that within each socioeconomic class there were generally more respiratory deaths in those areas where the annual average of sulfur dioxide was high.

With the advantage of an improved monitoring system, which went into operation in Chicago in 1966, and with this background from the studies already done, Dr. Carnow and his colleagues took a new approach to the relationship of air pollution and mortality, analyzing deaths, not on an annual basis, but on particular days. The city was divided into square miles, and the sulfur dioxide level for each square mile on each day in January 1966 was estimated from the measured concentrations at the nearest one, two, or three continuous-monitoring stations. (Chicago has eight such stations.)

The month was divided into three segments, or "tertiles," according to low, medium, or high sulfur dioxide concentration in each square mile. January was a month of heavy pollution, so that the "low" period averaged 0.08 part per million in the least polluted square miles of the city, and 0.13 in the most polluted. Deaths occurring in each ten-day period were compared within each square mile, and then totaled for the 212 square miles of the city.

One of the greatest difficulties in studies seeking the relationship between air pollution and death is in finding a way to hold all other factors constant, so that the health effects of air pollution can be measured. This has been most satisfactorily done in analyses of episodes of very high air pollution, for example, in London in 1952 and in New York in 1962. The number of deaths in those cities that would be expected in the same number of days, at the same time of year, with temperatures about the same, were calculated from experience in previous years. The expected deaths were compared with actual deaths during the pollution episode, and the "excess deaths" attributed to air pollution. These studies of episodes left an important question unanswered, since the "normal" periods that were compared with the episodes were not periods of *no* pollution, but merely periods of what had become "normal" pollution in those cities. The question was whether people were dying from the effects of air pollution at these "normal" levels. If people die of the effects of air pollution during "normal" periods, is there a threshold — a level of pollution below which no deaths will result?

Air pollution causes death and illness, particularly among nonwhites and in low socioeconomic groups.

In the study of Chicago in January 1966, the effect of air pollution was isolated by confining the study to a single month, so that seasonal variations could not be a factor. There was no significant difference in temperature between days when pollution levels were high and when they were low. Age, sex, race, and socioeconomic and cultural differences which might have affected the death rate were kept constant by dealing with the small segments of the population separately first, comparing the population of each square mile with itself on days of differing pollution.

This study did show a rise in deaths at the upper tertile of pollution as compared to the lower and middle tertiles, but no significant difference between the lower and middle tertiles. There was no significant rise in deaths among females when pollution levels rose.

The study showed a particularly striking rise in the deaths among men due to cardiovascular and respiratory disease. Nationwide statistics show that more men die from these causes than women. This suggests the reason why there appeared to be no increase in mortality among women when there was a rise in air pollution levels. These statistics, as well as the Chicago discovery of no mortality rise for females, also suggest that women are not as much endangered by air pollution as are men.

The results presented in detail by Dr. Carnow at the AMA medical research conference dealt with males 55 and over, and suggested that this may be another high-risk group. When deaths from all causes in this group were analyzed, there was no significant difference between the days with low pollution levels (sulfur dioxide averaging 0.08 part per million) and the days with middle pollution levels (sulfur dioxide averaging 0.1 part per million), but there was a clear and significant increase between the low and middle thirds of the month as compared with the third having high pollution levels (sulfur dioxide averaging 0.29 part per million). The same pattern was repeated for deaths due to coronary heart disease, all cardiovascular disease, bronchitis and emphysema, all respiratory disease, and even for deaths due to neoplasms (tumors). In regard to this last category, Dr. Carnow commented:

Various forms of cancer occur much more frequently among asbestos workers than in the general population.

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It was only when sulfur dioxide was 0.34 or higher that a significant increase was shown in either the nonwhite or the low socioeconomic group.

