

THE AMBIENT AIR*

EDITH IGLAUER

In the universe, our earth is a mere dot, but it is still the only celestial body we know about that supports life, and a primary requirement of life on this planet is the mixture of gases that surrounds us, called air. We cannot be deprived of one of its ingredients, oxygen, for more than six minutes without suffering fatal brain damage. This gives us no chance to be fussy as we move through our daily lives, so we take the air pretty much as it comes. The air we normally breathe is made up of about one-fifth oxygen, a little less than four-fifths nitrogen, argon, and traces of other gases, of water vapor in varying amounts, and, finally, of all the poisons, waste products, and general trash we hurl into it as if it were not the thing most necessary to our existence. There is plenty of air—between five and six quadrillion tons—but because we don't know whether there is any more like it anywhere else, we have to be sure we can manage with what we have. And what we have is essentially an ocean, with vertical limits, and with enough oxygen for normal breathing only in the lowest fifteen thousand feet of the troposphere. Most of the air we use is in the first two thousand feet of this shallow layer, and what we ordinarily breathe is in the first seven feet.

The mechanisms that enable us to use air are the lungs, and their most striking feature is their ability to clear themselves of impurities.

* Reprinted with permission from *The New Yorker*, April 13, 1968 (© 1968 The New Yorker Magazine, Inc.).

The nose, the mouth, and the throat are loaded with a rich assortment of bacteria; a few inches below them are the lungs—in a normal person sterile, beautifully designed structures that permit rapid and easy intake of air, from which oxygen is absorbed into the bloodstream in exchange for outgoing wastes. A few scientists have deliberately inhaled radioactive particles in order to map the path of impurities through the human body, and they have reported that impurities are normally cleared from the lungs in something between two and four hours. The marvellous cleansing devices without which the lungs would soon fill with lethal dust are cilia—small hairlike projections that are actually tiny pieces of tissue growing on the surface of the windpipe and the bronchial tubes. The cilia beat upward like small canoe paddles, pushing along a continuous stream of cohesive fluid—mucus—which carries impurities breathed in through the nose back up the throat to be swallowed into the digestive system.

The respiratory tract has been likened to a tree growing upside down in the human chest. The trunk is the windpipe, and the bronchi are the branches. Below the conducting airways are the primary functional units of the lungs—an abundant array of little air sacs called alveoli, which can be compared to the leaves of the tree. Here is where the real work of the lungs occurs; the entire blood supply of the body flows through this area, which is intricately interlaced with capillaries,

and it is here that the oxygen breathed in joins the bloodstream and carbon dioxide is removed. Impurities that somehow evade the sweep of the cilia and enter these more distant reaches of the lungs are not readily cleansed from the air sacs. Bacteria invading the alveoli are ordinarily ingested by special cells called macrophages and dissolved there by enzymes, but solid particles find their way between or into the air sacs and then into the lymph channels in the walls of the bronchial tree, where they may remain for weeks or indefinitely. In each human lung, there are three hundred million alveoli, and the classic saw in medical schools is that the combined surface area of the alveoli would cover a tennis court. The walls of the air sacs are diaphanous membranes, thinner than paper. When these walls progressively break down, making breathing more and more of an effort, the unhappy owner of the lungs is suffering from a disease called emphysema. Or, in other cases, the walls of the bronchial tubes may become chronically inflamed, causing an increased flow of mucus and coughing; this is bronchitis. Either way, the lungs have ceased to be an efficient breathing machine. For many years, the medical profession was rather bored by emphysema and bronchitis, but in recent decades sharp increases in deaths from these ailments, together with an even greater rise in deaths from lung cancer, particularly in the United States and Great Britain, have caused an enormous upsurge in research into respiratory ailments. Emphysema is now the fastest-growing cause of death in the United States; in Great Britain there is so much bronchitis that it is regarded as the national disease, and the incidence of lung cancer there has increased about a hundredfold in the last sixty years. Physicians and other scientists, intensifying their efforts to explain these assaults on

the lungs, are looking more and more closely into the effects of dirty air on the human body.

When scientists study a disease, they like to go after first a cause and then a cure, and to identify the disease and reproduce it in the laboratory. But there is no "air-pollution disease" to reproduce, and no set of symptoms that can be precisely attributed to air pollution alone. Thus, although discoveries are continuously made in the field of air pollution, scientists find difficulty in agreeing on their significance. It appears at this point that the increasing contamination of our air with the wastes from more fuel burned for more power, with the exhaust products from more internal-combustion engines, and with emissions from old and new industries in rapidly developing countries aggravates rather than causes respiratory disease. If there is a single obvious villain, it is in the form of personal air pollution that a victim creates for himself and breathes in high concentrations directly through his mouth into his lungs while smoking. However, it is known that the person who smokes amid the dirty air of a city has a greater chance of developing a pair of sick lungs than his compatriot who smokes in the country. This elusive difference is called "the urban factor"—which means that something in city living puts an extra burden on the lungs, increasing the risk of disease.

Although air pollution's role has not been precisely defined, any inquiry into its effects on the human body must start with one indisputable fact: When the supposedly benign ocean of air in which we live is overloaded with contaminants under certain adverse weather conditions, people in cities die—not just one or two people more than usual, but lots of them. This is the reality that has sent scientists by the hundred to work on air pollution; that has released millions of government dollars for

research on thousands of guinea pigs, mice, rats, dogs, cats, chickens, rabbits, and even donkeys, which spend their lives breathing varied mixtures of noxious air or being painted with ugly extracts of automobile exhausts or tobacco fumes; that has impelled the British government to appropriate money from its scarce supply to finance a country-wide air-sampling network and to subsidize a change in the individual heating arrangements of an entire nation; and that is causing other governments, most notably that of Japan, to take long second looks at their own air before such disasters strike their heavily industrialized and heavily populated cities.

In urban areas of the Northern Hemisphere during the months from October to February, what are called temperature inversions—in which a layer of cool air is trapped under a layer of warm air—have caused lethal events that are referred to by experts on air pollution as "incidents," "acute episodes," or "disasters," depending on their severity. Each time, a blanket of warm air has for several days imprisoned heavily polluted cooler air over a densely populated surface area, and when the winds have cleared the stagnant air away it has been discovered that there were many more deaths than would normally have been expected. In December, 1930, a heavily industrialized section of the Meuse Valley, in Belgium, had a bad three-day fog during which hundreds of people became ill and sixty died—more than ten times the normal number of deaths. Shortly afterward, during a thick nine-day fog in January, 1931, five hundred and ninety-two people in the Manchester and Salford area of England died—again a large jump in the death rate. But to us in the United States these events seemed remote—until 1948, when, in Donora, Pennsylvania, a small mill town dominated by steel and chemical plants, a four-day

fog filled with zinc sulphate and sulphur dioxide, among other pollutants, made almost half the fourteen thousand inhabitants sick. Twenty persons died. Ten years later, Donora residents who had been acutely ill during that episode were found to have a higher rate of sickness and to die at an earlier age than the average for all the townspeople.

