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AIRBORNE PARTICULATE MATTER

INTRODUCTION

Airborne particles have long been known to be a health hazard, ever since major cities became filled with soot in the early days of the industrial revolution. Historical pollution episodes like those in London in 1952 and Donora, Pennsylvania in 1948 raised awareness of the potential health threat of pollution. Approximately 4000 people died during the two-week London episode, and the deaths have been attributed to a combination of particles, SO₂ and acid aerosols. More recently, scientists have

turned their attention to smaller particles as a potential health threat, including "fine" and "ultrafine" particles (PM_{2.5} and PM_{0.1}, respectively). One concern with particle sizes is the ability of the particles to enter the respiratory tract and penetrate deeply into the lungs. Larger particles are more likely to be trapped in the nasal passages or the trachea, but smaller particles can be carried into the lung and enter the alveoli, or air exchange sacs.

On July 18, 1997, the U.S. Environmental Protection Agency established new air quality standards for particulate matter, adding new standards for PM_{2.5} while retaining PM₁₀ standards. Environment Canada

is also considering revisions to the air quality guidelines for particulate matter, and it is expected that proposed guidelines for PM₁₀ and PM_{2.5} will be published in Fall 1997.

PARTICULATE MATTER TERMS

There are a number of terms for particulate matter that are all based on the sampling method used. An historical PM measure that is still in use is **Total Suspended Particles (TSP)**, in which airborne particles are captured on a filter and weighed; particles ranging in size from 0 to about 40 microns are included.

In the U.S. and Canada, PM standards or guidelines are now based on **PM₁₀** or **PM_{2.5}**, where the "cut-off" diameter of the captured particles is 10 or 2.5 microns, respectively. **Coarse Particles (CP or PM_{10-2.5})** is determined by subtracting the fine particle concentration from PM₁₀. The particles are still collected on a filter and weighed, but the collection devices first exclude particles of larger sizes. TSP, PM₁₀ and PM_{2.5} are all measured as mass per volume of air, usually ug/m³.

Two historical PM measures that are still commonly used are **Coefficient of Haze (COH)** and **Black Smoke or British Smoke (BS)**. Both are measured by collecting particles on a filter or tape, and determining the quantity of particles by reflectiveness or opacity; BS is a measure of the "blackness" of the particle stain on the filter paper. These more indirect measures can be converted to ug/m³ if the monitors can be calibrated to another nearby collection device that measures PM by mass (such as PM₁₀).

Note: All particle sizes are described in terms of "aero-dynamic diameter" or the functional diameter equivalent to that of a spherical particle which settles at the same rate as the particles considered.

COMMUNITY HEALTH STUDIES

The results of numerous epidemiological studies indicate that an increase in PM concentration is associated with increased mortality, increased hospitalization for respiratory or cardiovascular diseases, increases in respiratory symptoms and decreased lung function.¹ In one review article, a 10 ug/m³ increase in PM₁₀ concentration was found to be associated with a 1% increase in daily mortality, and a 3% increase in asthma attacks, bronchodilator use and lower respiratory symptoms.²

Mortality: There are numerous studies that link exposure to PM with increased mortality. Two recent publications from the U.S. show associations with both long-term and short-term exposures. Pope et al.,³ using data from surveys of American Cancer Society volunteers from 151 U.S. metropolitan areas, found a relative risk ratio of 1.17 (1.09 to 1.26) for mortality from all causes with long-term exposure to PM_{2.5}. The availability of questionnaire data allowed the authors to adjust for factors such as cigarette smoking and occupational exposures. In the short-term study, Schwartz et al.⁴ found that a 10 ug/m³ increase in PM_{2.5} (from two days previously) was associated with a 1.5% (1.1% to 1.9%) increase in total mortality in six U.S. cities. For both studies, higher risk levels were found when considering mortality from cardiopulmonary diseases.

