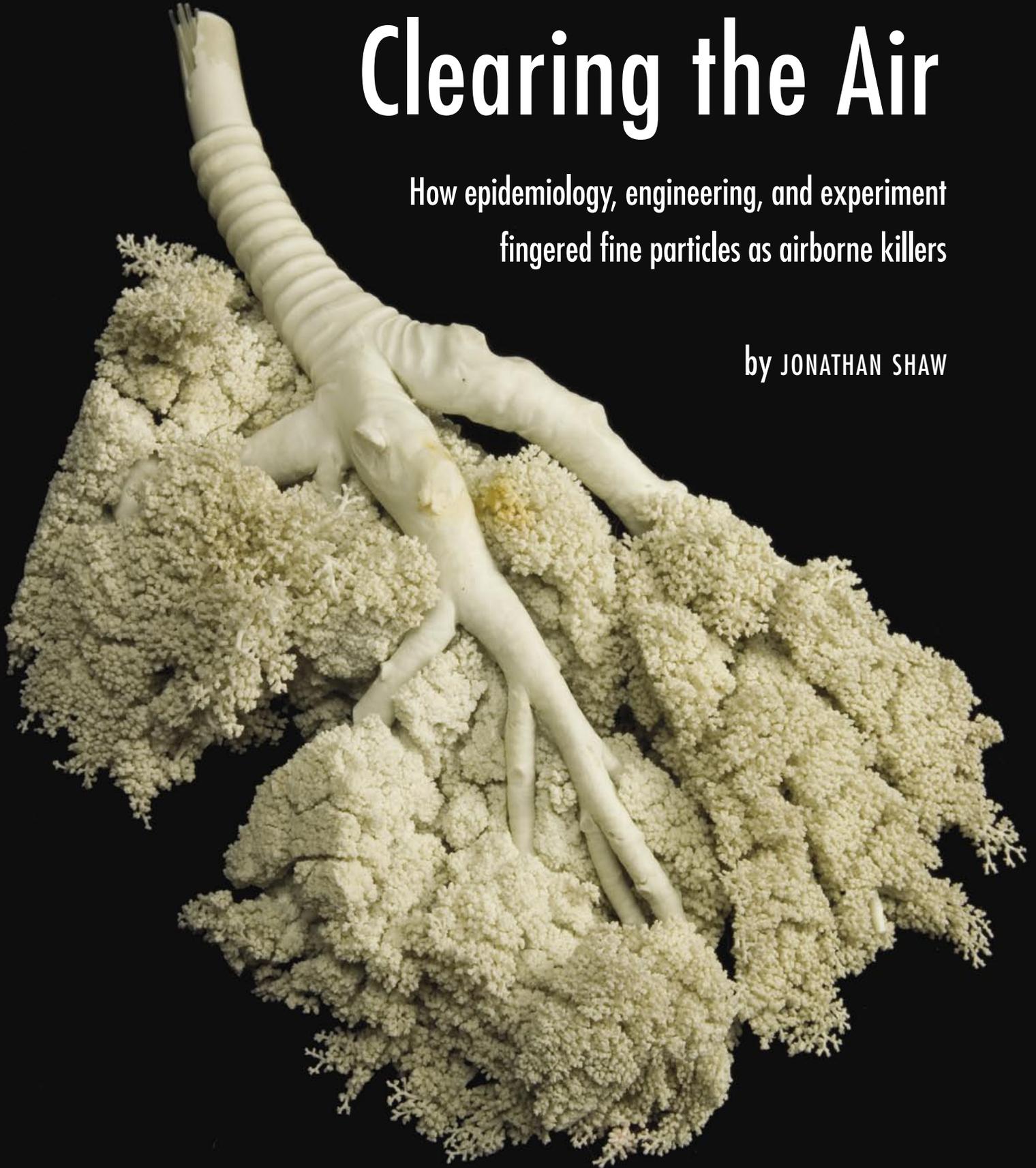


Clearing the Air

How epidemiology, engineering, and experiment
fingered fine particles as airborne killers

by JONATHAN SHAW



EVERY DAY, MOST OF US GET IN A CAR and drive. We adjust the temperature, maybe turn on the radio, and flip on the fan, never considering that the “fresh air” coming from outside is afloat with fine particles: combustion products, sulfates, nitrates, metal dust, and microscopic bits of ground-up concrete and rubber tire. Some of it comes from vehicle exhaust and some is stirred up and resuspended by passing cars and trucks. This fine-particle cloud, say scientists who have measured it, hangs like an invisible dome over busy highways. At rush hour, cars queue up in traffic, bumper to bumper, so that the air intake of one vehicle draws in the tailpipe emissions of another—or even worse, the exhaust from an older diesel bus or truck with a badly adjusted engine. Then the fan pumps particulate-laden combustion products into the car, filling the interior. Soon every cubic centimeter of air—roughly the size of a sugar cube—contains hundreds of thousands of tiny, invisible particles. When you breathe, these fine particles deposit far down in the gas-exchange regions known as the deep lung.