As Dr. Carnow pointed out:

> There are a number of possible explanations for these patterns. The first is that low socioeconomic or nonwhite individuals have increased resistance to disease and therefore show significant mortality only at very high sulfur dioxide levels. The higher death rate in these groups, the poor nutrition, the lack of available medical care, poor housing, and other factors suggest that this is not the case. The second possibility is that adaptation occurs to a higher degree at the low socioeconomic level and in nonwhite individuals. The increased death with the increase in pollutants suggests, however, that this is not so.

Dr. Carnow then proposed an alternative explanation, namely, that the most susceptible individuals in all population groups are killed by a combination of illness and environmental factors including *the usual levels of pollution* in the areas where they live, but since this happens all the time, their deaths appear as part of the normal death rate. A pronounced rise in pollution kills a number of people highly susceptible to pollution *and in addition* some people who are more resistant to pollution.

What he was suggesting was that two interrelated factors are responsible for the deaths: individual resistance and the level of pollution (a mixture of various gaseous and particulate pollutants). Each of these two factors in turn is highly complex. Resistance is determined by individual physiological differences which have interacted throughout a person's lifetime with such social effects as nutrition, such personal habits as smoking, and such environmental effects as previous exposure to pollution.

Dr. Carnow suggested that "in relation to morbidity and mortality, particularly in high-risk groups, but possibly in the entire population, there may be no threshold."

In other words, his hypothesis is that there may be a point where resistance is so low in an individual that even low levels of air pollution can precipitate illness and death, and that high levels of pollution can precipitate illness or death in less susceptible, more resistant individuals.

It should be emphasized that this is as yet only a theory. Dr. Carnow himself proposed to test it in a number of ways. The relationship between mortality and air pollution in Chicago during many months is being studied, since the preliminary results presented at the conference were for one month only. In addition, if his hypothesis is valid, people moving from areas with low pollution levels to areas with high pollution levels would be expected to die sooner than people of similar racial and socioeconomic background who remain in areas with low pollution levels. Studies are being designed to test whether this is indeed the case.

City Pollutants

Studies of two urban contaminants — asbestos and the hydrocarbon benzpyrene — revealed some of the ways in which living systems are affected by pollutants.

The discussion of asbestos was prepared by I. J. Selikoff, W. J. Nicholson, and A. M. Langer, and was presented by Dr. Selikoff, who published an article on asbestos in the March 1969 issue of *Environment*. Dr. Selikoff addressed himself primarily to asbestos air pollution in urban areas. He began by pointing out that among asbestos insulation workers in the New York metropolitan area approximately one in five deaths is due to lung cancer, one in ten is due to mesothelioma (a form of cancer which has been strongly linked to asbestos exposure), and one in ten to asbestosis (lungs severely scarred by inhalation of asbestos particles) and cor pulmonale (a heart condition resulting from disease of the lungs).

"These men have been exposed to amounts of asbestos surely greater than those in the community generally, and we would not expect their risk to be duplicated in the general population," Dr. Selikoff said, "but is *part* of their risk disseminated with the dusts from their work?"

Dr. Selikoff presented new data confirming previous findings of asbestos in the lungs of urban dwellers and new data on asbestos in samples of urban air. He also identified populations which may be especially vulnerable to the slowly developing asbestos-related diseases because of previous exposure to asbestos.

The various forms of cancer noted above occur much more frequently among asbestos workers than in the general population, even when there is little or no radiological evidence of asbestosis. Industry clean-up may reduce dust levels and prevent the asbestos-scarred lungs of asbestosis, yet this is not necessarily adequate to prevent cancer.