The British, who have been complaining about the unpleasant effects of coal smoke since the year 1273, when it was termed "prejudicial to health," were recording air-pollution episodes as far back as 1873, when, during a London fog, two hundred and sixty-eight unexpected deaths from bronchitis were noted. But it was not until a great fog blanketed London in 1952 that the sinister potential of air pollution became fully apparent to everyone. This fog lasted from December 5th to December 8th, and ten days later, when the complete mortality reports had been evaluated, a shocked world learned that there had been an astonishing four thousand more deaths in Greater London during that period than would normally have been expected. During previous temperature inversions over London, the excess mortality had been among the elderly and infants under a year old, but in this disaster the percentage increase in mortality was similar for all age groups. In Donora, victims had not begun dying until the third smoggy day, but in London a number of the unexpected deaths occurred within the first twenty-four hours; then when the air cleared the rate fell abruptly. The statistics indicated that almost all those who died unexpectedly had records of bronchitis, emphysema, or heart trouble, and that people in the last category were most vulnerable. It was small comfort to conjecture that most of the people who died would probably not have lived much longer anyway. Again, in January, 1956, a thousand extra

deaths in London were blamed on an extended fog. In that same year, Parliament passed a Clean Air Act, and Britain embarked on an extraordinary program to reduce the burning of soft coal. The effects of the program became apparent in 1962, for, in December of that year, London experienced a severe fog and inversion, and this time seven hundred excess deaths were recorded—still too many, of course, but an improvement over the previous figures. The lower mortality was attributed not only to less smoke but to generally better medical care (including wide use of antibiotics) and to greater public awareness—through newspaper, radio, and television coverage—of what was happening and of how to take precautions during a period of severe air pollution. Those with serious heart or respiratory ailments knew by then that they should stay indoors, that they must not smoke, that they should get plenty of rest, and that they should move slowly in order to decrease the demands on their respiratory systems. Frightening alarms were carefully avoided, yet far more than the normal number of people dropped dead in the streets from heart failure.

New York has the most severe air-pollution problem in the United States, but its air is still not as dirty as that of London, which has the reputation of being the cradle of air pollution. In November, 1953, early in 1963, and over the Thanksgiving weekend in 1966, New York had smog episodes that were bad, but not as deadly as London's. They have been carefully studied by Dr. Leonard Greenberg, who was New York City's first Commissioner of Air Pollution Control and is now Professor of Preventive Medicine at Albert Einstein College of Medicine, where he is working with a group of air-pollution specialists. He has calculated that there were approximately two hundred and twenty

excess deaths owing to air pollution in 1953, three hundred to three hundred and fifty in 1963, and one hundred and sixty-eight in 1966. These episodes are a warning that the city's air supply has limits and must be protected before it becomes so contaminated that the normal prevailing winds cannot disperse the filth. New Yorkers wake up almost every morning to a temperature inversion that usually extends about a thousand feet above the ground and lasts from six to eight o'clock, though even in good weather it is not fully burned away by the sun until about ten. The brilliant red sunsets so much admired in New York and Los Angeles are caused by large particles in the air, most of them produced by incomplete combustion in the generation of heat and power. Meteorologists and air-pollution experts do not enjoy these sunsets, and they get really depressed when they consider how the tendency of our growing population to cluster in relatively small areas will affect the air and the climate of the future. They say that in twenty years, when population increases will have caused Philadelphia to merge into New York and New York into Boston, so that there will be one long city with no open spaces in between, the wind patterns will change and only people living on the edges of this huge sprawl will get any rural breezes. The fellow at the center will be living on what is called a "heat island"—a phenomenon that is already occurring in a small way in several cities. At street level, New York is now, on the average, from ten to fifteen degrees warmer at night than the Westchester suburbs, owing to the heating of the buildings in winter and the storage of solar heat in the asphalt and concrete in summer. In fifty years, the Westchester suburbanite may be able to grow tropical plants in his yard, but the great mass of air around him may be so polluted that he will scarcely

dare step out into that yard.

In worldwide terms, the air is still clear. Pollutants, fortunately, are still subject to a huge natural removal cycle, which will operate as long as there is enough time or room for their dispersal. No one knows just when the limits of dispersal will be reached, but when they are, a slow worldwide buildup of background pollution levels in the air will begin. There is evidence of a tiny increase—something like two-tenths of one per cent—in the quantity of carbon dioxide in the atmosphere annually, because of the ever-mounting combustion of fuel. This has led to speculation about a possible “greenhouse effect,” whereby the radiative property of carbon dioxide will cause the planet to warm up enough so that the ice caps will slowly begin to melt.

The average person, however, is not worrying about melting ice caps when he looks up at the murky sky but is simply wondering what the air is doing to *him*. He may notice that his eyes itch and are red and teary. The tears, a natural eyewash, protect his eyes from whatever is in the air; if he consults his ophthalmologist, he will be told that the danger is slight as long as he doesn't irritate his eyes further by rubbing them. People who find smoggy days distasteful—you can actually taste and smell the sulphur in the air with even as little as one part per million—are apt to say they find it hard to breathe, or feel a bit stuffy, or have a headache. For asthmatics, the feeling of pressure is real, because most of them seem to be particularly sensitive to sulphur compounds. The presence of enough sulphur in the air to be noticeable usually indicates that there are other pollutants as well: hydrocarbons, nitrogen oxides (which are responsible for the brown smog clouds so often seen over our cities), ozone, and so on. The most cursory knowledge of the damage to vegetables in

Staten Island and New Jersey truck gardens near chemical industries, or to fruits and flowers exposed to California's highway smog, or to trees near smelters in Western states, coupled with the experience of discomfort in the eyes, throat, and chest when the air becomes overburdened and stagnant, makes us all wonder uneasily whether all living matter isn't being affected in ways we cannot see. Is there a relationship between emphysema and air pollution? Why do people who live in dirtier air get more colds and more bronchitis? Why is there more lung cancer in cities? No one knows what is happening to normal people continuously exposed, as all of us are, to varying levels of pollutants in what scientists poetically call the ambient air—another name for the everyday atmosphere—and the answers may be as elusive as the air itself.

The search for the pieces to this gigantic puzzle is being pursued through basic research on animals and human beings in the United States and Great Britain. In the United States, the Public Health Service directs a good part of the research in its own National Center for Air Pollution Control, and through contracts with university laboratories and special units as far away as Japan. The British, with a wistful look at the large sums of money available to their American colleagues, have assigned some of their investigations to a dozen or so handpicked scientists who form a group called the Air Pollution Research Unit, in London, and they have assigned other investigations to forty or so people who make their headquarters at the government's Warren Spring Laboratory, near the capital. In both the United States and Great Britain, the lab work is being reinforced by broad studies of the behavior of diseases in communities—studies that the British, with their talent for collecting and evaluating statistics, have developed into a fine art. If exact clin-

ical proof of damage to human health from air pollution continues to be as shadowy and inconclusive as it is now, these studies may be the most important work of all.

A large part of the American biological work is done on animals, but the British place their research emphasis on man, partly because in England there are stringent regulations on animal experimentation—which in any case is expensive—and also because they are skeptical about assumptions concerning the complex physiology of man that are made on the basis of work done on the simpler and often not altogether similar mechanisms of lower animals. “Extrapolation,” which in this context means inferring that results achieved from experiments on animals can be applied to man, is a fighting word in air-pollution circles. American scientists say that though most biological experimentation by necessity has to be conducted only on creatures other than man, the results *can* guide and suggest—that if a substance is found to be injurious to animals it seems advisable to expose man to the substance as little as possible. Final proof, if any, of the effects that our air has on our health will probably come about by relating laboratory findings to results of epidemiological investigations—studies of the distribution of disease among populations. This type of study is a sort of statistical embroidery, a matching and meshing of interrelated numbers—census figures, figures for deaths and illnesses, daily pollution and weather readings, employment statistics, hospital-admissions statistics, and the answers to special questionnaires that center on lung functions and smoking habits. Air pollution has at last become a fashionable worry, and local air problems are being examined everywhere—in such widely scattered spots as New Orleans, Genoa, Sheffield, and Osaka. Air does not halt at national borders, and neither do epidemiologists,

who are engaging in more and more joint enterprises wherever two or more countries share a patch of murky heaven, or wherever something can be learned from comparisons. A study of the pollution in the air in the Detroit River area between Detroit, Michigan, and Windsor, Ontario, was undertaken in 1960 by a joint American and Canadian commission, and it revealed both causes and effects of the deteriorating air, which was blackened by hand-fired coal-burning ships and by more than six thousand industrial plants in the vicinity. Often, such studies produce unexpected findings. For instance, an epidemiological comparison of chronic respiratory disease was made in two towns chosen because they were very similar in every respect except that of air pollution—Berlin, New Hampshire, whose pulp and paper mills cast a blue haze over the community, and Chilliwack, British Columbia, a town with very clean air. The comparison led the epidemiologists to the conclusion that cigarette smoking was more harmful than the level of air pollution, and also to the discovery that men working in the paper mills of Berlin had less disease than anybody else covered in the study—primarily because if they weren't healthy they wouldn't be hired or be kept on.

