Health Effects From Repeated Exposures to Low Concentrations of Air Pollutants

RICHARD A. PRINDLE, M.D., M.P.H., and EMANUEL LANDAU

ONE of the best documented facts in the whole complex field of air pollution is that it can, in certain circumstances, result in acute illness and sudden death. Everyone knows about the disasters in Belgium's Meuse Valley, in Donora, Pa., and in London. Continuing research is uncovering other such episodes, long after they have occurred. In the United States we plan to continue our search for further evidence from the past. We hope to develop eventually a warning system that will predict the weather and other conditions which made possible such abnormally high concentrations of air pollutants and thereby mitigate, or even eliminate, future air pollution disasters.

Nevertheless, although more Americans than ever before are doing research today in air pollution, an increasing proportion of this effort is devoted to the long-term effects of exposure to low pollutant levels.

Our approaches to the determination of chronic effects of pollution have been of two major kinds: (*a*) the repeated laboratory exposure of human and animal subjects to specific pollutants or mixtures; and (*b*) the epidemiologic approach, using the community as a field laboratory.

Laboratory Research

In pursuing the first kind of research, the Division of Air Pollution, Public Health Service, has encouraged attempts to develop techniques capable of measuring minute changes in physiology to supplement new knowledge of pollutant concentrations at levels which cause marked pathological variations or death. Accordingly, we have recently undertaken studies of physiological and metabolic activities. Unfortunately, because of lack of knowledge about the physiological effects of pollutants, the choice of metabolic activity to be studied must often be based on trial and error. In some cases, a chance observation by other investigators, discovered in a search of the literature or through personal communication, offers a clue which seems worth pursuing. In one such instance, because of the similarity of certain toxicological effects of ozone to those produced by ionizing radiation, our researchers are following, in rats exposed to ozone, the urinary excretion pattern of creatine and creatinine, known to be affected by radiation. Possible alterations in protein and purine metabolism after exposure to various pollutants are being sought by analyses of the urinary excretion of uric acid and amino acid nitrogen. Measurements of oxygen consumption, also in progress, may yield useful information during long-term inhalation exposures. These ap-

Both authors are with the Division of Air Pollution, Public Health Service. Dr. Prindle is deputy chief of the division, and Mr. Landau is chief of the Biometrics Section, Field Studies Branch. *The paper was given before the Verein Deutscher Ingenieure (DI) Kommission (Commission for Clean Air) June 20, 1962, at Düsseldorf, Germany. It is scheduled to be published in Staub in October 1962.*

proaches are then coupled with studies of pulmonary function for comparison with human disease states.

The following examples illustrate some of the various approaches in the study of the long-term effects of air pollution on animals and man. In a study using classic laboratory techniques, repeated inhalation of ozone at a concentration of 1 ppm (2.6 mg./m.^{3}), only slightly greater than that existing in some urban atmospheres, produced chronic bronchitis and bronchiolitis in small animals (*1*). The smaller bronchioles were partly occluded by hyperplastic or sloughed epithelium mixed with acute inflammatory exudate in guinea pigs which survived the experiments and were sacrificed at the end of more than 400 days of exposure. The bronchiolar walls displayed fibrosis extending into the alveolar ducts and alveoli. A mild degree of emphysema was considered to be secondary to the bronchial occlusion. The changes were less marked in rats and hamsters and inconsistent in three mice examined. No evidence of intrapulmonary injury was detected in two dogs whose lungs were examined microscopically, but the trachea and major bronchi showed slight epithelial injury. Rats and guinea pigs which died during the course of the exposure exhibited massive pneumonia; slight fibrosis was noted as early as the 25th day of exposure.

Groups of 9-month-old rats were exposed continuously up to 2 years to 1, 2, 4, 8, 16, and 32 ppm of sulfur dioxide to determine the long-term effects as manifested by survival, hematological response, and clinical symptoms (*2*). Exposed rats exhibited changes in skin, fur, and conjunctiva and respiratory distress of increasing severity with increasing concentrations of the gas. A marked difference in the death rate of the group exposed to the 32 ppm concentration (84 mg./m.^{3}) was observed, as compared with controls, and groups exposed to lesser concentrations of sulfur dioxide also began to die before the control group. All control animals survived the first 9 months. By 18 months and until the end of the experiment, the survival rate of rats at all exposures to SO_2 except 32 ppm was similar but distinctly different from that of control animals. The earlier age at death of exposed animals was considered compatible with a process of accelerated aging, possibly resulting from the stress of such exposure.