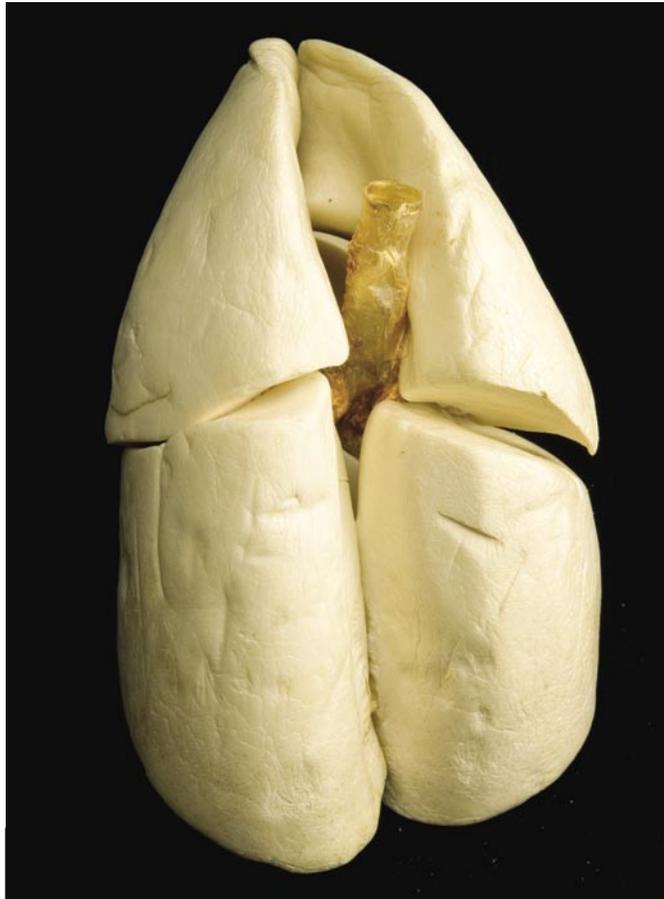
For most people, these brief exposures will not cause any lasting harm. After all, particles are all around us; we breathe them constantly. But in susceptible individuals, or people who are exposed to them long-term, some particles can cause real damage, or even kill.

The lungs are our main point of contact with the environment around us—we breathe 50 to 60 pounds of air a day—and that makes them particularly vulnerable to airborne incursions by gases and fine particles. At the Harvard School of Public Health (HSPH), epidemiologists, economists, engineers, aerosol physicists, veterinarians, and physiologists are working together to understand human influences on the air we breathe.

In the early 1990s, epidemiological research at the school began to suggest that fine particles from combustion sources such as power plants and vehicles (known as PM_{2.5}, or particles that are 2.5 microns or smaller in diameter) are more dangerous to human health than large particles or typical outdoor levels of pollutant gases such as ozone, sulfur dioxide, and carbon monoxide. Researchers comparing air quality in six cities across the United States were stunned when their data showed that people living in cities with the dirtiest air died on average two years earlier than

residents of cities with the cleanest air. The difference in death rates was linked to elevated levels of fine-particle pollution.

In public-health terms, a two-year shift in life expectancy is enormous—comparable to the protective effects of proper diet and exercise—so that the researchers themselves had doubts at first about their findings. But the association held up. Then came the difficult task of figuring out who was dying and why, an effort that continues to occupy HSPH’s innovative Center for Environmental Health, now in its forty-first year of operation under a grant from the National Institutes of Environmental Health Sciences. Because its work influences public policy here and abroad, understanding where particles come from, which ones are dangerous, and how they affect human health are all part of its research agenda.



Human lungs (above) are our main point of contact with the environment, and also our most vulnerable. A complex architecture of branching airways evenly distributes air to 300,000,000 thin-walled alveoli with a surface area the size of a tennis court. The airway cast (left) was made by pouring plastic into a lung which was then dissolved in a bath of acid.

THE STORY BEGINS with the lung itself, which has been a focus of the center’s research throughout its history. Lung diseases like cancer, emphysema, fibrosis, and asthma are almost all initiated or aggravated by the inhalation of particles and gases, says center director Joseph Brain, Drinker professor of environmental physiology. Because these diseases are generally incurable, public-health measures, rather than treatment, are the best medicine. For example, Brain notes that “97 to 98 percent of lung cancer would be eliminated if people didn’t smoke cigarettes and avoided environmental and workplace exposures to air pollution. That is why there has been a strong lung-biology program since the origin of the school, and a persistent interest in environmental and occupational lung disease.”

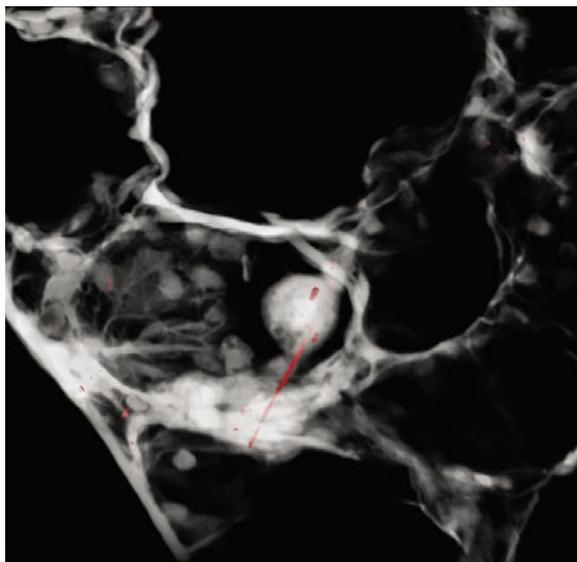
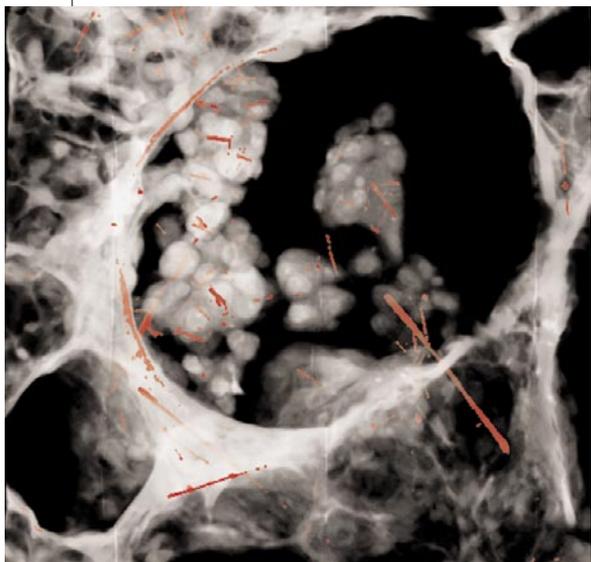
The lung has an enormous surface area, “the size of a tennis court,” Brain says. In addition, the alveolar walls where gas exchange takes place are “much thinner than the gut,