Selikoff and Hammond demonstrated in 1968 that "asbestos bodies" had been regularly found at autopsies in many cities of the world (these data were included in the *Environment* article the following year). However, asbestos bodies are coated fragments of asbestos, and it is difficult to analyze the cores. There remained doubt that the cores were necessarily asbestos. The question has now been answered, not by analysis of asbestos bodies, but by a direct search for asbestos fibers and fibrils that had not become coated. Asbestos bodies were found in a small sample of lung tissue of 1,449 out of 3,000

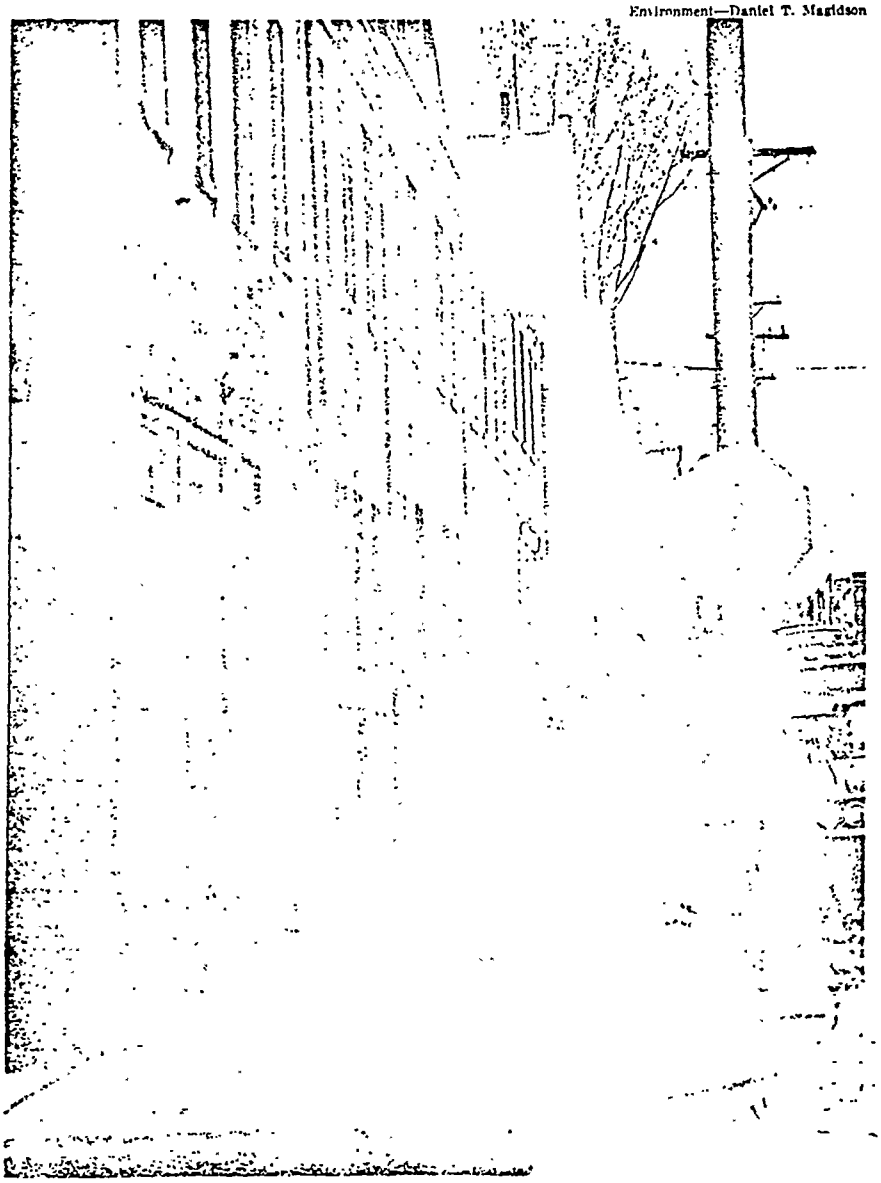
This suggests that aside from whatever specific effect the high level of pollutants have on the cardiovascular and respiratory systems, they appear to act as a death accelerator in regard to individuals with terminal disease.

One might interpret the results of this mortality study as an indication of a threshold level between the middle and high levels of pollution, that is, somewhere between 0.17 and 0.29 part per million of sulfur dioxide. It would seem that at 0.17 and below, deaths might have been due to other causes. Then deaths from pollution are represented by the difference between the 38 or 39 average deaths per day at the lower levels of pollution and the average of 52 deaths per day at the higher levels. One could then say that it appeared that about 13 deaths per day during the ten days of high pollution levels were caused by air pollution in January 1966. It would also seem that as long as sulfur dioxide remains below 0.17 part per million in any city, no pollution-related deaths would be expected.