It is becoming increasingly evident that oxides of sulfur, in concentrations attainable in community air, may affect the human respiratory tract. A research team at the Harvard School of Public Health has shown that the acute response in human beings resembles that in guinea pigs. Normal persons who inhaled either sulfuric acid mist or sulfur dioxide for brief periods exhibited markedly shallower, more rapid breathing (*3,4*). More recently another team of investigators at Harvard measured pulmonary function in healthy volunteers exposed to controlled levels of sulfur dioxide (*5*). During administration of the gas, all measurements of resistance showed an increase, greatest for pulmonary flow resistance (PFR) on quiet breathing, intermediate for PFR on panting and for airway resistance, and smallest for total respiratory resistance. Pulmonary flow resistance showed no change at 1 to 2 ppm of sulfur dioxide; it increased an average of 19 percent above control levels at 4 to 5 ppm and 49 percent at 8 to 19 ppm; and when sulfur dioxide was combined with aerosol, the increase was 72 percent. However, investigators at St. Luke's Hospital in Cleveland observed no changes in resistance in normal subjects exposed briefly to sulfur dioxide in concentrations of 2.5 to 23 ppm, combined with particulates and aerosols, whereas emphysematous subjects exhibited a decrease in airway resistance (*5*).

Although the acute effects of exposure to high concentrations of carbon monoxide are well documented, the chronic effects from long-term subtoxic doses are controversial. Recent findings suggest that, besides its known effects upon hemoglobin, carbon monoxide exposure may affect the eye and nervous system adversely. Since 1955, carbon monoxide levels in the Los Angeles atmosphere have been increasing by about 1 ppm (0.0012 mg./m.^{3}) per year. It is estimated that gasoline engine exhaust is the source of about 75 percent of the total carbon monoxide content of Los Angeles air, with significant contributions also from metallurgic and oil-refining operations.

Research workers (*6, 7*) have found an average blood carboxyhemoglobin of 3.8 percent,

not markedly different from average levels in groups with lesser degrees of exposure, in subjects exposed to carbon monoxide in their working environment, from smoking, or while commuting to work in private automobiles. The carbon monoxide concentration in a garage and automobile inspection center where the exposed group worked ranged from 10 to 150 ppm, (0.06 mg./m.^{3}); in the working environment of the control group, the ambient carbon monoxide level was less than 10 ppm (0.012 mg./m.^{3}). Although 17 of 68 exposed subjects, compared with 3 of 25 controls, complained of headache, dizziness, or unusual fatigue at the end of the workday, no relationship could be found between carboxyhemoglobin levels and occurrence of those symptoms.

In a preliminary study performed by Public Health Service scientists at Cincinnati, Ohio (*8*), the levels of carbon monoxide in the passenger compartment of stationary vehicles in heavy traffic were greatly increased, reaching a maximum of 370 ppm (0.44 mg./m.^{3}). Investigators at the University of Michigan (*9a,10*) sought to determine whether atmospheric carbon monoxide levels in urban areas might interfere with the driver's ability to operate his vehicle. Data collected from appropriate sites in Detroit for 1 year showed that median daily values of atmospheric carbon monoxide ranged between 0 and 20 ppm. During periods of high atmospheric stability and heavy traffic, concentrations reached 100 ppm at some sampling sites and persisted at this level for several hours. In homes several hundred feet from street sampling sites, concentrations approximated those in the street. Analysis of reports of more than 4,000 consecutive accidents involving almost 5,000 persons failed to reveal a higher accident rate associated with occupations in which high carbon monoxide exposure would be expected. In an attempt to relate the carbon monoxide content of the blood to air levels, a cigar smoker and a nonsmoker traveled in a police scout car for 8 hours for a distance of 130 miles. The carbon monoxide in the vehicular air, monitored continuously—reflecting outside traffic conditions and not influenced by any tobacco smoke in the car—averaged 17 ppm with a peak of 120 ppm when the engine was idling. The smoker's blood carboxyhemoglobin rose from 3.1 to 3.9 percent, the nonsmoker's from 0.8 to 1.2 percent.

