity (hospitalizations) and mortality data were collected in 2012 using the ESPS (Enqu□te Sant□ et Protection Sociale/Health, Health Care and Insurance Survey) survey data and the CepiDc (Centre d'□pid□miologie sur les Causes M□dicales de D□c□s/French Epidemiology Centre on Medical Causes of Death) database. Based on Poisson regression analyses, the long-term effect was estimated. A higher risk of all-cause mortality was observed using C□piDc database, with a relative risk of 1.024 (95% CI: 1.022, 1.026) and 1.029 (95% CI: 1.027, 1.031) for a 10 □g/m^3 increase in PM2.5 and PM10, respectively. Mortality due to cardiovascular and respiratory diseases likewise exhibited long-term associations with both PM2.5 and PM10. Using ESPS survey data, a significant risk was observed for both PM2.5 and PM10 in all-cause mortality and all-cause morbidity. Although a risk for higher all-cause mortality and morbidity was also present for NO_2, the cause-specific relative risk due to NO_2 was found to be lesser, as compared to PM. Nevertheless, cardiovascular and respiratory morbidity were related to NO_2, along with PM2.5 and PM10. However, the health effect of O_3 was seen to be substantially lower in comparison to the other pollutants.
CONCLUSION	Our study confirmed that PM has a long-term impact on mortality and morbidity. Exposure to NO_2 and O_3 could also lead to increased health risks.

표-651. PubMed 논문번호 30418257의 내용 요약

구분	내용
PubMed ID	30418257
TITLE	Cardiovascular Diseases and Long-term Self-reported Exposure to Pollution: RESULTS OF A NATIONAL EPIDEMIOLOGICAL STUDY IN LEBANON.
JOURNAL	Journal of cardiopulmonary rehabilitation and prevention: 10.1097/HCR.0000000000000378
AUTHORS	Salameh Pascale, Zeidan Rouba Karen, Hallit Souheil, Farah Rita, Chahine Mirna, Asmar Roland, Hosseini Hassan
BACKGROUND	Cardiovascular diseases (CVDs) are linked to high mortality and morbidity, particularly in developing countries. Some studies have linked indoor and outdoor pollution to CVD, but results are inconsistent. Our objective was to assess this association in Lebanon, a Middle Eastern country.
METHODS	A national cross-sectional study was conducted across Lebanon. CVD prevalence, which included prevalent ischemic heart and cerebrovascular diseases, was assessed. Moreover, in addition to self-reported items of pollution exposure, we assessed potential predictors of CVD, including sociodemographic characteristics, self-reported health information, and biological measurements.
RESULTS	We assessed the dose-effect relationship of pollution items in relation with CVD. Self-reported indoor and outdoor pollution exposures were associated with CVD, with or without taking biological values into account. Moreover, we found a dose-effect relationship of exposure with risk of disease (44% increase in risk of CVD for every additional pollution exposure item), after adjustment for sociodemographic and biological characteristics.
CONCLUSION	Although additional studies would be necessary to confirm these findings, interventions should start to sensitize the population about the effect of pollution on chronic diseases and the work of reducing pollution and improving air quality should be implemented to decrease the disease burden on the population and health system.

표-652. PubMed 논문번호 30446244의 내용 요약

구분	내용
PubMed ID	30446244
TITLE	Association of long-term PM〈sub〉2.5〈/sub〉 exposure with traditional and novel lipid measures related to cardiovascular disease risk.
JOURNAL	Environment international: 10.1016/j.envint.2018.11.001
AUTHORS	McGuinn Laura A, Schneider Alexandra, McGarrah Robert W, Ward-Caviness Cavin, Neas Lucas M, Di Qian, Schwartz Joel, Hauser Elizabeth R, Kraus William E, Cascio Wayne E, Diaz-Sanchez David, Devlin Robert B, McGuinn Laura A, Schneider Alexandra, McGarrah Robert W, Ward-Caviness Cavin, Neas Lucas M, Di Qian, Schwartz Joel, Hauser Elizabeth R, Kraus William E, Cascio Wayne E, Diaz-Sanchez David, Devlin Robert B
BACKGROUND	Fine particulate matter (PM2.5) exposure is associated with increased morbidity and mortality, particularly for cardiovascular disease. The association between long-term exposure to PM2.5 and measures of lipoprotein subfractions remains unclear. Therefore, we examined associations between long-term PM2.5 exposure and traditional and novel lipoprotein measures in a cardiac catheterization cohort in North Carolina.
METHODS	This cross-sectional study included 6587 patients who had visited Duke University for a cardiac catheterization between 2001 and 2010 and resided in North Carolina. We used estimates of daily PM2.5 concentrations on a 1 km-grid based on satellite measurements. PM2.5 predictions were matched to the address of each patient and averaged for the year prior to catheterization date. Serum lipids included HDL, LDL, and triglyceride-rich particle, and apolipoprotein B concentrations (HDL-P, LDL-P, TRL-P, and apoB, respectively). Linear and quantile regression models were used to estimate change in lipoprotein levels with each μg/m3 increase in annual average PM2.5. Models were adjusted for age, sex, race/ethnicity, history of smoking, area-level education, urban/rural status, body mass index, and diabetes.
RESULTS	For a 1-μg/m3 increment in PM2.5 exposure, we observed increases in total and small LDL-P, LDL-C, TRL-P, apoB, total cholesterol, and triglycerides. The percent change from the mean outcome level was 2.00% (95% CI: 1.38%, 2.64%) for total LDL-P and 2.25% (95% CI: 1.43%, 3.06%) for small LDL-P.
CONCLUSION	Among this sample of cardiac catheterization patients residing in North Carolina, long-term PM2.5 exposure was associated with increases in several lipoprotein concentrations. This abstract does not necessarily reflect U.S. EPA policy.

표-653. PubMed 논문번호 30447258의 내용 요약

구분	내용
PubMed ID	30447258
TITLE	VCAM-1-mediated neutrophil infiltration exacerbates ambient fine particle-induced lung injury.
JOURNAL	Toxicology letters: 10.1016/j.toxlet.2018.11.002
AUTHORS	Cui Anfeng, Xiang Meng, Xu Ming, Lu Peng, Wang Shun, Zou Yajuan, Qiao Ke, Jin Chengyu, Li Yijun, Lu Meng, Chen Alex F, Chen Sifeng, Cui Anfeng, Xiang Meng, Xu Ming, Lu Peng, Wang Shun, Zou Yajuan, Qiao Ke, Jin Chengyu, Li Yijun, Lu Meng, Chen Alex F, Chen Sifeng
BACKGROUND	Fine ambient particle matter (PM2.5) induces inflammatory lung injury; however, whether intratracheal administration of PM2.5 increases pulmonary polymorphonuclear leukocyte (PMN) infiltration, the mechanism of infiltration, and if these cells exacerbate PM2.5-induced lung injury are unknown.
METHODS	Using 32,704 subjects, the association between blood PMNs and ambient PM2.5 levels on the previous day was retrospectively analyzed. Neutropenia was achieved by injecting mice with PMN-specific antibodies. Inhibition of PMN infiltration was achieved by pretreating PMNs with soluble vascular cell adhesion molecule-1 (sVCAM-1). The effects of PMNs on PM2.5-induced lung injury and endothelial dysfunction were observed.
RESULT	Short-term PM2.5 (〉 75 μg/m3 air) exposure increased the PMN/white blood cell ratio and the PMN count in human peripheral blood observed during routine examination. A significant number of PM2.5-treated PMNs was able to bind sVCAM-1. In mice, intratracheally-instilled PM2.5 deposited in the alveolar space and endothelial cells, which caused significant lung edema, morphological disorder, increased permeability of the endothelial-alveolar epithelial barrier, and PMN infiltration with increased VCAM-1 expression. Depletion of circulatory PMNs inhibited these adverse effects. Replenishment of untreated PMNs, but not those pretreated with soluble VCAM-1, restored lung injury. In vitro, PM2.5 increased VCAM-1 expression and endothelial and epithelial monolayer permeability, and promoted PMN adhesion to, chemotaxis toward, and migration across these monolayers. PMNs, but not those pretreated with soluble VCAM-1, exacerbated these effects.
CONCLUSION	VCAM-1-mediated PMN infiltration was essential for a detrimental cycle of PM2.5-induced inflammation and lung injury. Results suggest that drugs that inhibit PMN function might prevent acute deterioration of chronic pulmonary and cardiovascular diseases triggered by PM2.5.

표-654. PubMed 논문번호 30477822의 내용 요약

구분	내용
PubMed ID	30477822
TITLE	Prenatal particulate air pollution exposure and cord blood homocysteine in newborns: Results from the ENVIRONAGE birth cohort.
JOURNAL	Environmental research: 10.1016/j.envres.2018.08.032
AUTHORS	Hogervorst Janneke G F, Madhloum Narjes, Saenen Nelly D, Janssen Bram G, Penders Joris, Vanpoucke Charlotte, De Vivo Immaculata, Vrijens Karen, Nawrot Tim S, Hogervorst Janneke G F, Madhloum Narjes, Saenen Nelly D, Janssen Bram G, Penders Joris, Vanpoucke Charlotte, De Vivo Immaculata, Vrijens Karen, Nawrot Tim S
INTRODUCTION	Particulate air pollution is probably causally related to increased risk of cardiovascular disease. Plasma homocysteine is an established cardiovascular disease risk factor. Recent studies show that exposure to particulate air pollution is associated with plasma homocysteine levels in adults but no studies on the association between prenatal air pollution and neonatal homocysteine levels exist.
METHODS	In 609 newborns of the ENVIRONAGE (ENVIRonmental influence ON early AGEing) birth cohort, we investigated the association between prenatal particulate matter exposure with a diameter ≤ 2.5 □m (PM2.5) and cord plasma homocysteine levels, and in a subset (n = 490) we studied the interaction with 11 single nucleotide polymorphism (SNPs) in oxidative stress-related genes (CAT, COMT, GSTP1, SOD2, NQO1 and HFE), through multiple linear regression. PM2.5 levels were obtained using a high resolution spatial temporal interpolation method. Homocysteine levels were measured by the homocysteine enzymatic assay on a Roche/Hitachi cobas c system. SNPs were assessed on the Biotrove OpenArray SNP genotyping platform.
RESULTS	In multivariable-adjusted models, cord plasma homocysteine levels were 8.1% higher (95% CI: 1.9 to 14.3%; p = 0.01) for each 5 □g/m³ increment in average PM2.5 exposure during the entire pregnancy. With regard to pregnancy trimesters, there was only an association in the 2nd trimester: 3.6% (95% CI: 0.9% to 6.4%; p = 0.01). The positive association between PM2.5 in and homocysteine was (borderline) statistically significantly modified by genetic variants in MnSOD (p interaction = 0.02), GSTP1 (p interaction = 0.07) and the sum score of the 3 studied SNPs in the CAT gene (p interaction=0.09), suggesting oxidative stress as an underlying mechanism of action.
CONCLUSIONS	Exposure to particulate air pollution in utero is associated with higher cord blood homocysteine levels, possibly through generating oxidative stress. Increased air pollution-induced homocysteine levels in early life might predispose for cardiovascular and other diseases later in life.

표-655. PubMed 논문번호 30489348의 내용 요약

구분	내용
PubMed ID	30489348
TITLE	Diesel Exhaust, Respirable Dust, and Ischemic Heart Disease: An Application of the Parametric g-formula.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000000954
AUTHORS	Neophytou Andreas M, Costello Sadie, Picciotto Sally, Brown Daniel M, Attfield Michael D, Blair Aaron, Lubin Jay H, Stewart Patricia A, Vermeulen Roel, Silverman Debra T, Eisen Ellen A
BACKGROUND	Although general population studies of air pollution suggest that particulate matter-diesel exhaust emissions in particular-is a potential risk factor for cardiovascular disease, direct evidence from occupational cohorts using quantitative metrics of exposure is limited. In this study, we assess counterfactual risk of ischemic heart disease (IHD) mortality under hypothetical scenarios limiting exposure levels of diesel exhaust and of respirable mine/ore dust in the Diesel Exhaust in Miners Study cohort.
METHODS	We analyzed data on 10,779 male miners from 8 nonmetal, noncoal mines-hired after diesel equipment was introduced in the respective facilities-and followed from 1948 to 1997, with 297 observed IHD deaths in this sample. We applied the parametric g-formula to assess risk under hypothetical scenarios with various limits for respirable elemental carbon (a surrogate for diesel exhaust), and respirable dust, separately and jointly.
RESULTS	The risk ratio comparing the observed risk to cumulative IHD mortality risk at age 80 under a hypothetical scenario where exposures to elemental carbon and respirable dust are eliminated was 0.79 (95% confidence interval [CI]: 0.64, 0.97). The corresponding risk difference was -3.0% (95% CI: -5.7, -0.3).
CONCLUSION	Our findings, based on data from a cohort of nonmetal miners, are consistent with the hypothesis that interventions to eliminate exposures to diesel exhaust and respirable dust would reduce IHD mortality risk.

표-656. PubMed 논문번호 The Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017 comparative risk assessment (CRA) is a comprehensive approach to risk factor quantification that offers a useful tool for synthesising evidence on risks and risk-outcome associations. With each annual GBD study, we update the GBD CRA to incorporate improved methods, new risks and risk-outcome pairs, and new data on risk exposure levels and risk-outcome associations.의 내용 요약

구분	내용
BACKGROUND	The Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017 comparative risk assessment (CRA) is a comprehensive approach to risk factor quantification that offers a useful tool for synthesising evidence on risks and risk-outcome associations. With each annual GBD study, we update the GBD CRA to incorporate improved methods, new risks and risk-outcome pairs, and new data on risk exposure levels and risk-outcome associations.
METHODS	We used the CRA framework developed for previous iterations of GBD to estimate levels and trends in exposure, attributable deaths, and attributable disability-adjusted life-years (DALYs), by age group, sex, year, and location for 84 behavioural, environmental and occupational, and metabolic risks or groups of risks from 1990 to 2017. This study included 476 risk-outcome pairs that met the GBD study criteria for convincing or probable evidence of causation. We extracted relative risk and exposure estimates from 46 749 randomised controlled trials, cohort studies, household surveys, census data, satellite data, and other sources. We used statistical models to pool data, adjust for bias, and incorporate covariates. Using the counterfactual scenario of theoretical minimum risk exposure level (TMREL), we estimated the portion of deaths and DALYs that could be attributed to a given risk. We explored the relationship between development and risk exposure by modelling the relationship between the Socio-demographic Index (SDI) and risk-weighted exposure prevalence and estimated expected levels of exposure and risk-attributable burden by SDI. Finally, we explored temporal changes in risk-attributable DALYs by decomposing those changes into six main component drivers of change as follows: (1) population growth; (2) changes in population age structures; (3) changes in exposure to environmental and occupational risks; (4) changes in exposure to behavioural risks; (5) changes in exposure to metabolic risks; and (6) changes due to all other factors, approximated as the risk-deleted death and DALY rates, where the risk-deleted rate is the rate that would be observed had we reduced the exposure levels to the TMREL for all risk factors included in GBD 2017.
FINDINGS	In 2017, 34·1 million (95% uncertainty interval [UI] 33·3-35·0) deaths and 1·21 billion (1·14-1·28) DALYs were attributable to GBD risk factors. Globally, 61·0% (59·6-62·4) of deaths and 48·3% (46·3-50·2) of DALYs were attributed to the GBD 2017 risk factors. When ranked by risk-attributable DALYs, high systolic blood pressure (SBP) was the leading risk factor, accounting for 10·4 million (9·39-11·5) deaths and 218 million (198-237) DALYs, followed by smoking (7·10 million [6·83-7·37] deaths and 182 million [173-193] DALYs), high fasting plasma glucose (6·53 million [5·23-8·23] deaths and 171 million [144-201] DALYs), high body-mass index (BMI; 4·72 million [2·99-6·70] deaths and 148 million [98·6-202] DALYs), and short gestation for birthweight (1·43 million [1·36-1·51] deaths and 139 million [131-147] DALYs). In total, risk-attributable DALYs declined by 4·9% (3·3-6·5) between 2007 and 2017. In the absence of demographic changes (ie, population growth and ageing), changes in risk exposure and risk-deleted DALYs would have led to a 23·5% decline in DALYs during that period. Conversely, in the absence of changes in risk exposure and risk-deleted DALYs, demographic changes would have led to an 18·6% increase in DALYs during that period. The ratios of observed risk exposure levels to exposure levels expected based on SDI (O/E ratios) increased globally for unsafe drinking water and household air pollution between 1990 and 2017. This result suggests that development is occurring more rapidly than are changes in

표-657. PubMed 논문번호 30524134의 내용 요약

구분	내용
PubMed ID	30524134
TITLE	Association Between Ambient Air Pollution and Cardiac Morpho-Functional Phenotypes: Insights From the UK Biobank Population Imaging Study.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.118.034856
AUTHORS	Aung Nay, Sanghvi Mihir M, Zemrak Filip, Lee Aaron M, Cooper Jackie A, Paiva Jose M, Thomson Ross J, Fung Kenneth, Khanji Mohammed Y, Lukaschuk Elena, Carapella Valentina, Kim Young Jin, Munroe Patricia B, Piechnik Stefan K, Neubauer Stefan, Petersen Steffen E
Background	Exposure to ambient air pollution is strongly associated with increased cardiovascular morbidity and mortality. Little is known about the influence of air pollutants on cardiac structure and function. We aim to investigate the relationship between chronic past exposure to traffic-related pollutants and the cardiac chamber volume, ejection fraction, and left ventricular remodeling patterns after accounting for potential confounders.
Methods	Exposure to ambient air pollutants including particulate matter and nitrogen dioxide was estimated from the Land Use Regression models for the years between 2005 and 2010. Cardiac parameters were measured from cardiovascular magnetic resonance imaging studies of 3920 individuals free from pre-existing cardiovascular disease in the UK Biobank population study. The median (interquartile range) duration between the year of exposure estimate and the imaging visit was 5.2 (0.6) years. We fitted multivariable linear regression models to investigate the relationship between cardiac parameters and traffic-related pollutants after adjusting for various confounders.
Results	The studied cohort was 62±7 years old, and 46% were men. In fully adjusted models, particulate matter with an aerodynamic diameter <2.5 μm concentration was significantly associated with larger left ventricular end-diastolic volume and end-systolic volume (effect size = 0.82%, 95% CI, 0.09-1.55%, P=0.027; and effect size = 1.28%, 95% CI, 0.15-2.43%, P=0.027, respectively, per interquartile range increment in particulate matter with an aerodynamic diameter <2.5 μm) and right ventricular end-diastolic volume (effect size = 0.85%, 95% CI, 0.12-1.58%, P=0.023, per interquartile range increment in particulate matter with an aerodynamic diameter <2.5 μm). Likewise, higher nitrogen dioxide concentration was associated with larger biventricular volume. Distance from the major roads was the only metric associated with lower left ventricular mass (effect size = -0.74%, 95% CI, -1.3% to -0.18%, P=0.01, per interquartile range increment). Neither left and right atrial phenotypes nor left ventricular geometric remodeling patterns were influenced by the ambient pollutants.
Conclusions	In a large asymptomatic population with no prevalent cardiovascular disease, higher past exposure to particulate matter with an aerodynamic diameter <2.5 μm and nitrogen dioxide was associated with cardiac ventricular dilatation, a marker of adverse remodeling that often precedes heart failure development.

표-658. PubMed 논문번호 30550348의 내용 요약

구분	내용
PubMed ID	30550348
TITLE	Pulmonary hypertension in patients with a history of intravenous drug use.
JOURNAL	Current medical research and opinion: 10.1080/03007995.2018.1558863
AUTHORS	McGee Michael, Whitehead Nicholas, Twaddell Scott, Collins Nicholas
OBJECTIVE	Pulmonary hypertension may be a consequence of intrinsic elevation in pulmonary vasculature resistance or complicate numerous other conditions affecting the cardiac and respiratory systems. In this review we sought to explore the relationship between pulmonary hypertension and intravenous drug use.
METHODS	A narrative review was conducted using PubMed MeSH search with further papers identified using a standard PubMed search with relevant key terms and various synonyms.
RESULTS	HIV infection may be associated with pulmonary hypertension due to indirect consequences of viral infection, venous thromboembolism or its therapies. Anti-retroviral infection may also influence plasma concentrations of commonly used treatments for pulmonary hypertension. Intravenous drug use is acknowledged as an important portal for the acquisition of hepatitis virus C infection, with portopulmonary hypertension a potential complication associated with poor prognosis. Interferon based therapy, used in treatment of chronic hepatitis C infection, may also play a causal role in the development of pulmonary hypertension. More recently, sofosbuvir has been linked to development or exacerbation of pulmonary arterial hypertension. Certain drugs of abuse may cause pulmonary hypertension due to properties that result in direct injury to the pulmonary vasculature. The potential for embolic phenomena, complicating venous thromboembolism, recurrent embolization of particulate matter or because of right-sided endocarditis, resulting in pulmonary hypertension is an important contributing factor in the pathophysiology in this unique cohort.
CONCLUSIONS	Eliciting a history of intravenous drug use is important and may be associated with a number of less common etiologies, each with specific diagnostic and therapeutic implications.

표-659. PubMed 논문번호 30551805의 내용 요약

구분	내용
PubMed ID	30551805
TITLE	The association between natural gas well activity and specific congenital anomalies in Oklahoma, 1997-2009.
JOURNAL	Environment international: 10.1016/j.envint.2018.12.011
AUTHORS	Janitz Amanda E, Dao Hanh Dung, Campbell Janis E, Stoner Julie A, Peck Jennifer D, Janitz Amanda E, Dao Hanh Dung, Campbell Janis E, Stoner Julie A, Peck Jennifer D
BACKGROUND	Natural gas drilling may pose multiple health risks, including congenital anomalies, through air pollutant emissions and contaminated water. Two recent studies have evaluated the relationship between natural gas activity and congenital anomalies, with both observing a positive relationship.
OBJECTIVES	We aimed to evaluate whether residence near natural gas wells is associated with critical congenital heart defects (CCHD), neural tube defects (NTD), and oral clefts in Oklahoma, the third highest natural gas producing state in the US.
METHODS	We conducted a retrospective cohort study among singleton births in Oklahoma (n = 476,600) to evaluate natural gas activity and congenital anomalies. We calculated an inverse distance-squared weighted (IDW) score based on the number of actively producing wells within a two-mile radius of the maternal residence during the month of delivery. We used modified Poisson regression with robust error variance to estimate prevalence proportion ratios (PPR) and 95% confidence intervals (CI) for the association between tertiles of natural gas activity (compared to no wells) and CCHD, NTD, and oral clefts adjusted for maternal education.
RESULTS	We observed an increased, though imprecise, prevalence of NTDs among children with natural gas activity compared to children with no wells (2nd tertile PPR: 1.34, 95% CI: 0.93, 1.93; 3rd tertile PPR: 1.20, 95% CI: 0.82, 1.75). We observed no association with CCHD or oral clefts overall. Specific CCHDs of common truncus, transposition of the great arteries, pulmonary valve atresia and stenosis, tricuspid valve atresia and stenosis, interrupted aortic arch, and total anomalous pulmonary venous connection were increased among those living in areas with natural gas activity compared to those living in areas without activity, though not statistically significant.
DISCUSSION	Our results were similar to previous studies for NTDs and specific CCHDs. Future directions include evaluating the association between specific phases of the drilling process and congenital anomalies to better refine the relevant exposure period.

표-660. PubMed 논문번호 30566375의 내용 요약

구분	내용
PubMed ID	30566375
TITLE	Long-Term Exposure to Ultrafine Particles and Incidence of Cardiovascular and Cerebrovascular Disease in a Prospective Study of a Dutch Cohort.
JOURNAL	Environmental health perspectives: 10.1289/EHP3047
AUTHORS	Downward George S, van Nunen Erik J H M, Kerckhoffs Jules, Vineis Paolo, Brunekreef Bert, Boer Jolanda M A, Messier Kyle P, Roy Ananya, Verschuren W Monique M, van der Schouw Yvonne T, Sluijs Ivonne, Gulliver John, Hoek Gerard, Vermeulen Roel
BACKGROUND	There is growing evidence that exposure to ultrafine particles (UFP; particles smaller than [Formula: see text]) may play an underexplored role in the etiology of several illnesses, including cardiovascular disease (CVD).
OBJECTIVES	We aimed o investigate the relationship between long-term exposure to ambient UFP and incident cardiovascular and cerebrovascular disease (CVA). As a secondary objective, we sought to compare effect estimates for UFP with those derived for other air pollutants, including estimates from two-pollutant models.
METHODS	Using a prospective cohort of 33,831 Dutch residents, we studied the association between long-term exposure to UFP (predicted via land use regression) and incident disease using Cox proportional hazard models. Hazard ratios (HR) for UFP were compared to HRs for more routinely monitored air pollutants, including particulate matter with aerodynamic diameter [Formula: see text] ([Formula: see text]), PM with aerodynamic diameter [Formula: see text] ([Formula: see text]), and [Formula: see text].
RESULTS	Long-term UFP exposure was associated with an increased risk for all incident CVD [[Formula: see text] per [Formula: see text]; 95% confidence interval (CI): 1.03, 1.34], myocardial infarction (MI) ([Formula: see text]; 95% CI: 1.00, 1.79), and heart failure ([Formula: see text]; 95% CI: 1.17, 2.66). Positive associations were also estimated for [Formula: see text] ([Formula: see text]; 95% CI: 1.01, 1.48 per [Formula: see text]) and coarse PM ([Formula: see text]; HR for all [Formula: see text]; 95% CI: 1.01, 1.45 per [Formula: see text]). CVD was not positively associated with [Formula: see text] (HR for all [Formula: see text]; 95% CI: 0.75, 1.28 per [Formula: see text]). HRs for UFP and CVAs were positive, but not significant. In two-pollutant models ([Formula: see text] and [Formula: see text]), positive associations tended to remain for UFP, while HRs for [Formula: see text] and [Formula: see text] generally attenuated towards the null.
CONCLUSIONS	These findings strengthen the evidence that UFP exposure plays an important role in cardiovascular health and that risks of ambient air pollution may have been underestimated based on conventional air pollution metrics. https://doi.org/10.1289/EHP3047.

표-661. PubMed 논문번호 30571166의 내용 요약

구분	내용
PubMed ID	30571166
TITLE	The Association of Ambient Air Pollution with Sleep Apnea: The Multi-Ethnic Study of Atherosclerosis.
JOURNAL	Annals of the American Thoracic Society: 10.1513/AnnalsATS.201804-248OC
AUTHORS	Billings Martha E, Gold Diane, Szpiro Adam, Aaron Carrie P, Jorgensen Neal, Gassett Amanda, Leary Peter J, Kaufman Joel D, Redline Susan R
RATIONALE	Air pollution may influence sleep through airway inflammation or autonomic nervous system pathway alterations. Epidemiological studies may provide evidence of relationships between chronic air pollution exposure and sleep apnea.
OBJECTIVES	To determine whether ambient-derived pollution exposure is associated with obstructive sleep apnea and objective sleep disruption.
METHODS	We analyzed data from a sample of participants in MESA (Multi-Ethnic Study of Atherosclerosis) who participated in both the Sleep and Air studies. Mean annual and 5-year exposure levels to nitrogen dioxide (NO2) and particulate matter ≤ 2.5 μm in aerodynamic diameter (PM2.5) were estimated at participants' homes using spatiotemporal models based on cohort-specific monitoring. Participants completed in-home full polysomnography and 7 days of wrist actigraphy. We used multivariate models, adjusted for demographics, comorbidities, socioeconomic factors, and site, to assess whether air pollution was associated with sleep apnea (apnea-hypopnea index ≥ 15) and actigraphy-measured sleep efficiency.
RESULTS	The participants (n = 1,974) were an average age of 68 (±9) years, 46% male, 36% white, 24% Hispanic, 28% black, and 12% Asian; 48% had sleep apnea and 25% had a sleep efficiency of ≤88%. A 10 ppb annual increase in NO2 exposure was associated with 39% greater adjusted odds of sleep apnea (95% confidence interval [CI], 1.03-1.87). A 5 μg/m3 greater annual PM2.5 exposure was also associated with 60% greater odds of sleep apnea (95% CI, 0.98-2.62). Sleep efficiency was not associated with air pollution levels in fully adjusted models.
CONCLUSIONS	Individuals with higher annual NO2 and PM2.5 exposure levels had a greater odds of sleep apnea. These data suggest that in addition to individual risk factors, environmental factors also contribute to the variation of sleep disorders across groups, possibly contributing to health disparities.

표-662. PubMed 논문번호 30575190의 내용 요약

구분	내용
PubMed ID	30575190
TITLE	Ambient fine particulate matter (PM2.5) exposure is associated with idiopathic ventricular premature complexes burden: A cohort study with consecutive Holter recordings.
JOURNAL	Journal of cardiovascular electrophysiology: 10.1111/jce.13829
AUTHORS	Tsai Tsung-Ying, Lo Li-Wei, Liu Shin-Huei, Cheng Wen-Han, Chou Yu-Hui, Lin Wei-Lun, Shinya Yamada, Lin Yenn-Jiang, Chang Shih-Lin, Hu Yu-Feng, Chung Fa-Po, Liao Jo-Nan, Chao Tze-Fan, Tuan Ta-Chuan, Chen Shih-Ann
BACKGROUND	Epidemiological evidence has shown an association between ambient fine particulate matter (PM2.5) exposure and cardiovascular mortality. Increased ventricular premature complex (VPC) burden can cause left ventricular dilatation and dysfunction. We aimed to investigate the relationship between acute PM2.5 exposure and VPC burden in patients without structural heart disease.
METHODS	We reviewed 26 820 patients who underwent 24-hour Holter electrocardiogram (ECG) recordings between 1 Jan 2013 and 1 Dec 2016. We enrolled patients with significant idiopathic (structurally normal heart) VPC burden defined as ≥30 VPCs/h (Lown grade 2) who had at least two Holter ECG recordings. The VPC burden between the studies on high and low PM2.5 exposure dates was compared in 24 and 12 hours time periods.
RESULT	Sixty-seven patients (31 men, 56.49 ± 18.35 years) were enrolled. Patients were exposed to 25.63 ± 11.47 and 14.66 ± 7.51 μg/m 3 of PM2.5 during the high and low study dates, respectively. The overall VPC counts (10,490.69 ± 10,681.63/day) and burden (10.22% ± 10.17%) were significantly higher on the days with higher PM2.5 exposure compared with low PM2.5 exposure dates (8293.31 ± 9009.09; P = 0.014% and 9.14% ± 12.73%, P = 0.012, respectively). Compared with low PM2.5 exposure dates, the VPC burden on high exposure dates was significantly higher from 9 am to 9 pm (5.85% ± 6.41% vs 4.84% ± 6.97%; P = 0.025) but not at nocturnal periods.
CONCLUSION	Our study demonstrated a significantly higher VPC burden on high PM2.5 exposure date. The burden was increased in the daytime but not at nighttime. This result suggests that daytime PM2.5 exposure may be associated with ventricular arrhythmia burden in the healthy population.

표-663. PubMed 논문번호 30577116의 내용 요약

구분	내용
PubMed ID	30577116
TITLE	The association between PM〈sub〉2.5〈/sub〉 exposure and neurological disorders: A systematic review and meta-analysis.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2018.11.218
AUTHORS	Fu Pengfei, Guo Xinbiao, Cheung Felix Man Ho, Yung Ken Kin Lam, Fu Pengfei, Guo Xinbiao, Cheung Felix Man Ho, Yung Ken Kin Lam
BACKGROUND	Recent systematic review and meta-analyses have tried to identify an association between PM2.5 exposure and stroke, but few could find a conclusive and comprehensive evidence. Moreover, the associations between PM2.5, neurodegenerative diseases and neurodevelopmental disorders have never been reviewed. We aimed to assess the effects of PM2.5 exposure on stroke, dementia, Alzheimer's disease, autism spectrum disorder (ASD), Parkinson's disease, and mild cognitive impairment (MCI).
METHODS	We searched PubMed and CNKI databases for articles published until June 2018. Studies were eligible for analysis if they were human studies and provided risk estimates with 95% CI. We screened 1645 articles and identified 80 eligible studies covering 26 countries across all continents except Antarctica. Risks of incidence and mortality were extracted and stratified by types of neurological disorders, PM2.5 concentration and duration of PM2.5 exposure.
RESULTS	We found significant association between PM2.5 exposure and stroke, dementia, Alzheimer's disease, ASD, Parkinson's disease. The risks of ischemic and hemorrhagic stroke were higher than that of stroke in general, and that hemorrhagic stroke had by far the highest mortality. The risk of stroke for heavily polluted countries was significantly higher than that of lightly polluted countries. Short- and long-term PM2.5 exposure was associated with increased risks of stroke (short-term odds ratio 1.01 [per 10 μg/m3 increase in PM2.5 concentrations], 95% CI 1.01-1.02; long-term 1.14, 95% CI 1.08-1.21) and mortality (short-term 1.02, 95% CI 1.01-1.04; long-term 1.15, 95% CI 1.07-1.24) of stroke. Long-term PM2.5 exposure was associated with increased risks of dementia (1.16, 95% CI 1.07-1.26), Alzheimer's disease (3.26, 95% 0.84-12.74), ASD (1.68, 95% CI 1.20-2.34), and Parkinson's disease (1.34, 95% CI 1.04-1.73).
CONCLUSIONS	There is a strong association between PM2.5 exposure and neurological disorders. National governments should exert greater efforts to improve air quality given its health implications.

표-664. PubMed 논문번호 30612847의 내용 요약

구분	내용
PubMed ID	30612847
TITLE	Long-term exposure of fine particulate matter air pollution and incident atrial fibrillation in the general population: A nationwide cohort study.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2018.12.048
AUTHORS	Kim In-Soo, Yang Pil-Sung, Lee Jinae, Yu Hee Tae, Kim Tae-Hoon, Uhm Jae-Sun, Pak Hui-Nam, Lee Moon-Hyoung, Joung Boyoung, Kim In-Soo, Yang Pil-Sung, Lee Jinae, Yu Hee Tae, Kim Tae-Hoon, Uhm Jae-Sun, Pak Hui-Nam, Lee Moon-Hyoung, Joung Boyoung
BACKGROUND	Although many studies have linked elevations in fine particulate matter (PM2.5) air pollution to adverse cardiovascular outcomes, long-term exposures of PM2.5 on air pollution-related incident atrial fibrillation (AF) in general population have not yet been investigated well.
METHODS	We included 432,587 subjects of general population not diagnosed with AF from the Korean National Health Insurance Service-National Sample Cohort from 2009 to 2013. Medical records were screened from January 2002 to investigate the subjects' disease-free baseline period. They were followed until December 2013. We matched subjects' residential ZIP code with hourly measurements of air pollutant (particulate and gaseous) concentrations and meteorological (temperature and humidity) data during the study period.
RESULTS	During 1,666,528 person·years, incident AF was observed in 5825 subjects (350/100,000 person·year). We found significant associations between incident AF and long-term average concentrations of PM2.5 (HR = 1.179[1.176-1.183] for 10 μg/m3 increments, p < 0.001), PM10 (HR = 1.034[1.033-1.036] for 10 μg/m3 increments, p < 0.001), and gaseous air pollutants during the study period. When dividing subjects into subgroups, these long-term exposures of PM2.5 effects were more profound in males (HR = 1.187[1.183-1.192], p < 0.001), older subjects (aged ≥ 60 years; HR = 1.194[1.188-1.200], p < 0.001), those who were obesity (body mass index ≥ 27.5 kg/m2, HR = 1.191[1.183-1.199], p < 0.001), subjects with previous myocardial infarction (HR = 1.203[1.186-1.221], p < 0.001), and history of hypertension (HR = 1.191[1.185-1.197], p < 0.001) (each interaction p < 0.05 compared to the opposite subgroup).
CONCLUSIONS	Even in the Asian general population, long-term exposure of PM2.5 is associated with the increased incidence of new-onset AF. It is more profound in obese male subjects > 60-year old and who have a history of hypertension or previous myocardial infarction.

표-665. PubMed 논문번호 30641259의 내용 요약

구분	내용
PubMed ID	30641259
TITLE	Case-crossover analysis of short-term particulate matter exposures and stroke in the health professionals follow-up study.
JOURNAL	Environment international: 10.1016/j.envint.2018.12.044
AUTHORS	Fisher Jared A, Puett Robin C, Laden Francine, Wellenius Gregory A, Sapkota Amir, Liao Duanping, Yanosky Jeff D, Carter-Pokras Olivia, He Xin, Hart Jaime E
BACKGROUND	Stroke is a leading cause of morbidity and mortality in the United States. Associations between short-term exposures to particulate matter (PM) air pollution and stroke are inconsistent. Many prior studies have used administrative and hospitalization databases where misclassification of the type and timing of the stroke event may be problematic.
METHODS	In this case-crossover study, we used a nationwide kriging model to examine short-term ambient exposure to PM10 and PM2.5 and risk of ischemic and hemorrhagic stroke among men enrolled in the Health Professionals Follow-up Study. Conditional logistic regression models were used to obtain estimates of odds ratios (OR) and 95% confidence intervals (CI) associated with an interquartile range (IQR) increase in PM2.5 or PM10. Lag periods up to 3 days prior to the stroke event were considered in addition to a 4-day average. Stratified models were used to examine effect modification by patient characteristics.
RESULTS	Of the 727 strokes that occurred between 1999 and 2010, 539 were ischemic and 122 were hemorrhagic. We observed positive statistically significant associations between PM10 and ischemic stroke (ORlag0-3 = 1.26; 95% CI: 1.03-1.55 per IQR increase [14.46 μg/m3]), and associations were elevated for nonsmokers, aspirin nonusers, and those without a history of high cholesterol. However, we observed no evidence of a positive association between short-term exposure to PM and hemorrhagic stroke or between PM2.5 and ischemic stroke in this cohort.
CONCLUSIONS	Our study provides evidence that ambient PM10 may be associated with higher risk of ischemic stroke and highlights that ischemic and hemorrhagic strokes are heterogeneous outcomes that should be treated as such in analyses related to air pollution.

표-666. PubMed 논문번호 30654247의 내용 요약

구분	내용
PubMed ID	30654247
TITLE	Short-term exposure to ambient air pollution and circulating biomarkers of endothelial cell activation: The Framingham Heart Study.
JOURNAL	Environmental research: 10.1016/j.envres.2018.10.027
AUTHORS	Li Wenyuan, Dorans Kirsten S, Wilker Elissa H, Rice Mary B, Ljungman Petter L, Schwartz Joel D, Coull Brent A, Koutrakis Petros, Gold Diane R, Keaney John F, Vasan Ramachandran S, Benjamin Emelia J, Mittleman Murray A, Li Wenyuan, Dorans Kirsten S, Wilker Elissa H, Rice Mary B, Ljungman Petter L, Schwartz Joel D, Coull Brent A, Koutrakis Petros, Gold Diane R, Keaney John F, Vasan Ramachandran S, Benjamin Emelia J, Mittleman Murray A
BACKGROUND	Short-term exposure to air pollution has been associated with cardiovascular events, potentially by promoting endothelial cell activation and inflammation. A few large-scale studies have examined the associations and have had mixed results.
METHODS	We included 3820 non-current smoking participants (mean age 56 years, 54% women) from the Framingham Offspring cohort examinations 7 (1998-2001) and 8 (2005-2008), and Third Generation cohort examination 1 (2002-2005), who lived within 50 km of a central monitoring station. We calculated the 1- to 7-day moving averages of fine particulate matter (PM2.5), black carbon (BC), sulfate (SO42-), nitrogen oxides (NOx), and ozone before examination visits. We used linear mixed effect models for P-selectin, monocyte chemoattractant protein 1 (MCP-1), intercellular adhesion molecule 1, lipoprotein-associated phospholipase A2 activity and mass, and osteoprotegerin that were measured up to twice, and linear regression models for CD40 ligand and interleukin-18 that were measured once, adjusting for demographics, life style and clinical factors, socioeconomic position, time, and meteorology.
RESULTS	We found negative associations of PM2.5 and BC with P-selectin, of ozone with MCP-1, and of SO42- and NOx with osteoprotegerin. At the 5-day moving average, a 5 □g/m3 higher PM2.5 was associated with 1.6% (95% CI: -2.8, -0.3) lower levels of P-selectin; a 10 ppb higher ozone was associated with 1.7% (95% CI: -3.2, -0.1) lower levels of MCP-1; and a 20 ppb higher NOx was associated with 2.0% (95% CI: -3.6, -0.4) lower levels of osteoprotegerin.
CONCLUSIONS	We did not find evidence of positive associations between short-term air pollution exposure and endothelial cell activation. On the contrary, short-term exposure to higher levels of ambient pollutants were associated with lower levels of P-selectin, MCP-1, and osteoprotegerin in the Framingham Heart Study.

표-667. PubMed 논문번호 30689640의 내용 요약

구분	내용
PubMed ID	30689640
TITLE	Association between temperature variability and daily hospital admissions for cause-specific cardiovascular disease in urban China: A national time-series study.
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1002738
AUTHORS	Tian Yaohua, Liu Hui, Si Yaqin, Cao Yaying, Song Jing, Li Man, Wu Yao, Wang Xiaowen, Xiang Xiao, Juan Juan, Chen Libo, Wei Chen, Gao Pei, Hu Yonghua
BACKGROUND	Epidemiological studies have provided compelling evidence of associations between ambient temperature and cardiovascular disease. However, evidence of effects of daily temperature variability on cardiovascular disease is scarce and mixed. We aimed to examine short-term associations between temperature variability and hospital admissions for cause-specific cardiovascular disease in urban China.
METHODS AND FINDINGS	We conducted a national time-series analysis in 184 cities in China between 2014 and 2017. Data on daily hospital admissions for ischemic heart disease, heart failure, heart rhythm disturbances, and ischemic stroke were obtained from the database of Urban Employee Basic Medical Insurance (UEBMI) including 0.28 billion enrollees. Temperature data were acquired from the China Meteorological Data Sharing Service Center. Temperature variability was calculated from the standard deviation (SD) of daily minimum and maximum temperatures over exposure days. City-specific associations between temperature variability and cardiovascular disease were examined with overdispersed Poisson models controlling for calendar time, day of the week, public holiday, and daily mean temperature and relative humidity. Random-effects meta-analyses were performed to obtain national and regional average associations. We also plotted exposure-response relationship curve using a natural cubic spline of temperature variability. There were 8.0 million hospital admissions for cardiovascular disease during the study period. At the national-average level, a 1-° C increase in temperature variability at 0-1 days (TV0-1) was associated with a 0.44% (0.32%-0.55%), 0.31% (0.20%-0.43%), 0.48% (0.01%-0.96%), 0.34% (0.01%-0.67%), and 0.82% (0.59%-1.05%) increase in hospital admissions for cardiovascular disease, ischemic heart disease, heart failure, heart rhythm disturbances, and ischemic stroke, respectively. The estimates decreased but remained significant when controlling for ambient fine particulate matter (PM2.5), NO2, and SO2 pollution. The main limitation of the present study was the unavailability of data on individual exposure to temperature variability.
CONCLUSIONS	Our findings suggested that short-term temperature variability exposure could increase the risk of cardiovascular disease, which may provide new insights into the health effects of climate change.

표-668. PubMed 논문번호 30700142의 내용 요약

구분	내용
PubMed ID	30700142
TITLE	Mediterranean Diet and the Association Between Air Pollution and Cardiovascular Disease Mortality Risk.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.118.035742
AUTHORS	Lim Chris C, Hayes Richard B, Ahn Jiyoung, Shao Yongzhao, Silverman Debra T, Jones Rena R, Thurston George D
BACKGROUND	Recent experimental evidence suggests that nutritional supplementation can blunt adverse cardiopulmonary effects induced by acute air pollution exposure. However, whether usual individual dietary patterns can modify the association between long-term air pollution exposure and health outcomes has not been previously investigated. We assessed, in a large cohort with detailed diet information at the individual level, whether a Mediterranean diet modifies the association between long-term exposure to ambient air pollution and cardiovascular disease mortality risk.
METHODS	The National Institutes of Health-American Association for Retired Persons Diet and Health Study, a prospective cohort (N=548 845) across 6 states and 2 cities in the United States and with a follow-up period of 17 years (1995-2011), was linked to estimates of annual average exposures to fine particulate matter and nitrogen dioxide at the residential census-tract level. The alternative Mediterranean Diet Index, which uses a 9-point scale to assess conformity with a Mediterranean-style diet, was constructed for each participant from information in cohort baseline dietary questionnaires. We evaluated mortality risks for cardiovascular disease, ischemic heart disease, cerebrovascular disease, or cardiac arrest associated with long-term air pollution exposure. Effect modification of the associations between exposure and the mortality outcomes by alternative Mediterranean Diet Index was examined via interaction terms.
RESULTS	For fine particulate matter, we observed elevated and significant associations with cardiovascular disease (hazard ratio [HR] per 10 μg/m3, 1.13; 95% CI, 1.08-1.18), ischemic heart disease (HR, 1.16; 95% CI, 1.10-1.23), and cerebrovascular disease (HR, 1.15; 95% CI, 1.03-1.28). For nitrogen dioxide, we found significant associations with cardiovascular disease (HR per 10 ppb, 1.06; 95% CI, 1.04-1.08) and ischemic heart disease (HR, 1.08; 95% CI, 1.05-1.11). Analyses indicated that Mediterranean diet modified these relationships, as those with a higher alternative Mediterranean Diet Index score had significantly lower rates of cardiovascular disease mortality associated with long-term air pollution exposure (P-interaction<0.05).
CONCLUSIONS	A Mediterranean diet reduced cardiovascular disease mortality risk related to long-term exposure to air pollutants in a large prospective US cohort. Increased consumption of foods rich in antioxidant compounds may aid in reducing the considerable disease burden associated with ambient air pollution.

표-669. PubMed 논문번호 30702928의 내용 요약

구분	내용
PubMed ID	30702928
TITLE	Associations between Coarse Particulate Matter Air Pollution and Cause-Specific Mortality: A Nationwide Analysis in 272 Chinese Cities.
JOURNAL	Environmental health perspectives: 10.1289/EHP2711
AUTHORS	Chen Renjie, Yin Peng, Meng Xia, Wang Lijun, Liu Cong, Niu Yue, Liu Yunning, Liu Jiangmei, Qi Jinlei, You Jinling, Kan Haidong, Zhou Maigeng
BACKGROUND	Coarse particulate matter with aerodynamic diameter between 2.5 and [Formula: see text] ([Formula: see text]) air pollution is a severe environmental problem in developing countries, but its challenges to public health were rarely evaluated.
OBJECTIVE	We aimed to investigate the associations between day-to-day changes in [Formula: see text] and cause-specific mortality in China.
METHODS	We conducted a nationwide daily time-series analysis in 272 main Chinese cities from 2013 to 2015. The associations between [Formula: see text] concentrations and mortality were analyzed in each city using overdispersed generalized additive models. Two-stage Bayesian hierarchical models were used to estimate national and regional average associations, and random-effect models were used to pool city-specific concentration-response curves. Two-pollutant models were adjusted for fine particles with aerodynamic diameter [Formula: see text] ([Formula: see text]) or gaseous pollutants.
RESULTS	Overall, we observed positive and approximately linear concentration-response associations between [Formula: see text] and daily mortality. A [Formula: see text] increase in [Formula: see text] was associated with higher mortality due to nonaccidental causes [0.23%; 95% posterior interval (PI): 0.13, 0.33], cardiovascular diseases (CVDs; 0.25%; 95% PI: 0.13, 0.37), coronary heart disease (CHD; 0.21%; 95% PI: 0.05, 0.36), stroke (0.21%; 95% PI: 0.08, 0.35), respiratory diseases (0.26%; 95% PI: 0.07, 0.46), and chronic obstructive pulmonary disease (COPD; 0.34%; 95% PI: 0.12, 0.57). Associations were stronger for cities in southern vs. northern China, with significant differences for total and cardiovascular mortality. Associations with [Formula: see text] were of similar magnitude to those for [Formula: see text] in both single- and two-pollutant models with mutual adjustment. Associations were robust to adjustment for gaseous pollutants other than nitrogen dioxide and sulfur dioxide. Meta-regression indicated that a larger positive correlation between [Formula: see text] and [Formula: see text] predicted stronger city-specific associations between [Formula: see text] and total mortality.
CONCLUSIONS	This analysis showed significant associations between short-term [Formula: see text] exposure and daily nonaccidental and cardiopulmonary mortality based on data from 272 cities located throughout China. Associations appeared to be independent of exposure to [Formula: see text], carbon monoxide, and ozone. https://doi.org/10.1289/EHP2711.

표-670. PubMed 논문번호 30706081의 내용 요약

구분	내용
PubMed ID	30706081
TITLE	Long-term exposure to ambient fine particulate matter (PM〈sub〉2.5〈/sub〉) and incident type 2 diabetes: a longitudinal cohort study.
JOURNAL	Diabetologia: 10.1007/s00125-019-4825-1
AUTHORS	Lao Xiang Qian, Guo Cui, Chang Ly-Yun, Bo Yacong, Zhang Zilong, Chuang Yuan Chieh, Jiang Wun Kai, Lin Changqing, Tam Tony, Lau Alexis K H, Lin Chuan-Yao, Chan Ta-Chien
AIMS/HYPOTHESIS	Information on the associations of long-term exposure to fine particulate matter (with an aerodynamic diameter less than 2.5 μm; PM2.5) with the development of type 2 diabetes is scarce, especially for south-east Asia, where most countries are experiencing serious air pollution. This study aimed to investigate the long-term effects of exposure to ambient PM2.5 on the incidence of type 2 diabetes in a population of Taiwanese adults.
METHODS	A total of 147,908 participants without diabetes, at least 18 years of age, were recruited in a standard medical examination programme between 2001 and 2014. They were encouraged to take medical examinations periodically and underwent at least two measurements of fasting plasma glucose (FPG). Incident type 2 diabetes was identified as FPG ≥7 mmol/l or self-reported physician-diagnosed diabetes in the subsequent medical visits. The PM2.5 concentration at each participant's address was estimated using a satellite-based spatiotemporal model with a resolution of 1 × 1 km2. The 2 year average of PM2.5 concentrations (i.e. the year of and the year before the medical examination) was treated as an indicator of long-term exposure to ambient PM2.5 air pollution. We performed Cox regression models with time-dependent covariates to analyse the long-term effects of exposure to PM2.5 on the incidence of type 2 diabetes. A wide range of covariates were introduced in the models to control for potential effects, including age, sex, education, season, year, smoking status, alcohol drinking, physical activity, vegetable intake, fruit intake, occupational exposure, BMI, hypertension and dyslipidaemia (all were treated as time-dependent covariates except for sex).
RESULTS	Compared with the participants exposed to the first quartile of ambient PM2.5, participants exposed to the second, third and fourth quartiles of ambient PM2.5 had HRs of 1.28 (95% CI 1.18, 1.39), 1.27 (95% CI 1.17, 1.38) and 1.16 (95% CI 1.07, 1.26), respectively, for the incidence of type 2 diabetes. Participants who drank occasionally or regularly (more than once per week) or who had a lower BMI (〈23 kg/m2) were more sensitive to the long-term effects of exposure to ambient PM2.5.
CONCLUSIONS/INTERPRETATION	Long-term exposure to ambient PM2.5 appears to be associated with a higher risk of developing type 2 diabetes in this Asian population experiencing high levels of air pollution.

표-671. PubMed 논문번호 30715377의 내용 요약

구분	내용
PubMed ID	30715377
TITLE	Childhood Exposure to Passive Smoking and Bone Health in Adulthood: The Cardiovascular Risk in Young Finns Study.
JOURNAL	The Journal of clinical endocrinology and metabolism: 10.1210/jc.2018-02501
AUTHORS	Juonala Markus, Pitk□nen Niina, Tolonen Sanna, Laaksonen Marika, Siev□nen Harri, Jokinen Eero, Laitinen Tomi, Sabin Matthew A, Hutri-K□h□nen Nina, Lehtim□ki Terho, Taittonen Leena, Jula Antti, Loo Britt-Marie, Impivaara Olli, K□h□nen Mika, Magnussen Costan G, Viikari Jorma S A, Raitakari Olli T
CONTEXT	Passive smoke exposure has been linked to the risk of osteoporosis in adults.
OBJECTIVE	We examined the independent effects of childhood passive smoke exposure on adult bone health.
DESIGN/SETTING	Longitudinal, the Cardiovascular Risk in Young Finns Study.
PARTICIPANTS	The study cohort included 1422 individuals followed for 28 years since baseline in 1980 (age 3 to 18 years). Exposure to passive smoking was determined in childhood. In adulthood, peripheral bone traits were assessed with peripheral quantitative CT (pQCT) at the tibia and radius, and calcaneal mineral density was estimated with quantitative ultrasound. Fracture data were gathered by questionnaires.
RESULTS	Parental smoking in childhood was associated with lower pQCT-derived bone sum index in adulthood ($\beta \pm$ SE, -0.064 ± 0.023 per smoking parent; P = 0.004) in multivariate models adjusted for age, sex, active smoking, body mass index, serum 25-OH vitamin D concentration, physical activity, and parental socioeconomic position. Similarly, parental smoking was associated with lower heel ultrasound estimated bone mineral density in adulthood ($\beta \pm$ SE, -0.097 ± 0.041 per smoking parent; P = 0.02). Parental smoking was also associated with the incidence of low-energy fractures (OR, 1.28; 95% CI, 1.01 to 1.62). Individuals with elevated cotinine levels (3 to 20 ng/mL) in childhood had lower bone sum index with pQCT ($\beta \pm$ SE, -0.206 ± 0.057; P = 0.0003). Children whose parents smoked and had high cotinine levels (3 to 20 ng/mL) had significantly lower pQCT-derived bone sum index compared with those with smoking parents but had low cotinine levels (<3 ng/mL) ($\beta \pm$ SE, -0.192 ± 0.072; P = 0.008).
CONCLUSIONS AND RELEVANCE	Children of parents who smoke have evidence of impaired bone health in adulthood.

표-672. PubMed 논문번호 30716571의 내용 요약

구분	내용
PubMed ID	30716571
TITLE	Association of long-term exposure to traffic-related PM〈sub〉10〈/sub〉 with heart rate variability and heart rate dynamics in healthy subjects.
JOURNAL	Environment international: 10.1016/j.envint.2019.01.031
AUTHORS	Meier-Girard Delphine, Delgado-Eckert Edgar, Schaffner Emmanuel, Schindler Christian, K□nzli Nino, Adam Martin, Pichot Vincent, Kronenberg Florian, Imboden Medea, Frey Urs, Probst-Hensch Nicole
BACKGROUND	Epidemiological evidence on the influence of long-term exposure to traffic-related particulate matter (TPM10) on heart rate variability (HRV) is weak.
OBJECTIVE	To evaluate the association of long-term exposure (10 years) with TPM10 on the regulation of the autonomic cardiovascular system and heart rate dynamics (HRD) in an aging general population, as well as potential modifying effects by the a priori selected factors sex, smoking status, obesity, and gene variation in selected glutathione S-transferases (GSTs).
METHODS	We analyzed data from 1593 SAPALDIA cohort participants aged ≥ 50 years. For each participant, various HRV and HRD parameters were derived from 24-hour electrocardiogram recordings. Each parameter obtained was then used as the outcome variable in multivariable mixed linear regression models in order to evaluate the association with TPM10. Potential modifying effects were assessed using interaction terms.
RESULTS	No association between long-term exposure to TPM10 and HRV/HRD was observed in the entire study population. However, HRD changes were found in subjects without cardiovascular morbidity and both HRD and HRV changes in non-obese subjects without cardiovascular morbidity. Subjects without cardiovascular morbidity with homozygous GSTM1 gene deletion appeared to be more susceptible to the effects of TPM10.
CONCLUSION	This study suggests that long-term exposure to TPM10 triggers adverse changes in the regulation of the cardiovascular system. These adverse effects were more visible in the subjects without cardiovascular disease, in whom the overall relationship between TPM10 and HRV/HRD could not be masked by underlying morbidities and the potential counteracting effects of related drug treatments.

표-673. PubMed 논문번호 30731313의 내용 요약

구분	내용
PubMed ID	30731313
TITLE	Short-term and long-term exposures to fine particulate matter constituents and health: A systematic review and meta-analysis.
JOURNAL	Environmental pollution (Barking, Essex : 1987): 10.1016/j.envpol.2018.12.060
AUTHORS	Yang Yang, Ruan Zengliang, Wang Xiaojie, Yang Yin, Mason Tonya G, Lin Hualiang, Tian Linwei, Yang Yang, Ruan Zengliang, Wang Xiaojie, Yang Yin, Mason Tonya G, Lin Hualiang, Tian Linwei
BACKGROUND	Fine particulate matter (Particulate matter with diameter ≤ 2.5 μm) is associated with multiple health outcomes, with varying effects across seasons and locations. It remains largely unknown that which components of PM2.5 are most harmful to human health.
METHODS	We systematically searched all the relevent studies published before August 1, 2018, on the associations of fine particulate matter constituents with mortality and morbidity, using Web of Science, MEDLINE, PubMed and EMBASE. Studies were included if they explored the associations between short term or long term exposure of fine particulate matter constituents and natural, cardiovascular or respiratory health endpoints. The criteria for the risk of bias was adapted from OHAT and New Castle Ottawa. We applied a random-effects model to derive the risk estimates for each constituent. We performed main analyses restricted to studies which adjusted the PM2.5 mass in their models.
RESULTS	Significant associations were observed between several PM2.5 constituents and different health endpoints. Among them, black carbon and organic carbon were most robustly and consistently associated with all natural, cardiovascular mortality and morbidity. Other potential toxic constituents including nitrate, sulfate, Zinc, silicon, iron, nickel, vanadium, and potassium were associated with adverse cardiovascular health, while nitrate, sulfate and vanadium were relevant for adverse respiratory health outcomes.
CONCLUSIONS	Our analysis suggests that black carbon and organic carbon are important detrimental components of PM2.5, while other constituents are probably hazardous to human health. However, more studies are needed to further confirm our results.

표-674. PubMed 논문번호 30735532의 내용 요약

구분	내용
PubMed ID	30735532
TITLE	Individual and area-level determinants associated with C-reactive protein as a marker of cardiometabolic risk among adults: Results from the German National Health Interview and Examination Survey 2008-2011.
JOURNAL	PloS one: 10.1371/journal.pone.0211774
AUTHORS	Steppuhn Henriette, Lau ß mann Detlef, Baumert Jens, Kroll Lars, Lampert Thomas, Pla ß Dietrich, Scheidt-Nave Christa, Heidemann Christin
BACKGROUND	High-sensitivity C-reactive protein (hsCRP) is a sensitive biomarker of systemic inflammation and is related to the development and progression of cardiometabolic diseases. Beyond individual-level determinants, characteristics of the residential physical and social environment are increasingly recognized as contextual determinants of systemic inflammation and cardiometabolic risks. Based on a large nationwide sample of adults in Germany, we analyzed the cross-sectional association of hsCRP with residential environment characteristics. We specifically asked whether these associations are observed independent of determinants at the individual level.
METHODS	Data on serum hsCRP levels and individual sociodemographic, behavioral, and anthropometric characteristics were available from the German Health Interview and Examination Survey for Adults (2008-2011). Area-level variables included, firstly, the predefined German Index of Socioeconomic Deprivation (GISD) derived from the INKAR (indicators and maps on spatial and urban development in Germany and Europe) database and, secondly, population-weighted annual average concentration of particulate matter (PM10) in ambient air provided by the German Environment Agency. Associations with log-transformed hsCRP levels were analyzed using random-intercept multi-level linear regression models including 6,768 participants aged 18-79 years nested in 162 municipalities.
RESULTS	No statistically significant association of PM10 exposure with hsCRP was observed. However, adults residing in municipalities with high compared to those with low social deprivation showed significantly elevated hsCRP levels (change in geometric mean 13.5%, 95%CI 3.2%-24.7%) after adjusting for age and sex. The observed relationship was independent of individual-level educational status. Further adjustment for smoking, sports activity, and abdominal obesity appeared to markedly reduce the association between area-level social deprivation and hsCRP, whereas all individual-level variables contributed significantly to the model.
CONCLUSIONS	Area-level social deprivation is associated with higher systemic inflammation and the potentially mediating role of modifiable risk factors needs further elucidation. Identifying and assessing the source-specific harmful components of ambient air pollution in population-based studies remains challenging.

표-675. PubMed 논문번호 30766556의 내용 요약

구분	내용
PubMed ID	30766556
TITLE	GATA4 molecular screening and assessment of environmental risk factors in a Moroccan cohort with tetralogy of Fallot.
JOURNAL	African health sciences: 10.4314/ahs.v18i4.11
AUTHORS	El Bouchikhi Ihssane, Belhassan Khadija, Moufid Fatima Zohra, Houssaini Mohammed Iraqui, Bouguenouch Laila, Samri Imane, Bouhrim Mohamed, Ouldim Karim, Atmani Samir
Background	Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart defect (CHD) with an incidence of 1/3600 live births. This disorder was associated with mutations in the transcription factors involved in cardiogenesis, like Nk2 homeobox5 (NKX2-5), GATA binding protein4 (GATA4) and T-BOX1 (TBX1). GATA4 contributes particularly to heart looping and differentiation of the second heart field.
Objectives	The aim of this study was to screen a Moroccan cohort with tetralogy of Fallot for GATA4 mutations, and to assess environmental risk factors that could be involved in the occurrence of this disorder.
Methods	Thirty-one non-syndromic TOF patients, enrolled between 5th April 2014 and 18th June 2015, were screened for GATA4 mutations using direct sequencing of GATA4 coding exons. Statistical assessment of different risk factors, which is a retrospective study, was carried out using Chi-square and Fisher's exact tests.
Results	We identified seven exonic variants in nine patients (two missense and five synonymous variants); in addition of eight intronic variants. Assessment of environmental risk factors shows significant association of maternal passive smoking with TOF in the Moroccan population.
Conclusion	The present study allowed, for the first time, the molecular and environmental characterisation of Moroccan TOF population. Our findings emphasise particularly the strong association of passive smoking with the emergence of tetralogy of Fallot.

표-676. PubMed 논문번호 30775931의 내용 요약

구분	내용
PubMed ID	30775931
TITLE	Exposure to Residential Greenness as a Predictor of Cause-Specific Mortality and Stroke Incidence in the Rome Longitudinal Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP2854
AUTHORS	Orioli Riccardo, Antonucci Chiara, Scortichini Matteo, Cerza Francesco, Marando Federica, Ancona Carla, Manes Fausto, Davoli Marina, Michelozzi Paola, Forastiere Francesco, Cesaroni Giulia
BACKGROUND	Living in areas with higher levels of surrounding greenness and access to urban green areas have been associated with beneficial health outcomes. Some studies suggested a beneficial influence on mortality, but the evidence is still controversial.
OBJECTIVES	We used longitudinal data from a large cohort to estimate associations of two measures of residential greenness exposure with cause-specific mortality and stroke incidence.
METHODS	We studied a population-based cohort of 1,263,721 residents in Rome aged [Formula: see text], followed from 2001 to 2013. As greenness exposure, we utilized the leaf area index (LAI), which expresses the tree canopy as the leaf area per unit ground surface area, and the normalized difference vegetation index (NDVI) within 300- and [Formula: see text] buffers around home addresses. We estimated the association between the two measures of residential greenness and the outcomes using Cox models, after controlling for relevant individual covariates and contextual characteristics, and explored potential mediation by air pollution [fine particulate matter with aerodynamic diameter [Formula: see text] [Formula: see text] and [Formula: see text]] and road traffic noise.
RESULTS	We observed 198,704 deaths from nonaccidental causes, 81,269 from cardiovascular diseases [CVDs; 29,654 from ischemic heart disease (IHD)], 18,090 from cerebrovascular diseases, and 29,033 incident cases of stroke. Residential greenness, expressed as interquartile range (IQR) increase in LAI within [Formula: see text], was inversely associated with stroke incidence {hazard ratio (HR) 0.977 [95% confidence interval (CI): 0.961, 0.994]} and mortality for nonaccidental [HR 0.988 (95% CI: 0.981, 0.994)], cardiovascular [HR 0.984 (95% CI: 0.974, 0.994)] and cerebrovascular diseases [HR 0.964 (95% CI: 0.943, 0.985)]. Similar results were obtained using NDVI with 300- or [Formula: see text] buffers.
CONCLUSIONS	Living in greener areas was associated with better health outcomes in our study, which could be partly due to reduced exposure to environmental hazards. Further research is required to understand the underlying mechanisms. https://doi.org/10.1289/EHP2854.

표-677. PubMed 논문번호 30790006의 내용 요약

구분	내용
PubMed ID	30790006
TITLE	Health effects of ultrafine particles: a systematic literature review update of epidemiological evidence.
JOURNAL	International journal of public health: 10.1007/s00038-019-01202-7
AUTHORS	Ohlwein Simone, Kappeler Ron, Kutlar Joss Meltem, K□nzli Nino, Hoffmann Barbara
OBJECTIVES	Due to their small size, ultrafine particles (UFP) are believed to exert higher toxicity than larger particles. As numerous studies on health effects of UFP have been published since the last systematic review in 2013, we aim to systematically review the new literature.
METHODS	We searched MEDLINE and the specialized LUDOK database for studies published between 01.01.2011 and 11.05.2017 investigating health effects of ambient air pollution-related UFP. We included epidemiologic studies containing UFP measures and quantifiable measures of associations. Relevant data were extracted on the basis of previously developed evaluation criteria.
RESULTS	We identified 85 original studies, conducting short-term (n = 75) and long-term (n = 10) investigations. Panel (n = 32), scripted exposure with predefined settings (n = 16) or time series studies (n = 11) were most frequent. Thirty-four studies adjusted for at least one other pollutant. Most consistent associations were identified for short-term effects on pulmonary/systemic inflammation, heart rate variability and blood pressure.
CONCLUSIONS	The evidence suggests adverse short-term associations with inflammatory and cardiovascular changes, which may be at least partly independent of other pollutants. For the other studied health outcomes, the evidence on independent health effects of UFP remains inconclusive or insufficient.

표-678. PubMed 논문번호 30795752의 내용 요약

구분	내용
PubMed ID	30795752
TITLE	Prevalence and factors associated with asthma among adolescents and adults in Uganda: a general population based survey.
JOURNAL	BMC public health: 10.1186/s12889-019-6562-2
AUTHORS	Kirenga Bruce J, de Jong Corina, Katagira Winceslaus, Kasozi Samuel, Mugenyi Levicatus, Boezen Marike, van der Molen Thys, Kamya Moses R
BACKGROUND	Recent large-scale population data on the prevalence of asthma and its risk factors are lacking in Uganda. This survey was conducted to address this data gap.
METHODS	A general population based survey was conducted among people ≥12 years. A questionnaire was used to collect participants socio-demographics, respiratory symptoms, medical history, and known asthma risk factors. Participants who reported wheeze in the past 12 months, a physician diagnosis of asthma or current use of asthma medications were classified as having asthma. Asthmatics who were ≥ 35 years underwent spirometry to determine how many had fixed airflow obstruction (i.e. post bronchodilator forced expiratory volume in one second/forced vital capacity (FEV1/FVC) ratio < lower limit of normal (LLN). Descriptive statistics were used to summarize participants' characteristics. Prevalence of asthma was calculated as a proportion of asthmatics over total survey population. To obtain factors independently associated with asthma, a random-effects model was fitted to the data.
RESULTS	Of the 3416 participants surveyed, 61.2% (2088) were female, median age was 30 years (IQR, 20-45) and 323 were found to have asthma. Sixteen people with asthma ≥35 years had fixed airflow obstruction. The prevalence of asthma was 11.0% (95% CI:8.9-13.2; males 10.3%, females 11.4%, urban 13.0% and rural 8.9%. Significantly more people with asthma smoked than non-asthmatics: 14.2% vs. 6.3%, p < 0.001, were exposed to biomass smoke: 28.0% vs. 20.0%, p < 0.001, had family history of asthma: 26.9% vs. 9.4%, p, < 0.001, had history of TB: 3.1% vs. 1.30%, p = 0.01, and had hypertension: 17.9% vs. 12.0%, p = 0. 003. In multivariate analysis smoking, (adjusted odds ratio (AOR), 3.26 (1.96-5.41, p < 0.001) family history of asthma, AOR 2.90 (98-4.22 p- < 0.001), nasal congestion, AOR 3.56 (2.51-5.06, p < 0.001), biomass smoke exposure, AOR 2.04 (1.29-3.21, p = 0.002) and urban residence, AOR 2.01(1.23-3.27, p = 0.005) were independently associated with asthma.
CONCLUSION	Asthma is common in Uganda and is associated with smoking, biomass smoke exposure, urbanization, and allergic diseases. Health care systems should be strengthened to provide asthma care. Measures to reduce exposure to the identified associated factors are needed.

표-679. PubMed 논문번호 30797734의 내용 요약

구분	내용
PubMed ID	30797734
TITLE	Fine particulate matter and incidence of metabolic syndrome in non-CVD patients: A nationwide population-based cohort study.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2019.01.010
AUTHORS	Lee Seulbi, Park Hyesook, Kim Soontae, Lee Eun-Kyung, Lee Jiyoung, Hong Young Sun, Ha Eunhee
BACKGROUND	It has been reported that particulate matter (PM) is associated with cardiovascular diseases (CVD) while metabolic syndrome is also an important risk factor for CVD. However, few studies have investigated the epidemiological association between PM and metabolic syndrome.
OBJECTIVE	To investigate the association between one-year exposure to PM with an aerodynamic diameter <2.5 μm (PM2.5) and the risk of metabolic syndrome in Korean adults without CVD.
METHODS	Exposure to PM2.5 was assessed using a Community Multiscale Air Quality (CMAQ) model. Metabolic syndrome was defined by National Cholesterol Education Program Adult Treatment Panel III. Andersen and Gill model with time-varying covariates, considering recurrent events, was used to investigate the association between one-year average PM2.5 and the risk of incident metabolic syndrome in 119,998 adults from the national health screening cohort provided by Korea National Health Insurance from 2009 to 2013.
RESULTS	Higher risk of metabolic syndrome, waist-based obesity, hypertension, hypertriglyceridemia, low HDL cholesterol, and hyperglycemia were significantly associated with a 10-μg/m3 increase in PM2.5 [adjusted hazard ratio (HR): 1.070, 1.510, 1.499, 1.468, 1.627 and 1.380, respectively]. In addition, the risk of metabolic syndrome associated with PM2.5 exposure was significant in the consistently obese group (obese at baseline and endpoint).
CONCLUSION	Exposure to one-year average PM2.5 is associated with an increased risk of metabolic syndrome and its components in adults without CVD. These associations are particularly prominent in the consistently obese group (obese at baseline and endpoint). Our findings indicate that PM2.5 affects the onset of MS and its components which may lead to increase the risk of CVD.

