
Systemic and vascular effects of circulating diesel exhaust particulate matter.

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OBJECTIVE: Numerous studies have found an association between transiently increased particulate matter air pollution and acute adverse cardiovascular health effects; however, the mechanisms underlying these effects are not clear. Translocation of ultra-fine ambient particulate matter has been proposed to play a key role in these acute side effects. This study was designed to determine the contribution of circulating (translocated) diesel exhaust particles (DEPs) to the systemic and vascular effects.

METHODS: C57 mice (10-week) received intravenous DEPs via tail vein injection. Following 1-h post-injection, inflammatory cytokines (IL-1β, IL-6 and TNF-α), peripheral blood cell counts, band cell counts, aortic endothelial function and vascular constriction were assessed. Thoracic aortae were isolated, and endothelial function was examined by measuring acetylcholine (ACh) and sodium nitroprusside (SNP)-stimulated vascular relaxation using a wire myograph. In addition, phenylephrine (PE)-stimulated vasoconstriction was also measured. The amount of DEPs deposited and trapped in tissues (the spleen,
liver, lungs and heart) were quantified. RESULTS: Acute systemic DEP exposure caused a significant increase in TNF-\(\alpha\), peripheral neutrophil and band cell counts. ACh and SNP-induced relaxation were not affected by acute systemic DEP exposure, neither was PE-stimulated constriction. There was a significantly increased DEP deposition in the spleen as well as in the liver. No significantly increased DEPs were detected in the lung and heart. CONCLUSION: Here we show that circulating DEPs induce a systemic response characterized by increased TNF-\(\alpha\), peripheral granulocytes, but does not impact endothelial function. Our study also suggests that circulating particles are rapidly removed from the circulation and predominantly sequestered in the spleen and liver.

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Health impact assessment of increasing public transport and cycling use in Barcelona: a morbidity and burden of disease approach.

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OBJECTIVE: Quantify the health impacts on morbidity of reduced car trips and increased public transport and cycling trips.

METHODS: A health impact assessment study of morbidity outcomes related to replacing car trips in Barcelona metropolitan (3,231,458 inhabitants). Through 8 different transport scenarios, the number of cases of disease or injuries related to physical activity, particulate matter air pollution <2.5 μm (PM2.5) and traffic incidents in travelers was estimated. We also estimate PM2.5 exposure and cases of disease in the general population.

RESULTS: A 40% reduction in long-duration car trips substituted by public transport and cycling trips resulted in annual reductions of 127 cases of diabetes, 44 of cardiovascular diseases, 30 of dementia, 16 minor injuries, 0.14 major injuries, 11 of breast cancer and 3 of colon-cancer, amounting to a total reduction of 302 Disability Adjusted Life Years per year in travelers. The reduction in PM2.5 exposure in the general population resulted in annual reductions of 7 cases of low birth weight, 6 of preterm birth, 1 of cardiovascular disease and 1 of lower respiratory tract infection.

CONCLUSIONS: Transport policies to reduce car trips could produce important health benefits in terms of reduced morbidity, particularly for those who take up active transportation.
Diesel exhaust particles modulate vascular endothelial cell permeability: implication of ZO-1 expression.