and far thinner than skin. That is good news when you are running the Boston Marathon, because you want up to three liters of oxygen to diffuse efficiently across that barrier each minute, but it also makes us vulnerable.” Each day we take in 20,000 liters of air—compared to just a few liters of food and drink—with little personal choice in the matter. “We breathe in viruses, bacteria, tobacco smoke, welding fumes, and urban particles,” he notes. “Whatever happens to be there can land in the deep lung.”

“Most large particles deposit in your nose,” says Rick Rogers, senior research scientist at HSPH and an expert in tagging and

imaging particles. Rogers's pictures show just where particles end up after they enter the respiratory system. Airborne particles larger than PM_{2.5} are removed in the upper and middle airways. Trapped in a mucus layer that lines the nose, trachea, and bronchi, they are carried up to the back of your throat on a moving carpet of mucus propelled by cilia pulsing a thousand times a minute. Then they are swallowed. But *fine* particles reach the alveoli, where there is no mucus lining (which would impair gas exchange). There the lungs employ a different kind of cleaning agent: macrophages, large white blood cells that eat foreign invaders such as bacteria and viruses by engulfing them, encasing them in an intracellular stomach, and then injecting bleach-like chemicals into the container. The invader dies, degrades, and is reabsorbed by the body along with the macrophage itself. In the

Macrophages are wonderful cleaners, but “like a housecleaner, every hundred hours or so they knock over a vase.”



Asbestos fibers (left)—long, thin, flexible, and durable—reach the deep lung and spear airway walls. Macrophages try to envelope and dissolve them (right), but the fibers are too long to be enclosed completely. Damaging enzymes and oxidants secreted by the macrophage leak out along the fiber and damage nearby lung tissue.

case of bacteria and viruses, “If we didn’t have these macrophages, we’d all be coming down with fatal lung infections,” Brain says. Macrophages also attack and attempt to degrade fine particles, but can be overwhelmed when there is “too much dust or too much smoke.”

Then the macrophages spill some of their weapons in what Brain terms “biological friendly fire. For example, smoke causes emphysema, which is destruction of lung tissue. But smoke doesn’t do it directly; it is the body’s powerful protective mechanisms that destroy the lungs.”

Once an airway or alveolar wall is gone, it is gone for good. The lung can’t regenerate. Macrophages are wonderful cleaners, Brain says, but “like a housecleaner, every hundred hours or so they knock over a vase.”

Sometimes macrophages can’t completely surround a foreign particle. Asbestos fibers, for example, are often very thin, less than 1 micron in diameter, but also very long: 10 to 30 microns from end to end. In theory, they shouldn’t be able to get past the upper airways. In fact, they become aligned with the airflow and penetrate the deep lung. When a macrophage engulfs an asbestos

fiber, it wraps itself around the middle, but the ends stick out, so the macrophage’s intracellular stomach is open at both ends. The reactive chemicals and digestive enzymes intended to dissolve the fiber instead leak into the surrounding tissue and break it down. The fiber persists, eventually leading to lung disease.

Understanding how asbestos causes disease, which may be primarily a question of its physical structure (coupled to flexibility and durability), could make it possible to identify new synthetic fibers with similar characteristics before they cause illness, Brain believes. “If we create something brand-new like Kevlar or nanofibers with no history of human exposure, but with the same physical characteristics [as asbestos], then we get worried. And if we put some in an animal lung and see inflammation similar to that seen from asbestos, we would suggest that exposures be kept low.”

That kind of cautionary advice could save many lives, because the latent period in lung disease is generally very long. Brain describes a man who “in college had a summer job working three months at a shipyard when he was 20, and now, 40 years later, has developed asbestos-related lung cancer” (mesothelioma, a disease so distinctive that it is diagnostic of asbestos exposure).