표-680. PubMed 논문번호 30799203의 내용 요약

구분	내용
PubMed ID	30799203
TITLE	Association of secondhand tobacco smoke exposure during childhood on adult cardiovascular disease risk among never-smokers.
JOURNAL	Annals of epidemiology: 10.1016/j.annepidem.2019.01.012
AUTHORS	Pistilli Maxwell, Howard Virginia J, Safford Monika M, Lee Brian K, Lovasi Gina S, Cushman Mary, Malek Angela M, McClure Leslie A
PURPOSE	Adult secondhand tobacco smoke (SHS) exposure is related to stroke and coronary heart disease (CHD) risk, but long-term effects are less clear. We evaluated whether childhood SHS exposure affects subsequent stroke or CHD risk among adult black and white never-smokers followed for stroke and CHD.
METHODS	In this prospective cohort study, inverse probability weights were calculated to correct for bias due to attrition and survey nonresponse. Cox proportional hazards models were used to assess hazard ratios and 95% confidence intervals for stroke or CHD, separately, by number of childhood household smokers.
RESULTS	Of 13,142 eligible participants, 6136 had childhood SHS exposure assessed. Baseline mean (SD) age was 63.5 (9.0), 65% were female, 30% black, 46% reported 0 childhood household smokers, 36% reported 1, and 18% reported 2+. In 60,649 person-years, 174 strokes were observed (2.9% of participants), and in 45,195 person-years, 114 CHD events were observed (2.1% of participants). The weighted and adjusted hazard ratios (95% confidence intervals) of stroke for 2+ versus 0 childhood household smokers was 1.66 (1.29-2.13) and was 1.15 (0.82-1.59) for CHD.
CONCLUSIONS	We observed a significant association between childhood SHS exposure and stroke, but not CHD, after age 45 years and adjusting for missing information.

표-681. PubMed 논문번호 30819252의 내용 요약

구분	내용
PubMed ID	30819252
TITLE	Genome-wide DNA methylation and long-term ambient air pollution exposure in Korean adults.
JOURNAL	Clinical epigenetics: 10.1186/s13148-019-0635-z
AUTHORS	Lee Mi Kyeong, Xu Cheng-Jian, Carnes Megan U, Nichols Cody E, Ward James M, Kwon Sung Ok, Kim Sun-Young, Kim Woo Jin, London Stephanie J
BACKGROUND	Ambient air pollution is associated with numerous adverse health outcomes, but the underlying mechanisms are not well understood; epigenetic effects including altered DNA methylation could play a role. To evaluate associations of long-term air pollution exposure with DNA methylation in blood, we conducted an epigenome-wide association study in a Korean chronic obstructive pulmonary disease cohort (N = 100 including 60 cases) using Illumina's Infinium HumanMethylation450K Beadchip. Annual average concentrations of particulate matter ≤ 10 μm in diameter (PM10) and nitrogen dioxide (NO2) were estimated at participants' residential addresses using exposure prediction models. We used robust linear regression to identify differentially methylated probes (DMPs) and two different approaches, DMRcate and comb-p, to identify differentially methylated regions (DMRs).
RESULTS	After multiple testing correction (false discovery rate < 0.05), there were 12 DMPs and 27 DMRs associated with PM10 and 45 DMPs and 57 DMRs related to NO2. DMP cg06992688 (OTUB2) and several DMRs were associated with both exposures. Eleven DMPs in relation to NO2 confirmed previous findings in Europeans; the remainder were novel. Methylation levels of 39 DMPs were associated with expression levels of nearby genes in a separate dataset of 3075 individuals. Enriched networks were related to outcomes associated with air pollution including cardiovascular and respiratory diseases as well as inflammatory and immune responses.
CONCLUSIONS	This study provides evidence that long-term ambient air pollution exposure impacts DNA methylation. The differential methylation signals can serve as potential air pollution biomarkers. These results may help better understand the influences of ambient air pollution on human health.

표-682. PubMed 논문번호 30852444의 내용 요약

구분	내용
PubMed ID	30852444
TITLE	Long-term exposure to ambient fine particulate matter and incidence of diabetes in China: A cohort study.
JOURNAL	Environment international: 10.1016/j.envint.2019.02.069
AUTHORS	Liang Fengchao, Yang Xueli, Liu Fangchao, Li Jianxin, Xiao Qingyang, Chen Jichun, Liu Xiaoqing, Cao Jie, Shen Chong, Yu Ling, Lu Fanghong, Wu Xianping, Zhao Liancheng, Wu Xigui, Li Ying, Hu Dongsheng, Huang Jianfeng, Liu Yang, Lu Xiangfeng, Gu Dongfeng
BACKGROUND	Diabetes caused substantial economic and health burden worldwide. However, the associations between air pollution and diabetes incidence were rarely reported in the developing countries, especially in China with relatively high PM2.5 concentrations.
OBJECTIVES	A cohort-based study was conducted to assess the diabetes incidence associated with long-term exposure to ambient PM2.5.
METHODS	We collected individual health data and risk factors from the project of Prediction for Atherosclerotic Cardiovascular Disease Risk in China (China-PAR Project) from 15 provinces over China. Diabetes was defined as fasting glucose levels ≥7.0 mmol/L at the follow-ups and/or the use of insulin or oral hypoglycemic agents and/or diagnosed medical history of diabetes during 2004 to 2015. Individual-level PM2.5 exposures were estimated from satellite-based PM2.5 concentrations (10 km spatial resolution) during the study period. Cox proportional hazards models with random intercepts of each cohort and region were employed to estimate the diabetes incidence attributable to PM2.5, after the adjustment for age, gender, body mass index, smoking status, education, work-related physical activity level, hypertension, urbanicity, county-level averaged years of education, and long-term levels of temperature and relative humidity.
RESULTS	A total of 88,397 subjects were analyzed with 580,928 person-years of follow-up after 2004, among which 6439 new cases of diabetes were observed. The mean age of the subjects was 51.7 years at baseline. For an increase of 10 μg/m3 in long-term PM2.5 exposure, the multivariable-adjusted percent increase in the diabetes incidence was estimated to be 15.66% (95% confidence interval: 6.42%, 25.70%). The adverse effects of PM2.5 were larger among females, rural subjects, non-smokers, normotensives, subjects younger than 65 years and subjects with body mass index <25 kg/m2.
CONCLUSIONS	Our findings provided evidence for the association of long-term exposure to PM2.5 with diabetes incidence in China. A sustained improvement of air quality will benefit the reduction for diabetes epidemic in China.

표-683. PubMed 논문번호 30860255의 내용 요약

구분	내용
PubMed ID	30860255
TITLE	Cardiovascular disease burden from ambient air pollution in Europe reassessed using novel hazard ratio functions.
JOURNAL	European heart journal: 10.1093/eurheartj/ehz135
AUTHORS	Lelieveld Jos, Klingm□ller Klaus, Pozzer Andrea, P□schl Ulrich, Fnais Mohammed, Daiber Andreas, M□nzel Thomas
AIMS	Ambient air pollution is a major health risk, leading to respiratory and cardiovascular mortality. A recent Global Exposure Mortality Model, based on an unmatched number of cohort studies in many countries, provides new hazard ratio functions, calling for re-evaluation of the disease burden. Accordingly, we estimated excess cardiovascular mortality attributed to air pollution in Europe.
METHODS AND RESULTS	The new hazard ratio functions have been combined with ambient air pollution exposure data to estimate the impacts in Europe and the 28 countries of the European Union (EU-28). The annual excess mortality rate from ambient air pollution in Europe is 790 000 [95% confidence interval (95% CI) 645 000-934 000], and 659 000 (95% CI 537 000-775 000) in the EU-28. Between 40% and 80% are due to cardiovascular events, which dominate health outcomes. The upper limit includes events attributed to other non-communicable diseases, which are currently not specified. These estimates exceed recent analyses, such as the Global Burden of Disease for 2015, by more than a factor of two. We estimate that air pollution reduces the mean life expectancy in Europe by about 2.2 years with an annual, attributable per capita mortality rate in Europe of 133/100 000 per year.
CONCLUSION	We provide new data based on novel hazard ratio functions suggesting that the health impacts attributable to ambient air pollution in Europe are substantially higher than previously assumed, though subject to considerable uncertainty. Our results imply that replacing fossil fuels by clean, renewable energy sources could substantially reduce the loss of life expectancy from air pollution.

표-684. PubMed 논문번호 30862234의 내용 요약

구분	내용
PubMed ID	30862234
TITLE	Association of short- and long-term exposure to air pollution with atrial fibrillation.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487319835984
AUTHORS	Kwon Oh Kyung, Kim Sun-Hwa, Kang Si-Hyuck, Cho Youngjin, Oh Il-Young, Yoon Chang-Hwan, Kim Sun-Young, Kim Ok-Jin, Choi Eue-Keun, Youn Tae-Jin, Chae In-Ho
BACKGROUND	Atrial fibrillation is a common cardiac arrhythmia and an important risk factor for stroke and cardiovascular morbidity. However, there is limited evidence regarding the association of air pollution with atrial fibrillation. This study aimed to compare the short-term and long-term effects of air pollution on atrial fibrillation.
DESIGN	A nationwide cohort from the Korean general population.
METHODS	Different analytical approaches were used for short-term and long-term effects. For the analysis of short-term effects, the daily incidence of emergency admissions for atrial fibrillation was identified. The relationship of atrial fibrillation with air pollutants, including PM2.5 (particulate matter ≤ 2.5 μm in aerodynamic diameter), PM10, carbon monoxide, sulphur dioxide, nitrogen dioxide and ozone, was analysed using a time-series analysis. The long-term effects of air pollution were analysed for subjects aged ≥30 years who resided in Seoul between 2007 and 2015 and had no history of atrial fibrillation.
RESULTS	During the study period, 1137 emergency visits were identified in Seoul as being associated with atrial fibrillation. A 10- μg/m3 increase in ambient PM2.5 was shown to significantly increase emergency admissions by 4.5% at lag day 3 (p = 0.038). No other pollutants showed a significant relationship with emergency atrial fibrillation admission. Among 124,010 residents in Seoul, 1903 developed atrial fibrillation at a median follow-up of 9.5 years (1.95 per 1000 person-years). Long-term exposure to air pollution had no significant impact on atrial fibrillation occurrence (p = 0.830 for PM2.5).
CONCLUSION	This study suggests that short-term exposure to PM2.5 triggers atrial fibrillation. However, we found no evidence linking atrial fibrillation with long-term exposure to air pollution.

표-685. PubMed 논문번호 30896633의 내용 요약

구분	내용
PubMed ID	30896633
TITLE	The relationship between smoking and stroke: A meta-analysis.
JOURNAL	Medicine: 10.1097/MD.0000000000014872
AUTHORS	Pan Biqi, Jin Xiao, Jun Liu, Qiu Shaohong, Zheng Qiuping, Pan Mingwo
BACKGROUND	Stroke is one of the leading causes of death and disability for adult men and women worldwide, and a number of studies have explored the influences of smoking on stroke. However, few studies have discussed the relationship between stroke and smoking with consideration of the following factors: sex, the number of cigarettes smoked per day, stroke subtype, and the follow-up duration. Consequently, we aimed to extend previous work by using a systematic review to explore the relationship between stroke and cigarette smoking in reference to the above factors.
METHODS	A systematic review was conducted using the PubMed, Embase, and Cochrane Central Register databases and the following search criteria: ["stroke" (MeSH) and "smoking" (MeSH)]. All analyses were conducted with Stata, and funnel plots and Egger regression asymmetry tests were used to assess publication bias.
RESULTS	The meta-analysis included 14 studies involving 303134 subjects. According to the meta-analysis, smokers had an overall increased risk of stroke compared with nonsmokers, with a pooled odds ratio (OR) of 1.61 (95% confidence interval [CI]: 1.34-1.93, P<.001). A subgroup analysis conducted based on smoking status revealed ORs of 1.92 (95% CI: 1.49-2.48) for current smokers and 1.30 (95% CI: 0.93-1.81) for former smokers. In addition, the relationship between stroke of any type and smoking status was also statistically significant; current smokers had an increased risk of stoke compared with nonsmokers (OR: 1.46, 95% CI: 1.04-2.07, P<.001), which was influenced by sex (men: OR: 1.54, 95% CI: 1.11-2.13, P=.002; women: OR: 1.88, 95% CI: 1.45-2.44, P<.023). From the analysis, we also observed that passive smoking increased the overall risk of stroke by 45% (OR: 1.45, 95% CI: 1.0-2.11, P<.05). Based on the dose-response meta-analysis, the risk of stroke increased by 12% for each increment of 5 cigarettes per day.

표-686. PubMed 논문번호 30905164의 내용 요약

구분	내용
PubMed ID	30905164
TITLE	Parental smoking and the risk of congenital heart defects in offspring: An updated meta-analysis of observational studies.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487319831367
AUTHORS	Zhao Lijuan, Chen Lizhang, Yang Tubao, Wang Lesan, Wang Tingting, Zhang Senmao, Chen Letao, Ye Ziwei, Zheng Zan, Qin Jiabi, Zhao Lijuan, Chen Lizhang, Yang Tubao, Wang Lesan, Wang Tingting, Zhang Senmao, Chen Letao, Ye Ziwei, Zheng Zan, Qin Jiabi
OBJECTIVE	Although previous reviews confirmed maternal active smoking was significantly associated with risk of fetal congenital heart defects (CHDs), association between maternal passive smoking and paternal smoking and risk of CHDs is inconclusive nowadays; furthermore, a complete overview is lacking. A meta-analysis of observational studies was conducted to assess the risk of CHDs associated with maternal active and passive smoking and paternal smoking.
METHODS	Seven electronic databases were searched for qualified research up to June 2018. We summarized study characteristics and the summary risk estimates were calculated using either the random-effect model or fixed-effect model. Sensitivity and subgroup analysis were carried out to identify the potential heterogeneity moderators.
RESULTS	One hundred and twenty-five studies involving 137,574 CHDs cases in 8,770,837 study participants were included. Overall, maternal active (risk ratio (RR) = 1.25; 95% confidence interval (CI): 1.16-1.34; p < 0.01) and passive (RR = 2.24, 95% CI: 1.81-2.77; p < 0.01) smoking as well as paternal active smoking (RR = 1.74, 95% CI: 1.48-2.06; p < 0.01) were significantly associated with CHDs risk. For specific CHD subtypes, our study showed that maternal active smoking was significantly associated with risk of atrial septal defect (RR = 1.27, 95% CI: 1.02-1.59; p = 0.03) and right ventricular outflow tract obstruction (RR = 1.43, 95% CI: 1.04-1.97; p = 0.03). Relevant heterogeneity moderators have been identified by subgroup analysis. Sensitivity analysis yielded consistent results.
CONCLUSION	Maternal active smoking, maternal passive smoking as well as paternal smoking all increased the risk of CHDs in offspring. Preventing parental smoking during peri-pregnancy is a priority for CHDs prevention.

표-687. PubMed 논문번호 30910154의 내용 요약

구분	내용
PubMed ID	30910154
TITLE	Characteristics of patients with endometrial hyperplasia under different air quality index conditions.
JOURNAL	Taiwanese journal of obstetrics & gynecology: 10.1016/j.tjog.2019.01.021
AUTHORS	Chang Fung-Wei, Hsu Ren-Jun, Liu Shu-Hui
OBJECTIVE	Air pollution has been widely recognized to pose a threat to health. Urban outdoor air pollution was listed as the 14th biggest risk factor for global deaths in 2004 in the Global Health Risks report published by the World Health Organization in 2009. Many past studies have indicated that exposure to environmental contaminants promotes changes in the internal mechanisms of diseases, including the infection of various systems in the body, hormonal changes, and vascular proliferation. These changes may be related to the severity of endometrial hyperplasia. Therefore, this study used the air quality monitoring data of the Environmental Protection Administration (EPA) to examine the effects of air pollutant concentration on patients with endometrial hyperplasia.
MATERIALS AND METHODS	This population-based nationwide study used data for 2002-2013 from the National Health Insurance Research Database of Taiwan. Patients who developed endometrial hyperplasia before 2002 were excluded. In total, 14,883 patients with endometrial hyperplasia were tracked. The exposure levels and air quality index (AQI) values in this study were based on the taiwan air quality monitoring network data collected by the EPA from 2000 to 2013. The data were further divided into the good air quality group ($AQI \leq 50$) and poor air quality group ($AQI > 50$). The study used linear regression model to estimate the correlation linking air pollutant concentration with endometrial hyperplasia.
RESULTS	The results indicated that, in comparison to endometrial hyperplasia patients who were exposed to air with good quality, those exposed to air with poor quality had a higher average age ($p < 0.001$) and higher proportion of living in southern Taiwan ($p < 0.001$), as well as higher rates of diabetes ($p < 0.001$), hyperlipidemia ($p < 0.001$), hypertension, cerebrovascular diseases ($p = 0.024$), cerebral vascular accidents ($p = 0.024$), and chronic kidney disease ($p < 0.001$).
CONCLUSION	The patients with endometrial hyperplasia in poor AQI area had severe comorbidity. Thus, attention must be paid to the improvement of air quality and the implementation of preventive measures against contaminants.

표-688. PubMed 논문번호 30922502의 내용 요약

구분	내용
PubMed ID	30922502
TITLE	Analysis of the Seasonal Trend of Congenital Heart Defects.
JOURNAL	The Journal of pediatrics: 10.1016/j.jpeds.2018.12.024
AUTHORS	Dong Yuanyuan, Zhang Yunting, Tong Shilu, Jiang Zhongyi, Xu Zhiwei, Li Xinyue, Wang Wei
OBJECTIVE	To determine the seasonal trend of congenital heart defects (CHDs) in China using hospital-based clinical data.
STUDY DESIGN	We included 40 501 patients with CHD hospitalized at the Shanghai Children Medical Center between 2006 and 2017. The birth rate of CHD in each month was adjusted by sex, year of birth, and monthly birth rate of the general population. Negative binomial regression models were used to assess the seasonal trend of CHD.
RESULTS	The included patients consisted of 22 600 boys (55.8%), resulting in a male-to-female ratio of 1.26:1. Among subtypes of CHDs, ventricular septal defects and atrial septal defects were the most common, accounting for 39.7% and 12.6%, respectively. A statistically significant seasonal trend in the monthly birth rate of patients with CHDs was found; the highest relative rate of CHD was found in October and the lowest in April. After adjusting for the potential confounders, the highest relative rate of CHD was found in October and the lowest in November.
CONCLUSIONS	There seems to be a significant monthly birth rate variation of CHDs in China. The highest relative rate of CHDs occurred in October, suggesting possible maternal exposure to environmental hazards from January to March. These hazards may include air pollution, virus infection, and unhealthy lifestyle behaviors during the Spring Festival.

표-689. PubMed 논문번호 30953813의 내용 요약

구분	내용
PubMed ID	30953813
TITLE	A systematic review of cardiovascular responses associated with am carbon and fine particulate matter.
JOURNAL	Environment international: 10.1016/j.envint.2019.02.027
AUTHORS	Kirrane E F, Luben T J, Benson A, Owens E O, Sacks J D, Dutton Madden M, Nichols J L
BACKGROUND	Exposure to fine particulate matter (PM2.5), an ambient air polluta mass-based standards promulgated under the Clean Air Act, and b carbon (BC), a common component of PM2.5, are both associated cardiovascular health effects.
OBJECTIVES	To elucidate whether BC is associated with distinct, or stronger, cardiovascular responses compared to PM2.5, we conducted a syst review. We evaluated the associations of short- and long-term BC related component elemental carbon (EC), with cardiovascular end including heart rate variability, heart rhythm, blood pressure and v function, ST segment depression, repolarization abnormalities, athe and heart function, in the context of what is already known about
DATA SOURCES	We conducted a stepwise systematic literature search of the PubMe Science and TOXLINE databases and applied Preferred Reporting Systematic Review and Meta-Analyses (PRISMA) guidelines for re results.
STUDY ELIGIBILITY CRITERIA	Studies reporting effect estimates for the association of quantitative measurements of ambient BC (or EC) and PM2.5, with relevant car endpoints (i.e. meeting inclusion criteria) were included in the revie Included studies were evaluated for risk of bias in study design and
STUDY APPRAISAL AND SYNTHESIS METHODS	Risk of bias evaluations assessed aspects of internal validity of stud based on study design, conduct, and reporting to identify potential related to confounding or other biases. Study results are presented comparison of the consistency of associations with PM2.5 and BC across studies.
RESULTS	Our results demonstrate similar associations for BC (or EC) and PM the cardiovascular endpoints examined. Across studies, association and PM2.5 varied in their magnitude and precision, and confidence were generally overlapping within studies. Where differences in the of the association between BC or EC and PM2.5 within a study co discerned, no consistent pattern across the studies examined was ap
LIMITATIONS	We were unable to assess the independence of the effect of BC, rela effect of PM2.5, on the cardiovascular system, nor was information to understand the impact of differential exposure misclassification.
CONCLUSIONS	Overall, the evidence indicates that both BC (or EC) and PM2.5 are with cardiovascular effects but the available evidence is not sufficie distinguish the effect of BC (or EC) from that of PM2.5 mass.

표-690. PubMed 논문번호 31010894의 내용 요약

구분	내용
PubMed ID	31010894
TITLE	Effects of occupational exposure to respirable quartz dust on acute myocardial infarction.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2018-105540
AUTHORS	Gellissen Johannes, Pattloch Dagmar, M□hner Matthias
OBJECTIVES	The aim of this study is to investigate the effects of occupational exposure to respirable quartz (RQ) on first acute myocardial infarction (AMI). RQ causes pulmonary diseases like silicosis and has also been linked to cardiovascular diseases. Inflammation is hypothesised as the underlying pathway.
METHODS	We performed a 1:3 matched case-control study nested in a cohort of male uranium miners. We included cases (identified from hospital records and validated according to WHO criteria) who had suffered their first AMI while still employed and <65 years of age. Controls were matched by date of birth and Wismut recruitment era. RQ exposure was derived from a job-exposure matrix. We performed a conditional logistic regression adjusted for smoking, metabolic syndrome and baseline erythrocyte sedimentation rate. Subgroups by date of birth and Wismut recruitment era were analysed to minimise the impact of pre-exposures.
RESULTS	The study base comprised 292 matched sets. The cumulative exposure ranged from 0 to 38.9 mg/m3-years RQ. The adjusted OR of the highest RQ tertile (>14.62 mg/m3-years) was 1.27 (95% CI 0.82 to 1.98). However, for miners born after 1928 and hired in the earliest recruitment era (1946-1954), a significantly elevated risk was seen in the highest RQ tertile (OR=6.47 [95% CI 1.33 to 31.5]; 50 matched sets).
CONCLUSIONS	An impact of quartz dust on first AMI was observed only in a small subgroup that had virtually no pre-exposure to RQ. Further studies on the basis of complete occupational history are required to substantiate this finding.

표-691. PubMed 논문번호 31021373의 내용 요약

구분	내용
PubMed ID	31021373
TITLE	Association of Air Pollution With Increased Risk of Peritonsillar Abscess Formation.
JOURNAL	JAMA otolaryngology- head & neck surgery: 10.1001/jamaoto.2019.0742
AUTHORS	Kim So Young, Kong Il Gyu, Min Chanyang, Choi Hyo Geun
Importance	Several studies reported an association between peritonsillar abscess forma and climate conditions, including seasonal changes; however, the results w inconsistent.
Objective	To evaluate the association between meteorological conditions and/or air pollution and peritonsillar abscess formation.
Design, Setting, and Participants	In this nested case-control study, 3819 participants with peritonsillar abscesses were matched (1:4) for age, sex, income, region of residence, hypertension, diabetes, and dyslipidemia with 15 276 control participants. Korean Health Insurance Review and Assessment Service-National Sampl Cohort (HIRA-NSC) data from 2002 through 2013 were used.
Exposures and Main Outcomes and Measures	The meteorological data included the mean daily temperature (° C), highe daily temperature (° C), lowest daily temperature (° C), daily temperature difference (° C), relative humidity (%), spot atmospheric pressure (hPa), sulfur dioxide ([SO2], parts per million [ppm]), nitrogen dioxide (NO2, pp ozone (O3, ppm), carbon monoxide (CO, ppm), and particulate matter le than 10 μg (PM10, μg/m3) for the previous 14 days, 10 days, 7 days, 5 d or 3 days before the matched index date. These factors were measured in or 273 locations hourly. The crude and adjusted odds ratios (aORs) and 9 confidence intervals (CIs) of meteorological data for peritonsillar abscess formation were analyzed using unconditional logistic regression analysis. Subgroup analyses were conducted according to age and sex.
Results	The male to female ratio of study participants was 1.43 (11 260 to 7835). Because the age groups were classified using 5-year intervals, the mean ag could not be defined. The mean differences of NO2 and PM10 concentrat for the 14 days between peritonsillar abscess group and control group wer 1.78 ppb (95% CI, 1.47-2.09) and 1.33 μg/m3 (95% CI, 0.67-1.99), respectively. The aORs of NO2 (0.1 ppm) and PM10 (10 μg/m3) during 14 days prior to the index date for peritonsillar abscess formation were 12 (95% CI, 8.4-19.5) and 1.04 (95% CI, 1.02-1.06), respectively. The other meteorological conditions did not reach statistical significance.
Conclusions and Relevance	Peritonsillar abscess formation was associated with high concentrations of NO2 and PM10.

표-692. PubMed 논문번호 31021973의 내용 요약

구분	내용
PubMed ID	31021973
TITLE	Association between maternal exposure to pollutant particulate matter 2.5 and congenital heart defects: a systematic review.
JOURNAL	JBI database of systematic reviews and implementation reports: 10.11124/JBISRIR-2017-003881
AUTHORS	Hall Katie C, Robinson Jennifer C
OBJECTIVE	The objective of this review was to determine if there was an association between maternal exposure to pollutant particulate matter 2.5 during the first trimester of pregnancy and congenital heart defects within the first year of life.
INTRODUCTION	The environment is recognized as an important determinant of health for both the individual and population. Air pollution specifically is a major environmental risk factor impacting health with links to asthma, heart disease, obesity, and fetal developmental complications. Of the commonly monitored air pollutants, particulate matter 2.5 has associations with health, especially among vulnerable populations such as children and pregnant women. A congenital heart defect is a fetal complication that impacts 34.3 million infants globally, with more than 80% of the diagnoses having an unknown etiology. Although environmental risk factors such as air pollution are thought to be a risk factor in the diagnosis of a congenital heart defect, epidemiologic research evidence is limited.
INCLUSION CRITERIA	This review considered studies that evaluated maternal exposure to the air pollutant particulate matter 2.5 during the first trimester (weeks 1-12) of fetal development. The primary outcome was a diagnosis of a congenital heart defect in an infant within the first year of life.
METHODS	A three-step search strategy was utilized in this review and included 11 databases and two websites. Studies published from January 2002 to September 2018 were eligible for inclusion. Only papers published in English were included. Eligible studies underwent critical appraisal by two independent reviewers using standardized critical appraisal instruments from JBI. Quantitative data were extracted from the included studies independently by two reviewers. Odds ratios (ORs) and 95% confidence intervals (CIs) were extracted for the individual outcome measures, specifically atrial septal defect, ventricular septal defect, and tetralogy of fallot, respectively. The defects were identified and pooled, where possible, in statistical meta-analysis. Where statistical pooling was not possible, findings were reported narratively.
RESULTS	Five studies were identified that met the inclusion criteria, including three cohort and two case-control designs. Each individual study identified at least one statistically significant increase or inverse association between particulate matter 2.5 and a congenital heart defect. An increased risk was identified with more than seven isolated and two groupings of congenital heart defects. An inverse risk was identified with two isolated and one grouping of congenital heart defects. Meta-analysis results were: atrial septal defect, OR = 0.65 (95% CI, 0.37 to 1.15); ventricular septal defect, OR=1.02 (95% CI, 075 to 1.37); and tetralogy of fallot, OR=1.16 (95% CI, 0.78 to 1.73), indicating no statistically significant findings.
CONCLUSION	There was no significant evidence to support an association between air pollutant particulate matter 2.5 and a congenital heart defect in the first year of life. However, few studies met the rigorous inclusion criteria, and the studies that did had high heterogeneity, making it difficult to complete a

표-693. PubMed 논문번호 31023432의 내용 요약

구분	내용
PubMed ID	31023432
TITLE	Cardiovascular Benefits of Fish-Oil Supplementation Against Fine Particulate Air Pollution in China.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2018.12.093
AUTHORS	Lin Zhijing, Chen Renjie, Jiang Yixuan, Xia Yongjie, Niu Yue, Wang Cuiping, Liu Cong, Chen Chen, Ge Yihui, Wang Weidong, Yin Guanjin, Cai Jing, Clement Viviane, Xu Xiaohui, Chen Bo, Chen Honglei, Kan Haidong
BACKGROUND	Few studies have evaluated the health benefits of omega-3 fatty acid supplementation against fine particulate matter (aerodynamic diameter <2.5 μm [PM2.5]) exposure in highly polluted areas.
OBJECTIVES	The authors sought to evaluate whether dietary fish-oil supplementation protects cardiovascular health against PM2.5 exposure in China.
METHODS	This is a randomized, double-blinded, and placebo-controlled trial among 65 healthy college students in Shanghai, China. Participants were randomly assigned to either the placebo group or the intervention group with dietary fish-oil supplementation of 2.5 g/day from September 2017 to January 2018, and received 4 rounds of health examinations in the last 2 months of treatments. Fixed-site PM2.5 concentrations on campus were measured in real time. The authors measured blood pressure and 18 biomarkers of systematic inflammation, coagulation, endothelial function, oxidative stress, antioxidant activity, cardiometabolism, and neuroendocrine stress response. Acute effects of PM2.5 on these outcomes were evaluated within each group using linear mixed-effect models.
RESULTS	The average PM2.5 level was 38 μg/m3 during the study period. Compared with the placebo group, the fish-oil group showed relatively stable levels of most biomarkers in response to changes in PM2.5 exposure. Between-group differences associated with PM2.5 exposure varied by biomarkers and by lags of exposure. The authors observed beneficial effects of fish-oil supplementation on 5 biomarkers of blood inflammation, coagulation, endothelial function, oxidative stress, and neuroendocrine stress response in the fish-oil group at a false discovery rate of <0.05.
CONCLUSIONS	This trial shows that omega-3 fatty acid supplementation is associated with short-term subclinical cardiovascular benefits against PM2.5 exposure among healthy young adults in China. (Effect of Dietary Supplemental Fish Oil in Alleviating Health Hazards Associated With Air Pollution; NCT03255187).

표-694. PubMed 논문번호 31030900의 내용 요약

구분	내용
PubMed ID	31030900
TITLE	Air pollution and humidity as triggering factors for stroke. Results of a 12-year analysis in the West Paris area.
JOURNAL	Revue neurologique: 10.1016/j.neurol.2019.03.002
AUTHORS	Hirel C, Berton L, Preda C, Richard O, Lambert Y, Pico F
BACKGROUND AND PURPOSE	Previous studies have suggested an association between stroke and meteorological factors, air pollution and acute respiratory infections as triggering factors. Often, these factors have been evaluated separately. We evaluated the association between all these environmental triggering factors and calls for suspected stroke in a suburb in west Paris from 2004 to 2015.
METHODS	We used data from the emergency medical dispatching center of all calls for suspected stroke (SAMU 78), climatic parameters (M□t□oFrance), pollution (AIRPARIF), and data from influenza epidemic surveillance networks (GROG and Sentinelles). The association between short-term exposure (1-day lag) to environmental triggering factors and stroke occurrence was analyzed using negative-binomial log linear regression model for counting time series.
RESULTS	Between 2004 and 2015, a total of 11,037 calls for suspected stroke were recorded. In bivariate analysis, there were associations between calls for suspected stroke and temperature (mean, maximum and minimum), humidity and influenza epidemic. In multivariable analysis, only two variables were associated with calls for suspected stroke: humidity [3.93% excess relative risk (ERR) of stroke per 10% increase in humidity; 95% confidence interval (CI), 1.42 to 6.51; P<0.002] and pollution on the "Air Parif Atmo" scale (2.86% ERR of stroke per 1 unit increase; 95% CI, 1.01 to 4.75; P=0.002).
CONCLUSIONS	This study suggests that short-term exposure to air pollution and a high level of humidity are associated with a significant excess relative risk of calls for suspected stroke.

표-695. PubMed 논문번호 31035115의 내용 요약

구분	내용
PubMed ID	31035115
TITLE	Associations of long-term exposure to ambient PM〈sub〉1〈/sub〉 with hypertension and blood pressure in rural Chinese population: The Henan rural cohort study.
JOURNAL	Environment international: 10.1016/j.envint.2019.04.037
AUTHORS	Li Na, Chen Gongbo, Liu Feifei, Mao Shuyuan, Liu Yisi, Hou Yitan, Lu Yuanan, Liu Suyang, Wang Chongjian, Xiang Hao, Guo Yuming, Li Shanshan
BACKGROUND	The epidemiological evidence on relationships between long-term exposure to particulate matter and hypertension and blood pressure has been inconclusive. Limited evidence was available for particulate matter with an aerodynamic diameter ≤ 1 μm (PM1) in rural areas of developing countries.
OBJECTIVE	This study aimed to investigate the associations between long-term exposure to PM1 and hypertension and blood pressure among rural Chinese population.
METHODS	This study included 39,259 participants who had completed the baseline survey from Henan Rural Cohort. Participants' exposure to PM1 was assessed by a satellite-based spatiotemporal model. The binary logistic regression model was used to examine the association between long-term PM1 exposure and hypertension, and multivariable linear regression model was used to investigate the associations between long-term PM1 exposure and systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and pulse pressure (PP). Moreover, we examined potential effect modifications by demographic, lifestyle and diet factors.
RESULTS	The mean concentration of PM1 for all participants during the 3-year before baseline survey was 59.98 μg/m3. Each 1 μg/m3 increase in PM1 concentration was significantly associated with an increase of 4.3% [Odds ratio(OR) = 1.043, 95% confidence interval(CI): 1.033, 1.053] in odds for hypertension, an increase of 0.401 mm Hg (95% CI, 0.335, 0.467), 0.328 mm Hg (95% CI, 0.288, 0.369), 0.353 mm Hg (95% CI, 0.307, 0.399) and 0.073 mm Hg (95% CI, 0.030, 0.116) in SBP, DBP, MAP and PP, respectively. Further stratified analyses showed that the effect of PM1 on hypertension and blood pressure could be modified by sex, lifestyle and diet.
CONCLUSIONS	This study suggests that long-term exposure to ambient PM1 increases the risk of hypertension and is associated with elevations in blood pressure in rural Chinese adults, especially in male and those with unhealthy habits.

표-696. PubMed 논문번호 31036481의 내용 요약

구분	내용
PubMed ID	31036481
TITLE	Traffic noise and other determinants of blood pressure in adolescence.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2019.04.012
AUTHORS	Enoksson Wallas Alva, Eriksson Charlotta, Edstedt Bonamy Anna-Karin, Gruzieva Olena, Kull Inger, □gren Mikael, Pyko Andrei, Sj□str□m Mattias, Pershagen G□ran
BACKGROUND	Exposure to traffic noise has been associated with hypertension in adults but the evidence in adolescents is limited. We investigated long-term road traffic noise exposure, maternal occupational noise during pregnancy and other factors in relation to blood pressure and prehypertension at 16 years of age.
METHODS	Systolic and diastolic blood pressure were measured in 2597 adolescents from the Swedish BAMSE birth cohort. Levels of road traffic noise were estimated at home addresses during lifetime and for the mother during pregnancy as well as maternal occupational noise exposure during pregnancy. Exposure to NOx from local sources was also assessed. Associations between noise or NOx exposure and blood pressure or prehypertension were analysed using linear and logistic regression.
RESULTS	The prevalence of prehypertension was higher among males and in those with overweight, low physical activity or overweight mothers. No strong or consistent associations were observed between pre- or postnatal exposure to road traffic noise and blood pressure at 16 years of age. However, inverse associations were suggested for systolic or diastolic blood pressure and prehypertension, which reached statistical significance among males (OR 0.80 per 10 dB Lden, 95% CI 0.65-0.99) and those with maternal occupational noise exposure ≥ 70 dB LAeq8h (OR 0.60, 95% CI 0.41-0.87). On the other hand, occupational noise exposure during pregnancy tended to increase systolic blood pressure and prehypertension risk in adolescence. No associations were seen for NOx exposure.
CONCLUSION	No conclusive associations were observed between pre- or postnatal noise exposure and blood pressure or prehypertension in adolescents.

표-697. PubMed 논문번호 31038389의 내용 요약

구분	내용
PubMed ID	31038389
TITLE	Air Pollution and Angioedema.
JOURNAL	Otolaryngology-head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery: 10.1177/0194599819846446
AUTHORS	Kedarisetty Suraj, Jones Evan, Tint Derrick, Soliman Ahmed M S
OBJECTIVE	To identify environmental factors that may precipitate angioedema.
STUDY DESIGN	Case series with chart review.
SETTING	An urban tertiary care medical center.
SUBJECTS AND METHODS	After institutional review board approval was obtained, a chart review of all patients who presented to Temple University Hospital with angioedema from January 2012 to December 2014 was performed. Patient demographics and hospital course were gathered. Environmental data on the dates of presentation, including precipitation, humidity, and air pollution, were obtained from regional Environmental Protection Agency online data banks and used for statistical analysis.
RESULTS	In total, 408 patients, representing 450 episodes of angioedema, met the inclusion criteria for the study. Most patients were female (58%), African American (74%), and on an angiotensin-converting enzyme inhibitor (ACE-I) upon presentation (61%). Days with unhealthy levels of air pollution were associated with an increased likelihood of angioedema presentation (odds ratio [OR], 1.82; confidence interval [CI], 1.01-3.30; P = .046). Subgroup analysis revealed that elevated ground-level ozone was the primary air pollutant significantly associated with angioedema presentations (OR, 4.95; CI 1.92-12.76; P = .0009). Higher ground-level ozone was also associated with ACE-I angioedema presentations (P = .017) but not with non-ACE-I angioedema presentations (P = .86). Air quality was not predictive of angioedema severity or need for intubation.
CONCLUSION	Angioedema is a complex, multifactorial disease resulting in potentially life-threatening complications. This is the first study to demonstrate that higher levels of air pollution, specifically ground-level ozone, are associated with significantly increased rates of angioedema episodes, although not severity.

표-698. PubMed 논문번호 31063398의 내용 요약

구분	내용
PubMed ID	31063398
TITLE	Long-Term Exposure to Ambient Ozone and Progression of Subclinical Arterial Disease: The Multi-Ethnic Study of Atherosclerosis and Air Pollution.
JOURNAL	Environmental health perspectives: 10.1289/EHP3325
AUTHORS	Wang Meng, Sampson Paul D, Sheppard Lianne E, Stein James H, Vedal Sverre, Kaufman Joel D
BACKGROUND	Long-term ozone ([Formula: see text]) exposure is associated with cardiovascular mortality, but little is known about the associations between [Formula: see text] and subclinical arterial disease.
OBJECTIVES	We studied the longitudinal association of exposure to [Formula: see text] and progression of key subclinical arterial markers in adults: intima-media thickness of common carotid artery ([Formula: see text]), carotid plaque (CP) burden, and coronary artery calcification (CAC).
METHODS	CAC was measured one to four times at baseline and at follow-up exams (1999-2012) by computed tomography (CT) in 6,619 healthy adults, recruited at age 45-84 y without cardiovascular disease (CVD), over a mean of 6.5 y (standard deviation: 3.5 y). [Formula: see text] and CP burden were quantified in 3,392 participants using carotid artery ultrasound imaging acquired over a mean of 9 y (1.7 y). Over 91% and 89% participants had at least one follow-up [Formula: see text] and CAC measurement, respectively. Residence-specific [Formula: see text] concentrations were estimated by a validated spatiotemporal model spanning from 1999 to 2012. This model relied on comprehensive monitoring data and geographical variables to predict individualized long-term average concentrations since baseline. Linear mixed models and logistic regression model were used to evaluate relationships of long-term average exposure to [Formula: see text] with longitudinal change in [Formula: see text], CAC, and CP formation, respectively.
RESULTS	Mean progression rates of [Formula: see text] and CAC were [Formula: see text] and [Formula: see text]. CP formation was identified in 55% of the subjects. A [Formula: see text] increase in long-term average [Formula: see text] exposure was associated with a [Formula: see text] [95% confidence interval (CI): 1.4, 9.7] greater increase in [Formula: see text] over 10 y. A [Formula: see text] increase in [Formula: see text] was also associated with new CP formation [odds ratio (OR): 1.2 (95% CI: 1.1, 1.4)] but not CAC progression [[Formula: see text] (95% CI: [Formula: see text], 2)]. Associations were robust in the analysis with extended covariate adjustment, including copollutants, i.e., nitrogen oxides ([Formula: see text]) and particulate matter with diameter [Formula: see text] ([Formula: see text]).
CONCLUSION	Over almost a decade of follow-up, outdoor [Formula: see text] concentrations were associated with increased rate of carotid wall thickness progression and risk of new plaque formation, suggesting arterial injury in this cohort. https://doi.org/10.1289/EHP3325.

표-699. PubMed 논문번호 31067132의 내용 요약

구분	내용
PubMed ID	31067132
TITLE	Health Effects of Household Solid Fuel Use: Findings from 11 Countries within the Prospective Urban and Rural Epidemiology Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP3915
AUTHORS	Hystad Perry, Duong MyLinh, Brauer Michael, Larkin Andrew, Arku Raphael, Kurmi Om P, Fan Wen Qi, Avezum Alvaro, Azam Igbal, Chifamba Jephat, Dans Antonio, du Plessis Johan L, Gupta Rajeev, Kumar Rajesh, Lanas Fernando, Liu Zhiguang, Lu Yin, Lopez-Jaramillo Patricio, Mony Prem, Mohan Viswanathan, Mohan Deepa, Nair Sanjeev, Puoane Thandi, Rahman Omar, Lap Ah Tse, Wang Yanga, Wei Li, Yeates Karen, Rangarajan Sumathy, Teo Koon, Yusuf Salim
BACKGROUND	Household air pollution (HAP) from solid fuel use for cooking affects 2.5 billion individuals globally and may contribute substantially to disease burden. However, few prospective studies have assessed the impact of HAP on mortality and cardiorespiratory disease.
OBJECTIVES	Our goal was to evaluate associations between HAP and mortality, cardiovascular disease (CVD), and respiratory disease in the prospective urban and rural epidemiology (PURE) study.
METHODS	We studied 91,350 adults 35-70 y of age from 467 urban and rural communities in 11 countries (Bangladesh, Brazil, Chile, China, Colombia, India, Pakistan, Philippines, South Africa, Tanzania, and Zimbabwe). After a median follow-up period of 9.1 y, we recorded 6,595 deaths, 5,472 incident cases of CVD (CVD death or nonfatal myocardial infarction, stroke, or heart failure), and 2,436 incident cases of respiratory disease (respiratory death or nonfatal chronic obstructive pulmonary disease, pulmonary tuberculosis, pneumonia, or lung cancer). We used Cox proportional hazards models adjusted for individual, household, and community-level characteristics to compare events for individuals living in households that used solid fuels for cooking to those using electricity or gas.
RESULTS	We found that 41.8% of participants lived in households using solid fuels as their primary cooking fuel. Compared with electricity or gas, solid fuel use was associated with fully adjusted hazard ratios of 1.12 (95% CI: 1.04, 1.21) for all-cause mortality, 1.08 (95% CI: 0.99, 1.17) for fatal or nonfatal CVD, 1.14 (95% CI: 1.00, 1.30) for fatal or nonfatal respiratory disease, and 1.12 (95% CI: 1.06, 1.19) for mortality from any cause or the first incidence of a nonfatal cardiorespiratory outcome. Associations persisted in extensive sensitivity analyses, but small differences were observed across study regions and across individual and household characteristics.
DISCUSSION	Use of solid fuels for cooking is a risk factor for mortality and cardiorespiratory disease. Continued efforts to replace solid fuels with cleaner alternatives are needed to reduce premature mortality and morbidity in developing countries. https://doi.org/10.1289/EHP3915.

표-700. PubMed 논문번호 31075749의 내용 요약

구분	내용
PubMed ID	31075749
TITLE	Complex relationships between greenness, air pollution, and mortality in a population-based Canadian cohort.
JOURNAL	Environment international: 10.1016/j.envint.2019.04.047
AUTHORS	Crouse Dan L, Pinault Lauren, Balram Adele, Brauer Michael, Burnett Richard T, Martin Randall V, van Donkelaar Aaron, Villeneuve Paul J, Weichenthal Scott
BACKGROUND	Epidemiological studies have consistently demonstrated that exposure to fine particulate matter (PM2.5) is associated with increased risks of mortality. To a lesser extent, a series of studies suggest that living in greener areas is associated with reduced risks of mortality. Only a handful of studies have examined the interplay between PM2.5, greenness, and mortality.
METHODS	We investigated the role of residential greenness in modifying associations between long-term exposures to PM2.5 and non-accidental and cardiovascular mortality in a national cohort of non-immigrant Canadian adults (i.e., the 2001 Canadian Census Health and Environment Cohort). Specifically, we examined associations between satellite-derived estimates of PM2.5 exposure and mortality across quintiles of greenness measured within 500 m of individual's place of residence during 11 years of follow-up. We adjusted our survival models for many personal and contextual measures of socioeconomic position, and residential mobility data allowed us to characterize annual changes in exposures.
RESULTS	Our cohort included approximately 2.4 million individuals at baseline, 194,270 of whom died from non-accidental causes during follow-up. Adjustment for greenness attenuated the association between PM2.5 and mortality (e.g., hazard ratios (HRs) and 95% confidence intervals (CIs) per interquartile range increase in PM2.5 in models for non-accidental mortality decreased from 1.065 (95% CI: 1.056-1.075) to 1.041 (95% CI: 1.031-1.050)). The strength of observed associations between PM2.5 and mortality decreased as greenness increased. This pattern persisted in models restricted to urban residents, in models that considered the combined oxidant capacity of ozone and nitrogen dioxide, and within neighbourhoods characterised by high or low deprivation. We found no increased risk of mortality associated with PM2.5 among those living in the greenest areas. For example, the HR for cardiovascular mortality among individuals in the least green areas was 1.17 (95% CI: 1.12-1.23) compared to 1.01 (95% CI: 0.97-1.06) among those in the greenest areas.
CONCLUSIONS	Studies that do not account for greenness may overstate the air pollution impacts on mortality. Residents in deprived neighbourhoods with high greenness benefitted by having more attenuated associations between PM2.5 and mortality than those living in deprived areas with less greenness. The findings from this study extend our understanding of how living in greener areas may lead to improved health outcomes.

표-701. PubMed 논문번호 31092656의 내용 요약

구분	내용
PubMed ID	31092656
TITLE	Implementation of clean cookstove interventions and its effects on pressure in low-income and middle-income countries: systematic
JOURNAL	BMJ open: 10.1136/bmjopen-2018-026517
AUTHORS	Onakomaiya Deborah, Gyamfi Joyce, Iwelunmor Juliet, Opeyemi Oluwasanmi Mofetoluwa, Obiezu-Umeh Chisom, Dalton Milena Ucheoma, Ojo Temitope, Vieira Dorice, Ogedegbe Gbenga, Olopa Christopher
OBJECTIVE	A review of the implementation outcomes of clean cookstove use, effects on blood pressure (BP) in low-income and middle-income (LMICs).
DESIGN	Systematic review of studies that reported the effect of clean cook BP among women, and implementation science outcomes in LMI
DATA SOURCES	We searched PubMed, Embase, INSPEC, Scielo, Cochrane Library Health and Web of Science PLUS. We conducted searches in Nov with a repeat in May 2018. We did not restrict article publication
ELIGIBILITY CRITERIA FOR SELECTING STUDIES	We included only studies conducted in LMICs, published in Englis of publication year and studies that examined the use of improved cookstove intervention on BP. Two authors independently screene article titles, abstracts and full-text articles to identify those that i following search terms: high BP, hypertension and or household a LMICs, cookstove and implementation outcomes.
RESULTS	Of the 461 non-duplicate articles identified, three randomised con (RCTs) (in Nigeria, Guatemala and Ghana) and two studies of pr design (in Bolivia and Nicaragua) met eligibility criteria. These ar evaluated the effect of cookstove use on BP in women. Two of th reported a mean reduction in diastolic BP of −2.8 mm Hg (−5.0, − p=0.01) for the Nigerian study; −3.0 mm Hg; (−5.7, −0.4; p=0.0 Guatemalan study; while the study conducted in Ghana reported non-significant change in BP. The pre-post studies reported a sig reduction in mean systolic BP of −5.5 mm Hg; (p=0.01) for the B study, and −5.9 mm Hg (−11.3, −0.4; p=0.05) for the Nicaraguan Implementation science outcomes were reported in all five studies reported feasibility, one reported adoption and one reported feasi adoption of cookstove interventions).
CONCLUSION	Although this review demonstrated that there is limited evidence o implementation of clean cookstove use in LMICs, the effects of cl cookstove on BP were significant for both systolic and diastolic B women. Future studies should consider standardised reporting of implementation outcomes.

표-702. PubMed 논문번호 31095432의 내용 요약

구분	내용
PubMed ID	31095432
TITLE	The Association between Long-Term Air Pollution and Urinary Catecholamines: Evidence from the Multi-Ethnic Study of Atherosclerosis.
JOURNAL	Environmental health perspectives: 10.1289/EHP3286
AUTHORS	Hajat Anjum, Diez Roux Ana V, Castro-Diehl Cecilia, Cosselman Kristen, Golden Sherita Hill, Hazlehurst Marnie F, Szpiro Adam, Vedal Sverre, Kaufman Joel D
BACKGROUND	Autonomic nervous system effects have been hypothesized as a mechanism of air pollutant health effects, though scant prior epidemiologic research has examined the association between air pollutants and catecholamines.
OBJECTIVES	To examine the association of long-term air pollutants with three urinary catecholamines: dopamine (DA), epinephrine (EPI), and norepinephrine (NE). As a secondary aim, we also examined the association between short-term (or acute) exposure to fine particulate matter [particulate matter with aerodynamic diameter [Formula: see text] ([Formula: see text])] and those catecholamines.
METHODS	We used data from the Multi-Ethnic Study of Atherosclerosis (MESA) and two of its ancillary studies, the MESA Air Pollution Study and the MESA Stress Study, to provide exposure and outcome data. DA, EPI, and NE from urine samples were collected from 2004 to 2006 from 1,002 participants in the New York, New York, and Los Angeles, California, study sites. Spatiotemporal models incorporated cohort-specific monitoring and estimated annual average pollutant concentrations ([Formula: see text], [Formula: see text], [Formula: see text] and black carbon) at participants' homes the year prior to urine collection. Secondarily, short-term [Formula: see text] was evaluated (day of, day prior, and 2- to 5-d lags prior to urine collection). Several covariates were considered confounders (age, race, sex, site, socioeconomic status, cardiovascular disease risk factors, psychosocial stressors, and medication use) in linear regression models.
RESULTS	A [Formula: see text] higher annual [Formula: see text] concentration was associated with 6.3% higher mean EPI level [95% confidence interval (CI): 0.3%, 12.6%]. A 2-[Formula: see text] higher annual ambient [Formula: see text] concentration was associated with 9.1% higher mean EPI (95% CI: 3.2%, 15.3%) and 4.4% higher DA level (95% CI: 1%, 7.9%). [Formula: see text], black carbon, and short-term [Formula: see text] exposures were not significantly associated with any of the catecholamines.
CONCLUSIONS	We found an association between EPI and long-term concentrations of [Formula: see text] and [Formula: see text] and an association between DA and long-term ambient [Formula: see text]. These novel findings provide modest support for the hypothesis that air pollutant exposures are related to sympathetic nervous system activation. https://doi.org/10.1289/EHP3286.

표-703. PubMed 논문번호 31096974의 내용 요약

구분	내용
PubMed ID	31096974
TITLE	Relations between air pollution and vascular development in 5-year old children: a cross-sectional study in the Netherlands.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-019-0487-1
AUTHORS	Ntarladima Anna-Maria, Vaartjes Ilonca, Grobbee Diederick E, Dijst Martin, Schmitz Oliver, Uiterwaal Cuno, Dalmeijer Geertje, van der Ent Cornelis, Hoek Gerard, Karssenberg Derek
BACKGROUND	Air pollution has been shown to promote cardiovascular disease in adults. Possible mechanisms include air pollution induced changes in arterial wall function and structure. Atherosclerotic vascular disease is a lifelong process and childhood exposure may play a critical role. We investigated whether air pollution is related to arterial wall changes in 5-year old children. To this aim, we developed an air pollution exposure methodology including time-weighted activity patterns improving upon epidemiological studies which assess exposure only at residential addresses.
METHODS	The study is part of an existing cohort study in which measurements of carotid artery intima-media thickness, carotid artery distensibility, elastic modulus, diastolic and systolic blood pressure have been obtained. Air pollution assessments were based on annual average concentration maps of Particulate Matter and Nitrogen Oxides at 5 m resolution derived from the European Study of Cohorts for Air Pollution Effects. We defined children's likely primary activities and for each activity we calculated the mean air pollution exposure within the assumed area visited by the child. The exposure was then weighted by the time spent performing each activity to retrieve personal air pollution exposure for each child. Time spent in these activities was based upon a Dutch mobility survey. To assess the relation between the vascular status and air pollution exposure we applied linear regressions in order to adjust for potential confounders.
RESULTS	Carotid artery distensibility was consistently associated with the exposures among the 733 5-years olds. Regression analysis showed that for air pollution exposures carotid artery distensibility decreased per standard deviation. Specifically, for NO2, carotid artery distensibility decreased by − 1.53 mPa− 1 (95% CI: −2.84, − 0.21), for NOx by − 1.35 mPa− 1 (95% CI: −2.67, − 0.04), for PM2.5 by − 1.38 mPa− 1 (95% CI: −2.73, − 0.02), for PM10 by − 1.56 mPa− 1 (95% CI: −2.73, − 0.39), and for PM2.5absorbance by − 1.63 (95% CI: −2.30, − 0.18). No associations were observed for the rest outcomes.
CONCLUSIONS	The results of this study support the view that air pollution exposure may reduce arterial distensibility starting in young children. If the reduced distensibility persists, this may have clinical relevance later in life. The results of this study further stress the importance of reducing environmental pollutant exposures.

표-704. PubMed 논문번호 31103472의 내용 요약

구분	내용
PubMed ID	31103472
TITLE	Ambient particulate air pollution and circulating C-reactive protein level: A systematic review and meta-analysis.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2019.05.005
AUTHORS	Liu Qisijing, Gu Xuelin, Deng Furong, Mu Lina, Baccarelli Andrea A, Guo Xinbiao, Wu Shaowei
BACKGROUND	Ambient particulate air pollution is a major threat to the cardiovascular health of people. Inflammation is an important component of the pathophysiological process that links air pollution and cardiovascular disease (CVD). A classical marker of inflammation-C-reactive protein (CRP), has been recognized as an independent predictor of CVD risk. Exposure to ambient particulate matter (PM) may cause systemic inflammatory response but its association with CRP has been inconsistently reported.
OBJECTIVES	To estimate the potential effects of short-term and long-term exposures to ambient particulate air pollution on circulating CRP level based on previous epidemiological studies.
METHODS	A systematic literature search of PubMed, Web of Science, Embase, and Scopus databases for publications up to January 2018 was conducted for studies reporting the association between ambient PM (PM2.5 or PM10, or both) and circulating CRP level. We performed a meta-analysis for the associations reported in individual studies using a random-effect model and evaluated the effect modification by major potential modifiers.
RESULTS	This meta-analysis comprised data from 40 observational studies conducted on 244,681 participants. These included 32 (27 PM2.5 studies and 13 PM10 studies) and 11 (9 PM2.5 studies and 5 PM10 studies) studies that investigated the associations of CRP with short-term and long-term exposure to particulate air pollution, respectively. A 10 μg/m3 increase in short-term exposure to PM2.5 and PM10 was associated with increases of 0.83 % (95% CI: 0.30%, 1.37%) and 0.39% (95% CI: -0.04%, 0.82%) in CRP level, respectively, and a 10 μg/m3 increase in long-term exposure to PM2.5 and PM10 was associated with much higher increases of 18.01% (95% CI: 5.96%, 30.06%) and 5.61% (95% CI: 0.79%, 10.44%) in CRP level, respectively. The long-term exposure to particulate air pollution was more strongly associated with CRP level than short-term exposure and PM2.5 had a greater effect on CRP level than PM10.
CONCLUSION	Exposure to ambient particulate air pollution is associated with elevated circulating CRP level suggesting an activated systemic inflammatory state upon exposure, which may explain the association between particulate air pollution and CVD risk.

표-705. PubMed 논문번호 31113399의 내용 요약

구분	내용
PubMed ID	31113399
TITLE	Effect of passive exposure to cigarette smoke on blood pressure in children and adolescents: a meta-analysis of epidemiologic studies.
JOURNAL	BMC pediatrics: 10.1186/s12887-019-1506-7
AUTHORS	Aryanpur Mahshid, Yousefifard Mahmoud, Oraii Alireza, Heydari Gholamreza, Kazempour-Dizaji Mehdi, Sharifi Hooman, Hosseini Mostafa, Jamaati Hamidreza
BACKGROUND	Hypertension is an emerging disease in children and adolescents resulting in future morbidities. Cigarette smoking is one of the most studied contributing factors in this regard; however, there are contradictory results among different studies. Therefore, the present meta-analysis tends to assess the relationship between passive exposure to cigarette smoke and blood pressure in children and adolescents.
METHOD	Medline, Embase, Scopus, EBSCO, and Web of Sciences were systematically reviewed for observational studies up to May, 2017, in which the relationship between cigarette smoking and hypertension were assessed in children and adolescents. The meta-analysis was performed with a fixed effect or random effects model according to the heterogeneity.
RESULTS	Twenty-nine studies were included in present meta-analysis incorporating 192,067 children and adolescents. Active smoking (pooled OR = 0.92; 95% CI: 0.79 to 1.05) or passive exposure to cigarette smoke (pooled OR = 1.01; 95% CI: 0.93 to 1.10) were not associated with developing hypertension in the study population. Despite the fact that active cigarette smoking did not significantly affect absolute level of systolic and diastolic blood pressure, it was shown that passive exposure to cigarette smoke leads to a significant increase in absolute level of systolic blood pressure (pooled coefficient = 0.26; 95% CI: 0.12 to 0.39).
CONCLUSION	Both active and passive cigarette smoking were not associated with developing hypertension in children and adolescents. However, passive cigarette smoke was associated with higher level of systolic blood pressure in children and adolescents.

표-706. PubMed 논문번호 31125079의 내용 요약

구분	내용
PubMed ID	31125079
TITLE	Favorable Changes in Biomarkers of Potential Harm to Reduce the Adverse Health Effects of Smoking in Smokers Switching to the Menthol Tobacco Heating System 2.2 for 3 Months (Part 2).
JOURNAL	Nicotine & tobacco research : official journal of the Society for Research on Nicotine and Tobacco: 10.1093/ntr/ntz084
AUTHORS	Haziza Christelle, de La Bourdonnaye Guillaume, Donelli Andrea, Skiada Dimitra, Poux Valerie, Weitkunat Rolf, Baker Gizelle, Picavet Patrick, L□dicke Frank
INTRODUCTION	Tobacco Heating System (THS) 2.2, a candidate modified-risk tobacco product, aims at offering an alternative to cigarettes for smokers while substantially reducing the exposure to harmful and potentially harmful constituents found in cigarette smoke.
METHODS	One hundred and sixty healthy adult US smokers participated in this randomized, three-arm parallel group, controlled clinical study. Subjects were randomized in a 2:1:1 ratio to menthol Tobacco Heating System 2.2 (mTHS), menthol cigarette, or smoking abstinence for 5 days in confinement and 86 subsequent ambulatory days. Endpoints included biomarkers of exposure to harmful and potentially harmful constituents (reported in our co-publication, Part 1) and biomarkers of potential harm (BOPH).
RESULTS	Compliance (protocol and allocated product exposure) was 51% and 18% in the mTHS and smoking abstinence arms, respectively, on day 90. Nonetheless, favorable changes in BOPHs of lipid metabolism (total cholesterol and high- and low-density cholesterol), endothelial dysfunction (soluble intercellular adhesion molecule-1), oxidative stress (8-epi-prostaglandin F2α), and cardiovascular risk factors (eg, high-sensitivity C-reactive protein) were observed in the mTHS group. Favorable effects in other BOPHs, including ones related to platelet activation (11-dehydrothromboxane B2) and metabolic syndrome (glucose), were more pronounced in normal weight subjects.
CONCLUSIONS	The results suggest that the reduced exposure demonstrated when switching to mTHS is associated with overall improvements in BOPHs, which are indicative of pathomechanistic pathways underlying the development of smoking-related diseases, with some stronger effects in normal weight subjects.
IMPLICATIONS	Switching to mTHS was associated with favorable changes for some BOPHs indicative of biological pathway alterations (eg, oxidative stress and endothelial dysfunction). The results suggest that switching to mTHS has the potential to reduce the adverse health effects of smoking and ultimately the risk of smoking-related diseases. Switching to mTHS for 90 days led to reductions in a number of biomarkers of exposure in smokers, relative to those who continued smoking cigarettes, which were close to those observed when stopping smoking (reported in our co-publication, Part 1). Initial findings suggest reduced levels of 8-epi-prostaglandin F2α and intercellular adhesion molecule 1, when switching to mTHS for 90 days. These changes are comparable to what is observed upon smoking cessation. In normal weight subjects, additional favorable changes were seen in 11-dehydrothromboxane B2, fibrinogen, homocysteine, hs-CRP, percentage of predicted forced expiratory volume in 1 second, systolic blood pressure, diastolic blood pressure, glucose, high-density lipoprotein, apolipoprotein A1, and

표-707. PubMed 논문번호 31128435의 내용 요약

구분	내용
PubMed ID	31128435
TITLE	Long-term exposure to atmospheric metals assessed by mosses and mortality in France.
JOURNAL	Environment international: 10.1016/j.envint.2019.05.004
AUTHORS	Lequy Emeline, Siemiatycki Jack, Leblond S□bastien, Meyer Caroline, Zhivin Sergey, Vienneau Danielle, de Hoogh Kees, Goldberg Marcel, Zins Marie, Jacquemin B□n□dicte
BACKGROUND	Long-term exposure to air pollution affects health, but little is known about exposure to atmospheric metals. Estimating exposure to atmospheric metals across large spatial areas remains challenging. Metal concentrations in mosses could constitute a useful proxy. Here, we linked moss biomonitoring and epidemiological data to investigate the associations between long-term exposure to metals and mortality.
METHODS	We modelled and mapped 13 atmospheric metals from a 20-year national moss biomonitoring program to derive exposure estimates across France. In the population-based Gazel cohort, we included 11,382 participants from low to intermediate population density areas and assigned modelled metals to their residential addresses. We distinguished between airborne metals that are primarily of natural origin and those primarily of anthropogenic origin. Associations were estimated between exposure to metals and mortality (natural-cause, cardiovascular and respiratory), using Cox models, with confounder adjustment at individual level.
FINDINGS	Between 1996 and 2017, there were 1313 deaths in the cohort (including 181 cardiovascular and 33 respiratory). Exposure to the anthropogenic metals was associated with an increased risk of natural-cause mortality (hazard ratio of 1.16 [1.08-1.24] per interquartile range of exposure), while metals from natural sources were not.
INTERPRETATION	Some atmospheric anthropogenic metals may be associated with excess mortality - even in areas with relatively low levels of exposure to air pollution. Consistent with the previous literature, our findings support the use of moss biomonitoring as a tool to assess health effects of air pollution exposure at individual level.

표-708. PubMed 논문번호 31166133의 내용 요약

구분	내용
PubMed ID	31166133
TITLE	Long-Term PM₁₀ Exposure and Cause-Specific Mortality in the Latium Region (Italy): A Difference-in-Differences Approach.
JOURNAL	Environmental health perspectives: 10.1289/EHP3759
AUTHORS	Renzi Matteo, Forastiere Francesco, Schwartz Joel, Davoli Marina, Michelozzi Paola, Stafoggia Massimo
BACKGROUND	The link between particulate matter (PM) exposure and adverse health outcomes has been widely evaluated using large cohort studies. However, the possibility of residual confounding and lack of information about the health effects of PM in rural and suburban areas are unsolved issues.
OBJECTIVE	Our aim was to estimate the effect of annual PM≤10□g (PM10) exposure on cause-specific mortality in the Latium region (central Italy, of which Rome is the main city) during 2006-2012 using a difference-in-differences approach.
METHODS	We estimated daily PM10 concentrations for each 1 km2 of the region from 2006 to 2012 by use of satellite data, land-use predictors, and meteorological parameters. For each of the 378 regional municipalities and each year, we averaged daily PM10 values to obtain annual mean PM10 exposures. We applied a variant of the difference-in-differences approach to estimate the association between PM10 and cause-specific mortality by focusing on within-municipality fluctuations of mortality rates and annual PM exposures around municipality means, therefore controlling by design for confounding from all spatial and temporal potential confounders. Analyses were also stratified by population size of the municipalities to obtain effect estimates in rural and suburban areas of the region.
RESULTS	In the period 2006-2012, we observed deaths due to three causes: 347,699 nonaccidental; 92,787 cardiovascular; and 16,509 respiratory causes. The annual average (standard deviation, SD) PM10 concentration was 21.9 (±4.9) □g/km3 in Latium. For each 1-□g/m3 increase in annual PM10 we estimated increases of 0.8% (95% confidence intervals (CIs): 0.2%, 1.3%), 0.9% (0.0%, 1.8%), and 1.4% (-0.4%, 3.3%) in nonaccidental, cardiovascular, and respiratory mortality, respectively. Similar results were found when we excluded the metropolitan area of Rome from the analysis. Higher effects were estimated in the smaller municipalities, e.g., those with population < 5,000 inhabitants.
CONCLUSION	Our study suggests a significant association of annual PM10 exposure with nonaccidental and cardiorespiratory mortality in the Latium region, even outside Rome and in suburban and rural areas. https://doi.org/10.1289/EHP3759.

표-709. PubMed 논문번호 31181010의 내용 요약

구분	내용
PubMed ID	31181010
TITLE	Long-term Exposure to Low Air Pollutant Concentrations and the Relationship with All-Cause Mortality and Stroke in Older Men.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000001034
AUTHORS	Dirgawati Mila, Hinwood Andrea, Nedkoff Lee, Hankey Graeme J, Yeap Bu B, Flicker Leon, Nieuwenhuijsen Mark, Brunekreef Bert, Heyworth Jane
BACKGROUND	Long-term air pollution exposure has been associated with increased risk of mortality and stroke. Less is known about the risk at lower concentrations. The association of long-term exposure to PM2.5, PM2.5 absorbance, NO2, and NOx with all-cause mortality and stroke was investigated in a cohort of men aged ≥ 65 years who lived in metropolitan Perth, Western Australia.
METHODS	Land use regression models were used to estimate long-term exposure to air pollutants at participant's home address (n = 11,627) over 16 years. Different metrics of exposure were assigned: baseline; year before the outcome event; and average exposure across follow-up period. The Mortality Register and Hospital Morbidity Data from the Western Australia Data Linkage System were used to ascertain mortality and stroke cases. Hazard ratios (HRs) and 95% confidence intervals were estimated using Cox proportional hazard models, adjusting for age, smoking, education, and body mass index for all-cause mortality. For fatal and hospitalized stroke, the models included variables controlled for all-cause mortality plus hypertension.
RESULTS	Fifty-four percent of all-participants died, 3% suffered a fatal stroke, and 14% were hospitalized stroke cases. PM2.5 absorbance increased the risk of all-cause mortality with adjusted HR of 1.12 (1.02-1.23) for baseline and average exposures, and 1.14 (1.02-1.24) for past-year exposure. There were no associations between PM2.5 absorbance, NO2, and NOx and stroke outcomes. However, PM2.5 was associated with reduced risks of fatal stroke.
CONCLUSION	Long-term exposure to PM2.5 absorbance was associated with all-cause mortality among older men exposed to low concentrations; and exposure to PM2.5 was associated with reduced risk of fatal stroke.

표-710. PubMed 논문번호 31181011의 내용 요약

구분	내용
PubMed ID	31181011
TITLE	Long-term Effects of Cumulative Average PM2.5 Exposure on the Risk of Hemorrhagic Stroke.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000001001
AUTHORS	Noh Juhwan, Sohn Jungwoo, Han Minkyung, Kang Dae Ryong, Choi Yoon Jung, Kim Hyeon Chang, Suh Il, Kim Changsoo, Shin Dong Chun
BACKGROUND	Epidemiological studies have revealed associations between the fine particle (PM2.5; aerodynamic diameter 〈2.5 μm) exposure and cardiovascular disease. Researchers have also recently begun investigating the association between PM2.5 exposure and hemorrhagic stroke (HS) and identifying subpopulations vulnerable to PM2.5 exposure. Long-term cumulative average PM2.5 exposure may affect the risk of HS, and these effects may be modified by risk factors.
METHODS	This retrospective study evaluated the effects of PM2.5 on the time-to-first-diagnosis of HS among 62,676 Seoul metropolitan city residents with 670,431 total person-years of follow-up; this cohort is a subset from a nationally representative cohort of 1,025,340 individuals from the Korean National Health Insurance Service database (2002-2013). A time-dependent Cox proportional hazards model was used to adjust for age, sex, household income, insurance type, body mass index, smoking status, medical history, and family history. The annual mean PM2.5 concentrations for 25 districts were used as the time-dependent variable. Subgroup analyses of the traditional risk factors of HS were performed to evaluate potential effect modifications.
RESULTS	Each 10-μg/m increment in cumulative average PM2.5 exposure was noticeably associated with HS (hazard ratio [HR] = 1.43; 95% confidence interval [CI]: 1.09-1.88). The adverse effects of PM2.5 exposure were modified by ≥65 years of age (HR = 2.00; 95% CI = 1.32, 3.02) and obesity (body mass index ≥25 kg/m; HR = 1.91; 95% CI = 1.28, 2.84).
CONCLUSIONS	Cumulative average PM2.5 exposure might increase the risk of HS. Elderly (≥65 years) and obese individuals may be more vulnerable to the effects of PM2.5 exposure.

