Non-respiratory Health Hazards of Particulate Matter

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ABSTRACT

Particulate matter or PM consists of a heterogeneous mixture of very small particles and liquid droplets suspended in air. The size of particles in PM is directly linked to their potential to cause health problems. Particles with diameter ≤ 10 μm are the particles that generally pass through the throat and nose and enter the lungs. Then, they can affect different body organs, and may cause serious health effects. Usually respiratory harms of PM are reported, however the importance of the non-respiratory health hazards should be highlighted, as well. In addition to short-term effects, exposure to PM from early life might be associated with low birth weight, increase in oxidative stress and endothelial dysfunction, which in turn might have long-term effects on chronic non-communicable diseases.

Environmental protection actions, notably for reducing the emission of PM, should be considered for public health measures taken into account for primordial/primary prevention of chronic diseases, especially in developing countries. Some studies have documented the effect of PM on inflammatory and pro-thrombotic factors implicated in the progression of cardiovascular diseases. In particular, the increase of platelet count and platelet hyper-reactivity towards agonists are emerging as markers of hematologic and hemostatic changes in response to the exposure to air pollutants.

Considering the emerging epidemic of chronic disease in low- and middle-income countries, the vicious cycle of rapid urbanization and increasing levels of air pollution, public health and regulatory policies for air quality protection should be integrated into the main priorities of the primary health care system and into the educational curriculum of health professionals.
INTRODUCTION

Air pollution is a global health issue with serious public health implications. One of the six common and harmful air pollutants is particulate matter (PM), also known as particle pollution. It is a complex mixture of very small particles and liquid droplets. Particle pollution consists of a number of components, containing acids (as nitrates and sulfates), organic chemicals, metals, and dust or soil particles. They generally pass through the throat and nose and enter the lungs, and then they can cause serious health effects. The size of particles is directly linked to their potential for causing health problems. EPA is concerned about particles that are 10 micrometers in diameter or smaller because those are the particles that generally pass through the throat and nose and enter the lungs. Once inhaled, these particles can affect the heart and lungs and cause serious health effects.

While most studies have focused on respiratory effects of PM, here we provide a summary of study findings on their non-respiratory health effects and their possible implications in various age groups.

Methods

We used electronic databases for a search of the literature to find relevant studies:

1. PubMed
2. Ovid MEDLINE(R)
3. Ovid MEDLINE(R) in process and other non-indexed citations
4. Allied and Complementary Medicine (AMED)
5. Cumulative Index to Nursing and Allied Health Literature (CINAHL)
6. Scopus and EMBASE
7. CAB Abstracts
8. Global Health

The following search terms were used: health, particulate matter, particulates, air pollution, air pollutants, as well as specific types of PM exposures, e.g. silica, diesel
exhaust, styrene, asphalt fumes, metal or welding fumes, which contain particles in
the fine or ultrafine size range.

The literature search was restricted to articles published in the English language from
1990 to 2011.

In a secondary search, we used other terms related to health problems as abnormal
growth/development, birth weight, prematurity, intrauterine growth retardation,
congenital defects, development, behavioral problems, neurocognitive decrements,
malignancy, cancer, mortality etc.

Data on study design and location, confounding factors, health outcomes, and study
findings were extracted from the selected studies. We also used secondary references
cited by the articles recognized in the primary search.

RESULTS

The main findings of the studies reviewed are presented in Table 1.

DISCUSSION

Findings from this review highlight the diverse non-respiratory health hazards of PM
exposure in different periods of life. It underscores the impact of PM exposure to
chronic non-communicable diseases with intermediate outcomes on their risk factors
from early life.

PM exposure may have adverse pregnancy outcomes such as congenital anomalies,
increased risk of abortion, preterm delivery, intrauterine growth retardation, and still
birth. Some of these conditions, as intrauterine growth retardation may have lifelong
effects, notably on the incidence of chronic non-communicable diseases.

The review of individual morbidity studies suggested an association of increases in
atherosclerotic cardiovascular diseases with increasing PM exposure.