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Exposure to air pollutants increases the incidence of cardiovascular disease. Recent toxicity studies revealed that ultra-fine particles (UFP, d(p)<100-200 nm), the major portion of particulate matter (PM) by numbers in the atmosphere, induced atherosclerosis. In this study, we posited that variations in chemical composition in diesel exhausted particles (DEP) regulated endothelial cell permeability to a different extent. Human aortic endothelial cells (HAEC) were exposed to well-characterized DEP (d(p)<100 nm) emitted from a diesel engine in either idling mode (DEP1) or in urban dynamometer driving schedule (UDDS) (DEP2). Horse Radish Peroxidase-Streptavidin activity assay showed that DEP2 increased endothelial permeability to a greater extent than DEP1 (control=0.077+/−0.005, DEP1=0.175+/−0.003, DEP2=0.265+/−0.006, n=3, p<0.01). DEP2 also
down-regulated tight junction protein, Zonular Occludin-1 (ZO-1), to a greater extent compared to DEP1. LDH and caspase-3 activities revealed that DEP-mediated increase in permeability was not due to direct cytotoxicity, and DEP-mediated ZO-1 down-regulation was not due to a decrease in ZO-1 mRNA. Hence, our findings suggest that DEP1 vs. DEP2 differentially influenced the extent of endothelial permeability at the post-translational level. This increase in endothelium permeability is implicated in inflammatory cell transmigration into subendothelial layers with relevance to the initiation of atherosclerosis.

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Can we identify sources of fine particles responsible for exercise-induced ischemia on days with elevated air pollution? The ULTRA study.


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Epidemiologic studies have shown that ambient particulate matter (PM) has adverse effects on cardiovascular health. Effective mitigation of the health effects requires identification of the most harmful PM sources. The objective of our study was to evaluate relative effects of fine PM [aerodynamic diameter <or= 2.5 microm (PM2.5)] from different sources on exercise-induced ischemia. We collected daily outdoor PM2.5 samples between autumn 1998 and spring 1999 in Helsinki, Finland. The mass of PM2.5 was apportioned between five sources. Forty-five elderly nonsmoking persons with stable coronary heart disease visited a clinic biweekly for submaximal exercise testing, during which the occurrence of ST segment depressions was recorded. Levels of PM2.5 originating from local traffic and long-range transport were associated with ST segment depressions >0.1 mV, with odds ratios at 2-day lag of 1.53 [95% confidence interval (CI), 1.19-1.97] and 1.11 (95% CI, 1.02-1.20) per 1 microg/m^3, respectively. In multipollutant models, where we used indicator elements for sources instead of source-specific PM2.5, only absorbance (elemental carbon), an indicator of local traffic and other combustion, was associated with ST segment depressions. Our results suggest that the PM fraction originating from combustion processes, notably traffic, exacerbates ischemic heart diseases associated with PM mass.

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Determinants of personal and indoor PM2.5 and absorbance among elderly subjects with coronary heart disease.


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Epidemiological studies have established an association between outdoor levels of fine particles (PM2.5) and cardiovascular health. However, there is little information on the determinants of PM2.5 exposures among persons with cardiovascular disease, a potentially susceptible population group. Daily outdoor, indoor and personal PM2.5 and absorbance (proxy for elemental carbon) concentrations were measured among elderly subjects with cardiovascular disease in Amsterdam, the Netherlands, and Helsinki, Finland, during the winter and spring of 1998–1999 within the framework of the ULTRA study. There were 37 non-smoking subjects in Amsterdam and 47 in Helsinki. In Amsterdam, where there were enough exposure events for analyses, exposure to environmental tobacco smoke (ETS) indoors was a major source of between-subject variation in PM2.5 exposures, and a strong determinant of PM2.5 and absorbance exposures. When the days with ETS were excluded, within-subject variation accounted for 89% of the total variation in personal PM2.5 and 97% in absorbance in Amsterdam. The respective
figures were 66% and 61% in Helsinki. In both cities, outdoor levels of PM2.5 and absorbance were major determinants of personal and indoor levels. Traffic was also an important determinant of absorbance: living near a major street increased exposure by 22%, and every hour spent in a motor vehicle by 13% in Amsterdam. The respective increases were 37% and 9% in Helsinki. Cooking was associated with increased levels of both absorbance and PM2.5. Our results demonstrate that by using questionnaires in connection with outdoor measurements, exposure estimation of PM2.5 and its combustion originating fraction can be improved among elderly persons with compromised health.

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Effects of ultrafine and fine particulate and gaseous air pollution on cardiac autonomic control in subjects with coronary artery disease: the ULTRA study.