Sometimes the structure of the lungs leads to different levels of particle exposure. Says Rogers, “We know if you have a mom and her seven-year-old standing at a bus stop and they get a blast of diesel exhaust, the child is going to get relatively much greater particle deposition.” Because of differences in

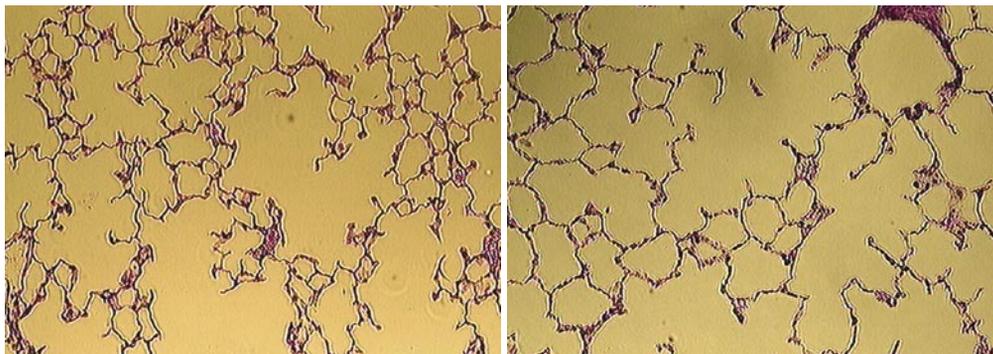
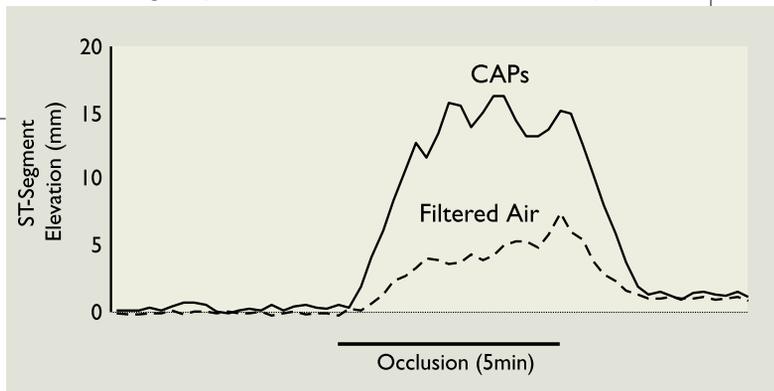
surface to lung volume, metabolic rate, and activity, the seven-year-old’s lungs will get two and a half times the dose of particles as the mother’s lungs. “We first predicted this theoretically,” says Rogers, “and the experimental evidence supports it. The seven-year-old has a fully alveolated lung with an enormous surface area, but a small chest volume, so there is a greater particle deposition relative to the adult, who has a much larger chest volume and a slower metabolism.”

ALTHOUGH LUNG BIOLOGY is fairly well understood, particle biology is not. Asbestos aside, the chemical and physical characteristics that make some particles especially dangerous have not been well defined. Is it even the particles themselves that are the culprits, or is it their surface characteristics? Is the number of particles more important than the mass? The need

for better understanding is especially true of ultra-fine or nano-sized particles that can be the same size as molecules in the body. These may actually enter the bloodstream by crossing the alveolar membrane or by passing *between* cells that make up the alveolar wall. Brain hopes to team with Flowers University Professor George Whitesides, a chemist, to explore this area. “What an academic lab like the one that I run can bring to this,” says Whitesides, “is that we can make particles in which the surface chemistry and composition are very well defined” (see “Thinking Small,” January-February, page 50). “If we knew more about particle biology,” says Brain, “we might be able to design out more of the health effects, or at least reduce them.”

The gaps in scientific knowledge about what particles and gases actually do after they enter the lung was an important obstacle to epidemiologists trying to figure out why air pollution was killing people. Even after the Six Cities study (which began in 1974) fingered fine particles as the

During a myocardial infarction in a laboratory animal, the heart responds differently in the presence of particle pollution (top curve) and filtered air (bottom curve), as seen in the change in part of an electrocardiogram.



If a mother and her seven-year-old child get a blast of diesel exhaust at a bus stop, the child's lungs will get a dose of particles two and a half times greater than the mother's, because the lungs of juveniles, who have a higher metabolism, are fully alveolated but have a smaller total volume. The images above show the smaller-sized alveolar structures in a juvenile (left) versus an adult rat.

culprits, the mechanism was not understood. “We could see effects in children as their lung function went down temporarily by up to 5 percent,” says Douglas Dockery, professor of environmental epidemiology. “So we postulated that the long-term effect of air pollution would be accelerated loss of lung function over a lifetime, leading to disability or death due to respiratory disease.”

But what they found in 1990, a full 15 years into the Six Cities study, was something entirely unexpected. People were losing lung function, but what was killing them were cardiovascular events such as heart attacks and dysrhythmias. And it was fine particles from power plants and other combustion sources such as automobiles and home heating that showed the strongest associations with these deaths. “At first we didn't believe it,” says Dockery. “We thought there must be something wrong.” Recognizing the groundbreaking nature of their findings, the group validated their results by finding the same reduced life expectancy in a much larger sample, using data mined from an existing American Cancer Society (ACS) study. In 1993, the group published their Six Cities findings in the *New England Journal of Medicine*. It is the most cited air-pollution paper in existence.

Science might have marched on from there as it usually does,

with other scientists replicating the results over years of similar longitudinal study. But in 1994, the American Lung Association sued the Environmental Protection Agency (EPA) over a point of law. The Clean Air Act requires the agency's administrator to set air-quality standards to protect the public health every five years, based on the best scientific evidence available. The particle standard had originally been set in 1970, and in 1987 the way it was

measured had been changed to PM₁₀ (particles smaller than 10 microns), though the standard was effectively the same. But nothing had happened during the subsequent seven years. A federal court ordered the EPA to review the scientific evidence on particle pollution.