Moreover, this review presents evidence circulating markers of inflammation and
coagulation, a reasonable biological mechanism linking PM exposure with
atherosclerotic diseases. The harmful effects of PM on cardiovascular system are
well-documented, but the underlying mechanisms remain to be determined. It is
suggested that time that exposure to PM may enhance atherogenesis. The human
blood vessel endothelium is a sensitive target for PM. The interactions of the
inflammation and coagulation systems are suggested as of the main mechanisms
involved in impairment of endothelial function and eventually cardiovascular
diseases. The effect of air pollution on inflammation, oxidative stress and
cardiocascular risk factors are documented in various age groups even in children and
adolescents. The inflammation process stimulates the coagulation system, and result
in increased secretion of tissue factor. Endothelial function has key roles in
anticoagulant and fibrinolytic systems. In vitro studies have demonstrated significant
decrease in endogenous anticoagulation activity, thrombomodulin , endothelial
protein C receptor antigen and culture of endothelial cells during the inflammation
process. A growing body of evidence suggests that the effects of air pollution on the
inflammation and the coagulation systems may have a role in endothelial dysfunction
and in turn in the progression of cardiovascular diseases. Atherogenesis starts from
the fetal life through interrelations of traditional risk factors with inflammatory,
immune, and endothelial biomarkers. PM might have various harmful effects on this
process from early life.
The relationship of air pollution with hematological factors remains controversial.
Some studies documented the association of short-term and long-term exposure to PM
with WBC count, whereas some other studies did not confirm such association. It is
suggested that differences in the extent of the response to PM is influenced by the
variations in the susceptibility of different persons. Some studies proposed that PM
might have carcinogenic effects, and might cause DNA damage.
Of special concern are the health effects of PM exposure in the pediatric age group
with various short-term and long-term harmful health effects.
We acknowledge that this review has some limitations and potential biases, some of
which are common to reviews in general. For instance, publication bias, which is the
propensity for manuscripts with positive findings to be published more than those
with null findings, is a potential limitation of all reviews.
Despite these limitations, the collective evidence from a broad range of studies and cardiovascular outcomes suggest an association of PM exposure with most chronic non-communicable disease and their potential risk factors. Future longitudinal studies should be conducted. Considering the emerging epidemic of chronic disease in low- and middle-income countries, the vicious cycle of rapid urbanization and increasing levels of air pollution, public health and regulatory policies for air quality protection should be integrated into the main priorities of the primary health care system and into the educational curriculum of health professionals.

In general, this review gives an overview of the importance of PM exposure in adverse health outcome of various body organs.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Location</th>
<th>Population studied</th>
<th>Aims</th>
<th>Findings</th>
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<tr>
<td>Fann N et al, 2011 (1)</td>
<td>USA</td>
<td>Populations aged 65-99</td>
<td>use the photochemical Community Multiscale Air Quality (CMAQ) model in conjunction with ambient monitored data to create fused surfaces of summer season average 8-hour ozone and annual mean PM(2.5) levels at a 12 km grid resolution across the continental United States.</td>
<td>These results show that despite significant improvements in air quality in recent decades, recent levels of PM(2.5) and ozone still pose a nontrivial risk to public health.</td>
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<tr>
<td>Richardson EA et al, 2011(2)</td>
<td>New Zealand</td>
<td>Adult population</td>
<td>This New Zealand study investigated whether exposure to particulate air pollution, PM10, was associated with mortality and health inequalities.</td>
<td>A dose-response relationship for respiratory disease mortality, including at concentrations below the existing annual average guideline value of 20 μg/m3.</td>
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<td>González-Santiago O et al, 2011 (3)</td>
<td>Mexico</td>
<td>Aerodynamics</td>
<td>The aim of this study was to analyze the temporal behavior of PM10 (particulate matter &lt; or =10 microm in aerodynamic diameter).</td>
<td>PM10 levels were high and exceed the annual limit of 50 microg/m3 set up by the Mexican standard Norma Oficial Mexicana NOM-025-SSA1-1993. These levels could have serious health effects. The southwest zone of MMA had the highest levels of PM10 during the period studied. Winter was the most polluted season, and summer was the least polluted season. Thursday and Friday were the most polluted days, and Sunday was the least polluted day.</td>
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PM10 were 8:00 to 10:00 a.m. and nighttime hours were the cleanest.