표-711. PubMed 논문번호 31189380의 내용 요약

구분	내용
PubMed ID	31189380
TITLE	Investigation of air pollution and noise on progression of thoracic aortic calcification: results of the Heinz Nixdorf Recall Study.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487319854818
AUTHORS	Hennig Frauke, Moebus Susanne, Reinsch Nico, Budde Thomas, Erbel Raimund, J□ckel Karl-Heinz, Lehmann Nils, Hoffmann Barbara, K□lsch Hagen, Hennig Frauke, Moebus Susanne, Reinsch Nico, Budde Thomas, Erbel Raimund, J□ckel Karl-Heinz, Lehmann Nils, Hoffmann Barbara, K□lsch Hagen
AIMS	Air pollution and noise are potential risk factors for subclinical atherosclerosis. Longitudinal analyses, especially on the interplay of these environmental factors, are scarce and inconsistent. Hence we investigated long-term traffic-related exposure to air pollution and noise with the development and progression of thoracic aortic calcification, a marker of subclinical atherosclerosis.
METHODS	We used baseline (2000-2003) and follow-up (2006-2008) data from the German Heinz Nixdorf Recall cohort study, including 4814 middle-aged adults. Residence-based air pollution (PM2.5 (aerodynamic diameter ≤ 2.5 □m), PM10, nitrogen dioxide and particle number), and noise was assessed with dispersion models. Thoracic aortic calcification was quantified from non-contrast enhanced electron beam computed tomography. The presence and extent of thoracic aortic calcification progression were analysed with multiple logistic and linear regression models, respectively, adjusting for age, sex, lifestyle variables, socioeconomic status and respective co-exposure.
RESULTS	We observed no association in the full study sample (n = 3155, mean age 59.1 (±7.6) years, 52.8% women). While an interquartile range in particle number and night-time noise yielded odds ratios of 1.20 (1.03, 1.40) and 1.21 (1.00, 1.46) for binary thoracic aortic calcification progression, and 0.02 (-0.01, 0.05) and 0.04 (0.00, 0.07) higher growth rates of thoracic aortic calcification in participants with baseline thoracic aortic calcification less than 10, negative findings were observed in those with baseline thoracic aortic calcification of 10 or greater. Results were similar for other pollutants and daytime noise.
CONCLUSION	Our study shows no overall associations. Subgroup analyses suggest independent associations of traffic-related air pollution and noise with the development and progression of subclinical atherosclerosis in participants with no or minor thoracic aortic calcification at baseline, in contrast to negative findings in those with advanced calcification.

표-712. PubMed 논문번호 31208937의 내용 요약

구분	내용
PubMed ID	31208937
TITLE	Methylome-wide association study provides evidence of particulate matter air pollution-associated DNA methylation.
JOURNAL	Environment international: 10.1016/j.envint.2019.03.071
AUTHORS	Gondalia Rahul, Baldassari Antoine, Holliday Katelyn M, Justice Anne E, M□ndez-Gir□ldez Ra□l, Stewart James D, Liao Duanping, Yanosky Jeff D, Brennan Kasey J M, Engel Stephanie M, Jordahl Kristina M, Kennedy Elizabeth, Ward-Caviness Cavin K, Wolf Kathrin, Waldenberger Melanie, Cyrys Josef, Peters Annette, Bhatti Parveen, Horvath Steve, Assimes Themistocles L, Pankow James S, Demerath Ellen W, Guan Weihua, Fornage Myriam, Bressler Jan, North Kari E, Conneely Karen N, Li Yun, Hou Lifang, Baccarelli Andrea A, Whitsel Eric A
BACKGROUND	DNA methylation (DNAm) may contribute to processes that underlie associations between air pollution and poor health. Therefore, our objective was to evaluate associations between DNAm and ambient concentrations of particulate matter (PM) ≤2.5, ≤10, and 2.5-10 μm in diameter (PM2.5; PM10; PM2.5-10).
METHODS	We conducted a methylome-wide association study among twelve cohort- and race/ethnicity-stratified subpopulations from the Women's Health Initiative and the Atherosclerosis Risk in Communities study (n = 8397; mean age: 61.5 years; 83% female; 45% African American; 9% Hispanic/Latino American). We averaged geocoded address-specific estimates of daily and monthly mean PM concentrations over 2, 7, 28, and 365 days and 1 and 12 months before exams at which we measured leukocyte DNAm in whole blood. We estimated subpopulation-specific, DNAm-PM associations at approximately 485,000 Cytosine-phosphate-Guanine (CpG) sites in multi-level, linear, mixed-effects models. We combined subpopulation- and site-specific estimates in fixed-effects, inverse variance-weighted meta-analyses, then for associations that exceeded methylome-wide significance and were not heterogeneous across subpopulations ($P < 1.0 \times 10^{-7}$; PCochran's $Q > 0.10$), we characterized associations using publicly accessible genomic databases and attempted replication in the Cooperative Health Research in the Region of Augsburg (KORA) study.
RESULTS	Analyses identified significant DNAm-PM associations at three CpG sites. Twenty-eight-day mean PM10 was positively associated with DNAm at cg19004594 (chromosome 20; MATN4; $P = 3.33 \times 10^{-8}$). One-month mean PM10 and PM2.5-10 were positively associated with DNAm at cg24102420 (chromosome 10; ARPP21; $P = 5.84 \times 10^{-8}$) and inversely associated with DNAm at cg12124767 (chromosome 7; CFTR; $P = 9.86 \times 10^{-8}$). The PM-sensitive CpG sites mapped to neurological, pulmonary, endocrine, and cardiovascular disease-related genes, but DNAm at those sites was not associated with gene expression in blood cells and did not replicate in KORA.
CONCLUSIONS	Ambient PM concentrations were associated with DNAm at genomic regions potentially related to poor health among racially, ethnically and environmentally diverse populations of U.S. women and men. Further investigation is warranted to uncover mechanisms through which PM-induced epigenomic changes may cause disease.

표-713. PubMed 논문번호 31228782의 내용 요약

구분	내용
PubMed ID	31228782
TITLE	Urine metabolites associated with cardiovascular effects from exposure of size-fractioned particulate matter in a subway environment: A randomized crossover study.
JOURNAL	Environment international: 10.1016/j.envint.2019.104920
AUTHORS	Zhang Yannan, Chu Mengtian, Zhang Jingyi, Duan Junchao, Hu Dayu, Zhang Wenlou, Yang Xuan, Jia Xu, Deng Furong, Sun Zhiwei
BACKGROUND	Ambient particulate matter (PM) is closely associated with morbidity and mortality from cardiovascular disease. Urine metabolites can be used as a non-invasive means to explore biological mechanisms for such associations, yet has not been performed in relation to different sizes of PM. In this randomized crossover study, we used metabolomics approach to explore the urine biomarkers linked with cardiovascular effects after PM exposure in a subway environment.
METHODS AND RESULTS	Thirty-nine subjects were exposed to PM for 4 h in subway system, with either a respirator intervention phase (RIP) with facemask and no intervention phase (NIP) in random order with a 2-week washout period. Electrocardiogram (ECG) parameters and ambulatory blood pressure (BP) were monitored during the whole riding period and urine samples were collected for metabolomics analysis. After exposure to PM for 4 h in subway system, 4 urine metabolites in male and 7 urine metabolites in female were screened out by UPLC/Q-TOF MS/MS-based metabolomics approach. Cardiovascular parameters (HRV and HR) predominantly decreased in response to all size-fractions of PM and were more sensitive in response to different size-fractioned PM in males than females. Besides LF/HF, most of the HRV indices decrease induced by the increase of all size-fractioned PM while PM1.0 was found as the most influential one on indicators of cardiovascular effects and urine metabolites both genders. Prolyl-arginine and 8-OHdG were found to have opposing role regards to HRV and HR in male.
CONCLUSION	Our data indicated that short-term exposure to PM in a subway environment may increase the risk of cardiovascular disease as well as affect urine metabolites in a size dependent manner (besides PM0.5), and male were more prone to trigger the cardiovascular events than female after exposure to PM; whereas wearing facemask could effectively reduce the adverse effects caused by PM.

표-714. PubMed 논문번호 31248666의 내용 요약

구분	내용
PubMed ID	31248666
TITLE	Mortality, morbidity, and risk factors in China and its provinces, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(19)30427-1
AUTHORS	Zhou Maigeng, Wang Haidong, Zeng Xinying, Yin Peng, Zhu Jun, Chen Wanqing, Li Xiaohong, Wang Lijun, Wang Limin, Liu Yunning, Liu Jiangmei, Zhang Mei, Qi Jinlei, Yu Shicheng, Afshin Ashkan, Gakidou Emmanuela, Glenn Scott, Krish Varsha Sarah, Miller-Petrie Molly Katherine, Mountjoy-Venning W Cliff, Mullany Erin C, Redford Sofia Boston, Liu Hongyan, Naghavi Mohsen, Hay Simon I, Wang Linhong, Murray Christopher J L, Liang Xiaofeng
BACKGROUND	Public health is a priority for the Chinese Government. Evidence-based decision making for health at the province level in China, which is home to a fifth of the global population, is of paramount importance. This analysis uses data from the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017 to help inform decision making and monitor progress on health at the province level.
METHODS	We used the methods in GBD 2017 to analyse health patterns in the 34 province-level administrative units in China from 1990 to 2017. We estimated all-cause and cause-specific mortality, years of life lost (YLLs), years lived with disability (YLDs), disability-adjusted life-years (DALYs), summary exposure values (SEVs), and attributable risk. We compared the observed results with expected values estimated based on the Socio-demographic Index (SDI).
FINDINGS	Stroke and ischaemic heart disease were the leading causes of death and DALYs at the national level in China in 2017. Age-standardised DALYs per 100 000 population decreased by 33·1% (95% uncertainty interval [UI] 29·8 to 37·4) for stroke and increased by 4·6% (-3·3 to 10·7) for ischaemic heart disease from 1990 to 2017. Age-standardised stroke, ischaemic heart disease, lung cancer, chronic obstructive pulmonary disease, and liver cancer were the five leading causes of YLLs in 2017. Musculoskeletal disorders, mental health disorders, and sense organ diseases were the three leading causes of YLDs in 2017, and high systolic blood pressure, smoking, high-sodium diet, and ambient particulate matter pollution were among the leading four risk factors contributing to deaths and DALYs. All provinces had higher than expected DALYs per 100 000 population for liver cancer, with the observed to expected ratio ranging from 2·04 to 6·88. The all-cause age-standardised DALYs per 100 000 population were lower than expected in all provinces in 2017, and among the top 20 level 3 causes were lower than expected for ischaemic heart disease, Alzheimer's disease, headache disorder, and low back pain. The largest percentage change at the national level in age-standardised SEVs among the top ten leading risk factors was in high body-mass index (185%, 95% UI 113·1 to 247·7]), followed by ambient particulate matter pollution (88·5%, 66·4 to 116·4).
INTERPRETATION	China has made substantial progress in reducing the burden of many diseases and disabilities. Strategies targeting chronic diseases, particularly in the elderly, should be prioritised in the expanding Chinese health-care system.
FUNDING	China National Key Research and Development Program and Bill & Melinda Gates Foundation.

표-715. PubMed 논문번호 31253484의 내용 요약

구분	내용
PubMed ID	31253484
TITLE	Interaction between neighborhood walkability and traffic-related air pollution on hypertension and diabetes: The CANHEART cohort.
JOURNAL	Environment international: 10.1016/j.envint.2019.04.070
AUTHORS	Howell Nicholas A, Tu Jack V, Moineddin Rahim, Chen Hong, Chu Anna, Hystad Perry, Booth Gillian L
BACKGROUND	Living in unwalkable neighborhoods has been associated with heightened risk for diabetes and hypertension. However, highly walkable environments may have higher concentrations of traffic-related air pollution, which may contribute to increased cardiovascular disease risk. We therefore aimed to assess how walkability and traffic-related air pollution jointly affect risk for hypertension and diabetes.
METHODS	We used a cross-sectional, population-based sample of individuals aged 40-74 years residing in selected large urban centres in Ontario, Canada on January 1, 2008, assembled from administrative databases. Walkability and traffic-related air pollution (NO2) were assessed using validated tools and linked to individuals based on neighborhood of residence. Logistic regression was used to estimate adjusted associations between exposures and diagnoses of hypertension or diabetes accounting for potential confounders.
RESULTS	Overall, 2,496,458 individuals were included in our analyses. Low walkability was associated with higher odds of hypertension (lowest vs. highest quintile OR = 1.34, 95% CI: 1.32, 1.37) and diabetes (lowest vs. highest quintile OR = 1.25, 95% CI: 1.22, 1.29), while NO2 exhibited similar trends (hypertension: OR = 1.09 per 10 p.p.b., 95% CI: 1.08, 1.10; diabetes: OR = 1.16, 95% CI: 1.14, 1.17). Significant interactions were identified between walkability and NO2 on risk for hypertension ($p < 0.0001$ and diabetes ($p < 0.0001$). At higher levels of pollution (40 p.p.b.), differences in the probability of hypertension (lowest vs. highest walkability quintile: 0.26 vs. 0.25) or diabetes (lowest vs. highest walkability quintile: 0.15 vs. 0.15) between highly walkable and unwalkable neighborhoods were diminished, compared to differences observed at lower levels of pollution (5 p.p.b.) (hypertension, lowest vs. highest walkability quintile: 0.21 vs. 0.13; diabetes, lowest vs. highest walkability quintile: 0.09 vs. 0.06).
CONCLUSIONS	Walkability and traffic-related air pollution interact to jointly predict risk for hypertension and diabetes. Although walkable neighborhoods appear to have beneficial effects, they may accentuate the harmful effects of air pollution on cardiovascular risk factors.

표-716. PubMed 논문번호 31272016의 내용 요약

구분	내용
PubMed ID	31272016
TITLE	Spatial variations in ambient ultrafine particle concentrations and risk of congenital heart defects.
JOURNAL	Environment international: 10.1016/j.envint.2019.104953
AUTHORS	Lavigne Eric, Lima Isac, Hatzopoulou Marianne, Van Ryswyk Keith, Decou Mary Lou, Luo Wei, van Donkelaar Aaron, Martin Randall V, Chen Hong, Stieb David M, Crighton Eric, Gasparrini Antonio, Elten Michael, Yasseen Abdool S, Burnett Richard T, Walker Mark, Weichenthal Scott
BACKGROUND	Cardiovascular malformations account for nearly one-third of all congenital anomalies, making these the most common type of birth defects. Little is known regarding the influence of ambient ultrafine particles (<0.1 μm) (UFPs) on their occurrence.
OBJECTIVE	This population-based study examined the association between prenatal exposure to UFPs and congenital heart defects (CHDs).
METHODS	A total of 158,743 singleton live births occurring in the City of Toronto, Canada between April 1st 2006 and March 31st 2012 were identified from a birth registry. Associations between exposure to ambient UFPs between the 2nd and 8th week post conception when the foetal heart begins to form and CHDs identified at birth were estimated using random-effects logistic regression models, adjusting for personal- and neighbourhood-level covariates. We also investigated multi-pollutant models accounting for co-exposures to PM2.5, NO2 and O3.
RESULTS	A total of 1468 CHDs were identified. In fully adjusted models, UFP exposures during weeks 2 to 8 of pregnancy were not associated with overall CHDs (Odds Ratio (OR) per interquartile (IQR) increase = 1.02, 95% CI: 0.96-1.08). When investigating subtypes of CHDs, UFP exposures were associated with ventricular septal defects (Odds Ratio (OR) per interquartile (IQR) increase = 1.13, 95% CI: 1.03-1.33), but not with atrial septal defect (Odds Ratio (OR) per interquartile (IQR) increase = 0.89, 95% CI: 0.74-1.06).
CONCLUSION	This is the first study to evaluate the association between prenatal exposure to UFPs and the risk of CHDs. UFP exposures during a critical period of embryogenesis were associated with an increased risk of ventricular septal defect.

표-717. PubMed 논문번호 31288180의 내용 요약

구분	내용
PubMed ID	31288180
TITLE	Acute effects of ambient temperature on hypotension hospital visits: A time-series analysis in seven metropolitan cities of Korea from 2011 to 2015.
JOURNAL	Environment international: 10.1016/j.envint.2019.104941
AUTHORS	Han Changwoo, Lim Youn-Hee, Lee Kyung-Shin, Hong Yun-Chul
BACKGROUND	Although blood pressure decreases in response to high ambient temperature, little is known about whether the ambient temperature can induce clinical hypotension events. Therefore, we conducted a time-series analysis to evaluate the association between hypotension hospital visits and ambient temperature in seven metropolitan cities of Korea.
METHODS	We used the National Health Insurance Database, which contains the complete hospital visit data of the entire Korean population. We collected hospital visit data of seven metropolitan cities and linked the number of daily hypotension hospital visits to city-level ambient temperature, relative humidity, and air pollution levels from 2011 to 2015. Time-series analysis using the Poisson generalized additive model was conducted for each metropolitan city and we meta-analyzed the time-series results using the random effect model.
RESULTS	There were 132,097 hospital visits for hypotension during our study period. A 1 degree Celsius (° C) increase in ambient temperature was associated with 1.1% increase in hospital visits for hypotension on lag day 0. Effects of ambient temperature lasted for 7 days, showing greater effects in shorter lag days. Subgroup analysis by sex and income groups showed similar results, but effects of ambient temperature on hypotension hospital visits was higher in the younger age group compared to older age group (aged over 65 years old). The results were unchanged when we applied cumulative lags, different case definitions, degrees of freedom per year, and multi-pollutant model adjusting for air pollutants.
CONCLUSIONS	Hospital visits for hypotension were positively associated with ambient temperature. Increased hypotension events in response to increased ambient temperature might explain the high cardiovascular mortality on hot days.

표-718. PubMed 논문번호 31289812의 내용 요약

구분	내용
PubMed ID	31289812
TITLE	PM2.5 air pollution and cause-specific cardiovascular disease mortality.
JOURNAL	International journal of epidemiology: 10.1093/ije/dyz114
AUTHORS	Hayes Richard B, Lim Chris, Zhang Yilong, Cromar Kevin, Shao Yongzhao, Reynolds Harmony R, Silverman Debra T, Jones Rena R, Park Yikyung, Jerrett Michael, Ahn Jiyoung, Thurston George D
BACKGROUND	Ambient air pollution is a modifiable risk factor for cardiovascular disease, yet uncertainty remains about the size of risks at lower levels of fine particulate matter (PM2.5) exposure which now occur in the USA and elsewhere.
METHODS	We investigated the relationship of ambient PM2.5 exposure with cause-specific cardiovascular disease mortality in 565 477 men and women, aged 50 to 71 years, from the National Institutes of Health-AARP Diet and Health Study. During 7.5 x 106 person-years of follow up, 41 286 cardiovascular disease deaths, including 23 328 ischaemic heart disease (IHD) and 5894 stroke deaths, were ascertained using the National Death Index. PM2.5 was estimated using a hybrid land use regression (LUR) geostatistical model. Multivariate Cox regression models were used to estimate relative risks (RRs) and 95% confidence intervals (CI).
RESULTS	Each increase of 10 μg/m3 PM2.5 (overall range, 2.9-28.0 μg/m3) was associated, in fully adjusted models, with a 16% increase in mortality from ischaemic heart disease [hazard ratio (HR) 1.16; 95% CI 1.09-1.22] and a 14% increase in mortality from stroke (HR 1.14; CI 1.02-1.27). Compared with PM2.5 exposure <8 μg/m3 (referent), risks for CVD were increased in relation to PM2.5 exposures in the range of 8-12 μg/m3 (CVD: HR 1.04; 95% CI 1.00-1.08), in the range 12-20 μg/m3 (CVD: HR 1.08; 95% CI 1.03-1.13) and in the range 20+ μg/m3 (CVD: HR 1.19; 95% CI 1.10-1.28). Results were robust to alternative approaches to PM2.5 exposure assessment and statistical analysis.
CONCLUSIONS	Long-term exposure to fine particulate air pollution is associated with ischaemic heart disease and stroke mortality, with excess risks occurring in the range of and below the present US long-term standard for ambient exposure to PM2.5 (12 □g/m3), indicating the need for continued improvements in air pollution abatement for CVD prevention.

표-719. PubMed 논문번호 31302482의 내용 요약

구분	내용
PubMed ID	31302482
TITLE	Green space, air pollution, traffic noise and cardiometabolic health in adolescents: The PIAMA birth cohort.
JOURNAL	Environment international: 10.1016/j.envint.2019.104991
AUTHORS	Bloemsma Lizan D, Gehring Ulrike, Klompmaker Jochem O, Hoek Gerard, Janssen Nicole A H, Lebret Erik, Brunekreef Bert, Wijga Alet H
BACKGROUND	Green space has been hypothesized to improve cardiometabolic health of adolescents, whereas air pollution and traffic noise may negatively impact cardiometabolic health.
OBJECTIVES	To examine the associations of green space, air pollution and traffic noise with cardiometabolic health in adolescents aged 12 and 16 years.
METHODS	Waist circumference, blood pressure, cholesterol and glycated hemoglobin (HbA1c) were measured in subsets of participants of the Dutch PIAMA birth cohort, who participated in medical examinations at ages 12 (n = 1505) and/or 16 years (n = 797). We calculated a combined cardiometabolic risk score for each participant, with a higher score indicating a higher cardiometabolic risk. We estimated exposure to green space (i.e. the average Normalized Difference Vegetation Index (NDVI) and percentages of green space in circular buffers of 300 m and 3000 m), air pollution (by land-use regression models) and traffic noise (using the Standard Model Instrumentation for Noise Assessments (STAMINA) model) at the adolescents' home addresses at the time of the medical examinations. We assessed associations of these exposures with cardiometabolic health outcomes at ages 12 and 16 by multiple linear regression, adjusting for potential confounders.
RESULTS	We did not observe consistent patterns of associations of green space, air pollution and traffic noise with the cardiometabolic risk score, blood pressure, total cholesterol levels, the total/HDL cholesterol ratio and HbA1c. We found inverse associations of air pollution with waist circumference at both age 12 and 16. These associations weakened after adjustment for region, except for particulate matter with a diameter of <2.5 μm (PM2.5) at age 12. The association of PM2.5 with waist circumference at age 12 remained after adjustment for green space and road traffic noise (adjusted difference − 1.42 cm [95% CI −2.50, −0.35 cm] per 1.16 μg/m3 increase in PM2.5).
CONCLUSION	This study does not provide evidence for beneficial effects of green space or adverse effects of air pollution and traffic noise on cardiometabolic health in adolescents.

표-720. PubMed 논문번호 31311712의 내용 요약

구분	내용
PubMed ID	31311712
TITLE	Particulate Air Pollutants and Trajectories of Depressive Symptoms in Older Women.
JOURNAL	The American journal of geriatric psychiatry : official journal of the Americ Association for Geriatric Psychiatry: 10.1016/j.jagp.2019.04.019
AUTHORS	Petkus Andrew J, Younan Diana, Wang Xinhui, Serre Marc, Vizuete Willian Resnick Susan, Espeland Mark A, Gatz Margaret, Chui Helena, Manson JoAnn E, Chen Jiu-Chiuan
OBJECTIVES	Although several environmental factors contribute to the etiology of late-life depressive symptoms, the role of ambient air pollution has been understudie Experimental data support the neurotoxicity of airborne particulate matter with aerodynamic diameter of ≤2.5 μm (PM2.5), but it remains unclear whether long-term exposure is associated with late-life depressive symptom Our secondary aim was to explore whether the observed associations betwe exposure and depressive symptoms are explained by dementia risk.
DESIGN, SETTING, AND PARTICIPANTS	Prospective community-dwelling cohort study from the Women's Health Initiative Study of Cognitive Aging (1999-2010). Our analyses included 1,9 older women (baseline age 73.3 ± 3.75) with no prior depression or cogniti impairment.
MEASUREMENTS	Participants completed annual assessments of depressive symptoms (15-item Geriatric Depression Scale). Average ambient PM2.5 exposure at the residential location was estimated by spatiotemporal modeling for the 3-yea preceding each neuropsychological assessment. Participants underwent separate annual examinations for incident dementia defined by DSM-IV. Latent-class mixture models examined the association between PM2.5 and identified trajectories of symptoms.
RESULTS	Six trajectories of depressive symptoms were identified. Across all women, PM2.5 exposure was positively associated with depressive symptoms. The effect was especially strong in two clusters with sustained depressive symptoms (n = 625 sustained-mild [31%]; n = 125 sustained-moderate; [6%]). Among those with sustained-moderate symptoms, the estimated adverse effect of PM2.5 exposure was greater than that of hypertension. Among women without dementia, associations were modestly attenuated.
CONCLUSION	Long-term exposure to ambient fine particles was associated with increased depressive symptoms among older women without prior depression or cognitive impairment.

표-721. PubMed 논문번호 31315297의 내용 요약

구분	내용
PubMed ID	31315297
TITLE	Association of Long-Term Exposure to Fine Particulate Matter and Cardio-Metabolic Diseases in Low- and Middle-Income Countries: A Systematic Review.
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph16142541
AUTHORS	Jaganathan Suganthi, Jaacks Lindsay M, Magsumbol Melina, Walia Gagandeep K, Sieber Nancy L, Shivasankar Roopa, Dhillon Preet K, Hameed Safraj Shahul, Schwartz Joel, Prabhakaran Dorairaj
METHODS	Multiple databases were searched for English articles with date limits until March 2018. We included studies investigating the association of long-term exposure to PM2.5 (defined as an annual average/average measure for 3 more days of PM2.5 exposure) and CMDs, such as hospital admissions, prevalence, and deaths due to CMDs, conducted in LMICs as defined by World Bank. We excluded studies which employed exposure proxy measures, studies among specific occupational groups, and specific episodes of air pollution.
RESULTS	A total of 5567 unique articles were identified, of which only 17 articles were included for final review, and these studies were from Brazil, Bulgaria, China, India, and Mexico. Outcome assessed were hypertension, type 2 diabetes mellitus and insulin resistance, and cardiovascular disease (CVD)-related emergency room visits/admissions, death, and mortality. Largely a positive association between exposure to PM2.5 and CMDs was found, and CVD mortality with effect estimates ranging from 0.24% to 6.11% increased per 10 μg/m3 in PM2.5. CVD-related hospitalizations and emergency room visits increased by 0.3% to 19.6%. Risk factors like hypertension had an odds ratio of 1.14, and type 2 diabetes mellitus had an odds ratio ranging from 1.14-1.32. Diversity of exposure assessment and health outcomes limited the ability to perform a meta-analysis.
CONCLUSION	Limited evidence on the association of long-term exposure to PM2.5 and CMDs in the LMICs context warrants cohort studies to establish the association.

표-722. PubMed 논문번호 31326714의 내용 요약

구분	내용
PubMed ID	31326714
TITLE	Long-term exposure to PM〈sub〉2.5〈/sub〉 and stroke: A systematic review and meta-analysis of cohort studies.
JOURNAL	Environmental research: 10.1016/j.envres.2019.108587
AUTHORS	Yuan Sheng, Wang Jiaxin, Jiang Qingqing, He Ziyu, Huang Yuchai, Li Zhengyang, Cai Luyao, Cao Shiyi
BACKGROUND	Stroke is one of the world's leading causes of death. Many studies have checked the relationship between short-term exposure to particulate matter (PM) and stroke, but few have focused on the effect of long-term exposure to PM2.5 (particulate matters with an aerodynamic diameter of ≤2.5 μm). This study aimed to quantitatively examine the relationship of long-term exposure to PM2.5 with stroke incidence and mortality.
METHODS	We identified relevant studies by searching the PubMed, EMBASE and MEDLINE. After the systematical review of pertinent studies, random-effect meta-analysis was conducted to investigate the association between long-term exposure to PM2.5 and stroke.
RESULTS	Our meta-analysis included 16 cohort studies with more than 2.2 million people and above 49 149 endpoint events (incident stroke and death from stroke). The pooled hazard ratio (HR) for each 5 μg/m3 increment in PM2.5 was 1.11 (95% CI: 1.05, 1.17) (CI for confidence interval) for incidence of stroke and 1.11 (95% CI:1.05, 1.17) for mortality of stroke. In the region-specific analysis, significant association between PM2.5 and incidence of stroke was found in North America (HR=1.09, 95% CI:1.05, 1.14) and Europe (HR=1.07, 95% CI:1.05, 1.10), while the pooled result of Asia showed no significance (HR=2.31, 95% CI:0.49, 10.95).
CONCLUSIONS	Long-term exposure to PM2.5 is an important risk factor for stroke. Since air quality is intimately related to everyone, policies aimed at reducing particulate matters will benefit public health a lot.

표-723. PubMed 논문번호 31339350의 내용 요약

구분	내용
PubMed ID	31339350
TITLE	Mortality Risk and Fine Particulate Air Pollution in a Large, Representative Cohort of U.S. Adults.
JOURNAL	Environmental health perspectives: 10.1289/EHP4438
AUTHORS	Pope C Arden, Lefler Jacob S, Ezzati Majid, Higbee Joshua D, Marshall Julian D, Kim Sun-Young, Bechle Matthew, Gilliat Kurtis S, Vernon Spencer E, Robinson Allen L, Burnett Richard T
BACKGROUND	Evidence indicates that air pollution contributes to cardiopulmonary mortality. There is ongoing debate regarding the size and shape of the pollution-mortality exposure-response relationship. There are also growing appeals for estimates of pollution-mortality relationships that use public data and are based on large, representative study cohorts.
OBJECTIVES	Our goal was to evaluate fine particulate matter air pollution ([Formula: see text]) and mortality using a large cohort that is representative of the U.S. population and is based on public data. Additional objectives included exploring model sensitivity, evaluating relative effects across selected subgroups, and assessing the shape of the [Formula: see text]-mortality relationship.
METHODS	National Health Interview Surveys (1986-2014), with mortality linkage through 2015, were used to create a cohort of 1,599,329 U.S. adults and a subcohort with information on smoking and body mass index (BMI) of 635,539 adults. Data were linked with modeled ambient [Formula: see text] at the census-tract level. Cox proportional hazards models were used to estimate [Formula: see text]-mortality hazard ratios for all-cause and specific causes of death while controlling for individual risk factors and regional and urban versus rural differences. Sensitivity and subgroup analyses were conducted and the shape of the [Formula: see text]-mortality relationship was explored.
RESULTS	Estimated mortality hazard ratios, per [Formula: see text] long-term exposure to [Formula: see text], were 1.12 (95% CI: 1.08, 1.15) for all-cause mortality, 1.23 (95% CI: 1.17, 1.29) for cardiopulmonary mortality, and 1.12 (95% CI: 1.00, 1.26) for lung cancer mortality. In general, [Formula: see text]-mortality associations were consistently positive for all-cause and cardiopulmonary mortality across key modeling choices and across subgroups of sex, age, race-ethnicity, income, education levels, and geographic regions.
DISCUSSION	This large, nationwide, representative cohort of U.S. adults provides robust evidence that long-term [Formula: see text] exposure contributes to cardiopulmonary mortality risk. The ubiquitous and involuntary nature of exposures and the broadly observed effects across subpopulations underscore the public health importance of breathing clean air. https://doi.org/10.1289/EHP4438.

표-724. PubMed 논문번호 31344384의 내용 요약

구분	내용
PubMed ID	31344384
TITLE	The cardiovascular effects of electronic cigarettes: A systematic review of experimental studies.
JOURNAL	Preventive medicine: 10.1016/j.ypmed.2019.105770
AUTHORS	Kennedy Ciaran D, van Schalkwyk May C I, McKee Martin, Pisinger Charlotta
BACKGROUND	Smoking is responsible for substantial cardiovascular morbidity and mortality. Electronic cigarettes have been advocated as a means to reduce this disease burden; by reducing exposure to harmful substances in smokers who are unable to quit. Concerns have been raised however, about cardiovascular effects of their use, with inhalants containing carbonyls and fine particulate matter. We systematically reviewed experimental studies of in vitro, animal, and human cardiovascular effects associated with electronic cigarette use.
METHODS	A literature search was conducted using Ovid MEDLINE & Embase databases, identifying experimental studies investigating cardiovascular effects of electronic cigarette use. Subsequently, Cochrane Risk of Bias tools were used to assess study quality. Any differences in outcomes by conflict of interest and risk of bias status were sought.
RESULTS	38 studies were included, investigating animals (n=6), humans (n=24) and human cardiovascular cells in vitro (n=8). 74.3% of studies found potentially harmful effects. Increased sympathetic nerve activity was observed in human studies, whilst platelet haemostatic processes, reactive oxygen species production and endothelial dysfunction were reported across all study types. Studies with conflicts of interest or median-high risk of bias were less likely to identify potentially harmful effects (p=0.0007, p=0.04 respectively).
DISCUSSION	Most studies suggest potential for cardiovascular harm from electronic cigarette use, through mechanisms that increase risk of thrombosis and atherosclerosis. Notably, studies with conflicts of interest are significantly less likely to identify concerning cardiovascular effects. Included studies examine healthy, adult participants, limiting generalisation to potential high-risk groups including individuals with established cardiovascular disease or young, non-smokers.

표-725. PubMed 논문번호 31347651의 내용 요약

구분	내용
PubMed ID	31347651
TITLE	The role of cardiovascular disease in the relationship between air pollution and incident dementia: a population-based cohort study.
JOURNAL	International journal of epidemiology: 10.1093/ije/dyz154
AUTHORS	Ilango Sindana D, Chen Hong, Hystad Perry, van Donkelaar Aaron, Kwong Jeffrey C, Tu Karen, Martin Randall V, Benmarhnia Tarik
BACKGROUND	Evidence suggests a link between air pollution and dementia. Cardiovascular disease (CVD) may be a potential determinant of dementia. This motivated us to quantify the contribution of CVD to the association between air pollution and dementia.
METHODS	A cohort of Canadian-born residents of Ontario, who participated in the 1996-2003 Canadian Community Health Surveys, was followed through 2013 or until dementia diagnosis. Exposure to nitrogen dioxide (NO2) and fine particulate matter (PM2.5) was estimated with a 3-year average and 5-year lag before dementia diagnosis. Incident CVD was evaluated as a mediator. We used multi-level Cox proportional and Aalen additive hazard regression models, adjusting for individual- and neighbourhood-level risk factors to estimate associations with NO2 and PM2.5. We estimated the total, direct and indirect effects of air pollution on dementia through cardiovascular disease.
RESULTS	This study included 34 391 older adults. At baseline, the mean age of this cohort was 59 years. The risk of dementia was moderately higher among those more exposed to NO2 (hazard ratio (HR) 1.10, 95% confidence interval (CI) 0.99-1.19; and 100 additional cases per 100 000 [standard error (SE) <100x10-5]) and PM2.5 [(HR 1.29, 95% CI 0.99-1.64; 200 additional cases per 100 000] [SE 100x10-5]) after adjusting for covariates; however, these estimates are imprecise. A greater proportion of the relationship between PM2.5 and dementia was mediated through CVD than NO2 for both scales.
CONCLUSIONS	These results suggest some of the association between air pollution and dementia is mediated through CVD, indicating that improving cardiovascular health may prevent dementia in areas with higher exposure to air pollution.

표-726. PubMed 논문번호 31350540의 내용 요약

구분	내용
PubMed ID	31350540
TITLE	Acute Blood Pressure and Cardiovascular Effects of Near-Roadway Exposures With and Without N95 Respirators.
JOURNAL	American journal of hypertension: 10.1093/ajh/hpz113
AUTHORS	Morishita Masako, Wang Lu, Speth Kelly, Zhou Nina, Bard Robert L, Li Fengyao, Brook Jeffrey R, Rajagopalan Sanjay, Brook Robert D
BACKGROUND	The risk for cardiovascular events increases within hours of near-roadway exposures. We aimed to determine the traffic-related air pollution (TRAP) and biological mechanisms involved and if reducing particulate matter <2.5 □m (PM2.5) inhalation is protective.
METHODS	Fifty healthy-adults underwent multiple 2-hour near-roadway exposures (Tuesdays to Fridays) in Ann Arbor during 2 separate weeks (randomized to wear an N95 respirator during 1 week). Monday both weeks, participants rested 2 hours in an exam room (once wearing an N95 respirator). Brachial blood pressure, aortic hemodynamics, and heart rate variability were repeatedly measured during exposures. Endothelial function (reactive hyperemia index [RHI]) was measured post-exposures (Thursdays). Black carbon (BC), total particle count (PC), PM2.5, noise and temperature were measured throughout exposures.
RESULTS	PM2.5 (9.3 ± 7.7 □g/m3), BC (1.3 ± 0.6 □g/m3), PC (8,375 ± 4,930 particles/cm3) and noise (69.2 ± 4.2 dB) were higher (P values <0.01) and aortic hemodynamic parameters trended worse while near-roadway (P values<0.15 vs. exam room). Other outcomes were unchanged. Aortic hemodynamics trended towards improvements with N95 respirator usage while near-roadway (P values<0.15 vs. no-use), whereas other outcomes remained unaffected. Higher near-roadway PC and BC exposures were associated with increases in aortic augmentation pressures (P values<0.05) and trends toward lower RHI (P values <0.2). N95 respirator usage did not mitigate these adverse responses (nonsignificant pollutant-respirator interactions). Near-roadway outdoor-temperature and noise were also associated with cardiovascular changes.
CONCLUSIONS	Exposure to real-world combustion-derived particulates in TRAP, even at relatively low concentrations, acutely worsened aortic hemodynamics. Our mixed findings regarding the health benefits of wearing N95 respirators support that further studies are needed to validate if they adequately protect against TRAP given their growing worldwide usage.

표-727. PubMed 논문번호 31371940의 내용 요약

구분	내용
PubMed ID	31371940
TITLE	Effects of air pollution on acute exacerbation of chronic obstructive pulmonary disease: a descriptive retrospective study (pol-AECOPD).
JOURNAL	International journal of chronic obstructive pulmonary disease: 10.2147/COPD.S192047
AUTHORS	Morantes-Caballero Jairo A, Fajardo Rodriguez Hugo Alberto
Purpose	Acute exacerbation of COPD (AECOPD) is among the most frequent causes for hospital admission, causing morbidity and mortality. Infection is the most frequent cause, and studies on pollution have shown higher hospital admission and mortality with inconsistent results. The objective was to identify if there is a change in levels of particulate matter (PM) during the days leading up to the symptom onset.
Patients and methods	A retrospective study was carried out on medical records of patients with AECOPD from a University Hospital. PM values of the consultation day, onset symptoms, and up to three previous days were recorded. Moreover, clinical presentation, laboratory findings, treatments, and hospital outcomes were recorded.
Results	A total of 250 medical records were included, mean age of 77 years, hospital stay mean of 6.7 days, 26.8% with no previous exposure was identified, coexistence with asthma was 5%, Obstructive Sleep Apnea Syndrome 15%, Pulmonary Hypertension 34%, antibiotic use 62%, ICU admission of 14% with non-invasive mechanical ventilation of 68%, and in-patient mortality of 2.4%. PM 2.5 of 48 hrs before onset symptoms median was 20.1 μg/m3 versus 15 and 16.5 for the day of symptoms and 3 days prior to onset symptoms ($p<0.001$). PM 10 of 46.65 μg/m3, versus 39 and 35.6, respectively ($p<0.001$). Expectoration OR 4.74; Purulence OR 6.58; Pleuritic pain OR 3.62; Antibiotic use OR 2.87, and corticoids use OR 2.62, all with $p<0.05$.
Conclusions	Patients with AECOPD have a higher median of particulate matter 48 hrs prior to symptomatic onset, as well as greater use of antibiotics and corticosteroids.

표-728. PubMed 논문번호 31375335의 내용 요약

구분	내용
PubMed ID	31375335
TITLE	Exposure to second hand smoke and 10-year (2002-2012) incidence of cardiovascular disease in never smokers: The ATTICA cohort study.
JOURNAL	International journal of cardiology: 10.1016/j.ijcard.2019.07.065
AUTHORS	Critselis Elena, Panagiotakos Demosthenes B, Georgousopoulou Ekavi N, Katsaounou Paraskevi, Chrysohoou Christina, Pitsavos Christos
BACKGROUND	Despite WHO Framework Convention of Tobacco Control (FCTC) adoption, effective implementation of national smoking bans remains pending in several countries. This study quantified the association of second hand smoke (SHS) exposure and 10-year cardiovascular disease (CVD) among never smokers in such settings.
METHODS	In 2001-2002, a sample of 1514 males and 1528 females (range: 18-89 years old) were randomly selected in Greece. Frequency and duration of SHS exposure (i.e. exposure extending >30 min/day) within the home and/or workplace were assessed by interview. Following a 10-year follow-up period (2002-2012), incidence of non-fatal and fatal CVD (ICD-10) was evaluated among n = 2020 participants. The analytic study sample consisted of all never smokers (n = 910).
RESULTS	Despite national smoking ban implementation (2009), 44.6% (n = 406) of never smokers reported SHS exposure. While SHS exposed never smokers exhibited a more favorable profile of CVD-related risk factors at baseline, they subsequently developed similar 10-year CVD incidence rates, at a younger mean age (p = 0.001), than their non-exposed counterparts. Following adjustment for several lifestyle and clinical factors, SHS exposed never smokers exhibited a two-fold elevated 10-year CVD risk (adj. HR: 2.04, 95% CI: 1.43-2.92), particularly among women (adj. HR: 2.45, 95% CI: 1.45-4.06). SHS exposure accounted for 32% excess Population Attributable Risk (PAR) for 10-year CVD events in never smokers, with highest rates (PAR: 52%) being among those exposed in the workplace.
CONCLUSION	The prevention of SHS associated CVD and related healthcare costs mandates additional strategies for securing the effective implementation of comprehensive WHO FCTC based national smoking bans.

표-729. PubMed 논문번호 31377785의 내용 요약

구분	내용
PubMed ID	31377785
TITLE	Ischaemic heart disease and stroke mortality by specific coal type among non-smoking women with substantial indoor air pollution exposure in China.
JOURNAL	International journal of epidemiology: 10.1093/ije/dyz158
AUTHORS	Bassig Bryan A, Dean Hosgood H, Shu Xiao-Ou, Vermeulen Roel, Chen Bingshu E, Katki Hormuzd A, Seow Wei Jie, Hu Wei, Portengen L□tzen, Ji Bu-Tian, Wong Jason Y Y, Ning Bofu, Downward George S, Li Jihua, Yang Kaiyun, Yang Gong, Gao Yu-Tang, Xiang Yong-Bing, Nagaradona Teja, Zheng Wei, Silverman Debra T, Huang Yunchao, Lan Qing
BACKGROUND	Lifetime use of bituminous ('smoky') coal is associated with nearly a 100-fold higher risk of lung cancer mortality compared with anthracite ('smokeless') coal use in rural Xuanwei, China, among women. Risk of mortality from ischaemic heart disease (IHD) and stroke for these coal types has not been evaluated.
METHODS	A cohort of 16 323 non-smoking women in Xuanwei, who were lifetime users of either smoky or smokeless coal, were followed up from 1976 to 2011. We estimated hazard ratios (HRs) and 95% confidence intervals (CI) to evaluate lifetime use of coal types and stoves in the home in relation to risk of IHD and stroke mortality.
RESULTS	Among lifetime users of smokeless coal, higher average exposure intensity (≥4 tons/year vs <2.5 tons/year, HR = 7.9, 95% CI = 3.5-17.8; Ptrend =<0.0001) and cumulative exposure (>64 ton-years vs ≤28 ton-years, HR = 6.5, 95% CI = 1.5-28.3; Ptrend =0.003) during follow-up and over their lifetime was associated with increased IHD mortality, and ventilated stove use dramatically reduced this risk (HR = 0.2, 95% CI 0.1-0.5). Higher cumulative exposure to smoky coal during follow-up showed positive associations with IHD mortality, but the evidence for other metrics was less consistent compared with associations with smokeless coal use.
CONCLUSIONS	Higher use of smokeless coal, which is burned throughout China and is generally regarded to be a cleaner fuel type, is associated with IHD mortality. Use of cleaner fuels or stove interventions may be effective in reducing the increasing burden of IHD in developing regions that currently rely on smokeless coal for cooking and heating.

표-730. PubMed 논문번호 31382185의 내용 요약

구분	내용
PubMed ID	31382185
TITLE	Short-term exposure to air pollution and incidence of stroke in the Women's Health Initiative.
JOURNAL	Environment international: 10.1016/j.envint.2019.105065
AUTHORS	Sun Shengzhi, Stewart James D, Eliot Melissa N, Yanosky Jeff D, Liao Duanping, Tinker Lesley F, Eaton Charles B, Whitsel Eric A, Wellenius Gregory A
BACKGROUND	Evidence of the association between daily variation in air pollution and risk of stroke is inconsistent, potentially due to the heterogeneity in stroke etiology.
OBJECTIVES	To estimate the associations between daily variation in ambient air pollution and risk of stroke and its subtypes among participants of the Women's Health Initiative, a large prospective cohort study in the United States.
METHODS	We used national-scale, log-normal ordinary kriging models to estimate daily concentrations of fine particulate matter (PM2.5), respirable particulate matter (PM10), nitrogen dioxide (NO2), nitrogen oxides (NOx), sulphur dioxide, and ozone at participant addresses. Stroke was adjudicated by trained neurologists and classified as ischemic or hemorrhagic. Ischemic strokes were further classified according to the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification. We used a time-stratified case-crossover approach to estimate the odds ratio (OR) of the risk of stroke associated with an interquartile range (IQR) increase in concentrations of each air pollutant. We performed stratified analysis to examine whether associations varied across subgroups defined by age at stroke onset, US census region, smoking status, body mass index, and prior history of diabetes mellitus, hypertension, heart or circulation problems, or arterial fibrillation at enrollment.
RESULTS	Among 5417 confirmed strokes between 1993 and 2012, 4300 (79.4%) were classified as ischemic and 924 (17.1%) as hemorrhagic. No association was observed between day-to-day variation in any pollutant and risk of total stroke, ischemic stroke, or specific etiologies of ischemic stroke. We observed a positive association between risk of hemorrhagic stroke and NO2 and NOx in the 3 days prior to stroke with OR of 1.24 (95% CI: 1.01, 1.52) and 1.18 (95% CI: 1.03, 1.34) per IQR increase, respectively. The observed associations with hemorrhagic stroke were more pronounced among non-obese participants.
CONCLUSIONS	In this large cohort of post-menopausal US women, daily NO2 and NOx were associated with higher risk of hemorrhagic stroke, but ambient levels of four other air pollutants were not associated with higher risk of total stroke, ischemic stroke, or ischemic stroke subtypes.

표-731. PubMed 논문번호 31408135의 내용 요약

구분	내용
PubMed ID	31408135
TITLE	Association Between Long-term Exposure to Ambient Air Pollution and Change in Quantitatively Assessed Emphysema and Lung Function.
JOURNAL	JAMA: 10.1001/jama.2019.10255
AUTHORS	Wang Meng, Aaron Carrie Pistenmaa, Madrigano Jaime, Hoffman Eric A, Angelini Elsa, Yang Jie, Laine Andrew, Vetterli Thomas M, Kinney Patrick L, Sampson Paul D, Sheppard Lianne E, Szpiro Adam A, Adar Sara D, Kirwa Kipruto, Smith Benjamin, Lederer David J, Diez-Roux Ana V, Vedal Sverre, Kaufman Joel D, Barr R Graham
Importance	While air pollutants at historical levels have been associated with cardiovascular and respiratory diseases, it is not known whether exposure to contemporary air pollutant concentrations is associated with progression of emphysema.
Objective	To assess the longitudinal association of ambient ozone (O3), fine particulate matter (PM2.5), oxides of nitrogen (NOx), and black carbon exposure with change in percent emphysema assessed via computed tomographic (CT) imaging and lung function.
Design, Setting, and Participants	This cohort study included participants from the Multi-Ethnic Study of Atherosclerosis (MESA) Air and Lung Studies conducted in 6 metropolitan regions of the United States, which included 6814 adults aged 45 to 84 years recruited between July 2000 and August 2002, and an additional 257 participants recruited from February 2005 to May 2007, with follow-up through November 2018.
Exposures	Residence-specific air pollutant concentrations (O3, PM2.5, NOx, and black carbon) were estimated by validated spatiotemporal models incorporating cohort-specific monitoring, determined from 1999 through the end of follow-up.
Main Outcomes and Measures	Percent emphysema, defined as the percent of lung pixels less than −950 Hounsfield units, was assessed up to 5 times per participant via cardiac CT scan (2000-2007) and equivalent regions on lung CT scans (2010-2018). Spirometry was performed up to 3 times per participant (2004-2018).
Results	Among 7071 study participants (mean [range] age at recruitment, 60 [45-84] years; 3330 [47.1%] were men), 5780 were assigned outdoor residential air pollution concentrations in the year of their baseline examination and during the follow-up period and had at least 1 follow-up CT scan, and 2772 had at least 1 follow-up spirometric assessment, over a median of 10 years. Median percent emphysema was 3% at baseline and increased a mean of 0.58 percentage points per 10 years. Mean ambient concentrations of PM2.5 and NOx, but not O3, decreased substantially during follow-up. Ambient concentrations of O3, PM2.5, NOx, and black carbon at study baseline were significantly associated with greater increases in percent emphysema per 10 years (O3: 0.13 per 3 parts per billion [95% CI, 0.03-0.24]; PM2.5: 0.11 per 2 μg/m3 [95% CI, 0.03-0.19]; NOx: 0.06 per 10 parts per billion [95% CI, 0.01-0.12]; black carbon: 0.10 per 0.2 μg/m3 [95% CI, 0.01-0.18]). Ambient O3 and NOx concentrations, but not PM2.5 concentrations, during follow-up were also significantly associated with greater increases in percent emphysema. Ambient O3 concentrations, but not other pollutants, at baseline and during follow-up were significantly associated with a greater decline in forced expiratory volume in 1 second per 10 years (baseline: 13.41 mL per 3

표-732. PubMed 논문번호 31419765의 내용 요약

구분	내용
PubMed ID	31419765
TITLE	Inflammation and acute traffic-related air pollution exposures among a cohort of youth with type 1 diabetes.
JOURNAL	Environment international: 10.1016/j.envint.2019.105064
AUTHORS	Puett Robin C, Yanosky Jeff D, Mittleman Murray A, Montresor-Lopez Jessica, Bell Ronny A, Crume Tessa L, Dabelea Dana, Dolan Lawrence M, D'Agostino Ralph B, Marcovina Santica M, Pihoker Catherine, Reynolds Kristi, Urbina Elaine, Liese Angela D
BACKGROUND	Evidence remains equivocal regarding the association of inflammation, a precursor to cardiovascular disease, and acute exposures to ambient air pollution from traffic-related particulate matter. Though youth with type 1 diabetes are at higher risk for cardiovascular disease, the relationship of inflammation and ambient air pollution exposures in this population has received little attention.
OBJECTIVES	Using five geographically diverse US sites from the racially- and ethnically-diverse SEARCH for Diabetes in Youth Cohort, we examined the relationship of acute exposures to PM2.5 mass, Atmospheric Dispersion Modeling System (ADMS)-Roads traffic-related PM concentrations near roadways, and elemental carbon (EC) with biomarkers of inflammation including interleukin-6 (IL-6), c-reactive protein (hs-CRP) and fibrinogen.
METHODS	Baseline questionnaires and blood were obtained at a study visit. Using a spatio-temporal modeling approach, pollutant exposures for 7 days prior to blood draw were assigned to residential addresses. Linear mixed models for each outcome and exposure were adjusted for demographic and lifestyle factors identified a priori.
RESULTS	Among the 2566 participants with complete data, fully-adjusted models showed positive associations of EC average week exposures with IL-6 and hs-CRP, and PM2.5 mass exposures on lag day 3 with IL-6 levels. Comparing the 25th and 75th percentiles of average week EC exposures resulted in 8.3% higher IL-6 (95%CI: 2.7%,14.3%) and 9.8% higher hs-CRP (95%CI: 2.4%,17.7%). We observed some evidence of effect modification for the relationships of PM2.5 mass exposures with hs-CRP by gender and with IL-6 by race/ethnicity.
CONCLUSIONS	Indicators of inflammation were associated with estimated traffic-related air pollutant exposures in this study population of youth with type 1 diabetes. Thus youth with type 1 diabetes may be at increased risk of air pollution-related inflammation. These findings and the racial/ethnic and gender differences observed deserve further exploration.

표-733. PubMed 논문번호 31433918의 내용 요약

구분	내용
PubMed ID	31433918
TITLE	Ambient Particulate Air Pollution and Daily Mortality in 652 Cities.
JOURNAL	The New England journal of medicine: 10.1056/NEJMoa1817364
AUTHORS	Liu Cong, Chen Renjie, Sera Francesco, Vicedo-Cabrera Ana M, Guo Yuming, Tong Shilu, Coelho Micheline S Z S, Saldiva Paulo H N, Lavigne Eric, Matus Patricia, Valdes Ortega Nicolas, Osorio Garcia Samuel, Pascal Mathilde, Stafoggia Massimo, Scortichini Matteo, Hashizume Masahiro, Honda Yasushi, Hurtado-D□az Magali, Cruz Julio, Nunes Baltazar, Teixeira Jo□o P, Kim Ho, Tobias Aurelio, □□iguez Carmen, Forsberg Bertil, □str□m Christofer, Ragettli Martina S, Guo Yue-Leon, Chen Bing-Yu, Bell Michelle L, Wright Caradee Y, Scovronick Noah, Garland Rebecca M, Milojevic Ai, Kysel□ Jan, Urban Ale□, Orru Hans, Indermitte Ene, Jaakkola Jouni J K, Ryti Niilo R I, Katsouyanni Klea, Analitis Antonis, Zanobetti Antonella, Schwartz Joel, Chen Jianmin, Wu Tangchun, Cohen Aaron, Gasparrini Antonio, Kan Haidong
BACKGROUND	The systematic evaluation of the results of time-series studies of air pollution is challenged by differences in model specification and publication bias.
METHODS	We evaluated the associations of inhalable particulate matter (PM) with an aerodynamic diameter of 10 μm or less (PM10) and fine PM with an aerodynamic diameter of 2.5 μm or less (PM2.5) with daily all-cause, cardiovascular, and respiratory mortality across multiple countries or regions. Daily data on mortality and air pollution were collected from 652 cities in 24 countries or regions. We used overdispersed generalized additive models with random-effects meta-analysis to investigate the associations. Two-pollutant models were fitted to test the robustness of the associations. Concentration-response curves from each city were pooled to allow global estimates to be derived.
RESULTS	On average, an increase of 10 μg per cubic meter in the 2-day moving average of PM10 concentration, which represents the average over the current and previous day, was associated with increases of 0.44% (95% confidence interval [CI], 0.39 to 0.50) in daily all-cause mortality, 0.36% (95% CI, 0.30 to 0.43) in daily cardiovascular mortality, and 0.47% (95% CI, 0.35 to 0.58) in daily respiratory mortality. The corresponding increases in daily mortality for the same change in PM2.5 concentration were 0.68% (95% CI, 0.59 to 0.77), 0.55% (95% CI, 0.45 to 0.66), and 0.74% (95% CI, 0.53 to 0.95). These associations remained significant after adjustment for gaseous pollutants. Associations were stronger in locations with lower annual mean PM concentrations and higher annual mean temperatures. The pooled concentration-response curves showed a consistent increase in daily mortality with increasing PM concentration, with steeper slopes at lower PM concentrations.
CONCLUSIONS	Our data show independent associations between short-term exposure to PM10 and PM2.5 and daily all-cause, cardiovascular, and respiratory mortality in more than 600 cities across the globe. These data reinforce the evidence of a link between mortality and PM concentration established in regional and local studies. (Funded by the National Natural Science Foundation of China and others.).

표-734. PubMed 논문번호 31444115의 내용 요약

구분	내용
PubMed ID	31444115
TITLE	Effects of short-term exposure to fine and ultrafine particles from indoor sources on arterial stiffness - A randomized sham-controlled exposure study.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2019.08.002
AUTHORS	Soppa Vanessa J, Shinnawi Samir, Hennig Frauke, Sasse Birgitta, Hellack Bryan, Kaminski Heinz, Quass Ulrich, Schins Roel P F, Kuhlbusch Thomas A J, Hoffmann Barbara
OBJECTIVES	Particulate air pollution is linked to adverse cardiovascular effects, including arterial stiffness. The aim of the study was to investigate the effect of short-term exposure to indoor fine and ultrafine particles on augmentation index (AIx), augmentation pressure (AP), and pulse wave velocity (PWV), early signs of vascular damage.
METHODS	We analyzed the association of particle emissions from typical indoor sources (candle burning - CB, toasting bread - TB, and frying sausages - FS) with changes in pulse wave analysis indices in 55 healthy adults in a randomized cross-over controlled exposure study. Particle mass concentration (PMC), size-specific particle number concentration (PNC) and lung-deposited particle surface area concentration (PSC) were measured during the 2 h exposure. AIx and AP were measured before, directly, 2, 4 and 24 h after exposure. PWV was measured directly and 24 h after exposure. We performed multiple mixed linear regression analyses of different particle metrics and AIx, AP and PWV.
RESULTS	The highest mean PMC was observed during FS reaching a maximum of 210 μg/m3 PM10. The maximal PNC for UFP <100 nm was reached during CB with 2.3 million particles/cm3. PSC was similar across all three exposures (about 3000 μm2/cm^3). Strongest associations between different particles metrics and arterial stiffness indices could be observed for UFP from CB and FS and for PMC from TB. The highest mean increase could be observed for the UFP fraction <10 nm, measured during CB, and AIx with an increase of 9.5%-points (95%-CI: 3.1; 15.9). PSC seemed to follow the pattern of PNC. PM10 and PM2.5 from TB led to clear changes in AIx with biggest increases for PM10 of 5.8%-points (95%-CI: 3.2; 8.4) 2 h after exposure and for PM2.5 of 8.1%-points (95%-CI: 2.5; 13.7) directly after exposure.
CONCLUSIONS	Our study indicates effects of indoor exposure to fine and ultrafine particles on systemic arterial stiffness indices that depend on the indoor source as well as on particle metric. Differences in size-specific physical characteristics of source-specific particles might account for these differential effects. We did not observe clear and stable associations of indoor particle exposure and PWV.

표-735. PubMed 논문번호 31449466의 내용 요약

구분	내용
PubMed ID	31449466
TITLE	Ambient Air Pollution and the Risk of Atrial Fibrillation and Stroke: A Population-Based Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP4883
AUTHORS	Shin Saeha, Burnett Richard T, Kwong Jeffrey C, Hystad Perry, van Donkelaar Aaron, Brook Jeffrey R, Goldberg Mark S, Tu Karen, Copes Ray, Martin Randall V, Liu Ying, Kopp Alexander, Chen Hong
BACKGROUND	Although growing evidence links air pollution to stroke incidence, less is known about the effect of air pollution on atrial fibrillation (AF), an important risk factor for stroke.
OBJECTIVES	We assessed the associations between air pollution and incidence of AF and stroke. We also sought to characterize the shape of pollutant-disease relationships.
METHODS	The population-based cohort comprised 5,071,956 Ontario residents, age 35-85 y and without the diagnoses of both outcomes on 1 April 2001 and was followed up until 31 March 2015. AF and stroke cases were ascertained using health administrative databases with validated algorithms. Based on annual residential postal codes, we assigned 5-y running average concentrations of fine particulate matter ([Formula: see text]), nitrogen dioxide ([Formula: see text]), and ozone ([Formula: see text]) from satellite-derived data, a land-use regression model, and a fusion-based method, respectively, as well as redox-weighted averages of [Formula: see text] and [Formula: see text] ([Formula: see text]) for each year. Using Cox proportional hazards models, we estimated the hazard ratios (HRs) and 95% confidence intervals (95% CIs) of AF and stroke with each of these pollutants, adjusting for individual- and neighborhood-level variables. We used newly developed nonlinear risk models to characterize the shape of pollutant-disease relationships.
RESULTS	Between 2001 and 2015, we identified 313,157 incident cases of AF and 122,545 cases of stroke. Interquartile range increments of [Formula: see text], [Formula: see text], [Formula: see text], and [Formula: see text] were associated with increases in the incidence of AF [HRs (95% CIs): 1.03 (1.01, 1.04), 1.02 (1.01, 1.03), 1.01 (1.00, 1.02), and 1.01 (1.01, 1.02), respectively] and the incidence of stroke [HRs (95% CIs): 1.05 (1.03, 1.07), 1.04 (1.01, 1.06), 1.05 (1.03, 1.06), and 1.05 (1.04, 1.06), respectively]. Associations of similar magnitude were found in various sensitivity analyses. Furthermore, we found a near-linear association for stroke with [Formula: see text], whereas [Formula: see text], [Formula: see text]-, and [Formula: see text] relationships exhibited sublinear shapes.
CONCLUSIONS	Air pollution was associated with stroke and AF onset, even at very low concentrations. https://doi.org/10.1289/EHP4883.

표-736. PubMed 논문번호 31470256의 내용 요약

구분	내용
PubMed ID	31470256
TITLE	The limits of single-group interrupted time series analysis in assessing the impact of smoke-free laws on short-term mortality.
JOURNAL	The International journal on drug policy: 10.1016/j.drugpo.2019.07.018
AUTHORS	Barrio Gregorio, Belza Mar□a J, Carmona Roc□o, Hoyos Juan, Ronda Elena, Regidor Enrique
BACKGROUND	Decreases in circulatory/respiratory morbimortality after the January-2006 Spanish partial smoke-free law have been found using designs without control groups, such as single-group interrupted time series (ITS), which are prone to biases. The aim was to reassess the law's impact on mortality using ITS designs with robustness checks.
METHODS	A comprehensive cohort of people aged ≥25 in each calendar-year of 2002-2007, living in 13 of 18 Spanish regions, was followed up between 01/2002 and 12/2007. The law included a smoking ban in indoor public and workplaces, allowing exceptions in catering, hospitality and leisure venues, and other interventions. Post-law changes in monthly coronary/respiratory mortality were estimated using segmented regression, adjusting for relevant covariates, including seasonality, extreme temperatures, influenza incidence and air pollution. The validity of results was assessed using control outcomes, hypothetical law dates, and non-equivalent control groups, analysing their results as difference-in-differences (DID) designs.
RESULTS	Significant immediate post-law decreases in coronary, respiratory and non-tobacco-related mortality were observed among people aged ≥70. A significant immediate post-law decrease in respiratory mortality (-12.7%) was also observed among people age 25-69, although this was neutralized by a subsequent upward trend before 1.5 years. More favourable post-law changes in coronary/respiratory mortality among the target (people aged 25-69) than control groups (people aged ≥70 or women aged ≥80) were not identified in DID designs. Establishing hypothetical law dates, immediate decreases began in February/March 2005 with maxima between April and July 2005.
CONCLUSIONS	After robustness checks, the results do not support a clear positive impact of the 2006 Spanish smoke-free law on short-term coronary/respiratory mortality. The favourable immediate changes observed pre- and post-law could derive mainly from the harvesting effect of the January-2005 cold wave. This highlights the risks of assessing the impact of health interventions using both morbimortality outcomes and designs without a control group and adequate robustness checks.

표-737. PubMed 논문번호 31476158의 내용 요약

구분	내용
PubMed ID	31476158
TITLE	Fine Particulate Matter Exposure and Cerebrospinal Fluid Markers of Vascular Injury.
JOURNAL	Journal of Alzheimer's disease : JAD: 10.3233/JAD-190563
AUTHORS	Shaffer Rachel M, Sheppard Lianne, Peskind Elaine R, Zhang Jing, Adar Sara D, Li Ge
BACKGROUND	Cerebrovascular diseases play an important role in dementia. Air pollution is associated with cardiovascular disease, with growing links to neurodegeneration. Prior studies demonstrate associations between fine particulate matter (PM2.5) and biomarkers of endothelial injury in the blood; however, no studies have evaluated these biomarkers in cerebrospinal fluid (CSF).
OBJECTIVE	We evaluate associations between short-term and long-term PM2.5 exposure with CSF vascular cell adhesion molecule-1 (VCAM-1) and e-selectin in cognitively normal and mild cognitive impairment (MCI)/Alzheimer's disease (AD) individuals.
METHODS	We collected CSF from 133 community volunteers at VA Puget Sound between 2001-2012. We assigned short-term PM2.5 from central monitors and long-term PM2.5 based on annual average exposure predictions linked to participant addresses. We performed analyses stratified by cognitive status and adjusted for key covariates with tiered models. Our primary exposure windows for the short-term and long-term analyses were 7-day and 1-year averages, respectively.
RESULTS	Among cognitively normal individuals, a 5 μg/m3 increase in 7-day and 1-year average PM2.5 was associated with elevated VCAM-1 (7-day: 35.4 (9.7, 61.1) ng/ml; 1-year: 51.8 (6.5, 97.1) ng/ml). A 5 μg/m3 increase in 1-year average PM2.5, but not 7-day average, was associated with elevated e-selectin (53.3 (11.0, 95.5) pg/ml). We found no consistent associations among MCI/AD individuals.
CONCLUSIONS	We report associations between short-term and long term PM2.5 and CSF biomarkers of vascular damage in cognitively normal adults. These results are aligned with prior research linking PM2.5 to vascular damage in other biofluids as well as emerging evidence of the role of PM2.5 in neurodegeneration.

표-738. PubMed 논문번호 31488269의 내용 요약

구분	내용
PubMed ID	31488269
TITLE	Early-Life Environmental Exposures and Blood Pressure in Children.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2019.06.069
AUTHORS	Warembourg Charline, Maitre L□a, Tamayo-Uria Ibon, Fossati Serena, Roumeliotaki Theano, Aasvang Gunn Marit, Andrusaityte Sandra, Casas Maribel, Cequier Enrique, Chatzi Lida, Dedele Audrius, Gonzalez Juan-Ramon, Gra□ulevi□ien□ Regina, Haug Line Smastuen, Hernandez-Ferrer Carles, Heude Barbara, Karachaliou Marianna, Krog Norun Hjertager, McEachan Rosemary, Nieuwenhuijsen Mark, Petraviciene Inga, Quentin Joane, Robinson Oliver, Sakhi Amrit Kaur, Slama R□my, Thomsen Cathrine, Urquiza Jose, Vafeiadi Marina, West Jane, Wright John, Vrijheid Martine, Basaga□a Xavier
BACKGROUND	Growing evidence exists about the fetal and environmental origins of hypertension, but mainly limited to single-exposure studies. The exposome has been proposed as a more holistic approach by studying many exposures simultaneously.
OBJECTIVES	This study aims to evaluate the association between a wide range of prenatal and postnatal exposures and blood pressure (BP) in children.
METHODS	Systolic and diastolic BP were measured among 1,277 children from the European HELIX (Human Early-Life Exposome) cohort aged 6 to 11 years. Prenatal (n = 89) and postnatal (n = 128) exposures include air pollution, built environment, meteorology, natural spaces, traffic, noise, chemicals, and lifestyles. Two methods adjusted for confounders were applied: an exposome-wide association study considering the exposures independently, and the deletion-substitution-addition algorithm considering all the exposures simultaneously.
RESULTS	Decreases in systolic BP were observed with facility density (β change for an interquartile-range increase in exposure: -1.7 mm Hg [95% confidence interval (CI): -2.5 to -0.8 mm Hg]), maternal concentrations of polychlorinated biphenyl 118 (-1.4 mm Hg [95% CI: -2.6 to -0.2 mm Hg]) and child concentrations of dichlorodiphenyldichloroethylene (DDE: -1.6 mm Hg [95% CI: -2.4 to -0.7 mm Hg]), hexachlorobenzene (-1.5 mm Hg [95% CI: -2.4 to -0.6 mm Hg]), and mono-benzyl phthalate (-0.7 mm Hg [95% CI: -1.3 to -0.1 mm Hg]), whereas increases in systolic BP were observed with outdoor temperature during pregnancy (1.6 mm Hg [95% CI: 0.2 to 2.9 mm Hg]), high fish intake during pregnancy (2.0 mm Hg [95% CI: 0.4 to 3.5 mm Hg]), maternal cotinine concentrations (1.2 mm Hg [95% CI: -0.3 to 2.8 mm Hg]), and child perfluorooctanoate concentrations (0.9 mm Hg [95% CI: 0.1 to 1.6 mm Hg]). Decreases in diastolic BP were observed with outdoor temperature at examination (-1.4 mm Hg [95% CI: -2.3 to -0.5 mm Hg]) and child DDE concentrations (-1.1 mm Hg [95% CI: -1.9 to -0.3 mm Hg]), whereas increases in diastolic BP were observed with maternal bisphenol-A concentrations (0.7 mm Hg [95% CI: 0.1 to 1.4 mm Hg]), high fish intake during pregnancy (1.2 mm Hg [95% CI: -0.2 to 2.7 mm Hg]), and child copper concentrations (0.9 mm Hg [95% CI: 0.3 to 1.6 mm Hg]).
CONCLUSIONS	This study suggests that early-life exposure to several chemicals, as well as built environment and meteorological factors, may affect BP in children.

표-739. PubMed 논문번호 31490535의 내용 요약

구분	내용
PubMed ID	31490535
TITLE	Long-term Cardiovascular Disease Risk Among Firefighters After the World Trade Center Disaster.
JOURNAL	JAMA network open: 10.1001/jamanetworkopen.2019.9775
AUTHORS	Cohen Hillel W, Zeig-Owens Rachel, Joe Cynthia, Hall Charles B, Webber Mayris P, Weiden Michael D, Cleven Krystal L, Jaber Nadia, Skerker Molly, Yip Jennifer, Schwartz Theresa, Prezant David J
Importance	Published studies examining the association between World Trade Center (WTC) exposure on and after September 11, 2001, and longer-term cardiovascular disease (CVD) outcomes have reported mixed findings.
Objective	To assess whether WTC exposure was associated with elevated CVD risk in Fire Department of the City of New York (FDNY) firefighters.
Design, Settings, and Participants	In this cohort study, the association between WTC exposure and the risk of CVD was assessed between September 11, 2001, and December 31, 2017, in FDNY male firefighters. Multivariable Cox regression analyses were used to estimate CVD risk in association with 2 measures of WTC exposure: arrival time to the WTC site and duration of work at the WTC site. Data analyses were conducted from May 1, 2018, to March 8, 2019.
Main Outcomes and Measures	The primary CVD outcome included myocardial infarction, stroke, unstable angina, coronary artery surgery or angioplasty, or CVD death. The secondary outcome (all CVD) included all primary outcome events or any of the following: transient ischemic attack; stable angina, defined as either use of angina medication or cardiac catheterization without intervention; cardiomyopathy; and other CVD (aortic aneurysm, peripheral arterial vascular intervention, and carotid artery surgery).
Results	There were 489 primary outcome events among 9796 male firefighters (mean [SD] age on September 11, 2001, was 40.3 [7.4] years and 7210 individuals [73.6%] were never smokers). Age-adjusted incident rates of CVD were higher for firefighters with greater WTC exposure. The multivariable adjusted hazard ratio (HR) for the primary CVD outcome was 1.44 (95% CI, 1.09-1.90) for the earliest arrival group compared with those who arrived later. Similarly, those who worked at the WTC site for 6 or more months vs those who worked less time at the site were more likely to have a CVD event (HR, 1.30; 95% CI, 1.05-1.60). Well-established CVD risk factors, including hypertension (HR, 1.41; 95% CI, 1.10-1.80), hypercholesterolemia (HR, 1.56; 95% CI, 1.28-1.91), diabetes (HR, 1.99; 95% CI, 1.33-2.98), and smoking (current: HR, 2.13; 95% CI, 1.68-2.70; former: HR, 1.55; 95% CI, 1.23-1.95), were significantly associated with CVD in the multivariable models. Analyses with the all-CVD outcome were similar.
Conclusions and Relevance	The findings of the study suggest a significant association between greater WTC exposure and long-term CVD risk. The findings appear to reinforce the importance of long-term monitoring of the health of survivors of disasters.