An important epidemiological method is to study twins, especially identical twins, who have been separated and live in different environments. A few years ago, Dr. Rune Cederlöf, of Stockholm, first investigated the respiratory and heart ailments of twins in relation to the "urban factor" in his own country, and he is now making a similar investigation here of more than seven thousand pairs of male twins. His Swedish studies showed that when one twin lived in the country and the other in the city and both smoked, the city twin was more susceptible to bronchitis and angina pectoris than the country twin, and that, in his words, "the

effect of being a smoker appears to be more pronounced when combined with exposure to air pollution. . . . There were no such noteworthy differences between non-smokers."

The urban factor is interrelated with so many other elements—smoking, occupation, economic class, and so on—that epidemiologists attempting to isolate it resort to all kinds of devices. In 1959, the Japanese tried the idea of systematically measuring the breathing capacities of schoolchildren ten and eleven years old who had not yet started either to smoke or to work. Epidemiologists went to Kawasaki, an industrial city between Tokyo and Yokohama, and chose children from two schools—one surrounded by industrial plants, the other on a hill twenty miles away in a rural environment. The breathing capacity of the schoolchildren in the industrial area was found to vary with levels of air pollution. They breathed to the full capacity of their lungs on days when the winds were doing their job of dispersing the pollutants, but when the air was still and the dustfall from metal-working factories, foundries, and electric-power plants in the area was so heavy that it soiled the books and papers in the classrooms, the children's breathing was much more shallow. In the rural neighborhood, there were no such variations; the children's breathing capacity simply increased with their size.

Startling differences in the ability to breathe the Japanese air, depending on its degree of pollution, began to be discovered by the United States Army in the late nineteen-forties during the occupation, when some of its personnel contracted a previously unknown ailment, originally called Yokohama Asthma and later renamed Tokyo-Yokohama Respiratory Disease. The industrialized Kanto Plain, where Tokyo and Yokohama are situated, is usually covered with

smog, which is especially dense in winter, and a number of Americans stationed in this area who had no medical or family history of asthma suddenly began to display all the symptoms of severe asthmatic attacks when the air pollution was particularly bad. Among flying personnel with these symptoms, the disease seemed to vanish when they flew to a height of five thousand feet or more above the contaminated area, but it returned again within minutes of their landing. Ninety-seven per cent of the six hundred and twenty individuals who were diagnosed as suffering from Tokyo-Yokohama Respiratory Disease were smokers, and all continued to have attacks as long as they remained in Japan. The attacks diminished or ceased when they left, but if they returned, even after as long as six years, the severe symptoms recommenced almost immediately. Biopsies showed the disease to be similar to the British form of bronchitis, and patients who had the ailment for some months, or during more than one tour of duty in Japan, developed permanent damage, progressing rapidly to emphysema. Lieutenant Colonel Harvey W. Phelps, a United States medical officer who has followed the history of the disease through the years, has decided that it is "induced . . . by the combined effects of cigarette smoking and the severe air pollution in the . . . area," and that the best treatment is early removal from Japan. When the United States Army sponsored a special investigation among the local population, no cases were found.

In San Marino, California, a suburb of Los Angeles, a high-school cross-country team was recently studied by the United States Public Health Service in an effort to discover whether variations in the purity of the air affected the performance of the runners in their gruelling two-mile races. The boys' records in competitive meets between 1959 and 1964 were com-

pared with air-pollution measurements for the days the meets were held. Runners ordinarily improve their performance as the season goes on, and this team, throughout each season, did better at each succeeding meet except in four instances. The four days on which the team did worse than it had done at the preceding meet were the four worst days of smog. The study also indicated that it was specifically the oxidant compounds of Los Angeles' renowned photochemical smog that caused the trouble, since on those four days the levels of oxidants were high but the levels of carbon monoxide and of solid particles were not; in fact, the very worst day of all for the team was the one on which the carbon-monoxide reading was lowest. The performances of the athletes indicated, moreover, that they were affected by the lowest measurable quantity of air pollution—one part per hundred million units of air, which in Los Angeles is considered a clear day. The practical significance of such a study is that if a smoggy day makes a young athlete just a little more tired, an older man with cardiac trouble had better not run for his bus on such a day, or attempt any other exertion that might tax his injured heart.

On quiet Keppel Street, in London, in the somewhat austere quarters of the London School of Hygiene and Tropical Medicine, the school's professor of epidemiology, Dr. Donald Reid, an urbane Scot with a diplomat's manner, is developing a new line of research. Assisted by a group of younger men he has trained, he is conducting studies of respiratory disease among people in different parts of the world who are doing basically the same type of work. In 1960, Dr. Reid began an intensive study of respiratory ailments among British postal employees, and this is now being duplicated among postal workers in the United States and Japan. The British study revealed

that post-office employees working outdoors as truck drivers and maintenance men in central London, where the foggy pollution was greatest, had much more serious chest illnesses than men doing the same jobs in relatively unpolluted areas. Men over fifty were most seriously affected, and among them especially, but not entirely, those who smoked. In outlying districts, men employed in the telephone branch of the post office were also included. In the United States, postal workers in New York City and telephone-company employees doing outdoor work in Washington, Baltimore, and Westchester have been investigated. The studies have established that in the past the British worker has run a much higher risk of serious lung trouble than his American counterpart.

Just how much worse the pollution of Britain's air has been nobody knows, but Americans have no cause for complacency. Recently, for instance, three University of Manitoba pathologists who had studied three hundred lungs from emphysema victims in St. Louis, where there is a relatively high degree of pollution, and three hundred more from victims in Winnipeg, where pollution is relatively light, reported that severe emphysema struck its sufferers (all of them cigarette smokers) earlier and killed them faster in St. Louis than it did in Winnipeg. A study made in Buffalo by a research group headed by Dr. Warren Winkelstein, of the University of Buffalo's School of Medicine, showed that between 1959 and 1961 deaths from all causes among men between fifty and sixty-nine on the same general economic level were a third higher in the section of the city with the highest level of pollution than they were in the least polluted part. Deaths from chronic respiratory diseases were twice as frequent, and similar findings by the late Dr. Louis Zeidberg in relation to acute respiratory diseases have been reported from Nash-

ville, Tennessee. Buffalo and Nashville, though they are not exactly clean cities, are not America's dirtiest, either.

If Britain continues its vigorous national program to clean up the air and we continue our careless, localized policy in regard to air pollution, the British may ultimately win the game of who breathes cleaner air. The British government is partly subsidizing a shift to natural gas for home heating, and this fuel, thanks to recent discoveries of it under the North Sea, will eventually be cheaper than coal. At the same time, the nationalized Central Electricity Generating Board is building new nuclear-power stations in isolated places; the generation of nuclear power does not pollute the air, although even the slightest possibility of an accident makes officials wary of building atomic stations near populous areas. In any event, the development of techniques for transporting high-voltage electricity economically over long distances has made it unnecessary for power plants to be built in such areas.