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<tr>
<th>Study</th>
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<tr>
<td>Fujita EM et al, 2011</td>
<td>USA</td>
<td>Adult population</td>
<td>Researchers at the Desert Research Institute measured volatile organic compounds (VOCs), including several mobile-source air toxics (MSATs), particulate matter with a mass mean aerodynamic diameter &lt; or = 2.5 μm (PM2.5), black carbon (BC), nitrogen oxides (NOx), particulate matter (PM), and carbon monoxide (CO) on highways in Los Angeles County during summer and fall 2004, to characterize the diurnal and seasonal variations in measured concentrations related to volume and mix of traffic. The on-road concentrations of benzene, toluene, ethylbenzene, and xylenes (BTEX) during the summer were 3.5 +/- 0.7 and 1.2 +/- 0.6 times higher during morning and afternoon commuting periods, respectively, compared to annual average 24-hour concentrations measured at air toxic monitoring network sites. These ratios were higher during the fall, with smaller diurnal differences (4.8 +/- 0.7 and 3.9 +/- 0.6 for morning and afternoon commuting periods, respectively), the results of this study show that it is necessary to account for the proportion of diesel trucks to total vehicular traffic because of the disproportionate contribution of diesel exhaust to BC and to directly emitted PM. Alternatively, easily measured pollutants such as CO and BC can serve as reasonable surrogates for MSATs (e.g., BTEX and BD) and DPC, respectively.</td>
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<tr>
<td>Mauderly JL et al, 2011</td>
<td>USA</td>
<td>Experimental study</td>
<td>This was the fourth study by the National Environmental Respiratory Center to create a database for responses of animal models to combustion-derived pollutant mixtures, to identify causal pollutants-regardless of source. Only 17 of 270 species-gender-time-outcome comparisons were significantly affected by exposure; some models showed no effects. There was strong evidence that PM participated meaningfully in only three responses.</td>
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<tr>
<td>Knuckles TL et al, 2011</td>
<td>New Mexico, USA</td>
<td>Young rats were exposed to whole diesel emissions (DE) adjusted to 300 μg/m(3) of particulate matter (containing 3.5 ppm NO) or 0, 3, or 10 ppm NO as a positive control. Epidemiological studies have associated traffic-related airborne pollution with adverse cardiovascular outcomes. Nitric oxide (NO) is a common component of fresh diesel and gasoline engine emissions that rapidly transforms both in the atmosphere and once inhaled. pure NO exposures led to a concentration-dependent increase in plasma nitrates compared to controls, which lasted for roughly 4 h postexposure. Whole DE exposure for 1 h also led to a doubling of plasma NOx. NAC injection increased the levels of plasma nitrates and nitrites (NOx) in the DE exposure group. Inhibition of nitric oxide synthase (NOS) by N(G)-nitro-L-arginine (L-NNA) did not block the rise in plasma NOx, demonstrating that the increase was entirely due to exogenous sources.</td>
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| Garrett P et al, 2011 | Lisbon, Portugal. | Adult population | Urban ambient air pollution exposures continue to be a global public health concern. Although air quality targets are often exceeded in Lisbon, the largest For PM(2.5) exposures, we found a statistically significant cause-specific cardiovascular mortality in the range 2.39% (95% C.I. 1.29%, 3.50%) per 10 μg/m(3) increase. A statistically significant cause-specific cardiovascular mortality was not observed in..."
### Study Descriptions