표-740. PubMed 논문번호 31491811의 내용 요약

구분	내용
PubMed ID	31491811
TITLE	The cross-sectional and longitudinal association between air pollution and salivary cortisol: Evidence from the Multi-Ethnic Study of Atherosclerosis.
JOURNAL	Environment international: 10.1016/j.envint.2019.105062
AUTHORS	Hajat Anjum, Hazlehurst Marnie F, Golden Sherita Hill, Merkin Sharon Stein, Seeman Teresa, Szpiro Adam A, Kaufman Joel D, Roux Ana Diez
BACKGROUND	Cortisol, a stress hormone released by the activation of the hypothalamic-pituitary-adrenal (HPA) axis, is critical to the body's adaptive response to physiological and psychological stress. Cortisol has also been implicated in the health effects of air pollution through the activation of the sympathetic nervous system. This study evaluates the cross-sectional and longitudinal association between several air pollutants and salivary cortisol.
METHODS	We used data from the Multi-Ethnic Study of Atherosclerosis (MESA), a cohort of 45-85 years old participants from six US cities. Salivary cortisol was evaluated at two time points between 2004 and 2006 and then again from 2010 to 2012. Cortisol samples were taken several times per day on two or three consecutive days. Particulate matter <2.5 μm in diameter (PM2.5), nitrogen dioxide (NO2) and nitrogen oxides (NOx) in the year prior to cortisol sampling were examined. We used piecewise linear mixed models that were adjusted for demographics, socioeconomic status and cardiovascular risk factors to examine both cross-sectional and longitudinal associations. Longitudinal models evaluated change in cortisol over time.
RESULTS	The pooled cross-sectional results revealed largely null results with the exception of a 9.7% higher wake-up cortisol associated with a 10 ppb higher NO2 (95% CI, -0.2%, 20.5%). Among all participants, the features of the cortisol curve became flatter over 5 years. The wake-to-bed slope showed a more pronounced flattening over time (0.014, 95% CI, 0.0, 0.03) with a 10 ppb higher NO2 level. Other air pollutants were not associated with change in cortisol over time.
CONCLUSIONS	Our results suggest only a moderate association between traffic related air pollution and cortisol. Very few epidemiologic studies have examined the long-term impact of air pollution on the stress response systems, thus warranting further exploration of these findings.

표-741. PubMed 논문번호 31492503의 내용 요약

구분	내용
PubMed ID	31492503
TITLE	Modifiable risk factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study.
JOURNAL	Lancet (London, England): 10.1016/S0140-6736(19)32008-2
AUTHORS	Yusuf Salim, Joseph Philip, Rangarajan Sumathy, Islam Shofiqul, Mente Andrew, Hystad Perry, Brauer Michael, Kutty Vellappillil Raman, Gupta Rajeev, Wielgosz Andreas, AlHabib Khalid F, Dans Antonio, Lopez-Jaramillo Patricio, Avezum Alvaro, Lanas Fernando, Oguz Aytekin, Kruger Iolanthe M, Diaz Rafael, Yusoff Khalid, Mony Prem, Chifamba Jephat, Yeates Karen, Kelishadi Roya, Yusufali Afzalhussein, Khatib Rasha, Rahman Omar, Zatonska Katarzyna, Iqbal Romaina, Wei Li, Bo Hu, Rosengren Annika, Kaur Manmeet, Mohan Viswanathan, Lear Scott A, Teo Koon K, Leong Darryl, O'Donnell Martin, McKee Martin, Dagenais Gilles, Yusuf Salim, Joseph Philip, Rangarajan Sumathy, Islam Shofiqul, Mente Andrew, Hystad Perry, Brauer Michael, Kutty Vellappillil Raman, Gupta Rajeev, Wielgosz Andreas, AlHabib Khalid F, Dans Antonio, Lopez-Jaramillo Patricio, Avezum Alvaro, Lanas Fernando, Oguz Aytekin, Kruger Iolanthe M, Diaz Rafael, Yusoff Khalid, Mony Prem, Chifamba Jephat, Yeates Karen, Kelishadi Roya, Yusufali Afzalhussein, Khatib Rasha, Rahman Omar, Zatonska Katarzyna, Iqbal Romaina, Wei Li, Bo Hu, Rosengren Annika, Kaur Manmeet, Mohan Viswanathan, Lear Scott A, Teo Koon K, Leong Darryl, O'Donnell Martin, McKee Martin, Dagenais Gilles
BACKGROUND	Global estimates of the effect of common modifiable risk factors on cardiovascular disease and mortality are largely based on data from separate studies, using different methodologies. The Prospective Urban Rural Epidemiology (PURE) study overcomes these limitations by using similar methods to prospectively measure the effect of modifiable risk factors on cardiovascular disease and mortality across 21 countries (spanning five continents) grouped by different economic levels.
METHODS	In this multinational, prospective cohort study, we examined associations for 14 potentially modifiable risk factors with mortality and cardiovascular disease in 155 722 participants without a prior history of cardiovascular disease from 21 high-income, middle-income, or low-income countries (HICs, MICs, or LICs). The primary outcomes for this paper were composites of cardiovascular disease events (defined as cardiovascular death, myocardial infarction, stroke, and heart failure) and mortality. We describe the prevalence, hazard ratios (HRs), and population-attributable fractions (PAFs) for cardiovascular disease and mortality associated with a cluster of behavioural factors (ie, tobacco use, alcohol, diet, physical activity, and sodium intake), metabolic factors (ie, lipids, blood pressure, diabetes, obesity), socioeconomic and psychosocial factors (ie, education, symptoms of depression), grip strength, and household and ambient pollution. Associations between risk factors and the outcomes were established using multivariable Cox frailty models and using PAFs for the entire cohort, and also by countries grouped by income level. Associations are presented as HRs and PAFs with 95% CIs.
FINDINGS	Between Jan 6, 2005, and Dec 4, 2016, 155 722 participants were enrolled and followed up for measurement of risk factors. 17 249 (11·1%) participants were from HICs, 102 680 (65·9%) were from MICs, and 35 793 (23·0%) from LICs. Approximately 70% of cardiovascular disease cases and deaths in the overall study population were attributed to modifiable risk factors. Metabolic

표-742. PubMed 논문번호 31492618의 내용 요약

구분	내용
PubMed ID	31492618
TITLE	Cardiovascular disease and long-term occupational exposure to ultrafine particles: A cohort study of airport workers.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2019.08.010
AUTHORS	M ø ller Karina Lauenborg, Brauer Charlotte, Mikkelsen Sigurd, Bonde Jens Peter, Loft Steffen, Helweg-Larsen Karin, Thygesen Lau Caspar, M ø ller Karina Lauenborg, Brauer Charlotte, Mikkelsen Sigurd, Bonde Jens Peter, Loft Steffen, Helweg-Larsen Karin, Thygesen Lau Caspar
AIM	To investigate if ischemic heart disease (IHD) and cerebrovascular disease is associated with long-term occupational exposure to ultrafine particles (UFP) outdoors at an airport.
METHODS AND RESULTS	This is a register-based follow-up study based on a cohort comprising an exposed group of 6515 men employed in unskilled work at Copenhagen Airport and a reference group of 61,617 men in unskilled work in other firms in greater Copenhagen during 1990-2012. The exposure was assessed from information on proportion of time spent on the airport apron for each calendar year (apron-years) and the primary exposure measure was cumulated apron-years. The cohort was merged to the National Patient Register that includes data on all contacts to public and private hospitals in Denmark and the Register of Causes of Death. Risk estimates were provided by Poisson regression and adjusted for age, calendar year and educational level. We found no associations between cumulative apron-years and IHD (IRR, 1.00; 95%CI, 0.97-1.03) or cerebrovascular disease (IRR, 1.00; 0.98-1.02) when adjusted for confounders.
CONCLUSION	In this large cohort study, we found no association between outdoor occupational exposure to UFP and IHD and cerebrovascular disease.

표-743. PubMed 논문번호 31504557의 내용 요약

구분	내용
PubMed ID	31504557
TITLE	Benefits of physical activity not affected by air pollution: a prospective cohort study.
JOURNAL	International journal of epidemiology: 10.1093/ije/dyz184
AUTHORS	Sun Shengzhi, Cao Wangnan, Qiu Hong, Ran Jinjun, Lin Hualiang, Shen Chen, Siu-Yin Lee Ruby, Tian Linwei
BACKGROUND	Physical activity (PA) is beneficial to human health, whereas long-term exposure to air pollution is harmful. However, their combined effects remain unclear. We aimed to estimate the combined (interactive) mortality effects of PA and long-term exposure to fine particulate matter (PM2.5) among older adults in Hong Kong.
METHODS	Participants aged ≥65 years from the Elderly Health Service Cohort (n = 66 820) reported their habitual PA at baseline (1998-2001) and were followed up till 31 December 2011. We used a satellite-based spatiotemporal model to estimate PM2.5 concentration at the residential address for each participant. We used Cox proportional hazards regression to assess the interaction between habitual PA and long-term exposure to PM2.5 on cardiovascular and respiratory mortality. We tested for additive interaction by estimating relative excess risk due to interaction and multiplicative interaction employing P-value for the interaction term.
RESULTS	The death risks were inversely associated with a higher volume of PA and were positively associated with long-term exposure to PM2.5. The benefits of PA were more pronounced for participation in traditional Chinese exercise (e.g. Tai Chi) and aerobic exercise (e.g. cycling). We found little evidence of interaction between PA (volume and type) and long-term exposure to PM2.5 on either additive or multiplicative scales.
CONCLUSIONS	In this cohort of older Chinese adults, PA may decrease the risk of mortality, be it in areas of relatively good or bad air quality. The beneficial mortality effects of habitual PA outweighed the detrimental effects of long-term exposure to air pollution in Hong Kong.

표-744. PubMed 논문번호 31539843의 내용 요약

구분	내용
PubMed ID	31539843
TITLE	Association of residential air pollution, noise, and greenspace with initial ischemic stroke severity.
JOURNAL	Environmental research: 10.1016/j.envres.2019.108725
AUTHORS	Vivanco-Hidalgo Rosa Maria, Avellaneda-G□mez Carla, Dadvand Payam, Cirach Marta, Ois □ngel, G□mez Gonz□lez Alejandra, Rodriguez-Campello Ana, de Ceballos Pablo, Basaga□a Xavier, Zabalza Ana, Cuadrado-Godia Elisa, Sunyer Jordi, Roquer Jaume, Wellenius Gregory A
BACKGROUND AND PURPOSE	A number of environmental risk factors of acute ischemic stroke have been identified, but few studies have evaluated the influence of the outdoor environment on stroke severity. We assessed the association of residential ambient fine particulate matter air pollution (PM2.5), noise, and surrounding greenspace with initial stroke severity.
METHODS	We obtained data on patients hospitalized with acute ischemic stroke from a hospital-based prospective stroke register (2005-2014) in Barcelona. We estimated residential PM2.5 based on an established land use regression model, greenspace as the average satellite-based Normalized Difference Vegetation Index (NDVI) within a 300 m buffer of the residence, and daily (Lday), evening (Levening), night (Lnight) and average noise (Lden) level at the street nearest to the residential address using municipal noise models. Stroke severity was assessed at the time of hospital presentation using the National Institute of Health Stroke Scale (NIHSS).We used logistic regression and binomial models to evaluate the associations of PM2.5, greenspace, and noise with initial stroke severity adjusting for potential confounders.
RESULTS	Among 2761 patients, higher residential surrounding greenspace was associated with lower risk of severe stroke (OR for NIHSS>5, 0.75; 95% CI: 0.60-0.95), while, living in areas with higher Lden was associated with a higher risk of severe stroke (OR, 1.30; 95% CI: 1.02-1.65). PM2.5 was not associated with initial stroke severity.
CONCLUSIONS	In an urban setting, surrounding greenspace and traffic noise at home are associated with initial stroke severity, suggesting an important influence of the built environment on the global burden of ischemic stroke.

표-745. PubMed 논문번호 31546738의 내용 요약

구분	내용
PubMed ID	31546738
TITLE	Associations Between Sub-Clinical Markers of Cardiometabolic Risk and Exposure to Residential Indoor Air Pollutants in Healthy Adults in Perth, Western Australia: A Study Protocol.
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph16193548
AUTHORS	Gilbey Suzanne E, Reid Christopher M, Huxley Rachel R, Soares Mario J, Zhao Yun, Rumchev Krassi
BACKGROUND	A growing body of epidemiological and clinical evidence has implicated air pollution as an emerging risk factor for cardiometabolic disease. Whilst individuals spend up to two-thirds of daily time in their domestic residential environment, very few studies have been designed to objectively measure the sub-clinical markers of cardiometabolic risk with exposure to domestic indoor air pollutants. This cross-sectional study aims to investigate associations between the components of domestic indoor air quality and selected sub-clinical cardiometabolic risk factors in a cohort of healthy adults living in Perth, Western Australia.
METHODS	One hundred and eleven non-smoking adults (65% female) living in non-smoking households who were aged between 35-69 years were recruited for the project. Study subjects were invited to participate in all sections of the study, which included: Domestic indoor air monitoring along with the concurrent 24 h ambulatory monitoring of peripheral and central blood pressure and measures of central hemodynamic indices, standardized questionnaires on aspects relating to current health status and the domestic environment, a 24 h time-activity diary during the monitoring period, and clinic-based health assessment involving collection of blood and urine biomarkers for lipid and glucose profiles, as well as measures of renal function and an analysis of central pulse wave and pulse wave velocity.
RESULTS	This study provides a standardized approach to the study of sub-clinical cardiometabolic health effects that are related to the exposure to indoor air pollution.
CONCLUSION	The findings of this study may provide direction for future research that will further contribute to our understanding of the relationship that exists between indoor air pollution and sub-clinical markers of cardiometabolic risk.

표-746. PubMed 논문번호 31558248의 내용 요약

구분	내용
PubMed ID	31558248
TITLE	Childhood Tobacco Smoke Exposure and Risk of Atrial Fibrillation in Adulthood.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2019.07.060
AUTHORS	Groh Christopher A, Vittinghoff Eric, Benjamin Emelia J, Dupuis Jos□e, Marcus Gregory M
BACKGROUND	Cigarette smoking is known to increase the risk of atrial fibrillation (AF), and a recent cross-sectional analysis suggested that parental smoking may be an AF risk factor.
OBJECTIVES	The purpose of this study was to assess if parental smoking predicts offspring AF in the Framingham Heart Study.
METHODS	This study analyzed Framingham Offspring cohort participants with parents in the Original cohort with known smoking status during the offspring's childhood. Framingham participants were evaluated every 2 to 8 years and were under routine surveillance for incident AF. The authors assessed AF incidence among Offspring participants exposed to parental smoking through age 18 years and performed a mediation analysis to determine the extent to which offspring smoking might explain observed associations.
RESULTS	Of 2,816 Offspring cohort participants with at least 1 parent in the Original cohort, 82% were exposed to parental smoking. For every pack/day increase in parental smoking, there was an 18% increase in offspring AF incidence (adjusted hazard ratio [HR]: 1.18; 95% confidence interval [CI]: 1.00 to 1.39; p = 0.04). Additionally, parental smoking was a risk factor for offspring smoking (adjusted odds ratio [OR]: 1.34; 95% CI: 1.17 to 1.54; p < 0.001). Offspring smoking mediated 17% (95% CI: 1.5% to 103.3%) of the relationship between parental smoking and offspring AF.
CONCLUSIONS	Childhood secondhand smoke exposure predicted increased risk for adulthood AF after adjustment for AF risk factors. Some of this relationship may be mediated by a greater propensity among offspring of smoking parents to smoke themselves. These findings highlight potential new pathways for AF risk that begin during childhood, offering new evidence to motivate smoking avoidance and cessation.

표-747. PubMed 논문번호 31561037의 내용 요약

구분	내용
PubMed ID	31561037
TITLE	Long-term exposure to greenspace and metabolic syndrome: A Whitehall II study.
JOURNAL	Environmental pollution (Barking, Essex : 1987): 10.1016/j.envpol.2019.113231
AUTHORS	de Keijzer Carmen, Basaga□a Xavier, Tonne Cathryn, Valent□n Ant□nia, Alonso Jordi, Ant□ Josep M, Nieuwenhuijsen Mark J, Kivim□ki Mika, Singh-Manoux Archana, Sunyer Jordi, Dadvand Payam
BACKGROUND	Metabolic syndrome is an important risk factor for non-communicable diseases, particularly type 2 diabetes, coronary heart disease, and stroke. Long-term exposure to greenspace could be protective of metabolic syndrome, but evidence for such an association is lacking. Accordingly, we investigated the association between long-term exposure to greenspace and risk of metabolic syndrome.
METHODS	The present longitudinal study was based on data from four clinical examinations between 1997 and 2013 in 6076 participants of the Whitehall II study, UK (aged 45-69 years at baseline). Long-term exposure to greenspace was assessed by satellite-based indices of greenspace including Normalized Difference Vegetation Index (NDVI) and Vegetation Continuous Field (VCF) averaged across buffers of 500 and 1000 m surrounding the participants' residential location at each follow-up. The ascertainment of metabolic syndrome was based on the World Health Organization (WHO) definition. Hazard ratios for metabolic syndrome were estimated using Cox proportional hazards regression models, controlling for age, sex, ethnicity, lifestyle factors, and socioeconomic status.
RESULTS	Higher residential surrounding greenspace was associated with lower risk of metabolic syndrome. An interquartile range increase in NDVI and VCF in the 500 m buffer was associated with 13% (95% confidence interval (CI): 1%, 23%) and 14% (95% CI: 5%, 22%) lower risk of metabolic syndrome, respectively. Greater exposure to greenspace was also associated with each individual component of metabolic syndrome, including a lower risk of high levels of fasting glucose, large waist circumference, high triglyceride levels, low HDL cholesterol, and hypertension. The association between residential surrounding greenspace and metabolic syndrome may have been mediated by physical activity and exposure to air pollution.
CONCLUSIONS	The findings of the present study suggest that middle-aged and older adults living in greener neighbourhoods are at lower risk of metabolic syndrome than those living in neighbourhoods with less greenspace.

표-748. PubMed 논문번호 31591299의 내용 요약

구분	내용
PubMed ID	31591299
TITLE	Short-Term Effects of Ambient Air Pollution on ST-Elevation Myocardial Infarction Events: Are There Potentially Susceptible Groups?
JOURNAL	International journal of environmental research and public health: 10.3390/ijerph16193760
AUTHORS	Pan Hsiu-Yung, Cheung Shun-Man, Chen Fu-Cheng, Wu Kuan-Han, Cheng Shih-Yu, Chuang Po-Chun, Cheng Fu-Jen
BACKGROUND	Air pollution exposure is associated with greater risk for cardiovascular events. This study aims to examine the effects of increased exposure to short-term air pollutants on ST-segment elevation myocardial infarction (STEMI) and determine the susceptible groups.
METHODS	Data on particulate matter PM2.5 and PM10 and other air pollutants, measured at each of the 11 air-quality monitoring stations in Kaohsiung City, were collected between 2011 and 2016. The medical records of non-trauma adult (>17 years) patients who had visited the emergency department (ED) with a typical electrocardiogram change of STEMI were extracted. A time-stratified and case-crossover study design was used to examine the relationship between air pollutants and daily ED visits for STEMI.
RESULTS	An interquartile range increment in PM2.5 on lag 0 was associated with an increment of 25.5% (95% confidence interval, 2.6%-53.4%) in the risk of STEMI ED visits. Men and persons with ≥3 risk factors (male sex, age, hypertension, diabetes, current smoker, dyslipidemia, history of myocardial infarction, and high body mass index) for myocardial infarction (MI) were more sensitive to the hazardous effects of PM2.5 (interaction: $p = 0.039$ and $p = 0.018$, respectively). The associations between PM10, NO2, and O3 and STEMI did not achieve statistical significance.
CONCLUSION	PM2.5 may play an important role in STEMI events on the day of exposure in Kaohsiung. Men and persons with ≥3 risk factors of MI are more susceptible to the adverse effects of PM2.5 on STEMI.

표-749. PubMed 논문번호 31601202의 내용 요약

구분	내용
PubMed ID	31601202
TITLE	Low concentrations of fine particle air pollution and mortality in the Canadian Community Health Survey cohort.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-019-0518-y
AUTHORS	Christidis Tanya, Erickson Anders C, Pappin Amanda J, Crouse Daniel L, Pinault Lauren L, Weichenthal Scott A, Brook Jeffrey R, van Donkelaar Aaron, Hystad Perry, Martin Randall V, Tjepkema Michael, Burnett Richard T, Brauer Michael
BACKGROUND	Approximately 2.9 million deaths are attributed to ambient fine particle air pollution around the world each year (PM2.5). In general, cohort studies of mortality and outdoor PM2.5 concentrations have limited information on individuals exposed to low levels of PM2.5 as well as covariates such as smoking behaviours, alcohol consumption, and diet which may confound relationships with mortality. This study provides an updated and extended analysis of the Canadian Community Health Survey-Mortality cohort: a population-based cohort with detailed PM2.5 exposure data and information on a number of important individual-level behavioural risk factors. We also used this rich dataset to provide insight into the shape of the concentration-response curve for mortality at low levels of PM2.5.
METHODS	Respondents to the Canadian Community Health Survey from 2000 to 2012 were linked by postal code history from 1981 to 2016 to high resolution PM2.5 exposure estimates, and mortality incidence to 2016. Cox proportional hazard models were used to estimate the relationship between non-accidental mortality and ambient PM2.5 concentrations (measured as a three-year average with a one-year lag) adjusted for socio-economic, behavioural, and time-varying contextual covariates.
RESULTS	In total, 50,700 deaths from non-accidental causes occurred in the cohort over the follow-up period. Annual average ambient PM2.5 concentrations were low (i.e. 5.9 μg/m3, s.d. 2.0) and each 10 μg/m3 increase in exposure was associated with an increase in non-accidental mortality (HR = 1.11; 95% CI 1.04-1.18). Adjustment for behavioural covariates did not materially change this relationship. We estimated a supra-linear concentration-response curve extending to concentrations below 2 μg/m3 using a shape constrained health impact function. Mortality risks associated with exposure to PM2.5 were increased for males, those under age 65, and non-immigrants. Hazard ratios for PM2.5 and mortality were attenuated when gaseous pollutants were included in models.
CONCLUSIONS	Outdoor PM2.5 concentrations were associated with non-accidental mortality and adjusting for individual-level behavioural covariates did not materially change this relationship. The concentration-response curve was supra-linear with increased mortality risks extending to low outdoor PM2.5 concentrations.

표-750. PubMed 논문번호 31605119의 내용 요약

구분	내용
PubMed ID	31605119
TITLE	Association between ambient and household air pollution with carotid intima-media thickness in peri-urban South India: CHAI-Project.
JOURNAL	International journal of epidemiology: 10.1093/ije/dyz208
AUTHORS	Ranzani Otavio T, Mil□ Carles, Sanchez Margaux, Bhogadi Santhi, Kulkarni Bharati, Balakrishnan Kalpana, Sambandam Sankar, Sunyer Jordi, Marshall Julian D, Kinra Sanjay, Tonne Cathryn
BACKGROUND	Evidence linking ambient air pollution with atherosclerosis is lacking from low- and middle-income countries. Additionally, evidence regarding the association between household air pollution and atherosclerosis is limited. We evaluated the association between ambient fine particulate matter [particulate matter with an aerodynamic diameter of ≤2.5 □m (PM2.5)] and biomass fuel use on carotid intima-media thickness (CIMT), a surrogate of atherosclerosis, in India.
METHODS	We analysed the third follow-up of the Andhra Pradesh Children and Parent Study cohort (2010-2012), which recruited participants from 28 peri-urban villages. Our primary outcome was mean CIMT, measured using a standardized protocol. We estimated annual average PM2.5 outdoors at residence using land-use regression. Biomass cooking fuel was self-reported. We fitted a within-between linear-mixed model adjusting for potential confounders.
RESULTS	Among 3278 participants (48% women, mean age 38 years), mean PM2.5 was 32.7 [range 24.4-38.2] □g/m3, and 60% used biomass. After confounder adjustment, we observed positive associations between within-village variation in PM2.5 and CIMT in all participants [1.79%, 95% confidence interval (CI), -0.31 to 3.90 per 1 □g/m3 of PM2.5] and in men (2.98%, 95% CI, 0.23-5.72, per 1 □g/m3 of PM2.5). Use of biomass cooking fuel was associated with CIMT in all participants (1.60%, 95% CI, -0.46 to 3.65), especially in women with an unvented stove (6.14%, 95% CI, 1.40-10.89). The point-estimate for the PM2.5 association was larger in sub-groups with higher cardiometabolic risk profile.
CONCLUSIONS	Ambient and household air pollution were positively associated with CIMT in a peri-urban population of India, although with limited precision for some estimates. We observed differences in the association between ambient and household air pollution and CIMT by gender.

표-751. PubMed 논문번호 31630066의 내용 요약

구분	내용
PubMed ID	31630066
TITLE	Association between dust storm occurrence and risk of suicide: Case-crossover analysis of the Korean national death database.
JOURNAL	Environment international: 10.1016/j.envint.2019.105146
AUTHORS	Lee Hyewon, Jung Jiyun, Myung Woojae, Baek Ji Hyun, Kang Jae Myeong, Kim Doh Kwan, Kim Ho
BACKGROUND	Asian dust storms (ADSs) have been associated with adverse health outcomes, including respiratory and cardiovascular diseases. Considering the increasing global desertification driven by climate change, it is necessary to assess dust storm-related adverse health effects for establishing appropriate public health interventions. Recent studies have found that ambient air pollution has negative effects on mental health including cognitive disorders, depression, and suicide. However, these studies mostly focused on traditional anthropogenic pollutants from traffic exhaust or fossil fuel power plants; the association between dust storms and suicidal death is yet to be determined.
OBJECTIVE	To assess the association between ADSs and suicide risk in Seoul, South Korea from 2002 to 2015.
METHODS	To determine whether increased risk of suicide is associated with occurrence of ADSs, we performed a time-stratified case-crossover study that linked the national death statistics database with ADS occurrence data from the Korea Meteorology Administration. Exposure to ADSs was compared between the day of suicide and control days, matched to the day of the week, month, and year. We further examined whether the effect of ADSs on suicide risk differed according to ADS duration and intensity.
RESULTS	Over the 14-year period, 30,704 people died by suicide and 133 ADSs were reported. Of these, 55 ADSs lasted over 2 days (long-duration ADSs), and 67 ADSs had higher levels of particulate matter < 10 μm in diameter (PM10) that exceeded the 50th percentile value over the total 133 ADS days (high-intensity ADSs). Exposure to ADS was associated with a 13.1% (95% confidence interval [CI], 4.5-22.4; P = .002) increase in suicide risk on the day of ADS occurrence. Long-duration and high-intensity ADSs were associated with a 19.8% (95% CI, 6.5-34.7; P = .003) and 17.0% (95% CI, 5.2-30.0; P = .004) increase in suicide risk, respectively. These associations remained robust after adjusting for local air pollution levels and meteorological factors. However, this association was not replicated in the unconstrained distributed lag model which revealed inferior goodness-of-fit to our data.
CONCLUSIONS	Exposure to ADSs was associated with an increased risk of suicide, especially on the same day. This study provides novel evidence of a relationship between ADSs and suicide. These findings could help in establishing public health interventions for suicide prevention as well as in establishing dust storm warning systems. Future studies are warranted to confirm if our findings are replicable and to elucidate the underlying mechanisms.

표-752. PubMed 논문번호 31645051의 내용 요약

구분	내용
PubMed ID	31645051
TITLE	Relation of secondhand smoke exposure to vascular phenotypes in children and adolescents.
JOURNAL	Pediatric research: 10.1038/s41390-019-0627-x
AUTHORS	Harbin Michelle M, Kelly Aaron S, Dengel Donald R, Rudser Kyle D, Evanoff Nicholas G, Ryder Justin R, Harbin Michelle M, Kelly Aaron S, Dengel Donald R, Rudser Kyle D, Evanoff Nicholas G, Ryder Justin R
BACKGROUND	Subclinical cardiovascular risks of secondhand smoke (SHS) exposure among children and adolescents remains insufficiently described.
METHODS	This was a cross-sectional study of 298 children and adolescents (48.0% male, body mass index: 27.0 ± 8.9 kg/m2), including 49 self-reported cases with SHS. Arterial elasticity and stiffness (distensibility, compliance, incremental elastic modulus [IEM]) were obtained via ultrasound imaging in the abdominal aorta, brachial, and carotid arteries. A one-way analysis of variance compared differences between groups, and multiple linear regression adjusted for covariates.
RESULTS	SHS was associated with lower abdominal aorta diameter distensibility (aDD) (13.4 ± 3.6% vs. 16.0 ± 5.2%, p = 0.009) and abdominal aorta cross-sectional distensibility (aCSD) (28.8 ± 8.3% vs. 35.1 ± 12.2%, p = 0.009), as well as higher abdominal aorta IEM (aIEM) (1241 ± 794 vs. 935 ± 388 mmHg, p = 0.001). After adjustment for covariates, aDD (p = 0.047), aCSD (p = 0.040), and aIEM (p = 0.017) remained significant; this significance persisted with the additional adjustment of percent body fat. Measures of brachial and carotid compliance and distensibility were not associated with SHS.
CONCLUSIONS	SHS was associated with abdominal aorta stiffness; the majority of vascular measures within the brachial and carotid artery remained unaffected following adjustment for covariates, including hypertension and adiposity. SHS may predispose individuals to increased abdominal aorta stiffness, an artery previously reported to exhibit increased susceptibility to atherosclerosis.

표-753. PubMed 논문번호 31663780의 내용 요약

구분	내용
PubMed ID	31663780
TITLE	Prenatal Exposure to <mml:math xmlns:mml="http://www.w3.org/1998/Math/MathML"><mml:mrow><mml:mi>P</mml:mi><m and Cardiac Vagal Tone during Infancy: Findings from a Multiethnic Birth Cohort.
JOURNAL	Environmental health perspectives: 10.1289/EHP4434
AUTHORS	Cowell Whitney J, Brunst Kelly J, Malin Ashley J, Coull Brent A, Gennings Chris, Kloog Itai, Lipton Lianna, Wright Robert O, Enlow Michelle Bosquet, Wright Rosalind J
BACKGROUND	The autonomic nervous system plays a key role in maintaining homeostasis and responding to external stimuli. In adults, exposure to fine particulate matter (PM2.5) has been associated with reduced heart rate variability (HRV), an indicator of cardiac autonomic control.
OBJECTIVES	Our goal was to investigate the associations of exposure to fine particulate matter (PM2.5) with HRV as an indicator of cardiac autonomic control during early development.
METHODS	We studied 237 maternal-infant pairs in a Boston-based birth cohort. We estimated daily residential PM2.5 using satellite data in combination with land-use regression predictors. In infants at 6 months of age, we measured parasympathetic nervous system (PNS) activity using continuous electrocardiogram monitoring during the Repeated Still-Face Paradigm, an experimental protocol designed to elicit autonomic reactivity in response to maternal interaction and disengagement. We used multivariable linear regression to examine average PM2.5 exposure across pregnancy in relation to PNS withdrawal and activation, indexed by changes in respiration-corrected respiratory sinus arrhythmia (RSAc)-an established metric of HRV that reflects cardiac vagal tone. We examined interactions with infant sex using cross-product terms.
RESULTS	In adjusted models we found that a 1-unit increase in PM2.5 (in micrograms per cubic meter) was associated with a 3.53% decrease in baseline RSAc (95% CI: -6.96, 0.02). In models examining RSAc change between episodes, higher PM2.5 was generally associated with reduced PNS withdrawal during stress and reduced PNS activation during recovery; however, these associations were not statistically significant. We did not observe a significant interaction between PM2.5 and sex.
DISCUSSION	Prenatal exposure to PM2.5 may disrupt cardiac vagal tone during infancy. Future research is needed to replicate these preliminary findings. https://doi.org/10.1289/EHP4434.

표-754. PubMed 논문번호 31663781의 내용 요약

구분	내용
PubMed ID	31663781
TITLE	Long-Term Exposure to Particulate Air Pollution, Black Carbon, and Their Source Components in Relation to Ischemic Heart Disease and Stroke.
JOURNAL	Environmental health perspectives: 10.1289/EHP4757
AUTHORS	Ljungman Petter L S, Andersson Niklas, Stockfelt Leo, Andersson Eva M, Nilsson Sommar Johan, Eneroth Kristina, Gidhagen Lars, Johansson Christer, Lager Anton, Leander Karin, Molnar Peter, Pedersen Nancy L, Rizzuto Debora, Rosengren Annika, Segersson David, Wennberg Patrik, Barregard Lars, Forsberg Bertil, Sallsten Gerd, Bellander Tom, Pershagen G□ran
BACKGROUND	Long-term exposure to particulate matter (PM) in ambient air has been associated with cardiovascular mortality, but few studies have considered incident disease in relation to PM from different sources.
OBJECTIVES	We aimed to study associations between long-term exposure to different types of PM and sources, and incident ischemic heart disease (IHD) and stroke in three Swedish cities.
METHODS	Based on detailed emission databases, monitoring data, and high-resolution dispersion models, we calculated source contributions to PM with aerodynamic diameter ≤10 μm (PM10), PM with aerodynamic diameter ≤2.5 μm (PM2.5), and black carbon (BC) from road wear, traffic exhaust, residential heating, and other sources in Gothenburg, Stockholm, and Ume□. Registry data for participants from four cohorts were used to obtain incidence of IHD and stroke for first hospitalization or death. We constructed time windows of exposure for same-year, 1- to 5-y, and 6- to 10-y averages preceding incidence from annual averages at residential addresses. Risk estimates were based on random effects meta-analyses of cohort-specific Cox proportional hazard models.
RESULTS	We observed 5,166 and 3,119 incident IHD and stroke cases, respectively, in 114,758 participants. Overall, few consistent associations were observed between the different air pollution measures and IHD or stroke incidence. However, same-year levels of ambient locally emitted BC (range: 0.01-4.6 μg/m3) were associated with a 4.0% higher risk of incident stroke per interquartile range (IQR), 0.30 μg/m3 [95% confidence interval (CI): 0.04, 7.8]. This association was primarily related to BC from traffic exhaust. PM10 (range: 4.4-52 μg/m3) and PM2.5 (range: 2.9-22 μg/m3) were not associated with stroke. Associations with incident IHD were observed only for PM2.5 exposure from residential heating.
DISCUSSION	Few consistent associations were observed between different particulate components and IHD or stroke. However, long-term residential exposure to locally emitted BC from traffic exhaust was associated with stroke incidence. The comparatively low exposure levels may have contributed to the paucity of associations. https://doi.org/10.1289/EHP4757.

표-755. PubMed 논문번호 31677868의 내용 요약

구분	내용
PubMed ID	31677868
TITLE	Long-term exposure to PM〈sub〉2.5〈/sub〉 and fasting plasma glucose in non-diabetic adolescents in Yogyakarta, Indonesia.
JOURNAL	Environmental pollution (Barking, Essex : 1987): 10.1016/j.envpol.2019.113423
AUTHORS	Yu Wenhua, Sulistyoningrum Dian Caturini, Gasevic Danijela, Xu Rongbin, Julia Madarina, Murni Indah Kartika, Chen Zhuying, Lu Peng, Guo Yuming, Li Shanshan, Yu Wenhua, Sulistyoningrum Dian Caturini, Gasevic Danijela, Xu Rongbin, Julia Madarina, Murni Indah Kartika, Chen Zhuying, Lu Peng, Guo Yuming, Li Shanshan
BACKGROUND	Indonesia is facing serious air pollution. However, very few studies have been conducted to examine the health risks of air pollution in Indonesia, particularly for adolescents.
OBJECTIVE	To assess the association between long-term exposure to ambient particles with a diameter of 〈2.5 μm (PM2.5) and fasting plasma glucose (FPG) in adolescents.
METHODS	A cross-sectional study was conducted in 482 adolescents aged 14-18 years in Yogyakarta, Indonesia in 2016. We finally included 469 (97.30%) participants who had no missing data for data analysis. We collected individual data on socio-demographics, behavioral habits, and health information through standardized questionnaires. Satellite-based PM2.5 concentrations from 2013 to 2016 were assigned based on participants' residential addresses. The association between PM2.5 and FPG was examined using a generalized linear regression model while FPG was modeled as a continuous variable. An ordered logistic regression model was used to assess the relationship between PM2.5 and FPG categories.
RESULTS	Every 1 $\mu g/m^3$ increase in PM2.5 was associated with a 0.34 mg/dL [95 confidence interval (95% CI): 0.08 mg/dL, 0.59 mg/dL] increase in FPG levels. Comparing with the low FPG level (under 86 mg/dL), every 1 $\mu g/m^3$ increase in PM2.5 was associated with a 10.20% (95% CI: 1.60%, 19.80%) increase in the odds of impaired fasting glucose (IFG) (100-125 mg/dL). Stratified analyses indicated greater effects on participants with hypertension [odds ratio (OR) = 1.30, 95% CI: 1.09, 1.57] and those had higher physical activities (OR = 1.36, 95% CI: 1.09, 1.57). Adolescents' sex, obesity status and different cutoff points of FPG did not modify the association between the exposure to PM2.5 and FPG levels.
CONCLUSION	Long-term exposure to PM2.5 was associated with increased FPG levels in Indonesian non-diabetic adolescents.

표-756. PubMed 논문번호 31689002의 내용 요약

구분	내용
PubMed ID	31689002
TITLE	Effect of PM2.5 on macrosomia in China: A nationwide prospective cohort study.
JOURNAL	Pediatric obesity: 10.1111/ijpo.12584
AUTHORS	Chen Shi, Wang Shirui, Li Tiantian, Zhu Huijuan, Liang Siyu, Xu Ke, Zhang Yuelun, Yuan Xianxian, Yang Yingying, Pan Hui, Shi Xiaoming, Chen Shi, Wang Shirui, Li Tiantian, Zhu Huijuan, Liang Siyu, Xu Ke, Zhang Yuelun, Yuan Xianxian, Yang Yingying, Pan Hui, Shi Xiaoming
BACKGROUND	Macrosomia is associated with both neonatal complications and adult diseases (obesity, diabetes mellitus, etc.). Previous studies have reported maternal exposure to PM2.5 might influence metabolism and fetal development and cause adverse pregnancy outcomes. Studies conducted in areas with low PM2.5 concentration have found relationship between gestational PM2.5 exposure and birth weight. However, the impact of air pollution on macrosomia has not been studied, especially in highly polluted areas.
OBJECTIVE	To evaluate the association between fine particulate matter (PM2.5) exposure during pregnancy and the risk of macrosomia.
METHODS	Data from preconception health examination and prenatal and postnatal records were collected from 1 January 2010 to 31 December 2012 in the National Free Preconception Health Examination Project. Monthly mean of PM2.5 concentration during pregnancy was estimated from satellite data using an ensemble machine learning model. A newborn with birth weight above 4000 g was defined as macrosomia. Logistic regression models were used to examine the association between maternal exposure to PM2.5 and the risk of macrosomia, after adjusting for maternal age, pre-pregnancy body mass index, parity, neonatal sex, duration of gestation, seasonality, educational level, smoking and drinking habits, past history of diabetes mellitus and hypertension, and family history of diabetes mellitus. Restricted cubic spline models were used to evaluate the dose-response relationship between the risk of macrosomia and PM2.5 concentration.
RESULTS	Of 177 841 singleton nonlow birth weight newborns included, 14 598 (8.2%) had macrosomia. The mean PM2.5 concentrations were 70.7, 71.5, and 80.9 μg/m3 in the first, second, and third trimesters. In full-adjusted logistic regression models, significant associations were found between increased risk of macrosomia and every 10 μg/m3 increase of PM2.5 concentration over the first (odds ratio [OR]: 1.045; 95% CI, 1.037-1.052), second (OR: 1.035; 95% CI, 1.028-1.043), and third (OR: 1.033; 95% CI, 1.026-1.039) trimesters. There was a nonlinear does-response association between PM2.5 concentration and the risk of macrosomia.
CONCLUSIONS	Maternal exposure to PM2.5 during pregnancy was associated with an increased risk of macrosomia in China.

표-757. PubMed 논문번호 31693516의 내용 요약

구분	내용
PubMed ID	31693516
TITLE	Evaluating the Sensitivity of PM2.5-Mortality Associations to the Spatial and Temporal Scale of Exposure Assessment.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000001136
AUTHORS	Crouse Dan L, Erickson Anders C, Christidis Tanya, Pinault Lauren, van Donkelaar Aaron, Li Chi, Meng Jun, Martin Randall V, Tjepkema Michael, Hystad Perry, Burnett Rick, Pappin Amanda, Brauer Michael, Weichenthal Scott
BACKGROUND	The temporal and spatial scales of exposure assessment may influence observed associations between fine particulate air pollution (PM2.5) and mortality, but few studies have systematically examined this question.
METHODS	We followed 2.4 million adults in the 2001 Canadian Census Health and Environment Cohort for nonaccidental and cause-specific mortality between 2001 and 2011. We assigned PM2.5 exposures to residential locations using satellite-based estimates and compared three different temporal moving averages (1, 3, and 8 years) and three spatial scales (1, 5, and 10 km) of exposure assignment. In addition, we examined different spatial scales based on age, employment status, and urban/rural location, and adjustment for O3, NO2, or their combined oxidant capacity (Ox).
RESULTS	In general, longer moving averages resulted in stronger associations between PM2.5 and mortality. For nonaccidental mortality, we observed a hazard ratio of 1.11 (95% CI = 1.08, 1.13) for the 1-year moving average compared with 1.23 (95% CI = 1.20, 1.27) for the 8-year moving average. Respiratory and lung cancer mortality were most sensitive to the spatial scale of exposure assessment with stronger associations observed at smaller spatial scales. Adjustment for oxidant gases attenuated associations between PM2.5 and cardiovascular mortality and strengthened associations with lung cancer. Despite these variations, PM2.5 was associated with increased mortality in nearly all of the models examined.
CONCLUSIONS	These findings support a relationship between outdoor PM2.5 and mortality at low concentrations and highlight the importance of longer-exposure windows, more spatially resolved exposure metrics, and adjustment for oxidant gases in characterizing this relationship.

표-758. PubMed 논문번호 31731170의 내용 요약

구분	내용
PubMed ID	31731170
TITLE	The correlation between PM〈sub〉2.5〈/sub〉 exposure and hypertensive disorders in pregnancy: A Meta-analysis.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2019.134985
AUTHORS	Sun Mengqi, Yan Wu, Fang Kacey, Chen Danrong, Liu Jiani, Chen Yi, Duan Junchao, Chen Rui, Sun Zhiwei, Wang Xu, Xia Yankai, Sun Mengqi, Yan Wu, Fang Kacey, Chen Danrong, Liu Jiani, Chen Yi, Duan Junchao, Chen Rui, Sun Zhiwei, Wang Xu, Xia Yankai
OBJECTIVE	To find the correlation between exposure to PM2.5 (fine particulate matter) and hypertensive disorders in pregnancy (HDP), and provide medical evidence for decreasing the incidence of hypertensive disorders in pregnancy.
METHOD	A combination of computer and manual retrieval was used to search for keywords in PubMed (385 records), Cochrane Library (20 records), Web of Science (419 records) and Embase (325 records). Finally, ten epidemiological articles were considered in this meta-analysis. Stata 13.0 was used to examine the heterogeneity among the studies and to calculate the combined effect value (OR, odds ratio) by selecting the corresponding models. Sensitivity analysis and publication bias test were also performed.
RESULTS	Meta-analysis indicated that there was an association between PM2.5 exposure (per 10 □g/m3 increase) and hypertensive disorders in pregnancy (OR = 1.52, 95% CI: 1.24-1.87). Exposure to PM2.5 (per 10 □g/m3 increase) enhanced the risk of pre-eclampsia (OR = 1.31, 95% CI: 1.07-1.61), but there was no evidence relating exposure to PM2.5 to gestational hypertension (OR = 1.35, 95% CI: 0.98-1.87).
CONCLUSION	There is a significant link between exposure to PM2.5 and hypertensive disorders in pregnancy. The first and the third trimester were more susceptible to PM2.5 exposure. It is recommended to further strengthen protective measures against PM2.5 during pregnancy.

표-759. PubMed 논문번호 31733955의 내용 요약

구분	내용
PubMed ID	31733955
TITLE	Effects of short-term exposure to particulate matters on heart rate variability: A systematic review and meta-analysis based on controlled animal studies.
JOURNAL	Environmental pollution (Barking, Essex : 1987): 10.1016/j.envpol.2019.113306
AUTHORS	Huang Fangfang, Wang Ping, Pan Xinjuan, Wang Yingfang, Ren Shuai, Huang Fangfang, Wang Ping, Pan Xinjuan, Wang Yingfang, Ren Shuai
BACKGROUND	Exposure to particulate matters (PM) is recognized as an important risk factor for cardiovascular disease. A change in cardiac autonomic function is one postulated mechanism leading to PM related cardiovascular events. This study therefore evaluated the associations of short-term exposure to PM and heart rate variability (HRV) parameters, which can reflect the cardiac autonomic function.
METHODS	Four electronic databases were searched for controlled studies of rodents published prior to December 2018. A systematic review and meta-analysis was conducted. Effect sizes were calculated for five main HRV parameters, including standard deviation of normal-to-normal intervals (SDNN), square root of mean squared differences between successive normal-to-normal intervals (rMSSD), low frequency (LF), high frequency (HF), and the ratio of LF and HF (LF/HF).
RESULTS	The review included 23 studies with 401 animals. Short-term exposure to PM by instillation yielded statistically significant effects on SDNN (Standardized Mean Difference [SMD] = -1.11, 95% Confidence Intervals [CI] = -2.22 to -0.01, P = 0.05), LF (SMD = -1.19, 95% CI = -1.99 to -0.40, P = 0.003) and LF/HF (SMD = -1.05, 95% CI = -2.03 to -0.07, P = 0.04). Short-term exposure to PM by inhalation only yielded statistically significant effect on LF/HF (SMD = -0.83, 95% CI = -1.39 to -0.27, P = 0.004). There was no evidence that animal model and exposure frequency influenced the relationship of PM and HRV.
CONCLUSIONS	Short-term exposure to PM can decrease HRV of rodents, affecting cardiac autonomic function. Exposure methods can influence the relationships of PM and HRV parameters. Further studies should focus on the effects of long-term PM exposure, on human beings, and potential influential factors of PM-HRV associations.

표-760. PubMed 논문번호 31747037의 내용 요약

구분	내용
PubMed ID	31747037
TITLE	Burden of Cause-Specific Mortality Associated With PM2.5 Air Pollution in the United States.
JOURNAL	JAMA network open: 10.1001/jamanetworkopen.2019.15834
AUTHORS	Bowe Benjamin, Xie Yan, Yan Yan, Al-Aly Ziyad
Importance	Ambient fine particulate matter (PM2.5) air pollution is associated with increased risk of several causes of death. However, epidemiologic evidence suggests that current knowledge does not comprehensively capture all causes of death associated with PM2.5 exposure.
Objective	To systematically identify causes of death associated with PM2.5 pollution and estimate the burden of death for each cause in the United States.
Design, Setting, and Participants	In a cohort study of US veterans followed up between 2006 and 2016, ensemble modeling was used to identify and characterize morphology of the association between PM2.5 and causes of death. Burden of death associated with PM2.5 exposure in the contiguous United States and for each state was then estimated by application of estimated risk functions to county-level PM2.5 estimates from the US Environmental Protection Agency and cause-specific death rate data from the Centers for Disease Control and Prevention.
Main Outcomes and Measures	Nonlinear exposure-response functions of the association between PM2.5 and causes of death and burden of death associated with PM2.5.
Exposures	Annual mean PM2.5 levels.
Results	A cohort of 4 522 160 US veterans (4 243 462 [93.8%] male; median [interquartile range] age, 64.1 [55.7-75.5] years; 3 702 942 [82.0%] white, 667 550 [14.8%] black, and 145 593 [3.2%] other race) was followed up for a median (interquartile range) of 10.0 (6.8-10.2) years. In the contiguous United States, PM2.5 exposure was associated with excess burden of death due to cardiovascular disease (56 070.1 deaths [95% uncertainty interval {UI}, 51 940.2-60 318.3 deaths]), cerebrovascular disease (40 466.1 deaths [95% UI, 21 770.1-46 487.9 deaths]), chronic kidney disease (7175.2 deaths [95% UI, 5910.2-8371.9 deaths]), chronic obstructive pulmonary disease (645.7 deaths [95% UI, 300.2-2490.9 deaths]), dementia (19 851.5 deaths [95% UI, 14 420.6-31 621.4 deaths]), type 2 diabetes (501.3 deaths [95% UI, 447.5-561.1 deaths]), hypertension (30 696.9 deaths [95% UI, 27 518.1-33 881.9 deaths]), lung cancer (17 545.3 deaths [95% UI, 15 055.3-20 464.5 deaths]), and pneumonia (8854.9 deaths [95% UI, 7696.2-10 710.6 deaths]). Burden exhibited substantial geographic variation. Estimated burden of death due to nonaccidental causes was 197 905.1 deaths (95% UI, 183 463.3-213 644.9 deaths); mean age-standardized death rates (per 100 000) due to nonaccidental causes were higher among black individuals (55.2 [95% UI, 50.5-60.6]) than nonblack individuals (51.0 [95% UI, 46.4-56.1]) and higher among those living in counties with high (65.3 [95% UI, 56.2-75.4]) vs low (46.1 [95% UI, 42.3-50.4]) socioeconomic deprivation; 99.0% of the burden of death due to nonaccidental causes was associated with PM2.5 levels below standards set by the US Environmental Protection Agency.
Conclusions and Relevance	In this study, 9 causes of death were associated with PM2.5 exposure. The burden of death associated with PM2.5 was disproportionally borne by black

표-761. PubMed 논문번호 31752939의 내용 요약

구분	내용
PubMed ID	31752939
TITLE	Air pollution and mortality in a large, representative U.S. cohort: multiple-pollutant analyses, and spatial and temporal decompositions.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-019-0544-9
AUTHORS	Lefler Jacob S, Higbee Joshua D, Burnett Richard T, Ezzati Majid, Coleman Nathan C, Mann Dalton D, Marshall Julian D, Bechle Matthew, Wang Yuzhou, Robinson Allen L, Arden Pope C
BACKGROUND	Cohort studies have documented associations between fine particulate matter air pollution (PM2.5) and mortality risk. However, there remains uncertainty regarding the contribution of co-pollutants and the stability of pollution-mortality associations in models that include multiple air pollutants. Furthermore, it is unclear whether the PM2.5-mortality relationship varies spatially, when exposures are decomposed according to scale of spatial variability, or temporally, when effect estimates are allowed to change between years.
METHODS	A cohort of 635,539 individuals was compiled using public National Health Interview Survey (NHIS) data from 1987 to 2014 and linked with mortality follow-up through 2015. Modelled air pollution exposure estimates for PM2.5, other criteria air pollutants, and spatial decompositions (< 1 km, 1-10 km, 10-100 km, > 100 km) of PM2.5 were assigned at the census-tract level. The NHIS samples were also divided into yearly cohorts for temporally-decomposed analyses. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) in regression models that included up to six criteria pollutants; four spatial decompositions of PM2.5; and two- and five-year lagged mean PM2.5 exposures in the temporally-decomposed cohorts. Meta-analytic fixed-effect estimates were calculated using results from temporally-decomposed analyses and compared with time-independent results using 17- and 28-year exposure windows.
RESULTS	In multiple-pollutant analyses, PM2.5 demonstrated the most robust pollutant-mortality association. Coarse fraction particulate matter (PM2.5-10) and sulfur dioxide (SO2) were also associated with excess mortality risk. The PM2.5-mortality association was observed across all four spatial scales of PM2.5, with higher but less precisely estimated HRs observed for local (< 1 km) and neighborhood (1-10 km) variations. In temporally-decomposed analyses, the PM2.5-mortality HRs were stable across yearly cohorts. The meta-analytic HR using two-year lagged PM2.5 equaled 1.10 (95% CI 1.07, 1.13) per 10 μg/m3. Comparable results were observed in time-independent analyses using a 17-year (HR 1.13, CI 1.09, 1.16) or 28-year (HR 1.09, CI 1.07, 1.12) exposure window.
CONCLUSIONS	Long-term exposures to PM2.5, PM2.5-10, and SO2 were associated with increased risk of all-cause and cardiopulmonary mortality. Each spatial decomposition of PM2.5 was associated with mortality risk, and PM2.5-mortality associations were consistent over time.

표-762. PubMed 논문번호 31756804의 내용 요약

구분	내용
PubMed ID	31756804
TITLE	Mortality and hospitalization associated to emissions of a coal power plant: A population-based cohort study.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2019.133757
AUTHORS	Minichilli Fabrizio, Gorini Francesca, Bustaffa Elisa, Cori Liliana, Bianchi Fabrizio
BACKGROUND	Coal-fired thermal power plants represent a significant source of air pollutants, especially sulfur dioxide (SO2) that has been associated with an increased risk of mortality and morbidity for respiratory and cardiovascular disease. A coal power plant in Vado Ligure (Italy) (CPPVL) started in 1970 was stopped in 2014 by the Prosecutor's Office on the grounds of environmental and health culpable disaster.
OBJECTIVE	To investigate the association between the exposure of residents to atmospheric pollutants emitted by CPPVL and the risk of mortality and hospitalization, considering both cancer and non-cancer causes in a population-based cohort study.
METHODS	SO2 and nitrogen oxides (NOx), estimated using the ABLE-MOLOCH-ADMS-Urban dispersion model, were selected as representative surrogates of exposure to CPPVL emissions (SO2-CPPVL) and cumulative emissions from other sources of pollution (NOx-MS), respectively. The relationship between each health outcome and categories of exposure to SO2-CPPVL was estimated by the Hazard Ratio (HR) using multiple sex-specific Cox regression models, adjusted for age, exposure to NOx-MS, and socio-economic deprivation index using SO2-CPPVL first quartile as a reference.
RESULTS	144,019 individuals were recruited (follow-up 2001-2013). An excess of mortality was found for all natural causes (men: 1.49; 95% CI 1.38-1.60; women: 1.49; 95% CI 1.39-1.59), diseases of the circulatory system (men: 1.41; 95% CI 1.24-1.56; women: 1.59; 95% CI 1.44-1.77), of the respiratory system (men: 1.90; 95% CI 1.47-2.45; women: 1.62; 95% CI 1.25-2.09), and of the nervous system and sense organs (men: 1.34; 95% CI 0.97-1.86; women: 1.38; 95% CI 1.03-1.83), and in men for trachea, bronchus, and lung cancers (1.59; 95% CI 1.26-2.00). Results of hospitalization analysis were consistent with those of mortality.
CONCLUSION	Results obtained, also when considering multiple sources of exposure, indicate that exposure to CPP emissions represents a risk factor for selected health outcomes as well as the urgently adoption of primary prevention measures and of a specific surveillance programme.

표-763. PubMed 논문번호 31775094의 내용 요약

구분	내용
PubMed ID	31775094
TITLE	The impact of air pollution on the incidence of diabetes and survival among prevalent diabetes cases.
JOURNAL	Environment international: 10.1016/j.envint.2019.105333
AUTHORS	Paul Lauren A, Burnett Richard T, Kwong Jeffrey C, Hystad Perry, van Donkelaar Aaron, Bai Li, Goldberg Mark S, Lavigne Eric, Copes Ray, Martin Randall V, Kopp Alexander, Chen Hong, Paul Lauren A, Burnett Richard T, Kwong Jeffrey C, Hystad Perry, van Donkelaar Aaron, Bai Li, Goldberg Mark S, Lavigne Eric, Copes Ray, Martin Randall V, Kopp Alexander, Chen Hong
PURPOSE	Growing evidence implicates ambient air pollutants in the development of major chronic diseases and premature mortality. However, epidemiologic evidence linking air pollution to diabetes remains inconclusive. This study sought to determine the relationships between selected air pollutants (nitrogen dioxide [NO2], fine particulate matter [PM2.5], ozone [O3], and oxidant capacity [Ox; the redox-weighted average of O3 and NO2]) and the incidence of diabetes, as well as the risk of cardiovascular or diabetes mortality among individuals with prevalent diabetes.
RESEARCH DESIGN AND METHODS	We followed two cohorts, which included 4.8 million Ontario adults free of diabetes and 452,590 Ontario adults with prevalent diabetes, from 2001 to 2015. Area-level air pollution exposures were assigned to subjects' residential areas, and outcomes were ascertained using health administrative data with validated algorithms. We estimated hazard ratios for the association between each air pollutant and outcome using Cox proportional hazards models, and modelled the shape of the concentration-response relationships.
RESULTS	Over the study period, 790,461 individuals were diagnosed with diabetes. Among those with prevalent diabetes, 26,653 died from diabetes and 64,773 died from cardiovascular diseases. For incident diabetes, each IQR increase in NO2 had a hazard ratio of 1.04 (95% CI: 1.03-1.05). This relationship was relatively robust to all sensitivity analyses considered, and exhibited a near-linear shape. There were also positive associations between incident diabetes and PM2.5, O3, and Ox, but these estimates were somewhat sensitive to different models considered. Among those with prevalent diabetes, almost all pollutants were associated with increased diabetes and cardiovascular mortality risk. The strongest association was observed between diabetes mortality and exposure to NO2 (HR = 1.08, 95% CI: 1.02-1.13).
CONCLUSIONS	Selected air pollutants, especially NO2, were linked to an increased risk of incident diabetes, as well as risk of cardiovascular or diabetes mortality among persons with prevalent diabetes. As NO2 is frequently used as a proxy for road traffic exposures, this result may indicate that traffic-related air pollution has the strongest effect on diabetes etiology and survival after diabetes development.

표-764. PubMed 논문번호 31785778의 내용 요약

구분	내용
PubMed ID	31785778
TITLE	Association between air pollution exposure and diabetic retinopathy among diabetics.
JOURNAL	Environmental research: 10.1016/j.envres.2019.108960
AUTHORS	Pan Shih-Chun, Huang Ching-Chun, Chin Wei-Shan, Chen Bing-Yu, Chan Chang-Chuan, Guo Yue Leon, Pan Shih-Chun, Huang Ching-Chun, Chin Wei-Shan, Chen Bing-Yu, Chan Chang-Chuan, Guo Yue Leon
BACKGROUND	Exposure to air pollution has been linked to adverse effects on vascular diseases. However, the effects of air pollution exposure on diabetic retinopathy (DR), a vascular disease, have not been studied.
OBJECTIVE	To determine the association of ambient air pollution exposure with DR risk.
METHODS	Patients newly diagnosed as having diabetes mellitus (DM) during 2003-2012 from Longitudinal Health Insurance Database 2005), a subset of National Health Insurance Research Database, were included as the study cohort. Newly diagnosed DR patients one year or later after DM diagnosis were identified as cases. Kriging was used to interpolate yearly concentrations of air pollutants at township levels and linked with every individual's residence in each year; average concentrations during the follow-up period were then calculated as personal exposure. Conditional logistic regressions with adjustments for age at DM diagnosis and comorbidities were applied.
RESULTS	Of newly diagnosed DM cases during 2003-2012, 579 were newly diagnosed as having DR over a mean follow-up period of 5.6 years. The Odds ratio (95% confidence interval) of DR occurrence for every 10- μg/m3 increase in particulate matter with ≤2.5 and 2.5-10- μm diameter was 1.29 (1.11-1.50) and 1.37 (1.17-1.61), respectively.
CONCLUSION	In patients with DM, the higher particulate matter exposure, the higher is the DR risk.

표-765. PubMed 논문번호 31831536의 내용 요약

구분	내용
PubMed ID	31831536
TITLE	Exposure to outdoor air pollution and its human-related health outcomes: an evidence gap map.
JOURNAL	BMJ open: 10.1136/bmjopen-2019-031312
AUTHORS	Sun Zhuanlan, Zhu Demi
OBJECTIVES	Outdoor air pollution is a serious environmental problem worldwide. Current systematic reviews (SRs) and meta-analyses (MAs) mostly focused on some specific health outcomes or some specific air pollution.
DESIGN	This evidence gap map (EGM) is to identify existing gaps from SRs and MAs and report them in broad topic areas.
DATA SOURCES	PubMed, Cochrane, Scopus and Web of Science were searched from their inception until June 2018. Citations and reference lists were traced.
ELIGIBILITY CRITERIA	SRs and MAs that investigated the impact of outdoor air pollution on human health outcomes were collected. This study excluded original articles and qualitative review articles.
DATA EXTRACTION AND SYNTHESIS	Characteristics of the included SRs and MAs were extracted and summarised. Extracted data included authors, publication year, location of the corresponding author(s), publication journal discipline, study design, study duration, sample size, study region, target population, types of air pollution and health outcomes.
RESULTS	Asia and North America published 93% of SRs and MAs included in this EGM. 31% of the SRs and MAs (27/86) included primary studies conducted in 5-10 countries. Their publication trends have increased during the last 10 years. A total of 2864 primary studies was included. The median number of included primary studies was 20 (range, 7-167). Cohort studies, case cross-over studies and time-series studies were the top three most used study designs. The mostly researched population was the group of all ages (46/86, 53%). Cardiovascular diseases, respiratory diseases and health service records were mostly reported. A lack of definite diagnostic criteria, unclear reporting of air pollution exposure and time period of primary studies were the main research gaps.
CONCLUSIONS	This EGM provided a visual overview of health outcomes affected by outdoor air pollution exposure. Future research should focus on chronic diseases, cancer and mental disorders.

표-766. PubMed 논문번호 31833635의 내용 요약

구분	내용
PubMed ID	31833635
TITLE	Association between passive smoking and cardiovascular disease: A systematic review and meta-analysis.
JOURNAL	IUBMB life: 10.1002/iub.2207
AUTHORS	Khoramdad Malihe, Vahedian-Azimi Amir, Karimi Leila, Rahimi-Bashar Farshid, Amini Hossein, Sahebkar Amirhossein, Khoramdad Malihe, Vahedian-Azimi Amir, Karimi Leila, Rahimi-Bashar Farshid, Amini Hossein, Sahebkar Amirhossein
BACKGROUND	The association between passive smoking (PS) and cardiovascular disease (CVD) has not yet been fully clarified.
OBJECTIVE	This meta-analysis was performed to evaluate the association between PS and the incidence of CVDs and mortality due to CVD.
METHODS	PubMed/Medicine, Science Direct, Scopus, Web of Knowledge, and ProQuest were searched to identify observational studies that met the inclusion criteria without time, language, age, gender, ethnicity, and design restrictions until July 30, 2018. In case-control studies, relative risk (RR) with 95% confidence interval (CI) was calculated for the relationship between PS and CVD incidence. Also, in cohort studies, hazard ratio (HR) with 95% CI was calculated for the relationship between PS and CVD mortality.
RESULTS	Eighteen studies (10 cohort and 8 case-control studies) were included with 10,672 participants (2,542 cases and 8,130 controls) in case-control studies and 2,313,935 participants in cohort studies. This meta-analysis in case-control studies revealed that the PS could increase the risk of CVD incidence by 28% (adjusted RR = 1.28 [95% CI 1.09, 1.50]), where the highest risk was associated with those who were exposed to second-hand smoke at home and at work (Adjusted RR = 1.41 [95% CI 0.73, 2.70]). Also, the meta-analysis in cohort studies indicated that PS was associated with a 12% higher increase in the risk of CVD mortality (Adjusted HR = 1.12 [95% CI 1.06, 1.20]) with the highest risk of mortality being observed for those who were exposed to second-hand smoking at home, work, and public places (Adjusted HR = 1.26 [95% CI 1.13, 1.40]).
CONCLUSIONS	PS is significantly associated with an increased risk of incidence and mortality of CVD.

표-767. PubMed 논문번호 31839460의 내용 요약

구분	내용
PubMed ID	31839460
TITLE	Long-term fine particulate matter exposure and cardiovascular mortality in the general population: a nationwide cohort study.
JOURNAL	Journal of cardiology: 10.1016/j.jjcc.2019.11.004
AUTHORS	Kim In-Soo, Yang Pil-Sung, Lee Jinae, Yu Hee Tae, Kim Tae-Hoon, Uhm Jae-Sun, Kim Jong-Youn, Pak Hui-Nam, Lee Moon-Hyoung, Joung Boyoung, Kim In-Soo, Yang Pil-Sung, Lee Jinae, Yu Hee Tae, Kim Tae-Hoon, Uhm Jae-Sun, Kim Jong-Youn, Pak Hui-Nam, Lee Moon-Hyoung, Joung Boyoung
BACKGROUND	Although eastern Asian countries are exposed to high levels of air pollution, the impact of long-term exposures to fine particulate matter (PM2.5) air pollution on all-cause and cardiovascular mortality is not well identified. We assessed the relationship between long-term PM2.5 exposure and all-cause/cardiovascular mortalities.
METHODS	We included 436,933 subjects who received national health examinations from the Korean National Health Insurance Service-based National Sample Cohort. We matched subjects' residential-address areas with hourly-measurements of PM2.5 concentration data. We estimated the risk of mortality with average PM2.5 exposure during the study period using a Cox proportional-hazards model.
RESULTS	During 1,683,271 person·years, all-cause and cardiovascular mortalities were observed in 6432 and 1603 subjects (382 and 95 per 100,000 person·years, respectively). An increase in 10 μg/m3 in PM2.5 was associated with increases in all-cause and cardiovascular mortalities by 3.4 % [2.7-4.1] and 4.7 % [3.6-5.8], respectively (each $p < 0.001$). PM2.5 was linearly and significantly correlated with these all-cause and cardiovascular mortalities above 18 μg/m3 of PM2.5 ($p < 0.001$), but it was not significant below 18 μg/m3 of PM2.5. To investigate the specific PM2.5 concentration for raising cardiovascular mortality more, we analyzed the sensitivities/specificities for different PM2.5 levels, and 18 μg/m3 showed the highest Youden's index (sensitivity + specificity-1) with c-index of 0.85 (0.84-0.86). PM2.5 effect on all-cause mortality was more profound in subjects with previous myocardial infarction compared to the opposite population.
CONCLUSIONS	In the Korean general population exposed to high-air pollution, long-term PM2.5 exposure was linearly associated with increased risk for all-cause and cardiovascular mortality, especially above 18 μg/m3 of PM2.5.

표-768. PubMed 논문번호 31865970의 내용 요약

구분	내용
PubMed ID	31865970
TITLE	Ambient Air Pollution and Mortality After Cardiac Transplantation.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2019.09.066
AUTHORS	Al-Kindi Sadeer G, Sarode Anuja, Zullo Melissa, Brook Jeff, Burnett Rick, Oliveira Guilherme H, Huang Wei, Brook Robert, Rajagopalan Sanjay
BACKGROUND	Heart transplant recipients are at high risk for mortality, with traditional risk scores performing modestly in predicting post-transplant survival, underscoring the importance of as yet unidentified factors in determining prognosis. In this analysis, the association between PM2.5 exposure levels and survival after heart transplantation were investigated.
OBJECTIVES	This study sought to study the association between PM2.5 exposure and mortality following heart transplantation.
METHODS	On the basis of the zip code of residence, mortality data in patients who underwent heart transplantation (2004 to 2015) in the United Network for Organ Sharing (UNOS) database were linked with validated estimates of fine particulate matter concentrations (particles with diameter <2.5 μm [PM2.5]; 1 × 1-km grids) for each calendar year during which a UNOS cardiac transplant recipient was at risk for death. Cox proportional hazard models were used to estimate the relationship between exposure and overall mortality adjusting for recipient, donor, and neighborhood variables.
RESULTS	A total of 21,800 patients with 86,713 patient-years of follow-up was included. Mean age at transplantation was 52.6 ± 12.6 years, 75% were male, 69% were white, and 39% had ischemic etiology of heart failure. Mean annual exposure to PM2.5 was 10.6 ± 2.3 μg/m3. At a median follow-up of 4.8 (95% confidence interval: 2.0 to 7.8) years, 5,208 patients (23.9%) had died. The estimated mortality hazard ratio, per 10 μg/m3 increment increase in annual PM2.5 exposure was 1.43 (95% confidence interval: 1.21 to 1.49). After adjusting for 30 recipient, donor, and neighborhood variables, the estimated mortality hazard ratio per 10 μg/m3 increment in annual exposure to PM2.5 was 1.26 (95% confidence interval: 1.11 to 1.43) relative increase in hazard of mortality. This association was consistent across subgroups.
CONCLUSIONS	This study provides evidence linking air pollution with mortality after heart transplantation. These results suggest an important influence of a key environmental factor in outcomes following heart transplantation, and supports the need for further studies in this population.

표-769. PubMed 논문번호 31880734의 내용 요약

구분	내용
PubMed ID	31880734
TITLE	Air Pollution, Physical Activity, and Cardiovascular Function of Patients With Implanted Cardioverter Defibrillators: A Randomized Controlled Trial of Indoor Versus Outdoor Activity.
JOURNAL	Journal of occupational and environmental medicine: 10.1097/JOM.0000000000001795
AUTHORS	Liu Ling, Urch Bruce, Nanthakumar Kumaraswamy, Chen Li, Smith-Doiron Marc, Brook Jeffrey R, Speck Mary, Silverman Frances, Stieb David M
OBJECTIVE	To investigate whether implanted cardioverter defibrillator (ICD) patients exercising indoors on higher air pollution (AP) days had reduced adverse cardiovascular effects compared with those exercising outdoors.
METHODS	Eighteen participants were randomly divided into control or intervention groups. Blood pressure (BP), pulse rate (PR), and oxygen saturation (O2SAT) were measured daily before and after participants walked outdoors for 30minutes. On days with higher forecast AP the intervention group exercised indoors.
RESULTS	AP was significantly associated with increased BP and PR, and reduced O2SAT. After adjustment for exercise levels, AP was associated with increased diastolic BP and PR in controls only. Significant improvements in cardiovascular measures over time were observed in both groups.
CONCLUSION	In ICD patients, reducing AP exposure may reduce adverse cardiovascular effects, while daily mild exercise may benefit cardiovascular function.

