HEALTH & CLEAN AIR
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The Particulars of Air Quality

The fog was thicker on that Friday morning than many people could ever remember. Through the day it steadily grew even thicker (and) people were already experiencing discomfort, and noticing the choking smell in the air....On (Saturday and) Sunday the fog continued and so did the deaths. The emergency services were no longer able to respond in any effective way. It is doubtful that many people perceived the nature of the calamity that had befallen them.

PETER BRIMBLECOMBE,
The Big Smoke

One-half century after the coal-induced "Great Fog" of 1952 claimed the lives of roughly 12,000 Londoners, smoke, soot, and dust remain lethal to young and old alike, but with a critical difference: then, the pollution contained particles large or dark enough to be seen as soot and smoke; today, these larger particles have been largely eliminated, so what remains is seen only cumulatively as a haze that, especially in the summer and in eastern North America, can seem natural, even benign. Yet the haze is neither natural nor benign. It consists mostly of exceedingly fine particles from the burning of coal, oil, and gas: they account for tens of thousands deaths a year in the United States alone.

Tiny enough for between 40 and 1,000 of them to fit on the width of a human hair, these particles cause death and illness over both the short and long term. On a daily basis, as their concentration rises, so too do deaths and illnesses, ranging from runny noses to emphysema. Over the longer term, particles are also associated with grave illness and injury. Indeed, researchers very recently concluded that the risk of dying from lung cancer from breathing these particles was the equivalent to that of living or working with a smoker.

Fine particles billow by the millions of tons each year from gasoline, diesel and jet engines, coal fired power plants, steel mills, and hundreds of other types of smokestacks and tail pipes, literally clouding virtually the entire continent. Visibility measurements from airport and other sites reflect concentrations of fine particles. On maps in which dense haze is shown in deepening shades of orange, that color has spread from a small, roughly circular area covering northern Ohio and bordering areas of Pennsylvania and Michigan in 1960 to a blanket over virtually every square mile east of the Mississippi River in 1990. In a few locations—Southern California is the most notable—fine particle levels have fallen. Visibility has also improved some-

what in some areas of the eastern United States recently, principally because of acid rain controls, but are still vastly worse than in the 1960s.

These particles find their way into even the youngest lungs. In Leicester, England, for example, physicians analyzed macrophages, which are specialized blood cells found in the lung, from children undergoing elective surgery. Ultrafine carbon particles—the sort that typify engine exhaust—were present in the macrophages of all of the children, even a 3-month-old infant. The levels in children who lived close to busy roads were roughly three times those of children living by quiet roads. When a similar examination was made of 14 non-smoking Canadian workers, 11 utility employees and 3 non-maintenance employees of a university, all contained ultrafine particles. "The demonstration of ultrafine particles in all 14 subjects independent of occupational exposure, suggests that there is environmental exposure to ultrafine particles," commented the researchers.

Pollution control programs initially focused on total suspended particulate matter (TSP), which is measured by weight. Then, regulations focused on smaller but still relatively large and heavy particles. These efforts have reduced the total mass of particles, but not necessarily the number. These remaining small particles easily penetrate building shells and pass through conventional heating and air conditioning filters, and when inhaled can reach the deepest recesses of the human lung. There, in ways that are still being unraveled, they inflict injuries that, in some cases, can ultimately lead to death.

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The numbers of particles have been rising for at least a half century, and there seems to be no end in sight. Consider, for example, that in 1950 there were no sales of jet fuel because there were no commercial jet aircraft. Today, however, the U.S. Energy Information Administration estimates that 1.7 million barrels of jet fuel are sold each day nationally, and when burned, each pound results in 100,000,000,000,000,000 particles. Similarly, motor vehicles, especially diesels, emit immense amounts of soot—classified by the California Air Resources Board as a toxic air contaminant. Since 1960, the miles traveled nationally by 18-wheel, on-the-road trucks has jumped 459 percent.