In a study of 237 persons involved in traffic accidents (including both drivers and pedestrians) and brought to the hospital for treatment, 50 percent of the drivers had less than 3 percent carboxyhemoglobin, 50 percent of the pedestrians had less than 2 percent, and only 3 persons had levels of 10 percent or more; in 1 of these the carboxyhemoglobin was 31.5 percent. It was concluded by the investigators that carbon monoxide concentrations in the general atmosphere of Detroit do not impair driving ability (*10*), but further work is now underway to substantiate or amend these findings.

Studies undertaken on animals have demonstrated that guinea pigs exposed to automobile exhaust, at a concentration several times normal, for 1, 2, and 4 weeks, were especially susceptible to severe pulmonary disease (*9b*). This came to light accidentally following an epidemic which produced pneumonia in the test animals. Significantly higher mortality occurred in the animals exposed to irradiated exhaust, comparable to heavy photochemical smog, than in animals exposed to nonirradiated exhaust or in control animals, which also experienced the epidemic but were exposed only to pure air. This finding parallels the results of another study in which animals exposed for only 2 hours to pure nitrogen dioxide at levels similar to those occasionally found in community atmospheres were much more susceptible to infection by certain pneumonia organisms (personal communication, Richard Ehrlich, Armour Research Institute, Chicago). More serious illnesses and more deaths occurred in this group than in the control animals, which were exposed to the same organisms but otherwise breathed only pure air.

Irradiated exhaust, that is, automobile exhaust which has been diluted with air and then exposed either to sunlight or to artificial light with ultraviolet components, is chemically different from exhaust which has not been irradiated. It has been shown that this irradiated gas is chemically similar to the so-called "photochemical smog" so notorious on our west coast. It also causes the same types of damage to vegetation as the "smog" found in California. Constituents include ozone, "oxidants" (oxygen-

containing compounds of high reactivity), other hydrocarbons, and oxides of nitrogen.

These ingredients appear to result from complex interactions due to photochemical action on the unburned hydrocarbons and oxides of nitrogen found in exhaust gases. Merely mixing ozone with hydrocarbons, such as gasoline vapors, can simulate this process to some degree. Because these ingredients appear to be more biologically potent, causing damage to plants and eye irritation in people, our recent studies have been focused on them to a large extent.

Physiological experimentation in which measures were made of respiratory function of guinea pigs, including pulmonary resistance, respiratory rate, and minute volume, has shown that the greatest changes occurred in those animals exposed to irradiated exhaust. In general, these changes have occurred when the animals have been exposed to concentrations two or more times the usual ambient levels. However, some physiological changes have occurred in animals at "community" levels, and certain specific pollutants have been observed to produce effects at or near these concentrations. This would appear to indicate that the observed maximum levels present in communities at this time are borderline with respect to causing immediate effects such as changes in pulmonary function and may be highly significant in their long-term effects.

Last year workers at the University of Southern California were able to produce true squamous cancers in the lungs of mice, similar in type to those found in human beings, by exposing the animals first to infection, then to air containing ozonized gasoline. In this experiment, one group of animals was exposed to a virus type of influenza and another was unexposed. Each of these groups was divided after recovery into two further groups, one exposed to purified air and the other to ozonized gasoline. In the animals receiving the infection alone, approximately 8 percent showed squamous changes in the bronchi consistent with healing processes after infection and occasionally demonstrated metaplastic changes. In the animals exposed to ozonized gasoline alone, there were no significant findings. In the uninfected animals exposed to pure air, the findings were negative. A striking 30 percent of the animals which had been infected and subsequently exposed to ozonized gasoline demonstrated the presence of squamous carcinoma. Interestingly enough, the male-female ratio was approximately 3 to 1, similar, in fact, to that found in humans and obviously not associated with smoking habits or occupation (*11,12*).

Epidemiologic Research

Considerable epidemiologic research has also been undertaken with the community used as a laboratory. While increasing effort has been devoted to the chronic effects of normal low levels of community pollutants, the Public Health Service has continued to support research into the extent of previously unreported air pollution disasters.

