



POLLUTED AIR— AND HEALTH

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A research investigator states that while air pollution is rarely, if ever, a sole and direct cause of disease, air pollutants—acting together—may intensify disease.

■ IN 1948 DONORA, PA., was a highly industrialized town of 14,000 people situated in a bend of the Monongahela River valley. Smoke and fumes poured from its industry, producing an atmosphere that was dirty and malodorous but which was not known to cause illness.

The disaster that occurred in October of 1948 in that city is now famous. Although the calamity at Donora was not the first or last air pollution disaster, many things about it are common to all others, and it provides the most concrete and inescapable evidence that under certain circumstances, uncontrolled air pollution can cause illness and death.

On Monday, October 25, a stable layer of air formed in the valley. From then until the smog was broken up by rain on Sunday, the 31st of October, this layer acted like a lid clamped down on the valley to prevent dispersal of pollutants. Two days after the episode started, the air was thick and heavy and visibility was markedly reduced.

Although a few persons started to feel ill on the second day, it was on the third day that large numbers became affected. Just as in other similar disasters, shortness of breath and cough were the most prominent symptoms, although some people experienced sore throat, headache, tears in the eyes, nausea and even vomiting. Between 8 p.m. and midnight of the third day, 40 per cent of the affected persons reported the onset of their illness. By the fourth day almost everybody who became ill during the episode was already sick. On the fourth day, 17 of the total of 20 deaths occurred. In all, 5,910 persons or 42.7 per cent of the population were affected to some degree by the smog.

As in all other disasters of this type, the elderly and those with pre-existing heart and lung disease were the most severely afflicted, although healthy persons and people in all age groups were affected.

Conditions that cause it not unique

The basic requirement for an air pollution episode—the concentration of numerous and active sources of smoke and gases—is not unique to Donora or to the Meuse Valley or London where similar disasters have occurred.

This requirement is met by many of our cities and

industrial areas. The freak of weather that is necessary to produce an episode cannot be prevented, although it can be predicted. Episodes of markedly increased air pollution with concentrations of gases as high as those thought to have been present in Donora and London have occurred in New York City, although there is little or no evidence of an associated increase in illness.

The symptoms of tears in the eyes, nasal discharge, sore throat, and cough, observed in the episodes, are the symptoms of irritation of the mucus membranes; probably a non-specific irritating effect of a large number of substances rather than the effect of a specific poison such as carbon monoxide. The tightness in the chest and the shortness of breath are also—indirectly at any rate—the result of irritation of the respiratory tree. They are produced by a narrowing of the bronchial passages with consequent increased airway resistance. One might easily deduce that people whose airway resistance is already increased by disease or whose pulmonary reserve is already impaired would have an increased incidence and severity of illness during the episode. This certainly fits the Donora pattern and the pattern seen in virtually every other air pollution disaster.

Therefore, our tentative conclusions from examining the “clinical cases” of the air pollution episodes are that there are noxious substances in the air that cause irritation of the mucus membranes and increased airway resistance.

A great deal of research has been done in the years since 1948 that bears on these conclusions and supports them, but fails to amplify them as much as one would like.

Pollutants that affect health

A number of irritant and potentially toxic substances have been identified, studied, and generally found to be noxious agents. These include substances secondary to industrial processes, emissions from automobiles, waste products from home heating, and many other secondary products of modern urban life.

However, to assume that the substances already identified represent the complete list of agents in urban air that may be dangerous or potentially toxic would be naive. We are limited in our knowledge of the health effects of air pollution by our knowledge of the substances in the air. New substances are continually being found.

Air pollution legislation directed solely at specific substances runs the hazard of becoming obsolete and failing to protect people from other toxic and irritating substances that have not yet been identified.

The experimental monitoring station set up in New York City's Lower East Side in conjunction with the Cornell studies on Air Pollution and Family Illness has produced some precise information about pollutants.