<table>
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<th>Study Authors</th>
<th>Location</th>
<th>Participants/Study Setting</th>
<th>Findings</th>
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<tr>
<td>Zota AR et al., 2011(8)</td>
<td>USA</td>
<td>Young children living near a mining-impacted Superfund site.</td>
<td>In over half of the homes, dust Pb, Zn, Cd, and As were higher than those in soil piles and the presence of chat predicted dust metals levels. Pb, Zn, Cd, and As dust levels were higher in homes with no known chat sources. In contrast, Mn concentrations were lower than in soil and were not associated with chat sources. Mn dust concentrations were predicted by soil concentrations and occupant density. These findings suggest that nearby outdoor sources of metal contaminants from mine waste may migrate indoors. Populations farther away from the mining site may also be exposed if secondary uses of chat are in close proximity to the home.</td>
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<tr>
<td>Jia X et al., 2011 (9)</td>
<td>Beijing, China.</td>
<td>Healthy elderly subjects</td>
<td>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. 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 pooled 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.</td>
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<td>Ritz C et al., 2011(10)</td>
<td>Canada</td>
<td>Mice exposed in utero to diesel exhaust particles.</td>
<td>The induction of inherited DNA sequence mutations arising in the germline (i.e., sperm or egg) of mice exposed in utero to diesel exhaust particles (DEPs) via maternal inhalation compared to unexposed controls was investigated in this study. We found no evidence for increased mutation frequencies in females exposed in utero to DEPs. In contrast, a statistically significant increase in the mutation frequency of male mice exposed in utero to DEPs (2-fold; Fisher's exact p&lt;0.05). These results in increased mutation</td>
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### Key Findings
- Young children living near hazardous waste sites may be exposed to environmental contaminants, yet few studies have conducted multi-media exposure assessments, including residential environments where children spend most of their time.

### Quantiative Impact
- In a city in Portugal, there is currently no study that has assessed the quantitative impact of these pollutants on daily mortality.