The EPA is required to set air-quality standards in the interests of human health without regard for the cost. But separately, the agency is also required to produce a cost-benefit analysis of its

regulations. Economists who calculate costs and benefits value a prevented asthma attack at hundreds of dollars, but a life lost at millions of dollars. When the Six Cities and ACS studies linked fine particles to increased death rates,

Dockery says, “The cost-benefit analysis flipped to show a huge benefit from controlling particle emissions.” The EPA issued a new standard in 1997. “Suddenly, we were talking about putting real controls on power plants that would have significant monetary costs,” says Dockery. “Industry mobilized to attack the scientific basis for the standards” (which to this day have not been enforced). “Their strategy was to identify ‘key studies’ used as the basis for the proposed standards. If they could discredit those studies, the scientific basis for the standards would be undermined.”

Dockery and his colleagues had the integrity of their science questioned; all their data were later independently examined—and ultimately validated.* “We are in an interesting quandary,” he says. “Congress wrote the law to protect even the most sensitive individuals, at a time when we thought we could define a level at which nobody would be adversely affected. But as we have become more so-

*Given the complexity of certain mathematical analyses in the Six Cities study linking PM_{2.5} to increased mortality, industry raised concerns and demanded the underlying data. But Harvard, concerned that any re-analysis be done to the highest scientific standards, sought instead an impartial third party to review the data, run new mathematical analyses, and publish the results. The work was performed by the Health Effects Institute, a public-private partnership set up by the EPA and industry in 1980 to resolve disputes of this kind. HEI's conclusions, which support the original findings, were published in 2000.

phisticated in our epidemiologic studies, it has become clear that...this concept—that there is a safe level at which you can protect everybody in the public against health effects—is not holding up. There are detectable health effects at even the lowest levels.”

People want science to define what is safe, Dockery adds. “Science can provide a method for estimating risk, but ultimately, if we decide that we are going to reduce air pollution to the lowest levels” by, say, getting rid of the most polluting diesel trucks, automobiles,

In a sliver of healthy lung (left), the alveoli are densely packed like foam. By contrast, the lung of a Welsh coal miner (right) is riddled with empty cavities, which form when the lung’s natural defense mechanisms against particles accidentally dissolve alveolar and airway walls. These never regenerate.



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and power plants, “that is a political decision.” Those kinds of decisions have other health implications, he points out. “If air pollution controls mean higher costs for electricity, gasoline, or home heating, this can also have health effects on the poor. Science can inform the debate, but won’t produce a magic number defining safe levels.”

ONE OF INDUSTRY’S COMPLAINTS about the epidemiological link between fine particles and cardiovascular deaths was that laboratory studies of healthy animals were showing few significant effects from breathing particles. “Our first experiments with animals were a failure,” says Brain. “We exposed healthy animals to high levels of fine particles generated in the lab and not much happened. Gradually, we realized that it didn’t make sense to expose healthy young rats. We needed animal models of heart disease, asthma, and fibrosis.” And they also wanted real air pollution—fine particles from outside air, not a surrogate like carbon soot.

Led by professor of environmental sciences Petros Koutrakis, a group of engineers and aerosol physicists within the school in 1994 created an “ambient particle concentrator”—a gad-

get as large as a room that takes air from outside and concentrates the particles to simulate the pollution of a bad summer day in Los Angeles—or in Santiago, Cairo, or Beijing. (Harvard now supplies these machines to other universities and to the EPA.) When the researchers exposed susceptible animals to real air pollution at concentrated levels, interesting things