Of the pollutants generally indicted for their effect on health, sulfur dioxide has been studied most extensively. The Cornell monitoring station in New York shows that the average values for sulfur dioxide are in the neighborhood of .15 to .2 parts per million (p.p.m.). This level is considered quite high—higher than expected and easily as high as that of London or any other American city.

One group of investigators has reported no change in pulmonary air flow resistance in man following exposure to sulfur dioxide in the range of 1 to 3 p.p.m. At 4 to 5 p.p.m. a small increase in airway resistance was noted, and at levels over 8 p.p.m. a definite increase in airway resistance was obtained.

In addition to the effect on airway resistance, sulfur oxides have been demonstrated to decrease the efficiency of the cleansing mechanism of the respiratory tract, thus slowing down the removal of mucus, debris, and foreign particles from the respiratory tree.

In these and most animal studies, the levels of sulfur dioxide required to produce the effects were considerably higher than the levels usually found in atmosphere air.

Ozone also has these irritating effects, as do several other substances. In the case of ozone, desensitization

may occur, and a tolerance to the lethal effects of ozone may be developed. Again, these effects are seen at concentrations higher than those found in urban air.

Even the famous Los Angeles oxidant smog when faithfully reproduced in the laboratory requires higher concentrations to cause eye irritation than are required for similar irritation in downtown Los Angeles.

Therefore, although the air pollution disasters give us reason to believe these pollutants should have distinct effects, when studied in the laboratory, out of the urban context, adverse effects are not seen at realistic pollutant levels.

The "unhealthy" man

Despite scientific evidence that low concentrations of substances such as sulfur dioxide are not harmful to healthy men, there is a further problem: we are not solely concerned with healthy men:

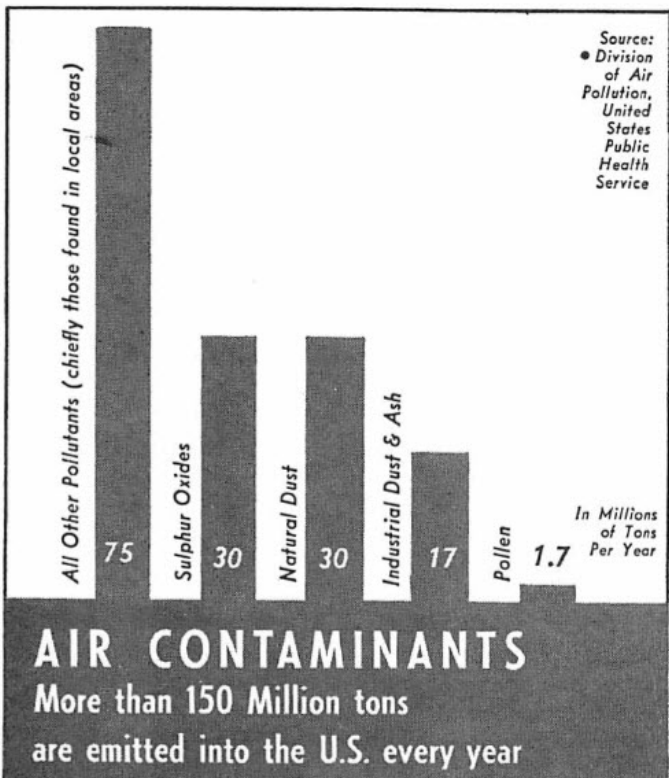
For example, within the "normal" population there would appear to be a group not otherwise distinguished who have an increased sensitivity to sulfur dioxide or who become sensitized after repeated exposure. These people show an increase in airway resistance more marked than the majority of subjects experimentally exposed to the same concentrations of sulfur dioxide and sulfuric acid mists. In one series of experiments, the investigators noted in themselves an increasing sensitivity to sulfur dioxide and sulfuric acid mist over the ten-month period in which the experiments were conducted. One of them developed "a moderately severe but extremely persistent bronchitis."

