

**Statistics Needed for
Determining the Effects of
the Environment on Health
Report of the Technical Consultant Panel
to the United States National Committee
on Vital and Health Statistics**

Recommendations concerning which statistical data on health effects of the environment should be collected, the ways in which recommended data should be collected, and any changes that should be made in the existing measurements of environmental factors in order to relate the environmental factors to health effects more efficiently.

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FOREWORD

This report, prepared under the auspices of the U.S. National Committee on Vital and Health Statistics, considers the needs for collecting statistics required in determining the health effects of a broad variety of environmental conditions. Although there is increasing public awareness of the importance of the environment on the health status of the U.S. population, there has been no systematic national effort to assess the problems. To meet this urgent need, this report was commissioned by the National Center for Health Statistics, the central health statistics collection unit for the Nation.

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STATISTICS NEEDED FOR DETERMINING THE EFFECTS OF THE ENVIRONMENT ON HEALTH

CHARGE TO CONSULTANTS

The past few decades have witnessed great changes in the physical and chemical contaminants of the human environment and an increasing recognition of the impact of both the physical and social environment on the health of human beings. Effects of environmental exposures cannot be isolated from effects of demographic factors and such habits as smoking. Therefore, studies of the effects of these changes in the environment on the health of the population have been inadequate to provide guidance for policies affecting control and protection.

The consultants on the Technical Consultant Panel to the U.S. National Committee on Vital and Health Statistics will recommend which statistical data on health effects of the environment should be collected, the ways in which recommended data should be collected, and any changes that should be made in the existing measures of environmental factors in order to relate better the environmental factors to the health effects.

The consultants will consider the major environmental contaminants known or suspected to significantly affect health, and will specify the nature of the health effects and offer evidence relevant to such statements. In doing so, the panel should consider such problems as low levels of exposure over long periods of time, variation in population susceptibility, interaction and synergistic effects, delayed manifestations of effects over many years or even over generations, the sequences of functional physiologic and pathologic response, and contrasts of exposure due to population mobility.

The consultants will review, for such en-

vironmental factors, the kinds of data now available on health effects, specify the data and analyses needed to estimate the health effects, and indicate the methods by which the data could be obtained in categories of methods such as the following:

Analysis of routinely collected data on the health of the population.

Special types of analysis which will aid in detecting and monitoring health reactions.

Special surveys of occupational and other exposed groups.

Disease registries in selected populations.

Monitoring of exposures and of health reactions.

Deficiencies of existing systems of measuring exposure should also be noted.

Listed below are the types of conditions and data that should be useful:

Relatively uncommon conditions with relatively high specificity, e.g., mesothelioma in relation to asbestos.

Relatively common conditions with poor specificity—e.g., coronary heart disease in relation to temperature, stress, or carbon monoxide exposures.

Overall patterns of mortality and morbidity, such as excess daily mortality, time of year effects, urban-rural differences, and consequences of migration.

Because of the wide variety of environmental contaminants, the consultants may restrict their consideration to those with the more deleterious health effects. When recommendations for collection of statistical data on health

effects have been prepared, priorities should be assigned in terms of the probable frequency and severity of the health effects in the population, and the relevance of such data for control and for prevention.

FINDINGS AND RECOMMENDATIONS

FINDINGS

Experience indicates that preventing those environmental conditions that affect human health is one of the most cost-effective strategies for preventing disease and impairment.

A large but difficult to estimate portion of the burden of such diseases as cancer and heart disease can be attributed to environmental exposures.

Occupational exposures to specific materials, because they are usually more intense and better defined than community exposures, can indicate agents that negatively affect health in the community—causing disease and impairing health or performance.

Identification of relationships between health and environmental conditions requires extensive epidemiological analysis of health and environmental statistics as well as the collection of new relevant data.

Potentially useful health statistics are available, but are not fully applied to environmental problems. Among these are occupation and industry statements recorded on death certificates, and data on industry and occupational morbidity and mortality available from the Social Security Administration, industrial organizations, and health care systems. Potentially useful data concerning environmental exposure are also available but are not fully linked to health status information. Sources of such data include the Environmental Protection Agency (EPA), the National Institute of Occupational Safety and Health (NIOSH), and the Food and Drug Administration (FDA).

Local health planning activities, especially those in health service agencies, need to evaluate their environmental health programs and activities on the bases of health statistics as well as on environmental exposure data.

RECOMMENDATIONS

The recommendations deal with several general topics, as follows:

The role and responsibility of the National Center for Health Statistics (NCHS) in regard to environmental health statistics and epidemiological studies of the environment as it affects health.

The improvement and standardization of statistical methods applicable to the study and monitoring of environmental health.

The extension and broadening of NCHS's current survey procedures to include more measures of environmental exposures.

The facilitation and performance of epidemiological studies using available data.

The establishment of new data systems specifically designed to collect information about environmental effects on health.

The following are the specific recommendations made by the panel:

1. Establish a national death index to permit determination of whether members of a cohort of persons of known environmental exposure have died, and if dead, where and when the death occurred.
2. Formally recognize the epidemiologic responsibilities and substantially strengthen the epidemiologic capability at NCHS; some specific responsibilities of such a program are
 - 2.1 Plan and carry out epidemiologic analyses of data obtained by the Center or other agencies concerned with the environment.

- 2.2 Plan, conduct, and demonstrate the usefulness of epidemiologic monitoring for prevention of unfavorable health effects of existing environmental exposures. Such procedures offer a possibility, as yet unproven, for evaluating new materials and technology whose health consequences are not otherwise known.
 - 2.3 Facilitate and support the pooling and interpretation of appropriate data among all Federal, State, and academic institutions concerned with environmental health phenomena.
 - 2.4 Provide increased opportunities for epidemiologists to use NCHS data for studies of the environment and health.
 - 2.5 Improve timeliness and accessibility of the annual national mortality summaries so that rates by time, area, age, race, sex, and other factors may be quickly and easily provided. This also applies to the 10 percent national monthly sample of deaths and will allow unusual patterns to be noted quickly by epidemiologists.
3. Provision by the NCHS of data in such format, detail, and timeliness that epidemiologic analysis can focus on environmental health problems by such procedures as
 - 3.1 Identification of excesses in mortality and morbidity in occupational groups.
 - 3.2 Analysis of variation in morbidity and mortality by age, race, sex, economic status, time of year, and geographic area in order to detect or obtain evidence pointing toward environmental effects.
 - 3.3 Detection of disease or impairment gradients which may be related to community air quality, including sulfur oxides and particulate matter, photochemical oxidants, and pollutants emitted from mines, mills, or smelters.
 - 3.4 Analysis of daily mortality in relation to environmental factors within and between selected metropolitan areas.
 - 3.5 Detection of disease or impairment which may be related to water quality including the presence of organic compounds, "hardness," heavy metals, and viral and other microbial agents.
 - 3.6 Early identification of rare types of mortality and morbidity associated with environmental exposures.
 - 3.7 Identification, in association with occupational exposures, of disease or impairment which may also be occurring as a result of community exposure.
4. Obtain data from the 1980 census on occupational and environmental exposures with the following objectives:
 - 4.1 To obtain occupational mortality rates by cause, adjusted (or specific) by age, race, and sex.
 - 4.2 To obtain income, education, or other socioeconomic gradients by cause, adjusted (or specific) by age, race, and sex.
 - 4.3 To obtain differential mortality rates by cause in locations with contrasting levels of air or water pollution or of urbanization, using age, race, sex-adjusted (or specific) data.
 - 4.4 To obtain by means of census or health survey procedures, data on differences by location in cigarette smoking, alcohol and drug use, and in dietary composition and motor vehicle use, comparable in time and location to data obtained in items 4.1-4.3 as just listed.
 - 4.5 To obtain, at least on a pilot basis, data on usual occupation and duration of residence in a given community from the 1980 census for comparison with death certificate data.
5. Develop and standardize statistical and data collection procedures relevant to environmental exposures for application to mortality data, data from the U.S. Bureau of the Census, and data collected by other government agencies. These

should be designed to permit comparisons with information from other countries, such as that from the United Kingdom—Registrar General's decennial report.¹

This effort will require a review of codes and coding procedures, particularly with respect to occupation, housing, and migration, for both the Center and the 1980 U.S. census.

6. Establish as ongoing activities of NCHS's Health Interview Survey (HIS) and Health Examination Survey (HES) the collection of data on environmental exposures and morbidity or other environmentally relevant effects including, but not limited to:
 - 6.1 Collection of water, food, or other samples which reflect environmental exposures in individual households.
 - 6.2 Sampling of human tissues or excreta to measure exposure to pollutants. Measurements must be based on population samples from which results can be extrapolated to defined populations. For pollutants that are stable in the body, estimation of the body burden offers several advantages over estimation of exposure based on analysis of environmental samples; these advantages include the ability to estimate the effects of multiple routes of exposure, more valid estimation of dose, and the opportunity to observe fluctuations in body burden and to detect active metabolites.
 - 6.3 Analysis of data on physiological, biochemical, or psychological reactions which may be related to environmental exposures.
7. Increase efforts to determine what proportion of morbidity is due to conditions at the place of employment. The experience in the California Health Department and the survey results in Oregon and Washington published by NIOSH² provide examples of practical and valid methods. The objectives would include
 - 7.1 Estimation of the extent and kinds of occupational disease as a means for its prevention and for equitable attribution of its costs.
 - 7.2 Delineation of problem areas reflecting possible inadequacies of health standards.
 - 7.3 Provision of information from pilot studies and from the Health Interview Survey to be used in studies of the prevention of occupational disease.
 - 7.4 Initiation of studies of long-term effects of carcinogenic, mutagenic, and teratogenic agents in the workplace.
8. Local health planning agencies should use mortality statistics and hospital admission and discharge data, as well as special surveys, to determine the amount and type of environmentally related disease present in their locality, and trends thereof. This should include
 - 8.1 Use of comparative mortality data by county. Such data are already published by site for cancer³ and are to be published for other diseases.
 - 8.2 Identification of environmental components and the use of this information for local health planning for the following diseases: cancer of urinary and respiratory tract, skin cancer, lymphoma and leukemia, bronchitis, asthma, emphysema, and other respiratory conditions. In interpreting data, the interaction between environmental exposures and smoking must be considered.
 - 8.3 Support by health service agencies on the development of local data on occupational injury and disease in relation to industrial employment and pollutant exposures.
 - 8.4 Epidemiologic monitoring based on such data to help evaluate local achievements in environmental health.

TYPES OF STATISTICAL INFORMATION AND ANALYSES CONSIDERED BY PANEL OF CONSULTANTS

The panel first considered routinely collected vital statistics, many of which are directly applicable to determining the health effects of environmental exposure since many specific causes of death imply a relevant environmental exposure. Included among these causes of death are the specific occupationally related conditions such as pneumoconioses and the specific intoxications and accidents that are related to environmental conditions.

Furthermore, many deaths and illnesses are related to environmental exposures, for example, those due to cancer, heart disease, and respiratory conditions. The proportion of cancer by site attributable to occupation and other environmental conditions according to "sound etiological hypotheses" is discussed in more detail later in this report (see subsection "Cancer" under "Types of Disease and Impairment to Which Environmental Contaminants Contribute"). The tabulation presented there based on an analysis by Higginson and Muir⁴ of the International Agency for Research on Cancer, reflects worldwide data and may represent a substantial understatement of the local effects of environmental conditions. Higginson has also said that, compared to the lowest cancer experience in a developed country, most other areas show an excess which suggests that 80 percent of cancer has an environmental origin.

It is necessary that other data not now included in the vital statistics system be utilized in looking for effects of environmental exposures. These include data on mortality among social security recipients and data on disability among social security groups. These data can be classified by industry, location, and in the case of disability, by occupation and cause of disability. Other sources of data include the information available through cancer registries and through registries of other specific diseases or agents, data obtained from various pension and insurance systems, data obtained from the Veterans Administration and from the military, and morbidity data obtained from school health and occupational health programs.