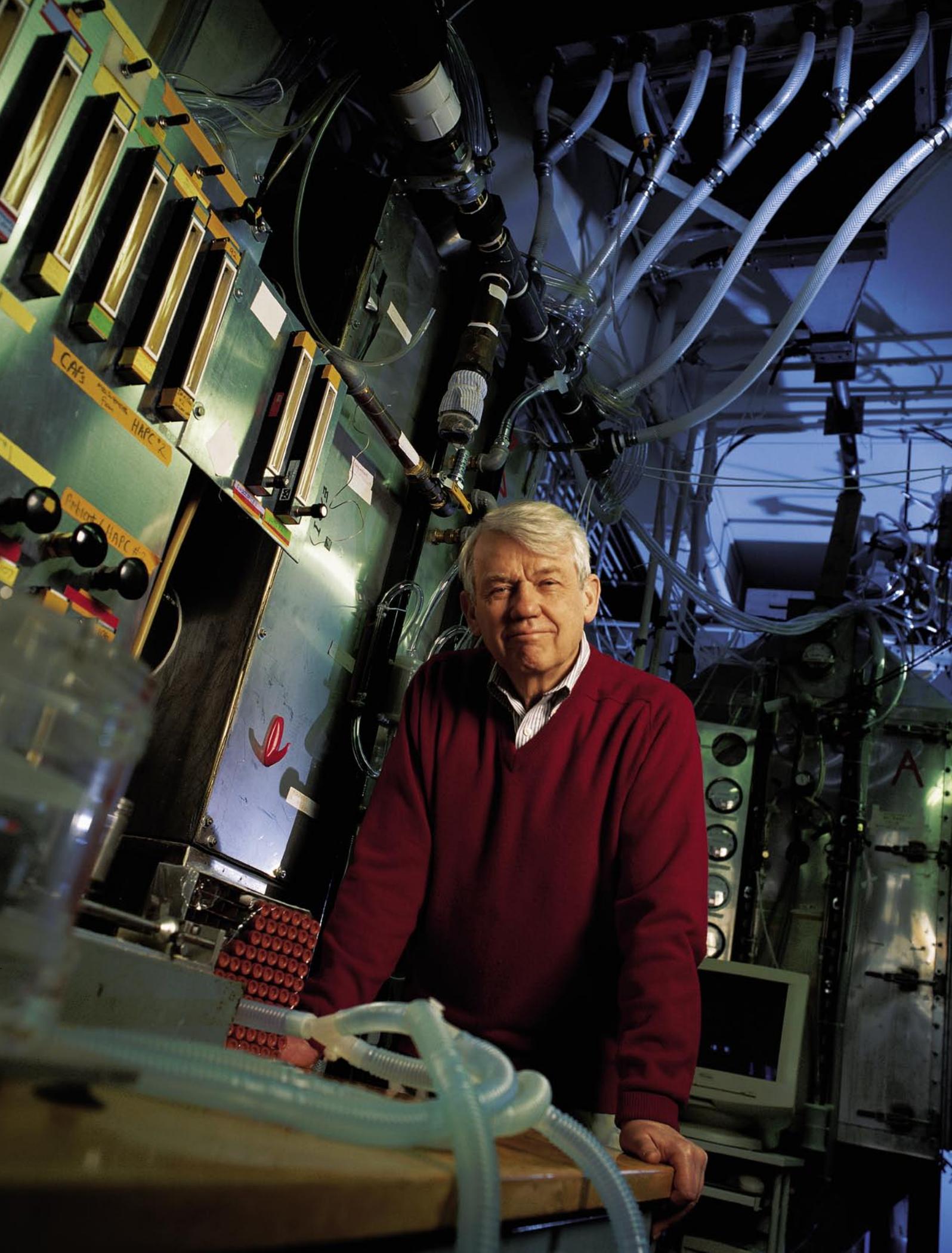
began to happen: cardiovascular changes, changes to the electrocardiogram, and changes in the blood. (The levels used are lower than many occupational exposures and lower than transient levels of pollution experienced in cars or on the street.)

“We have looked at these cardiac effects as a way of understanding the epidemiological findings,” says John Godleski, senior pulmonary pathologist at Brigham and Women’s Hospital and an associate professor in the department of environmental health. “We take an animal and implant a balloon occluder around its coronary artery. Then we occlude the artery to simulate a heart attack, and measure electrical changes.” (This is done for a short amount of time, so it does no permanent damage.) “If you expose the animal to filtered air you get one effect,” says Godleski, “and if you expose the same animal to particles you get a greater effect”—for example, an electrical wave that corresponds to the severity of a heart attack is greatly amplified in the presence of fine-particle pollution.

With this knowledge, researchers at the school conducted a study of several hundred people in Boston with implanted heart defibrillators to see how often they fired. (The defibrillator, as distinct from a pacemaker, administers a powerful shock when it detects an abnormal heart rhythm, and records the date and time.) The researchers found that even in Boston, which is relatively clean, when they plotted PM_{2.5} against the probability of firing, they got a straight line: the higher the fine-particle pollution, the more often the devices would fire, even at levels below the current EPA standard.

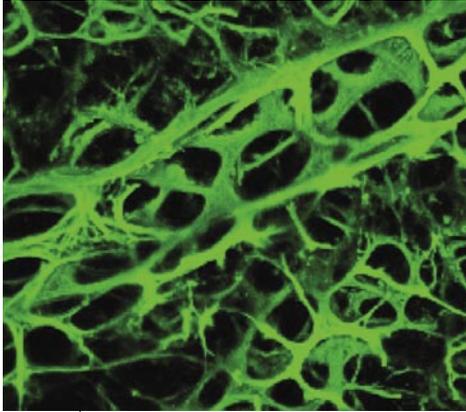
But how do fine particles cause heart attacks? “One hypothesis,” says Godleski, “since some of the effects are almost immedi-

Drinker professor of environmental physiology Joseph Brain with an ambient particle concentrator that allows researchers to expose animals (and soon humans) to higher levels of fine-particle pollution from the outside air, even on clear days. “Air has different particulate signatures,” says Brain. “Sometimes it comes from the Gulf, sometimes it comes from the Midwest, sometimes from Montreal; and sometimes it is dominated by local sources. If there is an east wind, it has sea salt in it.” A selective inlet in the device removes large particles; then the fine ones pass through a “virtual elutriator,” where the air has to make a sharp turn. “Gases can make the turn” and are siphoned off, says Brain, “but the particles have enough momentum that they can’t make the corner and so they go straight through.” The machine, “a complex and very sophisticated piece of engineering” developed by professor of environmental sciences Petros Koutrakis, can concentrate particles by a factor of 30, allowing researchers to simulate the air pollution of Mexico City, Houston, or Taipei.



ate, is that they must be neurally mediated.” Particles may stimulate nerve fibers in the lung. Signals relayed to the central nervous system may change the autonomic balance of the heart in ways that “make it more prone to arrhythmia and other effects,

The lung’s very smallest airways, alveolar ducts, like a Chinese finger puzzle or perforated pipes in a septic field, are full of holes. These alveolar entrance rings allow oxygen to percolate through.



which in turn create the potential for a fatality.”