According to Dr. Reid, British men between forty-five and fifty-four suffer twice as much lung cancer and five times as much general respiratory disease as their North American counterparts (although the latter have a greater chance of dying from heart disease). Women in the two countries follow similar, though less drastic, patterns. With the collaboration of the United States National Heart and Cancer Institutes and the Norwegian Cancer Registry, Dr. Reid is conducting another study, in which he keeps track of emigrants from England to the United States to see whether, in the generally cleaner American environment, the health of British respiratory systems improves, and in which he also collects data on Norwegian migrants here to see whether a group coming from a very pure atmosphere suffers more or less than the British group in

the more polluted American environment. This study suggests that the migrant retains a residual advantage or disadvantage from childhood. So far, the Norwegian immigrants have turned out to have lower susceptibility to both lung cancer and chest diseases than native Americans, and the British migrants seem to have about a one-third greater chance of contracting lung cancer than native Americans. However, their chance of contracting a respiratory disease is only a fifth of what it would have been if they had remained in Great Britain. Other studies have indicated that Britons who migrate to the cleaner air of Australia, New Zealand, and South Africa follow much the same pattern.

How long must the human body be exposed to British air before the pollution leaves an indelible imprint on the lungs? Will the current British smoke-control program make any difference? Dr. Reid, speculating on these questions with an American visitor recently in his pleasant office (which has a gas burner in the fireplace), said, "What happens to the British emigrant in America means that we are not innately condemned to have chest disease. If the British environment can be brought up to American levels, the frequency and severity of chest illness among the British should fall."

British epidemiologists, like their Japanese colleagues, consider children admirable experimental subjects. Two studies of children—one conducted in the steel town of Sheffield and the other in rural Wales—showed that children living in heavily polluted areas have more frequent and more serious ailments of the middle ear and disorders of the lower respiratory tract. This lead is being followed up by Dr. Reid and Dr. John Colley in a survey of twelve thousand children in parts of the country with widely different pollution levels. The most extraordinary of the recent studies involving children,

however, was made by one of Dr. Reid's younger associates, Dr. Walter Holland, of St. Thomas' Hospital Medical School in London, among fifteen thousand school-children in a county in southeastern England. The purpose of Dr. Holland's study was to account for variations in the lung functions and breathing levels of children. He discovered, unsurprisingly, that the children who breathed best lived in an unpolluted country atmosphere, that children from well-to-do homes breathed better and more deeply than poor children, and that children who had had severe diseases like pneumonia and bronchitis early in life were worse off than those who hadn't had them. What did surprise Dr. Holland was the answers the children gave to standard questions about smoking. Children between eleven and sixteen who smoked cigarettes had two to three times as many symptoms of respiratory trouble—coughing, phlegm, wheezing—as children who did not smoke, with youthful ex-smokers somewhere in between. Children who smoked five or more cigarettes a day had four times as many respiratory symptoms as non-smokers; in fact, they had already given themselves respiratory conditions comparable to those suffered by smokers in their forties. Dr. Holland defines a "smoker" as an adult who smokes a cigarette a day for a year or a child who smokes a cigarette a week for a year. Among the children he studied who were between the ages of nine and thirteen, nineteen per cent of the boys and five per cent of the girls said that they smoked regularly, and a third of the boys and not quite a quarter of the girls had started smoking but had given it up. In the fourteen-to-sixteen age group, twenty-seven per cent of the boys and thirty per cent of the girls smoked regularly, and almost half the boys and eight per cent of the girls smoked five or more cigarettes a day. Children of poorer parents smoked more than

children from wealthier families, and children who had lost a parent or who came from broken homes smoked most. "I never expected that we would show the difference in children between smokers and non-smokers and light and heavy smokers as clearly as we have," Dr. Holland commented as he read from his reports to a visitor. He still looked a trifle surprised by his findings. "Obviously, these children can't have been smoking very long, so the effect on their respiratory systems was produced over a very short period of time." Dr. Holland will next check the results, if any, that the extensive British propaganda campaign against smoking and the ban on television cigarette advertising are having on the statistics of the child smokers. "We are really trying to see if we can identify individuals who are particularly susceptible to disease and prevent its development," Dr. Holland explained. "Once it starts, there's nothing you can do about it. In any case, our findings about smoking certainly show that we can't single out air pollution as the one villain."

Among the many things that nobody knows about air pollution is exactly how many pollutants are dangerous. After the 1952 catastrophe, one pollutant that the British realized must be removed as fast as possible was the black smoke that poured from the millions of chimney pots on the roofs of their island's towns, where millions of small open fireplaces were burning soft coal. The almost unbelievable effort to change, in a few years, the habits of centuries in a democratic country is some sort of monument to human intelligence. It began in 1956, with the Clean Air Act, which established national jurisdiction over all domestic and industrial smoke and gave local authorities the power to create smoke-control areas. Since then, each local authority has been encouraged to draw up a program allowing everyone plenty of time

to convert from soft coal to natural gas, to electric heating, or to fuels manufactured from coal that have had the volatile, or "tarry-smoke," matter removed. (Oil is classified as "unauthorized fuel" but may be used for home heating if the local authorities approve.) The conversion from coal usually costs about twenty-five pounds, or sixty dollars, and when it has been completed and has been approved by the local public-health inspector, who is a very important man in an English town, seventy per cent of the expense is paid back to the homeowner by the local authority, which is, in turn, reimbursed for more than half of the amount by the central government. Because the old-fashioned coal-burning open fire is one of the most inefficient forms of heating known to man—about eighty per cent of the fuel is wasted—the conversions represent a sizable financial saving to homeowners in the long run. (Coal miners, however, who get some of their coal free and the rest at very low cost, don't like switching to another fuel, and are a very sticky problem.)

While the provisions for enforcement of the Clean Air Act include a threat of court action, gentle persuasion, locally and nationally, has worked wonders. The government periodically publishes lists of areas that have taken little or no action in conversion from coal, and invariably there is a sharp increase in activity shortly afterward. Initially, the regions with the heaviest smoke pollution were labelled Black Areas, and target dates for achieving smoke-free zones were generally set in the nineteen-eighties. But when word began to get around that with smoke control the sun seemed to be shining more often, the program gained momentum, and now many target dates have been advanced into the nineteen-seventies. Although coal still accounts for almost all of Britain's electric-power production, and a soot-free

Britain is still far in the future, the phasing out of coal for the heating of individual homes has been further hastened by the building of many new apartment buildings with efficient central-heating plants. Perhaps the greatest miracle is what has happened in London. Greater London leads the country in smoke control, with almost sixty per cent of its area smoke-free, and with emissions and ground-level concentrations of smoke about a third of what they were ten years ago. The famous square mile that is the old City of London is nearly free of smoke, and lately London, aware that they won't quickly get dirty again, has begun to wash the faces of its public buildings, revealing handsome stone exteriors that look gleaming white to those who remember the old blackness.

To epidemiologists, the most significant fact about London's clean-up is that during smog episodes there is now much less of an increase in sickness and death. In 1959 and 1960, in an effort to determine the effects of the Clean Air Act on human health, the government's Air Pollution Research scientists, who work under the direction of Dr. Patrick Lawther, handed out little pocket diaries to a thousand bronchitis patients in London and asked them to make a one-word notation every day for one winter, stating merely whether they felt better or worse than they felt the day before, or just the same. There turned out to be an extremely close correlation between the reports of feeling worse on certain days and high levels of pollution in the air. When the study was repeated five years later, this correlation was much less marked. Mr. Robert Waller, Dr. Lawther's chief colleague, who helped conduct the two studies and expects to conduct another in 1969 and 1970, is a cautious man, who hesitates to say whether this improvement can be attributed to better air or to the patients' increased knowledge of how to care

for themselves, but he does permit himself to describe the findings as "not bad," and even "encouraging." He is also pleased by the fact that there has been a decline in hospital admissions for diseases commonly associated with air pollution.