The literature of air pollution disasters was enriched recently by the publication of a paper reporting excess mortality, presumably due to elevated levels of pollutants resulting from an extended temperature inversion in New York City as long ago as November 1953. This excess mortality in the largest metropolis in the United States was determined retrospectively by an examination of death records (*13*). It parallels in that respect the experience of the 1952 disaster in London, the largest metropolitan area in Great Britain. However, while the London episode was studied almost concurrently, the study in the United States was made more than 5 years after the event.

In the 1953 incident, 220 excess deaths were attributed to cardiac and respiratory diseases, again paralleling the London episodes. These deaths must have been accompanied by increased morbidity. Unfortunately, the precise magnitude of this morbidity is uncertain, since it is extremely difficult to obtain reliable morbidity data for past years.

There are some possible sources of illness data, such as hospital admissions, physician visits, group medical practice usage, health surveys, and the like. It is obvious that it is no easy matter to collect such data after the lapse of nearly a decade. The less current the records, the greater is the danger that they may no longer exist. Therefore, it was gratifying to find than an examination of the records on

emergency room visits to the largest New York City hospitals for November 15–24, 1953, undertaken recently by the same group which reported on mortality in New York City, revealed about twice the expected number of visits by patients with respiratory and cardiac conditions (*14*).

The line of demarcation between an acute air pollution episode and the chronic long-term effects of low levels of air pollution can become quite blurred. This difficulty is exemplified when we look for causes of the large number of asthmatic responses to sublethal levels of pollutants which have been observed in New Orleans, Pasadena, and Nashville.

In New Orleans, it was demonstrated that there had been sharp periodic increases in emergency clinic visits to Charity Hospital by nonwhite asthmatics. This has occurred often enough so that adequate documentation is now possible (*9c*). The usual number of visits to Charity Hospital by asthmatics was 25 per day for the period 1953 to 1961.

Frequently, however, outbreaks of asthmatic attacks have seriously strained the facilities of the hospital. In August 1958, for example, an outbreak of asthma involved 100 people, with 3 deaths. There have been instances of daily admissions of 150 and even 200 Negro adult patients. Asthma outbreaks have been accurately predicted in advance on at least two occasions; the predictions were made on the basis of meteorological data which had shown that the outbreaks were associated with particular wind movements.

We are now able to report that these asthma outbreaks are thought to be related to particles of a silicon-containing compound emitted into the atmosphere as a result of poor combustion of garbage and refuse in the New Orleans city dumps. This could very possibly be an instance of an air pollutant acting as an allergen and creating an allergic response in certain susceptible individuals. Obviously, testing of skin and pulmonary sensitivity and further research are indicated to verify or disprove this hypothesis.

How are we to consider the response of asthmatics to insults to the respiratory tract in such diverse air pollution areas as Los Angeles and Nashville? Are these responses the product of acute or chronic insults? In the Los Angeles area a study was conducted from September 3 to December 9, 1946, of 137 bronchial asthma patients of 5 practicing physicians (*15*). The study revealed that the average number of patients afflicted on days when oxidant values were above a level that caused eye irritation was significantly greater than the average number on days when oxidant values were below this level. Similarly, the number of persons who had attacks on days when plants showed damage from air pollutants, a biological indicator, was significantly greater than the number on other days.

In Nashville, also, it was found that attack rates were significantly different when comparison was made of days with the highest and the lowest sulfur dioxide levels (*16*). The statistical significance was even greater when the daily data on asthma attacks were lagged 1 day to take account of possible delayed reactions to sulfur dioxide. A possibly corroborative finding was that the pattern of attacks for adult asthmatics reflected differences in air pollution levels in different sections of the city. Thus, the attack rate was three times as high in an area of high pollution as it was in a low-pollution area. It is particularly noteworthy that the sulfur dioxide levels in Nashville are not very high even at their worst.