Recently-published studies from other countries support these findings. Researchers in England⁴ found that a 10 ug/m³ increase in PM₁₀ was associated with a 1.1% increase in mortality for all causes (p=0.03), as well as with a 5% increase in death due to chronic obstructive pulmonary disease and a 1.7% increase in deaths from circulatory diseases. In Mexico City, an increase in total mortality was associated with a 100 ug/m³ increase in TSP (rate ratio 1.058, 95% CI 1.033-1.083).⁵

While many previous studies have found associations between PM exposure and mortality in adults, a recent report suggests that increased mortality among infants is associated with increased exposure to PM.⁶ Woodruff et al. obtained data on almost 4 million infants from 86 U.S. metropolitan areas, and the infants' PM₁₀ exposures during the first two months of life were classified as high, low or medium using ambient monitoring data (PM₁₀ range 11.9-68.8 ug/m³). In normal birth weight infants, high PM₁₀ exposure was associated with death from respiratory causes (OR 1.40, 95% CI 1.05, 1.85) and sudden infant death syndrome (OR 1.26, 95% CI 1.14, 1.39). An increased in death from respiratory causes was also found in low birth weight infants, though not statistically significant.

Hospital Admissions: Many, but not all, studies have found associations between PM exposure and hospitalization. In England,⁵ a 10 ug/m³ increase in PM₁₀ was associated with 2 to 6% increases in hospital admissions for asthma, bronchitis, pneumonia, all respiratory causes and cerebrovascular diseases. A recent study in Tucson, Arizona⁸ found an increase in admissions for cardiovascular disease (2.75%, 95% CI 0.52-5.04) for a change from 28 to 51 ug/m³ PM₁₀. A similar association was found with CO concentration, and the PM₁₀ and CO effects were found to be independent and additive. In the winter months in Santa Clara County, California,⁹ where wood stoves are commonly used as a heat source, significant increases in emergency room visits for asthma were found with a 60 ug/m³ increase in PM₁₀ when temperatures were below, but not when above, 41 degrees. Relative risks in different temperature categories ranged from 1.11 (95% CI 1.03-1.19) to 1.43 (95% CI 1.18-1.69).

However, in a reanalysis of data from an earlier study, Moolgavkar and colleagues¹⁰ found no associations between admissions for respiratory disease with pollutants in Birmingham, Alabama, and, in Minneapolis-St. Paul, only a small increase in admissions (3.4% increase) with PM₁₀ was found and that association was not statistically significant when other pollutants were considered. Mixed findings have also been reported in Canadian studies, though it should be noted that both used only data from the summer months, while PM levels can be higher in the winter. In Montreal,¹¹ ozone was found to be the strongest predictor of emergency room visits for respiratory illness but positive associations were also found with particles, with significant increases of 16%, 12% and 6% predicted for mean increases in PM₁₀, PM_{2.5} and sulfate concentration, respectively. The authors conclude that relative mass

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COMMUNITY HEALTH STUDIES (continued)

effects are $PM_{2.5} > PM_{10} >>$ sulfates. In contrast, Burnett et al.¹² found associations with admissions for respiratory or cardiac causes and numerous PM measures (PM_{10} , $PM_{2.5}$, COH, CP, sulfates and acid aerosols), but statistical significance was generally lost when additional pollutants (especially ozone and nitrogen dioxide) were considered in the models.

Respiratory Symptoms or Pulmonary Function Changes: Researchers in California¹³ found a significant increase in inhaler use with a 10 ug/m^3 increase in PM_{10} ($p < 0.02$) in a panel of asthmatic subjects (9 to 46 years of age); no significant effect on peak flow rate was found with PM exposure. Lower respiratory illness in children was found to increase with both a 20 ug/m^3 increase in PM_{10} (1.08, 95% CI 1.04-1.15) and a 10 ug/m^3 increase in $PM_{2.5}$ (1.21, 95% CI 1.08-1.35) in Mexico City.¹⁴ In Amsterdam, decreases in morning and evening peak flow rate (4% and 3%, respectively) and significant increases in 3 of 4 symptom categories were found over the range of 4 to 40 ug/m^3 BS; smaller effects were seen for ozone and PM_{10} .¹⁵ A recent report from the large Swiss study (nearly 10,000 adult subjects) indicates that a 3.4% decrease in FVC (a lung function measure) is associated with a 10 ug/m^3 increase in PM_{10} .¹⁶ In contrast, Thurston¹⁷ found ozone to be more strongly associated with pulmonary function changes than measures of acid aerosols and sulfates in a Connecticut asthma summer camp.