### Mortality Impact
- Report O(3) exposures to be associated with an increase of 1.11% (95%C.I. (0.58, 1.64)) for all-cause mortality in the population group ≥65 years and an increase of 0.96% (95%C.I. (0.56, 1.35)) for the general population. While our results showed a stronger association for cardiovascular mortality.
<table>
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<tr>
<th>Study</th>
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<th>Exposure Type</th>
<th>Summary</th>
<th>Findings</th>
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<tr>
<td>Humbert S et al, 2011 (11)</td>
<td>USA</td>
<td>Workers</td>
<td>This paper summarizes the work of an international expert group on the integration of human exposure to PM into life cycle impact assessment (LCIA), within the UNEP/SETAC Life Cycle Initiative. Estimated average intake fractions are greater for primary PM(10-2.5) than for primary PM(2.5) (21 versus 15), owing in part to differences in average emission height (lower, and therefore closer to people, for PM(10-2.5) than PM(2.5)). For indoor emissions, typical intake fraction values are ∼1000-7000.</td>
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<tr>
<td>Peltier RE et al, 2011 (12)</td>
<td>USA</td>
<td>Roadway emissions</td>
<td>We describe spatial and temporal patterns of seven chemical elements commonly observed in fine particulate matter (PM) and thought to be linked to roadway emissions that were measured at residential locations in New York City (NYC). Despite the fact that these elements are only a fraction of total PM(2.5) or PM(10-2.5) mass, the results have important implications for near-roadway exposures where elements with known causal links to health effects are shown to be at elevated concentrations in both the PM(2.5) and PM(10-2.5) size ranges.</td>
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<td>Lee PC et al, 2011 (13)</td>
<td>USA</td>
<td>Pregnant women</td>
<td>It is not well understood how air pollution leads to adverse pregnancy outcomes. One pathway may be through C-reactive protein, a biomarker of systemic inflammation that has been reported to increase the risk of preterm delivery. We examined whether air pollution influences serum concentrations of C-reactive protein in early pregnancy. Among nonsmokers, an observed 9.2 μg/m increase in PM10 (averaged over 28 days prior to the blood sample) was associated with an odds ratio of 1.41 for high C-reactive protein concentrations (95% confidence interval = 0.99-2.00). Similarly, a 4.6 μg/m increase in PM2.5 was associated with an odds ratio of 1.47 (1.05-2.06). The odds ratio was 1.49 (0.75-2.96) per 7.9 ppb increase in ozone during summer. There were no associations in smokers or for other air pollutants, and there was no evidence for effect-measure modification by obesity.</td>
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<td>Wu S et al, 2011 (14)</td>
<td>Beijing, China</td>
<td>Healthy adults</td>
<td>Recently, we reported association of several HRV indices with marked changes in particulate air pollution around the Beijing 2008 Olympic Games in a panel of healthy adults. We further investigated the cardiac effects of traffic-related air pollutants over wide exposure ranges with expanded data set in this panel of healthy adults. Results showed overall negative associations between pollutants with HRV indices, with differences in period-specific effects identified by stratifying the individuals into two different response groups (positive/negative), cardiac effects were stronger within each group. Exposure-response modeling identified changed curvilinear relationships between air pollution exposures and HRV indices with threshold effects.</td>
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<td>Author(s)</td>
<td>Location</td>
<td>Population</td>
<td>Study Type</td>
<td>Findings</td>
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<td>Barman SC et al, 2011 (15)</td>
<td>India</td>
<td>Adult population</td>
<td>In this study, SPM, RSPM, SO2, NOx and 7 trace metals associated with RSPM were estimated at 10 representative locations in urban area and one village area for control. Beside this, air quality index (AQI), health effects of different metals and mortality were assessed.</td>
<td>The 24 hr mean concentration of metals associated with RSPM was found to be higher than the control by 52.3, 271.8, 408.9, 75.8, 62.7, 487.54 and 189.5% for Fe, Cu, Pb, Zn, Ni, Mn and Cr respectively. The inter correlation of metals Pb with Mn, Fe and Cr; Zn with Ni and Cr; Mn with Fe and Cu with Cr showed significant positive relation either at p &lt; 0.05 or p &lt; 0.01 level. Metals Pb, Mn and Cr (p &lt; 0.01) and Cu (p &lt; 0.05) showed significant positive correlation with RSPM. These results indicate that ambient air quality in the urban area is affected adversely due to emission and accumulation of SPM, RSPM, SO2, NOx and trace metals.</td>
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<td>Coscollà C et al, 2011 (16)</td>
<td>Spain</td>
<td>Adult population</td>
<td>A confirmatory and sensitive procedure has been developed for the determination of 40 currently used pesticides (CUPs) in airborne particulate matter (PM10) at trace level. The proposed method includes extraction of PM10-bound pesticides by microwave-assisted extraction (MAE) followed by gel permeation chromatography (GPC) clean-up and determination by GC-MS/MS.</td>
<td>Eighteen out of 40 pesticides investigated were found in at least one sample (bifenthrin, chlorothalonil, chlorpyriphos-m, chlorpyriphos-ethyl, fipronil, fludioxonil, folpet, quinoxyfen, triadimefon, trifluralin, and vinclozoline), with concentrations ranging from 1.32 to 625.80 pg m⁻³.</td>
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<td>Nemmar A et al, 2011 (17)</td>
<td>United Arab Emirates</td>
<td>Experimental study</td>
<td>This study was undertaken to determine the cardiovascular effect of diesel exhaust on TO mice made hypertensive by implanting osmotic minipump infusing angiotensin II or vehicle (control). On day 13, the animals were intratracheally instilled with either DEP (15μg/mouse) or saline.</td>