Evidence from episodes in the Orient which afflicted American servicemen and their dependents also shows that air pollution appears to be an etiologic agent for a condition that was originally called "Yokohama asthma" (*17*). This is now referred to as "air pollution asthma." Some of the servicemen and their dependents were relieved of asthmatic attacks when they were moved out of affected areas in Japan and Okinawa. Conversely, some persons who were not evacuated quickly from the areas continued to manifest asthmatic symptoms. Accordingly, it appears that for some susceptible individuals this kind of asthmatic response may be truly a chronic effect of air pollution.

A further bit of evidence is based on recent data on illnesses of employee groups. These data indicate a high degree of relationship between respiratory illnesses lasting 8 days or more and levels of sulfates in selected cities in the United States (*18*). If this relationship

were to be more completely documented, it might explain some of the striking rise in the prevalence of chronic respiratory diseases which has been observed recently.

The best indication of all of the chronic effects of air pollution is undoubtedly provided by the statistics on chronic bronchitis in Great Britain (*19*). There, the disease is the third leading cause of death and the leading cause of disability.

In the United States there has been increasing acceptance of the view that a group of chronic respiratory diseases in this country, comprising emphysema, chronic bronchitis, bronchiectasis, and "other chronic interstitial pneumonia," is similar to the chronic bronchitis syndrome observed in Great Britain and that the apparent differences reported in the two countries may merely reflect differences in medical diagnostic criteria and terminology in patients with cases of differing severity and degree of infection.

We are aware that in the United States no evidence has been produced to demonstrate that air pollution is an etiologic agent for the emphysema syndrome. Nonetheless, there is an ever-mounting accumulation of evidence linking the two. Well known is the phenomenal rise of deaths from emphysema in the American population as a whole since 1950 (*20*). One may, with considerable certainty, ascribe part of the increase to the increasing acceptance of this classification as a cause of death, which in turn is due to the increasing discussion of chronic respiratory disease in the medical literature. Nevertheless, we have no reservation in stating that part, perhaps most, of the sevenfold increase in the frequency of this diagnosis as a cause of death is due to the greater prevalence of the disease.

One reason for assuming an air pollution factor as a cause of emphysema is the urban-rural comparison of mortality in the United States. Certainly, when the age-adjusted urban rates are double the rural rates, there would appear to be some factor which is directly related to residence in cities. Attention has been drawn previously to the fact that smoking differences among males, by residence, are relatively small and presumably do not account for the urban-rural ratio found for emphysema (*21*). The well-documented greater pollution of urban atmospheres as compared with rural points to a possible causal relationship.

It is recognized, of course, that there may also be an occupational exposure factor. The records of the Social Security Administration show that emphysema is the second leading cause of disability among male workers 50 years of age and older (*22*). It is clear that the evaluation of the role of air pollution in the increase in mortality from emphysema will have to take account of the occupational history of the decedent as well as his smoking habits.

Because of the increased interest in chronic respiratory diseases there has been a growing awareness of the need to inform people of the importance of certain symptoms. Accordingly, the National Tuberculosis Association has announced its intention of conducting a campaign during the spring of 1963 to alert people to the significance of "shortness of breath" and "chronic cough" (*23*). It is quite possible that people become so accustomed to these symptoms that they pay little attention or attach no importance to them. An increasing amount of data on the prevalence of emphysema and chronic bronchitis should be forthcoming as this educational campaign progresses.

In order to learn more about the long-term effects of air pollution, it was found necessary to conduct extensive field studies on selected populations. In 1959 the Division of Air Pollution of the Public Health Service, in cooperation with the Pennsylvania Department of Health, the Pennsylvania Electric Co., and others, undertook a study of two small communities in Pennsylvania, Seward and New Florence (*24*). The study had the elements of a natural laboratory setting inasmuch as the towns were virtually identical demographically. These towns, inhabited by about 1,000 persons each, are approximately 4 miles apart, with a soft-coal-burning electric power plant between them. The prevailing wind pattern was such that the town of Seward was subject to much higher levels of air pollution than New Florence. Thus, for the period of the study, the level of dustfall in Seward was three times that of New Florence, the level of sulfation was seven times as high, and the level of SO_2 was

at least nine times as high. Nevertheless, the SO_2 level in Seward was below that generally found in London (*25*).