표-770. PubMed 논문번호 31881529의 내용 요약

구분	내용
PubMed ID	31881529
TITLE	Identifying critical windows of prenatal particulate matter (PM〈sub〉2.5〈/sub〉) exposure and early childhood blood pressure.
JOURNAL	Environmental research: 10.1016/j.envres.2019.109073
AUTHORS	Rosa Maria Jos□, Hair Gleicy Macedo, Just Allan C, Kloog Itai, Svensson Katherine, Pizano-Z□rate Mar□a Luisa, Pantic Ivan, Schnaas Lourdes, Tamayo-Ortiz Marcela, Baccarelli Andrea A, Tellez-Rojo Martha M, Wright Robert O, Sanders Alison P, Rosa Maria Jos□, Hair Gleicy Macedo, Just Allan C, Kloog Itai, Svensson Katherine, Pizano-Z□rate Mar□a Luisa, Pantic Ivan, Schnaas Lourdes, Tamayo-Ortiz Marcela, Baccarelli Andrea A, Tellez-Rojo Martha M, Wright Robert O, Sanders Alison P
BACKGROUND	Exposure to air pollution is associated with increased blood pressure (BP) in adults and children. Some evidence suggests that air pollution exposure during the prenatal period may contribute to adverse cardiorenal health later in life. Here we apply a distributed lag model (DLM) approach to identify critical windows that may underlie the association between prenatal particulate matter ≤ 2.5 μm in diameter (PM2.5) exposure and children's BP at ages 4-6 years.
METHODS	Participants included 537 mother-child dyads enrolled in the Programming Research in Obesity, GRowth Environment, and Social Stress (PROGRESS) longitudinal birth cohort study based in Mexico City. Prenatal daily PM2.5 exposure was estimated using a validated satellite-based spatio-temporal model and BP was measured using the automated Spacelabs system with a sized cuff. We used distributed lag models (DLMs) to examine associations between daily PM2.5 exposure and systolic and diastolic BP (SBP and DBP), adjusting for child's age, sex and BMI, as well as maternal education, preeclampsia and indoor smoking report during the second and third trimester, seasonality and average postnatal year 1 PM2.5 exposure.
RESULTS	We found that PM2.5 exposure between weeks 11-32 of gestation (days 80-226) was significantly associated with children's increased SBP. Similarly, PM2.5 exposure between weeks 9-25 of gestation (days 63-176) was significantly associated with increased DBP. To place this into context, a constant 10 μg/m3 increase in PM2.5 sustained throughout this critical window would predict a cumulative increase of 2.6 mmHg (CI: 0.5, 4.6) in SBP and 0.88 mmHg (CI: 0.1, 1.6) in DBP at ages 4-6 years. In a stratified analysis by sex, this association persisted in boys but not in girls.
CONCLUSIONS	Second and third trimester PM2.5 exposure may increase children's BP in early life. Further work investigating PM2.5 exposure with BP trajectories later in childhood will be important to understanding cardiorenal trajectories that may predict adult disease. Our results underscore the importance of reducing air pollution exposure among susceptible populations, including pregnant women.

표-771. PubMed 논문번호 31888884의 내용 요약

구분	내용
PubMed ID	31888884
TITLE	Association between ambient fine particulate pollution and hospital admissions for cause specific cardiovascular disease: time series study in 184 major Chinese cities.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.l6572
AUTHORS	Tian Yaohua, Liu Hui, Wu Yiqun, Si Yaqin, Song Jing, Cao Yaying, Li Man, Wu Yao, Wang Xiaowen, Chen Libo, Wei Chen, Gao Pei, Hu Yonghua
OBJECTIVE	To estimate the risks of daily hospital admissions for cause specific major cardiovascular diseases associated with short term exposure to ambient fine particulate matter (aerodynamic diameter ≤2.5 μm; PM2.5) pollution in China.
DESIGN	National time series study.
SETTING	184 major cities in China.
POPULATION	8 834 533 hospital admissions for cardiovascular causes in 184 Chinese cities recorded by the national database of Urban Employee Basic Medical Insurance from 1 January 2014 to 31 December 2017.
MAIN OUTCOME MEASURES	Daily counts of city specific hospital admissions for primary diagnoses of ischaemic heart disease, heart failure, heart rhythm disturbances, ischaemic stroke, and haemorrhagic stroke among different demographic groups were used to estimate the associations between PM2.5 and morbidity. An overdispersed generalised additive model was used to estimate city specific associations between PM2.5 and cardiovascular admissions, and random effects meta-analysis used to combine the city specific estimates.
RESULTS	Over the study period, a mean of 47 hospital admissions per day (standard deviation 74) occurred for cardiovascular disease, 26 (53) for ischaemic heart disease, one (five) for heart failure, two (four) for heart rhythm disturbances, 14 (28) for ischaemic stroke, and two (four) for haemorrhagic stroke. At the national average level, an increase of 10 μg/m3 in PM2.5 was associated with a 0.26% (95% confidence interval 0.17% to 0.35%) increase in hospital admissions on the same day for cardiovascular disease, 0.31% (0.22% to 0.40%) for ischaemic heart disease, 0.27% (0.04% to 0.51%) for heart failure, 0.29% (0.12% to 0.46%) for heart rhythm disturbances, and 0.29% (0.18% to 0.40%) for ischaemic stroke, but not with haemorrhagic stroke (-0.02% (-0.23% to 0.19%)). The national average association of PM2.5 with cardiovascular disease was slightly non-linear, with a sharp slope at PM2.5 levels below 50 μg/m3, a moderate slope at 50-250 μg/m3, and a plateau at concentrations higher than 250 μg/m3. Compared with days with PM2.5 up to 15 μg/m3, days with PM2.5 of 15-25, 25-35, 35-75, and 75 μg/m3 or more were significantly associated with increases in cardiovascular admissions of 1.1% (0 to 2.2%), 1.9% (0.6% to 3.2%), 2.6% (1.3% to 3.9%), and 3.8% (2.1% to 5.5%), respectively.According to projections, achieving the Chinese grade 2 (35 μg/m3), Chinese grade 1 (15 μg/m3), and World Health Organization (10 μg/m3) regulatory limits for annual mean PM2.5 concentrations would reduce the annual number of admissions for cardiovascular disease in China. Assuming causality, which should be done with caution, this reduction would translate into an estimated 36 448 (95% confidence interval 24 441 to 48 471), 85 270 (57 129 to 113 494), and 97 516 (65 320 to 129 820), respectively.
CONCLUSIONS		

표-772. PubMed 논문번호 31888885의 내용 요약

구분	내용
PubMed ID	31888885
TITLE	Long term exposure to ambient fine particulate matter and incidence of stroke: prospective cohort study from the China-PAR project.
JOURNAL	BMJ (Clinical research ed.): 10.1136/bmj.l6720
AUTHORS	Huang Keyong, Liang Fengchao, Yang Xueli, Liu Fangchao, Li Jianxin, Xiao Qingyang, Chen Jichun, Liu Xiaoqing, Cao Jie, Shen Chong, Yu Ling, Lu Fanghong, Wu Xianping, Zhao Liancheng, Wu Xigui, Li Ying, Hu Dongsheng, Huang Jianfeng, Liu Yang, Lu Xiangfeng, Gu Dongfeng
OBJECTIVE	To study the effect of long term exposure to ambient fine particulate matter of diameter ≤2.5 μm (PM2.5) on the incidence of total, ischemic, and hemorrhagic stroke among Chinese adults.
DESIGN	Population based prospective cohort study.
SETTING	Prediction for Atherosclerotic Cardiovascular Disease Risk in China (China-PAR) project carried out in 15 provinces across China.
PARTICIPANTS	117 575 Chinese men and women without stroke at baseline in the China-PAR project.
MAIN OUTCOME MEASURES	Incidence of total, ischemic, and hemorrhagic stroke.
RESULTS	The long term average PM2.5 level from 2000 to 2015 at participants' residential addresses was 64.9 μg/m3, ranging from 31.2 μg/m3 to 97.0 μg/m3. During 900 214 person years of follow-up, 3540 cases of incident stroke were identified, of which 63.0% (n=2230) were ischemic and 27.5% (n=973) were hemorrhagic. Compared with the first quarter of exposure to PM2.5 (<54.5 μg/m3), participants in the highest quarter (>78.2 μg/m3) had an increased risk of incident stroke (hazard ratio 1.53, 95% confidence interval 1.34 to 1.74), ischemic stroke (1.82, 1.55 to 2.14), and hemorrhagic stroke (1.50, 1.16 to 1.93). For each increase of 10 μg/m3 in PM2.5 concentration, the increased risks of incident stroke, ischemic stroke, and hemorrhagic stroke were 13% (1.13, 1.09 to 1.17), 20% (1.20, 1.15 to 1.25), and 12% (1.12, 1.05 to 1.20), respectively. Almost linear exposure-response relations between long term exposure to PM2.5 and incident stroke, overall and by its subtypes, were observed.
CONCLUSIONS	This study provides evidence from China that long term exposure to ambient PM2.5 at relatively high concentrations is positively associated with incident stroke and its major subtypes. These findings are meaningful for both environmental and health policy development related to air pollution and stroke prevention, not only in China, but also in other low and middle income countries.

표-773. PubMed 논문번호 31891579의 내용 요약

구분	내용
PubMed ID	31891579
TITLE	Ambient particulate matter pollution and adult hospital admissions for pneumonia in urban China: A national time series analysis for 2014 through 2017.
JOURNAL	PLoS medicine: 10.1371/journal.pmed.1003010
AUTHORS	Tian Yaohua, Liu Hui, Wu Yiqun, Si Yaqin, Li Man, Wu Yao, Wang Xiaowen, Wang Mengying, Chen Libo, Wei Chen, Wu Tao, Gao Pei, Hu Yonghua
BACKGROUND	The effects of ambient particulate matter (PM) pollution on pneumonia in adults are inconclusive, and few scientific data on a national scale have been generated in low- or middle-income countries, despite their much higher PM concentrations. We aimed to examine the association between PM levels and hospital admissions for pneumonia in Chinese adults.
METHODS AND FINDINGS	A nationwide time series study was conducted in China between 2014 and 2017. Information on daily hospital admissions for pneumonia for 2014-2017 was collected from the database of Urban Employee Basic Medical Insurance (UEBMI), which covers 282.93 million adults. Associations of PM concentrations and hospital admissions for pneumonia were estimated for each city using a quasi-Poisson regression model controlling for time trend, temperature, relative humidity, day of the week, and public holiday and then pooled by random-effects meta-analysis. Meta-regression models were used to investigate potential effect modifiers, including cities' annual-average air pollutants concentrations, temperature, relative humidity, gross domestic product (GDP) per capita, and coverage rates by the UEBMI. More than 4.2 million pneumonia admissions were identified in 184 Chinese cities during the study period. Short-term elevations in PM concentrations were associated with increased pneumonia admissions. At the national level, a 10-μg/m3 increase in 3-day moving average (lag 0-2) concentrations of PM2.5 (PM $\leq$2.5 μm in aerodynamic diameter) and PM10 (PM $\leq$10 μm in aerodynamic diameter) was associated with 0.31% (95% confidence interval [CI] 0.15%-0.46%, P < 0.001) and 0.19% (0.11%-0.30%, P < 0.001) increases in hospital admissions for pneumonia, respectively. The effects of PM10 were stronger in cities with higher temperatures (percentage increase, 0.031%; 95% CI 0.003%-0.058%; P = 0.026) and relative humidity (percentage increase, 0.011%; 95% CI 0%-0.022%; P = 0.045), as well as in the elderly (percentage increase, 0.10% [95% CI 0.02%-0.19%] for people aged 18-64 years versus 0.32% [95% CI 0.22%-0.39%] for people aged $\geq$75 years; P < 0.001). The main limitation of the present study was the unavailability of data on individual exposure to PM pollution.
CONCLUSIONS	Our findings suggest that there are significant short-term associations between ambient PM levels and increased hospital admissions for pneumonia in Chinese adults. These findings support the rationale that further limiting PM concentrations in China may be an effective strategy to reduce pneumonia-related hospital admissions.

표-774. PubMed 논문번호 31926217의 내용 요약

구분	내용
PubMed ID	31926217
TITLE	The Association Between Secondhand Smoke Exposure and Survival for Patients With Heart Failure.
JOURNAL	Journal of cardiac failure: 10.1016/j.cardfail.2019.12.008
AUTHORS	Psotka Mitchell A, Rushakoff Joshua, Glantz Stanton A, De Marco Teresa, Fleischmann Kirsten E
BACKGROUND	The effect of secondhand tobacco smoke (SHS) exposure on patients with heart failure (HF) is uncertain. We investigated the association of mortality with SHS exposure for patients with HF.
METHODS	Nonsmokers with clinical HF were enrolled from 2003 to 2008 in a single-center longitudinal cohort study. The effect of SHS exposure determined by high-sensitivity urinary cotinine on mortality was estimated by multivariable proportional hazards modeling.
RESULTS	Mortality was assessed after median 4.3 years. Of 202 patients, enrollment urinary cotinine levels were below the limit of detection for 106 (52%) considered unexposed to SHS. The median detectable cotinine was 0.47 ng/mL (interquartile range: [0.28, 1.28]). Participants were 41% female, 65 ± 17 years old, and 57% white race. Elevated cotinine was associated with increased mortality after multivariate adjustment: hazard ratio (HR) per 1 ng/mL increase in urinary cotinine: 1.15, 95% confidence interval (CI): 1.08-1.23, P < .001. Higher age (HR per 5-year increase: 1.32, 95% CI: 1.22-1.43, P < .001), male sex (HR vs female: 1.52, 95% CI: 1.02-2.28, P = .040), and New York Heart Association class (HR for class III vs I: 2.91, 95% CI: 1.71-4.99, P < .001) were also associated with mortality.
CONCLUSIONS	SHS exposure is associated with a dose-dependent increase in mortality for patients with HF.

표-775. PubMed 논문번호 31930033의 내용 요약

구분	내용
PubMed ID	31930033
TITLE	Modifiable risk factors for carotid atherosclerosis: a meta-analysis and systematic review.
JOURNAL	Annals of translational medicine: 10.21037/atm.2019.10.115
AUTHORS	Ji Xi, Leng Xin-Yi, Dong Yi, Ma Ya-Hui, Xu Wei, Cao Xi-Peng, Hou Xiao-He, Dong Qiang, Tan Lan, Yu Jin-Tai
Background	Carotid atherosclerosis is a major cause of stroke, but the conclusion about risk factors for carotid atherosclerosis is still controversial. The aim of our present meta-analysis and systematic review was to explore the modifiable risk factors for carotid atherosclerosis.
Methods	We searched PubMed from January 1962 to October 2018 to include longitudinal and cross-sectional studies. The results were pooled using random effects model. Heterogeneity was measured by I2 statistic and publication bias was assessed by funnel plots.
Results	A total of 14,700 articles were screened, of which 76 with 27 factors were eligible. Our meta-analysis of cross-sectional studies indicated nine factors (hyperlipidemia, hyperhomocysteinemia, hypertension, hyperuricemia, smoking, metabolic syndrome, hypertriglyceridemia, diabetes, and higher low density lipoprotein) were significantly associated with the presence of carotid plaque, among which four (hyperlipidemia, hyperhomocysteinemia, hypertension, and hyperuricemia) could elevate the risk of atherosclerosis by at least 50%; and one factor (hypertension) was associated with increased carotid intima-media thickness. In the systematic review, another five factors [negative emotion, socioeconomic strain, alcohol, air pollution, and obstructive sleep apnea syndrome (OSAS)] were also related to the presence of atherosclerosis. The cross-sectional associations with most of the above 14 factors were further confirmed by longitudinal studies. Among them, the managements of 4 factors (hypertension, hyperlipidemia, diabetes and OSAS) were indicated to prevent carotid atherosclerosis by cohort studies.
Conclusions	Effective interventions targeting pre-existing disease, negative emotion, lifestyle and diet may reduce the risk of carotid atherosclerosis. Further good-quality prospective studies are needed to confirm these findings.

표-776. PubMed 논문번호 31931190의 내용 요약

구분	내용
PubMed ID	31931190
TITLE	Maternal air pollution exposure associated with risk of congenital heart defect in pre-pregnancy overweighted women.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2019.136470
AUTHORS	Yang Yin, Lin Qingmei, Liang Yin, Ruan Zengliang, Acharya Bipin Kumar, Zhang Shiyu, Qian Zhengmin, McMillin Stephen Edward, Hinyard Leslie, Sun Jia, Wang Changke, Ge Haibo, Wu Xueli, Guo Xiaoling, Lin Hualiang
OBJECTIVES	Prenatal exposure to air pollutant has been associated with congenital heart defect (CHD). However, no study has investigated this effect in pre-pregnancy overweighted women. This study aimed to evaluate gestational exposure to particulate pollutant (PM2.5) and gaseous air pollutants (O3 and NO2) on the risk of CHD, and explore the potential effect modifiers including maternal age, pre-pregnancy BMI and pregestational diseases.
METHODS	In this birth cohort study, a total of 63,213 pregnant women in Foshan, China were initially recruited and followed from their first hospital visit for pregnancy to delivery during 2015-2019. CHD cases were confirmed by the records in hospital- and population- based birth defect surveillance systems. Air pollutant exposures were estimated by the daily concentrations measured in air monitoring stations in each participant's residential county. Mixed-effects regression models, adjusted for potential confounding factors were applied to estimate the associations between air pollutant and CHD during the first three months of the pregnancy.
RESULTS	A total of 985 (1.6%) newborns were identified as CHD cases. For each 10 μg/m3 increase in ambient O3 during the 1st month, the OR values for CHD were 1.03 (95% CI: 0.94, 1.13) in pre-pregnancy normal weighted women and 1.24 (95% CI: 1.01, 1.53) in pre-pregnancy overweighted women. For each 10 μg/m3 increase in NO2 during the 3rd month, the OR values for CHD were 1.09 (95% CI: 1.01, 1.18) in pre-pregnancy normal weighted women and 1.27 (95% CI: 1.07, 1.51) in pre-pregnancy overweighted women. No significant associations were found between PM2.5 exposure and CHD in our analysis.
CONCLUSIONS	This study demonstrates that gaseous air pollutants (O3 and NO2) exposure during the cardiac embryogenesis period is associated with an increased risk of CHD, particularly for pre-pregnancy overweighted women.

표-777. PubMed 논문번호 31941383의 내용 요약

구분	내용
PubMed ID	31941383
TITLE	Short-Term Exposure to Waterpipe/Hookah Smoke Triggers a Hyperactive Platelet Activation State and Increases the Risk of Thrombogenesis.
JOURNAL	Arteriosclerosis, thrombosis, and vascular biology: 10.1161/ATVBAHA.119.313435
AUTHORS	Alarabi Ahmed B, Karim Zubair A, Ramirez Jean E Montes, Hernandez Keziah R, Lozano Patricia A, Rivera Jos□ O, Alshbool Fatima Z, Khasawneh Fadi T
OBJECTIVE	Cardiovascular disease is a major public health problem. Among cardiovascular disease's risk factors, tobacco smoking is considered the single most preventable cause of death, with thrombosis being the main mechanism of cardiovascular disease mortality in smokers. While tobacco smoking has been on the decline, the use of waterpipes/hookah has been rising, mainly due to the perception that they are less harmful than regular cigarettes. Strikingly, there are few studies on the negative effects of waterpipes on the cardiovascular system, and none regarding their direct contribution to thrombus formation. Approach and Results: We used a waterpipe whole-body exposure protocol that mimics real-life human exposure scenarios and investigated its effects, relative to clean air, on platelet function, hemostasis, and thrombogenesis. We found that waterpipe smoke (WPS)-exposed mice exhibited both shortened thrombus occlusion and bleeding times. Further, our results show that platelets from WPS-exposed mice are hyperactive, with enhanced agonist-induced aggregation, dense and α-granule secretion, αIIbβ3 integrin activation, phosphatidylserine expression, and platelet spreading, when compared with clean air-exposed platelets. Finally, at the molecular level, it was found that Akt (protein kinase B) and ERK (extracellular signal-regulated kinases) phosphorylation are enhanced in the WPS and in nicotine-treated platelets.
CONCLUSIONS	Our findings demonstrate that WPS exposure directly modulates hemostasis and increases the risk of thrombosis and that this is mediated, in part, via a state of platelet hyperactivity. The negative health impact of WPS/hookah, therefore, should not be underestimated. Moreover, this study should also help in raising public awareness of the toxic effects of waterpipe/hookah.

표-778. PubMed 논문번호 31941576의 내용 요약

구분	내용
PubMed ID	31941576
TITLE	The Effects of Fine Dust, Ozone, and Nitrogen Dioxide on Health.
JOURNAL	Deutsches Arzteblatt international: 10.3238/arztebl.2019.0881
AUTHORS	Ritz Beate, Hoffmann Barbara, Peters Annette
BACKGROUND	Air pollutants, especially fine dust, ozone, and nitrogen dioxide, pose a danger to health worldwide. In 2005, the World Health Organization (WHO), in order to protect public health, issued global recommendations for maximum levels of fine dust (10 μg/m3 for fine dust particles smaller than 2.5 μm [PM2.5]), ozone, and nitrogen dioxide. The recommended levels are regularly exceeded in many places in Germany.
METHODS	This review is based on relevant publications retrieved by a selective search in PubMed and, in part, on an expert statement issued in the name of the International Society for Environmental Epidemiology (ISEE) and the European Respiratory Society (ERS).
RESULTS	Air pollutants affect the entire body, from the beginning of intrauterine development all the way to the end of life, causing premature death mainly through lung and heart disease. An epidemiological study has shown, for example, that mor- tality rises approximately 7% for every incremental long-term exposure to 5 μg/m3 PM2.5 (95% confidence interval: [2; 13]). Aside from lung and heart disease, the carcinogenic effect of fine dust is now well established. High fine-dust exposure has also been linked to metabolic diseases. For example, in a meta-analysis of cohort studies, the incidence of type 2 diabetes mellitus was found to be associated with elevated fine dust concentrations, with a 25% relative risk increase [10; 43] for every 10 □g/m3 of PM2.5. More recent studies have shown that these substances cause harm even in concentrations that are below the recommended limits.
CONCLUSION	It is very important for public health that the current EU standards for rkedly lowered so that health risks can be further reduced, in accordance with the recommendations of the WHO.

표-779. PubMed 논문번호 31974293의 내용 요약

구분	내용
PubMed ID	31974293
TITLE	Lifetime cumulative exposure to rubber dust, fumes and N-nitrosamines and non-cancer mortality: a 49-year follow-up of UK rubber factory workers.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2019-106269
AUTHORS	Hidajat Mira, McElvenny Damien Martin, Ritchie Peter, Darnton Andrew, Mueller William, Agius Raymond M, Cherrie John W, de Vocht Frank
OBJECTIVES	To examine associations between occupational exposures to rubber dust, rubber fumes and N-nitrosamines and non-cancer mortality.
METHODS	A cohort of 36 441 males aged 35+ years employed in British rubber factories was followed-up to 2015 (94% deceased). Competing risk survival analysis was used to assess risks of dying from non-cancer diseases (respiratory, urinary, cerebrovascular, circulatory and digestive diseases). Occupational exposures to rubber dust, rubber fumes, N-nitrosamines were derived based on a population-specific quantitative job-exposure matrix which in-turn was based on measurements in the EU-EXASRUB database.
RESULTS	Exposure-response associations of increased risk with increasing exposure were found for N-nitrosomorpholine with mortality from circulatory diseases (subdistribution hazard ratio (SHR) 1.17; 95% CI 1.12 to 1.23), ischaemic heart disease (IHD) (SHR 1.19; 95% CI 1.13 to 1.26), cerebrovascular disease (SHR 1.19; 95% CI 1.07 to 1.32) and exposures to N-nitrosodimethylamine with respiratory disease mortality (SHR 1.41; 95% CI 1.30 to 1.53). Increased risks for mortality from circulatory disease, IHD and digestive diseases were found with higher levels of exposures to rubber dust, rubber fumes and N-nitrosamines sum, without an exposure-dependent manner. No associations were observed between rubber dust, rubber fumes and N-nitrosamines exposures with mortality from asthma, urinary disease, bronchitis, emphysema, liver disease and some digestive diseases.
CONCLUSIONS	In a cohort of rubber factory workers with 49 years of follow-up, increased risk for mortality from circulatory, cerebrovascular, respiratory and digestive diseases were found to be associated with cumulative occupational exposures to specific agents.

표-780. PubMed 논문번호 32014767의 내용 요약

구분	내용
PubMed ID	32014767
TITLE	Pet ownership in utero and in childhood decreases the effects of environmental tobacco smoke exposure on hypertension in children: A large population based cohort study.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2020.136859
AUTHORS	Xu Shu-Li, Liu Ai-Ping, Wu Qi-Zhen, Marks Tia, He Zhi-Zhou, Qian Zhengmin, McMillin Stephen Edward, Sun Jia, Appleton Allison A, Bloom Michael S, Lin Shao, Yu Hong-Yao, Zhou Yang, Liu Ru-Qing, Feng Dan, Hu Li-Wen, Yang Bo-Yi, Zeng Xiao-Wen, Sun Xiao, Dong Guang-Hui
BACKGROUND	Little is known about whether exposure to pets influences the association between hypertension and environmental tobacco smoke (ETS). The current study aims to examine the interaction of pet ownership on ETS exposure and the development of hypertension in children.
METHODS	From 2012 to 2013, a total of 9354 children, 5 to 17 years of age, were recruited from 62 schools in seven northeastern cities. BP in children was measured and hypertension was defined as an average diastolic blood pressure (DBP) or systolic blood pressure (SBP) at or above the 95th percentile for that child's age, sex, and height. Pet ownership in three different time periods (in utero, past 2 years, and currently) and ETS exposure data were collected from parents via a questionnaire. Two-level regressions were used for the data analyses.
RESULTS	The data show consistent, significant interactions between exposure to pets and effects from ETS. Children who were not exposed to pets experienced stronger effects from ETS on hypertension when compared to those exposed to pets, and the protective effect of pet ownership became stronger with a greater number of pets in the home. Exposure to in utero ETS was associated with hypertension [adjusted odds ratio (aOR) = 1.32, 95% confidence interval (CI): 1.13-1.54] only for those children without pet exposure in utero but not for those with pets (aOR = 0.75; 95% CI: 0.49-1.15) (pinteraction < 0.05). Moreover, household dog ownership was related to significantly lower effects of current ETS on hypertension (aOR = 0.80, 95% CI: 0.61-1.05) compared with children without dogs (aOR = 1.26, 95% CI: 1.11-1.44) (pinteraction = 0.001). Interaction associations between ETS and pet ownership were more robust for girls than for boys and for younger than older children.
CONCLUSION	This study indicates an inverse relationship between pet ownership and ETS, potentially pointing to pet ownership as protecting against the development of hypertension in children.

표-781. PubMed 논문번호 32017464의 내용 요약

구분	내용
PubMed ID	32017464
TITLE	A Spatiotemporal Analysis of Organ-Specific Lupus Flares in Relation to Atmospheric Variables and Fine Particulate Matter Pollution.
JOURNAL	Arthritis & rheumatology (Hoboken, N.J.): 10.1002/art.41217
AUTHORS	Stojan George, Kvit Anton, Curriero Frank C, Petri Michelle
OBJECTIVE	To identify potential clusters of systemic lupus erythematosus (SLE) organ-specific flares and their relationship to fine particulate matter pollution (PM2.5), temperature, ozone concentration, resultant wind, relative humidity, and barometric pressure in the Hopkins Lupus Cohort, using spatiotemporal cluster analysis.
METHODS	A total of 1,628 patients who fulfilled the Systemic Lupus International Collaborating Clinics classification criteria for SLE and who had a home address recorded were included in the analysis. Disease activity was assessed using the Lupus Activity Index. Assessment of rash, joint involvement, serositis, and neurologic, pulmonary, renal, and hematologic activity was quantified on a 0-3 visual analog scale (VAS). An organ-specific flare was defined as an increase in VAS of ≥1 point compared to the previous visit. Spatiotemporal clusters were detected using SaTScan software. Regression models were used for cluster adjustment and included individual, county-level, and environmental variables.
RESULTS	Significant clusters unadjusted for environmental variables were identified for joint flares ($P < 0.05$; n = 3), rash flares ($P < 0.05$; n = 4), hematologic flares ($P < 0.05$; n = 3), neurologic flares ($P < 0.05$; n = 2), renal flares ($P < 0.001$; n = 4), serositis ($P < 0.001$; n = 2), and pulmonary flares ($P < 0.001$; n = 2). The majority of the clusters identified changed in significance, temporal extent, or spatial extent after adjustment for environmental variables.
CONCLUSION	We describe the first spatiotemporal clusters of lupus organ-specific flares. Seasonal, as well as multi-year, cluster patterns were identified, differing in extent and location for the various organ-specific flare types. Further studies focusing on each individual organ-specific flare are needed to better understand the driving forces behind these observed changes.

표-782. PubMed 논문번호 32041074의 내용 요약

구분	내용
PubMed ID	32041074
TITLE	Association between long-term exposure to ambient air pollutants and excessive daytime sleepiness in Chinese rural population: The Henan Rural Cohort Study.
JOURNAL	Chemosphere: 10.1016/j.chemosphere.2020.126103
AUTHORS	Wang Yan, Mao Zhenxing, Chen Gongbo, Tu Runqi, Abdulai Tanko, Qiao Dou, Liu Xue, Dong Xiaokang, Luo Zhicheng, Wang Yikang, Li Ruiying, Huo Wenqian, Yu Songcheng, Guo Yuming, Li Shanshan, Wang Chongjian
BACKGROUND	Excessive daytime sleepiness is associated with many adverse consequences, including cardiovascular diseases and mortality. Although exposure to air pollution has been suggested in connection with excessive daytime sleepiness, evidence in China is scarce. The study aimed to explore the association between long-term exposure to air pollution and excessive daytime sleepiness in rural China.
METHODS	A lot of 27935 participants (60% females) from the Henan Rural Cohort Study were included in this analysis. A satellite-based spatiotemporal model estimated a 3-year average air pollution exposure to NO2 (nitrogen dioxide), PM1 (particulate matter with aerodynamic diameters not more than 1 μm) and PM2.5 (particulate matter with aerodynamic diameters not more than 2.5 μm) at the home address of participants before the baseline survey. Logistic regression was used to evaluate the odds ratio and 95% confidence interval between long-term air pollution and excessive daytime sleepiness.
RESULTS	The average concentrations of NO2, PM1 and PM2.5 during three years preceding baseline survey were 38.22 $\mu g/m^3$, 56.29 $\mu g/m^3$ and 72.30 $\mu g/m^3$. Exposure to NO2, PM1 and PM2.5 were all associated with excessive daytime sleepiness. Each 1 $\mu g/m^3$ increment of NO2, PM1 and PM2.5 were related to a 20% (OR: 1.20, 95% CI: 1.13-1.27), 10% (OR: 1.10, 95% CI: 1.05-1.16) and 17% (OR: 1.17, 95% CI: 1.10-1.23) increase of the prevalence of excessive daytime sleepiness.
CONCLUSION	The study demonstrated that long-term exposure to NO2, PM1 and PM2.5 were all associated with excessive daytime sleepiness. The impact of air pollution should be considered when treating individuals with excessive daytime sleepiness.

표-783. PubMed 논문번호 32046641의 내용 요약

구분	내용
PubMed ID	32046641
TITLE	Association between cumulative social risk, particulate matter environmental pollutant exposure, and cardiovascular disease risk.
JOURNAL	BMC cardiovascular disorders: 10.1186/s12872-020-01329-z
AUTHORS	Canterbury Ann, Echouffo-Tcheugui Justin B, Shpilsky Daniel, Aiyer Aryan, Reis Steven E, Erqou Sebhat
BACKGROUND	Long-term exposure to pollution has been shown to increase risk of cardiovascular disease (CVD) and mortality, and may contribute to the increased risk of CVD among individuals with higher social risk.
METHODS	Data from the community-based Heart Strategies Concentrating on Risk Evaluation (HeartSCORE) study were used to quantify Cumulative Social Risk (CSR) by assigning a score of 1 for the presence of each of 4 social risk factors: racial minority, single living, low income, and low educational status. 1-year average air pollution exposure to PM2.5 was estimated using land-use regression models. Associations with clinical outcomes were assessed using Cox models, adjusting for traditional CVD risk factors. The primary clinical outcome was combined all-cause mortality and nonfatal CVD events.
RESULTS	Data were available on 1933 participants (mean age 59 years, 66% female, 44% Black). In a median follow up time of 8.3 years, 137 primary clinical outcome events occurred. PM2.5 exposure increased with higher CSR score. PM2.5 was independently associated with clinical outcome (adjusted hazard ratio [HR]: 1.19 [95% CI: 1.00, 1.41]). Participants with ≥2 CSR factors had an adjusted HR of 2.34 (1.48-3.68) compared to those with CSR = 0. The association was attenuated after accounting for PM2.5 (HR: 2.16; [1.34, 3.49]). Mediation analyses indicate that PM2.5 explained 13% of the risk of clinical outcome in individuals with CSR score ≥ 2.
CONCLUSION	In a community-based cohort study, we found that the association of increasing CSR with higher CVD and mortality risks is partially accounted for by exposure to PM2.5 environmental pollutants.

표-784. PubMed 논문번호 32069739의 내용 요약

구분	내용
PubMed ID	32069739
TITLE	Associations between long-term exposure to air pollution and blood pressure and effect modifications by behavioral factors.
JOURNAL	Environmental research: 10.1016/j.envres.2019.109109
AUTHORS	Li Na, Chen Gongbo, Liu Feifei, Mao Shuyuan, Liu Yisi, Liu Suyang, Mao Zongfu, Lu Yuanan, Wang Chongjian, Guo Yuming, Xiang Hao, Li Shanshan
BACKGROUND	Studies on the hypertensive effect of long-term air pollution exposure were inconclusive and showed scarce evidence from rural areas in developing countries. In this context, we examined the associations of air pollution exposure with hypertension and blood pressure, and their effect modifiers in rural Chinese adults.
METHODS	We studied 39,259 participants from a cohort established in five rural regions of central China. Individual exposures to PM2.5 and PM10 (particulate matter with an aerodynamic diameter less than or equal to 2.5 μm and 10 μm) and nitrogen dioxide (NO2) was evaluated using satellite-based spatiotemporal models. Mixed-effect regression models were applied to examine the associations of long-term exposure to air pollution with hypertension and four blood pressure component measurements, including systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and pulse pressure (PP). Several potential effect modifiers related to demographic and behavioral factors were also examined.
RESULTS	The results showed that for each 1 μg/m3 increase in PM2.5, PM10 and NO2, the adjusted odds ratio of hypertension was 1.029 (95%CI: 1.001,1.057), 1.015 (95%CI: 1.001, 1.029) and 1.069 (95%CI: 1.038, 1.100), respectively. These three air pollutants were also associated with increased SBP (except for PM10), DBP and MAP. The hypertensive effects of air pollution were more pronounced among males, smokers, drinkers, individuals with a high-fat diet, and those with high-level physical activity.
CONCLUSION	Long-term exposure to PM2.5, PM10 and NO2 was associated with increased blood pressure and hypertension in rural Chinese adults, and the associations were modified by several behavioral factors.

표-785. PubMed 논문번호 32081278의 내용 요약

구분	내용
PubMed ID	32081278
TITLE	Long-Term Exposure to Fine Particulate Matter and Cardiovascular Disease in China.
JOURNAL	Journal of the American College of Cardiology: 10.1016/j.jacc.2019.12.031
AUTHORS	Liang Fengchao, Liu Fangchao, Huang Keyong, Yang Xueli, Li Jianxin, Xiao Qingyang, Chen Jichun, Liu Xiaoqing, Cao Jie, Shen Chong, Yu Ling, Lu Fanghong, Wu Xianping, Wu Xigui, Li Ying, Hu Dongsheng, Huang Jianfeng, Liu Yang, Lu Xiangfeng, Gu Dongfeng
BACKGROUND	Evidence of the effects of long-term fine particulate matter (PM2.5) exposure on cardiovascular diseases (CVDs) is rare for populations exposed to high levels of PM2.5 in China and in other countries with similarly high levels.
OBJECTIVES	The aim of this study was to assess the CVD risks associated with long-term exposure to PM2.5 in China.
METHODS	A nationwide cohort study, China-PAR (Prediction for Atherosclerotic Cardiovascular Disease Risk in China), was used, with 116,972 adults without CVD in 2000 being included. Participants were followed until 2015. Satellite-based PM2.5 concentrations at 1-km spatial resolution during the study period were used for exposure assessment. A Cox proportional hazards model with time-varying exposures was used to estimate the CVD risks associated with PM2.5 exposure, adjusting for individual risk factors.
RESULTS	Annual mean concentrations of PM2.5 at the China-PAR sites ranged from 25.5 to 114.0 μg/m3. For each 10 μg/m3 increase in PM2.5 exposures, the multivariate-adjusted hazard ratio was 1.251 (95% confidence interval: 1.220 to 1.283) for CVD incidence and 1.164 (95% confidence interval: 1.117 to 1.213) for CVD mortality. The slopes of concentration-response functions of PM2.5 exposure and CVD risks were steeper at high PM2.5 levels. In addition, older residents, rural residents, and never smokers were more prone to adverse effects of PM2.5 exposure.
CONCLUSIONS	This study provides evidence that elevated long-term PM2.5 exposures lead to increased CVD risk in China. The effects are more pronounced at higher PM2.5 levels. These findings expand the current knowledge on adverse health effects of severe air pollution and highlight the potential cardiovascular benefits of air quality improvement in China and other low- and middle-income countries.

표-786. PubMed 논문번호 32123898의 내용 요약

구분	내용
PubMed ID	32123898
TITLE	Loss of life expectancy from air pollution compared to other risk factors: a worldwide perspective.
JOURNAL	Cardiovascular research: 10.1093/cvr/cvaa025
AUTHORS	Lelieveld Jos, Pozzer Andrea, P□schl Ulrich, Fnais Mohammed, Haines Andy, M□nzel Thomas
AIMS	Long-term exposure of humans to air pollution enhances the risk of cardiovascular and respiratory diseases. A novel Global Exposure Mortality Model (GEMM) has been derived from many cohort studies, providing much-improved coverage of the exposure to fine particulate matter (PM2.5). We applied the GEMM to assess excess mortality attributable to ambient air pollution on a global scale and compare to other risk factors.
METHODS AND RESULTS	We used a data-informed atmospheric model to calculate worldwide exposure to PM2.5 and ozone pollution, which was combined with the GEMM to estimate disease-specific excess mortality and loss of life expectancy (LLE) in 2015. Using this model, we investigated the effects of different pollution sources, distinguishing between natural (wildfires, aeolian dust) and anthropogenic emissions, including fossil fuel use. Global excess mortality from all ambient air pollution is estimated at 8.8 (7.11-10.41) million/year, with an LLE of 2.9 (2.3-3.5) years, being a factor of two higher than earlier estimates, and exceeding that of tobacco smoking. The global mean mortality rate of about 120 per 100 000 people/year is much exceeded in East Asia (196 per 100 000/year) and Europe (133 per 100 000/year). Without fossil fuel emissions, the global mean life expectancy would increase by 1.1 (0.9-1.2) years and 1.7 (1.4-2.0) years by removing all potentially controllable anthropogenic emissions. Because aeolian dust and wildfire emission control is impracticable, significant LLE is unavoidable.
CONCLUSION	Ambient air pollution is one of the main global health risks, causing significant excess mortality and LLE, especially through cardiovascular diseases. It causes an LLE that rivals that of tobacco smoking. The global mean LLE from air pollution strongly exceeds that by violence (all forms together), i.e. by an order of magnitude (LLE being 2.9 and 0.3 years, respectively).

표-787. PubMed 논문번호 32146266의 내용 요약

구분	내용
PubMed ID	32146266
TITLE	Associations of long-term exposure to ambient PM〈sub〉2.5〈/sub〉 with mortality in Chinese adults: A pooled analysis of cohorts in the China-PAR project.
JOURNAL	Environment international: 10.1016/j.envint.2020.105589
AUTHORS	Yang Xueli, Liang Fengchao, Li Jianxin, Chen Jichun, Liu Fangchao, Huang Keyong, Cao Jie, Chen Shufeng, Xiao Qingyang, Liu Xiaoqing, Shen Chong, Yu Ling, Lu Fanghong, Wu Xianping, Wu Xigui, Li Ying, Zhao Liancheng, Hu Dongsheng, Huang Jianfeng, Lu Xiangfeng, Liu Yang, Gu Dongfeng
BACKGROUND	The concentration-response relationship between mortality and long-term exposure to fine particulate matter (PM2.5) has not been fully elucidated, especially at high levels of PM2.5 concentrations.
OBJECTIVE	We aimed to evaluate chronic effects of ambient PM2.5 exposure on deaths among Chinese adults in high-exposure settings.
METHODS	Participants of the Prediction for Atherosclerotic cardiovascular disease Risk in China (China-PAR) project were included from four prospective cohorts among Chinese adults aged ≥18 years old. The overall follow-up rate of the four cohorts was 93.4% until the recent follow-up survey that ended in 2015. The average of satellite-based PM2.5 concentrations during 2000-2015 at 1-km spatial resolution was assigned to each participant according to individual residence addresses. Based on the pooled analysis of individual data from the four cohorts, a Cox proportional hazards model was used to estimate the hazard ratio (HR) and corresponding 95% confidence intervals (95% CIs) for the association of PM2.5 exposure with mortality after multivariate adjustment.
RESULTS	A total of 116,821 participants were eligible in the final analysis. During a mean of 7.7 years of follow-up, 6,395 non-accidental deaths and 2,507 cardio-metabolic deaths occurred. The mean of PM2.5 concentration was 64.9 μg/m3 ranging from 31.2 μg/m3 to 97.0 μg/m3. For each 10 μg/m3 increment in PM2.5, the HR was 1.11 (95% CI: 1.08-1.14) for non-accidental mortality and 1.22 (95% CI: 1.16-1.27) for cardio-metabolic mortality. In addition, a weak exponential curve for the concentration-response association between mortality and PM2.5 was observed among Chinese adults.
CONCLUSIONS	Our study provided important evidence of the long-term effects of PM2.5 exposure on deaths among Chinese adults. The findings expand our knowledge on concentration-response relationship in high-exposure environments, which is essential to address the urgent challenge of reducing the disease burden attributable to PM2.5 exposure in rapidly industrializing countries such as China.

표-788. PubMed 논문번호 32150674의 내용 요약

구분	내용
PubMed ID	32150674
TITLE	Impact of secondhand smoke exposure in former smokers on their subsequent risk of coronary heart disease: evidence from the population-based cohort of the Tehran Lipid and Glucose Study.
JOURNAL	Epidemiology and health: 10.4178/epih.e2020009
AUTHORS	Sadeghi Masoumeh, Daneshpour Maryam S, Khodakarim Soheila, Momenan Amir Abbas, Akbarzadeh Mahdi, Soori Hamid
OBJECTIVES	Cigarette smoking is an established, strong, and modifiable risk factor for coronary heart disease (CHD). However, little research has investigated CHD risk in former smokers who continue to be exposed to others' cigarette smoke (former & secondhand smokers).
METHODS	In the Tehran Lipid and Glucose Study, a prospective population-based cohort (n=20,069) was followed up for a median period of 14.6 years. A subset of 8,050 participants of 30 years of age and older was analyzed, with first CHD events as the study outcome. Participants were categorized as never, former, current, secondhand, and former & secondhand smokers. Data on smoking intensity (cigarette/d) were also collected. A Cox proportional hazards regression model was applied to estimate the risk of CHD, taking into account the main potential confounders.
RESULTS	The mean age of participants was 46.10 ±11.38 years, and they experienced 1,118 first CHD events (with most CHD cases in former smokers) during the follow-up period. The risk of CHD was highest in current smokers, followed in order by former & secondhand, former, and secondhand smokers (hazard ratio [HR], 1.99; 95% confidence interval [CI], 1.65 to 2.39; HR, 1.55; 95% CI, 1.15 to 2.08; HR, 1.39; 95% CI, 1.12 to 1.72; HR, 1.27; 95% CI, 1.07 to 1.51, respectively), compared to never smokers. The risk of CHD increased with smoking intensity, which has been proposed as a preferable measure of smoking, indicating a dose-response pattern.
CONCLUSIONS	The elevated risk of CHD in former & secondhand smokers was a noteworthy finding, with possible implications for health policy; however, further research is needed.

표-789. PubMed 논문번호 32164596의 내용 요약

구분	내용
PubMed ID	32164596
TITLE	Association between PM〈sub〉2.5〈/sub〉 and risk of hospitalization for myocardial infarction: a systematic review and a meta-analysis.
JOURNAL	BMC public health: 10.1186/s12889-020-8262-3
AUTHORS	Farhadi Zeynab, Abulghasem Gorgi Hasan, Shabaninejad Hosein, Aghajani Delavar Mouloud, Torani Sogand
BACKGROUND	It is generally assumed that there have been mixed results in the literature regarding the association between ambient particulate matter (PM) and myocardial infarction (MI). The aim of this meta-analysis was to explore the rate of short-term exposure PM with aerodynamic diameters ≤2.5 μm (PM2.5) and examine its potential effect(s) on the risk of MI.
METHODS	A systematic search was conducted on databases like PubMed, Scopus, Web of Science, and Embase with components: "air pollution" and "myocardial infarction". The summary relative risk (RR) and 95% confidence intervals (95%CI) were also calculated to assess the association between the PM2.5 and MI.
RESULTS	Twenty-six published studies were ultimately identified as eligible candidates for the meta-analysis of MI until Jun 1, 2018. The results illustrated that a 10- μg/m 3 increase in PM2.5 was associated with the risk of MI (RR = 1.02; 95% CI 1.01-1.03; P ≤ 0.0001). The heterogeneity of the studies was assessed through a random-effects model with p 〈 0.0001 and the I2 was 69.52%, indicating a moderate degree of heterogeneity. We also conducted subgroup analyses including study quality, study design, and study period. Accordingly, it was found that subgroups time series study design and high study period could substantially decrease heterogeneity (I2 = 41.61, 41.78).
CONCLUSIONS	This meta-analysis indicated that exposure - response between PM2.5 and MI. It is vital decision makers implement effective strategies to help improve air pollution, especially in developing countries or prevent exposure to PM2.5 to protect human health.

표-790. PubMed 논문번호 32167232의 내용 요약

구분	내용
PubMed ID	32167232
TITLE	A time-to-event analysis on air pollutants with the risk of cardiovascular disease and mortality: A systematic review and meta-analysis of 84 cohort studies.
JOURNAL	Journal of evidence-based medicine: 10.1111/jebm.12380
AUTHORS	Pranata Raymond, Vania Rachel, Tondas Alexander Edo, Setianto Budhi, Santoso Anwar
OBJECTIVE	Air pollution is one of the most substantial problems globally. Aerodynamic toxic of particulate matter with <10 mm in diameter (PM10), or <2.5 mm (PM2.5), as well as nitric dioxide (NO2), have been linked with health issues. We aimed to perform a comprehensive analysis of the time-to-event for different types of air pollutants on cardiovascular disease (CVD) events based on cohort studies.
METHODS	A comprehensive search on topics that assesses air pollution and cardiovascular disease with keywords up until July 2019 was performed.
RESULTS	There were a total of 28 215 394 subjects from 84 cohorts. Increased PM2.5 was associated with composite CVD [HR 1.10 (1.02, 1.19)], acute coronary events [HR 1.15 (1.12, 1.17)], stroke [HR 1.13 (1.06, 1.19)], and hypertension [HR 1.07 (1.01, 1.14)], all-cause mortality [HR 1.07 (1.04, 1.09)], CVD mortality [HR 1.10 (1.07, 1.12)], and ischemic heart disease (IHD) mortality [HR 1.11 (1.07, 1.16)]. Association with AF became significant after removal of a study. Increased PM10 was associated with heart failure [HR 1.25 (1.04, 1.50)], all-cause mortality [HR 1.16 (1.06, 1.27)], CVD mortality [HR 1.17 (1.04, 1.30)], and IHD mortality [HR 1.03 (1.01, 1.05)]. Increased of NO2 was associated with increased composite CVD [HR 1.15 (1.02, 1.29)], atrial fibrillation [HR 1.01 (1.01, 1.02)], acute coronary events [HR 1.08 (1.02, 1.13)], all-cause mortality [HR 1.23 (1.14, 1.32)], CVD mortality [HR 1.17 (1.10, 1.25)], and IHD mortality [HR 1.05 (1.03, 1.08)].
CONCLUSION	Air pollutants are associated with an increased incidence of cardiovascular diseases, all-cause mortality, and CVD mortality.

표-791. PubMed 논문번호 32172126의 내용 요약

구분	내용
PubMed ID	32172126
TITLE	Short-term exposure to ambient particle gamma radioactivity is associated with increased risk for all-cause non-accidental and cardiovascular mortality.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2020.137793
AUTHORS	Huang Shaodan, Xiong Jianyin, Vieira Carolina L Z, Blomberg Annelise J, Gold Diane R, Coull Brent A, Sarosiek Kristopher, Schwartz Joel D, Wolfson Jack M, Li Jing, Koutrakis Petros
BACKGROUND	Recent studies have found that particulate matter (PM) attached radioactivity was associated with certain adverse health effects including increased blood pressure and lung dysfunction. However, there has been no investigation on the direct effect of PM radioactivity on mortality.
METHODS	Exposures to ambient PM gamma activities were determined using U.S. EPA RadNet data. Data on daily deaths were obtained from individual state Departments of Public Health. We used a generalized additive quasi-Poisson model to estimate the associations between two-day average ambient PM gamma activities (gamma2 through gamma9) with all-cause non-accidental and cardiovascular daily deaths for each of 18 US cities, for each season, adjusting for two-day average PM2.5 exposure, temperature, relative humidity, day of week and long-term trends. Subsequently, we used random-effects meta-analysis to estimate the overall effect in the 18 cities for each season.
RESULTS	We found that all-cause non-accidental daily mortality in spring season was positively associated with two-day average ambient PM gamma activities in spring, with significant results for gamma2, gamma5 and gamma6. Similarly, cardiovascular daily mortality was positively associated with two-day average ambient PM gamma activities, with significant results for gamma2, gamma4, gamma5, gamma6, gamma7 and gamma9. For the spring season, each interquartile range (IQR) increase of two-day averaged ambient PM gamma activity was associated with increase in all-cause daily deaths, ranging from 0.15% (95% Confidence Interval (CI): -0.36%, 0.65%) to 1.03 (95%CI: 0.18%, 1.89%). Each IQR was also associated with increase in cardiovascular daily deaths, ranging from 0.01% (95%CI: -0.89, 0.92) to 2.95% (95%CI: 1.33, 4.59). For other seasons overall we found statistically insignificant associations of PM radioactivity with mortality.
CONCLUSIONS	Our findings suggest that there are potential systemic toxic effects of inhalation of radionuclides attached to ambient air particles.

표-792. PubMed 논문번호 32193046의 내용 요약

구분	내용
PubMed ID	32193046
TITLE	Prenatal air pollution exposure and growth and cardio-metabolic risk in preschoolers.
JOURNAL	Environment international: 10.1016/j.envint.2020.105619
AUTHORS	Fossati Serena, Valvi Damaskini, Martinez David, Cirach Marta, Estarlich Marisa, Fern□ndez-Somoano Ana, Guxens M□nica, I□iguez Carmen, Irizar Amaia, Lertxundi Aitana, Nieuwenhuijsen Mark, Tamayo Ibon, Vioque Jesus, Tard□n Adonina, Sunyer Jordi, Vrijheid Martine
OBJECTIVES	We investigated the association between outdoor air pollutants exposure in the first trimester of pregnancy, and growth and cardio-metabolic risk at four years of age, and evaluated the mediating role of birth weight.
METHODS	We included mother-child pairs (N = 1,724) from the Spanish INMA birth cohort established in 2003-2008. First trimester of pregnancy nitrogen dioxide (NO2) and fine particles (PM2.5) exposure levels were estimated. Height, weight, waist circumference, blood pressure, and lipids were measured at four years of age. Body mass index (BMI) trajectories from birth to four years were identified.
RESULTS	Increased PM2.5 exposure in the first trimester of pregnancy was associated with decreased z-scores of weight (zWeight) and BMI (zBMI) (zWeight change per interquartile range increase in PM2.5 exposure = -0.12; 95% CI: -0.23, -0.01; zBMI change = -0.12; 95% CI: -0.23, -0.01). Higher NO2 and PM2.5 exposure was associated to a reduced risk of being in a trajectory with accelerated BMI gain, compared to children with the average trajectory. Birth weight partially mediated the association between PM2.5 and zWeight and zBMI. PM2.5 and NO2 were not associated with the other cardio-metabolic risk factors.
CONCLUSIONS	This comprehensive study of many growth and cardio-metabolic risk related outcomes suggests that air pollution exposure during pregnancy may be associated with delays in physical growth in the early years after birth. These findings imply that pregnancy exposure to air pollutants has a lasting effect on growth after birth and require follow-up at later child ages.

표-793. PubMed 논문번호 32211302의 내용 요약

구분	내용
PubMed ID	32211302
TITLE	Exposure to Wood Smoke and Associated Health Effects in Sub-Saharan Africa: A Systematic Review.
JOURNAL	Annals of global health: 10.5334/aogh.2725
AUTHORS	Bede-Ojimadu Onyinyechi, Orisakwe Orish Ebere
Background	Observational studies suggest that exposure to wood smoke is associated with a variety of adverse health effects in humans.
Objective	We aimed to summarise evidence from sub-Saharan Africa on levels of exposure to pollutants in wood smoke and the association between such exposures and adverse health outcomes.
Methods	PubMed and Google scholar databases were searched for original articles reporting personal exposure levels to pollutants or health outcomes associated with wood smoke exposure in Sub-Saharan African population.
Results	Mean personal PM2.5 and carbon monoxide levels in the studies ranged from 26.3 ± 1.48 μg/m3 to 1574 ± 287 μg/m3 and from 0.64 ± 2.12 ppm to 22 ± 2.4 ppm, respectively. All the reported personal PM2.5 exposure levels were higher than the World Health Organization's Air Quality Guideline (AQG) for 24-hour mean exposure. Use of wood fuels in domestic cooking is the major source of wood smoke exposure in this population. Occupational exposure to wood smoke included the use of wood fuels in bakery, fish drying, cassava processing and charcoal production. Females were exposed to higher levels of these pollutants than males of the same age range. Major determinants for higher exposure to wood smoke in SSA included use of unprocessed firewood, female gender and occupational exposure. We recorded strong and consistent associations between exposure to wood smoke and respiratory diseases including acute respiratory illness and impaired lung function. Positive associations were reported for increased blood pressure, low birth weight, oesophageal cancer, sick building syndrome, non-syndromic cleft lip and/or cleft palate and under-five mortality.
Conclusion	There is high level of exposure to wood smoke in SSA and this exposure is associated with a number of adverse health effects. There is urgent need for aggressive programs to reduce wood smoke exposure in this population.

표-794. PubMed 논문번호 32217384의 내용 요약

구분	내용
PubMed ID	32217384
TITLE	Comparative assessments of mortality from and morbidity of circulatory diseases in association with extreme temperatures.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2020.138012
AUTHORS	Lin Yu-Kai, Sung Fung-Chang, Honda Yasushi, Chen Yi-Jhih, Wang Yu-Chun
BACKGROUND	This study evaluated vulnerable subpopulation on mortality, emergency room visits (ERVs) and outpatient visits associated with ambient daily temperature from 2000 to 2014 using vital statistics and insurance claims of Taiwan.
METHODS	We used the distributed lag non-linear model to assess circulatory disease-specific deaths, ERVs, and outpatient visits by mean temperature after controlling particulate matter (PM10) and other covariates. Lag effect of temperature changes on health risks accumulated for 0-10 days associated with low temperature and for 0-5 days for high temperature were evaluated. Cause-specific pooled relative risk (RR) and 95% confidence intervals (CI) were estimated for the whole population of Taiwan using random-effects meta-analysis.
RESULTS	We used reference temperatures of 60th percentiles for mortality from circulatory diseases, 99th percentile for ERVs of circulatory diseases, 2nd percentile for ERVs of heart diseases and ischemic heart disease, 53th percentile for ERVs of cerebrovascular disease, and 12-16th percentiles for outpatient visits of circulatory diseases. The lag effects peaked at lag 4-5 day for low temperature exposure and at lag 0 for high temperature exposure. Pooled cold related health risk was the highest for mortality from and ERV of circulatory diseases with RR of 1.41 (95% CI: 1.34, 1.49) and 1.41 (95% CI: 1.35, 1.48), respectively, as daily mean temperatures was at 1st percentile (12.8 ° C). Heat related health risk was significant for mortality from heart diseases [RR = 1.12 (95% CI: 1.07, 1.18)] and ischemic heart diseases [RR = 1.13 (95% CI: 1.06, 1.20)] as daily mean temperatures was at 99th percentile (29.9 ° C).
CONCLUSIONS	Health authority should evaluate the effectiveness of adaptive policy, strategy, and actions responding to extreme temperatures to prevent mortality from circulatory diseases.

표-795. PubMed 논문번호 32227140의 내용 요약

구분	내용
PubMed ID	32227140
TITLE	Association Between Cardiovascular Disease and Long-term Exposure to Air Pollution With the Risk of Dementia.
JOURNAL	JAMA neurology: 10.1001/jamaneurol.2019.4914
AUTHORS	Grande Giulia, Ljungman Petter L S, Eneroth Kristina, Bellander Tom, Rizzuto Debora
Importance	Emerging yet contrasting evidence associates air pollution with incident dementia, and the potential role of cardiovascular disease (CVD) in this association is unclear.
Objective	To investigate the association between long-term exposure to air pollution and dementia and to assess the role of CVD in that association.
Design, Setting, and Participants	Data for this cohort study were extracted from the ongoing Swedish National Study on Aging and Care in Kungsholmen (SNAC-K), a longitudinal population-based study with baseline assessments from March 21, 2001, through August 30, 2004. Of the 5111 randomly selected residents in the Kungsholmen district of Stockholm 60 years or older and living at home or in institutions, 521 were not eligible (eg, due to death before the start of the study or no contact information). Among the remaining 4590 individuals, 3363 (73.3%) were assessed. For the current analysis, 2927 participants who did not have dementia at baseline were examined, with follow-up to 2013 (mean [SD] follow-up time, 6.01 [2.56] years). Follow-up was completed February 18, 2013, and data were analyzed from June 26, 2018, through June 20, 2019.
Exposures	Two major air pollutants (particulate matter ≤2.5 μm [PM2.5] and nitrogen oxide [NOx]) were assessed yearly from 1990, using dispersion models for outdoor levels at residential addresses.
Main Outcomes and Measures	The hazard of dementia was estimated using Cox proportional hazards regression models. The potential of CVD (ie, atrial fibrillation, ischemic heart disease, heart failure, and stroke) to modify and mediate the association between long-term exposure to air pollution and dementia was tested using stratified analyses and generalized structural equation modeling.
Results	At baseline, the mean (SD) age of the 2927 participants was 74.1 (10.7) years, and 1845 (63.0%) were female. Three hundred sixty-four participants with incident dementia were identified. The hazard of dementia increased by as much as 50% per interquartile range difference in mean pollutant levels during the previous 5 years at the residential address (hazard ratio [HR] for difference of 0.88 μg/m3 PM2.5, 1.54 [95% CI, 1.33-1.78]; HR for difference of 8.35 μg/m3 NOx, 1.14 [95% CI, 1.01-1.29]). Heart failure (HR for PM2.5, 1.93 [95% CI, 1.54-2.43]; HR for NOx, 1.43 [95% CI, 1.17-1.75]) and ischemic heart disease (HR for PM2.5, 1.67 [95% CI, 1.32-2.12]; HR for NOx, 1.36 [95% CI, 1.07-1.71]) enhanced the dementia risk, whereas stroke appeared to be the most important intermediate condition, explaining 49.4% of air pollution-related dementia cases.
Conclusions and Relevance	This study found that long-term exposure to air pollution was associated with a higher risk of dementia. Heart failure and ischemic heart disease appeared to enhance the association between air pollution and dementia, whereas stroke seemed to be an important intermediate condition between the association of air pollution exposure with dementia.

표-796. PubMed 논문번호 32243204의 내용 요약

구분	내용
PubMed ID	32243204
TITLE	Land-Use Change and Cardiometabolic Risk Factors in an Urbanizing Area of South India: A Population-Based Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP5445
AUTHORS	Mil□ Carles, Ranzani Otavio, Sanchez Margaux, Ambr□s Albert, Bhogadi Santhi, Kinra Sanjay, Kogevinas Manolis, Dadvand Payam, Tonne Cathryn
BACKGROUND	Land-use changes in city fringes due to urbanization can lead to a reduction of greenspace that may reduce its associated health benefits.
OBJECTIVES	We evaluated the association between changes in residential surrounding built-up land use and cardiometabolic risk factors in an urbanizing peri-urban area of south India and explored the mediating roles of air pollution, physical activity, and stress in these associations.
METHODS	We analyzed data on 6,039 adults from the third follow-up of the Andhra Pradesh Children and Parent Study (APCAPS) cohort (2010-2012). We generated trajectories of change in residential surrounding built-up land use (buffer areas) from 1995-2009 (stable, slow increase, fast increase) using remote sensing data and image classification methods. We estimated associations between built-up land use trajectories and natural log-transformed blood pressure, waist circumference, triglycerides, fasting glucose, and non-high-density lipoprotein (non-HDL) cholesterol using linear mixed models. We accounted for multiple mediators and the multilevel structure of the data in mediation analyses.
RESULTS	We observed positive associations between a fast increase in built-up land use within 300m of the home and all cardiometabolic risk factors. Compared with participants with stable trajectories, those with the largest increase in built-up land use had 1.5% (95% CI: 0.1, 2.9) higher systolic blood pressure, 2.4% (95% CI: 0.6, 4.3) higher diastolic blood pressure, 2.1% (95% CI: 0.5, 3.8) higher waist circumference, and 1.6% (95% CI: −0.6, 3.8) higher fasting glucose in fully adjusted models. Associations were positive, but not statistically significant, for triglycerides, fasting glucose, and non-HDL cholesterol. Physical activity and ambient particulate matter $\leq 2.5\,\mu$m in aerodynamic diameter (PM2.5) partially mediated the estimated associations. Associations between fast build-up and all cardiometabolic risk factors except non-HDL cholesterol were stronger in women than men.
DISCUSSION	Increases in built-up land use surrounding residences were consistently associated with higher levels of cardiometabolic risk factors. Our findings support the need for better integration of health considerations in urban planning in rapidly urbanizing settings. https://doi.org/10.1289/EHP5445.

표-797. PubMed 논문번호 32244108의 내용 요약

구분	내용
PubMed ID	32244108
TITLE	Quiet, clean, green, and active: A Navigation Guide systematic review of the impacts of spatially correlated urban exposures on a range of physical health outcomes.
JOURNAL	Environmental research: 10.1016/j.envres.2020.109388
AUTHORS	Rugel Emily Jessica, Brauer Michael
BACKGROUND	Recent epidemiologic analyses have considered impacts of multiple spatially correlated urban exposures, but this literature has not been systematically evaluated.
OBJECTIVES	To characterize the long-term impacts of four distinct spatially correlated urban environmental exposures - traffic-related air pollution (TRAP), noise, natural spaces, and neighborhood walkability - by evaluating studies including measures of at least two such exposures in relationship to mortality, cardiovascular disease, chronic respiratory disease, allergy, type 2 diabetes, or reproductive outcomes.
METHODS	Following the Navigation Guide framework, the literature was searched for studies published since 2003 and meeting predefined inclusion criteria. Identified studies were scored individually for risk of bias and all studies related to an exposure-group set were appraised for overall quality and strength of evidence.
RESULTS	A total of 51 individual studies (TRAP and noise: n = 29; TRAP and natural spaces: n = 10; noise and natural spaces: n = 2; TRAP, noise, and natural spaces: n = 7; TRAP, noise, natural spaces, and walkability: n = 3) were included. When TRAP and noise were considered jointly, evidence was sufficient for increased cardiovascular morbidity with higher noise exposures; sufficient for no effect of TRAP on CVD morbidity; sufficient for increased mortality with higher TRAP exposures, but limited for noise; and limited for increased adverse reproductive outcomes with higher TRAP exposures and no effect of noise. Looking at natural spaces and TRAP, there was limited evidence for lower risk of chronic respiratory disease and small increases in birthweight with greater natural space; this relationship with birthweight persisted after adjustment for noise as well. Evidence was inadequate for all other exposure groups and outcomes.
DISCUSSION	Studies that properly account for the complexity of relationships between urban form and physical health are limited but suggest that even highly correlated exposures may have distinct effects.
REVIEW REGISTRATION	PROSPERO 2018 CRD42018106050.

표-798. PubMed 논문번호 32276169의 내용 요약

구분	내용
PubMed ID	32276169
TITLE	Association between ambient air pollution and pregnancy complications: A systematic review and meta-analysis of cohort studies.
JOURNAL	Environmental research: 10.1016/j.envres.2020.109471
AUTHORS	Bai Wei, Li Yuanyuan, Niu Yaling, Ding Ye, Yu Xiao, Zhu Bo, Duan Ruixin, Duan Huawei, Kou Changgui, Li Yanbo, Sun Zhiwei
BACKGROUND	Pregnancy complications, such as gestational diabetes mellitus (GDM) and hypertensive disorders of pregnancy (HDP), have a great impact on public health. Exposure to ambient air pollution during pregnancy may cause pregnancy complications. The aim of our study is to explore the risk of trimester-specific maternal exposure to air pollutants on complications of pregnancy.
METHODS	PubMed, EMBASE, Web of Science, and Cochrane were systematically searched for cohort studies published before October 27, 2019 which reported the association between ambient air pollutants (PM2.5, PM10, CO, NO, NO2, NOx, O3, and SO2) and pregnancy complications (GDM, HDP, preeclampsia, and gestational hypertension) during different exposure windows. A meta-analysis was applied to combine relative risks (RRs) and their confidence intervals (CIs) from eligible studies. Quality assessment was conducted and Egger test was used to evaluate the publication bias. All statistical analyses were performed by STATA software (Version 15, StataCorp, College Station, Texas, USA).
RESULTS	This meta-analysis consisted of 33 cohort studies conducted on 22,253,277 pregnant women. Meta-analyses showed during the first trimester, there were significant associations of PM10 with gestational hypertension (RR = 1.07, 95% CI: 1.02-1.12 per 10 μg/m3, I2 = 0.0%), of SO2 with GDM (RR = 1.04, 95% CI: 1.00-1.08 per 1 ppb increment, I2 = 54.1%), of PM2.5 with preeclampsia (RR = 0.97, 95% CI: 0.95-1.00 per 5 μg/m3, I2 = 4.1%). During the entire pregnancy, PM2.5 significantly increased the risk of hypertensive disorders of pregnancy (RR = 1.18, 95% CI: 1.02-1.34 per 5 μg/m3, I2 = 85.1%). Egger test indicated that wide-scale publication bias was unlikely.
CONCLUSION	Maternal exposure to ambient air pollutants is associated with pregnancy complications especially during the first trimester. Further large multicenter cohort studies considering different constituents of pollutants, levels of disease severity, sensitive populations, and various exposure windows are warranted in the future research.

표-799. PubMed 논문번호 32278195의 내용 요약

구분	내용
PubMed ID	32278195
TITLE	Cardiorespiratory responses to fine particles during ambient PM〈sub〉2.5〈/sub〉 pollution waves: Findings from a randomized crossover trial in young healthy adults.
JOURNAL	Environment international: 10.1016/j.envint.2020.105590
AUTHORS	Zhao Yan, Xue Lijun, Chen Qiao, Kou Minghao, Wang Zemin, Wu Shaowei, Huang Jing, Guo Xinbiao
BACKGROUND	PM2.5 pollution waves (PPWs) are severe air pollution events with extremely high-level concentration of ambient PM2.5. PPWs, such as haze days, were suggested to be associated with increased cardiopulmonary mortality and morbidity. However, the biological mechanism response to ambient PM2.5 during PPWs is still unclear.
METHODS	A randomized crossover trial was conducted on 29 healthy young adults. Repeated health measurements were performed before, during and after two typical PPWs under filtered and sham indoor air purification, with a washout interval of at least 2 weeks. Health parameters including blood pressure (BP), pulmonary function, fractional exhaled nitric oxide (FeNO) and circulating biomarkers which reflect platelet activation, blood coagulation and systematic oxidative stress were measured.
RESULTS	Ambient PM2.5 levels elevated apparently during PPWs. Under sham purification, significant increase in FeNO and soluble P-selectin (sP-selectin) and decreases in pulmonary function were observed from pre-PPWs period to during-PPWs period. The changes in health biomarkers as mentioned above became attenuated and insignificant under filtered condition. For instance, sP-selectin increased by 12.0% (95% CI: 3.8%, 20.8%) during-PPWs periods compared with pre-PPWs periods under sham purification, while non-significant change was observed under filtered condition. Significant associations between time-weighted personal PM2.5 exposure and increased levels of health biomarkers including FeNO, sP-selectin, oxidized low-density lipoprotein (ox-LDL) and 8-isoprostane (8-isoPGF2α) were found.
CONCLUSION	PPWs could affect cardiopulmonary health through systematic oxidative stress, platelet activation and respiratory inflammation in healthy adults, and short-term indoor air purification could alleviate the adverse cardiopulmonary effects.

표-800. PubMed 논문번호 32278917의 내용 요약

구분	내용
PubMed ID	32278917
TITLE	Maternal air pollution exposure and congenital heart defects in offspring: A systematic review and meta-analysis.
JOURNAL	Chemosphere: 10.1016/j.chemosphere.2020.126668
AUTHORS	Hu Cheng-Yang, Huang Kai, Fang Yuan, Yang Xiao-Jing, Ding Kun, Jiang Wen, Hua Xiao-Guo, Huang Da-Yan, Jiang Zheng-Xuan, Zhang Xiu-Jun
BACKGROUND	Congenital heart defects (CHDs) has a multifactorial causation with a strong genetic component and many environmental triggers. Emerging body of empirical studies suggest that air pollution is an important contributor to the development of CHDs, however, there still remains some controversy over the current evidence, and to the authors' knowledge, no studies have reviewed the most recent evidence.
OBJECTIVES	We performed a systematic review and meta-analysis of epidemiological literature to investigate the relationship between maternal air pollution exposure and CHDs risk in offspring. The presence of heterogeneity and publication bias across available studies were also examined.
METHODS	An extensive literature search of epidemiological studies pertaining to air pollution and CHDs, published in English language, until August 1, 2019 was conducted. Summary risk estimates of pollution-outcome combinations were calculated for i) risk per specific increment of concentration and ii) risk at high versus low exposure level in each study using fixed-effect model or random-effects model.
RESULTS	A total of 26 studies were finally included. In the meta-analyses, high versus low carbon monoxide (CO) exposure was associated with an increased risk of tetralogy of Fallot [odds ratio (OR) = 1.21, 95% confidence interval (CI): 1.04-1.41], yet particulate matter ≤ 5 μm (PM2.5) exposure was marginally associated with it. Increased risk of atrial septal defects (ASDs) was found for each 10 μg/m3 and 10 ppb increment in particulate matter ≤ 10 μm (PM10) and ozone (O3) exposure, respectively (OR = 1.04, 95% CI: 1.00-1.09; OR = 1.09, 95% CI: 1.02-1.17). Categorical nitrogen dioxide (NO2) exposure was associated with an increased risk of coarctation of the aorta (OR for high versus low = 1.14, 95% CI: 1.02-1.26). Analyses for other combinations yielded none statistically significant associations. Sensitive analyses showed similar findings.
CONCLUSIONS	The summary effect estimates from this study suggest statistically significant associations between increased risk of specific CHDs subtypes and PM2.5, PM10, NO2, CO, and O3 exposures. Further studies, especially conducted in developing countries, with improvements in exposure assessing, outcome harmonizing, and mechanistic understanding are needed to elaborate the suggestive associations.