Potentially useful data, but in need of evaluation are

Reports obtained in coroners' offices from large cities that might reflect short-term fluctuations in mortality associated with environmental changes. (See appendix III.)

Data on the occurrence of occupational injury and occupational disease. These may be useful in epidemiologic monitoring. (See also appendixes I and II.)

The panel next considered the need for more adequate epidemiologic analysis of data which are available from various sources. Such analysis might detect differences among States, counties, or communities within a large metropolitan area in order to determine whether there are "hot spots" in one or more locations that would offer leads for a possible environmental cause. Such data have been analyzed with respect to cancer by a group at the National Cancer Institute^{5,6} and the implications are now being assessed by that Institute, the Environmental Protection Agency, and others. In addition, the intensive long-term study of statement of occupation recorded on death certificates in the State of Washington provides a useful form of analysis.⁷ The decennial reports of the Registrar General's Office in Great Britain¹ suggests additional analyses which should be considered in the United States. The objective of this type of analysis is to define the need for environmental exposure information applicable to environmental control measures to reduce exposures as well as the need for conducting prospective and retrospective epidemiologic studies.

The third major type of work considered by the panel involves the collection and interpretation of new data in which both environmental exposure and health reactions are obtained for defined populations or population cohorts. These represent a more active application of epidemiologic skills and staff to the problems of determining health effects associated with environmental conditions. The panel strongly recommends that such work be systematically undertaken using a variety of sources of health statistical information.

ELABORATION OF RECOMMENDATIONS

Recommendation 1: National Death Index

Recommendation 1 supports a national death index. This would provide information on whether or not a given person has died in a specified year in which death-registration area. Such a registry would be of invaluable assistance to environmental and other epidemiologists.

Recommendation 2: Strengthened Epidemiologic Capability of NCHS

The use of health statistics to determine the health effects of environmental conditions is fundamentally an epidemiologic problem, and yet there is no epidemiologic unit in the National Center for Health Statistics. The responsibilities of such a unit need not be limited to environmental health problems. Accordingly, the panel recommends that such a unit be established and that it be charged to carry out the epidemiologic analysis not only of data obtained in the Center but also of data available through other components of the Department of Health, Education, and Welfare, such as the Social Security Administration, and through other branches of government and of industry. Responsibility for such a program is authorized by the Federal legislation empowering the Center and the U.S. National Committee on Vital and Health Statistics to collect and analyze statistics on the determinants of health.

In addition NCHS should take an active role in bringing together the various organizations and agencies which can provide relevant data on a national or local basis and promote the use of comparable geographic and population bases for data on environmental exposures. Such efforts have been greatly needed and it is a natural function for the Center to carry them out.

There should be a provision for epidemiologic studies and monitoring to be carried out within the Federal Government by persons on short- or medium-term assignment. Such assignments could come from personnel in other Federal agencies, from State or local governments through the Intergovernmental Personnel Ex-

change Act of 1971, or from international assignments. Thus, we recommend that the epidemiologic unit provide opportunities for epidemiologists to work on any aspect of the Federal health statistical system, and we further recommend that the unit provide service for epidemiologists who are working elsewhere on environmental problems.

Recommendation 3: Provision of Data by NCHS

The charge to the panel provides guidelines to specify the sorts of data which are likely to indicate environmental contributions.

The certificate of death generally includes information on occupation, but such information is not being systematically utilized. The experience in the States of California and Washington and in Great Britain demonstrates that occupational analysis is worthwhile and the panel recommends (recommendation 3.1) that such analyses be made available. At the panel's request, the staff of NCHS prepared a review of a 0.167-percent systematic sample of the death records for 1 year and found that there were usable occupational statements in 90 percent of male deaths in the age group 20-44, 86 percent in the age group 45-64, and 73 percent in those 65 and over.

The variation in morbidity and mortality by time and by location indicates the likelihood, but does not prove the existence of, an environmental effect. It is therefore important to detect unfavorable locations and trends to see whether there are associated environmental changes. This effort might be incorporated into a more active epidemiologic monitoring program (recommendation 3.2).

There is no doubt that community air pollution exposures can result in aggravation of bronchitis, emphysema, and asthma. Morbidity and mortality from these diseases also are strongly associated with cigarette smoking. An excess of mortality from these causes has been reported in urban areas, but morbidity data from the National Health Interview Survey fail to show an urban excess. These data have not been analyzed with regard to probable air pollution exposure. This should be undertaken (recommendation 3.3). These analyses should

include fluctuations in daily mortality (recommendation 3.4). Experience has been that analysis of daily mortality requires a population base of about a million persons. (See "Daily Mortality Data" in appendix III.)

A pilot study is presently being supported in the National Health Examination Survey to determine the possible role of water hardness in relation to cardiovascular disease. The panel believes that studies relating water constituents to disease have been undersupported and should be augmented (recommendation 3.5).

The recent detection of angiosarcoma of the liver in persons exposed to vinylchloride has focused attention on the need for detecting rare types of disease that may specifically relate to certain types of exposure. A similar situation occurred with the relationship of mesothelioma to exposures to asbestos, either in the workplace or in the community. Nevertheless, the present vital statistics system is not efficient in detecting rare disease that have a peculiar clustering or that have a substantial increase. It is necessary, therefore, to evaluate the sensitivity of the health statistics system with respect to detecting uncommon diseases and causes of death (recommendation 3.6).

Recent studies of cancer by area show that high lung cancer rates occur in both men and women in counties with nonferrous metal smelters—evidence of communitywide effects of industrial pollutants.⁶ Other examples occur with respect to lead, cadmium, and asbestos. Thus industrial health effects may imply possibly communitywide effects and should be evaluated (recommendation 3.7).

Recommendation 4: Use of Data From 1980 Census

The British Registrar General's Office has analyzed occupational-social class gradients in mortality every 10 years, not only for men but also for women by their husbands' occupation and social class. We believe that the United States has been remiss in not performing similar studies. We recommend that a carefully planned occupational and environmental analysis be done for the next 3-year pericensal period (1979-81). The guidelines for such studies could

be developed following the review of the analysis of occupation done in this country in 1950,⁸ and by reference to the most recent report of the British Registrar General's Office,¹ which dealt with the 1961 British census data. We believe that this should represent an international collaborative effort, but that the plans should include studies on drug use, smoking, and alcohol use, as well as studies involving environmental exposure estimations. In preparation for this effort, it is necessary to review the classification, coding, and tabulation procedures currently in use to ensure that they are suitable for comparative studies over time within the United States as well as between different countries.

Recommendation 5: Standardizing Data Collection

Comparability between census and vital statistics procedures has been a problem in many studies of environmental factors and their influence on health. A common and well-standardized set of procedures for obtaining, recording, and coding occupation, industry, and materials to which people are exposed can increase such comparability and improve the cost-effectiveness of the collection of health data and environmental exposure data.

The work will involve development of compatible information and coding practices with, at least, the Census Bureau, dealing with housing and migration as well as industry, occupation, and migration. (See also "Health Effects and Occupational Exposures" near end of text.) Standardization to tobacco smoking histories would also be desirable. The work could be done by task groups and should be pretested early enough to be used in the 1980 census. The experience of NCHS with coding and tabulating data on illness, disability, and death will serve to further standardize these other variables.

Recommendation 6: Data Collection by HIS and HES

The National Center for Health Statistics has not used the Health Interview Survey and Health Examination Survey with maximum effectiveness to relate environmental exposures and their effects. The panel, therefore, believes that there

should be systematic effort to obtain relevant data to establish a more adequate monitoring of environmental health effects. Such an activity could extend to the collection of data on water use and water quality as supplied in the household, food consumption practices, the use of household agents such as pesticides, and materials that present an accident hazard, such as firearms or flammables.

In addition, the Health Examination Survey could be used for the collection of physiologic, biochemical, and psychological information that may be relevant to environmental exposures. The surveys have been a major source of data on smoking practices, but the data need to be supplemented so that population distributions of smoking can be obtained by State and by occupation and socioeconomic status. The increased sample size necessary for this purpose can easily be justified.

Sampling principles must be established for estimating exposure to pollutants within large and defined population groups. Although some work of this sort has already been done by the EPA, a coordinated program is needed and the involvement of the Center in interpreting the studies is important. Such measurements will add a physicochemical dimension to evaluation of exposure and may provide early evidence of possible disease or impairment.

Recommendation 7: Morbidity and Place of Employment

No national occupational morbidity reporting system exists in the United States. Ongoing programs in the State of California have been only marginally supported (see appendix I). Nevertheless, they have yielded much information of value for the early detection of occupational hazards.⁹ Some data are also compiled by the Bureau of Labor Statistics of the Depart-

ment of Labor. The recent pilot study in the States of Oregon and Washington indicates that a high proportion of chronic conditions among working-age adults may be related to conditions of employment.² A systematic effort is needed to extend these programs and to define the true dimensions of the environmental burden of disease and impairment. Such a program can provide early clues for predicting community hazards and thus help prevent them. The strengthened occupational disease reporting system should be funded for limited followup studies and for an ongoing monitoring operation that reflects the major types of industrial and occupational hazards in the country.¹⁰

Recommendation 8: HSA's and Environmental Health

Local health service agencies (HSA's) should consider environmental health problems and activities. Some guidelines are proposed in recommendation 8. The National Center provides data on mortality by county, but additional effort will be required to comply with Federal Regulation 122.107 (c) (Federal Register 41 #60 p. 12828, March 26, 1976) which states in part that: "The agency shall assemble and analyze data concerning . . . the environmental and occupational exposure factors affecting immediate and long term health conditions."

The purpose of this report as a whole is to facilitate compliance with this requirement, but from time to time data on diseases and environmental exposures conditions should be compiled, reviewed, and discussed. The role environmental factors play in cancer, heart disease, and developmental abnormalities is presented later in the report as a means of supporting activities that abet compliance with the Federal regulation (see "Types of Disease and Impairment to which Environmental Contaminants Contribute.")

MAJOR ENVIRONMENTAL CONTAMINANTS KNOWN OR SUSPECTED TO HAVE SIGNIFICANT EFFECTS ON HEALTH

A considerable number of environmental agents are thought to affect human health significantly given appropriate circumstances. These agents, and their related effects, are

shown in table 1; the effects are classified as "definite" or "possible." "Appropriate circumstances" may consist of characteristics of the exposed population, such as preexisting disease;

characteristics of the agent, such as concentration; or other characteristics of the specific exposure, such as the presence of other agents. By no means does each exposure produce the effects described, nor have some of the "possible" effects been definitely established. Furthermore, no attempt has been made to quantify the relationships. Further information about the effects of the specific agents described can be found in the World Health Organization monograph "Health Hazards of the Human Environment."¹¹

Quantitative estimates for many of the more important effects have been established by documents designated as criteria reports. Air quality criteria reports for community exposures are available for such pollutants as sulfur oxides,¹² particulate matter,¹³ ozone,¹⁴ carbon

monoxide,¹⁵ nitrogen dioxide,¹⁶ and hydrocarbons.¹⁷ Some examples of the air quality standards based on the criteria described in these reports are given in table 2.¹⁸ Criteria reports for occupational exposures are being prepared by consultants to the National Institute for Occupational Safety and Health (NIOSH). Dates of completion of criteria reports¹⁹ for some of the substances are given in table 3.

Estimates of the numbers of persons at risk because of occupational exposure to certain agents are also shown in table 3. These were prepared by NIOSH, which has also established a "severity rating" of the health effects based on the judgment of a panel. These evaluations of the health impact of the agents are used to determine priorities for preparing and issuing criteria documents and for conducting research.