But exposure to pollution was not the same throughout the city. Those square miles populated primarily by nonwhites and by all low socioeconomic groups were the areas of highest pollution levels. When the mortality and pollution data were related to race and socioeconomic status, a revealing picture emerged. The dividing line betwen increase and no increase in deaths continued to be between 0.17 and 0.29 part per million for whites and between 0.12 and 0.24 for the high socioeconomic group. However, no increase in deaths appeared in the nonwhite group when pollution levels averaged 0.21, or in the low socioeconomic group when they averaged 0.25.

(continued on page 29)

Environment—Daniel T. Magidson



Current research indicates that certain groups in the population—particularly elderly persons with respiratory disease and people from low socioeconomic backgrounds—are more susceptible to the ill-effects of air pollution.

Environment—Daniel P. Maddison

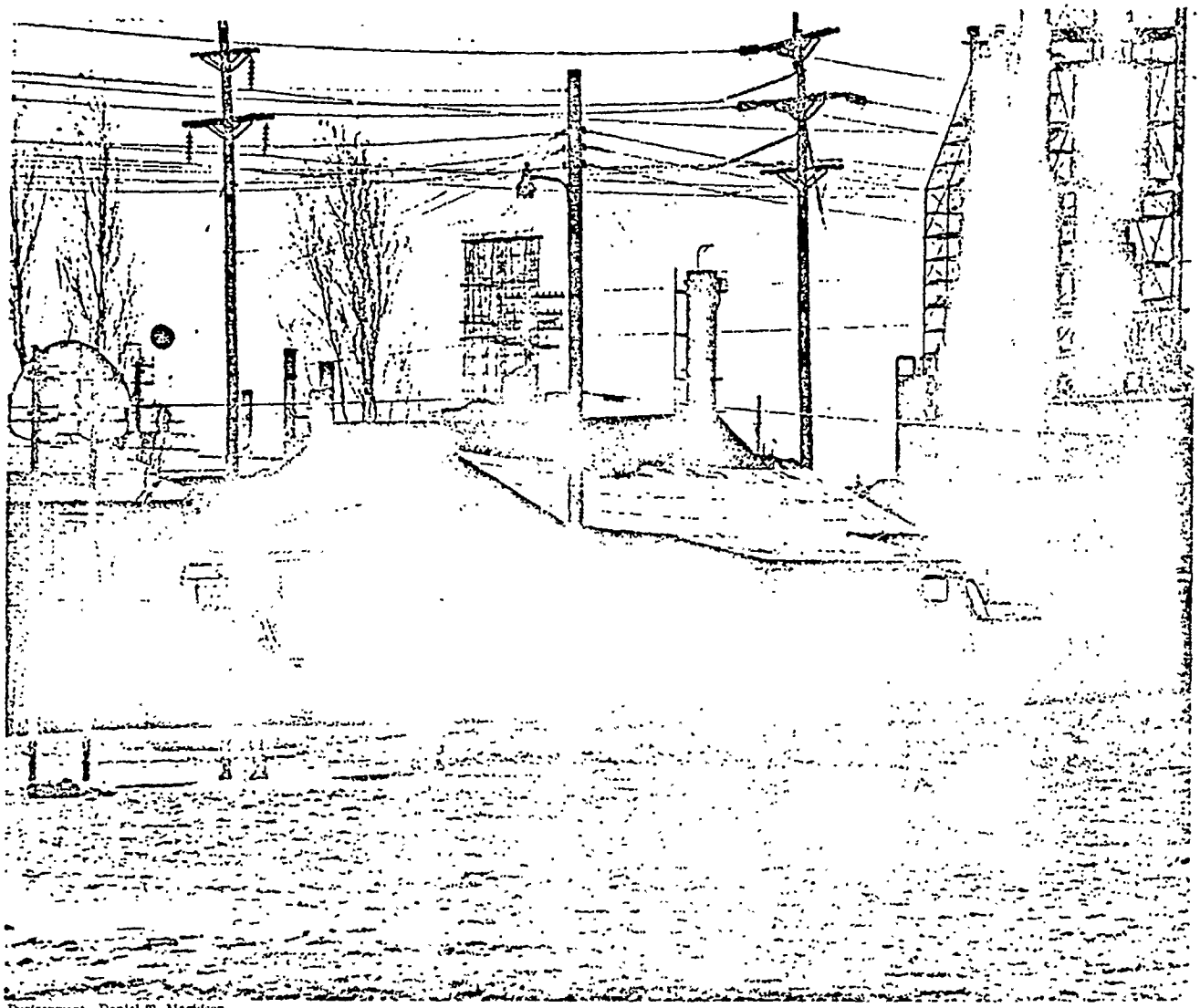
consecutive autopsies in New York. In 28 of these, a further very small portion of lung was examined by a technique that makes it possible to identify the exceedingly small asbestos fibers and fibrils. Chrysotile (a variety of asbestos commonly used in the United States) fibrils were found in all 28, although in four cases there were only 9 fibrils or less, too few for confidence in the count. In the remaining 24, from 10 to more than 200 fibrils were found.