표-801. PubMed 논문번호 32282436의 내용 요약

구분	내용
PubMed ID	32282436
TITLE	The Role of Ambient Particle Radioactivity in Inflammation and Endothelial Function in an Elderly Cohort.
JOURNAL	Epidemiology (Cambridge, Mass.): 10.1097/EDE.0000000000001197
AUTHORS	Blomberg Annelise J, Nyhan Marguerite M, Bind Marie-Ab□le, Vokonas Pantel, Coull Brent A, Schwartz Joel, Koutrakis Petros
BACKGROUND	The mechanisms by which exposure to particulate matter might increase risk of cardiovascular morbidity and mortality are not fully known. However, few existing studies have investigated the potential role of particle radioactivity. Naturally occurring radionuclides attach to particulate matter and continue to release ionizing radiation after inhalation and deposition in the lungs. We hypothesize that exposure to particle radioactivity increases biomarkers of inflammation.
METHODS	Our repeated-measures study included 752 men in the greater Boston area. We estimated regional particle radioactivity as a daily spatial average of gross beta concentrations from five monitors in the study area. We used linear mixed-effects regression models to estimate short- and medium-term associations between particle radioactivity and biomarkers of inflammation and endothelial dysfunction, with and without adjustment for additional particulate air pollutants.
RESULTS	We observed associations between particle radioactivity on C-reactive protein (CRP), intercellular adhesion molecule-1 (ICAM-1), and vascular cell adhesion molecule-1 (VCAM-1), but no associations with fibrinogen. An interquartile range width increase in mean 7-day particle radioactivity (1.2 × 10 Bq/m) was associated with a 4.9% increase in CRP (95% CI = 0.077, 9.9), a 2.8% increase in ICAM-1 (95% CI = 1.4, 4.2), and a 4.3% increase in VCAM-1 (95% CI = 2.5, 6.1). The main effects of particle radioactivity remained similar after adjustment in most cases. We also obtained similar effect estimates in a sensitivity analysis applying a robust causal model.
CONCLUSION	Regional particle radioactivity is positively associated with inflammatory biomarkers, indicating a potential pathway for radiation-induced cardiovascular effects.

표-802. PubMed 논문번호 32301991의 내용 요약

구분	내용
PubMed ID	32301991
TITLE	Association of Fine Particulate Matter Exposure With Bystander-Witnessed Out-of-Hospital Cardiac Arrest of Cardiac Origin in Japan.
JOURNAL	JAMA network open: 10.1001/jamanetworkopen.2020.3043
AUTHORS	Kojima Sunao, Michikawa Takehiro, Matsui Kunihiko, Ogawa Hisao, Yamazaki Shin, Nitta Hiroshi, Takami Akinori, Ueda Kayo, Tahara Yoshio, Yonemoto Naohiro, Nonogi Hiroshi, Nagao Ken, Ikeda Takanori, Sato Naoki, Tsutsui Hiroyuki
Importance	Out-of-hospital cardiac arrests (OHCAs) are a major public health concern and a leading cause of death worldwide. Exposure to ambient air pollution is associated with increases in morbidity and mortality and has been recognized as a leading contributor to global disease burden.
Objective	To examine the association between short-term exposure to particulate matter with a diameter of 2.5 μm or smaller (PM2.5) and the incidence of OHCAs of cardiac origin and with the development of initial cardiac arrest rhythm.
Design, Setting, and Participants	This case-control study used data from cases registered between January 1, 2005, and December 31, 2016, in the All-Japan Utstein Registry, a prospective, nationwide, population-based database for OHCAs across all 47 Japanese prefectures. These OHCA cases included patients who had bystander-witnessed OHCAs and for whom emergency medical services responders initiated resuscitation before hospital transfer. A case-crossover design was employed for the study analyses. A prefecture-specific, conditional logistic regression model to estimate odds ratios was applied, and a random-effects meta-analysis was used to obtain prefecture-specific pooled estimates. All analyses were performed from May 7, 2019, to January 23, 2020.
Main Outcomes and Measures	The main outcome was the association of short-term PM2.5 exposure with the incidence of bystander-witnessed OHCAs of cardiac origin. The differences in the distribution of initial cardiac arrest rhythm in OHCAs among those with exposure to PM2.5 were also examined.
Results	In total, 103 189 OHCAs witnessed by bystanders were included in the final analysis. Among the patients who experienced such OHCAs, the mean (SD) age was 75 (15.5) years, and 62 795 (60.9%) were men. Point estimates of the percentage increase for a 10- μg/m3 increase in PM2.5 at lag0-1 (difference in mean PM2.5 concentrations measured on the case day and 1 day before) demonstrated a statistically significantly higher incidence of OHCA across most of the 47 prefectures, without significant heterogeneity (I2 = 20.1%; P = .12). A stratified analysis found an association between PM2.5 exposure and OHCAs (% increase, 1.6; 95% CI, 0.1%-3.1%). An initial shockable rhythm, such as ventricular fibrillation or pulseless ventricular tachycardia (% increase, 0.6; 95% CI, -2.0% to 3.2%), was not associated with PM2.5 exposure. However, an initial nonshockable rhythm, such as pulseless electrical activity and asystole, was associated with PM2.5 exposure (% increase, 1.4; 95% CI, 0.1%-2.7%).
Conclusions and Relevance	Findings from this study suggest that increased PM2.5 concentration is associated with bystander-witnessed OHCA of cardiac origin that commonly presents with nonshockable rhythm. The results support measures to reduce PM2.5 exposure to prevent OHCAs of cardiac origin.

표-803. PubMed 논문번호 32334122의 내용 요약

구분	내용
PubMed ID	32334122
TITLE	Artificial intelligence-assisted analysis on the association between exposure to ambient fine particulate matter and incidence of arrhythmias in outpatients of Shanghai community hospitals.
JOURNAL	Environment international: 10.1016/j.envint.2020.105745
AUTHORS	Yang Mei, Zhou Runze, Qiu Xiangjun, Feng Xiangfei, Sun Jian, Wang Qunshan, Lu Qiufen, Zhang Pengpai, Liu Bo, Li Wei, Chen Mu, Zhao Yan, Mo Binfeng, Zhou Xin, Zhang Xi, Hua Yingxue, Guo Jin, Bi Fangfang, Cao Yajun, Ling Feng, Shi Shengming, Li Yi-Gang
BACKGROUND	Recently, the impact of fine particulate matter pollution on cardiovascular system is drawing considerable concern worldwide. The association between ambient fine particulate and the cardiac arrhythmias is not clear now.
OBJECTIVE	To study associations of ambient fine particulate with incidence of arrhythmias in outpatients.
METHODS	Data was collected from the remote electrocardiogram (ECG) system covering 282 community hospitals in Shanghai from June 24th, 2014 to June 23rd, 2016. ECG was performed for patients admitted to above hospitals with complaining of chest discomfort or palpitation, or for regular check-ups. Air quality data during this time period was obtained from China National Environment Monitoring Center. A generalized additive quasi-Poisson model was established to examine the associations between PM2.5 and cardiac arrhythmias.
RESULTS	Cardiac arrhythmias were detected in 202,661 out of 1,016,579 outpatients (19.9%) and fine particulate matter ranged from 6 to 219 μg/m3 during this period. Positive associations were evidenced between fine particulate matter level and prevalence of cardiac arrhythmia by different lag models. Per 10 μg/m3 increase in fine particulate matter was associated with a 0.584%(95%CI:0.346-0.689%, $p < 0.001$) increase of cardiac arrhythmia detected in these patient cohort at lag0-2. For different types of cardiac arrhythmias, an immediate arrhythmogenic effect of fine particulate matter (increase of the estimates of cardiac arrhythmia prevalence detected in daily outpatient visits) was found with paroxysmal supraventricular tachycardia; a lag effect was found with atrial fibrillation; and both immediate and lag effect was found with premature atrial contractions or atrial tachycardia, atrioventricular block. Moreover, the impact of fine particulate matter on cardiac arrhythmias was significantly greater in women (lag3 and lag0-4), and in people aged <65 years (lag0).
CONCLUSION	Ambient exposure to fine particulate matter is linked with increased risk of arrhythmias in outpatients visiting Shanghai community hospitals, with an immediate or lag effect. The arrhythmogenic effect varies among different types of cardiac arrhythmias.

표-804. PubMed 논문번호 32337473의 내용 요약

구분	내용
PubMed ID	32337473
TITLE	Fine particulate matter exposure and lipid levels among children in Mexico city.
JOURNAL	Environmental epidemiology (Philadelphia, Pa.): 10.1097/EE9.0000000000000088
AUTHORS	McGuinn Laura A, Coull Brent A, Kloog Itai, Just Allan C, Tamayo-Ortiz Marcela, Osorio-Y□□ez Citlalli, Baccarelli Andrea A, Wright Rosalind J, T□llez-Rojo Martha M, Wright Robert O
Background	Studies have identified associations between air pollution and lipid levels in adults, suggesting a mechanism by which air pollution contributes to cardiovascular disease. However, little is known about the association between early life air pollution exposure and lipid levels in children.
Methods	Participants included 465 mother-child pairs from a prospective birth cohort in Mexico City. Daily particulate matter <2.5 □m in diameter (PM2.5) predictions were estimated using a satellite-based exposure model and averaged over trimesters, the entire pregnancy, and the first year of life. We assessed associations with several lipid measures at 4-6 years of age, including total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), non-HDL-C, high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). Linear regression models were used to estimate change in lipid levels with each interquartile range increase in PM2.5. We additionally assessed if associations between PM2.5 and lipid levels varied across lipid quantiles using quantile regression. Models were adjusted for maternal education, body mass index, and age, child's age at study visit, prenatal environmental tobacco smoke, and season of conception.
Results	PM2.5 exposure during the third trimester was associated with increases in childhood total cholesterol, LDL-C, and non-HDL-C, and decreases in HDL-C and triglycerides. There was additionally an increasing trend in the effect estimate across higher quantiles of total cholesterol, LDL-C, and non-HDL-C during the third trimester and entire pregnancy period. There were no consistent associations for first year of life exposures.
Conclusion	In this longitudinal birth cohort in Mexico City, associations between prenatal PM2.5 and childhood lipid (total cholesterol, LDL-C, non-HDL-C) levels were greater for children at higher lipid quantiles.

표-805. PubMed 논문번호 32357880의 내용 요약

구분	내용
PubMed ID	32357880
TITLE	Machine learning approaches to predict peak demand days of cardiovascular admissions considering environmental exposure.
JOURNAL	BMC medical informatics and decision making: 10.1186/s12911-020-1101-8
AUTHORS	Qiu Hang, Luo Lin, Su Ziqi, Zhou Li, Wang Liya, Chen Yucheng
BACKGROUND	Accumulating evidence has linked environmental exposure, such as ambient air pollution and meteorological factors, to the development and severity of cardiovascular diseases (CVDs), resulting in increased healthcare demand. Effective prediction of demand for healthcare services, particularly those associated with peak events of CVDs, can be useful in optimizing the allocation of medical resources. However, few studies have attempted to adopt machine learning approaches with excellent predictive abilities to forecast the healthcare demand for CVDs. This study aims to develop and compare several machine learning models in predicting the peak demand days of CVDs admissions using the hospital admissions data, air quality data and meteorological data in Chengdu, China from 2015 to 2017.
METHODS	Six machine learning algorithms, including logistic regression (LR), support vector machine (SVM), artificial neural network (ANN), random forest (RF), extreme gradient boosting (XGBoost), and light gradient boosting machine (LightGBM) were applied to build the predictive models with a unique feature set. The area under a receiver operating characteristic curve (AUC), logarithmic loss function, accuracy, sensitivity, specificity, precision, and F1 score were used to evaluate the predictive performances of the six models.
RESULTS	The LightGBM model exhibited the highest AUC (0.940, 95% CI: 0.900-0.980), which was significantly higher than that of LR (0.842, 95% CI: 0.783-0.901), SVM (0.834, 95% CI: 0.774-0.894) and ANN (0.890, 95% CI: 0.836-0.944), but did not differ significantly from that of RF (0.926, 95% CI: 0.879-0.974) and XGBoost (0.930, 95% CI: 0.878-0.982). In addition, the LightGBM has the optimal logarithmic loss function (0.218), accuracy (91.3%), specificity (94.1%), precision (0.695), and F1 score (0.725). Feature importance identification indicated that the contribution rate of meteorological conditions and air pollutants for the prediction was 32 and 43%, respectively.
CONCLUSION	This study suggests that ensemble learning models, especially the LightGBM model, can be used to effectively predict the peak events of CVDs admissions, and therefore could be a very useful decision-making tool for medical resource management.

표-806. PubMed 논문번호 32357932의 내용 요약

구분	내용
PubMed ID	32357932
TITLE	Association of fine particulate matter exposure with acute noncardiovascular critical illnesses and in-hospital outcomes in patients receiving intensive cardiac care.
JOURNAL	BMC public health: 10.1186/s12889-020-08758-7
AUTHORS	Chen Fei, Liu Qi, Huang Baotao, Huang Fangyang, Li Yiming, Peng Yong, Chen Mao
BACKGROUND	The effect of short-term exposure to fine particulate matter (PM2.5) on the incidence of acute noncardiovascular critical illnesses (ANCIs) and clinical outcomes is unknown in patients with acute cardiovascular diseases.
METHODS	We conducted a retrospective study in 2337 admissions to an intensive cardiac care unit (ICCU) from June 2016 to May 2017. We used the 2-day average PM2.5 concentration before ICCU admission to estimate the individual exposure level, and patients were divided into 3 groups according to the concentration tertiles. Major ANCI was defined as the composite of acute respiratory failure, acute kidney injury, gastrointestinal hemorrhage, or sepsis. The primary endpoint was all-cause death or discharge against medical advice in extremely critical condition.
RESULTS	During the 12-month study period, the annual median concentration of PM2.5 in Chengdu, China was 48 μg/m3 (IQR, 33-77 μg/m3). More than 20% of admissions were complicated by major ANCI, and the primary endpoints occurred in 7.6% of patients during their hospitalization. The association of short-term PM2.5 exposure levels with the incidence of acute respiratory failure (adjusted OR [odds ratio] =1.31, 95% CI [confidence interval]1.12-1.54) and acute kidney injury (adjusted OR = 1.20, 95% CI 1.02-1.41) showed a significant trend. Additionally, there were numerically more cases of sepsis (adjusted OR = 1.21, 95% CI 0.92-1.60) and gastrointestinal hemorrhage (adjusted OR = 1.29, 95% CI 0.94-1.77) in patients with higher exposure levels. After further multivariable adjustment, short-term PM2.5 exposure levels were still significantly associated with incident major ANCI (adjusted OR = 1.32, 95% CI 1.12-1.56), as well as a higher incidence of the primary endpoint (adjusted OR = 1.52, 95% CI 1.09-2.12).
CONCLUSION	Short-term PM2.5 exposure before ICCU admission was associated with an increased risk of incident major ANCI and worse in-hospital outcomes in patients receiving intensive cardiac care.

표-807. PubMed 논문번호 32359859의 내용 요약

구분	내용
PubMed ID	32359859
TITLE	Short-term air pollution exposure is a risk factor for acute coronary syndromes in an urban area with low annual pollution rates: Results from a retrospective observational study (2011-2015).
JOURNAL	Archives of cardiovascular diseases: 10.1016/j.acvd.2020.03.013
AUTHORS	Gestro Massimo, Condemi Vincenzo, Bardi Luisella, Tomaino Laura, Roveda Eliana, Bruschetta Antongiulio, Solimene Umberto, Esposito Fabio
BACKGROUND	Epidemiological data suggest that air pollutants are risk factors for cardiovascular disease. Recent studies have questioned the adequacy of current legal pollutant limits, because concentrations lower than those recommended still affect cardiovascular morbidity and mortality.
AIM	To investigate the association between short-term exposure to air pollutants and the daily diagnosis of acute coronary syndrome (ACS) at the emergency department (ED) of S. Croce Hospital (Cuneo, Italy), between 2011 and 2015.
METHODS	We evaluated the effect of particulate matter (PM2.5-10), nitrogen dioxide and ozone as primary exposure, together with temperature and relative humidity as climatological control variables, on ED admissions for ACS (response variables). We studied residents aged ≥35 years, classified into three age groups (35-64, 65-74 and ≥75 years). Environmental data were analysed according to Poisson's regression, and conventional cardiovascular risk factors (CRFs; hypertension, diabetes, coronary artery disease, smoking and dyslipidaemia) were included as control variables.
RESULTS	ED admissions for ACS were 1625/391,689, with 298 in 2011 (0.183%), 305 in 2012 (0.188%), 347 in 2013 (0.214%), 341 in 2014 (0.21%) and 334 in 2015 (0.206%), with a general growth rate of 2.08% (from 2011 to 2015). The CRFs examined were confirmed to be highly associated with occurrence of ACS. Our study identified PM2.5 and temperature in all age groups to be additional risk factors, with PM2.5 exposure (P<0.01) being a particular risk for those aged ≥75 years. Dose-response models confirmed only PM2.5 as the main environmental risk factor in elderly patients (relative risk 1.06, 95% confidence interval 1.02-1.11; lag time 0-3 days). We also found a consistent relative risk for temperature in all age groups.
CONCLUSION	This study confirms the importance of PM2.5 as a risk factor for ACS, mostly in elderly patients, even in a city with low annual pollution rates.

표-808. PubMed 논문번호 32361533의 내용 요약

구분	내용
PubMed ID	32361533
TITLE	Personal exposure to particulate air pollution and vascular damage in peri-urban South India.
JOURNAL	Environment international: 10.1016/j.envint.2020.105734
AUTHORS	Ranzani Otavio T, Mil□ Carles, Sanchez Margaux, Bhogadi Santhi, Kulkarni Bharati, Balakrishnan Kalpana, Sambandam Sankar, Sunyer Jordi, Marshall Julian D, Kinra Sanjay, Tonne Cathryn
OBJECTIVE	Air pollution is a leading preventable risk factor for cardiovascular diseases. Previous studies mostly relied on concentrations at residence, which might not represent personal exposure. Personal air pollution exposure has a greater variability compared with levels of ambient air pollution, facilitating evaluation of exposure-response functions and vascular pathophysiology. We aimed to evaluate the association between predicted annual personal exposure to PM2.5 and black carbon (BC) and three vascular damage markers in peri-urban South India.
METHODS	We analyzed the third wave of the APCAPS cohort (2010-2012), which recruited participants from 28 villages. We used predicted personal exposure to PM2.5 and BC derived from 610 participant-days of 24 h average gravimetric PM2.5 and BC measurements and predictors related to usual time-activity. Outcomes included carotid intima-media thickness (CIMT), carotid-femoral pulse wave velocity (cf-PWV) and augmentation index (AIx). We fit linear mixed models, adjusting for potential confounders and accounting for the clustered data structure. We evaluated nonlinear associations using generalized additive mixed models.
RESULTS	Of the 3017 participants (mean age 38 years), 1453 (48%) were women. The average PM2.5 exposure was 51 □g/m3 (range 13-85) for men, and 61 □g/m3 (range 40-120) for women, while the average BC was 4 □g/m3 (range 3-7) for men and 8 □g/m3 (range 3-22) for women. A 10 μg/m3 increase of PM2.5 was positively associated with CIMT (0.026 mm, 95% CI 0.014, 0.037), cf-PWV (0.069 m/s, 95% CI 0.008, 0.131) and AIx (0.8%, 95% CI 0.3, 1.3) among men. The exposure-response function for PM2.5 and AIx among men showed non-linearity, particularly within the exposure range dominated by tobacco smoking and occupational exposures. Both PM2.5 and BC were positively associated with AIx among women (0.6%, 95% CI 0.2, 1.0, per 10 μg/m3 PM2.5; 0.5%, 95% CI 0.1, 0.8, per 2 μg/m3 BC).
CONCLUSIONS	Personal exposure to particulate matter was associated with vascular damage in a peri-urban population in South India. Personal exposure to particulate matter appears to have gender-specific effects on the type of vascular damage, potentially reflecting differences in sources of personal exposure by gender.

표-809. PubMed 논문번호 32376389의 내용 요약

구분	내용
PubMed ID	32376389
TITLE	Prevalence and risk factors of dry eye in 79,866 participants of the population-based Lifelines cohort study in the Netherlands.
JOURNAL	The ocular surface: 10.1016/j.jtos.2020.04.005
AUTHORS	Vehof Jelle, Snieder Harold, Jansonius Nomdo, Hammond Christopher J
PURPOSE	To investigate the prevalence of dry eye among all adult age categories and to discover independent risk factors by investigating a wide range of etiological categories.
METHODS	A cross-sectional association study including 79,866 voluntary participants aged 20-94 years of the population-based Lifelines Cohort Study in the Netherlands.
RESULTS	Overall, 9.1% of participants had dry eye disease as measured by the Women's Health Study dry eye questionnaire. Prevalence of dry eye symptoms were particularly prevalent in 20-30 years olds. Dry eye was associated with comorbidities in almost all body systems, including musculoskeletal, gastro-intestinal, ophthalmic, autoimmune, psychiatric, pain, functional, dermatological and atopic disorders. Numerous independent risk factors were discovered or confirmed, with strong associations for female sex, contact lens use, irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, eye surgery including cataract and laser refractive surgery, keratoconus, osteoarthritis, connective tissue diseases, atherosclerosis, Graves' disease, autistic disorder, depression, 'burnout', Crohn's disease, sarcoid, lichen planus, rosacea, liver cirrhosis, sleep apnea, sinusitis, thyroid function, and air pollution (NO2). High blood pressure and high BMI were strongly associated with less dry eye, as was current smoking, while ex-smokers had more dry eye. No clear link between dry eye and lipid or blood glucose levels was found.
CONCLUSIONS	This study on dry eye confirmed but also refuted many risk factors from smaller epidemiological studies, and discovered numerous new risk factors in multiple etiological categories. The finding that dry eye symptoms are particularly common in young adults is concerning, and warrants further study.

표-810. PubMed 논문번호 32387882의 내용 요약

구분	내용
PubMed ID	32387882
TITLE	Dose-related effect of secondhand smoke on cardiovascular disease in nonsmokers: Systematic review and meta-analysis.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2020.113546
AUTHORS	Zhang Dongdong, Liu Yiming, Cheng Cheng, Wang Yan, Xue Yuan, Li Wenjie, Li Xing
BACKGROUND	Despite the positive association between secondhand smoke (SHS) and cardiovascular diseases (CVD), no comprehensive assessment on the dose-response relationship between SHS and CVD has yet been reported. Therefore, a meta-analysis was conducted to update the binary association, and to elucidate the dose-response relationship of both self-reported and objectively measured SHS exposure with CVD.
METHODS	PubMed and Embase databases were searched for articles published up to November 12, 2019. Random-effects models were used to assess the summary odds ratios (ORs) of CVD with SHS exposure. Restricted cubic splines were used to fit the dose-response relationship.
RESULTS	Fifty-five eligible observational studies were included in this meta-analysis to investigate the association between SHS exposure and CVD. Based on the meta-analysis, the pooled OR of CVD was 1.22 (95% CI 1.17-1.28) for the self-reported SHS individuals as comparing to the non-exposed group. The result of restricted cubic splines showed a risk plateau of SHS exposure at 15 cigarettes per day (Pnon-linearity = 0.042), while other characteristics s of SHS exposure (amount, daily and cumulative duration, and cotinine) were in linear relationships with CVD (Pnon-linearity >0.05). In addition, an estimated of 6.77% (95% CI: 5.31%-8.46%) of all CVD cases in men and 7.15% (95% CI: 5.62%-8.93%) in women might be attributed to SHS exposure worldwide.
CONCLUSIONS	Exposure to SHS is associated with an increased risk of CVD regardless of the modes. And thus, well-enforced smoke-free laws could possibly reduce a substantial risk of CVD caused by SHS.

표-811. PubMed 논문번호 32389024의 내용 요약

구분	내용
PubMed ID	32389024
TITLE	Preexisting coronary heart disease and susceptibility to long-term effects of traffic-related air pollution: A matched cohort analysis.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487320921987
AUTHORS	Cohen Gali, Steinberg David M, Keinan-Boker Lital, Yuval Technion Center of Excellence in Exposure Science and Environmental Health, Technion Israel Institute of Technology, Israel, Levy Ilan, Chen Shimon, Shafran-Nathan Rakefet, Levin Noam, Shimony Tal, Witberg Guy, Bental Tamir, Shohat Tamar, Broday David M, Kornowski Ran, Gerber Yariv
BACKGROUND	Individuals with coronary heart disease are considered susceptible to traffic-related air pollution exposure. Yet, cohort-based evidence on whether preexisting coronary heart disease modifies the association of traffic-related air pollution with health outcomes is lacking.
AIM	Using data of four Israeli cohorts, we compared associations of traffic-related air pollution with mortality and cancer between coronary heart disease patients and matched controls from the general population.
METHODS	Subjects hospitalized with acute coronary syndrome from two patient cohorts (inception years: 1992-1993 and 2006-2014) were age- and sex-matched to coronary heart disease-free participants of two cycles of the Israeli National Health and Nutrition Surveys (inception years: 1999-2001 and 2005-2006). Ambient concentrations of nitrogen oxides at the residential place served as a proxy for traffic-related air pollution exposure across all cohorts, based on a high-resolution national land use regression model (50 m). Data on all-cause mortality (last update: 2018) and cancer incidence (last update: 2016) were retrieved from national registries. Cox-derived stratum-specific hazard ratios with 95% confidence intervals were calculated, adjusted for harmonized covariates across cohorts, including age, sex, ethnicity, neighborhood socioeconomic status, smoking, diabetes, hypertension, prior stroke and prior malignancy (the latter only in the mortality analysis). Effect-modification was examined by testing nitrogen oxides-by-coronary heart disease interaction term in the entire matched cohort.
RESULTS	The cohort (mean (standard deviation) age 61.5 (14) years; 44% women) included 2393 matched pairs, among them 2040 were cancer-free at baseline. During a median (25th-75th percentiles) follow-up of 13 (10-19) and 11 (7-17) years, 1458 deaths and 536 new cancer cases were identified, respectively. In multivariable-adjusted models, a 10-parts per billion nitrogen oxides increment was positively associated with all-cause mortality among coronary heart disease patients (hazard ratio = 1.13, 95% confidence interval 1.05-1.22), but not among controls (hazard ratio = 1.00, 0.93-1.08) (pinteraction = 0.003). A similar pattern was seen for all-cancer incidence (hazard ratioCHD = 1.19 (1.03-1.37), hazard ratioCHD-Free = 0.93 (0.84-1.04) (pinteraction = 0.01)). Associations were robust to multiple sensitivity analyses.
CONCLUSIONS	Coronary heart disease patients might be at increased risk for traffic-related air pollution-associated mortality and cancer, irrespective of their age and sex. Patients and clinicians should be more aware of the adverse health effects on coronary heart disease patients of chronic exposure to vehicle emissions.

표-812. PubMed 논문번호 32389507의 내용 요약

구분	내용
PubMed ID	32389507
TITLE	Incidence and Mortality of Lung Cancer Among Never Smokers in Relationship to Secondhand Smoking: Findings From the PLCO Trial.
JOURNAL	Clinical lung cancer: 10.1016/j.cllc.2020.04.009
AUTHORS	Abdel-Rahman Omar
PURPOSE	To assess the impact of secondhand smoking on the incidence and mortality of lung cancer among never smokers enrolled onto the Prostate, Lung, Colorectal, and Ovary (PLCO) study.
PATIENTS AND METHODS	Deidentified data sets from the PLCO study were accessed and never smokers who completed the supplementary questionnaire's questions related to history of exposure to secondhand smoking were included in the current study. Multivariate Cox regression analysis was conducted to evaluate the impact of adulthood and childhood secondhand smoking on lung cancer incidence and mortality.
RESULTS	A total of 49,569 participants were included in the current analysis. Using multivariate Cox regression analysis, participants with secondhand smoking most of their work time had a higher risk of lung cancer diagnosis (hazard ratio, 2.038; 95% confidence interval, 1.313-3.164; P = .002). Likewise, participants with secondhand smoking most of their adult living time had a higher risk of lung cancer diagnosis (hazard ratio, 1.809; 95% confidence interval, 1.161-2.819; P = .009). Moreover, participants with secondhand smoking most of the adult time had a higher risk of death from lung cancer (hazard ratio, 1.925; 95% confidence interval, 1.035-3.575; P = .038). Participants with secondhand smoking most of the adult time were also more likely to have had hypertension (P < .001), diabetes mellitus (P < .001), heart attack (P < .001), stroke (P = .028), chronic bronchitis (P < .001), and emphysema (P < .001).
CONCLUSION	Never smokers with a history of adult secondhand smoking had a higher probability of a subsequent diagnosis of lung cancer. Likewise, never smokers with a history of adult secondhand smoking were more likely to die from lung cancer.

표-813. PubMed 논문번호 32402982의 내용 요약

구분	내용
PubMed ID	32402982
TITLE	Ambient ozone pollution and years of life lost: Association, effect modification, and additional life gain from a nationwide analysis in China.
JOURNAL	Environment international: 10.1016/j.envint.2020.105771
AUTHORS	Li Jie, Yin Peng, Wang Lijun, Zhang Xiao, Liu Jiangmei, Liu Yunning, Zhou Maigeng
BACKGROUND	Ozone is one of the dominant air pollutants due to its impact on disease burden and increasing trend in concentration. However, evidence regarding short-term effect of ozone on years of life lost (YLL) is scarce.
METHODS	A national time-series study was conducted in 48 large Chinese cities from 2013 to 2017. Generalized additive model coupled with random effects model were used to estimate national-average associations of ozone with YLL. Potential modifiers and additional life gain due to avoidable YLL under certain scenario were also evaluated.
RESULTS	The average annual mean ozone concentration of these cities was 86.9 μg/m3. For 10 μg/m3 increase in 3-day moving average ozone concentration, we estimated 0.37% [95% confidence interval (CI): 0.29%, 0.46%] increase in YLL from nonaccidental causes, 0.38% (95% CI: 0.30%, 0.46%) increase in YLL from cardiovascular diseases, and 0.36% (95% CI: 0.16%, 0.56%) increase in YLL from respiratory diseases. Moreover, the associations were more evident in people with less education and in cities with lower carbon monoxide concentration or those located at north region with lower mean temperature. Finally, an estimated life of 0.055 (95% CI: 0.043, 0.068) years would be gained per deceased people if ozone concentration could fall to 100 μg/m3.
CONCLUSIONS	Our findings indicated robust associations between short-term exposure to ozone and YLL from nonaccidental causes and cardiopulmonary diseases. Relevant intervention design should take the heterogeneity of both individual- and city-level characteristics into account. Implementation of more stringent standard is beneficial for alleviating YLL caused by ozone.

표-814. PubMed 논문번호 32438827의 내용 요약

구분	내용
PubMed ID	32438827
TITLE	Long-Term Exposure to Air Pollution and Incidence of Myocardial Infarction: A Danish Nurse Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP5818
AUTHORS	Cramer Johannah, J ø rgensen Jeanette T, Hoffmann Barbara, Loft Steffen, Br□uner Elvira V, Prescott Eva, Ketzel Matthias, Hertel Ole, Brandt J ø rgen, Jensen Steen S, Backalarz Claus, Simonsen Mette K, Andersen Zorana J
BACKGROUND	Air pollution exposure has been linked to coronary heart disease, although evidence on PM2.5 and myocardial infarction (MI) incidence is mixed.
OBJECTIVES	This prospective cohort study aimed to investigate associations between long-term exposure to air pollution and MI incidence, adjusting for road traffic noise.
METHODS	We used data from the nationwide Danish Nurse Cohort on 22,882 female nurses (>44 years of age) who, at recruitment in 1993 or 1999, reported information on cardiovascular disease risk factors. Data on MI incidence was collected from the Danish National Patient Register until the end of 2014. Annual mean concentrations of particulate matter (PM) with a diameter <2.5 μg/m3 (PM2.5), PM10, nitrogen dioxide (NO2), and nitrogen oxides (NOx) at the nurses' residences since 1990 (PM10 and PM2.5) or 1970 (NO2 and NOx) were estimated using the Danish Eulerian Hemispheric Model/Urban Background Model/AirGIS (DEHM/UBM/AirGIS) dispersion model. We used time-varying Cox regression models to examine the association between 1- and 3-y running means of these pollutants, as well as 23-y running means of NO2 and NOx, with both overall and fatal incident MI. Associations were explored in three progressively adjusted models: Model 1, adjusted for age and baseline year; Model 2, with further adjustment for potential confounding by lifestyle and cardiovascular disease risk factors; and Model 3, with further adjustment for road traffic noise, modeled as the annual mean of a weighted 24-h average (Lden).
RESULTS	Of the 22,882 women, 641 developed MI during a mean follow-up of 18.6 y, 121 (18.9%) of which were fatal. Reported hazard ratios (HRs) were based on interquartile range increases of 5.3, 5.5, 8.1, and 11.5 μg/m3 for PM2.5, PM10, NO2, and NOx, respectively. In Model 1, we observed a positive association between a 3-y running mean of PM2.5 and an overall incident MI with an HR= 1.20 (95% CI: 1.07, 1.35), which attenuated to HR= 1.06 (95% CI: 0.92, 1.23) in Model 2. In Model 1 for incident fatal MI, we observed a strong association with a 3-y running mean of PM2.5, with an HR= 1.69 (95% CI: 1.33, 2.13), which attenuated to HR= 1.35 (95% CI: 1.01, 1.81) in Model 2. Similar associations were seen for PM10, with 3-y, Model 2 estimates for overall and fatal incident MI of HR= 1.06 (95% CI: 0.91, 1.23) and HR= 1.35 (95% CI: 1.01, 1.81), respectively. No evidence of an association was observed for NO2 or NOx. For all pollutants, associations in Model 2 were robust to further adjustment for road traffic noise in Model 3 and were similar for a 1-y running mean exposure.
CONCLUSIONS	We found no association between long-term exposure to PM2.5, PM10, NO2, or NOx and overall MI incidence, but we observed positive associations for PM2.5 and PM10 with fatal MI. We present novel findings that the association between PM and MI incidence is robust to adjustment for road traffic noise. https://doi.org/10.1289/EHP5818.

표-815. PubMed 논문번호 32438890의 내용 요약

구분	내용
PubMed ID	32438890
TITLE	Road Traffic Noise Exposure and Filled Prescriptions for Antihypertensive Medication: A Danish Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP6273
AUTHORS	Thacher Jesse D, Poulsen Aslak H, Roswall Nina, Hvidtfeldt Ulla, Raaschou-Nielsen Ole, Jensen Steen Solvang, Ketzel Matthias, Brandt J ø rgen, Overvad Kim, Tj ø nneland Anne, M□nzel Thomas, S ø rensen Mette
BACKGROUND	Epidemiological research on effects of transportation noise on incident hypertension is inconsistent.
OBJECTIVES	We aimed to investigate whether residential road traffic noise increases the risk for hypertension.
METHODS	In a population-based cohort of 57,053 individuals 50-64 years of age at enrollment, we identified 21,241 individuals who fulfilled our case definition of filling ≥2 prescriptions and ≥180 defined daily doses of antihypertensive drugs (AHTs) within a year, during a mean follow-up time of 14.0 y. Residential addresses from 1987 to 2016 were obtained from national registers, and road traffic noise at the most exposed fa□ade as well as the least exposed fa□ade was modeled for all addresses. Analyses were conducted using Cox proportional hazards models.
RESULTS	We found no associations between the 10-y mean exposure to road traffic noise and filled prescriptions for AHTs, with incidence rate ratios (IRRs) of 0.999 [95% confidence intervals (CI): 0.980, 1.019)] per 10-dB increase in road traffic noise at the most exposed fa□ade and of 1.001 (95% CI: 0.977, 1.026) at the least exposed fa□ade. Interaction analyses suggested an association with road traffic noise at the least exposed fa□ade among subpopulations of current smokers and obese individuals.
CONCLUSION	The present study does not support an association between road traffic noise and filled prescriptions for AHTs. https://doi.org/10.1289/EHP6273.

표-816. PubMed 논문번호 32450864의 내용 요약

구분	내용
PubMed ID	32450864
TITLE	Children's microvascular traits and ambient air pollution exposure during pregnancy and early childhood: prospective evidence to elucidate the developmental origin of particle-induced disease.
JOURNAL	BMC medicine: 10.1186/s12916-020-01586-x
AUTHORS	Luyten Leen J, Dockx Yinthe, Provost Eline B, Madhloum Narjes, Sleurs Hanne, Neven Kristof Y, Janssen Bram G, Bov□ Hannelore, Debacq-Chainiaux Florence, Gerrits Nele, Lefebvre Wouter, Plusquin Michelle, Vanpoucke Charlotte, De Boever Patrick, Nawrot Tim S
BACKGROUND	Particulate matter exposure during in utero life may entail adverse health outcomes later in life. The microvasculature undergoes extensive, organ-specific prenatal maturation. A growing body of evidence shows that cardiovascular disease in adulthood is rooted in a dysfunctional fetal and perinatal development, in particular that of the microcirculation. We investigate whether prenatal or postnatal exposure to PM2.5 (particulate matter with a diameter ≤ 2.5 μm) or NO2 is related to microvascular traits in children between the age of four and six.
METHODS	We measured the retinal microvascular diameters, the central retinal arteriolar equivalent (CRAE) and central retinal venular equivalent (CRVE), and the vessel curvature by means of the tortuosity index (TI) in young children (mean [SD] age 4.6 [0.4] years), followed longitudinally within the ENVIRONAGE birth cohort. We modeled daily prenatal and postnatal PM2.5 and NO2 exposure levels for each participant's home address using a high-resolution spatiotemporal model.
RESULTS	An interquartile range (IQR) increase in PM2.5 exposure during the entire pregnancy was associated with a 3.85-μm (95% CI, 0.10 to 7.60; p = 0.04) widening of the CRVE and a 2.87-μm (95% CI, 0.12 to 5.62; p = 0.04) widening of the CRAE. For prenatal NO2 exposure, an IQR increase was found to widen the CRVE with 4.03 μm (95% CI, 0.44 to 7.63; p = 0.03) and the CRAE with 2.92 μm (95% CI, 0.29 to 5.56; p = 0.03). Furthermore, a higher TI score was associated with higher prenatal NO2 exposure. We observed a postnatal effect of short-term PM2.5 exposure on the CRAE and a childhood NO2 exposure effect on both the CRVE and CRAE.
CONCLUSIONS	Our results link prenatal and postnatal air pollution exposure with changes in a child's microvascular traits as a fundamental novel mechanism to explain the developmental origin of cardiovascular disease.

표-817. PubMed 논문번호 32464360의 내용 요약

구분	내용
PubMed ID	32464360
TITLE	Ambient air pollutants aggravate association of snoring with prevalent hypertension: results from the Henan Rural Cohort.
JOURNAL	Chemosphere: 10.1016/j.chemosphere.2020.127108
AUTHORS	Zhang Haiqing, Li Shanshan, Chen Gongbo, Abdulai Tanko, Liu Xiaotian, Wang Yan, Liang Huiying, Hou Jian, Huo Wenqian, Mao Zhenxing, Wang Chongjian, Bie Ronghai
AIM	We aimed to assess if snoring and ambient air pollutants were jointly associated with prevalent hypertension in a cross-sectional study.
METHODS	A total of 28440 participants aged 18-79 years were obtained from the Henan Rural Cohort. Snoring evaluated using Pittsburgh sleep quality index (PSQI) scale was classified into 'Never', '<3 times/week' and '≥3 times/week' groups. Concentrations of air pollutants (PM1, PM2.5, PM10, and NO2) were evaluated by a satellite-based spatiotemporal model. The independent and joint associations between snoring and air pollutants on prevalence of hypertension were analyzed by logistic regression models.
RESULTS	The mean age of all participants was 56.0 ± 12.2 years. The frequencies and prevalence of participants with hypertension were 3666 (32.39%) in men and 5576(32.57%) in women, respectively. The odds ratio (OR) and 95% confidence interval (CI) of participants with snoring frequency of <3 times/week, ≥3 times/week was 1.10(1.02-1.20), and 1.15(1.08-1.23) for hypertension, compared to those without snoring. Participants with a snoring (≥3 times/week) and higher exposure concentrations of PM1, PM2.5, PM10, and NO2 had 2.58-fold(95% CI: 2.30-2.90), 3.03-fold(95% CI: 2.69-3.41), 2.89-fold(95% CI: 2.57-3.25) and 2.75-fold(95% CI: 2.44-3.10) for hypertension, compared to those without snoring and low concentrations of air pollutants. Additionally, participants with high PM1 and ≥3 times/week snoring (OR: 1.32, 95% CI: 1.18-1.48) was at a higher likelihood for prevalent hypertension, compared to those without snoring and with high PM1.
CONCLUSIONS	Snoring and high ambient air pollutants might be important predictors of hypertension, and higher concentration of PM1 might aggravate the association between snoring and hypertension.

표-818. PubMed 논문번호 32480112의 내용 요약

구분	내용
PubMed ID	32480112
TITLE	Physical activity attenuated association of air pollution with estimated 10-year atherosclerotic cardiovascular disease risk in a large rural Chinese adult population: A cross-sectional study.
JOURNAL	Environment international: 10.1016/j.envint.2020.105819
AUTHORS	Tu Runqi, Hou Jian, Liu Xiaotian, Li Ruiying, Dong Xiaokang, Pan Mingming, Mao Zhenxing, Huo Wenqian, Chen Gongbo, Guo Yuming, Li Shanshan, Wang Chongjian
BACKGROUND	Although long-term exposure to air pollution and physical inactivity are linked to increased risk for atherosclerotic cardiovascular diseases (ASCVD), however, the interactive effect of air pollution and physical activity (PA) on high 10-year ASCVD risk is largely unknown.
METHODS	A total of 31,162 individuals aged 35-74 years were derived from the Henan Rural Cohort Study, after individuals with personal histories of ASCVD or missing data on predictors of high 10-year ASCVD risk were excluded. Concentrations of air pollutants (nitrogen dioxide (NO2), particulate matter with an aerodynamics diameters ≤ 1.0 □m (PM1), ≤2.5 □m (PM2.5) or ≤10 □m (PM10)) of individuals were estimated using a spatiotemporal model based on satellites data. The metabolic equivalent (MET) of PA of each individual was evaluated using the formula: duration (hour/time) × frequency/week × MET coefficient of each type of activity. Logistic regression models were used to analyze associations between air pollutants, PA and high 10-year ASCVD risk. Interaction plots were used to describe interactive effects of air pollutants and PA on high 10-year ASCVD risk.
RESULTS	Each 1 □g/m3 increase in PM1, PM2.5, PM10 and NO2 were related to a 4.4% (odds ratio (OR): 1.044, 95% confidence interval (CI): 1.034, 1.056), 9.1% (OR: 1.091, 95% CI: 1.079, 1.104), 4.6% (OR: 1.046, 95% CI: 1.040, 1.051) or 6.4% (OR: 1.064, 95% CI: 1.055, 1.072) increase in high 10-year ASCVD risk (all p < 0.001), respectively; each one unit-increase in PA MET (hour/day) value was related to a 1.8% (OR: 0.982, 95% CI: 0.980, 0.985) decrease in high 10-year ASCVD risk. Negative interactive effects of PA and PM1, PM2.5, PM10 and NO2 on high 10-year ASCVD risk were observed (all p < 0.001).
CONCLUSION	Exposure to high levels of air pollutants were related to increase high 10-year ASCVD risk and these associations were attenuated by PA, implying that PA may be an effective method to the prevention of high 10-year ASCVD risk in highly polluted rural regions.

표-819. PubMed 논문번호 32480167의 내용 요약

구분	내용
PubMed ID	32480167
TITLE	Correlation between short-term air pollution exposure and unprovoked lung embolism. Prospective observational (Contamina-TEP Group).
JOURNAL	Thrombosis research: 10.1016/j.thromres.2020.04.033
AUTHORS	de Miguel-Diez Javier, Blasco-Esquivias Isabel, Rodriguez-Matute Consolacion, Bedate-Diaz Pedro, Lopez-Reyes Raquel, Fernandez-Capitan Carmen, Garcia-Fuika Sophe, Lobo-Beristain Jose Luis, Garcia-Lozaga Amaia, Quezada Carlos Andres, Murga-Arizabaleta Igor, Garcia-Ortega Alberto, Rodr□guez-Davila M ª Angeles, Marin-Barrera Lucia, Otero-Candelera Remedios, Praena-Fernandez Juan Manuel, Jara-Palomares Luis
BACKGROUND	The aim was to analyze the temporal relationship between short-term air pollution exposure and acute symptomatic unprovoked pulmonary embolism (PE).
PATIENTS/METHODS	We performed a prospective, multicenter study in consecutive patients diagnosed with acute symptomatic unprovoked PE from February 2012 to January 2013. We analyzed demographic and clinical data, patients' addresses, meteorological and air pollutants data (PM10, SO2, CO, NO2, ozone emission data). We considered the number of days the patient had symptoms, and the study period constituted the previous 30 days. Likewise, the mean annual data of the reference season were calculated as well as the data of the 30-day study period corresponding to the same dates in the previous 3 years in order to obtain the monthly mean of the different pollutants for each period.
RESULTS	A total of 162 patients with acute symptomatic PE were recruited (43.2% unprovoked PE). The air pollutants could be determined in 50% of the patients with unprovoked PE, and a final analysis was performed in 35 patients. In the multiple comparison analysis to verify a possible correlation between the study period and the annual median, only NO2 showed a statistically significant association ($p = 0.009$). When comparing the study period with the previous 3 years, only NO2 maintained a statistically significant association for the 3 study periods.
CONCLUSIONS	We found a relationship between short-term exposure to NO2 and the presence of unprovoked PE.

표-820. PubMed 논문번호 32484729의 내용 요약

구분	내용
PubMed ID	32484729
TITLE	Genome-Wide DNA Methylation in Peripheral Blood and Long-Term Exposure to Source-Specific Transportation Noise and Air Pollution: The SAPALDIA Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP6174
AUTHORS	Eze Ikenna C, Jeong Ayoung, Schaffner Emmanuel, Rezwan Faisal I, Ghantous Akram, Foraster Maria, Vienneau Danielle, Kronenberg Florian, Herceg Zdenko, Vineis Paolo, Brink Mark, Wunderli Jean-Marc, Schindler Christian, Cajochen Christian, R□□sli Martin, Holloway John W, Imboden Medea, Probst-Hensch Nicole
BACKGROUND	Few epigenome-wide association studies (EWAS) on air pollutants exist, and none have been done on transportation noise exposures, which also contribute to environmental burden of disease.
OBJECTIVE	We performed mutually independent EWAS on transportation noise and air pollution exposures.
METHODS	We used data from two time points of the Swiss Cohort Study on Air Pollution and Lung and Heart Diseases in Adults (SAPALDIA) from 1,389 participants contributing 2,542 observations. We applied multiexposure linear mixed-effects regressions with participant-level random intercept to identify significant Cytosine-phosphate-Guanine (CpG) sites and differentially methylated regions (DMRs) in relation to 1-y average aircraft, railway, and road traffic day-evening-night noise (Lden); nitrogen dioxide (NO2); and particulate matter (PM) with aerodynamic diameter <2.5 μm (PM2.5). We performed candidate (CpG-based; cross-systemic phenotypes, combined into "allostatic load") and agnostic (DMR-based) pathway enrichment tests, and replicated previously reported air pollution EWAS signals.
RESULTS	We found no statistically significant CpGs at false discovery rate <0.05. However, 14, 48, 183, 8, and 71 DMRs independently associated with aircraft, railway, and road traffic Lden; NO2; and PM2.5, respectively, with minimally overlapping signals. Transportation Lden and air pollutants tendentially associated with decreased and increased methylation, respectively. We observed significant enrichment of candidate DNA methylation related to C-reactive protein and body mass index (aircraft, road traffic Lden, and PM2.5), renal function and "allostatic load" (all exposures). Agnostic functional networks related to cellular immunity, gene expression, cell growth/proliferation, cardiovascular, auditory, embryonic, and neurological systems development were enriched. We replicated increased methylation in cg08500171 (NO2) and decreased methylation in cg17629796 (PM2.5).
CONCLUSIONS	Mutually independent DNA methylation was associated with source-specific transportation noise and air pollution exposures, with distinct and shared enrichments for pathways related to inflammation, cellular development, and immune responses. These findings contribute in clarifying the pathways linking these exposures and age-related diseases but need further confirmation in the context of mediation analyses. https://doi.org/10.1289/EHP6174.

표-821. PubMed 논문번호 32493701의 내용 요약

구분	내용
PubMed ID	32493701
TITLE	Occupational exposure to whole-body vibrations and pregnancy complications: a nationwide cohort study in Sweden.
JOURNAL	Occupational and environmental medicine: 10.1136/oemed-2020-106519
AUTHORS	Skr□der Helena, Pettersson Hans, Albin Maria, Gustavsson Per, Rylander Lars, Norl□n Filip, Selander Jenny
OBJECTIVES	Pregnancy complications are common contributors to perinatal mortality and morbidity. Still, the cause(s) of gestational hypertensive disorders and diabetes are largely unknown. Some occupational exposures have been inconsistently associated with pregnancy complications, but exposure to whole-body vibrations (WBV) has been largely overlooked even though it has been associated with adverse birth outcomes. Therefore, the aim was to assess whether occupational WBV exposure during pregnancy is associated with pregnancy complications in a nationwide, prospective cohort study.
METHODS	The Fetal Air Pollution Exposure cohort was formed by merging multiple Swedish, national registers containing information on occupation during pregnancy and diagnosis codes, and includes all working women who gave birth between 1994 and 2014 (n=1 091 044). WBV exposure was derived from a job-exposure matrix and was divided into categories (0, 0.1-0.2, 0.3-0.4 and ≥0.5 m/s2). ORs with 95% CIs were calculated using logistic regression adjusted for potential confounders.
RESULTS	Among women working full time (n=646 490), we found increased risks of all pregnancy complications in the highest exposure group (≥0.5 m/s2), compared with the lowest. The adjusted ORs were 1.76 (95% CI 1.41 to 2.20), 1.55 (95% CI 1.26 to 1.91) and 1.62 (95% CI 1.07 to 2.46) for preeclampsia, gestational hypertension and gestational diabetes, respectively, and were similar in all sensitivity analyses. There were no clear associations for part-time workers.
CONCLUSIONS	The results suggest that women should not be exposed to WBV at/above the action limit value of 0.5 m/s2 (European directive) continuously through pregnancy. However, these results need further confirmation.

표-822. PubMed 논문번호 Previous attempts to characterise the burden of chronic respiratory diseases have focused only on specific disease conditions, such as chronic obstructive pulmonary disease (COPD) or asthma. In this study, we aimed to characterise the burden of chronic respiratory diseases globally, providing a comprehensive and up-to-date analysis on geographical and time trends from 1990 to 2017.의 내용 요약

구분	내용
BACKGROUND	Previous attempts to characterise the burden of chronic respiratory diseases have focused only on specific disease conditions, such as chronic obstructive pulmonary disease (COPD) or asthma. In this study, we aimed to characterise the burden of chronic respiratory diseases globally, providing a comprehensive and up-to-date analysis on geographical and time trends from 1990 to 2017.
METHODS	Using data from the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017, we estimated the prevalence, morbidity, and mortality attributable to chronic respiratory diseases through an analysis of deaths, disability-adjusted life-years (DALYs), and years of life lost (YLL) by GBD super-region, from 1990 to 2017, stratified by age and sex. Specific diseases analysed included asthma, COPD, interstitial lung disease and pulmonary sarcoidosis, pneumoconiosis, and other chronic respiratory diseases. We also assessed the contribution of risk factors (smoking, second-hand smoke, ambient particulate matter and ozone pollution, household air pollution from solid fuels, and occupational risks) to chronic respiratory disease-attributable DALYs.
FINDINGS	In 2017, 544·9 million people (95% uncertainty interval [UI] 506·9-584·8) worldwide had a chronic respiratory disease, representing an increase of 39·8% compared with 1990. Chronic respiratory disease prevalence showed wide variability across GBD super-regions, with the highest prevalence among both males and females in high-income regions, and the lowest prevalence in sub-Saharan Africa and south Asia. The age-sex-specific prevalence of each chronic respiratory disease in 2017 was also highly variable geographically. Chronic respiratory diseases were the third leading cause of death in 2017 (7·0% [95% UI 6·8-7·2] of all deaths), behind cardiovascular diseases and neoplasms. Deaths due to chronic respiratory diseases numbered 3 914 196 (95% UI 3 790 578-4 044 819) in 2017, an increase of 18·0% since 1990, while total DALYs increased by 13·3%. However, when accounting for ageing and population growth, declines were observed in age-standardised prevalence (14·3% decrease), age-standardised death rates (42·6%), and age-standardised DALY rates (38·2%). In males and females, most chronic respiratory disease-attributable deaths and DALYs were due to COPD. In regional analyses, mortality rates from chronic respiratory diseases were greatest in south Asia and lowest in sub-Saharan Africa, also across both sexes. Notably, although absolute prevalence was lower in south Asia than in most other super-regions, YLLs due to chronic respiratory diseases across the subcontinent were the highest in the world. Death rates due to interstitial lung disease and pulmonary sarcoidosis were greater than those due to pneumoconiosis in all super-regions. Smoking was the leading risk factor for chronic respiratory disease-related disability across all regions for men. Among women, household air pollution from solid fuels was the predominant risk factor for chronic respiratory diseases in south Asia and sub-Saharan Africa, while ambient particulate matter represented the leading risk factor in southeast Asia, east Asia, and Oceania, and in the Middle East and north Africa super-region.
INTERPRETATION	Our study shows that chronic respiratory diseases remain a leading cause of death and disability worldwide, with growth in absolute numbers but sharp declines in several age-standardised estimators since 1990. Premature mortality from chronic respiratory diseases seems to be highest in regions with less-resourced health systems on a per-capita basis.
FUNDING	Bill & Melinda Gates Foundation.

표-823. PubMed 논문번호 32559440의 내용 요약

구분	내용
PubMed ID	32559440
TITLE	Associations of outdoor fine particulate air pollution and cardiovascular disease in 157 436 individuals from 21 high-income, middle-income, and low-income countries (PURE): a prospective cohort study.
JOURNAL	The Lancet. Planetary health: 10.1016/S2542-5196(20)30103-0
AUTHORS	Hystad Perry, Larkin Andrew, Rangarajan Sumathy, AlHabib Khalid F, Avezum □lvaro, Calik Kevser Burcu Tumerdem, Chifamba Jephat, Dans Antonio, Diaz Rafael, du Plessis Johan L, Gupta Rajeev, Iqbal Romaina, Khatib Rasha, Kelishadi Roya, Lanas Fernando, Liu Zhiguang, Lopez-Jaramillo Patricio, Nair Sanjeev, Poirier Paul, Rahman Omar, Rosengren Annika, Swidan Hany, Tse Lap Ah, Wei Li, Wielgosz Andreas, Yeates Karen, Yusoff Khalid, Zato□ski Tomasz, Burnett Rick, Yusuf Salim, Brauer Michael
BACKGROUND	Most studies of long-term exposure to outdoor fine particulate matter (PM2·5) and cardiovascular disease are from high-income countries with relatively low PM2·5 concentrations. It is unclear whether risks are similar in low-income and middle-income countries (LMICs) and how outdoor PM2·5 contributes to the global burden of cardiovascular disease. In our analysis of the Prospective Urban and Rural Epidemiology (PURE) study, we aimed to investigate the association between long-term exposure to PM2·5 concentrations and cardiovascular disease in a large cohort of adults from 21 high-income, middle-income, and low-income countries.
METHODS	In this multinational, prospective cohort study, we studied 157 436 adults aged 35-70 years who were enrolled in the PURE study in countries with ambient PM2·5 estimates, for whom follow-up data were available. Cox proportional hazard frailty models were used to estimate the associations between long-term mean community outdoor PM2·5 concentrations and cardiovascular disease events (fatal and non-fatal), cardiovascular disease mortality, and other non-accidental mortality.
FINDINGS	Between Jan 1, 2003, and July 14, 2018, 157 436 adults from 747 communities in 21 high-income, middle-income, and low-income countries were enrolled and followed up, of whom 140 020 participants resided in LMICs. During a median follow-up period of 9·3 years (IQR 7·8-10·8; corresponding to 1·4 million person-years), we documented 9996 non-accidental deaths, of which 3219 were attributed to cardiovascular disease. 9152 (5·8%) of 157 436 participants had cardiovascular disease events (fatal and non-fatal incident cardiovascular disease), including 4083 myocardial infarctions and 4139 strokes. Mean 3-year PM2·5 at cohort baseline was 47·5 μg/m3 (range 6-140). In models adjusted for individual, household, and geographical factors, a 10 μg/m3 increase in PM2·5 was associated with increased risk for cardiovascular disease events (hazard ratio 1·05 [95% CI 1·03-1·07]), myocardial infarction (1·03 [1·00-1·05]), stroke (1·07 [1·04-1·10]), and cardiovascular disease mortality (1·03 [1·00-1·05]). Results were similar for LMICs and communities with high PM2·5 concentrations (>35 μg/m3). The population attributable fraction for PM2·5 in the PURE cohort was 13·9% (95% CI 8·8-18·6) for cardiovascular disease events, 8·4% (0·0-15·4) for myocardial infarction, 19·6% (13·0-25·8) for stroke, and 8·3% (0·0-15·2) for cardiovascular disease mortality. We identified no consistent associations between PM2·5 and risk for non-cardiovascular disease deaths.
INTERPRETATION	Long-term outdoor PM2·5 concentrations were associated with increased risks of cardiovascular disease in adults aged 35-70 years. Air pollution is an

표-824. PubMed 논문번호 32565497의 내용 요약

구분	내용
PubMed ID	32565497
TITLE	Association Between Traffic Count and Cardiovascular Mortality: A Prospective Cohort Study in Taiwan.
JOURNAL	Journal of epidemiology: 10.2188/jea.JE20200082
AUTHORS	Pan Wen-Chi, Yeh Szu-Yu, Wu Chih-Da, Huang Yen-Tsung, Chen Yu-Cheng, Chen Chien-Jen, Yang Hwai-I
BACKGROUND	Exposure to traffic-related pollution is positively associated with cardiovascular diseases (CVD), but little is known about how different sources of traffic pollution (eg, gasoline-powered cars, diesel-engine vehicles) contribute to CVD. Therefore, we evaluated the association between exposure to different types of engine exhaust and CVD mortality.
METHODS	We recruited 12,098 participants from REVEAL-HBV cohort in Taiwan. The CVD mortality in 2000-2014 was ascertained by the Taiwan Death Certificates. Traffic pollution sources (2005-2013) were based on information provided by the Directorate General of Highway in 2005. Exposure to PM2.5 was based on a land-use regression model. We applied Cox proportional hazard models to assess the association of traffic vehicle exposure and CVD mortality. A causal mediation analysis was applied to evaluate the mediation effect of PM2.5 on the relationship between traffic and CVD mortality.
RESULTS	A total of 382 CVD mortalities were identified from 2000 to 2014. We found participants exposed to higher volumes of small car and truck exhausts had an increased CVD mortality. The adjusted hazard ratio (HR) was 1.10 for small cars (95% confidence interval [CI], 0.94-1.27; P-value = 0.23) and 1.24 for truck (95% CI, 1.03-1.51; P-value = 0.03) per one unit increment of the logarithm scale. The findings were still robust with further adjustment for different types of vehicles. A causal mediation analysis revealed PM2.5 had an over 60% mediation effect on traffic-CVD association.
CONCLUSIONS	Exposure to exhaust from trucks or gasoline-powered cars is positively associated with CVD mortality, and air pollution may play a role in this association.

표-825. PubMed 논문번호 32589457의 내용 요약

구분	내용
PubMed ID	32589457
TITLE	Long-Term Greenspace Exposure and Progression of Arterial Stiffness: The Whitehall II Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP6159
AUTHORS	de Keijzer Carmen, Foraster Maria, Basaga□a Xavier, Tonne Cathryn, Garcia Lucia Alonso, Valent□n Ant□nia, Kivim□ki Mika, Nieuwenhuijsen Mark J, Alonso Jordi, Ant□ Josep M, Singh-Manoux Archana, Sunyer Jordi, Dadvand Payam
BACKGROUND	Arterial stiffness, and its progression with age, is an important indicator of cardiovascular aging. Greenspace exposure may protect against arterial stiffness by promoting physical activity, fostering social cohesion, and reducing stress and exposure to air pollution and noise.
OBJECTIVES	The aim of this study was to investigate the association of long-term exposure to outdoor greenspace with arterial stiffness and its progression over time.
METHODS	This prospective cohort study was based on 4,349 participants (55-83 years of age) of the Whitehall II Study, United Kingdom. Arterial stiffness was assessed in two medical examinations (2007-2009 and 2012-2013) by measuring the carotid-femoral pulse wave velocity (cf-PWV). Residential surrounding greenspace was characterized using satellite-based indices of greenspace including normalized difference vegetation index (NDVI), enhanced vegetation index (EVI), and vegetation continuous fields (VCF) across buffers of 500 and 1,000m surrounding the participants' residential locations at each follow-up. The association between the greenspace indicators and baseline cf-PWV and 4-year progression of cf-PWV was assessed using linear mixed-effects models with the participant as a random effect, controlling for demographic, lifestyle, and (individual and area) socioeconomic factors.
RESULTS	No statistically significant associations were observed between residential surrounding greenspace and baseline or 4-y progression of cf-PWV; interquartile range (IQR) increases in NDVI, EVI, and VCF in the 500-m buffer were associated with -0.04m/s [95% confidence interval (CI): -0.12, 0.04], -0.03m/s (95% CI: -0.10, 0.05), and -0.02m/s (95% CI: -0.08, 0.04) in baseline cf-PWV and 0.06m/s (95% CI: -0.02, 0.14), 0.05m/s (95% CI: -0.03, 0.14), and 0.00m/s (95% CI: -0.09, 0.09) in 4-y progression in cf-PWV, respectively. The associations were similar when using 1,000-m buffers.
CONCLUSIONS	We did not observe any consistent association between residential surrounding greenspace and arterial stiffness. https://doi.org/10.1289/EHP6159.

표-826. PubMed 논문번호 32590280의 내용 요약

구분	내용
PubMed ID	32590280
TITLE	Long-term exposure to ambient source-specific particulate matter and its components and incidence of cardiovascular events - The Heinz Nixdorf Recall study.
JOURNAL	Environment international: 10.1016/j.envint.2020.105854
AUTHORS	Rodins Vitalijs, Lucht Sarah, Ohlwein Simone, Hennig Frauke, Soppa Vanessa, Erbel Raimund, J□ckel Karl-Heinz, Weimar Christian, Hermann Dirk M, Schramm Sara, Moebus Susanne, Slomiany Uta, Hoffmann Barbara
BACKGROUND	Few studies have examined the risk of long-term exposure to source-specific airborne pollutants on incidence of cerebrovascular and cardiovascular events.
OBJECTIVES	We aimed to estimate the effect of long-term exposure to source-specific air pollution and particulate matter (PM) components on incidence of stroke, coronary heart disease (CHD), and total cardiovascular events (CVE) in the population-based Heinz Nixdorf Recall study (HNR).
METHODS	We used baseline (2000-2003) and 14-year follow-up data of the HNR Study, an ongoing population-based prospective cohort study in Western Germany. Participants' residential mean exposures to NO2 and total and source-specific PM10, PM2.5, accumulation mode particle number concentration (PNAM), and PM components were modelled using a dispersion and chemical transport model. We used Cox regression to evaluate the effect of pollutants (per 1 μg/m3 increase and per interquartile range - IQR) on risk of stroke and CHD, adjusting for socio-demographic characteristics, lifestyle risk factors and nighttime traffic noise exposure.
RESULTS	In 4,105 included participants (aged 45-76 at baseline, 52.5% women), we observed 118 cases of first stroke and 373 cases of first CHD during 46,748 person-years under risk. The median survival time within the cohort was 13.3 years. No effect of exposure to ambient air pollution on risk of CHD was observed, but distinct effects were observed for stroke. Ambient traffic-specific PM showed a stronger effect on stroke than industry-specific PM: hazard ratios (95% confidence interval) for total, traffic-specific, and industry-specific PM2.5 were 1.16 (1.02-1.34), 2.53 (1.07-5.97), and 1.27 (1.03-1.56) per 1 μg/m3 increase, respectively. PM components showed no substantially different effects from those of total PM per IQR, but higher associations were observed for NH4 and SO4 per 1 μg/m3. However, the exposure contrast of ammonium and sulfate components was very low.
CONCLUSION	Traffic-specific PM exhibited stronger effects than total and industry-specific PM on risk of stroke. Among components, NH4 and SO4 showed higher effects. No effect was observed for PM and CHD.

표-827. PubMed 논문번호 32590284의 내용 요약

구분	내용
PubMed ID	32590284
TITLE	Short-term exposure to particulate matter (PM₁₀ and PM_{2.5}), nitrogen dioxide (NO₂), and ozone (O₃) and all-cause and cause-specific mortality: Systematic review and meta-analysis.
JOURNAL	Environment international: 10.1016/j.envint.2020.105876
AUTHORS	Orellano Pablo, Reynoso Julieta, Quaranta Nancy, Bardach Ariel, Ciapponi Agustin
BACKGROUND	Air pollution is a leading cause of mortality and morbidity worldwide. Short-term exposure (from one hour to days) to selected air pollutants has been associated with human mortality. This systematic review was conducted to analyse the evidence on the effects of short-term exposure to particulate matter with aerodynamic diameters less or equal than 10 and 2.5 □m (PM10, PM2.5), nitrogen dioxide (NO2), and ozone (O3), on all-cause mortality, and PM10 and PM2.5 on cardiovascular, respiratory, and cerebrovascular mortality.
METHODS	We included studies on human populations exposed to outdoor air pollution from any source, excluding occupational exposures. Relative risks (RRs) per 10 □g/m3 increase in air pollutants concentrations were used as the effect estimates. Heterogeneity between studies was assessed using 80% prediction intervals. Risk of bias (RoB) in individual studies was analysed using a new domain-based assessment tool, developed by a working group convened by the World Health Organization and designed specifically to evaluate RoB within eligible air pollution studies included in systematic reviews. We conducted subgroup and sensitivity analyses by age, sex, continent, study design, single or multicity studies, time lag, and RoB. The certainty of evidence was assessed for each exposure-outcome combination. The protocol for this review was registered with PROSPERO (CRD42018087749).
RESULTS	We included 196 articles in quantitative analysis. All combinations of pollutants and all-cause and cause-specific mortality were positively associated in the main analysis, and in a wide range of sensitivity analyses. The only exception was NO2, but when considering a 1-hour maximum exposure. We found positive associations between pollutants and all-cause mortality for PM10 (RR: 1.0041; 95% CI: 1.0034-1.0049), PM2.5 (RR: 1.0065; 95% CI: 1.0044-1.0086), NO2 (24-hour average) (RR: 1.0072; 95% CI: 1.0059-1.0085), and O3 (RR: 1.0043; 95% CI: 1.0034-1.0052). PM10 and PM2.5 were also positively associated with cardiovascular, respiratory, and cerebrovascular mortality. We found some degree of heterogeneity between studies in three exposure-outcome combinations, and this heterogeneity could not be explained after subgroup analysis. RoB was low or moderate in the majority of articles. The certainty of evidence was judged as high in 10 out of 11 combinations, and moderate in one combination.
CONCLUSIONS	This study found evidence of a positive association between short-term exposure to PM10, PM2.5, NO2, and O3 and all-cause mortality, and between PM10 and PM2.5 and cardiovascular, respiratory and cerebrovascular mortality. These results were robust through several sensitivity analyses. In general, the level of evidence was high, meaning that we can be confident in the associations found in this study.

표-828. PubMed 논문번호 32594998의 내용 요약

구분	내용
PubMed ID	32594998
TITLE	Associations of long-term exposure to air pollutants, physical activity and platelet traits of cardiovascular risk in a rural Chinese population.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2020.140182
AUTHORS	Hou Jian, Duan Yanying, Liu Xiaotian, Li Ruiying, Tu Runqi, Pan Mingming, Dong Xiaokang, Mao Zhenxing, Huo Wenqian, Chen Gongbo, Guo Yuming, Li Shanshan, Wang Chongjian
BACKGROUND	Long-term exposure to air pollutants relate to increase risk of cardiovascular diseases that may be partially attributable to platelet dysfunction. Physical activity (PA) may attenuate inflammation to modulate platelet function. Thus, this study aimed to evaluate associations of air pollutants and PA with platelet traits of cardiovascular risk.
METHODS	A total of 31,282 participants were obtained from the Henan Rural Cohort (n = 39,259). The concentrations of particulate matter (PM) (PM with an aerodynamic diameter ≤1.0 μm (PM1), ≤2.5 μm (PM2.5), or ≤10 μm (PM10)) and nitrogen dioxide (NO2) were evaluated by using a spatiotemporal model incorporated into satellites data. Independent and combined effects of air pollutants and PA on platelet traits were analyzed by linear mixed models.
RESULTS	Positive associations of PM1, PM2.5, PM10 and NO2 with platelet indices (mean platelet volume (MPV), platelet distribution width (PDW) and platelet large cell ratio (P-LCR), the MPV to platelet counts (PLT) ratio (MPVP)) were observed, whereas negative associations of PM10 and NO2 with PLT or plateletcrit (PCT) were observed; negative interaction effects of PM2.5 PM10 and NO2 and PA on MPV, PDW or P-LCR were found; negative interaction effects of PM1, PM2.5 and PM10 and PA on PCT were observed.
CONCLUSIONS	Long-term exposure to air pollutants were related to increase platelet size and these associations were attenuated by increased PA, implying that PA is a costless and affordable method to decrease adverse effects on platelet traits in relation to air pollutants.

표-829. PubMed 논문번호 32604256의 내용 요약

구분	내용
PubMed ID	32604256
TITLE	Reduced Fine Particulate Matter Air Pollution Exposures Using In-Home Portable Air Cleaners: PILOT RESULTS OF THE CARDIAC REHABILITATION AIR FILTER TRIAL (CRAFT).
JOURNAL	Journal of cardiopulmonary rehabilitation and prevention: 10.1097/HCR.0000000000000516
AUTHORS	Bard Robert L, Rubenfire Melvyn, Fink Samantha, Bryant Joseph, Wang Lu, Speth Kelly, Zhou Nina, Morishita Masako, Brook Robert D
PURPOSE	Fine particulate matter (PM2.5) air pollution is a leading risk factor for cardiovascular disease. Even low levels common to millions of Americans pose health risks. However, no study has tested protective measures such as in-home portable air cleaners (PACs) among at-risk cardiac patients. We conducted a pilot phase of the Cardiac Rehabilitation Air Filter Trial (CRAFT)-a randomized, double-blind, crossover study of outpatient cardiac rehabilitation patients at Michigan Medicine.
METHODS	During a routine visit, patients were provided with 2 PACs to run continuously for 5 d in both the bedroom and the main living space. PACs were randomized as active (with HEPA filter) versus sham. On day 4, subjects wore a personal PM2.5 monitor for 24-hr without activity restrictions. After a 1-wk washout, patients crossed over to the opposite mode.
RESULTS	Patients (n = 20; 4 women) were elderly (70.8 ± 9.6 yr) nonsmokers with cardiovascular disease living near the facility (10.7 ± 6.0 mi). Compared with sham, active in-home PAC use significantly lowered personal-level 24-hr PM2.5 exposures by 43.8% (-12.2 μg·m; 95% CI, -24.2 to -0.2). Sensitivity analyses corroborated the reductions in most patients.
CONCLUSION	An inexpensive in-home PAC can effectively lower personal PM2.5 exposures in cardiac patients. These benefits occurred even in a region with overall good air quality and if maintained over the long-term could translate into major reductions in cardiovascular events.