TYPES OF DISEASE AND IMPAIRMENT TO WHICH ENVIRONMENTAL CONTAMINANTS CONTRIBUTE

Selected disease categories on which environmental exposures have an impact are the subject considered now. Not all diseases and organ systems afflicted by environmental contaminants are included here, and their omission should not be interpreted as implying no environmental effect.

CANCER

A table showing estimates of the percent of cancers at selected sites for which there are "sound etiological hypotheses" was published in 1973 by Higginson and Muir⁴ (table 4). In view of reports since 1973, these estimates should be considered conservative.

Proportions of cancer attributed to occupational or other environmental factors will be higher among adult males than in the total population because a higher proportion of males are employed in industry. In addition, the overall age-adjusted incidence rate (excluding nonmelanotic skin cancer) for males is higher, estimated at 346.8 per 100,000 in the Third National Cancer Survey²⁰ compared with 270.2 for females.

For more than half the cancers observed in the Third National Cancer Survey, an external causative agent is suspected on the basis of "sound etiological hypotheses." About 5 percent of all cancers are believed to be related to occupation. Since occupational exposure to carcinogens occurs mainly in males, the implication is that about 10 percent of male cancers are related to occupation. These are conservative estimates based on available direct evidence. In addition, indirect evidence from population migration data suggests that environmental factors are not yet identified for cancer of stomach, colon, rectum, lung, and breast. Geographic variation is marked for cancer of the cervix, liver, bladder, and esophagus;²¹ and environmental factors are being sought for these sites as well.

In its publication "Cancer Rates and Risks"²² the National Cancer Institute has shown that the occurrence of cancer varies with socioeconomic status, cigarette smoking, diet, alcohol, radiation, and occupation.

Socioeconomic Status

The association with low socioeconomic status is generally quite marked for cancer of the

cervix, esophagus, and stomach, but smaller and in the opposite direction for cancer of the female breast. The lowest socioeconomic group has the highest incidence for cancers of the buccal cavity and respiratory system.

The relationships between cancer risk and socioeconomic status are not fully understood, but differences in general way of life, quality of medical care, and degree of exposure to carcinogenic materials in the environment may be contributing factors.

Smoking

No one now seriously disputes that lung cancer deaths occur much more frequently among cigarette smokers than among nonsmokers, and recent research has been directed toward elucidating the meaning of this correlation.²³ Epidemiologic studies show that cigarette smokers as well as pipe and cigar smokers have a significantly higher risk of developing laryngeal cancer than nonsmokers do, and that smoking contributes to the development of cancer of the oral cavity, esophagus, and lip. A significant association has also been shown between cigarette smoking and cancer of the urinary bladder.

Certain occupational exposures have been found to be associated with an increased risk of dying from lung cancer. Cigarette smoking appears to interact with some of these exposures to produce much higher lung cancer death rates than those which occur among nonsmokers with similar occupational exposures.

Diet and Alcohol

Observations on human populations have so far uncovered relatively few forms of cancer that can be linked with food intake.²⁴ The high incidence of cancer of the oropharynx and esophagus among residents of the far north of Sweden and Finland may be related to multiple dietary deficiencies. Iodine deficiency may be related to development of cancer of the thyroid. Undernourishment or malnourishment can contribute to the high frequency of cirrhosis of the liver and the later appearance of liver cancer among some groups of African Negroes, Chinese, Japanese, and others. Aflatoxin, a liver carcin-

ogen, has been identified as a contaminant of nuts and other foodstuffs in areas of high liver cancer incidence.

Geographic differences have suggested a relationship between the highly refined Western diet rich in starches and deficient in bulk with the elevated risks for bowel cancer in North America and Western Europe.

Cancers of the stomach and large bowel among the Japanese in Hawaii are being studied using migrant groups who experienced different lengths of exposure to their countries of origin. From studies of Japanese migrant groups, stomach cancer has been associated with consumption of foods preserved or pickled in salt; large bowel cancer has been similarly associated with the consumption of beef. The study of Scandinavian immigrants to the United States²⁵ has led to a suggestion that inadequate intake of fruit and vegetables could be related to high stomach cancer rates.

The association between excessive alcohol consumption and cancers of the buccal cavity, pharynx, larynx, and esophagus is now well established.

Radiation

There is clear evidence that radiation can cause cancer in human beings.²⁶ Although at present the number of tumors induced by artificial radiation constitutes only a very tiny fraction of all human cancer, the hazard potential will increase because of increasing use of radioactive substances in industry and medicine. Only monitoring and control activities can minimize this hazard.

Further investigations are needed to measure with greater precision the frequency of radiation-induced malignancies and to determine the relation between radiation dose, latent period, and cancer incidence rates.

Occupation

Even before the advent of the modern industrial era, an association between occupation and the occurrence of cancer was recognized. However, immense growth of modern industry has brought with it an increasing number and diversity of carcinogenic substances,

many of which are associated with particular occupations or industries. Some of the known occupational cancers are listed in table 5.²²

Mortality statistics of broad occupational groups provide additional leads (table 6).²² More detailed tables show that miners, laborers, and transportation workers have an increased risk of cancer, and that men in agricultural pursuits (nearly all of whom live in rural areas) have comparatively low mortality rates for all cancer sites except skin.⁸ These findings are confirmed by similar data collected in England and Wales.¹

The combination of a long latent period between exposure and the development of cancer, changes in exposure with time, incomplete diagnosis of some types of tumor, errors in reporting occupation in censuses and on death certificates, effects attributable to nonoccupational agents, and other factors make it difficult to detect small increases in cancer risk in specific occupation groups.

NONMALIGNANT RESPIRATORY CONDITIONS

Most of the same environmental exposures that contribute to excess rates of respiratory cancer also contribute to elevated rates of morbidity and mortality for bronchitis and pulmonary emphysema and, to a lesser extent, asthma. These three conditions, often collectively designated as "chronic obstructive pulmo-

nary disease," are found much more frequently among smokers than among nonsmokers,²³ have higher mortality among low income urban populations compared with upper income or rural residents, and show a predilection for individuals working in certain occupations.⁷ Fatal asthma is twice as common among other races as among the white race. Fatal tuberculosis is three times as common. Emphysema and lung cancer are five times as common in males as in females. While the prevalence and incidence of chronic interstitial pneumonia is more than three times that of active tuberculosis, the fatality rate is substantially less. Some of these patterns are shown in the morbidity data in tables 7 and 8 and in the mortality data in table A.^{27,28}

Mortality data by occupation for both malignant and nonmalignant chronic respiratory disease are shown for certain occupational groups in table B.⁸ Some parallelism is noted between malignant and nonmalignant respiratory diseases with respect to occupational distribution. For certain occupations, mortality is increased both for malignant and nonmalignant respiratory disease in men aged 20-64.

Six prospective studies and one retrospective study of the relative contribution that smoking makes to mortality from chronic obstructive bronchopulmonary disease permits the inference that approximately 90 percent of chronic obstructive pulmonary disease is associated with cigarette smoking.²³ A further contribution is made by variables associated with economic status and exposure to pollution.

Table A. Mortality rates for respiratory conditions associated with environmental exposures, by color and sex: United States, 1968

Condition	Total		White		Other		
	Both sexes	Male	Female	Male	Female	Male	Female
	Rate per 100,000						
Chronic bronchitis	2.7	4.3	1.2	4.6	1.3	2.0	0.5
Emphysema	12.1	20.9	3.7	22.6	4.0	9.1	1.5
Asthma	1.3	1.3	1.4	1.1	1.2	2.8	2.8
Pneumoconiosis	0.8	1.6	0	1.8	0	0.5	0
Chronic interstitial pneumonia	1.7	2.3	1.2	2.4	1.2	1.7	0.7
Malignant disease of respiratory system	31.8	53.5	11.1	54.7	11.4	44.3	8.4

SOURCE: National Center for Health Statistics: *Vital Statistics of the United States, 1968*, Vol. II, Part A. DHEW Pub. No. (HSM) 72-1101. Health Services and Mental Health Administration, Washington. U.S. Government Printing Office, 1972.

Table B shows selected examples of such occupations. Since bronchitis and asthma are often associated with environmental exposure, but are at least potentially reversible, they provide a warning of the possibility of other diseases related to such exposures and thus point the way to preventing later irreversible effects such as lung cancer. On the other hand, both groups of diseases are strongly associated with cigarette smoking, and such occupations may include a large proportion of smokers. The data shown in table B are more than 25 years old and may be reflecting occupational exposures occurring considerably farther back. Recent data are badly needed for study of the relationship between occupation and chronic disease of the lungs and other organs.

Overall mortality from chronic lung diseases has risen sharply during the period since 1950, probably reflecting the interaction of smoking, occupational changes, and increased air pollution. Since 1968, however, this rate has leveled off.

These trends are difficult to interpret since 1968 because of the increase in certification to "chronic obstructive lung" (or pulmonary) disease. These certifications are assumed to rep-

resent deaths that previously had been coded to emphysema with bronchitis, or bronchitis with emphysema. A new code, 519.3, was finally introduced in 1971, but even when deaths attributed to this code are added to emphysema mortality, the rates are not increasing as much as they did prior to 1968. Deaths allocated to this code combined with deaths allocated to the codes for emphysema and chronic bronchitis are shown in figure 1. Although overall mortality is still increasing from this group, between 1969 and 1973 the high rates for persons aged 65-85 years are accompanied by a decrease in the age-specific rates for men under 55. The change in pattern of mortality from chronic conditions among young people may be predictive of what will occur on a larger scale among the whole population. For example, the decrease in mortality from chronic nonmalignant respiratory diseases among young men may reflect a decreased influence of cigarette smoking and possibly a change in working conditions. A similar decrease in lung cancer mortality in young men in Great Britain has now extended to older men.²⁹ Among women, however, the upward trend continues at all ages, although the rates are not as high. Women have increased their use of

Table B. Observed numbers of deaths and standardized mortality ratios (SMR) for selected occupational groups exhibiting excess mortality from malignant and nonmalignant respiratory disease for males aged 20-64 years: United States, 1950

Occupational group	Mortality from malignant neoplasms of trachea, bronchus, and lung		Nonmalignant respiratory disease other than influenza and pneumonia	
	Deaths	SMR	Deaths	SMR
Barbers, etc.	95	¹ 151	29	126
Cooks, except private household	91	¹ 165	41	¹ 195
Machinists and job setters	190	¹ 138	66	125
Molders, metal	34	¹ 227	30	¹ 500
Painters (construction), paperhangers, and glaziers	212	¹ 167	64	² 133
Taxicab drivers and chauffeurs	77	¹ 188	36	¹ 225
Laborers, wood products, etc.	414	¹ 138	20	154
Laborers, primary metal industries	82	¹ 167	49	¹ 258
Laborers, transportation equipment	30	¹ 200	26	¹ 433
Operatives, etc., primary metal industries	77	¹ 145	41	¹ 195

¹SMR significantly above 100 at $p \leq .01$.

²SMR significantly above 100 at $p \leq .05$.

SOURCE: National Office of Vital Statistics: Mortality by occupation and cause of death among men 20-64 years of age: United States, 1950, by L. Guralnick. *Vital Statistics—Special Reports*, Vol. 53, No. 3. Public Health Service, Washington, D.C., Sept. 1963.