The purpose of the study was to determine the long-term effects of low concentrations of air pollutants. An attempt was made to include the entire adult population of both sexes 30 years old and over. In addition to X-rays, the study used the long Medical Research Council questionnaire, with slight revisions, chiefly in terms of a much more detailed work-experience history. A battery of pulmonary function tests, including the body plethysmograph, was administered to the study group. The analysis, which was scheduled for completion in the summer of 1962, takes into account such variables as smoking and occupational and residence exposure of the townspeople.

In the preliminary report, one significant finding was that the average airway resistance (measured by the body plethysmograph) was higher in Seward than in New Florence even after differences in height and age were taken into account.

A curious finding was that the male population of the polluted area was almost 1 inch shorter than that of New Florence. One would rightfully hesitate to attribute this difference in height to the difference in the environment. Yet this possibility should not be dismissed arbitrarily because of its apparent implausibility. One may only say that differences of this sort would have to be documented in many other communities before we could accept the hypothesis that the stature of the inhabitants was related to exposure to air pollutants rather than to ethnic or socioeconomic differences.

Since this study was completed, considerable effort has been made by the industry to reduce the pollution in the area. A restudy some time in the future might prove of considerable interest in evaluating the possible benefits of such reduction in pollutant levels as may have been achieved.

The long-term effects of the Donora disaster have also been studied in the United States (*26*). The resurvey of Donora 10 years after the disastrous smog of 1948 has shown that the persons who became ill during the outbreak have had a less favorable morbidity and mortality experience than the persons who were not affected in 1948. While it is true that those who became ill were probably less healthy to begin with than those who did not, it is quite likely that chronic effects due to unusually high levels of air pollution have been manifested in the affected group. Further, it is possible that repeated exposure to air pollution, even at very low levels, may have contributed to the long-term unfavorable experience.

The responsibility of air pollutants for the increasing frequency of lung cancer in the United States is at the moment a matter of some disagreement. Authoritative quantitative estimates of the role of air pollution as an etiologic agent do not exist, and only informed guesses can be made. Nevertheless, it is our thesis that, without decrying the importance of cigarette smoking as a factor, air pollution is also an important etiologic agent. This is not a novel idea. The World Health Organization report on lung cancer mentioned a number of possible etiologic agents and noted the prominence of air pollution in the list (*27*). Once again the sharp urban-rural differential in mortality rates for this disease is manifested. Also, lung cancer mortality rates appear to be related to the size of the urban area, the larger areas having the higher age-standardized mortality ratios.

The studies by Dean (*28*) and Eastcott (*29*) on migrants from Britain to South Africa and New Zealand suggest the role of air pollution as a causative factor in lung cancer. Unfortunately, there is no completed comparable study as yet in the United States. A study of British and Scandinavian migrants to the United States is underway, but we will have to wait several years for the results.

Conclusions

The great volume and variety of air pollutants in the United States offers unparalleled opportunities to study the chronic effects of low-level air pollution on health. In what other country is there amassed the concentration of automobiles found in Los Angeles with its resultant oxidant type of smog? The lethal concentration of pollutants in Donora in 1948 created a far different air pollution problem than is found currently on the west coast. In the United States, air pollution arises from

many and varied sources in every category, industrial, residential, municipal, and automotive, and makes necessary a wider range of research activities than in many other countries.

In summary, we in the United States are devoting considerable time and effort to this important question: Does longtime exposure to low concentrations of air pollution result in adverse health effects? Our preliminary answer, based on both laboratory and epidemiologic studies, is yes. The evidence as yet is only qualitative; much more will have to be done before the necessary quantitative answers are found on which to base rational control standards. Hopefully, with the data the Public Health Service is able to collect and that amassed by researchers in Europe and throughout the world, this goal can be attained.

REFERENCES

(*1*) Stokinger, H. E., Wagner, W. D., and Dobrogorski, O. J.: Ozone toxicity studies. III. Chronic injury to lungs of animals following exposure at a low level. A.M.A. Arch. Indust. H. 16: 514–522, December 1957.

(*2*) Ball, C. O. T., et al.: Survival of rats chronically exposed to sulfur dioxide. Physiologist 3: 15, August 1960.