Particles are a product of uncontrolled burning, and the struggle to eliminate, or at least reduce, such pollution dates to at least 1306, when smoke became so intolerable in London that the King banned the burning of coal. This was a no-nonsense prohibition, for the penalty for violation was death. Today, nearly seven centuries later, the world's governments are beginning to focus their efforts squarely on this deadly component of smoke. This issue of the *Newsletter* explores the sources of particulate matter, its contribution to human death and illness, and the policies and measures available to minimize this threat.

What is a Particle?

"Particle" is a catch-all word. Defining it is not unlike trying to describe the contents of a Halloween goody bag after a night of trick-or-treating. The term encompasses a wide variety of solids and liquids, including diesel soot, fly ash, ocean spray, dust and soils, dozens of metals ranging from arsenic to zinc, unburned and partially burned gasoline, tiny pieces of tires and brake pads, and scores of chemicals—alkanes, alkenes, aromatics, cyclic olefins, terpenes, to name a few of the more common ones—as well as numerous biological materials such as mold and disease-causing spores.

Defining particles is complicated even further by the fact that many of them are not emitted directly from a tailpipe or smoke stack, but instead are created by atmospheric chemical reactions. For example, when sulfur-containing coal, gasoline or diesel fuels are burned, a colorless gas, sulfur dioxide, is formed. It ages to form liquid fine particles that are droplets of sulfuric acid (a constituent of acid rain), which in turn, form extremely fine, solid particle sulfates. Much the same happens to oxides of nitrogen: all these, in turn, react with the thousands of organic chemicals, yielding an atmospheric soup that simply cannot be completely described. "No single analytical technique is currently capable of analyzing the entire range of organic compounds present in the atmosphere as particulate matter," explained the U.S. Environmental Protection Agency in a 2001 draft document.

In part because of the difficulty of defining particles according to character-

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istics such as their chemical identities, they are instead classified according to size. Part of the rationale for this is that whether a particle is an acid or metal, granite or gasoline, size determines how it will behave in the atmosphere and how far it will travel into the lung. Large particles—dust blown from fields or eroded from rocks, for example—travel shorter distances and are generally captured in the upper airways. Extremely small particles, emitted by the trillions times trillions from diesels, jet aircraft, powerplants, and others sources, can travel thousands of miles remaining suspended in air for weeks or months. When inhaled, they can penetrate to the very deepest parts of the lung.

As a general matter—and like most generalizations, this one has exceptions—particles are described as coarse, fine, and ultrafine. Coarse particles are larger than 2.5 microns, or two and one-half one-millionths of a meter. Much of this larger fraction consists of soil, street dust, pollen, and mold, though it can include toxic metals and biologically hazardous materials. Fine particles are 2.5 microns and smaller, and components include a wide variety of organic chemicals, compounds of lead, cadmium, vanadium and other metals, as well as pure carbon from diesels and power plants, often coated with chemicals that can cause cancer and other serious illnesses. Ultrafine particles, with a size of one-tenth of a micron or smaller, also include metals, organic compounds, and carbon spheres. By way of comparison, a human hair is about 100 microns, so roughly 40 fine particles could be placed on its width, or 1,000 or more ultrafine particles. For ease of reference, the shorthand method of referring to particulate matter (PM) by size is, for example, $PM_{2.5}$ or PM_{10}.

The anthrax terrorist attacks of 2001 graphically illustrate how such exceedingly small size can facilitate toxicity. Anthrax spores are between 2 and 6 microns in diameter, or somewhat larger than many fine particles of air pollution. Because of their extremely small size, the anthrax spores escaped from sealed envelopes and, after entering the air, quickly dispersed throughout entire buildings, clinging to walls, floors, machinery, furniture, and other surfaces. When inhaled, the spores were too small to be captured in the upper airways, so they lodged in the lung's deepest reaches, where they were engulfed by macrophages, then transported to lymph centers to be destroyed. Yet the spores survive this process and so, too, do air pollutants. Indeed, the anthrax spores germinate in the lymph nodes to form toxin-producing bacteria that kill about half their victims in the absence of intervention and antibiotics. Fine particles also survive the removal process, but exactly how is unclear. There can be no doubt of this, because they have been counted by electron microscope in lung tissues of cadavers. In residents of Mexico City, scientists found roughly two billion particles in every gram of dry lung tissue. Levels in the lung of residents of Vancouver, British Columbia—a much less polluted city—were lower, but still remarkable: about 280 million per gram of dry lung tissue.