표-830. PubMed 논문번호 32619910의 내용 요약

구분	내용
PubMed ID	32619910
TITLE	Fine particulate matter constituents and cause-specific mortality in China: A nationwide modelling study.
JOURNAL	Environment international: 10.1016/j.envint.2020.105927
AUTHORS	Yang Jun, Zhou Maigeng, Li Mengmeng, Yin Peng, Hu Jianlin, Zhang Chunlin, Wang Hao, Liu Qiyong, Wang Boguang
BACKGROUND	Fine particulate matter (with aerodynamic diameter ≤2.5 □m, PM2.5) causes huge disease burden worldwide. However, evidence is still inadequate and inconsistent on the relationships between PM2.5 constituents and mortality, especially in low resource settings.
OBJECTIVES	To evaluate the impact of PM2.5 constituents on cause-specific mortality in China.
METHODS	We obtained daily mortality data for 161 communities in 2011-2013 from the Disease Surveillance Point system in China. Daily concentrations of major PM2.5 constituents, including organic carbon (OC), elemental carbon (EC), sulphate (SO42-), nitrate (NO3-) and ammonium (NH4+), were estimated by using the modified Community Multiscale Air Quality model. For each community, we applied quasi-Poisson regression and polynomial distributed lag models to estimate the effects of PM2.5 constituents on cause-specific mortality. Then, the pooled effect estimates were calculated by a random-effect meta-analysis based on the restricted maximum likelihood estimation. Stratification analyses were performed by region, gender, age group and education level to identify the vulnerable populations.
RESULTS	Each interquartile range change of EC, OC, SO42-, NO3- and NH4+ at lag 0-3 day was associated with increments in non-accidental mortality of 0.45% (95%CI: 0.21, 0.69), 1.43% (0.97, 1.89), 0.71% (0.28, 1.15), 0.70% (0.10, 1.30) and 0.95% (0.39, 1.51), respectively. The associations were stronger for the deaths from cardiovascular disease and myocardial infarction, the elderly, illiterates, and people living in the South region.
CONCLUSIONS	Our findings suggest positive associations between PM2.5 constituents and cause-specific mortality, particularly for myocardial infarction.

표-831. PubMed 논문번호 32634667의 내용 요약

구분	내용
PubMed ID	32634667
TITLE	Short-term exposure to carbon monoxide and myocardial infarction: A systematic review and meta-analysis.
JOURNAL	Environment international: 10.1016/j.envint.2020.105901
AUTHORS	Lee Kuan Ken, Spath Nicholas, Miller Mark R, Mills Nicholas L, Shah Anoop S V
BACKGROUND	Previous studies suggest an association between short-term exposure to carbon monoxide and myocardial infarction. We performed a systematic review and meta-analysis to assess current evidence on this association to support the update of the World Health Organization (WHO) Global Air Quality Guidelines.
METHODS	We searched Medline, Embase and Cochrane Central Register of Controlled Trials to update the evidence published in a previous systematic review up to 30th September 2018 for studies investigating the association between short-term exposure to ambient carbon monoxide (up to lag of seven days) and emergency department visits or hospital admissions and mortality due to myocardial infarction. Two reviewers assessed potentially eligible studies and performed data extraction independently. Random-effects meta-analysis was used to derive the pooled risk estimate per 1 mg/m3 increase in ambient carbon monoxide concentration. Risk of bias in individual studies was assessed using a domain-based assessment tool. The overall certainty of the body of evidence was evaluated using an adapted certainty of evidence assessment framework.
RESULTS	We evaluated 1,038 articles from the previous review and our updated literature search, of which, 26 satisfied our inclusion criteria. Overall, myocardial infarction was associated with exposure to ambient carbon monoxide concentration (risk ratio of 1.052, 95% confidence interval 1.017-1.089 per 1 mg/m3 increase). A third of studies were assessed to be at high risk of bias (RoB) due to inadequate adjustment for confounding. Using an adaptation of the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework, the overall evidence was assessed to be of moderate certainty.
CONCLUSIONS	This review demonstrated that the pooled risk ratio for myocardial infarction was 1.052 (95% CI 1.017-1.089) per 1 mg/m3 increase in ambient carbon monoxide concentration. However, very few studies originated from low- and middle-income countries.

표-832. PubMed 논문번호 32641588의 내용 요약

구분	내용
PubMed ID	32641588
TITLE	Long-Term Exposure to Particulate Matter and Mortality from Cardiovascular Diseases in Japan: The Ibaraki Prefectural Health Study (IPHS).
JOURNAL	Journal of atherosclerosis and thrombosis: 10.5551/jat.54148
AUTHORS	Takeuchi Ayano, Nishiwaki Yuji, Okamura Tomonori, Milojevic Ai, Ueda Kayo, Asakura Keiko, Takebayashi Toru, Hasegawa Shuichi, Sairenchi Toshimi, Irie Fujiko, Ota Hitoshi, Nitta Hiroshi
AIM	To examine the association between long-term exposure to suspended particulate matter (SPM) and cardiovascular mortality in Japan after controlling for known major confounding factors among a large middle and elderly cohort study in Ibaraki Prefecture, Japan.
METHODS	We followed 91,808 residents (men 34%) who undertook a national health check-up at age 40-79 years for 17 years (1993-2010). Two different exposure indices were adopted: baseline SPM concentration (in the year 1990) and average SPM concentration for the first (average of 1990 and 1995) and the second half (average of 2005 to 2009) of the study period. Sex-specific adjusted risk ratios (RRs) for cardiovascular mortality were calculated using general mixed Poisson regression models after adjusting the age, BMI, history of diabetes mellitus and hypertension, creatinine, glutamic pyruvic transaminase, total cholesterol, high-density lipoprotein cholesterol, smoking, alcohol, and temperature. The variation between seven medical administration areas was also taken into account as a random effect.
RESULTS	Baseline SPM concentration was associated with an increased risk of mortality from all cardiovascular diseases, coronary artery disease, and stroke. The adjusted RRs (95% confidence interval [CI]) per 10 □g/m3 increase in SPM concentration for all cardiovascular mortality were 1.147 (1.014-1.300) for men and 1.097 (0.985-1.222) for women. The point estimate of RR was highest for non-hemorrhagic stroke in men (1.248 [0.991-1.571]), although CI overlapped the unity. The RRs seemed slightly lower in the second half than in the first half, though the CIs widened in the second half.
CONCLUSION	Our results suggest that long-term exposure to SPM is associated with an increased risk of all cardiovascular mortality for men in Ibaraki, Japan.

표-833. PubMed 논문번호 32644760의 내용 요약

구분	내용
PubMed ID	32644760
TITLE	Disease assimilation: The mortality impacts of fine particulate matter on immigrants to Canada.
JOURNAL	Health reports: 10.25318/82-003-x202000300002-eng
AUTHORS	Erickson Anders C, Christidis Tanya, Pappin Amanda, Brook Jeffrey R, Crouse Daniel L, Hystad Perry, Li Chi, Martin Randall V, Meng Jun, Pinault Lauren, von Donkelaar Aaron, Weichenthal Scott, Tjepkema Michael, Burnett Richard T, Brauer Michael
BACKGROUND	Immigrants make up 20% of the Canadian population; however, little is known about the mortality impacts of fine particulate matter (PM2.5) air pollution on immigrants compared with non-immigrants, or about how impacts may change with duration in Canada.
DATA AND METHODS	This study used the 2001 Canadian Census Health and Environment Cohort, a longitudinal cohort of 3.5 million individuals, of which 764,000 were classified as immigrants (foreign-born). Postal codes from annual income tax files were used to account for mobility among respondents and to assign annual PM2.5 concentrations from 1998 to 2016. Exposures were estimated as a three-year moving average prior to the follow-up year. Cox survival models were used to determine hazard ratios (HRs) for cause-specific mortality, comparing the Canadian and foreign-born populations, with further stratification by year of immigration grouped into 10-year cohorts.
RESULTS	Differences in urban-rural settlement patterns resulted in greater exposure to PM2.5 for immigrants compared with non-immigrants (mean = 9.3 vs. 7.5 μg/m3), with higher exposures among more recent immigrants. In fully adjusted models, immigrants had higher HRs per 10 μg/m3 increase in PM2.5 concentration compared with Canadian-born individuals for cardiovascular mortality (HR [95% confidence interval] = 1.22 [1.12 to 1.34] vs. 1.12 [1.07 to 1.18]) and cerebrovascular mortality (HR = 1.25 [1.03 to 1.52] vs. 1.03 [0.93 to 1.15]), respectively. However, tests for differences between the two groups were not significant when Cochran's Q test was used. No significant associations were found for respiratory outcomes, except for lung cancer in non-immigrants (HR = 1.10 [1.02 to 1.18]). When stratified by year of immigration, differences in HRs across varied by cause of death.
DISCUSSION	In Canada, PM2.5 is an equal-opportunity risk factor, with immigrants experiencing similar if not higher mortality risks compared with non-immigrants for cardiovascular-related causes of death. Some notable differences also existed with cerebrovascular and lung cancer deaths. Continued reductions in air pollution, particularly in urban areas, will improve the health of the Canadian population as a whole.

표-834. PubMed 논문번호 32682146의 내용 요약

구분	내용
PubMed ID	32682146
TITLE	Associations between particulate matter air pollution, presence and progression of subclinical coronary and carotid atherosclerosis: A systematic review.
JOURNAL	Atherosclerosis: 10.1016/j.atherosclerosis.2020.06.018
AUTHORS	Jilani Mohammad Hashim, Simon-Friedt Bridget, Yahya Tamer, Khan Ali Younas, Hassan Syed Z, Kash Bita, Blankstein Ron, Blaha Michael J, Virani Salim S, Rajagopalan Sanjay, Cainzos-Achirica Miguel, Nasir Khurram
BACKGROUND AND AIMS	Long-term exposure to particulate matter (PM) air pollution has been linked with increased cardiovascular events and mortality, however, studies had shown inconsistent associations between PM exposure and subclinical atherosclerosis.
METHODS	We performed an updated systematic literature review to identify studies evaluating the associations between PM and subclinical atherosclerosis, measured using presence/progression of coronary artery calcium (CAC) or carotid intima-media thickness (CIMT) in adult populations. Quality was assessed using the Newcastle-Ottawa scale.
RESULTS	Eighteen studies were included: 5 cohorts and 13 cross-sectional. Amongst 7 studies that evaluated the associations between PM and prevalence of CAC, 4 reported significantly higher odds of detectable CAC>0 or CAC>400 with increased PM exposure. Nine studies evaluated the association between exposure to at least one of the particulates and CIMT; of these, 6 reported significant independent associations. Two studies evaluated PM2.5 and CAC progression, with 1 reporting a greater progression of CAC with increased exposure to PM, while 3 out of 4 studies evaluating CIMT progression showed no significant difference in CIMT progression with a higher PM2.5 exposure. Additionally, 3 studies found significant associations between proximity to major roadways and measures of subclinical atherosclerosis. Among null studies, most displayed non-significant trends towards higher atherosclerosis burden with higher PM exposure.
CONCLUSIONS	Overall, available observational studies support a positive association between PM exposure and subclinical atherosclerosis. Further longitudinal studies are needed to better establish this relationship and assess the efficacy of previously identified interventions on mitigation of clinical cardiovascular disease.

표-835. PubMed 논문번호 32686482의 내용 요약

구분	내용
PubMed ID	32686482
TITLE	Independent and Opposing Associations of Habitual Exercise and Chronic PM〈sub〉2.5〈/sub〉 Exposures on Hypertension Incidence.
JOURNAL	Circulation: 10.1161/CIRCULATIONAHA.120.045915
AUTHORS	Guo Cui, Zeng Yiqian, Chang Ly-Yun, Yu Zengli, Bo Yacong, Lin Changqing, Lau Alexis Kh, Tam Tony, Lao Xiang Qian
BACKGROUND	We investigated the joint associations of habitual physical activity (PA) and long-term exposure to fine particulate matter (PM2.5) with the development of hypertension in a longitudinal cohort in Taiwan.
METHODS	We selected 140 072 adults (≥18 years of age) without hypertension who joined a standard medical screening program with 360 905 medical examinations between 2001 and 2016. PM2.5 exposure was estimated at each participant's address using a satellite data-based spatiotemporal model with 1 km2 resolution. Information on habitual PA and a wide range of covariates was collected using a standard self-administered questionnaire. We used the Cox regression model with time-dependent covariates to examine the joint associations.
RESULTS	The mean age of all observations was 41.7 years, and 48.8% were male. The mean value for systolic and diastolic blood pressure was 112.5 and 68.7mm Hg, respectively. Approximately 34.2% of all observations were inactive (0 metabolic equivalence values-hours), 29.8% had moderate-PA (median [interquartile range]; 3.75 [3.38 to 4.38] metabolic equivalence values-hours), and 36.0% had high-PA (15.7 [10.3 to 24.8] metabolic equivalence values-hours). The mean±SD of PM2.5 was 26.1±7.3 μg/m3. The prevalence of cardiovascular disease, diabetes mellitus, and cancer was 2.1%, 2.9%, and 1.5%, respectively. After adjusting for a wide range of covariates (including a mutual adjustment for PA or PM2.5), a higher PA level was associated with a lower risk of hypertension (hazard ratio [HR] for the moderate- and high-PA was 0.93 [95% CI, 0.89-0.97] and 0.92 [95% CI, 0.88-0.96], respectively, as compared with the inactive-PA), whereas a higher level of PM2.5 was associated with a higher risk of hypertension (HR for the moderate- and high-PM2.5 was 1.37 [95% CI, 1.32-1.43] and 1.92 [95% CI, 1.81-2.04], respectively, as compared with the low-PM2.5 group]. No significant interaction was observed between PA and PM2.5 (HR 1.01 [95% CI, 1.00-1.02]).
CONCLUSIONS	A high-PA and low PM2.5 exposure were associated with a lower risk of hypertension. The negative association between PA and hypertension remained stable in people exposed to various levels of PM2.5, and the positive association between PM2.5 and hypertension was not modified by PA. Our results indicated that PA is a suitable hypertension prevention strategy for people residing in relatively polluted regions.

표-836. PubMed 논문번호 32711561의 내용 요약

구분	내용
PubMed ID	32711561
TITLE	Impact of airborne particulate matter on skin: a systematic review from epidemiology to in vitro studies.
JOURNAL	Particle and fibre toxicology: 10.1186/s12989-020-00366-y
AUTHORS	Dijkhoff Irini M, Drasler Barbara, Karakocak Bedia Begum, Petri-Fink Alke, Valacchi Giuseppe, Eeman Marc, Rothen-Rutishauser Barbara
BACKGROUND	Air pollution is killing close to 5 million people a year, and harming billions more. Air pollution levels remain extremely high in many parts of the world, and air pollution-associated premature deaths have been reported for urbanized areas, particularly linked to the presence of airborne nano-sized and ultrafine particles.
MAIN TEXT	To date, most of the research studies did focus on the adverse effects of air pollution on the human cardiovascular and respiratory systems. Although the skin is in direct contact with air pollutants, their damaging effects on the skin are still under investigation. Epidemiological data suggested a correlation between exposure to air pollutants and aggravation of symptoms of chronic immunological skin diseases. In this study, a systematic literature review was conducted to understand the current knowledge on the effects of airborne particulate matter on human skin. It aims at providing a deeper understanding of the interactions between air pollutants and skin to further assess their potential risks for human health.
CONCLUSION	Particulate matter was shown to induce a skin barrier dysfunction and provoke the formation of reactive oxygen species through direct and indirect mechanisms, leading to oxidative stress and induced activation of the inflammatory cascade in human skin. Moreover, a positive correlation was reported between extrinsic aging and atopic eczema relative risk with increasing particulate matter exposure.

표-837. PubMed 논문번호 32718236의 내용 요약

구분	내용
PubMed ID	32718236
TITLE	Life-threatening and major cardiac events during long-distance races: updates from the prospective RACE PARIS registry with a systematic review and meta-analysis.
JOURNAL	European journal of preventive cardiology: 10.1177/2047487320943001
AUTHORS	Gerardin Beno□t, Guedeney Paul, Bellemain-Appaix Anne, Levasseur Thomas, Mustafic Hazrije, Benamer Hakim, Monsegu Jacques, Lamhaut Lionel, Montalescot Gilles, Aubry Pierre, Collet Jean-Philippe
AIMS	Limited data exist regarding the incidence and aetiology of life-threatening events such as major cardiac events or exertional heat stroke during long-distance races. We aimed to provide an updated incidence, etiology and prognosis of life-threatening events during long-distance races.
METHODS	The prospective RACE PARIS registry recorded all life-threatening events/fatal events occurring during 46 marathons, half-marathons and other long-distance races in the Paris area between 2006 and 2016, comprising 1,073,722 runners. Event characteristics were determined by review of medical records and interviews with survivors.
RESULTS	The incidence of life-threatening events, exertional heat stroke and major cardiac events was 3.35 per 100,000, 1.02 per 100,000 and 2.33 per 100,000, respectively, including 18 sudden cardiac arrests (1.67 per 100,000). The main aetiology of sudden cardiac arrest was myocardial ischaemia (11/18), due to acute coronary thrombosis (6/11), stable atherosclerotic coronary artery disease (2/11), coronary dissection (1/11), anomalous connection (1/11) or myocardial bridging (1/11). A third of participants with ischaemia-related major cardiac events presented with pre-race clinical symptoms. Major cardiac events were more frequent in the case of a high pollution index (6.78 per 100,000 vs. 2.07 per 100,000, odds ratio 3.27, 95% confidence interval 1.12-9.54). Case fatality was low (0.19 per 100,000). Similarly, we report in a meta-analysis of eight long-distance race registries comprising 16,223,866 runners a low incidence of long-distance race-related sudden cardiac arrest (0.82 per 100,000) and fatality (0.39 per 100,000). Death following sudden cardiac arrest was strongly associated with initial asystole or pulseless rhythm.
CONCLUSION	Long-distance race-related life-threatening events remain rare although serious events. Better information for runners on the risk of pre-race clinical symptoms, outside air pollution and temperature may reduce their incidence.

표-838. PubMed 논문번호 32726349의 내용 요약

구분	내용
PubMed ID	32726349
TITLE	Cooking fuels use and carotid intima-media thickness during early pregnancy of women in Myanmar.
JOURNAL	PloS one: 10.1371/journal.pone.0236151
AUTHORS	Min Myo, Taneepanichskul Nutta
BACKGROUND	Fuels burned in households for cooking cause indoor air pollution, exposing those who are cooking. Despite the mounting evidence of the effects of fuels use on health, few studies focus on the effect of cooking fuels have on carotid intima-media thickness (CIMT), a surrogate atherosclerosis biomarker in the early stages of pregnancy. This study aimed to examine the association between the use of cooking fuels and CIMT during early trimester of pregnancy among cooking women in Myanmar.
METHODOLOGY	In this cross-sectional study, a part of an ongoing birth cohort analysis, a total of 192 cooking pregnant women over 18 years with gestational weeks less than 18 were recruited from 15 rural health centers in Nay Pyi Taw from September to November 2019. Sociodemographic data, residential data, and fuels use data were collected with semi-structured questionnaires in face-to-face interviews. Anthropometric, hemodynamic, blood lipids, and ultrasound CIMT measurements were performed under standard protocols. Multiple linear regression was modeled to explore associations.
RESULTS	The study included 70 firewood fuel users, 26 charcoal fuel users, and 96 electricity fuel users. Following adjustments for potential confounding factors, firewood use was significantly associated with the increase of all CIMT analyzed. Importantly, a greater increase of mean CIMT of the right common carotid artery (RCCA; β = 0.033 mm; 95%CI: 0.006, 0.058; P<0.05) had significant association with charcoal use compared to firewood use (β = 0.029 mm; 95%CI: 0.010, 0.049; P<0.05).
CONCLUSIONS	Our findings demonstrate that the indoor use of cooking fuels that cause indoor air pollution, such as firewood and charcoal, is a considerable risk factor for human health and is associated with increased CIMT, wherein charcoal use contributes to more increase of mean CIMT of the RCCA. Measures to prevent health risks related to the use of such fuels should be instituted early on during pregnancy.

표-839. PubMed 논문번호 32736159의 내용 요약

구분	내용
PubMed ID	32736159
TITLE	Long-term exposure to low levels of air pollution and mortality adjusting for road traffic noise: A Danish Nurse Cohort study.
JOURNAL	Environment international: 10.1016/j.envint.2020.105983
AUTHORS	So Rina, J ø rgensen Jeanette Therming, Lim Youn-Hee, Mehta Amar J, Amini Heresh, Mortensen Laust H, Westendorp Rudi, Ketzel Matthias, Hertel Ole, Brandt J ø rgen, Christensen Jesper H, Geels Camilla, Frohn Lise M, Sisgaard Torben, Br□uner Elvira Vaclavik, Jensen Steen Solvang, Backalarz Claus, Simonsen Mette Kildevæld, Loft Steffen, Cole-Hunter Tom, Andersen Zorana Jovanovic
BACKGROUND	The association between air pollution and mortality is well established, yet some uncertainties remain: there are few studies that account for road traffic noise exposure or that consider in detail the shape of the exposure-response function for cause-specific mortality outcomes, especially at low-levels of exposure.
OBJECTIVES	We examined the association between long-term exposure to particulate matter [(PM) with a diameter of 〈2.5 □m (PM2.5), 〈10 □m (PM10)], and nitrogen dioxide (NO2) and total and cause-specific mortality, accounting for road traffic noise.
METHODS	We used data on 24,541 females (age 〉 44 years) from the Danish Nurse Cohort, who were recruited in 1993 or 1999, and linked to the Danish Causes of Death Register for follow-up on date of death and its cause, until the end of 2013. Annual mean concentrations of PM2.5, PM10, and NO2 at the participants' residences since 1990 were estimated using the Danish DEHM/UBM/AirGIS dispersion model, and annual mean road traffic noise levels (Lden) were estimated using the Nord2000 model. We examined associations between the three-year running mean of PM2.5, PM10, and NO2 with total and cause-specific mortality by using time-varying Cox Regression models, adjusting for individual characteristics and residential road traffic noise.
RESULTS	During the study period, 3,708 nurses died: 843 from cardiovascular disease (CVD), 310 from respiratory disease (RD), and 64 from diabetes. In the fully adjusted models, including road traffic noise, we detected associations of three-year running mean of PM2.5 with total (hazard ratio; 95% confidence interval: 1.06; 1.01-1.11), CVD (1.14; 1.03-1.26), and diabetes mortality (1.41; 1.05-1.90), per interquartile range of 4.39 μg/m3. In a subset of the cohort exposed to PM2.5 〈 20 □g/m3, we found even stronger association with total (1.19; 1.11-1.27), CVD (1.27; 1.01-1.46), RD (1.27; 1.00-1.60), and diabetes mortality (1.44; 0.83-2.48). We found similar associations with PM10 and none with NO2. All associations were robust to adjustment for road traffic noise.
DISCUSSION	Long-term exposure to low-levels of PM2.5 and PM10 is associated with total mortality, and mortality from CVD, RD, and diabetes. Associations were even stronger at the PM2.5 levels below EU limit values and were independent of road traffic noise.

표-840. PubMed 논문번호 32738766의 내용 요약

구분	내용
PubMed ID	32738766
TITLE	Associations of long-term exposure to ambient air pollution with cardiac conduction abnormalities in Chinese adults: The CHCN-BTH cohort study.
JOURNAL	Environment international: 10.1016/j.envint.2020.105981
AUTHORS	Cao Han, Li Bingxiao, Peng Wenjuan, Pan Li, Cui Ze, Zhao Wei, Zhang Han, Tang Naijun, Niu Kaijun, Sun Jixin, Han Xiaoyan, Wang Zhengfang, Liu Kuo, He Huijing, Cao Yajing, Xu Zhiyuan, Shan Anqi, Meng Ge, Sun Yanyan, Guo Chunyue, Liu Xiaohui, Xie Yunyi, Wen Fuyuan, Shan Guangliang, Zhang Ling
BACKGROUND	Evidence regarding the effects of long-term and high-level ambient air pollution exposure on cardiac conduction systems remains sparse.
OBJECTIVES	To investigate the associations of long-term exposure to air pollution and cardiac conduction abnormalities in Chinese adults and explore the susceptibility characteristics.
METHODS	In 2017, a total of 27,047 participants aged 18-80 years were recruited from the baseline survey of the Cohort Study on Chronic Disease of Communities Natural Population in Beijing, Tianjin and Hebei (CHCN-BTH). The three year (2014-2016) average pollutant concentrations were assessed by a spatial statistical model for PM2.5 and air monitoring stations for PM10, SO2, NO2, O3 and CO. Residential proximity to a roadway was calculated by neighborhood analysis. Associations were estimated by two-level generalized linear mixed models. Stratified analyses related to demographic characteristics, health behaviors, and cardiometabolic risk factors were performed. Two-pollutant models were used to evaluate the possible role of single pollutants.
RESULTS	We detected significant associations of long-term air pollutant exposure with increased heart rate (HR), QRS and QTc, such that an interquartile range increase in PM2.5 was associated with 3.63% (95% CI: 3.07%, 4.19%), 1.21% (95% CI: 0.83%, 1.60%), and 0.13% (95% CI: 0.07%, 0.18%) changes in HR, QRS and QTc, respectively. Compared to the other pollutants, the estimates of PM2.5 remained the most stable across all two-pollutant models. Similarly, significant associations were observed between living closer to a major roadway and higher HR, QRS and QTc. Stratified analyses showed generally greater association estimates in older people, males, smokers, alcohol drinkers, and those with obesity, hypertension and diabetes.
CONCLUSIONS	Long-term exposure to ambient air pollution was associated with cardiac conduction abnormalities in Chinese adults, especially in older people, males, smokers, alcohol drinkers, and those with cardiometabolic risk factors. PM2.5 may be the most stable pollutant to reflect the associations.

표-841. PubMed 논문번호 32777785의 내용 요약

구분	내용
PubMed ID	32777785
TITLE	Short-Term Air Pollution as a Risk for Stroke Admission: A Time-Series Analysis.
JOURNAL	Cerebrovascular diseases (Basel, Switzerland): 10.1159/000510080
AUTHORS	Byrne Colm Patrick, Bennett Kathleen E, Hickey Anne, Kavanagh Paul, Broderick Brian, O'Mahony Margaret, Williams David J
BACKGROUND	The harmful effects of outdoor air pollution on stroke incidence are becoming increasingly recognised. We examined the impact of different air pollutants (PM2.5, PM10, NO2, ozone, and SO2) on admission for all strokes in two Irish urban centres from 2013 to 2017.
METHODS	Using an ecological time series design with Poisson regression models, we analysed daily hospitalisation for all strokes and is-chaemic stroke by residence in Dublin or Cork, with air pollution level monitoring data with a lag of 0-2 days from exposure. Splines of temperature, relative humidity, day of the week, and time were included as confounders. Analysis was also performed across all four seasons. Data are presented as relative risks (RRs) and 95% confidence intervals (95% CI) per interquartile range (IQR) increase in each pollutant.
RESULTS	There was no significant association between all stroke admission and any individual air pollutant. On seasonal analysis, during winter in the larger urban centre (Dublin), we found an association between all stroke cases and an IQR increase in NO2 (RR 1.035, 95% CI: 1.003-1.069), PM10 (RR 1.032, 95% CI: 1.007-1.057), PM2.5 (RR 1.024, 95% CI: 1.011-1.039), and SO2 (RR 1.035, 95% CI: 1.001-1.071). There was no significant association found in the smaller urban area of Cork. On meta-analysis, there remained a significant association between NO2 (RR 1.013, 95% CI: 1.001-1.024) and PM2.5 (1.009, 95% CI 1.004-1.014) per IQR increase in each.
DISCUSSION	Short-term air pollution in winter was found to be associated with hospitalisation for all strokes in a large urban centre in Ireland. As Ireland has relatively low air pollution internationally, this highlights the need to introduce policy changes to reduce air pollution in all countries.

표-842. PubMed 논문번호 32783534의 내용 요약

구분	내용
PubMed ID	32783534
TITLE	Exposure to Road Traffic Noise and Incidence of Acute Myocardial Infarction and Congestive Heart Failure: A Population-Based Cohort Study in Toronto, Canada.
JOURNAL	Environmental health perspectives: 10.1289/EHP5809
AUTHORS	Bai Li, Shin Saeha, Oiamo Tor H, Burnett Richard T, Weichenthal Scott, Jerrett Michael, Kwong Jeffrey C, Copes Ray, Kopp Alexander, Chen Hong
BACKGROUND	Epidemiological evidence for the association between traffic-related noise and the incidence of major cardiovascular events such as acute myocardial infarction (AMI) and congestive heart failure (CHF) is inconclusive, especially in North America.
OBJECTIVES	We evaluated the associations between long-term exposure to road traffic noise and the incidence of AMI and CHF.
METHODS	Our study population comprised ~1 million people 30-100 years of age who lived in Toronto, Canada, from 2001 to 2015 and were free of AMI (referred to as the AMI cohort) or CHF (the CHF cohort) at baseline. Outcomes were ascertained from health administrative databases using validated algorithms. Annual average noise levels were estimated as the A-weighted equivalent sound pressure level over the 24-h period (LAeq24) and during nighttime (LAeqNight), respectively, using propagation modeling, and assigned to participants' annual six-digit postal code addresses during follow-up. We calculated hazard ratios (HRs) and 95% confidence intervals (CIs) for incident AMI and CHF in relation to LAeq24 and LAeqNight using random-effects Cox proportional hazards models adjusting for individual- and census tract-level covariates, including traffic-related air pollutants [e.g., ultrafine particles (UFPs) and nitrogen dioxide].
RESULTS	During follow-up, there were 37,441 AMI incident cases and 95,138 CHF incident cases. Each interquartile range change in LAeq24 was associated with an increased risk of incident AMI (HR=1.07; 95% CI: 1.06, 1.08) and CHF (HR=1.07; 95% CI: 1.06, 1.09). Similarly, LAeqNight was associated with incident AMI (HR=1.07; 95% CI: 1.05, 1.08) and CHF (HR=1.06; 95% CI: 1.05, 1.07). These results were robust to various sensitivity analyses and remained elevated after controlling for long-term exposure to UFPs and nitrogen dioxide. We found near-linear relationships between noise and the incidence of AMI and CHF with no evidence of threshold values.
CONCLUSION	In this large cohort study in Toronto, Canada, chronic exposure to road traffic noise was associated with elevated risks for AMI and CHF incidence. https://doi.org/10.1289/EHP5809.

표-843. PubMed 논문번호 32783836의 내용 요약

구분	내용
PubMed ID	32783836
TITLE	Short-term PM〈sub〉2.5〈/sub〉 exposure and circulating von Willebrand factor level: a meta-analysis.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2020.140180
AUTHORS	Liang Qingqing, Sun Mengqi, Wang Fenghong, Ma Yuexiao, Lin Lisen, Li Tianyu, Duan Junchao, Sun Zhiwei
BACKGROUND	Ambient fine particulate matter (PM2.5) is a major threat to cardiovascular health. Endothelial dysfunction is the initiating event associated with the PM2.5-induced cardiovascular disease (CVD). A sensitive marker of endothelial function-circulating von Willebrand factor (vWF), is an independent predictor of adverse clinical outcome in CVD patients. PM2.5 exposure may cause CVD, but the reports of relationship between short-term PM2.5 exposure and circulating vWF are inconsistent.
OBJECTIVE	To explore the influence of short-term PM2.5 exposure on circulating vWF.
METHODS	By using a combination of computer and manual retrieval, a systematic literature retrieval was conducted on PubMed, Cochrane Library, Web of Science, Embase and Scopus databases up to October 2019. The heterogeneity among studies was tested by Stata 12.0, and the pooled %-change (percentage change per 10 μg/m3 increase in PM2.5) and its 95% confidence interval (95%CI) were calculated by using random effect model. Sensitivity analysis and publication bias detection were also carried out.
RESULTS	12 articles were included in this meta-analysis. Short-term PM2.5 exposure (per 10 μg/m3 increase) was associated with the increased vWF (%-change = 0.41, 95%CI: 0.11-0.71). The pooled effect estimates of subgroup with PM2.5 exposure level 〈 25 μg/m3 was higher (%-change = 8.26; 95%CI: 1.99-14.53) than that with PM2.5 exposure level ≥ 25 μg/m3 (%-change = 0.36; 95%CI: 0.09-0.63).
CONCLUSION	Short-term PM2.5 exposure is associated with the increased circulating vWF. It suggests that short-term PM2.5 exposure causes endothelial dysfunction.

표-844. PubMed 논문번호 32798956의 내용 요약

구분	내용
PubMed ID	32798956
TITLE	Adverse health impacts of cooking with kerosene: A multi-country analysis within the Prospective Urban and Rural Epidemiology Study.
JOURNAL	Environmental research: 10.1016/j.envres.2020.109851
AUTHORS	Arku Raphael E, Brauer Michael, Duong MyLinh, Wei Li, Hu Bo, Ah Tse Lap, Mony Prem K, Lakshmi P V M, Pillai Rajamohanan K, Mohan Viswanathan, Yeates Karen, Kruger Lanthe, Rangarajan Sumathy, Koon Teo, Yusuf Salim, Hystad Perry
BACKGROUND	Kerosene, which was until recently considered a relatively clean household fuel, is still widely used in low- and middle-income countries for cooking and lighting. However, there is little data on its health effects. We examined cardiorespiratory effects and mortality in households using kerosene as their primary cooking fuel within the Prospective Urban Rural Epidemiology (PURE) study.
METHODS	We analyzed baseline and follow-up data on 31,490 individuals from 154 communities in China, India, South Africa, and Tanzania where there was at least 10% kerosene use for cooking at baseline. Baseline comorbidities and health outcomes during follow-up (median 9.4 years) were compared between households with kerosene versus clean (gas or electricity) or solid fuel (biomass and coal) use for cooking. Multi-level marginal regression models adjusted for individual, household, and community level covariates.
RESULTS	Higher rates of prevalent respiratory symptoms (e.g. 34% [95% CI:15-57%] more dyspnea with usual activity, 44% [95% CI: 21-72%] more chronic cough or sputum) and lower lung function (differences in FEV1: -46.3 ml (95% CI: -80.5; -12.1) and FVC: -54.7 ml (95% CI: -93.6; -15.8)) were observed at baseline for kerosene compared to clean fuel users. The odds of hypertension was slightly elevated but no associations were observed for blood pressure. Prospectively, kerosene was associated with elevated risks of all-cause (HR: 1.32 (95% CI: 1.14-1.53)) and cardiovascular (HR: 1.34 (95% CI: 1.00-1.80)) mortality, as well as major fatal and incident non-fatal cardiovascular (HR: 1.34 (95% CI: 1.08-1.66)) and respiratory (HR: 1.55 (95% CI: 0.98-2.43)) diseases, compared to clean fuel use. Further, compared to solid fuel users, those using kerosene had 20-47% higher risks for the above outcomes.
CONCLUSIONS	Kerosene use for cooking was associated with higher rates of baseline respiratory morbidity and increased risk of mortality and cardiorespiratory outcomes during follow-up when compared to either clean or solid fuels. Replacing kerosene with cleaner-burning fuels for cooking is recommended.

표-845. PubMed 논문번호 32800152의 내용 요약

구분	내용
PubMed ID	32800152
TITLE	Time-weighted average of fine particulate matter exposure and cause-specific mortality in China: a nationwide analysis.
JOURNAL	The Lancet. Planetary health: 10.1016/S2542-5196(20)30164-9
AUTHORS	Dong Zhaomin, Wang Hao, Yin Peng, Wang Lijun, Chen Renjie, Fan Wenhong, Xu Yilu, Zhou Maigeng
BACKGROUND	Most previous assessments of the hazardous effects attributable to fine particulate matter (PM2·5) exposure have used ambient PM2·5 as an exposure metric, resulting in substantial bias in effect estimates. We did a study to examine the association between cause-specific mortality and the time-weighted average of PM2·5 exposure after accounting for indoor exposure in 267 cities in China.
METHODS	We did a nationwide study, using Laser Egg air quality monitors in 36 cities to obtain data for indoor PM2·5 concentrations from 18 484 anonymised households between Nov 1, 2015 and July 2, 2018. We developed and validated a nationwide indoor PM2·5 prediction model for a further 302 cities by retrieving raw records of hourly concentrations from residents' air sensors; the model was used to predict indoor PM2·5 during 2013 to 2018. Daily ambient PM2·5 concentration data were estimated by averaging hourly ambient PM2·5 concentrations obtained from China's National Urban Air Quality Real-time Publishing Platform. Daily numbers of deaths from all non-accidental causes were obtained from 324 cities from the Disease Surveillance Point System of China between Jan 1, 2013, to Dec 31, 2017, and calculated for 267 cities that had an average daily mortality above three, and data for PM2·5 concentrations and meteorological information for at least 1 year between 2013 and 2017. We used distributed lag non-linear models to estimate city-specific associations between cause-specific mortality and reconstructed PM2·5 exposure by considering indoor PM2·5 exposure. We combined the city-specific effect estimates at the national level using a random effects meta-analysis.
FINDINGS	13 972 records of daily indoor PM2·5 concentrations for 36 cities, extracted from 47 459 183 raw records from the sensors were included for modelling indoor PM2·5 levels. The nationwide indoor PM2·5 concentration was 40 μg/m3 (SD 21) between 2013 and 2017, which was approximately 20% lower than the ambient PM2·5 concentration of 50 μg/m3 (42). An increase of 10 μg/m3 in time-averaged PM2·5 exposure concentrations was associated with increased daily mortality estimates of 0·44% (95% CI 0·33-0·54) for total non-accidental causes, 0·50% (0·37-0·63) for cardiovascular diseases, 0·46% (0·28-0·63) for coronary heart disease, 0·49% (0·32-0·66) for stroke, 0·59% (0·39-0·79) for respiratory diseases, and 0·69% (0·45-0·92) for chronic obstructive pulmonary disease, respectively. Compared with previous estimations based on ambient PM2·5, our estimates approximately doubled the size of the effects related to PM2·5.
INTERPRETATION	This nationwide study revealed a higher mortality risk attributed to time-averaged indoor and ambient PM2·5 exposure compared with the risk associated with ambient PM2·5 exposure alone, which indicates that caution should be exercised when using ambient PM2·5 as a surrogate for PM2·5 exposure.
FUNDING	National Natural Science Foundation of China (Youth Program) and the Fundamental Research Project of Beihang University.

표-846. PubMed 논문번호 32810504의 내용 요약

구분	내용
PubMed ID	32810504
TITLE	Acute exposure to traffic-related air pollution alters antioxidant status in healthy adults.
JOURNAL	Environmental research: 10.1016/j.envres.2020.110027
AUTHORS	Cosselman Kristen E, Allen Jason, Jansen Karen L, Stapleton Patricia, Trenga Carol A, Larson Timothy V, Kaufman Joel D
BACKGROUND	Exposure to traffic-related air pollution is associated with an increased risk of cardiovascular and respiratory disease. Evidence suggests that inhaled pollutants precipitate these effects via multiple pathways involving oxidative stress.
OBJECTIVE	Postulating that a decrease in circulating antioxidant levels reflect an oxidative response, we investigated the effect of inhaled diesel exhaust (DE) on the ratio of reduced to oxidized glutathione (GSH/GSSG) in healthy adults, and whether pre-exposure antioxidant supplementation blunted this response. We also examined exposure-related changes in antioxidant/stress response leukocyte gene expression (GCLc, HMOX-1, IL-6, TGFβ) and plasma IL-6 levels.
METHODS	Nineteen nonsmoking adults participated in a double-blind, randomized, four-way crossover study. Each subject completed 120-min exposures to filtered air and DE (200 μg/m3), with and without antioxidant pretreatment. Antioxidant comprised 1000 mg ascorbate for 7 days and 1200 mg N-acetylcysteine 1 day prior to exposure, with 1000 mg and 600 mg, respectively, administered 2 h prior to exposure. Whole blood glutathione was measured pre- and post-exposure; plasma IL-6 and mRNA expression were quantified pre, during and post exposure.
RESULTS	Diesel exhaust exposure was associated with significantly decreased GSH/GSSG ($p = 0.001$) and a 4-fold increase in IL-6 mRNA ($p = 0.01$) post exposure. Antioxidant pretreatment did not significantly mediate the effect of DE exposure on GSH/GSSG, though appeared to decrease the effect of exposure on IL-6 mRNA expression.
CONCLUSIONS	Acute DE inhalation induced detectable oxidative effects in healthy adults, which were not significantly attenuated by the selected antioxidant pre-treatment. This finding supports the premise that oxidative stress is one mechanism underlying the adverse effects of traffic-related air pollution.

표-847. PubMed 논문번호 32835677의 내용 요약

구분	내용
PubMed ID	32835677
TITLE	Association between short-term exposure to ambient particulate air pollution and biomarkers of oxidative stress: A meta-analysis.
JOURNAL	Environmental research: 10.1016/j.envres.2020.110105
AUTHORS	Li Zichuan, Liu Qisijing, Xu Zhouyang, Guo Xinbiao, Wu Shaowei
BACKGROUND	Exposure to ambient particulate air pollution contributes substantially to the mortality and morbidity due to cardiovascular diseases (CVD), respiratory diseases and neurodegenerative diseases. Several hypothetical mechanisms have been proposed to explain these associations, particularly oxidative stress. Malondialdehyde (MDA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and Superoxide Dismutase (SOD) are typical biomarkers of oxidative stress and have been frequently investigated. However, the association between exposure to ambient particulate matter (PM) and these biomarkers has not been well established.
OBJECTIVES	Evaluate the association between ambient particulate air pollution and biomarkers of oxidative stress based on existing epidemiological studies.
METHODS	A systematic literature search was conducted in databases of Science Direct, PubMed, Web of Science, and Scopus up to April 24, 2020 to summarize epidemiological studies reporting the association between exposure to ambient PM (PM2.5, PM10, or both) and biomarkers of oxidative stress, and a meta-analysis was performed for the associations reported in individual studies using a random-effect model.
RESULTS	This meta-analysis included 23 epidemiological studies (13 identified for 8-OHdG, 11 identified for MDA and 5 identified for SOD). A 10 μg/m3 increase in short-term exposure to ambient PM2.5 was associated with pooled percent changes of 2.10% (95% CIs: -0.13%, 4.38%), 1.60% (95% CIs: 0.21%, 3.01%) and -0.61% (95% CIs: -1.92%, 0.72%) in 8-OHdG, MDA and SOD, respectively.
CONCLUSION	Short-term exposure to ambient PM2.5 was associated with a significantly increased level of MDA, indicating that ambient particulate air pollution may contribute to increased oxidative stress.

표-848. PubMed 논문번호 32835970의 내용 요약

구분	내용
PubMed ID	32835970
TITLE	Short-term effects of ambient nitrogen dioxide on years of life lost in 48 major Chinese cities, 2013-2017.
JOURNAL	Chemosphere: 10.1016/j.chemosphere.2020.127887
AUTHORS	Li Jie, Zhang Xiao, Li Guoxing, Wang Lijun, Yin Peng, Zhou Maigeng
BACKGROUND	Evidence on the acute effect of short-term exposure to nitrogen dioxide (NO2) on years of life lost (YLL) is rare, especially in multicity setting.
METHODS	We conducted a time series study among 48 major Chinese cities covering more than 403 million people from 2013 to 2017. The relative percentage changes of NO2-YLL were estimated by generalized additive models in each city, then were pooled to generate average effects using random-effect models. In addition, stratified analyses by individual demographic factors and temperature as well as meta-regression analyses incorporating city-specific air pollutant concentrations, meteorological conditions, and socioeconomic indicators were performed to explore potential effect modification.
RESULTS	A 10 μg/m3 increase in two-day moving average (lag01) NO2 concentration was associated with 0.64% (95% CI: 0.47%, 0.81%), 0.47% (95% CI: 0.27%, 0.68%), and 0.68% (95% CI: 0.34%, 1.02%) relative increments in YLL due to nonaccidental causes, cardiovascular diseases (CVD), and respiratory diseases (RD), respectively. These associations were generally robust to the adjustment of co-pollutants, except for NO2-CVD that might be confounded by fine particulate matter. The increased YLL induced by NO2 were more pronounced in elderly people, hotter days, and cities characterized by less severe air pollution or higher temperature.
CONCLUSIONS	Our results demonstrated robust evidence on the associations between NO2 exposure and YLL due to nonaccidental causes, CVD, and RD, which provided novel evidence to better understand the disease burden related to NO2 pollution and to facilitate allocation of health resources targeting high-risk subpopulation.

표-849. PubMed 논문번호 32846171의 내용 요약

구분	내용
PubMed ID	32846171
TITLE	Association of long-term exposure to ambient air pollutants with prolonged sleep latency: The Henan Rural Cohort Study.
JOURNAL	Environmental research: 10.1016/j.envres.2020.110116
AUTHORS	Wang Yan, Liu Xiaotian, Chen Gongbo, Tu Runqi, Abdulai Tanko, Qiao Dou, Liu Xue, Dong Xiaokang, Luo Zhicheng, Wang Yikang, Li Ruiying, Huo Wenqian, Yu Songcheng, Guo Yuming, Li Shanshan, Wang Chongjian
BACKGROUND	Prolonged sleep latency is associated with far-reaching public health consequences. Although evidence about the effect of air pollution on sleep problem has been shown, the effect on sleep latency remained unknown. The study aimed to analyze the association between long-term exposure to air pollution and prolonged sleep latency in rural China.
METHODS	In all, 27935 participants were included in the study from Henan Rural Cohort Study. A satellite-based spatiotemporal model was used to evaluate the 3-year average concentration of air pollutants at the home address of participants before the baseline survey. Air pollutants included NO2 (nitrogen dioxide), PM1 (particulate matter with aerodynamic diameters ≤1 μm), PM2.5 (particulate matter with aerodynamic diameters ≤ 2.5 μm), and PM10 (particulate matter with aerodynamic diametes ≤ 10 μm). A logistic regression model was conducted to assess the odds ratio (OR) and 95% confidence interval (95% CI) between air pollutants and prolonged sleep latency.
RESULTS	There were 5825 (20.85%) participants with prolonged sleep latency. The average concentration of NO2, PM1, PM2.5, and PM10 were 38.22 (2.54) μg/m3, 56.29 (1.75) μg/m3, 72.30 (1.87) μg/m3, and 130.01 (4.58) μg/m3. The odds ratio (95%CI) of prolonged sleep latency with an IQR increase of NO2, PM1, PM2.5, and PM10 were 1.59 (1.33-1.90), 1.23 (1.13-1.33), 1.28 (1.13-1.45) and 1.43 (1.22-1.67). The stratified analysis showed the effect of air pollutants was stronger among those with stroke.
CONCLUSION	Long-term exposure to NO2, PM1, PM2.5 and PM10 were associated with prolonged sleep latency. The adverse impact of air pollution should be considered when treating sleep problems.

표-850. PubMed 논문번호 32846176의 내용 요약

구분	내용
PubMed ID	32846176
TITLE	Long-term exposure to air pollution and atherosclerosis in the carotid arteries in the Malm□ diet and cancer cohort.
JOURNAL	Environmental research: 10.1016/j.envres.2020.110095
AUTHORS	Hassl□f Helena, Moln□r Peter, Andersson Eva M, Spanne M□rten, Gustafsson Susanna, Stroh Emilie, Engstr□m Gunnar, Stockfelt Leo
BACKGROUND	Long-term exposure to air pollution increases the risk of cardiovascular morbidity and mortality, but the mechanisms are not fully known. Current evidence suggests that air pollution exposure contributes to the development of atherosclerosis. There are few studies investigating associations between air pollution and carotid plaques, a well-known precursor of cardiovascular disease.
METHODS	A Swedish population-based cohort (aged 45-64 years at recruitment) was randomly selected from the Malm□ Diet and Cancer study between 1991 and 1994, of which 6103 participants underwent ultrasound examination of the right carotid artery to determine carotid plaque presence and carotid intima media thickness (CIMT). Participants were assigned individual residential air pollution exposure (source-specific PM2.5, PM10, NOx, BC) at recruitment from Gaussian dispersion models. Logistic and linear regression models, adjusted for potential confounders and cardiovascular risk factors, were used to investigate associations between air pollutants and prevalence of carotid plaques, and CIMT, respectively.
RESULTS	The prevalence of carotid plaques was 35%. The mean levels of PM2.5 and PM10 at recruitment were 11 and 14 μg/m3, most of which was due to long range transport. The exposure contrast within the cohort was relatively low. PM2.5 exposure was associated with carotid plaques in a model including age and sex only (OR 1.10 (95% CI 1.01-1.20) per 1 μg/m3), but after adjustment for cardiovascular risk factors and socioeconomic status (SES) the association was weak and not significant (OR 1.05 (95% CI 0.96-1.16) per 1 μg/m3). The pattern was similar for PM10 and NOx exposure. Associations between air pollutants and plaques were slightly stronger for long-term residents and in younger participants with hypertension. There was no clear linear trend between air pollution exposure and plaque prevalence. Non-significant slightly positive associations were seen between air pollution exposures and CIMT.
CONCLUSIONS	In this large, well-controlled cross-sectional study at low exposure levels we found no significant associations between air pollution exposures and subclinical atherosclerosis in the carotid arteries, after adjusting for cardiovascular risk factors and SES. Further epidemiological studies of air pollution and intermediate outcomes are needed to explain the link between air pollution and cardiovascular events.

표-851. PubMed 논문번호 32918184의 내용 요약

구분	내용
PubMed ID	32918184
TITLE	Association between fine particulate matter and atrial fibrillation in implantable cardioverter defibrillator patients: a systematic review and meta-analysis.
JOURNAL	Journal of interventional cardiac electrophysiology : an international journal of arrhythmias and pacing: 10.1007/s10840-020-00864-1
AUTHORS	Yue Chao, Yang Fan, Wang Luyi, Li Fengwei, Chen Yingtai
PURPOSE	Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia with several risk factors. Recent studies have suggested that the exposure to fine particulate matter (PM2.5) increased the incidence of AF, but there is no meta-analysis of AF occurrence due to the exposure to PM2.5 in implantable cardioverter defibrillator (ICD) patients.
METHODS	We conducted a systematic review of publication using PubMed, Embase, the Cochrane library, and Web of Science to explore the association between PM2.5 and AF within ICD patients. The chosen studies were published until June 11, 2020. The I2 statistic and Q test were used to examine statistical heterogeneity across studies. Further sensitivity analyses were carried out to ascertain the reason for heterogeneity. Fixed or random-effect model was used to combine the effects. Final result was presented as the OR with 95% CI of increased incidence of AF for every 10 μg/m3 PM2.5 concentration increased.
RESULTS	After screening our analysis contained four studies and involved 1689 AF events from 572 patients. After using the random-effect model to combine the included study result, the overall OR was 1.24 (95% CI 1.00-1.53).
CONCLUSION	Our meta-analysis indicated that PM2.5 exposure had an adverse effect on AF incidence in ICD patients.

표-852. PubMed 논문번호 32920133의 내용 요약

구분	내용
PubMed ID	32920133
TITLE	Active ingredients targeting Nrf2 in the Mongolian medicine Qiwei Putao powder: Systematic pharmacological prediction and validation for chronic obstructive pulmonary disease treatment.
JOURNAL	Journal of ethnopharmacology: 10.1016/j.jep.2020.113385
AUTHORS	Deng Ya-Xin, Zhong Jing, Liu Zi-Jing, Wang Xiao-Qin, Zhang Bo
ETHNOPHARMACOLOGY RELEVANCE	Qiwei Putao powder (Uzhumu-7 in Mongolian) is a traditional Mongolian medicine, which has been widely used for alleviating cough and dyspnea, especially in aged individuals in both Inner Mongolia Autonomous Region and Xinjiang Uygur Autonomous Region of China. However, the active ingredients and exact pharmacological mechanism remain unclear.
MATERIALS AND METHODS	The protective effect of Qiwei Putao powder (QPP) on mice with cigarette smoke (CS)- and lipopolysaccharide (LPS)-induced chronic obstructive pulmonary disease (COPD) was assessed by histopathological hematoxylin and eosin staining, lung coefficient determination and measurement of cytokine levels. The bioactive ingredients and potential targets of the QPP were screened and detected with network pharmacology method and ultra performance liquid chromatography coupled with quadrupole time-of-fligh mass spectrometry (UPLC-Q/TOF-MS). The mechanism and efficacy of active ingredients were further validated in COPD mice with immunohistochemistry tests, cytokine level measurement and RT-PCR. The expression levels of nuclear factor erythroid 2-related factor 2 (Nrf2) in the nucleus, interleukin (IL)-1β, superoxide dismutase (SOD), malondialdehyd (MDA) and tumor necrosis factor-alpha (TNF-α) were detected by enzyme-linked immunosorbent assay (ELISA) kits to evaluate oxidative stre and inflammatory conditions in vivo after treatment. The expression of Nrf and downstream genes was detected by RT-PCR.
RESULTS	QPP can alleviate pathological changes in the lung during COPD progressio Sixty-one bioactive molecules were identified in QPP, 42 candidate compounds present in UPLC-Q/TOF-MS and 30 predicted COPD-related targets were generated by in silico analysis. A therapeutic network was constructed with all potential targets to predict the preventive effects of the targets on respiratory disease as well as cardiovascular diseases, nervous system diseases, musculoskeletal diseases and bacterial infections. Targets related to inflammation, immunity and oxidative stress (prostaglandin-endoperoxide synthase 2, PTGS2; Nrf2; heat shock protein 90 alpha class A1, HSP90AA1; nitric oxide synthase, NOS2A; etc.) influenced COPD progression the most. We found that Nrf2 promotes a cel antioxidant response and is a key common target in the response to treatme with isoliquiritigenin (ISL), pterostilbene (PTE) and quercetin (QUE), the highly absorbed active ingredients in the formula. The data showed a strong synergistic protective role of these three molecules against the death of huma type II alveolar adenocarcinoma (A549) cells through Nrf2 activation following H2O2 exposure and provide pharmacological mechanism of QPP COPD treatment.

표-853. PubMed 논문번호 32930777의 내용 요약

구분	내용
PubMed ID	32930777
TITLE	Association of Fine Particulate Matter and Risk of Stroke in Patients With Atrial Fibrillation.
JOURNAL	JAMA network open: 10.1001/jamanetworkopen.2020.11760
AUTHORS	Rhinehart Zachary J, Kinnee Ellen, Essien Utibe R, Saul Melissa, Guhl Emily, Clougherty Jane E, Magnani Jared W
Importance	Air pollution is associated with cardiovascular outcomes. Specifically, fine particulate matter measuring 2.5 μm or less (PM2.5) is associated with thrombosis, stroke, and myocardial infarction. Few studies have examined particulate matter and stroke risk in individuals with atrial fibrillation (AF).
Objective	To assess the association of residential-level pollution exposure in 1 year and ischemic stroke in individuals with AF.
Design, Setting, and Participants	This cohort study included 31 414 individuals with AF from a large regional health care system in an area with historically high industrial pollution. All participants had valid residential addresses for geocoding and ascertainment of neighborhood-level income and educational level. Participants were studied from January 1, 2007, through September 30, 2015, with prospective follow-up through December 1, 2017. Data analysis was performed from March 14, 2018, to October 9, 2019.
Exposures	Exposure to PM2.5 ascertained using geocoding of addresses and fine-scale air pollution exposure surfaces derived from a spatial saturation monitoring campaign and land-use regression modeling. Exposure to PM2.5 was estimated annually across the study period at the residence level.
Main Outcomes and Measures	Multivariable-adjusted stroke risk by quartile of residence-level and annual PM2.5 exposure.
Results	The cohort included 31 414 individuals (15 813 [50.3%] female; mean [SD] age, 74.4 [13.5] years), with a median follow-up of 3.5 years (interquartile range, 1.6-5.8 years). The mean (SD) annual PM2.5 exposure was 10.6 (0.7) μg/m3. A 1-SD increase in PM2.5 was associated with a greater risk of stroke after both adjustment for demographic and clinical variables (hazard ratio [HR], 1.08; 95% CI, 1.03-1.14) and multivariable adjustment that included neighborhood-level income and educational level (HR, 1.07; 95% CI, 1.00-1.14). The highest quartile of PM2.5 exposure had an increased risk of stroke relative to the first quartile (HR, 1.36; 95% CI, 1.18-1.58). After adjustment for clinical covariates, income, and educational level, risk of stroke remained greater for the highest quartile of exposure relative to the first quartile (HR, 1.21; 95% CI, 1.01-1.45).
Conclusions and Relevance	This large cohort study of individuals with AF identified associations between PM2.5 and risk of ischemic stroke. The results suggest an association between fine particulate air pollution and cardiovascular disease and outcomes.

표-854. PubMed 논문번호 32941838의 내용 요약

구분	내용
PubMed ID	32941838
TITLE	Vehicular traffic density and cognitive performance in the ELSA-Brasil study.
JOURNAL	Environmental research: 10.1016/j.envres.2020.110208
AUTHORS	Rocha Douglas, Suemoto Claudia K, Souza Santos Itamar, Lotufo Paulo A, Bense□or Isabela, Gouveia Nelson
BACKGROUND	Despite the knowledge about the deleterious effects of air pollutants and their influence on mortality and morbidity due to respiratory and cardiovascular diseases, little is known about the relationship between atmospheric pollutants and neurological diseases. Recently, studies from high-income countries have suggested an association between exposures to air pollutants with cognitive impairment. Thus, we investigated the association of air pollution with cognitive performance in the participants of the Brazilian Longitudinal Study of Adult Health (ELSA-Brasil).
METHODS	Cognitive function was evaluated using the word list, the verbal fluency, and the trail making tests (TMT). Pollutant exposure was evaluated indirectly using the distance-weighted traffic density (DWTD) of participants' residence and workplace. We investigated the cross-sectional association between DWTD and cognitive test scores using adjusted linear regression models for sociodemographic and clinical variables.
RESULTS	3050 were included (mean age = 52.1 ± 9.2 years old, 56.5% women, and 63.6% white). In the simple linear regression models, participants in the higher tertile of combined DWTD (residence and workplace) presented better cognitive performance in all tests when compared to participants in the lower tertile. The DWTD was not associated with cognitive performance in adjusted linear models especially when adjusted for socioeconomic variables (age, sex, education, and race). We found similar results when we investigated the association of cognitive performance with DTWD near participants' workplace and residence separately.
CONCLUSION	Air pollutants were not associated with worse cognitive performance in a large sample of middle-aged and older adults.

표-855. PubMed 논문번호 32943053의 내용 요약

구분	내용
PubMed ID	32943053
TITLE	Correlation between exposure to fine particulate matter and hypertensive disorders of pregnancy in Shanghai, China.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-020-00655-1
AUTHORS	Su Xiujuan, Zhao Yan, Yang Yingying, Hua Jing
BACKGROUND	Association between fine particulate matter (PM2.5) and hypertensive disorders of pregnancy (HDP) is inconsistent and appears to change in each trimester. We aim to investigate the association of exposure to ambient PM2.5 in early pregnancy with HDP.
METHODS	A retrospective cohort study was performed among 8776 women with singleton pregnancy who attended the antenatal clinic before 20 gestational weeks in a tertiary women's hospital during 2014-2015. Land use regression models were used to predict individual levels of PM2.5 exposure.
RESULTS	The average PM2.5 concentration during the first 20 gestational weeks ranged from 28.6 to 74.8 μg m- 3 [median, 51.4 μg m- 3; interquartile range, 47.3-57.8 μg m- 3]. A total of 440 (5.0%) women was diagnosed with HDP. The restricted cubic spline showed a positive exposure-response relationship between the PM2.5 concentration and risk of HDP. We observed an association between PM2.5 exposure during the first trimester with HDP (RR = 3.89 per 10 μg m- 3, 95% CI: 1.45-10.43), but not during the second trimester (RR = 0.71 per 10 μg m- 3, 95% CI: 0.40-1.27). Compared with their counterparts, nulliparous women who were exposed to high levels of PM2.5 in the index pregnancy had a higher risk of developing HDP [the relative excess risk due to interaction was 0.92 (0.46-1.38)].
CONCLUSION	Our findings suggest that PM2.5 exposure during the first trimester is associated with the development of HDP. The effect estimate is more obvious for nulliparous women than multiparous women.

표-856. PubMed 논문번호 33010596의 내용 요약

구분	내용
PubMed ID	33010596
TITLE	Particulate matter exposure and biomarkers associated with blood coagulation: A meta-analysis.
JOURNAL	Ecotoxicology and environmental safety: 10.1016/j.ecoenv.2020.111417
AUTHORS	Sun Mengqi, Liang Qingqing, Ma Yuexiao, Wang Fenghong, Lin Lisen, Li Tianyu, Sun Zhiwei, Duan Junchao
OBJECTIVE	Find the correlation between particulate matter (PM) and biomarkers related to blood coagulation, offer medical evidence to sensitive indicators and carry out early diagnosis of cardiovascular diseases.
METHOD	A combination of computer and manual retrieval was used to search for the keywords in PubMed (584 records), Cochrane Library (28 records), Web of Science (162 records) and Embase (163 records). Finally, a total of 25 articles were included in this meta-analysis. Stata 13.0 was applied to examine the heterogeneity among the studies and to calculate the combined effect estimates, percent variation (%) and 95% CI by selecting corresponding models. Additionally, sensitivity analysis and publication bias test were also conducted.
RESULTS	Meta-analysis indicated that there was an association between PM2.5 exposure (per 10 □g/m3 increase) and fibrinogen. With the increase of PM2.5 exposure (per 10 □g/m3 increase), the content of fibrinogen revealed a high level (2.26%; 95% CI: 1.08-3.44%); and the increase of UFPs exposure (per 5000/cm3 increase) was correlated with some biomarkers such as cell surface antigen and protein ligand including ICAM-1, sCD40L, P-selectin, E-selectin and PAI-1 that indirectly related to blood coagulation, yielding a percent variation of 10.83% (95% CI: 3.49%-18.17%).
CONCLUSION	This meta-analysis expounded that PM-related biomarkers were associated with blood coagulation, and the relationship with fibrinogen was much stronger.

표-857. PubMed 논문번호 33017176의 내용 요약

구분	내용
PubMed ID	33017176
TITLE	Air Pollution and Progression of Atherosclerosis in Different Vessel Beds-Results from a Prospective Cohort Study in the Ruhr Area, Germany.
JOURNAL	Environmental health perspectives: 10.1289/EHP7077
AUTHORS	Hennig Frauke, Geisel Marie Henrike, K□lsch Hagen, Lucht Sarah, Mahabadi Amir Abbas, Moebus Susanne, Erbel Raimund, Lehmann Nils, J□ckel Karl-Heinz, Scherag Andr□, Hoffmann Barbara
OBJECTIVES	Due to inconsistent epidemiological evidence on health effects of air pollution on progression of atherosclerosis, we investigated several air pollutants and their effects on progression of atherosclerosis, using carotid intima media thickness (cIMT), coronary calcification (CAC), and thoracic aortic calcification (TAC).
METHODS	We used baseline (2000-2003) and 5-y follow-up (2006-2008) data from the German Heinz Nixdorf Recall cohort study, including 4,814 middle-aged adults. Residence-based long-term air pollution exposure, including particulate matter (PM) with aerodynamic diameter ≤2.5 μm (PM2.5), (PM10), and nitrogen dioxide (NO2) was assessed using chemistry transport and land use regression (LUR) models. cIMT was quantified as side-specific median IMT assessed from standardized ultrasound images. CAC and TAC were quantified by computed tomography using the Agatston score. Development (yes/no) and progression of atherosclerosis (change in cIMT and annual growth rate for CAC/TAC) were analyzed with logistic and linear regression models, adjusting for age, sex, lifestyle variables, socioeconomic status, and traffic noise.
RESULTS	While no clear associations were observed in the full study sample (mean age 59.1 (±7.6) y; 53% female), most air pollutants were marginally associated with progression of atherosclerosis in participants with no or low baseline atherosclerotic burden. Most consistently for CAC, e.g., a 1.5 μg/m3 higher exposure to PM2.5 (LUR) yielded an estimated odds ratio of 1.19 [95% confidence interval (CI): 1.03, 1.39] for progression of CAC and an increased annual growth rate of 2% (95% CI: 1%, 4%).
CONCLUSION	Our study suggests that development and progression of subclinical atherosclerosis is associated with long-term air pollution in middle-aged participants with no or minor atherosclerotic burden at baseline, while overall no consistent associations are observed. https://doi.org/10.1289/EHP7077.

표-858. PubMed 논문번호 33054842의 내용 요약

구분	내용
PubMed ID	33054842
TITLE	Effect of probiotics on nasal and intestinal microbiota in people with high exposure to particulate matter ≤ 2.5 μm (PM2.5): a randomized, double-blind, placebo-controlled clinical study.
JOURNAL	Trials: 10.1186/s13063-020-04759-4
AUTHORS	Wu Yongcan, Pei Caixia, Wang Xiaomin, Wang Mingjie, Huang Demei, Wang Fei, Xiao Wei, Wang Zhenxing
BACKGROUND	Extended exposure to high concentrations of PM2.5 changes the human microbiota profile, which in turn may increase morbidity and mortality due to respiratory system damage. A balanced microecosystem is crucial to human health, and certain health-related problems may be addressed by effective microecosystem regulation. Recent studies have confirmed that probiotics may reduce the incidence of respiratory diseases. However, few studies have investigated probiotic treatment outcomes in subjects exposed to high concentrations of PM2.5.
METHODS	This study is designed as a prospective, randomized, participants- and assessor-blinded, placebo-controlled trial. One hundred and twenty eligible volunteers recruited from October 2019 to July 2020 in downtown Chengdu, China, will be treated with either probiotics or placebo over 4 consecutive weeks. The primary outcome will be 16SrRNA sequencing assay data from nasal and intestinal secretions. Secondary outcomes will be pulmonary function, score on a gastrointestinal symptom rating scale, COOP/WONCA charts, and the Short-Form Health Survey 36 for quality of life. Results will be analyzed to assess differences in clinical efficacy between groups. Six-month follow-up examinations will evaluate the long-term value of probiotics on cardiovascular and respiratory disease end-point events.
DISCUSSION	We will explore the characteristics of nasal and intestinal microbiota in a population with high exposure to PM2.5. Probiotics and placebo interventions will be tested for efficacy in microbial balance regulation, effects on lung and physical functions, and quality of life improvement. This study is expected to provide reliable evidence to support the widespread promotion of probiotics in clinical practice for the protection of individuals with high exposure to PM2.5.
TRIAL REGISTRATION	Chinese Clinical Trial Registry ChiCTR1900025469 . Registered on 27 August 2019.

표-859. PubMed 논문번호 33068582의 내용 요약

구분	내용
PubMed ID	33068582
TITLE	Long-term air pollution exposure and self-reported morbidity: A longitudinal analysis from the Thai cohort study (TCS).
JOURNAL	Environmental research: 10.1016/j.envres.2020.110330
AUTHORS	Paoin Kanawat, Ueda Kayo, Ingviya Thammasin, Buya Suhaimee, Phosri Arthit, Seposo Xerxes Tesoro, Seubsman Sam-Ang, Kelly Matthew, Sleigh Adrian, Honda Akiko, Takano Hirohisa
BACKGROUND	Several studies have shown the health effects of air pollutants, especially in China, North American and Western European countries. But longitudinal cohort studies focused on health effects of long-term air pollution exposure are still limited in Southeast Asian countries where sources of air pollution, weather conditions, and demographic characteristics are different. The present study examined the association between long-term exposure to air pollution and self-reported morbidities in participants of the Thai cohort study (TCS) in Bangkok metropolitan region (BMR), Thailand.
METHODS	This longitudinal cohort study was conducted for 9 years from 2005 to 2013. Self-reported morbidities in this study included high blood pressure, high blood cholesterol, and diabetes. Air pollution data were obtained from the Thai government Pollution Control Department (PCD). Particles with diameters ≤10 μm (PM10), sulfur dioxide (SO2), nitrogen dioxide (NO2), ozone (O3), and carbon monoxide (CO) exposures were estimated with ordinary kriging method using 22 background and 7 traffic monitoring stations in BMR during 2005-2013. Long-term exposure periods to air pollution for each subject was averaged as the same period of person-time. Cox proportional hazards models were used to examine the association between long-term air pollution exposure with self-reported high blood pressure, high blood cholesterol, diabetes. Results of self-reported morbidity were presented as hazard ratios (HRs) per interquartile range (IQR) increase in PM10, O3, NO2, SO2, and CO.
RESULTS	After controlling for potential confounders, we found that an IQR increase in PM10 was significantly associated with self-reported high blood pressure (HR = 1.13, 95% CI: 1.04, 1.23) and high blood cholesterol (HR = 1.07, 95%CI: 1.02, 1.12), but not with diabetes (HR = 1.05, 95%CI: 0.91, 1.21). SO2 was also positively associated with self-reported high blood pressure (HR = 1.22, 95%CI: 1.08, 1.38), high blood cholesterol (HR = 1.20, 95%CI: 1.11, 1.30), and diabetes (HR = 1.21, 95%CI: 0.92, 1.60). Moreover, we observed a positive association between CO and self-reported high blood pressure (HR = 1.07, 95%CI: 1.00, 1.15), but not for other diseases. However, self-reported morbidities were not associated with O3 and NO2.
CONCLUSIONS	Long-term exposure to air pollution, especially for PM10 and SO2 was associated with self-reported high blood pressure, high blood cholesterol, and diabetes in subjects of TCS. Our study supports that exposure to air pollution increases cardiovascular disease risk factors for younger population.