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Previous studies have shown an association between elevated concentrations of
particulate air pollution and cardiovascular morbidity and mortality. Therefore, the association between daily variation of ultrafine and fine particulate air pollution and cardiac autonomic control measured as heart rate variability (HRV) was studied in a large multicenter study in Amsterdam, the Netherlands, Erfurt, Germany, and Helsinki, Finland. Elderly subjects (n=37 in Amsterdam, n=47 in both Erfurt and Helsinki) with stable coronary artery disease were followed for 6 months with biweekly clinical visits. During the visits, ambulatory electrocardiogram was recorded during a standardized protocol including a 5-min period of paced breathing. Time and frequency domain analyses of HRV were performed. A statistical model was built for each center separately. The mean 24-h particle number concentration (NC) (1,000/cm(3)) of ultrafine particles (diameter 0.01–0.1 microm) was 17.3 in Amsterdam, 21.1 in Erfurt, and 17.0 in Helsinki. The corresponding values for PM2.5 were 20.0, 23.1, and 12.7 microg/m(3). During paced breathing, ultrafine particles, NO(2), and CO were at lags of 0–2 days consistently and significantly associated with decreased low-to-high frequency ratio (LF/HF), a measure of sympathovagal balance. In a pooled analysis across the centers, LF/HF decreased by 13.5% (95% confidence interval: -20.1%, -7.0%) for each 10,000/cm(3) increase in the NC of ultrafine particles (2-day lag). PM2.5 was associated with reduced HF and increased LF/HF in Helsinki, whereas the opposite was true in Erfurt, and in Amsterdam, there were no clear associations between PM2.5 and HRV. The results suggest that the
cardiovascular effects of ambient ultrafine and PM2.5 can differ from each other and that their effect may be modified by the characteristics of the exposed subjects and the sources of PM2.5.

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Personal, indoor, and outdoor exposures to PM2.5 and its components for groups of cardiovascular patients in Amsterdam and Helsinki.


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The aim of the investigation was to assess the relations between pairs of personal, indoor, and outdoor levels of fine particles and their components with respect to effects for older subjects with cardiovascular disease. In the framework of a study funded by the European Union (Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air; referred to as ULTRA)*, panel studies were conducted in Amsterdam (The Netherlands) and Helsinki (Finland). Concentrations of outdoor particulate matter 2.5 pm or smaller in aerodynamic diameter (PM2.5) were measured at a fixed site in each
location. With HEI funding, each subject's personal and indoor PM2.5 exposure was measured every other week for 6 months during the 24-hour period preceding intensive health measurements. Particle reflectance was measured as a marker for diesel exhaust. Elemental content of more than 50% of the personal and indoor samples and all corresponding outdoor samples was measured using x-ray fluorescence (XRF). Ion content (sulfate, nitrate) was measured using chromatography. For Amsterdam, 337 personal and 409 indoor measurements were collected from 37 subjects; for Helsinki, 336 personal and 503 indoor measurements were collected from 47 subjects. Median personal, indoor, and outdoor PM2.5 concentrations were 13.6, 13.6, and 16.5 microg/m3 in Amsterdam and 9.2, 9.2, and 11.1 microg/m3 in Helsinki. In both cities, personal and indoor PM2.5 concentrations were lower than and highly correlated with outdoor concentrations (median correlation coefficient [R] 0.7-0.8). For most elements, personal and indoor concentrations were also highly correlated with outdoor concentrations. The highest correlations (median R > 0.9) were found for sulfur (S), sulfate, and particle reflectance (reported as the absorption coefficient). Reflectance was a useful proxy for elemental carbon (EC), but site-specific calibration with EC data is necessary.

The findings of this study support using fixed-site measurements as a measure of exposure to PM in time-series studies linking the day-to-day variations in PM to the day-to-day variations in health endpoints, especially for components of PM that are generally associated with fine particles and have few
indoor sources.