(*3*) Amdur, M. O., Silverman, L., and Drinker, P.: Inhalation of sulfuric acid mist by human subjects. A.M.A. Arch. Indust. Hyg. & Occup. Med. 6: 305–313, October 1952.

(*4*) Amdur, M. O., Melvin, W. W., Jr., and Drinker, P.: Effects of inhalation of sulfur dioxide by man. Lancet 2: 758–759, October 10, 1953.

(*5*) Research in air pollution; conference report. Pub. Health Rep. 75: 1173–1189, December 1960.

(*6*) Hofreuter, D. H.: The automotive exhaust problem. A.M.A. Arch. Environ. H. 2: 559–563, May 1961.

(*7*) Hofreuter, D. H., Catcott, E. J., and Xintaras, C.: Carboxyhemoglobin in men exposed to carbon monoxide, A.M.A. Arch. Environ. H. 4: 81–85, January 1962.

(*8*) Rose, A. H., Jr., Stahman, R. C., and Stevenson, H. J. R.: Exhaust contamination in passenger cars. Technical Report A61–2. Robert A. Taft Sanitary Engineering Center, Public Health Service, Cincinnati, Ohio, February 1961.

(*9*) U.S. Public Health Service: Six years of research in air pollution, July 1, 1955, to June 30, 1961. Washington, D.C., 1961, (*a*) p. 196, (*b*) pp. 204–208, (*c*) p. 137.

(*10*) Clayton, G. D., Cook, W. A., and Fredrick, W. G.: A study of the relationship of street level carbon monoxide concentrations to traffic accidents. Am. Indust. Hyg. A. J. 21: 46–54, February 1960.

(*11*) Wiseley, D. V., Kotin, P., Fowler, P. R., and Trivedi, J.: The combined effect of repeated viral infection on pulmonary tumor induction in C57 black mice. Proc. Am. A. Cancer Res. 3: 278 (1961).

(*12*) Kotin, P.: Combination of stimuli in experimental lung cancer. Presented at the Fifth Air Pollution Medical Research Conference, Los Angeles, Calif., December 4–7, 1961.

(*13*) Greenburg, L., et al.: Report of an air pollution incident in New York City, November 1953. Pub. Health Rep. 77: 7–16, January 1962.

(*14*) Greenburg, L., Field, F., Reed, J. I., and Erhardt, C. L.: Air pollution and morbidity in New York City. J.A.M.A. In press.

(*15*) Schoettlin, C. E., and Landau, E.: Air pollution and asthmatic attacks in the Los Angeles area. Pub. Health Rep. 76: 545–548, June 1961.

(*16*) Zeidberg, L. D., Prindle, R. A., and Landau, E.: The Nashville air pollution study: I. Sulfur dioxide and bronchial asthma; a preliminary report. Am. Rev. Resp. Dis. 84: 489–503, October 1961.

(*17*) Phelps, H. W., Sobel, G. W., and Fisher, N. E.: Air pollution asthma among military personnel in Japan. J.A.M.A. 175: 990–993, March 18, 1961.

(*18*) Dohan, F. C.: Air pollutants and incidence of respiratory disease. A.M.A. Arch. Environ. H. 3: 387–395, October 1961.

(*19*) Ogilvie, A. G., and Newell, D. J.: Chronic bronchitis in Newcastle-Upon-Tyne. Livingstone, Ltd., Edinburgh and London, 1957, p. 2.

(*20*) U.S. National Office of Vital Statistics: Vital Statistics of the United States: 1950, 1959. U.S. Government Printing Office, Washington, D.C., 1952, 1961.

(*21*) Prindle, R. A.: Some considerations in the interpretation of air pollution health effects data. J. Air Pollut. Control A. 9: 12–19 (1959).

(*22*) U.S. Social Security Administration: Disability applicants under the Old-Age Survivors and Disability Insurance Program, 1960. Social Security Administration, Washington, D.C., January 1962, tables 10 and 11.

(*23*) Williams, G.: Program possibilities in respiratory diseases. Presented at the 58th annual meeting of the National Tuberculosis Association, Miami Beach, Fla., May 20–23, 1962.

(*24*) Prindle, R. A., et al.: Comparison of pulmonary function and other parameters in two communities with widely different air pollution levels. Am. J. Pub. Health. In press.