<td>The number of leukocytes and interleukin 6 concentration in plasma, however, were not affected in any of the animals. The PaO(2) was decreased, and PaCO(2) increased in DEP-treated HT mice compared to NT mice treated with DEP (P&lt;0.05). The number of circulating platelets was significantly increased in DEP-treated HT versus saline-treated HT and DEP-treated NT mice. Moreover, in NT mice, DEP exposure induced a prothrombotic effect in pial arterioles compared with saline-treated NT mice (P&lt;0.05). Interestingly, in DEP-treated HT mice, the prothrombotic events were significantly aggravated compared with saline-treated HT and DEP-treated NT mice. The direct addition of DEP (0.1-1μg/ml) to untreated mouse blood significantly induced in vitro platelet aggregation in a dose-dependent fashion, and these effects were more pronounced in blood of HT mice.</td>
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<tr>
<td>Nemmar A et al, 2011 (18)</td>
<td>UAE</td>
<td>Experimental study</td>
<td>Experimental approach. We investigated the acute (4 and 18h) effects of diesel exhaust particles (DEP) on cardiopulmonary parameters in mice, and the protective effect of thymoquinone, an ingredient of Nigella sativa. Mice were exposed to DEP or saline.</td>
<td>Four-h after DEP administration, there were no significant changes in the number of leukocytes, red blood cells, and plasma interleukin-6 concentration. However, at 18h post-exposure, both were significantly increased in DEP-treated mice compared with saline-treated mice. Interestingly, DEP exposure induced systemic inflammation characterized by high interleukin-6 (IL-6) concentration, and Superoxide dismutase (SOD) activity. Furthermore, DEP reduced platelets number and aggravated pial arteriole thrombosis. The addition of DEP (0.1-1μg/ml) to untreated mouse blood induced in vitro platelet aggregation in a dose-dependent manner, and these effects were more pronounced in blood of DEP-exposed mice.</td>
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<tr>
<td>Study</td>
<td>Location</td>
<td>Participants</td>
<td>Exposure</td>
<td>Health Effects</td>
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<tr>
<td>Peters A et al, 2001</td>
<td>USA</td>
<td>772 patients</td>
<td>Ambient particulate air pollution</td>
<td>Elevated concentrations of ambient particulate air pollution have been associated with increased hospital admissions for cardiovascular disease. Whether high concentrations of ambient particles can trigger the onset of acute myocardial infarction (MI), however, remains unknown.</td>
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<tr>
<td>Tonne C et al, 2007</td>
<td>USA</td>
<td>5,049 cases</td>
<td>Long-term exposure to particulate air pollution</td>
<td>Long-term exposure to particulate air pollution has been associated with an increased risk of dying from cardiopulmonary and ischemic heart disease, yet few studies have evaluated cardiovascular end points other than mortality. We investigated the relationship between long-term exposure to traffic and occurrence of acute myocardial infarction (AMI) in a case-control study.</td>
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<td>Pope CA 3rd et al, 2006</td>
<td>USA</td>
<td>12,865 patients</td>
<td>Ambient fine particulate pollution</td>
<td>Recent evidence suggests that long-term exposure to particulate air pollution contributes to pulmonary and systemic oxidative stress, inflammation, progression of atherosclerosis, and risk of ischemic heart disease and death. Short-term exposure may contribute to complications of atherosclerosis, such as plaque vulnerability, thrombosis, and acute ischemic events. These findings are inconclusive and controversial and require further study. This study evaluates the role of short-term exposure to particulate air pollution with a 4% increase in the odds of AMI per kilometer (95% CI, 3.6%).</td>
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Ambient fine particulate pollution (particles with an aerodynamic diameter ≤ 2.5 microm; PM2.5) elevated by 10 microg/m3 was associated with increased risk of acute ischemic coronary events (unstable angina and myocardial infarction) equal to 4.5% (95% confidence interval, 1.1 to 8.0). Effects were larger for those with angiographically demonstrated coronary artery disease.
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<th>Study</th>
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<th>Methodology</th>
<th>Findings</th>
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<td>Siddique et al., 2010 (22)</td>
<td>Delhi, India</td>
<td>Cross-sectional study of 969 school-going children (ages 9-17) and 850 age- and sex-matched children from rural areas were assessed.</td>
<td>The prevalence of attention-deficit hyperactivity disorder (ADHD) was assessed in two childhood populations.</td>
<td>There are positive associations between particulate exposure and mortality. Increase of PM(2.5) was not associated with stroke mortality. A 10 µg/m(3) increase in PM(10) was associated with a 1.09% increase in stroke daily attack and 0.70% increase in stroke daily mortality. As for PM(2.5), OR appeared to be 1.001 with a 10 µg/m(3) increase in stroke daily attack and 1.052 for daily mortality.</td>
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<td>Zhou et al., 2010 (23)</td>
<td>China</td>
<td>Meta-analysis method was used to pollysynthetically analyze 16 quantitative studies about the associations between particulate air pollution and stroke daily attack or mortality.</td>
<td>Subjects exposed to higher levels of traffic derived air pollution reported more ocular discomfort symptoms and presented greater tear film instability, suggesting that the ocular discomfort symptoms and tear breakup time could be used as convenient bioindicators of the adverse health effects of traffic derived air pollution exposure.</td>
<td>Recent advances include a better understanding related to aerosol dosimetry of the influence of susceptible populations on dose, including various diseases such as chronic obstructive pulmonary disease and bronchitis, as well as various human physiological characteristics (size, age, gender); UF PM, including transport of UF particles to the brain via the olfactory nerves and through the lung to other organs; hot spots in the lung resulting in localized high doses; and advancements in dosimetric modeling.</td>