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[Indoor air quality and occupational health, past and present].
[Article in Italian]

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The expression "indoor pollution" of residential, office and public buildings appeared for the first time in western societies toward the end of the '60s to indicate a complex phenomenon as important to public health as that of the "outdoor air pollution" or even more so. The demonstration of the toxic effects of passive smoking, radon, and other chemical and biological pollutants present in office and residential environments has prompted a wide spectrum of research into health risks, has led to position-taking by national and international authorities, and has given rise to a new scientific multi-disciplinary field of research, with respective international associations, scientific journals, and international conferences attended by thousands of participants. In Italy, since 1988, several scientific conferences have been organised and these have led to institutional initiatives such as the Commission set up in 1990 by the Italian
Environment Ministry and the Commission set up in 1998 by the Italian Ministry of Health. The latter produced a Prevention Plan for Health Protection and Promotion in Indoor Environments which, for the first time, tackles indoor air pollution in a systematic way and provides an overall picture of the health and economic impact of the main illnesses related to indoor pollution on society. Decree 626/94 has also been affected, in some way, by these new scientific findings and in art. 33 has produced an update of the old art. 9 of the Decree 303/56. The attention to the subject of indoor air quality, in addition to that of offices and commercial buildings, has turned in more recent years to special environments such as schools and hospitals, resulting in the production of important publications. The modern frontier of research on indoor and outdoor air hazards is represented by fine air particulate matter. A large number of worldwide epidemiological studies have revealed that the daily variation in fine and ultra-fine particle air concentration in urban areas is associated with the simultaneous daily variation in the morbidity and mortality of the general population. The particle-linked increase in mortality has been attributed to respiratory and cardiovascular toxic effects, but the mechanisms by which urban air particles (indoor and outdoor) induce worsening of respiratory and cardio-vascular diseases are so far unknown and are the subject of intense investigation. Workers employed in the tertiary sector (offices, trade, banking, hospitals, schools, etc.) now account for 80% of the Italian labour force and the
occupational physician is increasingly requested to assess the risk and monitor the health status of tertiary sector workers. These working environments are believed to be healthy and lacking in specific health risk factors, but such a belief is often only the result of the limited knowledge that employers, workers and the physicians themselves have about these environments and the results of international research studies over the last forty years. This issue is surely at the centre of the interest of our discipline and of public health throughout the developed western world and represents an ongoing challenge for the occupational physician, with new research topics and new problems to deal with. Recent issues include SARS and the defence of buildings and the air of working environments against terrorism attacks, such as the use of anthrax dust.

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Health effects of particles in ambient air.


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A summary of a critical review by a working group of the German commission on Air Pollution Prevention of VDI and DIN of the actual data on
exposure and health
effects (excluding cancer) of fine particulate air pollution is
presented. EXPOSURE: Typical ambient particle concentrations for
PM10 (PM2.5) in
Germany are in the range of 10–45 (10–30) microg/m3 as annual
mean and 50–200
(40–150) microg/m3 as maximum daily mean. The ratio of
PM2.5/PM10 generally
amounts between 0.7 and 0.9.
HEALTH EFFECTS: During the past 10 years many new
epidemiological and
toxicological studies on health effects of particulate matter
(PM) have been
published. In summary, long-term exposure against PM for years
or decades is
associated with elevated total, cardiovascular, and infant
mortality. With
respect to morbidity, respiratory symptoms, lung growth, and
function of the
immune system are affected. Short-term studies show consistent
associations of
exposure to daily concentrations of PM with mortality and
morbidity on the same
day or the subsequent days. Patients with asthma, COPD,
pneumonia, and other
respiratory diseases as well as patients with cardio-vascular
diseases and
diabetes are especially affected. The strongest associations
are found for PM2.5
followed by PM10, with no indication of a threshold value for
the health effects.
The data base for ultra fine particles is too small for final
conclusions. The
available toxicological data support the epidemiological
findings and give hints
as to the mechanisms of the effects.
CONCLUSION: The working group concludes that a further
reduction of the limit
values proposed for 2005 will substantially reduce health risks
due to
particulate air pollution. Because of the strong correlation of
PM10 with PM2.5
at most German sites there is no specific need for limit values of PM2.5 for Germany in addition to those of PM10.