Another hypothesis suggests that, because particles cause inflammation of the lungs, inflammatory agents

DIESEL ENGINES provide more power per unit of carbon dioxide (a greenhouse gas) than gasoline engines: they are more efficient. With concerns mounting over global climate change, more diesel automobiles are appearing in Europe. In the United States, where low-sulfur diesel fuel will be introduced next year, new diesel engines will also produce about 1 percent as much particulate emissions (by weight) as they did just 20 to 30 years ago. But older diesel engines—which can remain in service for 20 years or more—should either be retired or be retrofitted to burn the new fuels and carefully maintained in order to reduce their particulate emissions, say public-health researchers.

one can take a pill that will reduce the risk of a heart attack, so might a drug someday combat the effects of particle pollution in susceptible individuals.

EVEN AFTER LABORATORY STUDIES VALIDATED the epidemiology of the Six Cities study, questions were raised about the nature of susceptible individuals. So began what Dockery calls “a horrible discussion” that asked, “If people are dying from air pollution, are they people who were going to die soon anyway? Are we just advancing their date of death by a day, and if so [get-

ting back to economic considerations], is that really worth a million dollars [to change]?” After much investigation, it now appears that air pollution is in fact shortening lives by many years.

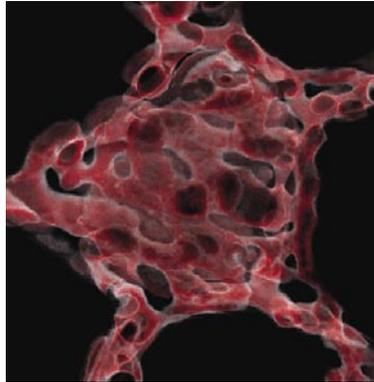
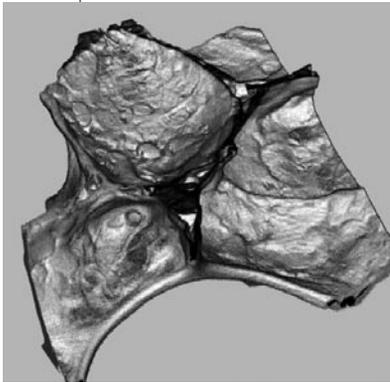
“If you get sick with influenza or pneumonia,” Dockery explains, “you might be in trouble for a few days, but if you recover, you can go on and live for another 20 or 30 years. But if, during the period when

you are sick, air pollution pushes you over the edge, then you are talking about substantial decreases in life expectancy.”

In children, a group made susceptible by their high metabolisms and developing organs, exposure to fine-particle pollution appears to cause small, permanent reductions in lung function. That is less a concern when the victims are young and have plenty of reserve lung capacity. But as people age, they lose about 1 percent of their lung function per year (1.5 percent for smokers). After 50 years, in one’s early seventies, this represents a 50 percent reduction in lung capacity (75 percent in smokers). The consequence, Dockery says, is that “at the end of their lives, when they *need* the reserve capacity, these kids will be a couple of years further along in terms of the decline of their lung function.” “If we could go before Congress and show them a sick baby, that would have a lot of influence,” he notes. “But we’re showing statistical associations, which are not as emotionally powerful. That is why we rely on our colleagues who do toxicology studies to provide mechanistic understanding.”

What those toxicology studies are beginning to show is that in these susceptible individuals, certain kinds of exposures—to combustion products, urban particles, toxic organics, and metals such as zinc, vanadium, and iron—have effects more adverse than simple carbon or resuspended crustal dust. “Saskatoon, Saskatchewan, on a windy day has lots of dust blowing around,” says Brain, “but it is unlikely to be associated with cardiac changes.” Other studies have shown that people, often poor, who live close to highways have the biggest particle exposures and experience the biggest health effects. If you live within 50 feet of a busy highway, for example, you will be exposed to combustion-related pollutants again and again as the particles are emitted and resuspended by every passing vehicle, intensifying the exposure.

The center is currently studying the confluence of two of these factors—metals and resuspension—in children in Tar Creek, Oklahoma, a place where there have been zinc and lead mines for a hundred years. Ten Native American tribes and other people live next to what look like mountains in an otherwise flat part of the



The seams where cells meet are clearly visible in this image of an alveolar wall (left). When the lung becomes chronically inflamed or infected, tiny cracks can develop between the cells in this wall, allowing fluid from the blood behind it (right) to leak through.

produced there may affect the heart in a negative way. Vasoconstrictors such as endothelin, for example, are secreted by the lungs when inflamed. The fact that mortality peaks 18 to 20 hours after the peak in a particle-pollution event (such as a smoggy day in summer) lends some support to this possibility; think of the way a sunburn can develop over time, after you leave the beach.

Finally, particles may pass through the lungs and actually reach the heart directly. This is thought to be the least likely scenario, says Godleski, but there is some evidence for it. Rogers is actively exploring the possibility with high-resolution laser-scanning microscopy.

Godleski’s current research involves understanding the origins of the electrical effects he has observed: are there variations in the flow of blood in the heart, or in the coronary vessels themselves? Are there changes in blood pressure that create more work for the heart? He believes that if we can understand the mechanisms well, there is the potential for therapy. Just as

state. One hundred to 200 feet high, they are mountains of mining waste, called chat piles, and are used to make dirt roads. “You see trucks driving along them and resuspending the stuff,” says Brain, who has done animal studies showing that a major pathway of metals to the brain is along the olfactory nerve after they have been dissolved in nasal mucus. Center scientists are measuring markers of several metals in the blood of newborns at Tar Creek and looking for developmental, neurobehavioral outcomes such as autism, low IQ, and attention deficit disorder.