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Though some of the pollution particles remain in the lung, others remain in the lymph nodes, while still others enter the blood stream. What happens next is something of a mystery that scientists are anxious to solve.

One possibility is that particles somehow change levels of ingredients of the blood, which was found in a study in 112 persons over the age of 60 in Edinburgh and Belfast. In a study of 47 Finnish patients with stable heart disease, exposure to fine and ultrafine particles was consistently associated with an electro-cardiogram abnormality often found in those with heart injury. And, in Chapel Hill, North Carolina when elderly volunteers were exposed to concentrated Chapel Hill particles for two hours, their heart rate variability dropped significantly, which is often a predictor of heart illness and death. Another study, this of people exposed to the Asian wildfires of 1998, suggests that particles somehow trigger an inflammatory reaction that, in turn, leads to a cascade of other adverse events. One of the most provocative and useful studies found that PM_{10} exposure resulted in an increase in C-reactive protein, an index of inflammation that is associated with increased rates of coronary artery disease. One painstaking study at a time, the mechanism by which particles cause illness and death is being revealed. Notwithstanding these uncertainties, it is nevertheless clear that small particles—especially those from burning coal, oil, and other materials, but also some of those in the coarse fraction—trigger illness and death.

Effects of Fine Particles

Mortality. The evidence that particulate kills is compelling. Studies have shown that increases in daily particle levels are followed by increases in daily deaths in Amsterdam, Athens, Barcelona, Basel, Berlin, Birmingham, Boston, Chicago, Cincinnati, Detroit, Dublin, Erfurt, Eastern Tennessee, London, Los Angeles, Lyon, Madison, Milan, Minneapolis, Mexico City, New York, Philadelphia, Provo, Rotterdam, Santiago, Santa Clara,

Steubenville, St. Louis, Sao Paolo, Topeka, Valencia, and Zurich.

The largest of these studies tracked the health histories of 552,138 adults in 151 metropolitan areas from 1982 through 1989 and accounted for smoking, obesity, age, alcohol use, and other potential confounding factors. In some studies, motor vehicle exhaust seems to play a critical role, but adverse health effects have also been linked to steel mills, peat-fired power plants, and a variety of other sources of particles. The association between mortality and fine particulate matter is, in the words of Dr. Douglas Dockery of the Harvard University School of Public Health "consistent (and) robust."

The effects are associated with chronic and acute exposures alike. In a study of 772 patients in Boston who had suffered heart attacks, for example, researchers found that as concentrations of particles rose—both PM_{10} and $PM_{2.5}$—the frequency of heart attacks did the same a few hours to one day later. Longer term exposures—years or even decades—are equally dangerous: when researchers analyzed data from more than 500,000 people in an average of 51 metropolitan districts in a study dating to 1982, they found that when PM_{10} concentrations increased by 10 micrograms per cubic meter, deaths from all causes rose 4 percent; from cardiopulmonary illness by 6 percent; and, from lung cancer by 8 percent.

A brief review of some of these studies provides some sense of the breadth and depth of research that makes it possible for sober and conservative observers to accept the seemingly counter-intuitive proposition that something that can barely be seen, and even then only as haze, could be killing more people than automobile accidents. Consider the following:

- Researchers from Johns Hopkins, Harvard and other universities examined data from 90 cities in different regions of the United States, covering all geographic areas. Daily levels of air pollution from 1987 to 1994 were compared to death and hospital records. The researchers found not only a link between exposure to particles and death, but "strong evidence of association between PM_{10} levels and exacerbation of chronic heart and lung disease sufficiently severe to warrant hospitalization."
- Responding to the rapidly accumulating body of evidence from the United States that air pollution was linked to mortality, the European Union founded the "Air Pollution and Health—A European Approach," or APHEA, study. Eleven teams of researchers from 10 different nations studied European cities with a total population of 25 million. As in North America, when particle levels rose, so did mortality and hospital admissions. When British Smoke, or BS, a measure of particle concentrations, rose by 50 micrograms per cubic meter, mortality increased by 2.2 percent.
- In addition to comparing air pollution levels with deaths, researchers collected particle samples from the Canadian cities of Montreal, Ottawa-Hull, Toronto, Windsor, Winnipeg, Edmonton, Calgary, and Vancouver for chemical analysis. Carbon was the dominant constituent of the total particulate matter, while sulfur, which is a contaminant of coal and diesel fuel, had the highest correlation with fine particles. As in other studies, increases in mortality were linked to higher levels of particles, but the association was strongest with $PM_{2.5}$.

[Some have argued that particle-caused deaths are merely "harvesting"—the acceleration by a few days of death in the elderly or frail that would have occurred in any event—but several studies have expressly addressed and discounted such claims.]

Two of the most largest and most persuasive of the studies linking fine particles to illness and death are the Harvard Six Cities Study and the American Cancer Society Study (ACS). Published in the mid-1990s, they were sharply criticized by polluting industries and a few scientists who challenged the existence of a causal connection between fine particles and death. These criticisms, in turn, prompted two other studies (a) a re-analysis of the Six Cities and ACS work and (b) the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), which was entirely new research on hospitalization and deaths associated with air pollution in major U.S. cities.

The re-analysis by independent investigators validated the original studies, confirming that they were sound science. In addition, NMMAPS found strong evidence linking daily increases in particle pollution in the twenty largest U.S. cities to not only increases in death, but increased hospital admissions for cardiovascular disease, pneumonia, and chronic obstructive pulmonary disease. These left no doubt that Americans were being placed at grave risk by particles. And, while the elderly and the ill may be at greatest risk from particles, the second largest group at risk are society's most defenseless: infants and children.

Infant and Child Mortality. The linkage between infant and child mortality and exposure to particles is the subject of considerably fewer studies than those of adults, but they are persuasive nonetheless. The results are consistent and they are reinforced by other studies of illness, suggesting a continuum of effects. In the Czech Republic, for example, researchers examined all births between 1989 and 1991. For each infant death, they random-

ly selected 20 other children of the same sex who had been born on the same day as the deceased, then examined 24-hour air pollution levels in the districts where each lived for the period between the birth and death. In all, they studied 2,494 infant deaths and, of these, the 133 due to respiratory causes were linked to increased levels of not only particles, but also sulfur dioxide and oxides of nitrogen. The researchers' conclusion was that "the effects of air pollution on infant mortality are specific for respiratory causes in [the period between one month and one year of age], are independent of socioeconomic factors, and are not mediated by birth weight or gestational age."

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Similarly, after scientists compared respiratory mortality in Sao Paulo, Brazil, of children under age five to daily levels of sulfur dioxide, carbon monoxide, ozone and PM_{10}, each was associated with an increased risk of death. As concentrations rose, so, too, did mortality and this was "quite evident after a short period of exposure (2 days)". The estimated proportions of respiratory deaths attributed to CO, SO_2, and PM_{10} when considered individually, were around 15 percent, 13 percent, and 7 percent, respectively.

A quite intriguing study was done by economists at the National Bureau of Economic Research in Washington, D.C., who examined the relationship between infant mortality rates and decreases in particulate matter emissions because of a recession. They found that as levels of particles fell, so did neonatal mortality (death before the age of 28 days). Focusing specifically on Pennsylvania, they found that when the levels of total particulate matter dropped roughly 25 percent, infant deaths within one year of birth attributable to "internal" causes (e.g. respiratory and cardiopulmonary deaths) fell by 14 percent.