Within our population there are also an increasingly large number of individuals who already have chronic obstructive ventilatory disease—for example, asthma, bronchitis, and emphysema. In these diseases in which there is increased airway resistance, there appears to be an increased sensitivity to sulfur dioxide and sulfuric acid mists. Atmospheric levels of these substances which produce no apparent change in normal persons may be capable of producing measurable worsening of the airway obstruction in individuals whose lungs are already compromised.

Pollution levels in cities

Polluted air is a complex reactive mixture. It may be that it is the interactions between pollutants, or as yet undiscovered pollutants, or other factors such as rates of change of pollutant concentration which cause illness.

Our understanding of air pollution is still primitive and does not allow us to find the specific causes of illness. Still, if ordinary levels of air pollution do produce harmful effects, there should be evidence in urban populations of illness that is different in type or degree or mortality rates that are increased over those illnesses and mortality rates of rural populations. There have been extensive surveys of morbidity and mortality in urban versus rural populations. Studies of the prevalence of chronic bronchitis and its relation to residence have been done in great detail in Great Britain where chronic





Much of the evidence on the effect of pollutants on health is related to healthy men. But there is a further problem: We are not solely concerned with healthy people. The number of individuals with chronic respiratory disease continues to grow.

bronchitis is a major cause of morbidity and mortality. In England it has been clearly demonstrated that bronchitis rates from city to city tend to parallel the levels of sulfur dioxide in those cities. But studies patterned precisely on the British methods have failed to show similar levels of bronchitis in this country. The prevalence of bronchitis in New York City postal employees, in Westchester County telephone workers and in California telephone workers has been remarkably similar and does not parallel the exposure to sulfur dioxide. The prevalence, incidentally, is about equal to that found in rural England and about half that of London.

Although British and American lungs are probably not different at birth, and sulfur dioxide is always sulfur dioxide, the context of urban life in Great Britain and the United States is undeniably different in many ways. It may be the difference between unidentified environmental factors in the city life of the two countries that probably accounts for the fact that the results do not parallel each other.

Mortality rates for cities such as Los Angeles, where air pollution is high, have been examined and compared with the rates in the remainder of the United States. No significant differences have been found that could be attributed to atmospheric pollution.

Mortality studies have certain disadvantages at best, but in the area of air pollution the disadvantages may be even greater. Often, in studies attempting to relate air pollution to mortality, pollution levels are considered only in relation to the respiratory tract and are considered as a direct cause of death. When such studies are done with the concept that air pollution, acting as a disease intensifier, may be a *contributing* stress to the diseases of many bodily systems, then more suggestive correlations have been found.

Admittedly, in a number of carefully done epidemiological studies, there has been little correlation between the levels of sulfur dioxide and the prevalence of asthma, bronchitis, and other respiratory diseases. But even in these excellent investigations, the basic assumption underlying the study method was that air pollution would have a direct and quantitative effect in the production of respiratory disease or changes in respiratory physi-

ology. When a less dramatic effect is sought, the results may be more positive. F. C. Dohan, in studying occupational groups in cities with different degrees of air pollution, showed a positive correlation between minor upper respiratory illness and the levels of air pollution.

A study by William Spicer and his group attempted to correlate atmospheric pollution and extensive pulmonary function studies done daily on a small group of patients with obstructive ventilatory disease. The patients became better and worse together but it was not possible to implicate any one pollutant. The authors concluded that the problem was complex and involved combination of factors with subtle variations around the theme of environmental changes.

Air pollution part of urban stress

The positive evidence is slim but I have tried to show what has made us believe that air pollution is rarely if ever a sole and direct cause of diseases, but is rather a part of urban stress; one factor in the production of multifactorial diseases such as chronic bronchitis and other respiratory diseases.

We may never produce evidence showing a specific effect from a specific level of one pollutant, but evidence of complex effects from the complex of pollutants is highly probable and just as important.

Atmospheric pollution is the child of weather conditions and emission sources. Sufficient information is already present about both sources and meteorology on which to base sound, fair, and effective controls. Because such controls must cross political boundaries and involve many aspects of urban life, initiation of them is difficult, but it should not take disaster or proof of death to make them a reality.

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