표-860. PubMed 논문번호 33069303의 내용 요약

구분	내용
PubMed ID	33069303
TITLE	Adverse health effects associated with household air pollution: a systematic review, meta-analysis, and burden estimation study.
JOURNAL	The Lancet. Global health: 10.1016/S2214-109X(20)30343-0
AUTHORS	Lee Kuan Ken, Bing Rong, Kiang Joanne, Bashir Sophia, Spath Nicholas, Stelzle Dominik, Mortimer Kevin, Bularga Anda, Doudesis Dimitrios, Joshi Shruti S, Strachan Fiona, Gumy Sophie, Adair-Rohani Heather, Attia Engi F, Chung Michael H, Miller Mark R, Newby David E, Mills Nicholas L, McAllister David A, Shah Anoop S V
BACKGROUND	3 billion people worldwide rely on polluting fuels and technologies for domestic cooking and heating. We estimate the global, regional, and national health burden associated with exposure to household air pollution.
METHODS	For the systematic review and meta-analysis, we systematically searched four databases for studies published from database inception to April 2, 2020, that evaluated the risk of adverse cardiorespiratory, paediatric, and maternal outcomes from exposure to household air pollution, compared with no exposure. We used a random-effects model to calculate disease-specific relative risk (RR) meta-estimates. Household air pollution exposure was defined as use of polluting fuels (coal, wood, charcoal, agricultural wastes, animal dung, or kerosene) for household cooking or heating. Temporal trends in mortality and disease burden associated with household air pollution, as measured by disability-adjusted life-years (DALYs), were estimated from 2000 to 2017 using exposure prevalence data from 183 of 193 UN member states. 95% CIs were estimated by propagating uncertainty from the RR meta-estimates, prevalence of household air pollution exposure, and disease-specific mortality and burden estimates using a simulation-based approach. This study is registered with PROSPERO, CRD42019125060.
FINDINGS	476 studies (15·5 million participants) from 123 nations (99 [80%] of which were classified as low-income and middle-income) met the inclusion criteria. Household air pollution was positively associated with asthma (RR 1·23, 95% CI 1·11-1·36), acute respiratory infection in both adults (1·53, 1·22-1·93) and children (1·39, 1·29-1·49), chronic obstructive pulmonary disease (1·70, 1·47-1·97), lung cancer (1·69, 1·44-1·98), and tuberculosis (1·26, 1·08-1·48); cerebrovascular disease (1·09, 1·04-1·14) and ischaemic heart disease (1·10, 1·09-1·11); and low birthweight (1·36, 1·19-1·55) and stillbirth (1·22, 1·06-1·41); as well as with under-5 (1·25, 1·18-1·33), respiratory (1·19, 1·18-1·20), and cardiovascular (1·07, 1·04-1·11) mortality. Household air pollution was associated with 1·8 million (95% CI 1·1-2·7) deaths and 60·9 million (34·6-93·3) DALYs in 2017, with the burden overwhelmingly experienced in low-income and middle-income countries (LMICs; 60·8 million [34·6-92·9] DALYs) compared with high-income countries (0·09 million [0·01-0·40] DALYs). From 2000, mortality associated with household air pollution had reduced by 36% (95% CI 29-43) and disease burden by 30% (25-36), with the greatest reductions observed in higher-income nations.
INTERPRETATION	The burden of cardiorespiratory, paediatric, and maternal diseases associated with household air pollution has declined worldwide but remains high in the world's poorest regions. Urgent integrated health and energy strategies are needed to reduce the adverse health impact of household air pollution, especially in LMICs.
FUNDING	British Heart Foundation, Wellcome Trust.

표-861. PubMed 논문번호 In an era of shifting global agendas and expanded emphasis on non-communicable diseases and injuries along with communicable diseases, sound evidence on trends by cause at the national level is essential. The Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) provides a systematic scientific assessment of published, publicly available, and contributed data on incidence, prevalence, and mortality for a mutually exclusive and collectively exhaustive list of diseases and injuries.의 내용 요약

구분	내용
BACKGROUND	In an era of shifting global agendas and expanded emphasis on non-communicable diseases and injuries along with communicable diseases, sound evidence on trends by cause at the national level is essential. The Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) provides a systematic scientific assessment of published, publicly available, and contributed data on incidence, prevalence, and mortality for a mutually exclusive and collectively exhaustive list of diseases and injuries.
METHODS	GBD estimates incidence, prevalence, mortality, years of life lost (YLLs), years lived with disability (YLDs), and disability-adjusted life-years (DALYs) due to 369 diseases and injuries, for two sexes, and for 204 countries and territories. Input data were extracted from censuses, household surveys, civil registration and vital statistics, disease registries, health service use, air pollution monitors, satellite imaging, disease notifications, and other sources. Cause-specific death rates and cause fractions were calculated using the Cause of Death Ensemble model and spatiotemporal Gaussian process regression. Cause-specific deaths were adjusted to match the total all-cause deaths calculated as part of the GBD population, fertility, and mortality estimates. Deaths were multiplied by standard life expectancy at each age to calculate YLLs. A Bayesian meta-regression modelling tool, DisMod-MR 2.1, was used to ensure consistency between incidence, prevalence, remission, excess mortality, and cause-specific mortality for most causes. Prevalence estimates were multiplied by disability weights for mutually exclusive sequelae of diseases and injuries to calculate YLDs. We considered results in the context of the Socio-demographic Index (SDI), a composite indicator of income per capita, years of schooling, and fertility rate in females younger than 25 years. Uncertainty intervals (UIs) were generated for every metric using the 25th and 975th ordered 1000 draw values of the posterior distribution.
FINDINGS	Global health has steadily improved over the past 30 years as measured by age-standardised DALY rates. After taking into account population growth and ageing, the absolute number of DALYs has remained stable. Since 2010, the pace of decline in global age-standardised DALY rates has accelerated in age groups younger than 50 years compared with the 1990-2010 time period, with the greatest annualised rate of decline occurring in the 0-9-year age group. Six infectious diseases were among the top ten causes of DALYs in children younger than 10 years in 2019: lower respiratory infections (ranked second), diarrhoeal diseases (third), malaria (fifth), meningitis (sixth), whooping cough (ninth), and sexually transmitted infections (which, in this age group, is fully accounted for by congenital syphilis; ranked tenth). In adolescents aged 10-24 years, three injury causes were among the top causes of DALYs: road injuries (ranked first), self-harm (third), and interpersonal violence (fifth). Five of the causes that were in the top ten for ages 10-24 years were also in the top ten in the 25-49-year age group: road injuries (ranked first), HIV/AIDS (second), low back pain (fourth), headache disorders (fifth), and depressive disorders (sixth). In 2019, ischaemic heart disease and stroke were the top-ranked causes of DALYs in both the 50-74-year and 75-years-and-older age groups. Since 1990, there has been a marked shift towards a greater proportion of burden due to YLDs from non-communicable diseases and injuries. In 2019, there were 11 countries where non-communicable disease and injury YLDs constituted more than half of all disease burden. Decreases in age-standardised DALY rates have

표-862. PubMed 논문번호 Rigorous analysis of levels and trends in exposure to leading risk factors and quantification of their effect on human health are important to identify where public health is making progress and in which cases current efforts are inadequate. The Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2019 provides a standardised and comprehensive assessment of the magnitude of risk factor exposure, relative risk, and attributable burden of disease.의 내용 요약

구분	내용
BACKGROUND	Rigorous analysis of levels and trends in exposure to leading risk factors and quantification of their effect on human health are important to identify where public health is making progress and in which cases current efforts are inadequate. The Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2019 provides a standardised and comprehensive assessment of the magnitude of risk factor exposure, relative risk, and attributable burden of disease.
METHODS	GBD 2019 estimated attributable mortality, years of life lost (YLLs), years of life lived with disability (YLDs), and disability-adjusted life-years (DALYs) for 87 risk factors and combinations of risk factors, at the global level, regionally, and for 204 countries and territories. GBD uses a hierarchical list of risk factors so that specific risk factors (eg, sodium intake), and related aggregates (eg, diet quality), are both evaluated. This method has six analytical steps. (1) We included 560 risk-outcome pairs that met criteria for convincing or probable evidence on the basis of research studies. 12 risk-outcome pairs included in GBD 2017 no longer met inclusion criteria and 47 risk-outcome pairs for risks already included in GBD 2017 were added based on new evidence. (2) Relative risks were estimated as a function of exposure based on published systematic reviews, 81 systematic reviews done for GBD 2019, and meta-regression. (3) Levels of exposure in each age-sex-location-year included in the study were estimated based on all available data sources using spatiotemporal Gaussian process regression, DisMod-MR 2.1, a Bayesian meta-regression method, or alternative methods. (4) We determined, from published trials or cohort studies, the level of exposure associated with minimum risk, called the theoretical minimum risk exposure level. (5) Attributable deaths, YLLs, YLDs, and DALYs were computed by multiplying population attributable fractions (PAFs) by the relevant outcome quantity for each age-sex-location-year. (6) PAFs and attributable burden for combinations of risk factors were estimated taking into account mediation of different risk factors through other risk factors. Across all six analytical steps, 30 652 distinct data sources were used in the analysis. Uncertainty in each step of the analysis was propagated into the final estimates of attributable burden. Exposure levels for dichotomous, polytomous, and continuous risk factors were summarised with use of the summary exposure value to facilitate comparisons over time, across location, and across risks. Because the entire time series from 1990 to 2019 has been re-estimated with use of consistent data and methods, these results supersede previously published GBD estimates of attributable burden.
FINDINGS	The largest declines in risk exposure from 2010 to 2019 were among a set of risks that are strongly linked to social and economic development, including household air pollution; unsafe water, sanitation, and handwashing; and child growth failure. Global declines also occurred for tobacco smoking and lead exposure. The largest increases in risk exposure were for ambient particulate matter pollution, drug use, high fasting plasma glucose, and high body-mass index. In 2019, the leading Level 2 risk factor globally for attributable deaths was high systolic blood pressure, which accounted for 10·8 million (95% uncertainty interval [UI] 9·51-12·1) deaths (19·2% [16·9-21·3] of all deaths in 2019), followed by tobacco (smoked, second-hand, and chewing), which accounted for 8·71 million (8·12-9·31) deaths (15·4% [14·6-16·2] of all deaths in 2019). The leading Level 2 risk factor for attributable DALYs globally in 2019 was child and maternal malnutrition,

표-863. PubMed 논문번호 33074736의 내용 요약

구분	내용
PubMed ID	33074736
TITLE	Association between Kawasaki Disease and Prenatal Exposure to Ambient and Industrial Air Pollution: A Population-Based Cohort Study.
JOURNAL	Environmental health perspectives: 10.1289/EHP6920
AUTHORS	Buteau Stephane, Belkaibech Sabrina, Bilodeau-Bertrand Marianne, Hatzopoulou Marianne, Smargiassi Audrey, Auger Nathalie
BACKGROUND	Environmental factors may contribute to the development of Kawasaki disease in children, but prenatal environmental exposures are understudied.
OBJECTIVE	We used a population-based cohort to investigate whether prenatal exposure to outdoor air pollution is associated with the incidence of Kawasaki disease in childhood.
METHODS	We performed a longitudinal cohort study of all children born in Quebec, Canada, between 2006 and 2012. Children were followed for Kawasaki disease from birth until 31 March 2018. We assigned prenatal air pollutant exposure according to the residential postal code at birth. The main exposure was annual average concentration of ambient fine particulate matter [PM $\leq 2.5\ \mu$m in aerodynamic diameter (PM2.5) and nitrogen dioxide (NO2) from satellite-based estimates and land-use regression models. As secondary exposures, we considered industrial PM2.5, NO2, and sulfur dioxide (SO2) emissions estimated from dispersion models. We estimated hazard ratios (HRs) using Cox proportional hazards models, adjusted for maternal age, parity, sex, multiple birth, maternal smoking during pregnancy, socioeconomic status, birth year, and rural residence. We considered single and multipollutant models. We performed several sensitivity analyses, including assessing modifying effects of maternal comorbidities (e.g., diabetes, preeclampsia).
RESULTS	The cohort comprised 505,336 children, including 539 with Kawasaki disease. HRs for each interquartile range increase in ambient air pollution were 1.16 (95% CI: 0.96, 1.39) for PM2.5 and 1.12 (95% CI: 0.96, 1.31) for NO2. For industrial air pollution, HRs were 1.07 (95% CI: 1.01, 1.13) for SO2, 1.09 (95% CI: 0.99, 1.20) for NO2, and 1.01 (95% CI: 0.97, 1.05) for PM2.5. In multipollutant models, associations for ambient PM2.5 and NO2 (i.e., from all sources) were robust to adjustment for industrial pollution, and vice versa.
DISCUSSION	In this population-based cohort study, both prenatal exposure to ambient and industrial air pollution were associated with the incidence of Kawasaki disease in childhood. Further studies are needed to consolidate the observed associations. https://doi.org/10.1289/EHP6920.

표-864. PubMed 논문번호 33081800의 내용 요약

구분	내용
PubMed ID	33081800
TITLE	The modifying role of physical activity in the cross-sectional and longitudinal association of health-related quality of life with physiological functioning-based latent classes and metabolic syndrome.
JOURNAL	Health and quality of life outcomes: 10.1186/s12955-020-01557-z
AUTHORS	Cerletti Paco, Keidel Dirk, Imboden Medea, Schindler Christian, Probst-Hensch Nicole
BACKGROUND	Single cardio-metabolic risk factors are each known modifiable risk factors for adverse health and quality of life outcomes. Yet, evidence on the clustered effect of these parameters and the metabolic syndrome (MetS) on health-related quality of life (HRQoL) is still limited and mostly cross-sectional. The objectives of this study were to identify clusters of cardio-metabolic physiological functioning, to assess their associations with HRQoL in comparison with the MetS, to elucidate the modifying role of physical activity, and to assess differences in health service utilization.
METHODS	This study is based on longitudinal data from two time points (2010/11 & 2017/18) of the Swiss Study on Air Pollution and Lung and Heart Diseases (SAPALDIA). Latent class analysis (LCA) grouped participants based on a priori selected cardio-metabolic and MetS related physiological functioning variables (Body mass index, body fat, glycated hemoglobin, blood triglycerides, blood pressure). The 36-item Short-Form Health Survey (SF-36) was used to assess HRQoL. Quantile regressions were performed with and without adjustment for physical activity, to detect independent associations of the latent classes, MetS and physical activity with HRQoL. To assess the modifying role of physical activity, we additionally grouped participants based on the combination of physical activity and latent classes or MetS, respectively. Logistic regressions were used to investigate health service utilization as outcome.
RESULTS	The LCA resulted in three classes labeled "Healthy" (30% of participants in 2017/18), "At risk" and "Unhealthy" (29%). The Unhealthy class scored lowest in all physical component scores of HRQoL. Compared to healthy and active participants, inactive participants in the "Unhealthy" class showed lower scores in the physical functioning domain both cross-sectionally (- 9.10 (- 12.02; - 6.18)) and longitudinally. This group had an odds ratio of 2.69 (1.52; 4.74) for being hospitalized in the previous 12 months.
CONCLUSIONS	These results point to subjects with adverse cardio-metabolic physiological functioning and low activity levels as an important target group for health promotion and maintenance of well-being. The promotion of physical activity at the early stages of aging seems pivotal to mitigate the impact of the MetS on HRQoL at higher age.

표-865. PubMed 논문번호 33091388의 내용 요약

구분	내용
PubMed ID	33091388
TITLE	Long-term effects of PM〈sub〉2·5〈/sub〉 on neurological disorders in the American Medicare population: a longitudinal cohort study.
JOURNAL	The Lancet. Planetary health: 10.1016/S2542-5196(20)30227-8
AUTHORS	Shi Liuhua, Wu Xiao, Danesh Yazdi Mahdieh, Braun Danielle, Abu Awad Yara, Wei Yaguang, Liu Pengfei, Di Qian, Wang Yun, Schwartz Joel, Dominici Francesca, Kioumourtzoglou Marianthi-Anna, Zanobetti Antonella
BACKGROUND	Accumulating evidence links fine particulate matter (PM2·5) to premature mortality, cardiovascular disease, and respiratory disease. However, less is known about the influence of PM2·5 on neurological disorders. We aimed to investigate the effect of long-term PM2·5 exposure on development of Parkinson's disease or Alzheimer's disease and related dementias.
METHODS	We did a longitudinal cohort study in which we constructed a population-based nationwide open cohort including all fee-for-service Medicare beneficiaries (aged ≥65 years) in the contiguous United States (2000-16) with no exclusions. We assigned PM2·5 postal code (ie, ZIP code) concentrations based on mean annual predictions from a high-resolution model. To accommodate our very large dataset, we applied Cox-equivalent Poisson models with parallel computing to estimate hazard ratios (HRs) for first hospital admission for Parkinson's disease or Alzheimer's disease and related dementias, adjusting for potential confounders in the health models.
FINDINGS	Between Jan 1, 2000, and Dec 31, 2016, of 63 038 019 individuals who were aged 65 years or older during the study period, we identified 1·0 million cases of Parkinson's disease and 3·4 million cases of Alzheimer's disease and related dementias based on primary and secondary diagnosis billing codes. For each 5 μg/m3 increase in annual PM2·5 concentrations, the HR was 1·13 (95% CI 1·12-1·14) for first hospital admission for Parkinson's disease and 1·13 (1·12-1·14) for first hospital admission for Alzheimer's disease and related dementias. For both outcomes, there was strong evidence of linearity at PM2·5 concentrations less than 16 μg/m3 (95th percentile of the PM2·5 distribution), followed by a plateaued association with increasingly larger confidence bands.
INTERPRETATION	We provide evidence that exposure to annual mean PM2·5 in the USA is significantly associated with an increased hazard of first hospital admission with Parkinson's disease and Alzheimer's disease and related dementias. For the ageing American population, improving air quality to reduce PM2·5 concentrations to less than current national standards could yield substantial health benefits by reducing the burden of neurological disorders.
FUNDING	The Health Effects Institute, The National Institute of Environmental Health Sciences, The National Institute on Aging, and the HERCULES Center.

표-866. PubMed 논문번호 33099063의 내용 요약

구분	내용
PubMed ID	33099063
TITLE	Urban environment during early-life and blood pressure in young children.
JOURNAL	Environment international: 10.1016/j.envint.2020.106174
AUTHORS	Warembourg Charline, Nieuwenhuijsen Mark, Ballester Ferran, de Castro Montserrat, Chatzi Leda, Esplugues Ana, Heude Barbara, Maitre L□a, McEachan Rosemary, Robinson Oliver, Slama R□my, Sunyer Jordi, Urquiza Jose, Wright John, Basaga□a Xavier, Vrijheid Martine
BACKGROUND	The urban environment is characterised by many exposures that may influence hypertension development from early life onwards, but there is no systematic evaluation of their impact on child blood pressure (BP).
METHODS	Systolic and diastolic blood pressure were measured in 4,279 children aged 4-5 years from a multi-centre European cohort (France, Greece, Spain, and UK). Urban environment exposures were estimated during pregnancy and childhood, including air pollution, built environment, natural spaces, traffic, noise, meteorology, and socioeconomic deprivation index. Single- and multiple-exposure linear regression models and a cluster analysis were carried out.
RESULTS	In multiple exposure models, higher child BP, in particular diastolic BP, was observed in association with higher exposure to air pollution, noise and ambient temperature during pregnancy, and with higher exposure to air pollution and higher building density during childhood (e.g., mean change [95% confidence interval] for an interquartile range increase in prenatal NO2 = 0.7 mmHg[0.3;1.2]). Lower BP was observed in association with higher temperature and better street connectivity during childhood (e.g., temperature = -1.1[-1.6;-0.6]). Some of these associations were not robust in the sensitivity analyses. Mother-child pairs were grouped into six urban environment exposure clusters. Compared to the cluster representing the least harmful urban environment, the two clusters representing the most harmful environment (high in air pollution, traffic, noise, and low in green space) were both associated with higher diastolic BP (1.3[0.1;2.6] and 1.5[0.5;2.5]).
CONCLUSION	This first large systematic study suggests that living in a harmful urban environment may impact BP regulation in children. These findings reinforce the importance of designing cities that promote healthy environments to reduce long-term risk of hypertension and other cardiovascular diseases.

표-867. PubMed 논문번호 33119678의 내용 요약

구분	내용
PubMed ID	33119678
TITLE	The effect of air-pollution and weather exposure on mortality and hospital admission and implications for further research: A systematic scoping review.
JOURNAL	PloS one: 10.1371/journal.pone.0241415
AUTHORS	Abed Al Ahad Mary, Sullivan Frank, Dem□ar Ur□ka, Melhem Maya, Kulu Hill
BACKGROUND	Air-pollution and weather exposure beyond certain thresholds have serious effects on public health. Yet, there is lack of information on wider aspects including the role of some effect modifiers and the interaction between air-pollution and weather. This article aims at a comprehensive review and narrative summary of literature on the association of air-pollution and weather with mortality and hospital admissions; and to highlight literature gaps that require further research.
METHODS	We conducted a scoping literature review. The search on two databases (PubMed and Web-of-Science) from 2012 to 2020 using three conceptual categories of "environmental factors", "health outcomes", and "Geographical region" revealed a total of 951 records. The narrative synthesis included all original studies with time-series, cohort, or case cross-over design; with ambient air-pollution and/or weather exposure; and mortality and/or hospital admission outcomes.
RESULTS	The final review included 112 articles from which 70 involved mortality, 30 hospital admission, and 12 studies included both outcomes. Air-pollution was shown to act consistently as risk factor for all-causes, cardiovascular, respiratory, cerebrovascular and cancer mortality and hospital admissions. Hot and cold temperature was a risk factor for wide range of cardiovascular, respiratory, and psychiatric illness; yet, in few studies, the increase in temperature reduced the risk of hospital admissions for pulmonary embolism, angina pectoris, chest, and ischemic heart diseases. The role of effect modification in the included studies was investigated in terms of gender, age, and season but not in terms of ethnicity.
CONCLUSION	Air-pollution and weather exposure beyond certain thresholds affect human health negatively. Effect modification of important socio-demographics such as ethnicity and the interaction between air-pollution and weather is often missed in the literature. Our findings highlight the need of further research in the area of health behaviour and mortality in relation to air-pollution and weather, to guide effective environmental health precautionary measures planning.

표-868. PubMed 논문번호 33120275의 내용 요약

구분	내용
PubMed ID	33120275
TITLE	The relationship between long-term exposure to PM_{2.5} and hypertension in women : A meta-analysis.
JOURNAL	Ecotoxicology and environmental safety: 10.1016/j.ecoenv.2020.111492
AUTHORS	Ma Yuexiao, Sun Mengqi, Liang Qingqing, Wang Fenghong, Lin Lisen, Li Tianyu, Duan Junchao, Sun Zhiwei
OBJECTIVE	Gender difference and PM2.5 exposure all have effects on hypertension, change of estrogen level in different women's stage bring complex influence on blood pressure. Then we conduct this meta-analysis to investigate the association between long-term exposure (at least one year) to fine particulate matter (PM2.5) and hypertension in adult non-pregnant women.
METHOD	Four major databases: PubMed, Cochrane Library, Web of Science and Embase were searched with specific search terms, and 11 studies were finally selected. The meta-analysis module of software Stata 12.0 was used for data processing with the effect values hazard ratio (HR) and odds ratio (OR) respectively.
RESULTS	After sensitivity analysis, we removed a study with highly heterogeneity and finally included 10 studies. Meta-analysis results showed that exposure to PM2.5 (per 10 μg/m3 increase) was associated with hypertension in non-pregnancy adult women, HR = 1.23, 95%CI: 1.08-1.40; OR = 1.07, 95%CI: 1.00-1.14. And subgroup analysis showed that menopause, non-White and diabetes are the key risk factors of hypertension when exposed to PM2.5.
CONCLUSION	This is the first meta-analysis to explore the association between PM2.5 and non-pregnancy women, and calculate OR and HR respectively for the first time. Exposure to PM2.5 could increase the risk of hypertension in non-pregnancy women, and the combined 'HR' was much higher than 'OR'.

표-869. PubMed 논문번호 33142159의 내용 요약

구분	내용
PubMed ID	33142159
TITLE	Association between air pollutants and atrial fibrillation in general population: A systematic review and meta-analysis.
JOURNAL	Ecotoxicology and environmental safety: 10.1016/j.ecoenv.2020.111508
AUTHORS	Yue Chao, Yang Fan, Li Fengwei, Chen Yingtai
BACKGROUND	Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia with several risk factors. Recent studies have suggested that the exposure to air pollutants may increase the prevalence of AF, we evaluated those studies systematically to better elucidate the correlation between exposure to air pollution and AF.
METHOD	We conducted a systematic review of publications using PubMed, Embase, the Cochrane library and Web of Science to explore the association between air pollutants and AF within the general population. The chosen studies were published until 7 July 2020. According to different study designs, we divided the outcomes into "short-term-exposure group" and "long-term-exposure group" for each pollutant. We used I2 statistics and Q-test to examine statistical heterogeneity, and sensitivity analysis to exclude the heterogeneous study. Fixed or random-effect model was used to combine the effects. Final result was presented as the OR and 95% CI of AF prevalence for every 10 μg/m3 increase in the concentration of PM2.5 and PM10;10 ppb increase in the concentration of SO2 ,NO2 ,O3; and 1 ppm increase in the CO concentration.
RESULTS	Our analysis contain 18 studies. Underlying short-term exposure effect, for each increment of 10 μg/m3 in the PM2.5 concentration, the combined OR of AF prevalence was 1.01(1.00-1.02), for PM10 was 1.03(1.01-1.05). For a 10 ppb increment in the concentration of SO2, NO2, and O3 was 1.05(1.01-1.09), 1.03(1.01-1.04), and 1.01(0.97-1.06), respectively, for a 1 ppm increase of CO concentration was 1.02(0.99-1.06). Underlying long-term-exposure effect for each increment of 10 μg/m3 in the PM2.5 concentration; the combined OR of AF prevalence was 1.07(1.04-1.10) and that for PM10 was 1.03(1.03-1.04) For a 10 ppb increment in the NO2 concentration was 1.02(1.00-1.04).
CONCLUSION	Our meta-analysis indicated that all air pollutants exposure had an adverse effect on AF prevalence in general population.

표-870. PubMed 논문번호 33153486의 내용 요약

구분	내용
PubMed ID	33153486
TITLE	Association between coarse particulate matter and inflammatory and hemostatic markers in a cohort of midlife women.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-020-00663-1
AUTHORS	Davis Emilie, Malig Brian, Broadwin Rachel, Ebisu Keita, Basu Rupa, Gold Ellen B, Qi Lihong, Derby Carol A, Park Sung Kyun, Wu Xiangmei May
BACKGROUND	Exposure to particulate matter air pollution has been associated with cardiovascular disease (CVD) morbidity and mortality; however, most studies have focused on fine particulate matter (PM2.5) exposure and CVD. Coarse particulate matter (PM10-2.5) exposure has not been extensively studied, particularly for long-term exposure, and the biological mechanisms remain uncertain.
METHODS	We examined the association between ambient concentrations of PM10-2.5 and inflammatory and hemostatic makers that have been linked to CVD. Annual questionnaire and clinical data were obtained from 1694 women (≥ 55 years old in 1999) enrolled in the longitudinal Study of Women's Health Across the Nation (SWAN) at six study sites from 1999 to 2004. Residential locations and the USEPA air monitoring network measurements were used to assign exposure to one-year PM10-2.5, as well as co-pollutants. Linear mixed-effects regression models were used to describe the association between PM10-2.5 exposure and markers, including demographic, health and other covariates.
RESULTS	Each interquartile (4 μg/m3) increase in one-year PM10-2.5 exposure was associated with a 5.5% (95% confidence interval [CI]: 1.8, 9.4%) increase in levels of plasminogen activator inhibitor-1 (PAI-1) and 4.1% (95% CI: -0.1, 8.6%) increase in high-sensitivity C-creative Protein (hs-CRP). Stratified analyses suggested that the association with PAI-1 was particularly strong in some subgroups, including women who were peri-menopausal, were less educated, had a body mass index lower than 25, and reported low alcohol consumption. The association between PM10-2.5 and PAI-1 remained unchanged with adjustment for PM2.5, ozone, nitrogen dioxide, and carbon monoxide.
CONCLUSIONS	Long-term PM10-2.5 exposure may be associated with changes in coagulation independently from PM2.5, and thus, contribute to CVD risk in midlife women.

표-871. PubMed 논문번호 33153628의 내용 요약

구분	내용
PubMed ID	33153628
TITLE	Particulate Matter and Temperature: Increased Risk of Adverse Clinical Outcomes in Patients With Atrial Fibrillation.
JOURNAL	Mayo Clinic proceedings: 10.1016/j.mayocp.2020.05.046
AUTHORS	Rivera-Caravaca Jos□ Miguel, Rold□n Vanessa, Vicente Vicente, Lip Gregory Y H, Mar□n Francisco
OBJECTIVE	To test the hypothesis that particulate matter with an aerodynamic diameter of less than 10 μm (PM10) and temperature are associated with an increased risk of adverse clinical outcomes in patients with atrial fibrillation (AF) taking vitamin K antagonists (VKAs).
PATIENTS AND METHODS	We included patients with AF whose condition was stable while taking VKAs (international normalized ratio, 2.0 to 3.0) for 6 months seen in a tertiary hospital (recruitment from May 1, 2007, to December 1, 2007). During a median follow-up of 6.5 years (interquartile range, 4.3 to 7.9 years), ischemic strokes, major bleeding, adverse cardiovascular events, and mortality were recorded. From 2007 to 2016, data on average temperature and PM10 were compared with clinical outcomes.
RESULTS	The study group included 1361 patients (663 [48.7%] male; median age, 76 years [interquartile range, 71 to 81 years]). High PM10 and low temperatures were associated with higher risk of major bleeding (adjusted hazard ratio [aHR], 1.44; 95% CI, 1.22 to 1.70 and aHR, 1.03; 95% CI, 1.01 to 1.05, respectively) and mortality (aHR, 1.50; 95% CI, 1.34 to 1.69 and aHR, 1.04; 95% CI, 1.02 to 1.06, respectively); PM10 was also associated with ischemic stroke and temperature with cardiovascular events. The relative risk (RR) for cardiovascular events and mortality increased in months in the lower quartile of temperature (RR, 1.12; 95% CI, 1.04 to 1.21 and RR, 1.41; 95% CI, 1.15 to 1.74, respectively). Comparing seasons, there were higher risks of cardiovascular events in spring, autumn, and winter than in summer, whereas the risk of mortality increased only in winter.
CONCLUSION	In patients with AF taking VKAs, high PM10 and low temperature were associated with increased risk of ischemic stroke and cardiovascular events, respectively. Both factors increased major bleeding and mortality risks, which were higher during colder months and seasons.

표-872. PubMed 논문번호 33167999의 내용 요약

구분	내용
PubMed ID	33167999
TITLE	Incident cardiovascular disease and particulate matter air pollution in South Korea using a population-based and nationwide cohort of 0.2 million adults.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-020-00671-1
AUTHORS	Kim Ok-Jin, Lee Soo Hyun, Kang Si-Hyuck, Kim Sun-Young
BACKGROUND	While many studies reported the association between long-term exposure to particulate matter air pollution (PM) and cardiovascular disease (CVD), few studies focused on incidence with relatively high-dose exposure using a nationwide cohort. This study aimed to investigate the association between long-term exposure to PM10 and PM2.5 and incidence of CVD in a nationwide and population-based cohort in South Korea where the annual average concentration of PM2.5 is above 20 μg/m3.
METHODS	We selected 196,167 adults in the National Health Insurance Service-National Sample Cohort (NHIS-NSC) constructed based on the entire South Korean population. Incidence of four CVD subtypes including ischemic heart disease (IHD), myocardial infarction, heart failure, and stroke, and total CVD including all four was identified as the first diagnosis for 2007-2015. To assess individual exposures, we used annually-updated district-level residential addresses and district-specific PM concentrations predicted by a previously developed universal kriging prediction model. We computed individual-level long-term PM concentrations for four exposure windows: previous 1, 3, and 5 year(s) and 5 years before baseline. We applied time-dependent Cox proportional hazards models to estimate hazard ratios (HRs) of incident CVDs per 10 μg/m3 increase in PM10 and PM2.5 after adjusting for individual- and area-level characteristics.
RESULTS	During 1,578,846 person-year, there were 33,580 cases of total incident CVD. Average PM10 and PM2.5 concentrations for the previous 5 years were 52.3 and 28.1 μg/m3, respectively. A 10 μg/m3 increase in PM2.5 exposed for the previous 5 years was associated with 4 and 10% increases in the incidence of total CVD (95% confidence interval: 0-9%) and IHD (4-16%), respectively. HRs tended to be higher with earlier exposure for IHD and more recent exposure for stroke. The estimated shape of the concentration-response relationship showed non-linear patterns. We did not find evidence of the association for PM10.
CONCLUSIONS	Using a population-based nationwide cohort exposed to relatively high PM concentration, this study confirmed the association between PM2.5 and CVD incidence that was reported in previous studies mostly with low-dose environments. The magnitude and the shape of the association were generally consistent with previous findings.

표-873. PubMed 논문번호 33183785의 내용 요약

구분	내용
PubMed ID	33183785
TITLE	Ambient sulfur dioxide and years of life lost from stroke in China: a time-series analysis in 48 cities.
JOURNAL	Chemosphere: 10.1016/j.chemosphere.2020.128857
AUTHORS	Wu Ziting, Li Jie, Huang Jing, Wang Yuxin, Cao Ru, Yin Peng, Wang Lijun, Zeng Qiang, Pan Xiaochuan, Zhou Maigeng, Li Guoxing
BACKGROUND AND OBJECTIVES	Sulfur dioxide (SO2) is a ubiquitous air pollutant and its concentration in China remains at a higher level in the world. However, evidence regarding short-term effect of SO2 on years of life lost (YLL) from stroke is scarce. We aim to estimate the short-term association between SO2 pollution and YLL for stroke and the related excess life years and economic loss.
METHODS	A national time-series study was conducted in 48 Chinese cities from 2013 to 2017. Generalized additive model coupled with random-effects model were used to explore the effects of SO2 on YLL from stroke. Stratified analyses were performed by demographical and geographical factors, and the effect modification of city-level factors was estimated. In addition, the related economic loss was calculated using the method of the value per statistical life year (VSLY).
RESULTS	Averaged daily mean SO2 concentration was 27.1 μg/m3 in 48 Chinese cities from 2013 to 2017. Per 10 μg/m3 increase in the concentration of SO2 (lag03) was associated with an increment of 0.70% (95% confidence interval: 0.27%,1.13%), 0.51% (-0.01%,1.04%), 0.71% (0.14%,1.28%) increase in YLL from total stroke, hemorrhagic and ischemic stroke, respectively. The effect of short-term ambient SO2 exposure on YLL from stroke was more pronounced in the less-educated population and those living in the south. The corresponding excess economic loss during the study period due to SO2-related YLL from stroke accounted for 0.08% (0.03%, 0.13%) of the GDP in China.
CONCLUSIONS	Our results provide evidence from China that short-term exposure to SO2 is positively associated with YLL from stroke and its major subtypes in certain subgroups of population. This study calls for greater awareness of the adverse health effect due to SO2 in China and other developing countries, as well as local-specific implementation of air pollution mitigation measures.

표-874. PubMed 논문번호 33213879의 내용 요약

구분	내용
PubMed ID	33213879
TITLE	Long-term exposure to fine particulate matter relates with incident myocardial infarction (MI) risks and post-MI mortality: A meta-analysis.
JOURNAL	Chemosphere: 10.1016/j.chemosphere.2020.128903
AUTHORS	Zhu Wentao, Cai Jiajie, Hu Yuchen, Zhang Haodan, Han Xiao, Zheng Huiqiu, Wu Jing
BACKGROUND	Air pollution has become a global challenge, and a growing number of studies have suggested possible relationships between long-term exposure to fine particulate matter (PM2.5) and risks of cardiovascular events, specifically, myocardial infarction (MI). However, the recently reported results were inconsistent. We thus performed a meta-analysis and sought to assess whether long-term exposure to PM2.5 relates with incident MI risks and post-MI mortality.
METHODS	EMBASE, Web of Science and PubMed were searched for all potentially eligible studies published before August 2, 2020 using a combination of keywords related to PM2.5 exposure, its long-term effects and myocardial infarction. Key information was extracted, and calculated hazard ratio (HR) values were combined by selecting corresponding models according to heterogeneity test. A sensitivity analysis and a publication bias assessment were also performed to determine the reliability of the results.
RESULTS	Of the initially identified 2100 citations, 12 studies met our inclusion criteria and observed a total population of approximately 7.2 million. Pooled estimates (per 10 μg/m3 increase) indicated a statistically significant association between long-term PM2.5 exposure and MI incidence (HR = 1.10, 95% CI: 1.02-1.18) or post-MI mortality (HR = 1.07, 95% CI: 1.04-1.09). Results for MI incidence from Egger's linear regression method (P = 0.515) and Begg's test (P = 0.711) showed no obvious publication bias.
CONCLUSION	Our quantitative analysis reveals a significant link between long-term PM2.5 exposure and greater MI incidence risks or higher post-MI mortality. Our findings may therefore have implications for individual protection and policy support to improve public health.

표-875. PubMed 논문번호 33221594의 내용 요약

구분	내용
PubMed ID	33221594
TITLE	Short-term differences in cardiac function following controlled exposure to cookstove air pollution: The subclinical tests on volunteers exposed to smoke (STOVES) study.
JOURNAL	Environment international: 10.1016/j.envint.2020.106254
AUTHORS	Cole-Hunter Tom, Dhingra Radhika, Fedak Kristen M, Good Nicholas, L'Orange Christian, Luckasen Gary, Mehaffy John, Walker Ethan, Wilson Ander, Balmes John, Brook Robert D, Clark Maggie L, Devlin Robert B, Volckens John, Peel Jennifer L
BACKGROUND	Exposure to household air pollution from solid fuel combustion for cooking and heating is an important risk factor for premature death and disability worldwide. Current evidence supports an association of ambient air pollution with cardiovascular disease but is limited for household air pollution and for cardiac function. Controlled exposure studies can complement evidence provided by field studies.
OBJECTIVES	To investigate effects of short-term, controlled exposures to emissions from five cookstoves on measures of cardiac function.
METHODS	Forty-eight healthy adults (46% female; 20-36 years) participated in six, 2-h exposures ('treatments'), including emissions from five cookstoves and a filtered-air control. Target fine particulate matter (PM2.5) exposure-concentrations per treatment were: control, 0 □g/m3; liquefied petroleum gas, 10 □g/m3; gasifier, 35 □g/m3; fan rocket, 100 □g/m3; rocket elbow, 250 □g/m3; and three stone fire, 500 □g/m3. Participants were treated in a set (pre-randomized) sequence as groups of 4 to minimize order bias and time-varying confounders. Heart rate variability (HRV) and cardiac repolarization metrics were calculated as 5-min means immediately and at 3 h following treatment, for analysis in linear mixed-effects models comparing cookstove to control.
RESULTS	Short-term differences in SDNN (standard deviation of duration of all NN intervals) and VLF (very-low frequency power) existed for several cookstoves compared to control. While all cookstoves compared to control followed a similar trend for SDNN, the greatest effect was seen immediately following three stone fire (β = −0.13 ms {%}; 95% confidence interval = −0.22, −0.03%), which reversed in direction at 3 h (0.03%; −0.06, 0.13%). VLF results were similar in direction and timing to SDNN; however, other HRV or cardiac repolarization results were not similar to those for SDNN.
DISCUSSION	We observed some evidence of short-term, effects on HRV immediately following cookstove treatments compared to control. Our results suggest that cookstoves with lower PM2.5 emissions are potentially capable of affecting cardiac function, similar to stoves emitting higher PM2.5 emissions.

표-876. PubMed 논문번호 33245887의 내용 요약

구분	내용
PubMed ID	33245887
TITLE	Application of high-resolution metabolomics to identify biological pathways perturbed by traffic-related air pollution.
JOURNAL	Environmental research: 10.1016/j.envres.2020.110506
AUTHORS	Li Zhenjiang, Liang Donghai, Ye Dongni, Chang Howard H, Ziegler Thomas R, Jones Dean P, Ebelt Stefanie T
BACKGROUND	Substantial research has investigated the adverse effects of traffic-related air pollutants (TRAP) on human health. Convincing associations between TRAP and respiratory and cardiovascular diseases are known, but the underlying biological mechanisms are not well established. High-resolution metabolomics (HRM) is a promising platform for untargeted characterization of molecular mechanisms between TRAP and health indexes.
OBJECTIVES	We examined metabolic perturbations associated with short-term exposures to TRAP, including carbon monoxide (CO), nitrogen dioxide (NO2), ozone (O3), fine particulate matter (PM2.5), organic carbon (OC), and elemental carbon (EC) among 180 participants of the Center for Health Discovery and Well-Being (CHDWB), a cohort of Emory University-affiliated employees.
METHODS	A cross-sectional study was conducted on baseline visits of 180 CHDWB participants enrolled during 2008-2012, in whom HRM profiling was determined in plasma samples using liquid chromatography-high-resolution mass spectrometry with positive and negative electrospray ionization (ESI) modes. Ambient pollution concentrations were measured at an ambient monitor near downtown Atlanta. Metabolic perturbations associated with TRAP exposures were assessed following an untargeted metabolome-wide association study (MWAS) framework using feature-specific Tobit regression models, followed by enriched pathway analysis and chemical annotation.
RESULTS	Subjects were predominantly white (76.1%) and non-smokers (95.6%), and all had at least a high school education. In total, 7821 and 4123 metabolic features were extracted from the plasma samples by the negative and positive ESI runs, respectively. There are 3421 features significantly associated with at least one air pollutant by negative ion mode, and 1691 features by positive ion mode. Biological pathways enriched by features associated with the pollutants are primarily involved in nucleic acids damage/repair (e.g., pyrimidine metabolism), nutrient metabolism (e.g., fatty acid metabolism), and acute inflammation (e.g., histidine metabolism and tyrosine metabolism). NO2 and EC were associated most consistently with these pathways. We confirmed the chemical identity of 8 metabolic features in negative ESI and 2 features in positive ESI, including metabolites closely linked to oxidative stress and inflammation, such as histamine, tyrosine, tryptophan, and proline.
CONCLUSIONS	We identified a range of ambient pollutants, including components of TRAP, associated with differences in the metabolic phenotype among the cohort of 180 subjects. We found Tobit models to be a robust approach to handle missing data among the metabolic features. The results were encouraging of further use of HRM and MWAS approaches for characterizing molecular mechanisms underlying exposure to TRAP.

표-877. PubMed 논문번호 33254849의 내용 요약

구분	내용
PubMed ID	33254849
TITLE	The effects of indoor air pollution from solid fuel use on cognitive function among middle-aged and older population in China.
JOURNAL	The Science of the total environment: 10.1016/j.scitotenv.2020.142460
AUTHORS	Luo Yanan, Zhong Yijing, Pang Lihua, Zhao Yihao, Liang Richard, Zheng Xiaoying
OBJECTIVES	Growing evidence has linked outdoor air pollution exposure with higher risk of cognitive impairments. However, the role of indoor air pollution in cognitive decline is not well elaborated. By using nationally representative longitudinal data, this study aimed to explore the effects of indoor air pollution from solid fuel use on cognitive function among middle-aged and older individuals in China.
METHODS	Data were obtained from 2011 to 2015 waves of CHARLS (China Health and Retirement Longitudinal Study). Scores from the Telephone Interview of Cognitive Status and figure drawing/word recall tests were used to measure cognitive function in 39,482 individuals. Exposure to indoor air pollution was measured as use of solid fuel for cooking. Solid fuel was defined as coal, biomass charcoal, wood, and straw; clean fuel was defined as liquefied gas, natural gas, and electricity. Linear mixed effect models were applied to examine the effect of indoor air pollution from solid fuel use on cognitive function.
RESULTS	Participants had an average global cognitive function of 9.67 (SD = 4.13). Solid fuel users made up 49.71% of participants, but this proportion was much greater among those living in rural areas (64.22%). Compared with clean fuel users, solid fuel users had worse cognitive function. On average, solid fuel users had a 0.81 (95%CI: −0.89, −0.73) lower global cognition score, 0.63 (95%CI: −0.69, −0.57) lower mental health score, and 0.16 (95%CI: −0.22, −0.14) lower episodic memory score. These effects were stronger among participants who are female, aged 65 years old and above, have education level of primary school and below, or have cardiovascular diseases.
CONCLUSIONS	These results provide evidence for the role of indoor air pollution in neurobehavioral disorders in China. Promotion of practices like expanded use of clean fuel and improved stoves in households may be crucial to significantly reduce indoor air pollution and protect mental health.

표-878. PubMed 논문번호 33261557의 내용 요약

구분	내용
PubMed ID	33261557
TITLE	Acute effect of ambient fine particulate matter on heart rate variability: an updated systematic review and meta-analysis of panel studies.
JOURNAL	Environmental health and preventive medicine: 10.1186/s12199-020-00912-2
AUTHORS	Niu Zhiping, Liu Feifei, Li Baojing, Li Na, Yu Hongmei, Wang Yongbo, Tang Hong, Chen Xiaolu, Lu Yuanan, Cheng Zilu, Liu Suyang, Chen Gongbo, Zhang Yuxiao, Xiang Hao
BACKGROUND	Decreased heart rate variability (HRV) is a predictor of autonomic system dysfunction, and is considered as a potential mechanism of increased risk of cardiovascular disease (CVD) induced by exposure to particulate matter less than 2.5 μm in diameter (PM2.5). Previous studies have suggested that exposure to PM2.5 may lead to decreased HRV levels, but the results remain inconsistent.
METHODS	An updated systematic review and meta-analysis of panel studies till November 1, 2019 was conducted to evaluate the acute effect of exposure to ambient PM2.5 on HRV. We searched electronic databases (PubMed, Web of Science, and Embase) to identify panel studies reporting the associations between exposure to PM2.5 and the four indicators of HRV (standard deviation of all normal-to-normal intervals (SDNN), root mean square of successive differences in adjacent normal-to-normal intervals (rMSSD), high frequency power (HF), and low frequency power (LF)). Random-effects model was used to calculate the pooled effect estimates.
RESULTS	A total of 33 panel studies were included in our meta-analysis, with 16 studies conducted in North America, 12 studies in Asia, and 5 studies in Europe. The pooled results showed a 10 μg/m3 increase in PM2.5 exposure which was significantly associated with a − 0.92% change in SDNN (95% confidence intervals (95%CI) − 1.26%, − 0.59%), − 1.47% change in rMSSD (95%CI − 2.17%, − 0.77%), − 2.17% change in HF (95%CI − 3.24%, − 1.10%), and − 1.52% change in LF (95%CI − 2.50%, − 0.54%), respectively. Overall, subgroup analysis suggested that short-term exposure to PM2.5 was associated with lower HRV levels in Asians, healthy population, and those aged ≥ 40 years.
CONCLUSION	Short-term exposure to PM2.5 was associated with decreased HRV levels. Future studies are warranted to clarity the exact mechanism of exposure to PM2.5 on the cardiovascular system through disturbance of autonomic nervous function.

표-879. PubMed 논문번호 33287833의 내용 요약

구분	내용
PubMed ID	33287833
TITLE	Synergistic health effects of air pollution, temperature, and pollen exposure: a systematic review of epidemiological evidence.
JOURNAL	Environmental health : a global access science source: 10.1186/s12940-020-00681-z
AUTHORS	Anenberg Susan C, Haines Shannon, Wang Elizabeth, Nassikas Nicholas, Kinney Patrick L
BACKGROUND	Exposure to heat, air pollution, and pollen are associated with health outcomes, including cardiovascular and respiratory disease. Studies assessing the health impacts of climate change have considered increased exposure to these risk factors separately, though they may be increasing simultaneously for some populations and may act synergistically on health. Our objective is to systematically review epidemiological evidence for interactive effects of multiple exposures to heat, air pollution, and pollen on human health.
METHODS	We systematically searched electronic literature databases (last search, April 29, 2019) for studies reporting quantitative measurements of associations between at least two of the exposures and mortality from any cause and cardiovascular and respiratory morbidity and mortality specifically. Following the Navigation Guide systematic review methodology, we evaluated the risk of bias of individual studies and the overall quality and strength of evidence.
RESULTS	We found 56 studies that met the inclusion criteria. Of these, six measured air pollution, heat, and pollen; 39 measured air pollution and heat; 10 measured air pollution and pollen; and one measured heat and pollen. Nearly all studies were at risk of bias from exposure assessment error. However, consistent exposure-response across studies led us to conclude that there is overall moderate quality and sufficient evidence for synergistic effects of heat and air pollution. We concluded that there is overall low quality and limited evidence for synergistic effects from simultaneous exposure to (1) air pollution, pollen, and heat; and (2) air pollution and pollen. With only one study, we were unable to assess the evidence for synergistic effects of heat and pollen.
CONCLUSIONS	If synergistic effects between heat and air pollution are confirmed with additional research, the health impacts from climate change-driven increases in air pollution and heat exposure may be larger than previously estimated in studies that consider these risk factors individually.

표-880. PubMed 논문번호 33289704의 내용 요약

구분	내용
PubMed ID	33289704
TITLE	Accelerated epigenetic age as a biomarker of cardiovascular sensitivity to traffic-related air pollution.
JOURNAL	Aging: 10.18632/aging.202341
AUTHORS	Ward-Caviness Cavin K, Russell Armistead G, Weaver Anne M, Slawsky Erik, Dhingra Radhika, Kwee Lydia Coulter, Jiang Rong, Neas Lucas M, Diaz-Sanchez David, Devlin Robert B, Cascio Wayne E, Olden Kenneth, Hauser Elizabeth R, Shah Svati H, Kraus William E
BACKGROUND	Accelerated epigenetic age has been proposed as a biomarker of increased aging, which may indicate disruptions in cellular and organ system homeostasis and thus contribute to sensitivity to environmental exposures.
METHODS	Using 497 participants from the CATHGEN cohort, we evaluated whether accelerated epigenetic aging increases cardiovascular sensitivity to traffic-related air pollution (TRAP) exposure. We used residential proximity to major roadways and source apportioned air pollution models as measures of TRAP exposure, and chose peripheral arterial disease (PAD) and blood pressure as outcomes based on previous associations with TRAP. We used Horvath epigenetic age acceleration (AAD) and phenotypic age acceleration (PhenoAAD) as measures of age acceleration, and adjusted all models for chronological age, race, sex, smoking, and socioeconomic status.
RESULTS	We observed significant interactions between TRAP and both AAD and PhenoAAD. Interactions indicated that increased epigenetic age acceleration elevated associations between proximity to roadways and PAD. Interactions were also observed between AAD and gasoline and diesel source apportioned PM2.5.
CONCLUSION	Epigenetic age acceleration may be a biomarker of sensitivity to air pollution, particularly for TRAP in urban cohorts. This presents a novel means by which to understand sensitivity to air pollution and provides a molecular measure of environmental sensitivity.

표-881. PubMed 논문번호 33296779의 내용 요약

구분	내용
PubMed ID	33296779
TITLE	Manuscript title: Long—term residential exposure to environmental/transportation noise and the incidence of myocardial infarction.
JOURNAL	International journal of hygiene and environmental health: 10.1016/j.ijheh.2020.113666
AUTHORS	Yankoty Larisa I, Gamache Philippe, Plante C□line, Goudreau Sophie, Blais Claudia, Perron St□phane, Fournier Michel, Ragettli Martina S, Fallah-Shorshani Masoud, Hatzopoulou Marianne, Liu Ying, Smargiassi Audrey
BACKGROUND	Cardiovascular effects of environmental noise are a growing concern. However, the evidence remains largely limited to the association between road traffic noise and hypertension and coronary heart diseases.
OBJECTIVES	To investigate the association between long-term residential exposure to environmental/transportation noise and the incidence of myocardial infarction (MI) in the adult population living in Montreal.
METHODS	An open cohort of adults aged 45 years old and over, living on the island of Montreal and free of MI before entering the cohort was created for the years 2000-2014 with the Quebec Integrated Chronic Disease Surveillance System; a systematic surveillance system from the Canadian province of Quebec starting in 1996. Residential noise exposure was calculated in three ways: 1) total ambient noise levels estimated by Land use regression (LUR) models; 2) road traffic noise estimated by a noise propagation model CadnaA and 3) distances to transportation sources (roads, airport, railways). Incident MI was based on diagnostic codes in hospital admission records. Cox models with time-varying exposures (age as the time axis) were used to estimate the associations with various adjustments (material deprivation indicator, calendar year, nitrogen dioxide, stratification for sex). Indirect adjustment based on ancillary data for smoking was performed.
RESULTS	1,065,414 individuals were followed (total of 9,000,443 person-years) and 40,718 (3.8%) developed MI. We found positive associations between total environmental noise, estimated by LUR models and the incidence of MI. Total noise LUR levels ranged from ~44 to ~79 dBA and varied slightly with the metric used. The adjusted hazard ratios (HRs) (also adjusted for smoking) were 1.12 (95% Confidence Intervals [CI]: 1.08-1.15), 1.11 (95%CI: 1.07-1.14) and 1.10 (95%CI: 1.06-1.14) per 10 dBA noise levels increase respectively in Level Accoustic equivalent 24 h (LAeq24 h), Level day-evening-night (Lden) and night level (Lnight). We found a borderline negative association between road noise levels estimated with CadnaA and MI (HR: 0.99 per 10 dBA; 95%CI: 0.98-1.00). Distances to major roads and highways were not associated with MI while the proximity to railways was positively associated with MI (HR for ≤100 vs 〉 1000 m: 1.07; 95%CI: 1.01-1.14). A negative association was found with the proximity to the airport noise exposure forecast (NEF25); HR (〈1 vs 〉1000 m) = 0.88 (95%CI: 0.81-0.96).
CONCLUSIONS	These associations suggest that exposure to total environmental noise at current urban levels may be related to the incidence of MI. Additional studies with more accurate road noise estimates are needed to explain the counterintuitive associations with road noise and specific transportation sources.

표-882. PubMed 논문번호 33303450의 내용 요약

구분	내용
PubMed ID	33303450
TITLE	Regular running in an air-polluted environment: physiological and anthropometric protocol for a prospective cohort study (Healthy Aging in Industrial Environment Study - Program 4).
JOURNAL	BMJ open: 10.1136/bmjopen-2020-040529
AUTHORS	Cipryan Lukas, Kutac Petr, Dostal Tomas, Zimmermann Matthew, Krajcigr Miroslav, Jandackova Vera, Sram Radim, Jandacka Daniel, Hofmann Peter
INTRODUCTION	Ambient air pollution is a global environmental problem, which causes adverse health effects and premature deaths worldwide. Although regular exercise and physical activity have evident health benefits, the influence of long-term air pollution exposure during regular outdoor running has not been definitively clarified.
METHODS AND ANALYSIS	This study protocol describes the physiological and anthropometric perspectives of the 'Healthy Aging in Industrial Environment' Study - Programme 4 (4HAIE). The 4HAIE research project is intended to be a single-centre, prospective, longitudinal and multidisciplinary cohort study. The presented study protocol describes the cross-sectional measurements and analyses. Overall, 1500 adult participants (age 18-65 years), runners and inactive individuals, living in a high or low air-polluted area of the Czech Republic will be recruited. We will measure and analyse biomarkers of oxidative stress and inflammation in the blood, exercise capacity (graded exercise test and spiroergometry), blood pressure, lung function (spirometry), cardiac autonomic regulation and anthropometry (body composition).
ETHICS AND DISSEMINATION	The 4HAIE study protocol has already been approved by the Ethics Committee of the University of Ostrava (3/2018). A detailed participant information sheet will be provided to each individual prior to obtaining their written informed consent. The study poses little to no risk to participants. The findings of this study will be disseminated at regional and international conferences, in peer-reviewed journals and via social and broadcast media.

표-883. PubMed 논문번호 33323443의 내용 요약

구분	내용
PubMed ID	33323443
TITLE	Association of short-term exposure to ambient PM〈sub〉2.5〈 hospital admissions and 30-day readmissions in end-stage re patients: population-based retrospective cohort study.
JOURNAL	BMJ open: 10.1136/bmjopen-2020-041177
AUTHORS	Wyatt Lauren H, Xi Yuzhi, Kshirsagar Abhijit, Di Qian, Ward Cavin, Wade Timothy J, Cascio Wayne E, Rappold Ana G
OBJECTIVES	To examine the effect of short-term exposure to ambient fine matter (PM2.5) on all-cause, cardiovascular and respiratory- admissions and readmissions among patients receiving outpat haemodialysis.
DESIGN	Retrospective cohort study.
SETTING	Inpatient hospitalisation claims identified from the US Renal I 530 US counties.
PARTICIPANTS	All patients receiving in-centre haemodialysis between 2008 a
PRIMARY AND SECONDARY OUTCOME MEASURES	Risk of all-cause, cardiovascular and respiratory-related hosp and 30-day all-cause and cause-specific readmission followi cardiovascular, and respiratory-related discharges. Readmissi evaluated for early (1-7 days postdischarge) and late (8-30 d postdischarge) readmission time periods. Relative risk is expre μg/m3 of PM2.5.
RESULTS	Same-day ambient PM2.5 was associated with increased hosp risk for cardiovascular causes (0.9%, 95% CI 0.2 to 1.7). Grea PM2.5-related associations were observed with 30-day readm Early-readmission risk was increased by 1.6%-1.8% followin (1.6%, 95% CI 0.6% to 2.6%), cardiovascular (1.8%, 95% CI and respiratory (1.8%, 95% CI 0.4% to 3.2%) discharges; wh late-readmission risk increased by 1.2%-1.3% following all- cardiovascular discharges. PM2.5-related associations with re were greatest for certain cause-specific readmissions ranging dysrhythmia and conduction disorder, heart failure, chronic o pulmonary disease, other non-cardiac chest pain or respirato pneumonia. Following all-cause discharges, the cause-specifi early-readmission risk was increased by 6.5% (95% CI 3.5% pneumonia, 4.8% (95% CI 2.3% to 7.4%) for dysrhythmia an disorder, 3.7% (95% CI 1.4% to 6.0%) for heart failure and 2 1.2% to 4.2%) for other non-cardiac chest pain or respirator syndrome-related causes.
CONCLUSIONS	Daily ambient PM2.5 was associated with an increased risk o admissions and 30-day readmissions following cardiopulmon discharges in a vulnerable end-stage renal disease population. week following discharge, greater PM2.5-related risk of reho identified for some diagnoses.

표-884. PubMed 논문번호 33326859의 내용 요약

구분	내용
PubMed ID	33326859
TITLE	The Risk of Spread of Infection During Craniotomy/Craniostomy on Patients with Active Coronavirus Disease 2019 (COVID-19) Infection: Myth or Fact?
JOURNAL	World neurosurgery: 10.1016/j.wneu.2020.12.040
AUTHORS	Singh Apinderpreet, Salunke Pravin, Chhabra Rajesh, Sethi Sunil, Sahoo Sushanta K, Karthigeyan Madhivanan, Gendle Chandrasekhar, Kumar Rakesh, Gupta Sunil
OBJECTIVES	Craniotomies/craniostomies have been categorized as aerosol-generating procedures and are presumed to spread coronavirus disease 2019 (COVID-19). However, the presence of severe acute respiratory distress syndrome coronavirus 2 virus in the generated bone dust has never been proved. Our objective is to evaluate the presence of virus in the bone dust (aerosol) generated during emergency neurosurgical procedures performed on patients with active COVID-19. This would determine the true risk of disease transmission during the surgery.
METHODS	Ten patients with active COVID-19 infection admitted to our institute in 1 month required emergency craniotomy/craniostomy. The bone dust and mucosal scrapings form paranasal sinuses (if opened) collected during these procedures were tested for the virus using reverse transcription polymerase chain reaction. The entire surgical team was observed for any symptoms related to COVID-19 for 14 days following surgery.
RESULTS	Nine patients had moderate viral load in their nasopharyngeal cavity, as detected on reverse transcription polymerase chain reaction. None of the samples of bone dust from these 10 patients tested positive. Mucosal scrapping obtained in 1 patient in which mastoid air cells were inadvertently opened tested negative as well. No health workers from the operating room developed COVID-19-related symptoms.
CONCLUSIONS	The bone dust generated during craniotomy/stomy of active patients does not contain the virus. The procedure on an active patient is unlikely to spread the disease. However, a study with larger cohort would be confirmatory.

표-885. PubMed 논문번호 33395948의 내용 요약

구분	내용
PubMed ID	33395948
TITLE	Comparison of associations between mortality and air pollution exposure estimated with a hybrid, a land-use regression and a dispersion model.
JOURNAL	Environment international: 10.1016/j.envint.2020.106306
AUTHORS	Klompmaker Jochem O, Janssen Nicole, Andersen Zorana J, Atkinson Richard, Bauwelinck Mariska, Chen Jie, de Hoogh Kees, Houthuijs Danny, Katsouyanni Klea, Marra Marten, Oftedal Bente, Rodopoulou Sophia, Samoli Evangelia, Stafoggia Massimo, Strak Maciej, Swart Wim, Wesseling Joost, Vienneau Danielle, Brunekreef Bert, Hoek Gerard
INTRODUCTION	To characterize air pollution exposure at a fine spatial scale, different exposure assessment methods have been applied. Comparison of associations with health from different exposure methods are scarce. The aim of this study was to evaluate associations of air pollution based on hybrid, land-use regression (LUR) and dispersion models with natural cause and cause-specific mortality.
METHODS	We followed a Dutch national cohort of approximately 10.5 million adults aged 29+ years from 2008 until 2012. We used Cox proportional hazard models with age as underlying time scale and adjusted for several potential individual and area-level socio-economic status confounders to evaluate associations of annual average residential NO2, PM2.5 and BC exposure estimates based on two stochastic models (Dutch LUR, European-wide hybrid) and deterministic Dutch dispersion models.
RESULTS	Spatial variability of PM2.5 and BC exposure was smaller for LUR compared to hybrid and dispersion models. NO2 exposure variability was similar for the three methods. Pearson correlations between hybrid, LUR and dispersion modeled NO2 and BC ranged from 0.72 to 0.83; correlations for PM2.5 were slightly lower (0.61-0.72). In general, all three models showed stronger associations of air pollutants with respiratory disease and lung cancer mortality than with natural cause and cardiovascular disease mortality. The strength of the associations differed between the three exposure models. Associations of air pollutants estimated by LUR were generally weaker compared to associations of air pollutants estimated by hybrid and dispersion models. For natural cause mortality, we found a hazard ratio (HR) of 1.030 (95% confidence interval (CI): 1.019, 1.041) per 10 □g/m3 for hybrid modeled NO2, a HR of 1.003 (95% CI: 0.993, 1.013) per 10 □g/m3 for LUR modeled NO2 and a HR of 1.015 (95% CI: 1.005, 1.024) per 10 □g/m3 for dispersion modeled NO2.
CONCLUSION	Air pollution was positively associated with natural cause and cause-specific mortality, but the strength of the associations differed between the three exposure models. Our study documents that the selected exposure model may contribute to heterogeneity in effect estimates of associations between air pollution and health.

표-886. PubMed 논문번호 Traffic-related air pollution (TRAP) contributes significantly to ambient air pollution, especially in urban settings. Air pollution has been established as a risk factor for hypertension and cardiovascular disease in adults, but this effect is less studied in other susceptible populations. There is increasing evidence that air pollution may adversely affect hypertensive disorders of pregnancy (e.g., gestational hypertension, preeclampsia, eclampsia).의 내용 요약

구분	내용
INTRODUCTION	Traffic-related air pollution (TRAP) contributes significantly to ambient air pollution, especially in urban settings. Air pollution has been established as a risk factor for hypertension and cardiovascular disease in adults, but this effect is less studied in other susceptible populations. There is increasing evidence that air pollution may adversely affect hypertensive disorders of pregnancy (e.g., gestational hypertension, preeclampsia, eclampsia).
OBJECTIVE	Because reports indicate that air pollution may be linked to hypertensive disorders, the National Toxicology Program (NTP) conducted a systematic review to evaluate whether exposure to TRAP during pregnancy is associated with hypertensive disorders of pregnancy.
METHODS	A systematic review protocol was developed and utilized for this evaluation that followed the Office of Health Assessment and Translation approach for conducting literature-based health assessments. This evaluation considered a range of traffic-related air pollutant measurements (e.g., fine particulate matter [PM2.5]) and traffic measures (e.g., proximity to major roads) in the literature search. Confidence ratings and level-of-evidence conclusions were developed for bodies of evidence for a given exposure measure when there was sufficient evidence (i.e., more than three studies). Changes in blood pressure during pregnancy, gestational hypertension, preeclampsia, eclampsia, or hemolysis, elevated liver enzyme levels, and low platelet count (HELLP) syndrome were considered as measures of hypertension. Hazard conclusions were developed using a two-step process. First, confidence ratings were developed for individual air pollutants (e.g., PM2.5, nitrogen oxides [NOx]) and traffic measures (traffic density and proximity to major roads). Overall hazard conclusions were then developed for TRAP, considering the combined bodies of evidence across different individual measures of traffic-related pollutants.
RESULTS AND EVIDENCE SYNTHESIS	The literature search and screening process identified 18 relevant epidemiological studies and one relevant animal study (from 344 potentially relevant references) that met the objective and the inclusion criteria. The human bodies of evidence for traffic-related PM2.5 and NO2 present a consistent pattern of findings that exposure to these pollutants is associated with the development of hypertensive disorders of pregnancy. There is a similar pattern of findings, but a smaller effect size, for bodies of evidence that residing in high-traffic density regions or in close proximity to major roads are associated with developing hypertensive disorders during pregnancy. There is a moderate level of evidence in the combined human body of evidence based primarily on the TRAP air pollutant studies with support from the traffic measures studies. There is an inadequate level of evidence in the animal body of evidence due to the lack of experimental animal studies identified for these measures. Evidence for other traffic-related pollutants that were identified (i.e., carbon monoxide [CO], black carbon [BC], and elemental carbon [EC]), including one animal study for CO, were few in number or provided inconsistent results across studies, and level-of-evidence conclusions were not reached.
DISCUSSION AND CONCLUSION	NTP concludes that exposure to TRAP is presumed to be a hazard to pregnant women for developing hypertensive disorders of pregnancy. This conclusion was based on moderate confidence and moderate level of evidence in the combined body of evidence from human studies reporting on multiple

표-887. PubMed 논문번호 33778334의 내용 요약

구분	내용
PubMed ID	33778334
TITLE	Indoor and outdoor road traffic noise and incident diabetes mellitus: Results from a longitudinal German cohort study.
JOURNAL	Environmental epidemiology (Philadelphia, Pa.): 10.1097/EE9.0000000000000037
AUTHORS	Ohlwein Simone, Hennig Frauke, Lucht Sarah, Matthiessen Clara, Pundt Noreen, Moebus Susanne, J□ckel Karl-Heinz, Hoffmann Barbara
Methods	We used data from 3,396 participants of age 45-75 years of the Heinz Nixdorf Recall study being non-diabetic at baseline (2000-2003). T2DM was defined via blood glucose level, incident intake of an anti-diabetic drug during follow-up or self-reported physician diagnosis at follow-up examination (2005-2008). Weighted 24-h (Lden) and night-time (Lnight) mean road traffic noise was assessed according to the European Union directive 2002/49/EC. Road traffic noise exposure indoors was modeled taking into account the participants' room orientation, ventilation behavior and window insulation (n = 2,697). We applied Poisson regression analyses to estimate relative risks (RRs) of incident T2DM, adjusting for demographic characteristics, lifestyle factors, and air pollution exposure (NO2 or PM2.5).
Results	A 10-dB(A) increase in outdoor road traffic noise (Lden) was associated with an RR of 1.09 (95% confidence interval, 0.96-1.24) for T2DM in the fully adjusted model. Models including PM2.5 or NO2 yielded RRs of 1.09 (0.96-1.24) and 1.11 (0.97-1.27), respectively. In analyses with road traffic noise (Lden) exposure indoors, we observed similar RRs with smaller confidence intervals (1.11 [1.01-1.21]).
Conclusions	Our analyses suggest that long-term exposure to indoor and outdoor road traffic noise may increase the risk of developing T2DM, independent of air pollution exposure.

표-888. PubMed 논문번호 33778339의 내용 요약

구분	내용
PubMed ID	33778339
TITLE	Long-term exposure to fine particulate matter and natural-cause and cause-specific mortality in Japan.
JOURNAL	Environmental epidemiology (Philadelphia, Pa.): 10.1097/EE9.0000000000000051
AUTHORS	Yorifuji Takashi, Kashima Saori, Tani Yasunari, Yamakawa Junji, Doi Hiroyuki
Background	A number of studies have linked long-term exposure to particulate matter with aerodynamic diameter <2.5 □m (PM2.5) with mortality, but most of these studies were conducted in Europe and North America. Studies in Asian countries had been conducted at relatively high exposures. We evaluated the association of long-term exposure to PM2.5 and natural-cause and cause-specific mortality in Japan, where PM2.5 levels are relatively low compared with levels in other Asian countries.
Methods	A cohort of 75,531 participants underwent basic health checkups in Okayama City in 2006 or 2007. We followed the participants until the end of 2016. Average PM2.5 levels from 2006 to 2010 were obtained and assigned to the participants by geographical location. We used the Cox proportional hazard models to estimate hazard ratios for a 5- μg/m3 increase in PM2.5 levels for natural-cause or cause-specific mortality, adjusting for potential confounders.
Results	PM2.5 exposure was associated with increased risk of mortality; the hazard ratios were 1.29 (95% confidence interval = 1.18, 1.41) for mortality from natural causes, 1.16 (1.02, 1.32) for cardiorespiratory mortality, and 1.63 (1.13, 2.34) for lung cancer mortality. PM2.5 exposure was more strongly associated with cardiorespiratory mortality from hypertension, pneumonia and influenza, and chronic obstructive pulmonary disease than with ischemic heart disease or cerebrovascular disease. Elderly participants and smokers tended to have higher effect estimates.
Conclusion	Long-term exposure to PM2.5 can increase the risk of natural-cause, cardiorespiratory, and lung cancer mortality in Japan.

표-889. PubMed 논문번호 33778342의 내용 요약

구분	내용
PubMed ID	33778342
TITLE	Associations between ambient air pollution and noise from road traffic with blood pressure and insulin resistance in children from Denmark.
JOURNAL	Environmental epidemiology (Philadelphia, Pa.): 10.1097/EE9.0000000000000069
AUTHORS	Pedersen Marie, Halldorsson Thorhallur I, Ketzel Matthias, Grandstr□m Charlotta, Raaschou-Nielsen Ole, Jensen Steen S, Grunnet Louise G, Vaag Allan, S ø rensen Mette, Olsen Sjurdur F
Background	Road traffic is a major source of air pollution and noise. Both exposures may contribute to increased blood pressure and metabolic disease; however, few studies have examined these relationships in children.
Objectives	We aimed to investigate whether long-term exposures to air pollution and noise from road traffic were associated with increased blood pressure and insulin resistance in children.
Methods	Cardiometabolic outcomes were derived from a follow-up examination of 629 children (10-15 years old) enrolled in the Danish National Birth Cohort. We evaluated associations with prenatal and postnatal residential exposure to nitrogen dioxide (NO2) and noise from road traffic (Lden) using historical addresses and linear regression models.
Results	A 10-unit increase in postnatal exposure to NO2 and Lden was associated with a 0.31 (-0.87, 1.48) and 0.18 (-0.61, 0.96) mmHg changes in diastolic blood pressure, respectively. In contrast, both exposures were associated with decreased systolic blood pressure. After adjustment and mutual adjustment for NO2, exposure to Lden was associated with a statistical significant decrease in systolic blood pressure both during prenatal and postnatal life, but the majority of the associations evaluated did not reach statistical significance. Inverse associations were observed for plasma fasting glucose, insulin, and HOMA of insulin resistance for both exposures, exposure windows, before and after adjustment.
Conclusions	The findings do not support evidence of associations between long-term exposures to air pollution and road traffic noise, increased blood pressure, and a metabolic profile characteristic of increased risk for glucose intolerance or type 2 diabetes later in life.