Illness. In addition to their linkage to death, fine particles are associated with a litany of lesser ills, including runny or stuffy noses, sinusitis, sore throat, wet cough, head colds, hayfever, burning or red eyes, wheezing, dry cough, phlegm, shortness of breath, and chest discomfort or pain, as well as hospital admissions for asthma and bronchitis. Increases in fine particle levels are accompanied by higher rates of chronic cough, asthma, and emphysema, even among non-smoking Seventh-Day Adventists. Bronchitis and chronic cough increase in school children as do emergency room and hospital admissions. In Utah, when particulate levels rose, hospital admissions of children for respiratory illnesses tripled.

Sources of Fine Particles

Whatever quality makes soot particles dangerous, it is clear that size alone is not the explanation. When combustion-related particles were instilled in the lungs of guinea pigs pathologic changes followed within 24 hours. Yet no pathologic changes were observed following a similar instillation of "natural" dust from the Mount St. Helen's volcanic eruptions.

Similarly, in a study in Spokane, Washington, researchers found that mortality increased when PM_{10} levels from vehicles increased, but not when the particles were due to dust storms. In addition, when researchers examined mortality data in six eastern U.S. cities, they found that death increased in association with levels of fine particles from combustion, but not those of "crustal," or natural origin.

Policy Implications

The information in this *Newsletter* identifies and roughly quantifies an ill-health burden of major proportions. Whatever the exact number of deaths due to fine particles may be, it clearly rivals or exceeds those in 1998 from motor vehicle accidents (43,501), suicide (30,575,) breast cancer (42,086), or leukemia (20,234).

Particles cannot be avoided through the simple expedient of going indoors. Even if respirators could eliminate the smallest fine or ultrafine particles, people do not wear them nor should they be expected to. The only means of reducing exposure and, hence, death and illness that are avoidable, is effective governmental action. There are two levels of government that can provide such protection in the United States—federal and state. The record suggests that if public health is to be safeguarded, it is incumbent on states to act.

Federal Level. The federal law designed to protect the public from air pollution, the Clean Air Act, requires the U.S. Environmental Protection Agency to establish ambient standards that protect public health, and technology-based emission standards for power plants, refineries, cement kilns, and other large polluters. At present there is, at the least, disarray in both sets of these standards.

Ambient standards for $PM_{2.5}$ and PM_{10} that were first proposed in 1997 have already been delayed for five years by lawsuits filed by truck drivers and owners, as well as other polluting industries. Although virtually all their claims were rejected by the U.S. Supreme Court,

the rules must now be revised by an administration viewed by some as overtly hostile to environmental protection.

Moreover, even if the rules were put in place immediately, it is doubtful that they could fully comply with the mandate of the Clean Air Act that ambient standards protect the health of sensitive populations (e.g., the elderly and those with heart disease) with an adequate margin of safety. Clearly, at levels of fine particles allowed by the federal short-term standard (65 micrograms over a 24-hour period) studies find adverse health effects, including death. Indeed, the California Office of Environmental Health Hazard Assessment recently recommended to the state California Air Resources Board a 24-hour $PM_{2.5}$ standard of 25 micrograms per cubic meter, while the corresponding federal standard establishes a 24-hour limit of 65. One explanation for this discrepancy is that the U.S. standard was promulgated six years ago, a defense that ignores the explicit mandate of the Clean Air Act that standards be reviewed and revised, if necessary, every five years.

To help assure that ambient standards are achieved and to provide a bit of a safety net, the Clean Air Act also requires the development and imposition of technology-based standards for cars, trucks, buses, power plants, steel mills, refineries, and other sources of air pollution. Though the evidence that fine particles pose grave risks to the health of Americans began accumulating in the mid-1970s—approaching three decades ago—there are virtually no fine or ultrafine particle performance standards for motor vehicles, power plants, refineries steel mills, and other sources. Indeed, neither the U.S. Environmental Protection Agency nor the states systematically collect data on emissions of fine particles from such major sources such as commercial jet aircraft or non-diesel engines.