A visitor to London can actually observe the contrast between air that has been cleaned up and air that is still polluted. From the roof of the Town Hall of the Borough of Southwark, south of the Thames in the center of Greater London, he can look out on a smoke-free zone to the south, where the air is clear, and then, turning slightly to the east, he can see a yet-to-be converted Black Area, distinctly marked by low puffs of smoke rising from rows of chimney pots. To the north, beside the Thames, he can also see a great white plume from the smokestack of Bankside Power Station. London's Bankside and Battersea stations, the former oil-burning, the latter coal-burning, are the only large power plants in the world where sulphur dioxide is literally washed out. Water from the Thames, to which an alkali—common chalk—has been added, is used to wash the gases before they are discharged, and in the resulting chemical reaction the sulphur dioxide in the smoke is turned into calcium sulphate, in solid particles, which are discharged, with the water, into the river. The process requires huge quantities of water, and there is a distinct limit to the amount of calcium sulphate that even as large a river as the Thames can carry away safely, so no more such installations will be built on this stretch of the river. Meanwhile, the Central Electricity Generating Board is experimenting with towering smokestacks, one of them more than eight hundred feet high, to dissipate the emissions over as wide an area as possible. The big stacks have helped to prevent any increase in ground-level concentrations of sulphur dioxide despite a substantial increase in

fuel consumption, and at some sites outside London there has even been a decrease.

A National Survey of Smoke and Sulphur Dioxide has been conducted throughout England and Wales by the government's laboratory at Warren Spring, an hour's drive north of London. For the past five and a half years, under the supervision of an immensely energetic physician named Dr. Marjorie Clifton, the staff has been evaluating reams of statistics that have poured in daily from six hundred locations throughout the country. Although the survey's intensive-sampling period has ended, regular measurements will continue to come in from key sites, and Dr. Clifton hopes, with the aid of a giant computer, to predict levels of pollution in British communities in time for them to take steps to avert disasters.

Back in London, the Air Pollution Research Unit is based at the Medical College of St. Bartholomew's Hospital, one of the world's great medical-instruction centers and the city's oldest hospital, dating back to the twelfth century. Among ancient buildings surrounding a grassy courtyard, where medical students play croquet on sunny days, the Unit works in a set of extremely modern laboratories behind a set of double doors over which an enormous "No Smoking" sign is mounted. Here, Dr. Lawther devises endless experiments—to be conducted on himself, on his staff, and on the people of London in general. The rooms include physics, chemistry, and physiology laboratories, and a workshop where a trained mechanic constructs and repairs delicate equipment for monitoring air and for measuring human respiratory reactions. Two prominent features of the physiology laboratory are a low green leather couch, where human subjects can lie down while they breathe carbon monoxide into their systems, and a glass-walled smoke chamber—a room inside a room—

which contains a desk and chairs, where one or two people can sit and work while they inhale smoke released through a blackened overhead vent from a small iron coal stove nearby. The predominating spirit around the place, one of quiet efficiency, is often jolted by the wild figure of the director, who, his laboratory smock flapping and his prematurely white hair in disarray, rushes through on the run between meetings, clinic hours, and speeches on the dangers of air pollution. Dr. Lawther is disturbed by the thought that a great deal of money is being spent around the world for what he regards as very bad air-pollution research when free co-operation is readily available from any sensible person who wants to breathe clean air. All London is Dr. Lawther's outdoor laboratory, and he and his staff have analyzed the street-level air pollution in places like busy Fleet Street, where they have shown that during maximum-traffic hours on weekdays the air in the middle of the street contains three times as much smoke and four times as much lead as the air in London's quieter neighborhoods, and also that carbon monoxide periodically reaches the maximum acceptable limit for industrial plants—one hundred parts per million units of air.

Dr. Lawther's favorite experimental animal is himself, and next is his colleague Waller, who is particularly sensitive to sulphur dioxide. When the sulphur dioxide in the air reaches ten parts per million, Waller wheezes audibly, like an asthmatic, in a consistently severe reaction. Dr. Lawther also uses other members of his staff for "double-blind" experiments, in which neither the subject nor the technician knows exactly what is going on. If the subject knew what substances he was breathing, Dr. Lawther believes, there could be a psychological reaction that would affect his respiration, and if the technician knew what substances he was administering, he might un-

wittingly weight his findings. The experiments are fitted in with the daily routine, and it is not uncommon for a staff member to interrupt a conversation in the middle of a sentence, look at his watch, disappear without a word, and reappear a few minutes later to resume speaking as if nothing had happened. Every morning, Dr. Lawther and Waller, who live in the same borough, take trains fifteen minutes apart to London Bridge, cross it, and walk for twenty minutes through the same city streets to the laboratory, Dr. Lawther arriving at nine-fifteen and Waller at nine-thirty. The object is to measure the effect of air pollution on the breathing of individuals who have just walked through an ordinary city street. Immediately after his arrival, each man enters a booth-like transparent-plastic chamber called a body plethysmograph, which is equipped to measure the respiration of its occupant. One morning some weeks ago, a visitor watched the two men follow each other into the plethysmograph, and they presented an interesting contrast. Dr. Lawther, who had already put on his white working coat, rushed into the chamber, sat down on a stool, and, even before a girl technician could close the door, began to read mail from a manila folder he was carrying. The Doctor continued to read from the folder as he waited briefly to acclimatize himself, put on a nose-clip so that he would be breathing entirely through his mouth, and took hold of a mouthpiece, through which he breathed ten times. He was still reading as the technician opened the door and he departed. Waller arrived on the dot of nine-thirty, took off his jacket, and entered the plethysmograph in his shirtsleeves, sat down on the stool with his heels caught in the lower rungs so that his long legs were out of the way, and folded his hands in his lap. He waited patiently until it was time to start breathing into the machine, then adjusted the nose-

clip and carefully breathed into the mouthpiece. When he came out of the box, he told the watching visitor that in Dr. Lawther's case the results of the daily tests had shown a relationship between his "airway resistance," or difficulty in breathing, and the concentration of pollutants in the outdoor atmosphere, his airway resistance increasing when the pollution was higher than average. "In my case, the association with air pollution is not striking at all—unless the sulphur dioxide level is particularly high," Waller continued. "I am a more variable creature, and my airway resistance changes very readily, in response to all kinds of things. A long series of measurements made with a portable device called a Peak Flow Meter have shown that colds, emotional disturbances, pretty girls, and situations of undue stress such as driving in central-London traffic or having to give a lecture affect the results. Some people breathe more easily when a little excitement releases adrenalin or some other substance that reduces any bronchial spasm that exists. We have found that exercise doesn't seem to affect us particularly. We've tried long runs through the city streets and long swims in a pool. Provided the air pollution was not exceptionally high, these activities didn't have any effect. Running during periods of high pollution did."

One of the Unit's main findings has been that the effects of pollution on breathing wear off just minutes or seconds after the subject removes himself from the polluted atmosphere. "You may wonder whether the pollution really matters," Waller said. "But if the subject stays in the pollution, it doesn't wear off." One reaction that the Air Pollution Research Unit watches for is a bronchospasm—a contraction of the airways to the lungs when certain substances are breathed. The problem, Waller said, is to identify the substances precisely. "It's rather

easy to see what *doesn't* affect us but hard to identify what does, and although we have this glorious mixture of air pollutants in London, we're not sure which components we're after," he explained. "We do have abundant evidence in our epidemiological studies that the sharp peaks of mortality and hospital morbidity that we see on our charts are caused by air pollution of some kind, rather than just by unusual weather conditions. But when you try to tackle *which* pollutant it is, you are really in deep water. Our studies have indicated that although there are members of the population who, like me, are very sensitive to sulphur dioxide, the majority do not react to low concentrations of it."