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<td>Power et al., 2010 (24)</td>
<td>USA</td>
<td>In a Cohort of Older Men, 680 older men (mean ± age 71 ± 7 y) between 1996 and 2007.</td>
<td>To assess the association between black carbon, a marker of traffic-related air pollution, and cognition in older men.</td>
<td>Recent advances include a better understanding related to aerosol dosimetry of the influence of susceptible populations on dose, including various diseases such as chronic obstructive pulmonary disease and bronchitis, as well as various human physiological characteristics (size, age, gender); UF PM, including transport of UF particles to the brain via the olfactory nerves and through the lung to other organs; hot spots in the lung resulting in localized high doses; and advancements in dosimetric modeling.</td>
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<td>Novaes et al., 2010 (25)</td>
<td>São Paulo, Brazil</td>
<td>A panel study involving 55 volunteers was carried out in São Paulo, Brazil.</td>
<td>To explore the clinical relevance of chronic exposure to ambient levels of traffic derived air pollution on the ocular surface.</td>
<td>Results did not provide strong evidence of an association between exposure to PM in the prior 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.</td>
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<td>Phalen et al., 2010 (26)</td>
<td>USA</td>
<td>The doses delivered to subjects inhaling air-pollutant particles, the concept of a dose metric (also called an indicator) has emerged. An ideal dose metric has the following properties: it is measurable; it is expressible in physical and temporal scientific units; and it has a causal relationship to one or more biological responses.</td>
<td>Significant positive associations were found with doctor visits, with an excess risk (ER) of 4.13% for allergic disease and 6.58% (95% CI: 3.82% - 9.34%) for pollinosis per 100 grains/1000 mm(2) increase in pollen, in single-pollutant models. Results suggest that level of airborne pollen may have a stronger effect than ambient air pollutants on allergic disease.</td>
<td>The authors found a weak association between black smoke and congenital malformations of cardiac chambers, only when using exposure as a continuous variable. However, when the authors used quartiles of exposure, odds ratios did not show a dose-response relation for consecutive quartiles. For sulfur dioxide, the results were not indicative of any association.</td>
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<td>Puett et al., 2010 (27)</td>
<td>USA</td>
<td>Using two prospective cohorts, the Nurses’ Health Study (NHS) and the Health Professionals Follow-Up Study, investigated the relationship of incident type 2 DM with PM2.5, PM10, and PM10-2.5 exposures in the prior 12 months and distance to roadways.</td>
<td>The authors found a weak association between black smoke and congenital malformations of cardiac chambers, only when using exposure as a continuous variable. However, when the authors used quartiles of exposure, odds ratios did not show a dose-response relation for consecutive quartiles. For sulfur dioxide, the results were not indicative of any association.</td>
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<tr>
<td>Authors</td>
<td>Location</td>
<td>Data Collection and Analysis</td>
<td>Health Effects</td>
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<td>Zhuang et al., 2010</td>
<td>Beijing, China</td>
<td>The monitoring data of daily air pollution, along with the daily numbers of outpatients visits at the Allergy Department of Beijing Shijitan Hospital from April to September in 2004 were collected.</td>
<td>The systemic pro-inflammatory and pro-thrombotic response to the inhalation of fine and ultrafine particulate matters is seemingly associated with platelet activation. This association may have a clinical significance, particularly in the presence of cardiometabolic risk factors, and may indicate the need for anti-platelet treatment.</td>
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<td>Dadvand et al., 2010</td>
<td>England</td>
<td>used registry-based data on congenital heart disease for the population of the northeast of England in 1985-1996.</td>
<td>Investigate the association between maternal exposure to ambient air pollution and congenital heart disease.</td>
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<td>Poursafa, 2010</td>
<td>Iran</td>
<td>Review</td>
<td>The results indicate a potential association between PM exposure and emergency department visits for suicide attempts as the result of depression.</td>
<td>Association of PM exposure with suicidal ideations.</td>
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<tr>
<td>Szyszkwicz et al., 2010</td>
<td>Canada</td>
<td>Emergency visit data were collected in a hospital in Vancouver, Canada</td>
<td>The effects of ambient air pollution on emergency department visits for suicide attempts were investigated.</td>
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<td>Brunekreef et al., 2009</td>
<td>Netherlands</td>
<td>a randomly selected subcohort of 5000 older adults participating in the ongoing Netherlands Cohort Study (NLCS) on diet and cancer.</td>
<td>Increased systemic inflammation and vascular oxidative stress.</td>
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<tr>
<td>Carmichael et al., 2009</td>
<td>Review</td>
<td>Asia calculated over a 4-year period</td>
<td>Aerosol distributions in Asia calculated over a 4-year period and constrained by satellite observations of aerosol optical depth (AOD) are presented.</td>
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<td>Nurkiewicz et al., 2008</td>
<td>Australia</td>
<td>closer examination by toxicologists of vascular responses following PM exposure</td>
<td>Each 10 mg/m3 elevation in ambient Black carbon (BC) concentrations (10% of the total AOD, and contributing significantly to atmospheric warming (its warming potential is approximately 55% of that due to CO2)) associated with approximately a 1% increase in the daily total number of deaths.</td>
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</tbody>
</table>