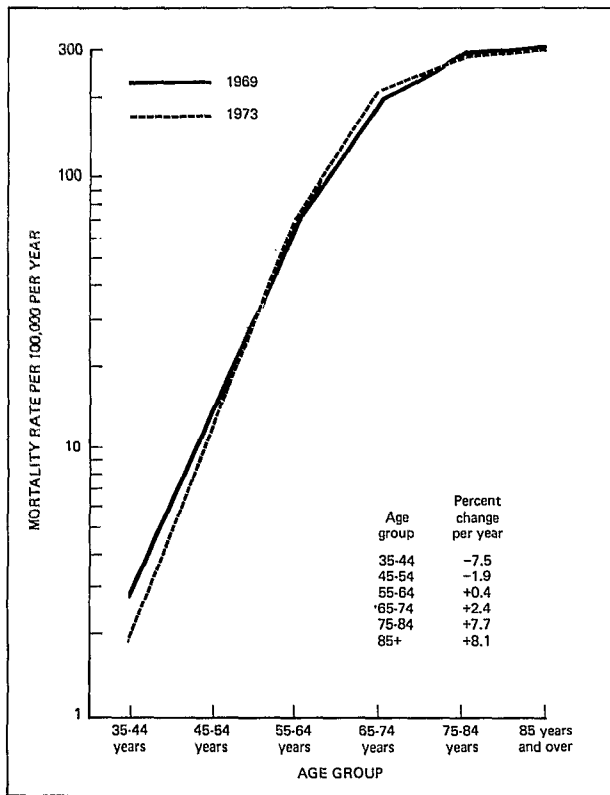


Figure 1. Age-specific mortality for white males from emphysema, bronchitis, asthma, and obstructive lung disease: United States, 1969 and 1973

cigarettes in many age groups, but the proportion of men who smoke has declined in every age group.³⁰

CARDIOVASCULAR CONDITIONS

The epidemiologic understanding of the determinants of adult-acquired, noninfectious heart disease, particularly coronary heart disease (CHD), is probably as advanced as that for any chronic disease. The list of identified personal risk factors is long and includes personal habits such as diet and cigarette smoking that interact with the environment, as well as blood pressure, serum cholesterol, blood glucose, uric acid, body mass, sedentariness, and personality type. In spite of successes in understanding and predicting CHD in populations, no more than perhaps 50 percent of cases are explained by the established risk factors.³¹

Carbon Monoxide

There is convincing clinical experimental evidence that exposure to carbon monoxide (CO) and raised carboxyhemoglobin (COHb) levels exacerbate symptoms of angina, and there is laboratory evidence of myocardial ischemia with reduced exercise tolerance.³² Increased case fatality rates associated with increased COHb suggest that the prognosis of acute myocardial infarct is affected by exposure to CO. Some laboratory evidence implicates chronic CO exposure as a possible atherogenic agent. This has yet to be demonstrated in man by epidemiologic evidence.

Meteorological and Seasonal Influences

Strong statistical associations exist between season, yearly temperature changes, and mortality attributed to CHD. In temperate climates, deaths increase in winter and decrease in summer. No doubt infectious agents are a contributor. Coincident changes have been reported in serum cholesterol. Whether this is a direct effect of temperature or secondary to changes in diet, exercise, or other environmental factors is not clear. Increases occur in daily mortality in association with marked changes in temperature and humidity.³³

Water Hardness

There is a large and controversial literature on the inverse relationship between water hardness and cardiovascular disease (CVD).³⁴ Recent reviews have given attention to:

Statistical associations.

Possible mechanisms by which trace metals may cause increased susceptibility to sudden deaths from arrhythmia.

Natural experiments, i.e., the experience of selected geographic areas with respect to CVD mortality before and after changing water quality.

Social Environment

A large body of evidence suggests that the transition of a society from rural, agrarian to an

urbanized, industrialized economy is accompanied by a rise in CHD. We do not know how much of this can be attributed to increases in the risk factors just identified, how much to changes in personal habits, how much to social and psychologic stress, and how much to unidentified agents in the physicochemical environment.

Industrial-Occupational Agents

One industrial agent, carbon disulfide (CS₂), is associated with CHD in epidemiologic studies. There is also some supporting clinical and laboratory evidence suggesting a mode of action through production of hypercholesterolemia. The evidence is far from conclusive or even convincing; however, it does suggest a systematic search for occupational groups with experience analogous to that of viscose rayon workers' excess of CHD and exposure to CS₂.

One additional agent, freon, is a possible provocation of arrhythmia in occupationally exposed pathologists and in laboratory animals.

DEVELOPMENTAL ABNORMALITIES

Pregnancy Outcome

We are presently limited in our ability to describe or understand possible effects of a multitude of chemicals and other environmental factors, either independently or through interactions, on the outcome of human pregnancies. However, inability to perceive ill effects gives no assurance that they are not occurring. Several of the reproductive processes are known to be affected by exposure to toxic substances.

The maternal organism.—During pregnancy the maternal organism is more vulnerable. Detoxification mechanisms are altered and changes in protein, carbohydrate, and lipid metabolism occur which may affect the impact of environmental agents on pregnancy duration and outcome. We do not know what initiates spontaneous labor but we do recognize a group of substances that can stimulate or retard the progress of labor, and it is conceivable that substances with similar action might exist among

the large number of chemicals in the environment.

Embryonic development.—The developing product of conception within the first 8 weeks has a high and variable susceptibility to environmental influences. This is the time when a great majority of teratological effects are induced and the conditions of exposure and types of agents are of particular concern in relation to structural defects.

Fetal development.—From eight weeks after conception to birth, the fetus, generally considered less vulnerable than the embryo, is subject to adverse influences likely to lead to physical growth retardation and postnatal functional abnormalities. In experimental rodent species, transplacental carcinogens are most active during the latter half of gestation.

Infant development.—The infant may be unusually susceptible to chemicals in a variety of ways; for example, its immature metabolic system may be unable to cope with foreign substances or even with an excess of endogenous substances.

Lactation.—Certain substances can decrease the quantity of milk and alter its quality; others may be excreted in the milk, rendering it unpalatable to the infant; and still others may be excreted in the milk that can have direct toxic effects on the infant.

Postnatal function of the child.—Since certain developmental processes are not complete until after birth—e.g., physical growth, and structural and physiologic maturation of the central nervous system and some endocrine glands—it is to be expected that environmental contaminants may detrimentally influence these postnatal developments. Aside from observations on postnatal survival and growth rates, little has been done in the way of experimental studies on postnatal functional alterations induced by environmental chemicals. An exception is the increasing concern about behavioral changes that may be induced either by prenatal exposure to substances for which the pregnant mother is the intermediary or by direct exposure of the immature child.

The latter aspects of the reproductive process have received more attention than the earlier ones, for which there are no human data.

Teratogens

The cause of deviations occurring during prenatal development that are not "normal variants"—i.e., not compatible with "normal" function in the offspring, and in which genetic control is diverted from its normal course by additional factors—is a field of study that requires urgent attention. Postconception events connected with environmental factors, particularly chemical exposures, need emphasis. Diversion from normal embryonic and fetal development may take several forms. At the extreme, development may cease altogether and the conceptus dies. In less extreme form there may be retardation of intrauterine growth and development for some stage in gestation. Gross structural or functional anomalies may accompany this phenomenon.

Over 80 percent of known clinical congenital malformations and spontaneous abortions are estimated to be of unknown etiology. Roughly 12 percent can be traced to genetic factors, with a few percent more to known "environmental insults."³⁵ By contrast, it can be shown in the laboratory that a very broad range of chemical agents can produce, under the proper conditions, some type of serious developmental deviation. Thus, substances already found to be embryopathic in animals range from highly toxic substances such as anti-tumor drugs to commonplace consumer items such as aspirin.

Despite laboratory data indicating teratogenic potential for a wide variety of environmental factors in animals, only a few have been proved or are strongly suspected to be embryopathic in man. The requisite epidemiologic studies are lacking.

Those chemical factors known to be embryopathic in man include:

Methylmercury

Aminopterin

Thalidomide

Iodine deficiency (cretinism)

Steroid hormones with androgenic activity

Carbon monoxide (hypoxia)

Those factors strongly suspected of affecting human prenatal development include:

Cortisone

Vitamin A deficiency

Diethylstilbestrol

It has been difficult to obtain valid health statistics reflecting prenatal health impacts. Data on spontaneous abortion and the abnormalities associated with abortion or other abnormal pregnancy outcomes in selected groups will be of great help. Among groups of special interest would be those in which either parent had a well-characterized occupational exposure.

Somatic Effects

Alterations in the reproductive processes by exposure to certain environmental chemicals and the unique effects of mutagens and especially teratogens on the maternal-fetal biological unit describe only the earliest aspect of developmental abnormalities. Data relevant to toxic effects during reproduction are limited, compared with the potential for data on impairment due to somatic effects in the postnatal years. These effects can be observed either relatively soon after individual exposure or after periods ranging from a few months to several years.

Improper and insufficient nutrition still contributes to disease, disability, and death in a great proportion of the world's population. The high frequency of malnutrition as soon as breast milk no longer serves as the sole source of food combined synergistically with infections to which this vulnerable age group is heavily exposed makes infant mortality and developmental abnormalities an important field for study.

In connection with dietary intake and environmental factors, the special vulnerability of infants to methemoglobinemia should be studied. Ingestion of nitrates or nitrites with drinking water has been shown in infants 30-60 days old to be related to elevated methemoglobin levels.³⁶

Technological and social developments have multiplied the hazards to which populations, especially in urban areas, are exposed.³⁷ Of

particular significance are the harmful effects of chemicals on the central nervous system, whose developmental process is not completed until after birth. There is thus a critical period of vulnerability during postnatal life and infancy. In these stages of cerebral maturation, many chemical compounds in common industrial use can produce serious and irreversible damage. For example, lead poisoning in children can produce irreparable brain damage with permanent mental retardation. A high degree of environmental exposure often occurs in children in industrialized areas, and direct ingestion of lead pigment paints is also a major source of exposure.³⁸ Peroneal nerve conduction velocity has been shown to diminish with increasing blood lead levels in children living in the vicinity of a smelter.³⁹

Studies of the effects on mortality and morbidity of chronic exposures to air pollutants strongly suggest that long-term exposure of infants and children to air pollution can impair respiratory health. Aggravation of chronic bronchitis, asthma, and pulmonary emphysema have all been considered in association with community air pollution. There is evidence that bronchitis can be caused by air pollution exposures over many years.⁴⁰⁻⁴²

To minimize the obvious importance of smoking, occupation, and previous medical history on studies of respiratory mortality and morbidity, a number of epidemiologic studies on school children have been carried out. The results of such studies indicate an increase in respiratory illness in children who reside in areas of high pollution, as compared with areas of low pollution.⁴³⁻⁴⁵ It has been suggested that such increased rates of illness cause higher rates of chronic pulmonary disease in this population in later life.

DISEASE RELATED TO INGESTED WATER POLLUTANTS OR CONSTITUENTS

Waterborne Diseases

During the period 1961-70, 130 waterborne disease outbreaks occurred in the United States.⁴⁶ Waterborne disease outbreaks are no longer on the decline in the United States as

indicated in table C.^{46,47} The number of cases per outbreak is indicated in table D^{46,47} and the types of illnesses observed are listed in table E. Two agents never before associated with documented waterborne outbreaks in the United States appeared during the 1961-70 period: enteropathogenic *Escherichia coli* (EEC), associated with adult disease, and *Giardia lamblia*. The deaths reported over a 25-year period were associated with chemical poisoning, typhoid shigellosis, amebiasis, enteropathogenic *E. coli*,

Table C. Average annual number of waterborne disease outbreaks, by type of system: United States, 1938-70

Years	All systems	Public systems	Private systems
Number of outbreaks			
1971-74 ¹	25	221	4
1966-70	14	4	10
1961-65	11	3	8
1956-60	12	5	7
1951-55	10	3	7
1946-50	23	6	17
1938-45	38	12	26

¹Craun, G. F., et al.: Review of the causes of waterborne disease outbreaks in the U.S.—1971-1974. *J. Am. Water Works Assoc.* 68(8): 420-424, Aug. 1976.

²Includes public and semipublic systems.

SOURCE: Craun, G. F., and McCabe, L. J.: Review of the causes of waterborne disease outbreaks. *J. Am. Water Works Assoc.* 65(1): 74-84, Jan. 1973.