Recently, the Unit has been studying the possible effects on human behavior of exposure to carbon monoxide in quantities so small that it induces no perceptible symptoms. When carbon monoxide is inhaled, it attaches itself more readily to the red blood cells than oxygen does, and detaches itself less readily, and while it rides around through the bloodstream it reduces the blood's ability to absorb oxygen. Temporarily, anyone with carbon monoxide in his blood becomes slightly anemic. It has been established that when we breathe air containing as much as a thousand parts of carbon monoxide per million our mental processes and nervous system are seriously affected and we suffer impairment of vision and severe headache, but many people believe that at lower levels our efficiency may be reduced somewhat. In busy streets, drivers and others may be exposed to concentrations in the range of ten to one hundred parts of carbon monoxide per million, and while nobody knows whether these relatively low concentrations have any effect, everybody is beginning to wonder. Dr. Brian Commins, who heads Dr. Lawther's chemistry laboratory, has recently perfected a new technique for

making carbon-monoxide measurements from a blood sample as small as that from a pricked finger. (A syringe of blood used to be needed.) The Unit scientists are now taking samples of their own blood after they walk through busy streets or as they drive their cars. One fact that has emerged is that the amount of carbon monoxide breathed during exposure to traffic fumes is less than the amount breathed while smoking a cigarette. Dr. Commins and other experimenters are wondering whether a person who smokes while he is driving may not expose himself to sufficient carbon monoxide to suffer minor behavioral distortions.

Some of the answers to the questions being asked at London's Air Pollution Research Unit may be coming before long from a toxicology laboratory at the Harvard University School of Public Health, in Boston. There, eighteen years ago, a young biochemist named Dr. Mary Amdur began inquiring into the toxicity of sulphuric-acid mist by exposing guinea pigs to it in different doses and combinations. Sulphur dioxide is the air pollutant we hear most about, because it is easy to measure and the amount of it in the air tells the experts that a lot of other pollutants are there, too, but laboratory experiments in which both animals and human beings have been exposed to doses of sulphur dioxide by itself, administered in the amounts found in the ambient air, have seemed to show that it's harmless except to the small percentage of sensitive people like Waller. Yet, especially during smoggy periods, something injurious to human lungs is in the air, and people die. The key to the puzzle probably lies in the fact that sulphur dioxide is an unstable compound. It combines with other substances, and, at a rate depending on atmospheric conditions, it oxidizes into sulphuric-acid mist. There is always some sulphuric-acid mist around wherever sulphur

dioxide is produced, even in a clear, dry climate, but the conversion to sulphuric-acid mist is much greater in regions or periods of high humidity. When scientists talk about sulphur dioxide as a hazard, they really mean that the hazard is probably sulphuric-acid mist.

Dr. Amdur, a small, willowy woman who is now associate professor of toxicology in the school's Department of Physiology, became interested in sulphuric-acid mist when she and some of her co-workers at Harvard began studying its toxicity after the 1948 Donora smog episode. She told a visitor recently, "We began by exposing guinea pigs for eight hours to various concentrations of sulphuric-acid mist to find what toxicologists call the L.D. 50, meaning the lethal dose fifty per cent—the point at which you kill fifty per cent of the animals. You start there as a standard procedure with any new chemical, and then you examine the pathology—the nature and extent of the fatal organic damage. The next step is to find out what level produces such damage but doesn't kill. Finally, you look at the subtle biological changes produced when you neither kill the animal nor seriously damage its lungs—changes that would occur in industrial or air-pollution situations."

Dr. Amdur explained that she works with guinea pigs because their sensitivity to sulphuric-acid mist, especially in the low concentrations that exist in our air, is higher than that of rats or rabbits, and their reactions to it are not dissimilar to those shown by human beings in far more limited experiments. "Rats can breathe huge quantities of the mist without batting an eyelash, and rabbits are quite happy with amounts that would do a guinea pig in," she said. When irritants are introduced into the atmosphere that the guinea pig breathes, its bronchial tubes constrict. This means that more effort is required for it to breathe,

which could help to explain the extra deaths—especially among infants, the sick, and the aged—during air-pollution episodes. Any categorical statement of a possible association between such animal experiments and human experience is, however, considered by Dr. Amdur and her colleagues unwarranted extrapolation.

One finding that has particularly interested Dr. Amdur is that her animals' airway resistance increased only slightly when they inhaled sulphur dioxide by itself but increased by four hundred per cent when the sulphur dioxide was combined with sulphuric-acid mist or with droplets of water containing small particles of sulphates of vanadium, iron, and manganese. This appears to be a classic example of a synergistic effect—one in which a combination of substances produces a greater effect than could have been expected on the basis of their individual effects. Dr. Amdur also noticed such a synergistic effect when she combined sulphur dioxide with zinc-ammonium sulphate—a compound that had been found on the filter of an air-conditioner during the Donora fog. Dr. Amdur believes that water droplets are essential to this synergism, and fogs, of course, provide plenty of droplets.

Evidence from several other laboratories has suggested that microscopic particles may elude our protective mechanisms and, acting not only as irritants themselves but as carriers of other irritants, invade and lodge in the lower lungs, where the protection is haphazard compared to the wonderful action of the cilia and mucus in the conducting airways. Dr. Amdur has found that when she gives her guinea pigs a dose of irritant particles, the animals' breathing returns to normal more slowly than it does when they have been exposed to irritant gases, and their recovery is slower still when they have received a combination of both particles and gases. Dr. Amdur does

not know what happens to the particles in her guinea pigs' lungs, but she has lately discovered that the irritant responses, or airway resistances, increase as the particles' size decreases. She has used particles as small as three-tenths of a micron—a micron is a thousandth of a millimetre—and plans to use even smaller ones as soon as she figures out the mechanics of producing and measuring them.

Although Dr. Amdur's experiments have focussed on acute air-pollution episodes, in which her guinea pigs are exposed to irritants for definite periods rather than continuously, she is one of the few experts willing to stick their necks out and try to help set some standards for sulphur-dioxide levels in the ambient air. In 1959, California health officials who wanted to set such standards asked her for a report. After carefully describing the relative toxicities of sulphuric acid and sulphur dioxide alone, as well as the factors of particle size and synergism, she suggested some tentative numerical standards—although California finally adopted more conservative ones. "Someone had to be first to give some numbers," she said. "At least, we've got data to use as a basis for thinking about air-pollution criteria. Certainly it seems evident that we cannot judge entirely on the basis of sulphur dioxide. The coal and power people have tried frantically to say that sulphur dioxide does no harm, but I've pointed out that this is deliberately avoiding the issue, because I don't think synergism can be dismissed. I know it's cheaper to pollute the air, but do we have to put the burden of irrefutable proof on the Public Health Service that somebody killed Grandma before they are allowed to set up controls?"

In the spring of 1967, in fact, the United States Public Health Service, in an act of sheer heroism, published a fat document entitled "Air Quality Criteria for

Sulphur Oxides," giving the levels at which its scientists believed that sulphur compounds "begin to harm our health and foul our environment," and expressing the hope that these criteria would "set the levels we must aim for in our drive for clean, breathable air." Outdoor conditions differ from laboratory conditions in that sulphur dioxide is always present with particulate matter and sulphuric-acid droplets; experiments like Dr. Amdur's have shown that sulphur dioxide is considerably more toxic when accompanied by other such pollutants. Therefore, the government criteria, which are based on epidemiological studies of actual situations in large cities such as Nashville, suggest much lower acceptable levels of sulphur dioxide than might be expected—less than one-tenth part sulphur dioxide per million parts of air as a daily average. The regulations for the control of sulphur-dioxide emissions from federal buildings are based on comparable levels. At the first hints of national standard-setting—although no actual standards had been drawn up—the anticipated howls were heard from a wide range of industries and their spokesmen, since sulphur compounds are produced in very many basic-industrial processes. The fight has just begun, but the American Petroleum Institute, the Edison Electric Institute, the National Coal Association, the National Coal Policy Conference, and the United Mine Workers have expressed their displeasure with the Public Health Service recommendations in the sharpest possible terms.