NOT ALL PARTICLES COME FROM OUTSIDE. The EPA tracks, maps, and publishes on-line forecasts of particle and ozone exposures around the country to help people make healthy choices about what time of day to exercise (morning, on days when ozone is predicted to be high) and when to restrict outdoor activity (for children, in particular, when particle counts are up).

But fine particles penetrate a typical home very efficiently, says Yamaguchi professor of environmental health and human habitation John Spengler, and ground-level ozone reacts with household compounds to form particles indoors, where people spend most of their time. Spengler pioneered studies of pollution in cars, subways, homes, and other indoor spaces. He says that ozone indoors creates particles and irritating gases through chemical reactions with common products containing limonene or pinene—cleaning liquids with a scent—and camphor (such as Ben-Gay). Although ozone from photocopiers and printers does react with the terpenes in toner to form particles, office buildings with filtered mechanical ventilating systems typically have much lower particle concentrations than outdoor air. That is because nowadays, in most office environments, there is neither smoking nor cooking, the two primary indoor sources of particles in the United States.

Increasingly, Spengler’s work on indoor air pollution has led him to embrace a global agenda. “The majority of the world’s population is still heating and cooking with biomass fuels,” he points out: “wood, crop residues, animal dung, and charcoal. There is a huge price being paid from particle pollution, particularly among women and children, because they are doing the cooking. In terms of the human toll,” he says, “it is the equivalent of several World Trade Center disasters every day in the developing world: 4 million deaths a year, mostly children.”

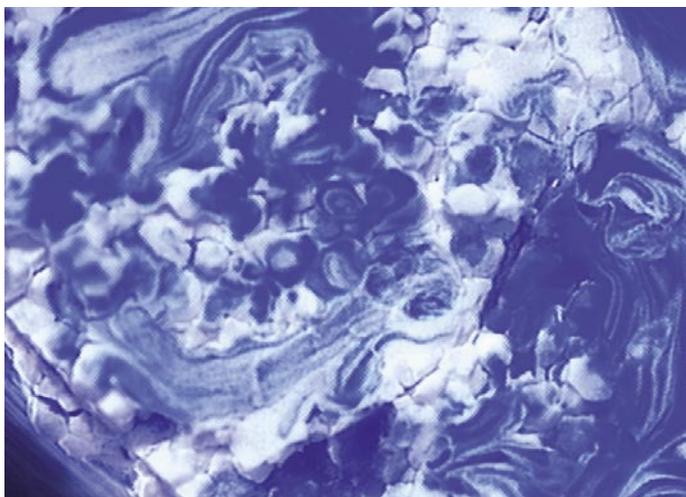
One Westerner living this lifestyle in a Nepalese village for a

year described it to Spengler in graphic terms: “I quickly learned not to stand up” inside dwellings, in order to avoid the smoke that collected near the ceiling, she told him. In the morning, she would wake, not to the sound of roosters, but to a “gut-wrenching hacking, followed by spitting” that could be heard throughout the village as its residents coughed up the mucus secreted in their lungs during the night.

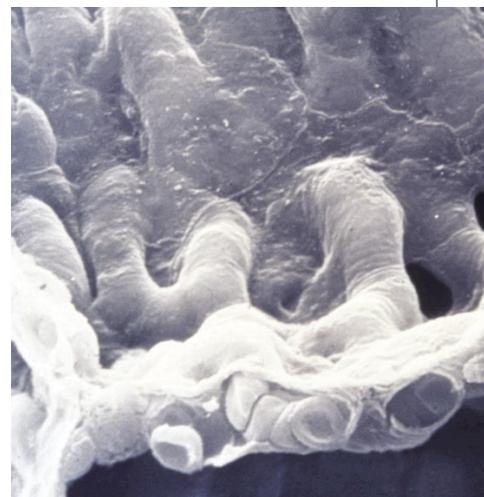
“This is a daily experience,” says Spengler. “Sure, we are worried about particles domestically and sure, there are health effects documented in the U.S. population and yes, we need to get tougher in our regulations, but we really ought to face up to this problem in global civil society.”

“We have an obligation in public health to go beyond just doing the research,” Spengler says. “We are here to improve public health, and that goes beyond just studying problems.” Domestically, that has meant assessing the health costs associated with power plants and vehicles—and showing what we as a society are paying that is *not* reflected in the price of electricity or the

“The majority of the world’s population is still heating and cooking with biomass fuels: wood, crop residues, animal dung, and charcoal.”



In an effort to understand why particles deposit where they do, researchers have learned that there is a high degree of mixing in the lung because of chaotic air flow.



The barrier separating the outside air from blood in the human lung is vanishingly thin, just 1 micron across—one-tenth the diameter of red blood cells, the doughnut-shaped objects (foreground) seen in the capillaries of an alveolar wall.

price of gas at the pump. And it has meant advocating tighter controls on particle emissions from existing diesel engines, not just new ones.

Globally, he says, the strategies for improving human health are not the same as in the United States. The answer abroad is to eradicate poverty, but the hope is that, as their life circumstances improve, developing societies don’t just migrate from one set of hazards to another. “As people gain in wealth, they move up the energy ladder,” Spengler says. The challenge is to ensure that they don’t trade open cooking fires for highways clogged with cars idling in rush-hour traffic. ▽

Jonathan Shaw ’89 is managing editor of this magazine.

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