Table D. Number of cases of waterborne disease per outbreak, by type of system: United States, 1938-74

Years	All systems	Public systems	Private systems
Illness per outbreak			
1971-74 ¹	171	24,210	28
1966-70	114	166	93
1961-65	680	2,603	39
1956-60	103	207	23
1951-55	125	333	33
1946-50	114	292	43
1938-45	340	1,000	50

¹Craun, G. F., et al.: Review of the causes of waterborne disease outbreaks in the U.S.—1971-1974. *J. Am. Water Works Assoc.* 68(8): 420-424, Aug. 1976.

²Includes public and semipublic systems.

SOURCE: Craun, G. F., and McCabe, L. J.: Review of the causes of waterborne disease outbreaks. *J. Am. Water Works Assoc.* 65(1): 74-84, Jan. 1973.

Table E. Number of outbreaks and cases of waterborne disease, by type of system and illness: United States, 1961-70

Illness	All systems		Private systems		Public systems	
	Out-breaks	Cases	Out-breaks	Cases	Out-breaks	Cases
Total	130	46,374	95	6,564	35	39,810
Gastroenteritis	39	26,546	25	4,498	14	22,048
Infectious hepatitis	¹ 30	903	22	664	¹ 8	239
Shigellosis	19	1,666	16	939	3	727
Typhoid	14	104	14	104		
Salmonellosis	² 9	16,706	² 4	96	5	16,610
Chemical poisoning	9	46	7	42	2	4
Enteropathogenic <i>E. coli</i>	4	188	4	188		
Giardiasis	3	176	1	19	2	157
Amebiasis	3	39	2	14	1	25

¹One gastroenteritis outbreak also included 7 cases of infectious hepatitis.

²One gastroenteritis outbreak was preceded by outbreak of 38 cases of salmonellosis.

SOURCE: Craun, G. F., and McCabe, L. J.: Review of the causes of waterborne disease outbreaks. *J. Am. Water Works Assoc.* 65(1): 74-84, Jan. 1973.

salmonellosis, and infectious hepatitis. Figure 2 shows the relationship between typhoid fever and infectious hepatitis cases and shows that while typhoid fever generally decreased infectious hepatitis increased.

One well-documented infectious hepatitis outbreak involving 90 cases resulted from a series of events including a cross-connection and reduced pressure in the mains resulting from a fire.⁴⁸ Plants producing very clear water tend to show low bacterial counts accompanied by low incidence of viral disease.⁴⁹ Production of good quality water should be measured by several criteria, including filtered water turbidity, bacteria as indicated by plate counts and by presumptive and confirmed coliform determinations, and use of chlorination. Plants treating polluted water achieved low virus rates in cities where qualified operators produced a superior product. The investigation of the efficiency of water treatment processes for the removal of poliovirus Type 3 showed that with proper conditioning of the water, removals of over 99 percent were attained.⁵⁰

Clustering of multiple sclerosis patients in Mansfield, Massachusetts, suggested that the etiologic agent was probably the water supply.⁵¹ It was hypothesized that exposure had occurred when the patients were about 14 years old, and the incubation period was estimated to be about 23 years.

Fungi potentially pathogenic for man have

been isolated from sewage and polluted water,⁵² and thus may affect the health of sewage workers. Weil's disease (leptospirosis) is held to be a compensable disease as in infectious hepatitis. Occupational morbidity studies of sewage

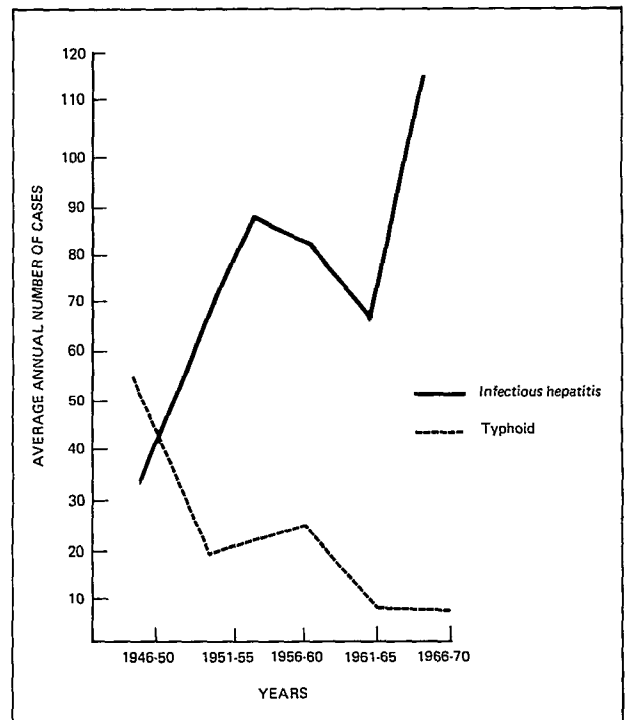


Figure 2. Average annual number of typhoid and hepatitis cases occurring in waterborne outbreaks—1946-70

workers can provide a guide for community protection as well.

Water reuse.—Four factors relevant to the utilization of reclaimed waters as a significant portion of community water supply are microorganisms, toxic minerals, nontoxic minerals, and stable organics. One article⁵³ focuses on the stable organic fraction, and proposes that bioassay techniques may be the best available means of assessing the toxicity of organics. In areas with various types of water reuse, the health statistics system should be systematically monitored for comparison with control areas.

MUTATIONS

A disturbing type of laboratory data on environmental health effects is the type dealing with mutation, the persisting alteration of the genetic materials in germ cells. Most of what is known derives from pioneering studies of radiation effects on fruit flies.⁵⁴ At present, substantial advance has led to mutagenicity

testing by use of special bacterial strains⁵⁵ and mammalian microsomes. A substantial number of known environmental mutagens are also carcinogens (135 out of 138 carcinogens tested have been shown to be mutagens).

While concern about human mutation is real and efforts to avoid it are biologically and ethically justified, we have not actually demonstrated increased stable mutations in humans due to environmental agents. What has been shown is alteration in somatic mutational indexes such as increased chromosomal breaks in lymphocytes as a result of exposure to ozone, benzene, or radiation.

Nevertheless, there is reason to believe that a proportion of birth defects is due to mutations,³⁵ and that more vigorous applications of epidemiological procedures may be able to demonstrate gradients that may reflect environmental exposures.

The health statistics system may make a useful contribution to the control and prevention of mutation by establishing improved procedures for detection and analysis of genetically related birth defects in human populations.

EPIDEMIOLOGIC METHODS APPLICABLE TO ENVIRONMENTAL HEALTH

A very brief overview is now offered of the methods of epidemiology and their more useful applications to national health data systems, particularly for identifying and assessing environmental health hazards.⁵⁶

DATA AVAILABLE FOR ANALYSES

Epidemiologic analysis of health measurements by age, race, sex, place, and time is the customary initial approach to describing any problem within the context of this report. The epidemiologic measures for any given disease may be incidence, prevalence, case fatality, or mortality. Temporal and spatial variation may reflect exposure and response to environmental pollutants. Epidemiologic analysis is also applicable to variations in states of health, as in

pulmonary function, cholinesterase levels, or body burden estimates based on blood, lead, carbon monoxide, or arsenic, lead, or mercury in hair or nails.

Stratification of a population into broad age groupings by life epochs (infancy, preschool, school, adult, retirement) provides subsets that obviously differ in many parameters—physiological, psychological, and social; however, they also differ grossly in their usual range of activity and potentials for exposure to different and unique local environments. Demonstrations of excesses of diseases and/or rapid changes within one but not others of these groups provide biologic populations indicators of responses to changing environments. The coincident decreases in infant and childhood death rates and increases in adult mortality from coronary heart disease, cancer of the lung, and

chronic obstructive pulmonary disease, particularly in white males, are graphic illustrations of this phenomenon occurring over a period of decades. A similar, more rapid change in black-white ratios of the same diseases has occurred—white excesses for some cancer sites, nationally at the time of the 1950 census, were replaced by black excesses by 1967.⁵⁷ Several competing hypotheses are raised: black entry into the urban-industrial environmental complex leading to increased exposure to deleterious physico-chemical agents; increased stress of the modern, affluent society as one of the prices to be paid for equal opportunity; and change in smoking patterns of black males.

The collection and analysis of routine vital statistics identifies some of these phenomena; their explanation requires more detailed information, possibly coming from secondary analysis of morbidity data over time in relation to person characteristics (such as that in Social Security Administration files, described in appendix IV). Although definitive answers almost invariably require *ad hoc* epidemiologic studies, much in the way of identification of problems and generation of hypotheses can be achieved by the analysis of data collected primarily for other purposes.

GROUP COMPARISONS

In general, epidemiological methods appropriate for use in examining environmental health problems are of two types. The first type involves comparisons between an exposed and a control group, or among groups with gradients in exposure. The second is concerned with comparisons within a group whose exposure varies over time.

The between-group comparison may involve groups which differ in exposure because of their place of work, their occupation, or other circumstances related to their way of life, for example, a particular hobby (as in exposure to lead in making pottery), or a personal habit (such as smoking, diet, consumption of drugs or medication). Alternatively, however, differences in exposure may be defined in terms of the general community or geographic area, such as exposure to community air pollution or com-

munity water supplies. The greatest problem in doing a study comparing the health experience of two or more groups is their comparability with respect to other significant variables. This can be approached by matching individuals on some of the more important and easily ascertained characteristics, such as age, sex, economic status, ethnic group, and smoking habits. Matching on relatively straightforward variables, however, involves a preliminary census to obtain the information necessary for matching; and this must then be followed by the actual matching and sample selection and by whatever procedure is suitable for obtaining data on the variables involved in the hypothesis being tested. More commonly, exposed and control populations are selected which are believed on more general evidence (for example, census data) to share about the same demographic characteristics but to differ with respect to the variables of primary interest (e.g., exposure to air pollution). Random sampling is then relied upon to prevent significant bias with respect to other background variables. Usually, information is also collected on these variables, and, if the two groups differ significantly, the variables are taken into account during analysis of the data, either by making comparisons within subgroups of the exposed and control groups (which requires larger sample sizes than would otherwise be needed) or by multivariate statistical analysis.

An alternative to defining the groups in terms of exposure is to define them in terms of effect, looking for differences in exposure which may discriminate among the groups.

In contrast to between-group comparisons, the study of an exposed group over time involves using the group as its own control, thus avoiding the problem of obtaining comparable exposed and control population groups. However, characteristics of these groups can change over time. The comparisons are made of variation of health experience of the group with variation in environmental exposure. This method is applicable principally to acute effects since it may involve rapidly fluctuating exposure, as in day-to-day variation in exposure to air pollution. One of the analytical problems associated with this method is that of handling temporal variation of both health effects and environmental exposure. For example, seasonal

variation in morbidity or mortality occurring coincidentally with seasonal variation in exposure may or may not indicate causation.

COMMUNITY EXPOSURE VERSUS OCCUPATIONAL EXPOSURES

The first evidence that some aspect of the environment is associated with a deleterious effect on health often occurs in an occupationally defined group. Recognition of the association is a function of several factors characteristic of the occupational setting: (1) the group is defined and accessible to followup; (2) the exposure is uniform, or similar, for the defined group; (3) the exposure may consist of substances different from those experienced by the general population; and (4) the exposure may be to a higher concentration than that experienced by the general population, resulting in more clearly definable health effects.

Community exposures, in contrast, tend to be: (1) associated with groups that are poorly defined and difficult to follow; (2) diffuse and variable over a geographically defined group; (3) related to substances at levels difficult to measure for general population exposure; and (4) related to low levels of concentrations which result in subtle or long term, if any, health effects.

However, once a substance has been shown to have health effects in an occupational or other environmental setting, it is proper to be concerned with the possible health effects of community exposures to low concentrations (for example, exposure to asbestos). In addition, community exposures may occur which have no analog in the workplace (exposure to community air pollution, to nitrates in drinking water).