Still, it might be possible to reduce levels of particulate particles through controls on major sources of pollution. In November, 1999, the U.S. Environmental Protection Agency and the Department of Justice did just that, launching a national initiative that targeted electric utilities throughout the Midwest and Southeast whose coal-fired power plants were alleged to be violating the Clean Air Act. Most recently, however, that effort has been sidelined by the Bush Administration in favor of a proposal to make emission reductions voluntary, and delaying cuts until 2018. As part of the Bush initiative, the "new source review" provisions of the Clean Air Act that U.S. EPA and Justice had been seeking to enforce would be repealed.

The significance of this action is underscored by a study of nine power plants in Illinois. It concluded that reducing their emissions would annually save 190 lives, lessen emergency room visits by 2,532, eliminate 13,290 asthma attacks, and avoid 168,900 restricted activity days. The authors noted that "Pre-1980 coal-fired power plants currently contribute about half of the electricity generation in the US and are responsible for 97% of power plant SO_2, and 85% of power plant NO_2 emissions"

State Level. In the absence of effective federal action—indeed with evidence of hostility toward air pollution controls altogether—the information in this *Newsletter* suggests that state and local governments should identify sources of fine particles and target them for aggressive controls if the burden of human death and illness is to be lessened. In addition, those states that promulgate their own ambient standards have the opportunity to redress the shortcoming of the current federal standard. The state with the earliest opportunity to act in each of these areas is most likely California, which is in the midst of revising its ambient standard and conducting an ongoing review of the adequacy of the state implementation plan. ■

FOR EXPERT READERS

Why are Small Particles Dangerous?

Recent challenges from industry—carried all the way to the Supreme Court in the United States—asserted that no causal inference could be made without understanding the mechanism of action of the inhaled particles. This precept was denied by the judges in both of the courts that considered the matter. Nevertheless, knowledge of the mechanisms involved, and also of the components of the $PM_{2.5}$ which account for its toxicity, are both important considerations. The active pursuit of the study of mechanisms has constituted one of the more interesting sleuthing challenges in recent air pollution/health research. There have been two main ideas (which are not necessarily in conflict). The first is that changes in recorded heart

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rate variability, together with tachycardia, may signal an electrical cause for an acute and fatal heart attack. The second is that observed changes in the blood, particularly an increase in fibrinogen and an increase in the percentage of circulating banded neutrophils, may predispose to myocardial infarction. The first theory was extensively addressed in the draft EPA Criteria Document for Particulate Pollution, which devoted a special appendix to it. The second has recently received strong reinforcement from two papers, as follows:

1) FUJI, T., HAYASHI, S., HOGG, J.C., VINCENT, R., & VAN EEDEN, S.F. Particulate matter induces cytokine expression in human bronchial epithelial cells. Am J Respir Cell Mol. Biol 25; 265-271, 2001. Cytokine messenger RNA measured by ribonuclease protection assay and cytokine protein production by enzyme-linked immunosorbent assay. Primary human bronchial epithelial cells (HBEC) obtained from fresh lung specimens obtained after surgery, then cultured to confluence (to show that cells were viable) and then exposed to a suspension of ambient particulate matter (10 to 500 micrograms/ml) with a diameter less than 10 microns for 2, 8, and 24 hours. Particles were obtained from air filters collected in Ottawa by the Environmental Health Protectorate from that city's ambient air. Supernatant collected at 24 hours. It was found that concentrations of LIF (leukemia inhibitory factor), GM-CSF (granulocyte macrophage colony-stimulating factor), IL-1alpha (interleukin), and IL-8 were all increased in a dose dependent manner after exposure to PM_{10} compared to production of these cytokines in non exposed cultures. It was also shown that the soluble fraction of PM_{10} did not increase these levels, and hence was inactive. The authors conclude: "that primary HBECs exposed to ambient PM_{10} produce proinflammatory mediators that contribute to the local and systemic inflammatory response, and we speculate that these mediators may have a role in the pathogenesis of cardiopulmonary disease associated with particulate air pollution." Figure shows particles "internalized" by the epithelial cells.