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Cardiovascular implications of exposure to traffic air pollution during exercise.

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Regular aerobic exercise is recommended by physicians to improve health and longevity. However, individuals exercising in urban regions are often in contact with air pollution, which includes particles and gases associated with respiratory disease and cancer. We describe the recent evidence on the cardiovascular effects of air pollution, and the implications of exercising in polluted environments, with a view to informing clinicians and other health professionals. There is now strong evidence that fine and ultra fine particulate matter present in air pollution increases cardiovascular morbidity and mortality. The main mechanisms of disease appear to be related to an increase in the pathogenic processes associated with atherosclerosis. People exercising in environments pervaded by air contaminants are probably at
increased risk, due to an exercise-induced amplification in respiratory uptake, lung deposition and toxicity of inhaled pollutants. We make evidence-based recommendations for minimizing exposure to air-borne toxins while exercising, and suggest that this advice be passed on to patients where appropriate.

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Particulate air pollution and risk of ST-segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study.


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Comment in

BACKGROUND: Daily variations in ambient particulate air pollution have been associated with cardiovascular mortality and morbidity. We therefore assessed the associations between levels of the 3 main modes of urban aerosol distribution and the occurrence of ST-segment depressions during repeated exercise tests.
METHODS AND RESULTS: Repeated biweekly submaximal exercise tests were performed during 6 months among adult subjects with stable coronary heart disease in Helsinki, Finland. Seventy-two exercise-induced ST-segment depressions >0.1 mV occurred during 342 exercise tests among 45 subjects. Simultaneously, particle mass <2.5 microm (PM2.5) and the number concentrations of ultrafine particles (particle diameter 10 to 100 nm [NC(0.01-0.1)]) and accumulation mode particles (100 to 1000 nm [NC(0.1-1)]) were monitored at a central site. Levels of particulate air pollution 2 days before the clinic visit were significantly associated with increased risk of ST-segment depression during exercise test. The association was most consistent for measures of particles reflecting accumulation mode particles (odds ratio 3.29; 95% CI, 1.57 to 6.92 for NC(0.1-1) and 2.84; 95% CI, 1.42 to 5.66 for PM2.5), but ultrafine particles also had an effect (odds ratio 3.14; 95% CI, 1.56 to 6.32), which was independent of PM2.5. Also, gaseous pollutants NO2 and CO were associated with an increased risk for ST-segment depressions. No consistent association was observed for coarse particles. The associations tended to be stronger among subjects who did not use beta-blockers.

CONCLUSIONS: The present results suggest that the effect of particulate air pollution on cardiovascular morbidity is at least partly mediated through increased susceptibility to myocardial ischemia.

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Particulate air pollution and acute health effects.

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Epidemiological studies have consistently shown an association between particulate air pollution and not only exacerbations of illness in people with respiratory disease but also rises in the numbers of deaths from cardiovascular and respiratory disease among older people. Meta-analyses of these studies indicate that the associations are unlikely to be explained by any confounder, and suggest that they represent cause and effect. We propose that the explanation lies in the nature of the urban particulate cloud, which may contain up to 100 000 nanometer-sized particles per mL, in what may be a gravimetric concentration of only 100–200 micrograms/m3 of pollutant. We suggest that such ultra-fine particles are able to provoke alveolar inflammation, with release of mediators capable, in susceptible individuals, of causing exacerbations of lung disease and of increasing blood coagulability, thus also explaining the observed increases in cardiovascular deaths associated with urban pollution episodes. This hypothesis is testable both experimentally and epidemiologically.