Rigid controls may very well have to be set up before we know how, and how much, air pollutants harm us, but sometimes facts do fall into place. A classic case occurred not long ago in Reading, Pennsylvania, where a smelter was refining the metal beryllium, which is used in various alloys. Beryllium is well known to be toxic to human beings who are exposed to it at

high levels, for then it causes berylliosis, a chronic and debilitating lung disease in which the sufferer simply wastes away. An argument had been going on intermittently through the nineteen-forties over whether smokestack emissions from processes in which beryllium was involved were toxic at the relatively low levels that prevailed when the fumes were diffused in the atmosphere. On the basis of small-scale experiments with animals, the United States Public Health Service at first put no controls on emissions of beryllium, but an accumulation of alarming evidence finally caused the Atomic Energy Commission, which used the metal in reactors and was therefore a large purchaser, to notify its contractors that in the future their factories would have to meet certain safety standards. The smelter in Reading refused to comply, and although it received no more contracts from the Atomic Energy Commission, it continued to produce beryllium for private industry. The Pennsylvania health authorities went on record as saying that on the basis of the federal data available there was no danger. But by 1959 twenty-five cases of berylliosis had been reported around Reading, and all concerned were forced to change their minds. A number of victims have sued the company, and one has been awarded damages of more than a hundred thousand dollars. The Pennsylvania health authorities have tightened their standards for safeguards to be used in refining beryllium, and the Reading plant has complied. The Reading berylliosis cases are particularly interesting to air-pollution experts because they show that even supposedly low-level emissions of a known toxic pollutant can be exceedingly dangerous.

The techniques of working with particles, developed by Dr. Amdur in Boston and Dr. Lawther and his staff in London, among others, have been further developed at

New York University's Institute of Environmental Medicine, which has a laboratory in Sterling Forest, a planned recreational, educational, and residential community in the foothills of the Ramapos, an hour from New York City. There, Dr. Roy Albert uses iron-oxide particles of uniform size and puts a minute radioactive tag on them that gives off gamma rays, enabling a detector to follow their journey through the chest of an experimental animal and measure the factors that govern the body's rejection or retention of them. He has found that some particles move through the lungs and into the stomach in from two to four hours, others remain in the lungs for many weeks, and those that penetrate beyond the bronchial tubes into the alveoli stay longest of all. Dr. Albert, who also conducts experiments on human beings in the city, works at Sterling Forest with donkeys—eleven of them. One evening about two years ago, he happened to be watching a TV commercial that featured a little old winemaker and a donkey; after observing the docility of the donkey under somewhat trying circumstances, the Doctor bought one for the Institute—a female named Abby. He later bought two more females, and then he picked up Anthony, a male, which was so much easier to handle that the Institute bought seven more males. Donkeys have about the same weight and lung size as humans (although their lungs seem to clear out impurities more rapidly), but their special virtue as laboratory animals is their ability to stand still for hours without sedation. Dr. Albert has devised a mask that resembles a feed bag with a hose on each side, through which the donkeys breathe a special mixture of radiated particles of a certain size and concentration. Detectors attached to the donkeys' sides follow the course of the particles in their chests and abdomens. In one experimental session, after

half-hour exposures to sulphur dioxide at from six to seven hundred parts per million—relatively large doses—during which mucus poured from the animals' noses and eyes, the donkeys developed coughs that lasted several days and persistent defects in lung clearance.

In the same laboratory where the donkeys are tested are rows of steel-and-glass chambers containing albino rats, which are handled by means of rubber gloves built into trapdoors, so that no bare hand ever has to be put inside the cages. Here, Dr. Norton Nelson has been testing the cancer-inducing qualities of a worrisome compound that scientists have christened benzo(a)pyrene, which is produced in city air by the inefficient combustion of fuel—particularly automobile fuel—and is also found in cigarette smoke. Thirty rats have been continuously exposed to what Dr. Nelson refers to as "pure mountain air"—the regular outdoor air of Sterling Forest—and thirty others to "contaminated air," containing heavy concentrations of sulphur dioxide. Both groups of rats have also been exposed, for an hour a day five days a week, to benzo(a)pyrene dust. The question is: Does the sulphur dioxide increase the carcinogenic effect of the benzo(a)pyrene sufficiently to give the animals lung cancer? The animals were put into the chambers when they were six weeks old, and both groups remained healthy throughout the first year. But at fifteen months one of the rats in the "city" air developed cancer, and there have since been two more cases—three out of thirty, or ten per cent. Dr. Nelson recently explained, "The inhalation of benzo(a)pyrene without sulphur dioxide failed to produce lung cancer, even though the concentration was raised to the point at which skin cancer was produced. Furthermore, the lung cancer that has been produced in the current experiments is of the type most frequently found in man."

At the Sloan-Kettering Institute for Cancer Research, in New York, Dr. Ernest Wynder and Dr. Dietrich Hoffmann, who pioneered in investigating the role of cigarettes in lung cancer, have collected particulate matter from polluted air in Detroit, Los Angeles, and the New York area for tests on experimental animals. Their samples, which are in solution form, are bottles of ugly-looking dark-brown liquid labelled according to place of origin, and they represent every kind of combustion emission, collected in various commercial areas and at busy street intersections. These are pure extracts of poison, and are, of course, much more toxic than the same substances diffused in the atmosphere. Dr. Wynder and Dr. Hoffmann have applied these liquids—or "tar extracts"—in very high concentrations to the skins of mice and in a good many cases eventually produced tumors. The most carcinogenic tar was from Detroit, the least toxic was from Scarsdale. Despite these experiments, the two doctors do not suggest that there is any major correlation between air pollution and lung cancer. They remain convinced that cigarettes are the major cause of the disease. "The final proof can come only from epidemiological data," Dr. Wynder says. "I am sure we are surrounded by carcinogens in what we breathe and eat, but most of these are in minute amounts that we can handle reasonably well. I think our lungs can handle reasonably well whatever we inhale in cities. But cigarettes bypass the protective mechanism of the nasal passage and overwhelm our lungs with smoke."

Dr. Wynder and Dr. Hoffmann have carried out several comparative studies of smoking and air pollution as factors in the production of lung cancer. The benzene extract of a gasoline-engine exhaust produces more skin tumors in mice than tobacco does, but Dr. Wynder warns that this does not necessarily mean that the ex-

haust gases will produce cancer in man. The exhaust gases, he points out, are diluted several thousand times in the air before they reach the lungs, whereas the lungs are directly exposed to undiluted tobacco smoke. One of the effects of tobacco smoke that has been demonstrated by the Wynder-Hoffmann experiments and others is a slowing down or cessation of the beating movement of the cilia and the flow of mucus—both vital guardians of the lungs.

Dr. Wynder's firm belief that air pollution plays a very minor role in lung cancer is reinforced by epidemiological findings. He cites the fact that lung cancer in the United States is six times as prevalent among men as among women although both sexes are similarly exposed to general air pollution. A possible explanation of this disparity is the longer record of heavy-smoking habits among men. In Los Angeles, Dr. Wynder made a study of Seventh-Day Adventists, whose religion does not permit smoking or drinking, and found them to have only ten per cent as much lung cancer as the rest of the population in that polluted city. This ratio also applied to cancer of the mouth, the larynx, and the esophagus—occupational hazards for those who make a life-work of heavy drinking—but all other cancers occurred among Seventh-Day Adventists in Los Angeles with the same frequency as they did among the general population. A more recent California study, made by state health officials, has further confirmed Dr. Wynder's beliefs. This report, based on data from various parts of the state, indicates that whereas smokers have five times as much chance of dying from lung cancer as non-smokers, when allowances are made for smoking habits, age, and length of residence, fewer people generally die of lung cancer in smoggy Los Angeles than in the relatively cleaner air of San Francisco and San Diego. In Italy, with

two Italian doctors, Dr. Wynder himself examined the population of a city peculiarly free from general air pollution—the city of Venice, where there are neither automobiles nor any large manufacturing establishments, except some glass factories on a nearby island. In Venice, lung cancer is the most common cause of death among men but is not at all common among women—a reflection of the national smoking pattern in Italy, where more than half the men over sixteen smoke cigarettes regularly and ninety per cent of the women over sixteen don't smoke at all. In both Italy and the United States, and elsewhere, too, the rate of lung cancer is higher among city dwellers generally, a condition that Dr. Wynder attributes to a number of factors—that people living in cities tend to smoke more, that sufferers from lung cancer often move to a city for treatment shortly before they die, that cancer is more commonly reported in cities, and that occupations common in cities expose workers directly to particles that may contribute to the development of cancer, such as metal and wood dust and particles of paint, asbestos, and chromate. The fact that there is more than twice as much lung cancer among men in Great Britain as there is among men in the United States Dr. Wynder attributes primarily to national differences in smoking habits and preferences. The British take more puffs per cigarette and also smoke a cigarette farther down—and the butt of a cigarette is known to contain more smoke condensate. Also, the British prefer cigarettes made of tobacco that is flue-cured and that experiments have shown to be more carcinogenic than air-cured tobacco, which is used in American cigarettes.