(*25*) World Health Organization: Air pollution. WHO Monograph Series No. 46, Geneva, 1961.

(*26*) Ciocco, A., and Thompson, D. J.: A follow-up of Donora ten years after: methodology and find-

ings. Am. J. Pub. Health 51: 155–164, February 1961.
(27) World Health Organization: Epidemiology of cancer of the lung; report of a study group. WHO Technical Report Series No. 192, Geneva, 1960, pp. 5–6.
(28) Dean, G.: Lung cancer among white South Africans. Report on a further study. Brit. M.J. 16: 1599–1605, December 16, 1961.
(29) Eastcott, D. F.: The epidemiology of lung cancer in New Zealand. Lancet 1: 37–39, January 7, 1956.

Program Notes

Fluoridation has reduced tooth decay among children in St. Paul, Minn., by 35 percent in the past 5 years. This conclusion is based on results of two dental surveys made on more than 20,000 pupils 5–15 years old.

« »

Three hundred and two cases of "battered child syndrome" (serious injury to children caused by severe beating or other mistreatment) were reported in 1 year by 71 hospitals; 33 children died and 85 suffered permanent brain injury. Reports from 77 district attorneys counted 447 cases during 1 year; 45 children died and 29 suffered permanent brain damage, according to C. H. Kemp, F. N. Silverman, B. F. Steele, W. Droegemueller, and H. K. Silver in the *Journal of the American Medical Association*, July 7, 1962.

« »

In "The Effects of Smoking," *Scientific American*, July 1962, Dr. E. Cuyler Hammond summarizes all the important statistical studies made to date, and also gives an account of recent studies of the direct physical effects of tobacco smoke on human tissue throughout the body.

« »

More than $50 million in tax money is used every year for the removal of litter along major highways. The U.S. Forest Service budgets $2½ million each year for sanitation and litter removal from the national forests. Litterbugs cost Los Angeles County taxpayers more than $7 million annually. Washington, D.C., spends nearly $3 million for cleanup.

« »

Every year about 600 U.S. motorists are killed by carbon monoxide fumes, reports the National Safety Council. CO may also be a factor in some of the 180,000 accidents a year caused by drivers who black out at the wheel.

« »

The United States now has 262,399 doctors, a net gain of 4,500 in 1961, compared with a net gain of 4,330 in 1960, according to the American Medical Association. Licenses to practice medicine were issued to 8,023 persons in 1961. Approximately 3,500 physicians died that year.

« »

Ten deaths due to parathion occurred in Florida during 1961. The Florida Pest Control Association sued to prevent the State board of health from enforcing regulations to control highly toxic pesticides, contending that this was a responsibility of the department of agriculture.

While the State board of health was prevented from enforcing safety controls, it alerted the public to recognize the dangers in using highly toxic materials and urging precautions.

« »

In the United States, about 22 million persons, 13 percent of the general population, are edentulous. Of persons over 35 years old, 30 percent have no teeth. (*Dental Abstracts* 7: 400, July 1962.)

« »

About 160,000 Louisiana and Texas cattle lost an average of 10 pounds each while trying to elude mosquitoes in the spring of 1962. Losses were estimated at $1.6 million, not counting the value of hundreds of animals which died as a result of the pests. Some cattle suffocated when insects clogged their nostrils; calves were mired trying to evade the pests.

« »

A 20-bed unit for emotionally disturbed blind children has opened in the children's section of Creedmoor State Hospital, Queens Village, N.Y.

« »

The Future Doctor Program at the North Conway (N.H.) Memorial Hospital each fall offers "learn and earn" training to a few mature high school students with good grades and an interest in studying medicine. Candidates are recommended to the hospital by local high school authorities. Preference is given juniors; seniors are second choice. The hospital administrator interviews all candidates for his selection.

Students report to the hospital each afternoon for 1 week early in the school year to learn basic nursing techniques. They are then given on-the-job instruction in outpatient and emergency departments on the nursing floors. Usually students work every other weekend and vacation day during the school year, and are given preference for full-time work during summer vacation. Participants, paid on an hourly basis, buy and maintain their uniforms.

This program was chosen as a blue ribbon exhibit by the New England Hospital Assembly.