Preliminary measurements of the amount of asbestos in the air of New York City, Philadelphia, and suburban Ridgewood, N.J., varied from one-hundredth to one-tenth of a microgram per cubic meter. The highest value was detected in Philadelphia. Since urban air typically contains particulates in amounts from 75 to 200 or more micrograms per cubic meter of air, amounts of asbestos in the hundredths of a microgram appear to be very little, but Dr. Selikoff pointed out that chrysotile asbestos easily fragments into fibrils so small that a thousandth of a microgram of this material could represent a million fibrils.

A common practice in the building of high-rise office buildings is to spray fireproofing material containing from 10 to 30 percent asbestos onto the girders and other structural components. Not only are workers engaged in the spraying or otherwise occupied on the same building exposed to the spray, but extensive fallout in the vicinity can often be seen. Dr. Selikoff reported air measurements from five sites near such a source:

CHRYSOTILE CONTENT OF NEW YORK CITY AIR IN VICINITY OF SPRAY-FIREPROOFING WITH ASBESTOS-CONTAINING MATERIALS

Sampling Site	Asbestos in Micrograms Per Cubic Meter of Air
Downwind from source	.045-.180
45 degrees from source	.015-.030
Upwind from source	.020
Downwind from source	.045
Upwind from source	020



Environment—Daniel T. Magidson

Pollution of the air affects everyone, but some are more affected than others. The boys pictured on the left, for example, breathe less polluted air on their suburban playground than the boys on the right, who play against a backdrop of the city's industry.

Since these measurements were made, New York City has instituted regulations to control this use of asbestos. The high Philadelphia measurements referred to previously were made near sites where spraying was taking place, but *after* control regulations had gone into effect.

Since asbestos fibers are mineral, they may persist in the environment a long time. Dr. Selikoff reported:

> We have found amosite asbestos fibers in the settled dust and in the household air within homes which had been occupied fifteen years before by workmen of an amosite factory. . . . both settled dust and ambient [surrounding] air in a construction workman's home contained chrysotile fibers at levels beyond those usually observed as background. Neighborhood contamination from factory sources may also be associated with long persistence of the mineral fibers. In preliminary studies, we have found this to be true of both superficial soil contamination and settled dust on attic rafters, in such neighborhoods.

There are clearly certain occupational groups at risk from asbestos inhalation, including the four million men in the construction industry, those who have worked in this industry in the past, and workers in asbestos factories. The fact that British investigators have found asbestos in the lungs of shipyard workers points to another group at risk, as Dr. Selikoff showed:

given level of asbestos before control measures are instituted?