HEALTH EFFECTS AND COMMUNITY EXPOSURES

The term "community exposure" is used in this report to refer to individuals whose exposure is not limited to the workplace. It includes subgroups of the population which might be at special risk because of other exposures or unusual susceptibility. These groups might include, for example, those with proximity to a

point source, and individuals of specific sex, age, ethnic backgrounds, or economic status, which could result in more than usual exposure to certain foods, types of housing, or greater susceptibility to the effects of nitrates in drinking water or to chronic respiratory disease.

Approaches to studying the relationship between health and environmental exposures in the community may be classified in several ways, for example, according to the method of selecting the population at risk, according to the analytic method to be employed, or according to the kinds of contrast to be made. For convenience, these will be discussed here under the following headings: geographic comparisons, comparisons over time, and dose-response relationships. A study may, of course, employ a combination of these.

Geographic Comparisons

Geographic comparisons are those in which the exposure is defined by geographic area. Thus, for example, the health experience of individuals living in areas with heavy air pollution may be compared with the health experience of individuals living in lightly polluted areas. One shortcoming of this kind of study is that a single measure or combination of measures of exposure is used to represent exposure of the entire group within each area. The contrasts studied may consist of high-low comparisons between two areas, or of a gradation of exposures using several areas.

If a measurement is available of the effect of a pollutant on each individual, the analysis may consist of testing for the differences in mean values between areas. The possibility also exists of using geographic comparisons to estimate a dose-response relationship by quantifying the exposure for several areas and testing for a significant gradient. In interpreting apparent geographic differences in health, it is important to take into account the possible differences in factors other than the one of primary interest. Migration and retirement rates in particular need to be considered when possible.

Comparisons Over Time

Comparisons over time are those in which the exposure is described primarily in terms of a

time interval. In air pollution studies, for example, one may relate health effects to pollution measurements representing a day (or less), a week, a month, a year, or longer. For intervals of less than a year, seasonal variation in both exposure and effect must be taken into account since coincidental seasonal variation could imply a relationship which may or may not represent a causative effect. Usually a relatively short time interval is used since pollution may vary greatly over time. The week seems to be a reasonable compromise since the day-of-the-week effect in morbidity and mortality can then be discounted. Usually correlation and regression analysis are used since the number of exposure-effect pairs tends to be large and represents many gradations of exposure. However, some kind of seasonal adjustment is usually necessary. As with geographic comparisons, it may be possible to estimate dose-response relationships provided enough levels of exposure are measured.

Temporo-Spatial Comparison

The combination of geographic comparisons with comparisons over time offers several advantages. First, if the same variation over time in relation to pollution is found in several areas, it suggests that the phenomenon does not result from variation with a third concomitant variable peculiar to a single situation. Similarly, geographic differences may be found to exist regardless of relatively small time variations in exposure. And finally, it may be found that a stronger effect of temporal variations may occur in more heavily polluted areas. Thus temporo-spatial analysis tends to yield more specific indexes of environmental effect than do either geographic (spatial) or comparisons over time (temporal).

Dose-Response Relationships

The study of dose-response relationships usually involves estimating exposure for individuals, rather than for groups, and relating this exposure to indices of health, both exposure and effects being measured on a continuous scale or at least one with many possible values. Estimation of the relationship itself then consists of fitting a curve to the data points. This could also be done with grouped data. In both these methods, observations are usually taken directly

on individuals rather than by assuming a uniform exposure on the basis of geographic location or exposure during a given period of time. For example, one might start by measuring the exposure of each individual to carbon monoxide by use of some sort of personal monitoring method; and measuring such effects as change in carboxyhemoglobin or expired air carbon monoxide concentration or visual acuity. Thus, a set of two individual measurements is obtained for each person. As described previously, dose-response analysis can also be carried out on data geographically defined or using a temporal sequence, but exposure data then become less precise.

EPIDEMIOLOGIC MONITORING AND SURVEILLANCE

Surveillance is defined here as collecting and analyzing data as it becomes available in a time sequence in order to detect departures from some established or expected value or norm. In the context of environmental health, departures from normality in either exposure (as in air pollution) or health effects are of interest. The goal is to be able to take useful and timely corrective action. In order to determine when departures from normality occur, expected values must be calculated along with an estimate of variance to provide confidence limits. One could then determine whether deviations of environmental variables are associated with morbidity or mortality. Conversely, departures from expected values of morbidity or mortality might indicate the need to look for variations in environmental exposure variables.

EFFECT OF INTERACTIVE AND CONTRIBUTORY VARIABLES

The importance of taking into account variables other than those of primary interest was mentioned earlier in the discussion of geographic variation. These variables may themselves be environmental, or may be other characteristics of the individuals being studied. One may wish merely to "control for" or eliminate the effect of other factors known or suspected to have an effect, or alternatively, these factors may be of interest in themselves, either in terms

of their own effect, or to detect interactions between them and the primary variable. The method of analysis depends upon the choice among these alternatives. If control of the other variables is the main objective, then a standardization procedure may be used, the analysis may proceed within several categories of the variables, or multivariate analysis may be used. If the effect of these variables themselves is an objective, then multivariate analysis may be preferred.

RARE EVENTS

In looking for environmental influences on health, one also needs to consider the rare health event. Rare events may result from environmental exposures, but because data need to be accumulated over long time periods or in large populations to detect an increase, associations may be particularly difficult to establish. What appear to be rare events, however, may not actually be rare; they may result from errors in coding or diagnosis of a common event. Furthermore, they may be overlooked because of lack of specificity of coding, either because they tend to be grouped with more common conditions for coding purposes or because they cannot be distinguished from them because of lack of information. For example, the information recorded on the death certificates may not be adequate for coding, as in the case of mesothelioma of pleura or peritoneum and angiosarcoma of the liver, both of which require specification of cell type. In other cases the mechanism by which a condition develops needs to be described in addition to a physical description of the result.

HEALTH EFFECTS AND OCCUPATIONAL EXPOSURES

Existing Records

Death certificates, in spite of their shortcomings, are a useful source of information on

occupational mortality. However, they currently show "last" occupation rather than "usual" occupation. Studies are needed to determine the extent this reflects the major, or at least a significant, occupational exposure. This can be deduced to some extent by comparing length in last occupation, also available on the death certificate, with age. Other alternatives are to compare data from death certificates with information from next of kin, social security information, or union records. Given that the occupation as shown on the death certificate has some validity, the next problem is to code and tabulate the information in such a way that occupational risks are adequately reflected. Several codes are now in use, and comparative work is being carried out to evaluate their usefulness. It is equally important to obtain and code information on industry worked in, since this may reflect occupational risks, in a more valid way, than occupation *per se*. One further step would be to code hazardous substances to which a worker has been exposed.

Special Ascertainment of Exposure

Detailed occupational histories should be obtained from cancer registries, union records, or social security records. An example of the latter can be found in statistical reports of occupation of disabled workers by cause of disability.⁵⁸ Interviews of surviving employees or of next of kin can be used to obtain fairly detailed histories, but these are time consuming and depend to a large degree on memory. Furthermore, interviews must be carefully structured so that information of the desired specificity is obtained. One approach is to first obtain a detailed chronological history; follow this with a check list of occupations and industries of special interest; and finally to inquire about exposures to known or suspected toxic substances. Standardized procedures for such work have been developed in a few centers, and need to be more generally utilized.



REFERENCES

- ¹General Register Office: *The Registrar-General's Decennial Supplement, England and Wales: 1961 Occupational Mortality Tables*. London. H. M. Stationery Office, 1971. p. 528.
- ²Center for Disease Control, National Institute for Occupational Safety and Health: *Pilot Study for Development of an Occupational Disease Surveillance Method*. Contract No. HSM 99-72-69. Public Health Service. Washington. U.S. Government Printing Office, May 1975.
- ³Mason, T. J., and McKay, F. W.: *U.S. Cancer Mortality by County; 1950-69*. DHEW Pub. No. (NIH) 74-615. Washington. U.S. Government Printing Office, 1973.
- ⁴Higginson, J., and Muir, C.: Epidemiology, in J. Higginson, ed., *Cancer Medicine*, New York. Lea & Febiger, 1974.
- ⁵Hoover, R., and Fraumeni, J. F., Jr.: Cancer mortality in U.S. counties with chemical industries. *Environ. Res.* 9: 196-207, Apr. 1975.
- ⁶Blot, W. J., and Fraumeni, J. F., Jr.: Arsenical air pollution and lung cancer. *Lancet* ii: 142-144, July 26, 1975.
- ⁷Milham, S., Jr.: *Occupational Mortality in Washington State, 1950-1971*, NIOSH, Washington State Department of Social and Health Services, Pub. No. (NIOSH) 76-175 (three volumes). U.S. Government Printing Office. Washington, 1977.
- ⁸National Office of Vital Statistics: Mortality by occupation and cause of death among men 20-64 years of age: United States, 1950, by L. Guralnick. *Vital Statistics—Special Reports*, Vol. 53, No. 3. Public Health Service. Washington, D.C., Sept. 1963.
- ⁹Baginsky, E.: Occupational illness and accidents reported from California shipyards. *Environ. Res.* 11: 271-279, Apr. 1976.
- ¹⁰Stanford Research Institute: *Evaluation of Existing Data Sources of Occupational Injury and Illness Statistics*. Prepared for Occupational Safety and Health Administration. Contract J-9-F-R0147. Menlo Park, Calif., June 1976.
- ¹¹World Health Organization: *Health Hazards of the Human Environment*. Geneva. World Health Organization, 1972.
- ¹²U.S. Department of Health, Education, and Welfare: *Air Quality Criteria for Sulfur Oxides*. AP-50, Washington. U.S. Government Printing Office, 1969.
- ¹³U.S. Department of Health, Education, and Welfare: *Air Quality Criteria for Particulate Matter*. AP-49, Washington. U.S. Government Printing Office, 1969.
- ¹⁴U.S. Department of Health, Education, and Welfare: *Air Quality Criteria for Photochemical Oxidants*. AP-63, Washington. U.S. Government Printing Office, 1970.
- ¹⁵U.S. Department of Health, Education, and Welfare: *Air Quality Criteria for Carbon Monoxide*. AP-62, Washington. U.S. Government Printing Office, 1970.
- ¹⁶Environmental Protection Agency: *Air Quality Criteria for Nitrogen Oxides*. AP-84, Washington. U.S. Government Printing Office, 1971.
- ¹⁷U.S. Department of Health, Education, and Welfare: *Air Quality Criteria for Hydrocarbons*. AP-64, Washington. U.S. Government Printing Office, 1970.
- ¹⁸Environmental Protection Agency: *EPA Regulations 40 CFR50*. Washington. U.S. Government Printing Office, and Commerce Clearing House, Inc.: *Pollution Control Guide*. Chicago. 1974.
- ¹⁹Council on Environmental Quality: *Environmental Quality-1975*, Seventh Annual Report of the Council on Environmental Quality, Washington. U.S. Government Printing Office, pp. 49-53, 1976.
- ²⁰National Cancer Institute: The third national cancer survey: Incidence data. *National Cancer Institute Monograph 41*. DHEW Pub. No. (NIH) 75-787, Washington. U.S. Government Printing Office, Mar. 1975.
- ²¹Doll, R.: Epidemiology of cancer: Current perspectives. *Am. J. Epidemiol.* 104: 396-404, Oct. 1976.
- ²²Levin, D. L., Devesa, S. S., Godwin, J. D., and Silverman, D. T.: *Cancer Rates and Risks*. DHEW Pub. No. (NIH) 75-691. Washington. U.S. Government Printing Office, 1974.
- ²³Center for Disease Control: *Health Consequences of Smoking*. DHEW Pub. No. (HSM) 71-7513. Atlanta. Public Health Service. 1971 and Public Health Service, Center for Disease Control: *Health Consequences of Smoking*. DHEW Pub. No. (CDC) 76-8704. Atlanta. Public Health Service. 1975.
- ²⁴Berg, John W.: Diet, in J. F. Fraumeni, Jr., ed., *Person at High Risk Cancer—An Approach to Cancer Etiology and Control*. New York. Academic Press, Dec. 1974. pp. 201-224.
- ²⁵Bjelke, E.: Epidemiological studies of cancer of the stomach, colon, and rectum with special emphasis on the

role of diet. *Scand. J. Gastroenterol.* 9 (suppl. 31): 1974.