Whatever the relative roles of smoking and general air pollution may be, there are unquestionably increasing amounts of very toxic substances floating about. High on

the list are asbestos and lead. Smokers working where they are exposed to asbestos dust have a rate of lung cancer eight times that of the general population, according to Dr. Cuyler Hammond, director of the American Cancer Society. Asbestos is used for brake linings, and some asbestos is given off every time the brakes are applied on an automobile. Although there is no evidence that inhalation of such fractional amounts hurts anybody, Dr. Hammond has written, "With the rapid growth of asbestos utilization (the five hundred thousand tons per year world production in 1930 has risen to four million tons per year now), it may be difficult for cigarette smokers to avoid inhaling air contaminated with asbestos." Lead, too, is under close scrutiny. It has always been one of the "body burdens"—a favorite scientific phrase—that all of us carry around with us, because it settles in our bones. Our supplies of food and water contain twenty times as much lead as they did in primitive days, but, as far as anybody knows, our intake of lead is still well below that associated with lead poisoning. However, in the last few decades the levels of lead not only in our food and water but in our air have been rising so steadily that scientists are beginning to wonder if this added lead burden may be contributing to some unidentified illnesses, or even to diseases we know about already. Some experts believe that the whole toxicology of lead needs reevaluation. Almost all gasolines are now leaded, and the lead is emitted from car exhausts in very fine particles, which the gasoline and lead companies would very much like to have us believe are harmless. Who knows? Nobody.

Air pollution has been a matter of official concern to the federal government only since 1955, when Congress gave the United States Public Health Service a small appropriation for systematic research on the subject. In the years imme-

diately after that, most of the research was done under contract in non-government laboratories, although work that had been started in what was formerly called the Sanitary Engineering Center, in Cincinnati, was continued. One of the more important experiments was conducted in California by Dr. Leslie Chambers, who installed a colony of mice right next to a Los Angeles freeway and concluded that lung cells destroyed by the prevalent smog could be regenerated by younger mice but not by older ones. In 1960, the Public Health Service created a Division of Air Pollution to supervise its medical and engineering programs. In 1967, the Division was reorganized and named the National Center for Air Pollution Control, with headquarters in Washington and laboratories in Cincinnati; Durham, North Carolina; and Ypsilanti, Michigan.

In Cincinnati, two six-cylinder automobile engines have been set up to run so that they simulate the average pattern of everyday driving, and pipes collect the exhaust gas, some of which is inhaled directly by experimental animals and some of which is piped into two huge steel boxes, each more than six hundred cubic feet in volume. Here, the gas is irradiated with light to produce the sort of photochemical smog that was once believed to be distinctive to Los Angeles but is now seen increasingly in other traffic-clogged cities. A whiff of this synthetic smog from a briefly opened valve has the sickening smell of the exhaust fumes from an old bus. One of the scientists conducting experiments with this synthetic smog is a veterinarian, Dr. David Coffin, who is chief of the Experimental Pathology Unit at the Cincinnati laboratory, and whose special interest is the interaction of air pollutants and infectious bacteria. Dr. Coffin has observed that animals that have been exposed to various pollutants are more susceptible to bacteria

than animals that have breathed normal air. When, for example, mice have been exposed first to ozone—a common pollutant from auto exhausts that is toxic to human beings—and then to streptococcus-pneumonia organisms, they have been more likely to develop pneumonia. Dr. Coffin has also learned from postmortems that animals exposed to bacteria but not to polluted air had no streptococci left in their lungs about six hours after exposure, whereas animals exposed to the combination of smog and bacteria eliminated the bacteria so slowly that the streptococci had a chance to grow and infect the lungs.

In another section of the Cincinnati laboratory, a biochemist named Dr. F. G. Hueter has since 1962 been exposing rodents to raw tail-pipe exhaust diluted with clean air in various proportions and to different concentrations of photochemical smog. Dr. Hueter has observed that guinea pigs that inhale smog are more susceptible to pulmonary infections and pneumonia, and that mice breathing photochemical smog exhibit signs of chronic disease (reflected in an elevated white-blood-cell count) after eighteen months of exposure in the latter third of their life-span. A group of mice exposed to irradiated exhaust during their fertile period had lower fertility rates—fewer females had litters, those that did had them less frequently—and the rate of survival of baby mice between the first and the tenth days was markedly reduced. Rats exposed either to raw exhaust or to smog developed abnormal, non-functioning lung tissue, which was not present in control animals living in clean air. Finally, mice exposed to either raw exhaust or irradiated air were less active for a period of time but then adapted themselves to the mixture and resumed their normal behavior. The mice exposed to the raw exhaust made the adaptation in twelve days, but it took the mice who inhaled

irradiated air twenty-four days to readjust themselves. In a new series of experiments, Dr. Hueter is going to study the effects on monkeys of inhaled lead from gasoline additives, and of systematic exposure to tail-pipe exhausts (irradiated and non-irradiated), to atmospheres containing sulphur dioxide and sulphuric-acid mist like those produced by heating and power plants, and to mixtures of nitrogen compounds. The laboratory is also studying the effects on eighty-six beagles of low levels of the sort of pollution that is regularly found in the average city air.

The trio of ailments in which general air pollution is believed to play at least an aggravating role—bronchitis, emphysema, and lung cancer—do not promise such obvious clues as, for instance, the Reading berylliosis cases. Nobody is sure at the moment quite how to evaluate the fact that nitrogen dioxide, which, like sulphur dioxide, is always present in our urban atmosphere and at low levels does not, as far as is known, affect human beings, has produced emphysema-like enlargements of the air sacs in the lungs of rats that have inhaled it at low levels over a fairly long period in a California laboratory. High concentrations of nitrogen dioxide to which human beings have been exposed accidentally—for example, in fires involving X-ray film—have caused pneumonia and death. Both cigarette smoke and automobile exhaust contain several hundred parts of nitrogen dioxide per million—concentrations that would be fatal in a continuous exposure. Nitrogen oxides are among the principal compounds earmarked for further research, particularly because they are employed in many new chemical processes and in secret gasoline additives of unknown toxicity.

Dogs, rats, mice, guinea pigs, donkeys, men, women, and children—a whole world breathing, and nobody knows exactly what we are breathing or exactly how it af-

fects us. Dr. Robert Horton, an epidemiologist who was formerly a professor at the University of Michigan and now presides, from a desk in Cincinnati, over the government's Health Effects Research Program, spent several hours discussing the progress of air-pollution research with a caller not long ago, and then, in a matter-of-fact voice, said, "The British reduced cholera and typhoid in the nineteenth century before they knew bacteria existed, and we may have to regulate our air supply before we have complete knowledge about air pollution. The methods we have for detecting excess deaths are so crude that there has to be a pretty big excess for us to realize that it's there at all. What we *do* know is that people get killed by air pollution, and I don't see any excuse for there being enough air pollution to kill people. Do you?"