More than with most aspects of industrial health, the exposure of workers in the construction industry is closely related to the exposure of nonworkers, since most construction not only takes place in the open air, but in highly populated areas. The demonstrable effect of occupational levels obviously calls for protection of this group, with the added advantage that asbestos in urban air would be reduced at the same time, protecting other city dwellers from less, but possibly significant, exposure.

Airborne Cancer-Producing Substances

There were a number of papers at the AMA conference describing laboratory experiments which were primarily of interest to specialists, but one of these had a clear and immediate relevance to the problem of air pollution as a possible cause of cancer. This was a paper by T. Timothy Crocker, Thomas V. O'Donnell, and Lora L. Nunes of the University of California, presented by Dr. Crocker.

Previous experiments have established that benzpyrene (a hydrocarbon, technically benzo(a)pyrene), which is found in urban air, can produce cancer in rodents and dogs. Besides being carcinogenic (capable of inducing the abnormal cell changes that distinguish cancer), it is also toxic (capable of killing cells). The same is true of an extract of the organic fraction of particulates from urban air (the benzene-soluble fraction). The complete composition of this extract is not known. Dr. Crocker and his colleagues have developed a laboratory method of testing these pollutants on respiratory tissue or whole respiratory structures of primates whose physiology is closer to that of humans than is the physiology of rodents and dogs, and even on those of humans.

Generally speaking, if changes take place in monkey and human tissue similar to those that take place in rodents and dogs, then we are a step closer to being able to say that the abnormal cell changes of cancer would take place in living primates, just as they are known to take place in living dogs and mice or hamsters.

Monkey cells in organ culture were not killed by the air pollution extract, but benzpyrene did appear to be toxic to monkey cells — more toxic than to dog cells.

Cancers did not develop under experimental conditions, but pathologic cell changes were found in some monkey tissue exposed to benzpyrene and also in some exposed to air pollution extract. Dr. Crocker told *Environment* that subsequent experiments with bronchial tissue from human fetuses have produced results quite similar to those with monkeys.

He cautioned, however, that the ultimate test of whether these pathologic changes would actually lead to cancer is the ability of the abnormal cell growth to invade adjacent cells, and this has not been demonstrated in his experiments. He also pointed out that the body's protective mechanisms cannot come into play in the organ cultures as they can in life, and this must also be taken into consideration.

Breathing Easier

Finally, to conclude on a happier note, a few words about a study that might have been called "Berlin Revisited": A group from Harvard University School of Public Health (B. G. Ferris, Jr., I. T. T. Higgins, M. W. Higgins, J. M. Peters, W. F. VanGanse, and M. D. Goldman) surveyed chronic respiratory disease in Berlin, New Hampshire in 1961 and again in 1966-67. Dr. Peters reported that they found the town in slightly better health on the return visit. Age-specific rates for chronic respiratory disease in nonsmokers, exsmokers, and smokers of from less than 4 to more than 45 cigarettes a day, were almost invariably slightly greater in 1961 than in 1967. One exception was females smoking more than 45 cigarettes a day, but there were only six persons in this category, so the disease rate was probably not reliable.

The investigators attributed the reduced incidence of chronic respiratory disease to a drop in air pollution. Unfortunately, the air monitoring data, particularly in 1961, were scanty, so that it is difficult to say just how great an improvement in air quality it took to achieve a drop from about 20 percent disease incidence among nonsmoking men in 1961 and about 18 percent among nonsmoking women, to about 12 percent for both groups in 1967.

Nevertheless, it's good to have evidence that air quality *can* improve, and that improvement *does* bring better health. □

As the level of sulfur dioxide in the air rises, so does the rate of acute illness. The picture of Boston, at left, was taken in October 1969, when the concentration of sulfur dioxide in Kenmore Square (circled) was 0.35 parts per million. This is more than double the highest level recorded in Chicago during the month of the mortality study described in this article.