²⁶National Academy of Sciences, National Research Council, Advisory Committee on the Biological Effects of Ionizing Radiations: *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation*. Washington, 1972.

²⁷National Center for Health Statistics: Prevalence of chronic respiratory conditions: United States, 1970. *Vital and Health Statistics*. Series 10-No. 84. DHEW Pub. No. (HRA) 74-1511. Health Resources Administration. Washington. U.S. Government Printing Office, Sept. 1973.

²⁸National Center for Health Statistics: *Vital Statistics of the United States, 1968*. DHEW Pub. No. (HSM) 72-1101. Health Services and Mental Health Administration, Washington. U.S. Government Printing Office, 1972.

²⁹Higgins, I. T. T.: Trends in respiratory cancer mortality. *Arch. Environ. Health* 28: 121-129, Mar. 1974.

³⁰Center for Disease Control: *Adult Use of Tobacco—1975*. Public Health Service. Center for Disease Control and National Institutes of Health, June 1976.

³¹Kuller, L. H.: Epidemiology of cardiovascular diseases: Current perspectives. *Am. J. Epidemiol.* 104: 425-462, Oct. 1976.

³²Goldsmith, John R., and Aronow, Wilbert S.: Carbon monoxide and coronary heart disease. *Environ. Res.* 10: 236-248, Dec. 1975.

³³Ellis, F. P., Nelson, F., and Pincus, L.: Mortality during heat waves in New York City, July 1972 and August and September 1973. *Environ. Res.* 10: 1-13, Aug. 1975.

³⁴Heyden, S.: The hard facts behind the hard-water theory and ischemic heart disease. *J. Chron. Dis.* 29: 149-157, Mar. 1976.

³⁵Crow, J. F.: Impact of various types of genetic damage and risk assessment, in *Evaluation of Chemical Mutagenicity Data in Relation to Population Risk, Environmental Health Perspectives*. DHEW Pub. No. (NIH) 74-218. Washington. U.S. Government Printing Office, 1974.

³⁶Shearer, L. A., et al.: Methemoglobin levels in infants in an area with high nitrate water supply. *Am. J. Public Health* 62: 1174-1180, Sept. 1972.

³⁷Panel on Chemicals and Health Report of the President's Science Advisory Committee: *Chemicals and Health*. Washington. National Science Foundation, Sept. 1973.

³⁸National Academy of Sciences, National Research Council Committee on Biological Effects of Atmospheric Pollutants, Division of Medical Sciences: *Lead; Airborne Lead in Perspective*. Washington, 1972.

³⁹Landrigan P.: Increased lead absorption with anemia and sub-clinical neuropathy in children near a lead smelter. *Proceedings of the International Conference on Heavy Metals in the Environment*. Toronto. Oct. 27-31, 1975.

⁴⁰Douglas, J. W. B., and Waller, R. E.: Air pollution and respiratory infection in children. *Br. J. Prev. Soc. Med.* 20: 1-8, Jan. 1966.

⁴¹Lunn, J. E., Knowelden, J., and Handyside, A. J.: Patterns of respiratory illness in Sheffield infant school children. *Br. J. Prev. Soc. Med.* 21: 7-16, Jan. 1967.

⁴²Lunn, J. E., Knowelden, J., and Roe, J. W.: Patterns of respiratory illness in Sheffield infant school children. A follow-up study. *Br. J. Prev. Soc. Med.* 24: 223-228, Nov. 1970.

⁴³Holland, W. W., Halil, T., Bennett, A. E., and Elliot, A.: Factors influencing the onset of chronic respiratory disease. *Br. Med. J.* 2: 205-208, Apr. 1969.

⁴⁴Colley, J. R. T., and Reid, D. D.: Urban and social origins of childhood bronchitis in England and Wales. *Br. Med. J.* 2: 213-217, Apr. 1970.

⁴⁵Zapletal, A., Jech, J., Paul, T., and Samanek, M.: Pulmonary function studies in children living in an air-polluted area. *Am. Rev. Respir. Dis.* 107: 400-409, Mar. 1973.

⁴⁶Craun, G. F., and McCabe, L. J.: Review of the causes of waterborne disease outbreaks. *J. Am. Water Works Assoc.* 65(1): 74-84, Jan. 1973.

⁴⁷Craun, G. F., et al.: Review of the causes of waterborne disease outbreaks in the U.S.—1971-1974. *J. Am. Water Works Assoc.* 68(8): 420-424, 1976.

⁴⁸Taylor, F. B.: The Holy Cross episode. *J. Am. Water Works Assoc.* 64(4): 230-232, Apr. 1972.

⁴⁹Hudson, H. E., Jr.: High quality water production and viral disease. *J. Am. Water Works Assoc.* 54(10): 1265-1272, 1962 and Woodward, R. L.: Discussion. *J. Am. Water Works Assoc.* 54(10): 1271-1274, Oct. 1962.

⁵⁰Robeck, G. G., Clarke, N. A., and Dostal, K. A.: Effectiveness of water treatment processes in virus removal. *J. Am. Water Works Assoc.* 54(10): 1275-1290, Oct. 1962 and Hartung, H. O.: Discussion. *J. Am. Water Works Assoc.* 54(10): 1290-1292, Oct. 1962.

⁵¹Eastman, R., Sheridan, J., and Poskanzer, D. C.: Multiple sclerosis clustering in a small Massachusetts community with possible common exposure 23 years before onset. *N. Engl. J. Med.* 209 (15): 793-794, Oct. 11, 1973.

⁵²Dixon, F. R., and McCabe, L. J.: Health aspects of wastewater treatment. *J. Water Pollut. Control Fed.* 36(8): 984-989, Aug. 1964.

⁵³Ongerth, H. J., Spath, D. P., Crook, J., and Greenberg, A. E.: Public health aspects of organics in water. *J. Am. Water Works Assoc.* 65(7): 495-498, July 1973.

⁵⁴Muller, H. J.: Artificial Transmutation of the Gene. *Science* 66: 84, 1972. pp. 84-87.

⁵⁵Ames, B. N., McCann, J., and Yamaski, F.: Methods for detecting carcinogens and mutagens with the salmonella/mammalian-microsome mutagenicity test. *Mutagen Res.* 31: 347-364, 1975.

⁵⁶Goldsmith, J. R.: Statistical problems and strategies in environmental epidemiology, in L. M. LeCam, J.

Neyman, and E. L. Scott, eds., *Proceedings of the Sixth Berkeley Symposium on Mathematical Statistics and Probability*, Vol. VI. Berkeley, University of California, 1972. ch. 1.

⁵⁷Burbank, F., and Fraumeni, J. F., Jr.: U.S. cancer mortality: Nonwhite predominance. *J. Natl. Cancer Inst.* 49: 649-659, Sept. 1972.

⁵⁸Occupational Health Program and Social Security Administration, Bureau of Disability Insurance: *Occupational*

Characteristics of Disabled Workers; by Disabling Condition, Disability Insurance Benefit Awards Made in 1959-1962 to Men Under Age 65. PHS Pub. No. 1531. Public Health Service. Washington. U.S. Government Printing Office, 1967.

⁵⁹National Bureau of Economic Research: Conference on Research in Income and Wealth, *The Anatomy of Earnings*. Ann Arbor, Mich., May 1974. Unpublished.

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Table 1. Definite and possible health effects of environmental pollutants and exposures

[Items in parentheses refer to effects other than those directly affecting human health status]

Agent, pollutant, or source	Definite effect	Possible effect
COMMUNITY AIR POLLUTION—A		
Sulfur dioxide (effects of sulfur oxides may be due to sulfur, sulfur trioxide, sulfuric acid, or sulfate salts)	1. Aggravation of asthma and chronic bronchitis 2. Impairment of pulmonary function 3. Sensory irritation	
Sulfur oxides and particulate matter from combustion sources	4. Short-term increase in mortality 5. Short-term increase in morbidity 6. Aggravation of bronchitis and cardiovascular disease 7. Contributory role in etiology of chronic bronchitis and emphysema 8. Contributory role to respiratory disease in children	9. Contributory role in etiology of lung cancer 10. Increase in chronic respiratory disease
Particulate matter (not otherwise specified)		
Oxidants	11. Aggravates emphysema, asthma, and bronchitis 12. Impairs lung function in patients with bronchitis-emphysema 13. Eye and respiratory irritation and impairment in performance of student athletes	
Ozone	15. Impairs lung function	14. Increased probability of motor-vehicle accidents
Carbon monoxide	17. Impairs exercise tolerance in patients with cardiovascular disease	16. Acceleration of aging, possibly due to lipid peroxidation and related processes
Nitrogen dioxide		18. Increased general mortality and coronary mortality rates 19. Impairment of central nervous system function 20. Causal factor in atherosclerosis 21. Factor in pulmonary emphysema 22. Impairment of lung defenses such as mast cells and macrophages or altered lung function
Lead	23. Increased storage in body	24. Impairment of hemoglobin and porphyrin synthesis
Hydrogen sulfide	25. Increased mortality from acute exposures 26. Causes sensory irritation	
Mercaptans		27. Headache, nausea, and sinus affections
Asbestos	28. Produces pleural calcification 29. Malignant mesothelioma, asbestosis	30. Contributes to chronic pulmonary disease (asbestos and lung cancer)
Organophosphorus pesticides	31. Acute fatal poisoning 32. Acute illness 33. Impaired cholinesterase activity	
Other odorous compounds		34. Headache and sinus affections
Beryllium	35. Berylliosis with pulmonary impairment	
Airborne microorganisms	36. Airborne infections	
FOOD AND WATER CONTAMINANTS—B		
Bacteria	1. Epidemic and endemic gastrointestinal infections (typhoid, cholera, shigellosis, salmonellosis, leptospirosis, etc.)	2. Secondary interaction with malnutrition and with nitrates in water (cf., no. 15)
Viruses	3. Epidemic hepatitis and other viral infections	4. Eye and skin inflammation from swimming
Protozoa and metazoa	5. Amoebiasis, schistosomiasis, hydatidosis and other parasitic infections	
Metals	6. Lead poisoning 7. Mercury poisoning (through food chains) 8. Cadmium poisoning (through food chains) 9. Arsenic poisoning 10. Chromium poisoning	11. Epidemic nephropathy 12. "Blackfoot" disease
Nitrates	13. Methemoglobinemia (with bacterial interactions)	14. Increase in cardiovascular disease
"Softness" factor		
Sulfates and/or phosphates	15. Gastrointestinal hypermotility	
Fluorides	16. Fluorosis of teeth when in excess	

Table 1. Definite and possible health effects of environmental pollutants and exposures—Con.