2) VAN EEDEN, S.F., TAN, W.C., SUWA, T., MUKAE, H., TERASHIMA, T., FUJII, T., QUI, D., VINCENT, R., & HOGG, J.C. Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM_{10}), Am J Respir Crit Care Med 164; 826-830, 2001. Human alveolar macrophages (AM) harvested from bronchial lavage specimens (BAL) from a noninvolved segment or lobe of lungs resected for small peripheral tumours. These were more than 90% viable. All specimens tested for endotoxin contamination. Urban PM_{10} preparation (EHC 93) came from filters from Ottawa. Identified cytokines also measured in the blood of young army cadets exposed to Asian smoke in April 1998 in Singapore (see {10607}). AM Cells were incubated with residual oil fly ash (ROFA), ambient urban particles (EHC 93) inert carbon particles, and latex particles of different sizes (0.1, 1.0, and 10 microns) for 24 hours. The latex, inert carbon and ROFA particles all showed a similar maximum TNF (tumour necrosis factor alpha) response, whereas EHC 93 showed a greater maximum response that was similar to lipopolysaccharide (LPS). EHC 93 (Ottawa PM_{10}) also resulted in a broad spectrum of proinflammatory cytokines, (IL-6, MIP-1alpha, and GM-CSF), with no difference in the anti-inflammatory cytokine IL-10. Analysis of blood samples taken during the exposure of the army cadets in Singapore to the PM_{10} from the Asian fires of 1998, showed elevated levels of IL-1beta, IL-6, and GM-CSF during the exposure time. Authors conclude: "These results show that a range of different particles stimulate AM (human alveolar macrophages) to produce proinflammatory cytokines and these cytokines are also present in the blood of subjects during an episode of acute atmospheric air pollution. We postulate that these cytokines induced a systemic response that has an important role in the pathogenesis of the cardiopulmonary adverse health effects associated with atmospheric pollution." These papers do not signal an end to the debate on the relative importance of different factors in the intermediary steps between small particle inhalation and the adverse health outcomes documented in epidemiological studies—but perhaps they might be considered as indicating "an end to the beginning." ■

Worth Noting

The Air In There

Assessments of threats to the health of those who live in developing nations tend to focus on those that are highly visible, such as malnutrition and communicable disease. Indeed, it seems logical that many more would die from such threats than from air pollution—but perhaps that's not necessarily so, according to two recent studies.

When researchers examined causes of death and recorded indoor levels of air pollution—principally from burning wood, dried dung and other forms of biomass—in Papua, New Guinea, Kenya, India, Nepal, China, and Gambia, they concluded that there were 4.1 million deaths annually from acute respiratory illnesses in children under five in developing countries. This compares to 3.0 million from intestinal disease, and 0.68 million from malaria.

Another study examined the use of biomass cooking fuels and the prevalence of tuberculosis in India, examining data from 260,162 people aged 20 and over in a Family Health Survey conducted in 1992-3. The scientists concluded that the "results strongly suggest that use of biomass fuels for cooking substantially increases the risk of tuberculosis in India."

Missing School

Kids often seem to be magnets for illnesses, especially colds, sore throats, and the like. Not surprisingly, respiratory illnesses are cited as the leading cause of school absenteeism. However, part of the explanation for all that illness and missed school may be air pollution, not just youth.

When researchers at the University of Southern California compared school absences for respiratory illness to levels of air pollution, they found a remarkable fit: as ozone, or smog, levels rose, so did missed school due to sore throats, cough, asthma attacks and other respiratory illnesses. For an increase in concentration of 20 parts per billion, a common day-to-day variation in highly-polluted Southern California, absenteeism for respiratory causes jumped 83 percent.

That same 20 ppb increases was associated with sharp jumps in specific ailments. Upper respiratory illnesses rose 45 percent, while lower respiratory illnesses with wet cough jumped 174 percent.

A CITIZEN'S GUIDE TO AIR POLLUTION

Written and edited by the Newsletter's co-editor, Dr. David Bates, and others, this comprehensive, authoritative and insightful book will soon be available for a modest price from the David Suzuki Foundation, www.davidsuzuki.org

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