**Note:** The table provides a summary of studies and their findings related to particulate matter and its effects on health.
<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Population</th>
<th>Objective</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simpson R et al., 2005 (35)</td>
<td>Austria</td>
<td>Brisbane, Melbourne, Perth and Sydney population</td>
<td>Investigating the health effects of air pollution on daily mortality</td>
<td>Respiratory morbidity has been reported.</td>
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<tr>
<td>Pope et al., 2002 (36)</td>
<td>USA</td>
<td>The risk factor data for approximately 500,000 adults were linked with air pollution data for metropolitan areas</td>
<td>To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality</td>
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<tr>
<td>Kelishadi et al.; 2009 (37)</td>
<td>Iran</td>
<td>Population-based sample of children and adolescents</td>
<td>To study the association of PM exposure with stress oxidative and insulin resistance</td>
<td>Independent association of PM with stress oxidative and insulin resistance</td>
</tr>
<tr>
<td>Poursafa et al.; 2010 (38)</td>
<td>Iran</td>
<td>Population-based sample of children and adolescents</td>
<td>To study the association of PM exposure with endothelial dysfunction</td>
<td>Independent association of PM exposure with endothelial dysfunction</td>
</tr>
<tr>
<td>Poursafa et al.; 2011 (39)</td>
<td>Iran</td>
<td>Population-based study in the pediatric age group</td>
<td>To study the association of genetic factors with the effects of PM exposure with endothelial dysfunction</td>
<td>Association of PM exposure with markers of endothelial dysfunction, independent of genetic factors</td>
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<tr>
<td>Poursafa et al.; 2011 (40)</td>
<td>Iran</td>
<td>Population-based study in the pediatric age group</td>
<td>To study the association of the effects of PM exposure with hematologic factors</td>
<td>Independent association of PM exposure with increased white blood cell count, decreased red blood cell count, and mean corpuscular volume.</td>
</tr>
</tbody>
</table>
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2. Richardson EA, Pearce J, Kingham S. Is particulate air pollution associated with health and health inequalities in New Zealand? Health Place. 2011 May 20. [Epub ahead of print]