Other health effects: In a comprehensive research project currently underway in the Teplice region of the Czech Republic,¹⁸ the effects of air pollution on numerous health endpoints is being studied. Preliminary findings indicate that recent exposures to both SO_2 and TSP are associated with decreases in sperm viability, though not total sperm count. In Beijing,¹⁹ a study of approximately 75,000 births (mother's first child) indicates an association between low birth weight and both SO_2 and TSP exposure during the third trimester; the authors report that the proportion of low birth weight attributable to air pollution is 13%. The increased Odds Ratios for low birth weight ($< 2500 \text{ g}$) for a 100 ug/m^3 change in pollutant were 1.11 (95% CI 1.06-1.16) for SO_2 and 1.10 (95% CI 1.05-1.14) for TSP.

AIRBORNE PARTICLES IN THE U.S. AND CANADA

The PM standards recently established by the U.S. EPA are 15 ug/m^3 for the annual mean and 65 ug/m^3 for a daily mean concentration of $PM_{2.5}$. For PM_{10} , the levels of 50 ug/m^3 (annual mean) and 150 ug/m^3 (24-hour) remain in place. (For each standard there are different averaging times or other parameters used in determining compliance.)

Scientists from Environment Canada and Health Canada²⁰ recently published a report on PM measurements from 19 Canadian locations. For 14 urban areas, the average TSP level was 55.2 ug/m^3 , with a maximal concentration of 572 ug/m^3 . Average daily concentrations of PM_{10} and $PM_{2.5}$ were 27.6 ug/m^3 (maximum 175 ug/m^3) and 13.9 ug/m^3 (maximum 89 ug/m^3), respectively. Approximately 44% of TSP was found to be PM_{10} , and $PM_{2.5}$ comprises about 49% of PM_{10} . Levels of $PM_{2.5}$ increased from summer to winter, presumably due to increases from combustion sources in the heating season. PM_{10} was slightly increased during the winter months; CP levels decreased somewhat in winter.

Using monitoring data from 42 non-urban sites in the U.S.,²¹ $PM_{2.5}$ concentrations were found to increase from west to east, with annual mean concentrations of 4 and 12 ug/m^3 , respectively, while PM_{10} annual mean concentrations ranged from 18 ug/m^3 in southeastern states to 8 ug/m^3 in central western states. The ratio of $PM_{2.5}$ to PM_{10} ranged from 45% in the western states to 65% in eastern U.S. states.

BIOLOGICAL MECHANISMS FOR PM EFFECTS

Numerous toxicological studies and several clinical studies have been published recently that offer insight into the biological means by which PM exposure would result in adverse respiratory or cardiovascular effects.

Increased inflammatory response with PM exposure is indicated in several recent studies. U.S. EPA researchers²² have found evidence for effects mediated by the transition metal

(iron, nickel, vanadium, etc.) content of particles. Effects seen included recruitment of immune cells (neutrophils, eosinophils, monocytes) and inflammation of the lung tissue. German researchers²³ have found both the water-soluble (i.e. metals) and solvent-soluble (i.e. polynuclear aromatic hydrocarbons) components of particles can exacerbate inflammatory reactions, though the effects were somewhat different. The aqueous component of particles was associated with increased immune chemical levels (PGE_2 and IL-8), while increased production of reactive oxygen species was found with the organic PM component. Studies have also suggested that PM exposures may exacerbate allergic responses, and a recent study reports that grass pollen allergen binds to diesel exhaust particles, which could allow allergens to become concentrated in polluted air and thus trigger attacks of allergic asthma.²⁴

It was hypothesized that inflammatory reactions may result in changes in blood chemistry that result in the cardiac effects found in epidemiological studies of PM exposure. Recently, German researchers²⁵ reported significant increases in plasma viscosity in men during an air pollution episode during Winter 1984-5. Increases were found to be associated with three measured pollutants, TSP, SO_2 and CO, but the associations were only statistically significant for SO_2 and CO among women.



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