[Items in parentheses refer to effects other than those directly affecting human health status]

Agent, pollutant, or source	Definite effect	Possible effect
LAND POLLUTION—C		
Human excreta	1. Schistosomiasis, taeniasis hookworm, and other infections	
Sewage		2. Typhus, plague, leptospirosis, and other infectious diseases
Industrial and radioactive waste	3. Storage and effects from toxic metals and other substances through food chains	
Pesticides—lead arsenate	4. Increased storage of heavy metals in smokers of tobacco grown on treated areas	
THERMAL EXPOSURES—D		
Cold damp	1. Excess mortality from respiratory disease and fatal exposure 3. Excess morbidity from respiratory and related diseases and morbidity from exposure	2. Contributes to excess mortality and morbidity from other causes
Cold dry	5. Mortality from frostbite and exposure	4. Rheumatism
Hot dry	7. Morbidity from frostbite and respiratory disease 8. Heatstroke mortality 9. Excess mortality attributed to other causes 10. Morbidity from heatstroke and from other causes 11. Impaired function; aggravation of renal and circulatory diseases	6. Impaired lung function
Hot damp	12. Increase in skin affections 14. Heat-exhaustion mortality 15. Excess mortality from other causes 16. Heat-related morbidity 17. Impaired vigor and circulatory function 18. Aggravation of renal and circulatory disease	13. Increase in prevalence of infectious agents and vectors
RADIATION AND MICROWAVES—E		
Natural sunlight	1. Fatalities from acute exposure 2. Morbidity due to "burn" 3. Skin cancer 4. Interaction with drugs in susceptible individuals	
Diagnostic X-ray	6. Skin cancer and other skin changes	5. Increase in malignant melanoma
Therapeutic radiation	9. Skin cancer 10. Increase in leukemia	7. Contributing factors to leukemia 8. Alteration in fecundity
Industrial uses of radiation and mining of radioactive ores	14. Acute accidental deaths 15. Radiation morbidity 16. Uranium nephritis 17. Lung cancer in cigarette-smoking miners	11. Increase in other cancers 12. Acceleration of aging 13. Mutagenesis
Nuclear power and reprocessing plants		18. Increase in adjacent community morbidity or mortality
Microwaves		19. Increase in cancer incidence 20. Community disaster 21. Alteration in human genetic material 22. Tissue damage
NOISE AND VIBRATIONS—F		
Traffic Aircraft (including sonic boom)	2. Permanent hearing loss	1. Progressive hearing loss
Vibrations		3. Aggravation or cause of mental illness 4. Articular and muscular disease 5. Adverse effects on nervous system

Table 1. Definite and possible health effects of environmental pollutants and exposures—Con.

[Items in parentheses refer to effects other than those directly affecting human health status]

Agent, pollutant, or source	Definite effect	Possible effect
HOUSING AND HOUSEHOLD AGENTS—G		
Heating, cooking, and refrigeration	1. Acute fatalities from carbon monoxide, fires and explosions, and discarded refrigerators	2. Increase in diseases of the respiratory tract in infants
Fumes and dust	3. Acute illness from fumes 4. Aggravation of asthma	5. Increase in chronic respiratory disease
Crowding	6. Spread of acute and contribution to chronic disease morbidity and mortality	
Structural factors (including electrical wiring, stoves, and thin walls)	7. Accidental fatality 8. Accidental injury 9. Morbidity and mortality from lack of protection from heat or cold 10. Morbidity and mortality due to fire or explosion	
Paints and solvents	11. Childhood lead-poisoning fatalities, associated mental impairment, and anemia 12. Renal and hepatic toxicity 13. Fatalities	
Household equipment and supplies (including pesticides)	14. Fatalities from fire and injury 15. Morbidity from fire and injury 16. Fatalities from poisoning 17. Morbidity from poisoning	
Toys, beads, and painted objects	18. Mortality and morbidity	
Urban design	19. Increased accident risks	20. Contribution to mental illness

Table 2. National primary ambient air quality standards

Pollutant	Averaging time	Frequency parameter	Concentration	
			$\mu\text{g}/\text{m}^3$	ppm
Carbon monoxide	1 hour	Annual maximum ¹	40,000	35
	8 hours	Annual maximum	10,000	9
Hydrocarbons (nonmethane)	3 hours (6 to 9 a.m.)	Annual maximum	² 160	² 0.24
Nitrogen dioxide	1 year	Arithmetic mean	100	0.05
Photochemical oxidants	1 hour	Annual maximum	160	0.08
Particulate matter	24 hours	Annual maximum	260	...
	24 hours	Annual geometric mean	75	...
Sulfur dioxide	24 hours	Annual maximum	365	0.14
	1 year	Arithmetic mean	80	0.03

¹Not to be exceeded more than once per year.

²As a guide in devising implementation plans for achieving oxidant standards.

SOURCE: EPA Regulations 40 CFR 50; and Commerce Clearing House, Inc.: *Pollution Control Guide*, 1974.

Table 3. Exposure estimates, selected harmful effects, and date of criteria document for selected occupational agents

Substance or agent	Exposure estimate	Selected harmful effects	Date of criteria document if available
Noise	7,500,000	Hearing and communications impairment	1972
Lasers	120,000	Cataract	---
Hot environments	1,300,000	Heat stroke, heat exhaustion	1972
Ultraviolet radiation	320,000	Sunburn, eye irritation, skin cancer	1972
Inorganic lead	83,000	Colic, neurological impairment, anemia	1973
Carbon monoxide	2,000,000	Impairment of oxygen transport by blood	1972
Inorganic mercury	150,000	Stomatitis, tremor, psychic disturbance	1973
Beryllium and its compounds	30,000	Respiratory irritation and granuloma, chronic changes in other organs, possibly cancer	1972
(Ethyl) parathion	250,000	Headache, sweating, coma	---
Methyl parathion	150,000		
Coal tar, pitch	50,000	Skin irritation and sensitization, possibly cancer	---
Benzene	2,000,000	Bone marrow depression, leukemia	1974
Fluorides	350,000	Respiratory, eye and skin irritation	1975
Chromium	160,000	Skin irritation, sensitization, ulcer, possibly cancer	1975
Boron trifluoride and other compounds, boron	50,000	Chest symptoms, central nervous system reactions	---
Arsenic	1,500,000	Skin reactions, sensitization, possibly cancer	1975
Carbon tetrachloride	160,000	Liver and kidney failure	---
Cadmium	100,000	Gastrointestinal irritation, kidney disease, possible emphysema from inhalation	---

Table 4. Percent distribution of cancers at selected sites for which there are sound etiologic hypotheses

Site	Exogenous factors				Congenital, familial or acquired	Un-known
	Cul-tural	Occu-pational	Iatro-genic	Miscel-laneous		
	Percent distribution					
Mouth (140,141,143,144)	90	1	-	5	-	<5
Salivary gland (142)	-	-	-	-	-	100
Esophagus (150)	80	-	-	4	<1	±15
Stomach (151)	4	-	-	1	-	95
Colon and rectum (153,154)	-	-	-	1	<1	99
Liver (155.0)	70	-	-	1	-	30
Liver (155.0) Africa	-	-	-	-	-	100
Lung (162)	80	1-2	-	±8	-	<10
Breast (170)	-	-	-	-	-	100
Cervix uteri (171)	-	-	-	-	-	100
Corpus uteri (172)	-	-	-	-	-	100
Ovary (175)	-	-	-	-	-	100
Other female genitals (176)	-	-	<1	-	-	99
Prostate and testis (177,178)	-	-	-	-	-	100
Penis (179.0)	-	<1	-	95	-	<5
Bladder (181.0) Western Industrial	50	10-20	<1	-	-	30-40
Bladder (181.0) Africa	-	-	-	50	-	50
Skin (190,191)	-	2	-	80	10	<8
Brain tumors (193)	-	-	2	-	<1	98
Leukemia and lymphoma (200-205)						
Children	-	-	<7	-	1	92
Adults	-	-	<1	-	-	99

SOURCE: Higginson, J., and Muir, C.: Epidemiology, in J. Higginson, ed., *Cancer Medicine*, New York. Lea & Febiger, 1974.

Table 5. Carcinogenic agents that may be associated with various occupations

Agent	Sites of cancer	Areas where noted
Specific agents:		
Arsenic	Skin, lung	United States, Great Britain, Germany, France, Argentina, Taiwan, African countries
Coal tar, pitch	Skin, lung	United States, Great Britain
Petroleum	Skin, lung	United States, France, Great Britain, Austria
Shale oils	Skin	United States, Great Britain
Lignite tar, paraffin	Skin	Great Britain, France
Creosote oils	Skin	United States, Great Britain
Anthracene oils	Skin	Great Britain
Soot carbon black	Skin	United States, Great Britain
Mustard gas	Lung	Japan
Cutting (mineral) oils	Skin, possibly respiratory and upper alimentary tract	Great Britain, Australia
Products of coal carbonization	Lung, bladder	Great Britain, United States, Japan
Sunlight	Skin	United States, Argentina, Australia, France, et al.
Chromates	Lung	United States, Great Britain, Germany, Canada
Asbestos	Lung, pleura, peritoneum, gastrointestinal tract	United States, Great Britain, Germany, Canada, S. Africa, Holland, Australia, USSR, Italy, et al.
Aromatic amines, dyes, chemicals used in rubber industry	Bladder, possibly biliary tract, salivary glands	United States, Germany, Great Britain, Switzerland, et al.
X-rays, radium	Skin, lung, leukemia	United States and many other areas
Nickel	Lung, nasal cavity and sinus	Great Britain, Norway, Canada
Benzol	Leukemia	United States, et al.
Isopropyl oil	Lung, larynx, nasal sinus	United States
Radioactive chemicals	Bones, nasal sinus	United States
Chemicals (various)	Lymphoma, pancreas	United States
Vinyl chloride	Liver	United States, Germany, Sweden, et al.
Nonspecific (occupations):		
Wood furniture workers	Nasal cavity, sinuses	Great Britain, United States
Leather workers	Nasal cavity, sinuses, bladder	Great Britain, United States
Soft coal workers	Stomach	United States (1 report)
Machinists, sheet metal workers, carpenters, construction painters	Lung	United States, Great Britain

SOURCE: Modified from Levin, D. L., Devesa, S. S., Godwin, J. D., and Silverman, D. T.: *Cancer Rates and Risks*. DHEW Pub. No. (NIH) 75-691. Washington. U.S. Government Printing Office, 1974.

Table 6. Standardized mortality ratios for all sites of cancer combined, and for cancer of selected sites for white males aged 20-64 years, by broad occupational group: United States, 1950

Broad occupational group	All sites	Buccal cavity and pharynx	Esophagus	Stomach	Large intestine	Rectum	Lung and bronchus	Bladder	Skin	Leukemia and leukemia
All persons, aged 20-64	100	100	100	100	100	100	100	100	100	100
Professional, technical, and kindred workers	91	83	68	64	124	111	82	97	93	114
Farmers and farm managers	81	62	35	91	81	64	55	73	133	127
Managers, officials, and proprietors, except farm	95	83	64	74	110	94	95	85	105	111
Clerical and kindred workers	92	100	77	69	118	103	94	115	93	93
Sales workers	102	93	70	75	114	113	103	111	137	106
Craftsmen, foremen, and kindred workers	111	107	117	108	104	118	132	125	132	105
Operative and kindred workers	101	105	122	102	105	111	110	99	128	92
Service workers, except private household	109	141	149	102	106	119	125	123	---	91
Laborers, including farm laborers and foremen	105	133	166	116	95	94	103	96	213	96
Farm laborers and foremen	59	64	85	79	53	32	53	76	---	72
Laborers, except farm and mine	123	161	182	130	108	113	123	104	172	106

SOURCE: Levin, D. L., Devesa, S. S., Godwin, J. D., and Silverman, D. T.: *Cancer Rates and Risks*. DHEW Pub. No. (NIH) 75-691. Washington. U.S. Government Printing Office, 1974.

Table 7. Prevalence of chronic bronchitis reported in health interviews, by age, family income, and place of residence: United States, 1970

Family income and place of residence	All ages	Under 17 years	17-44 years	45-64 years	65 years and over
Total	32.7	38.9	23.2	35.4	41.2
<u>Family income</u>					
Less than \$3,000	39.5	30.0	28.3	54.6	46.3
\$3,000-\$4,999	35.0	36.2	28.4	34.3	43.8
\$5,000-\$6,999	31.0	36.4	17.6	43.4	39.0
\$7,000-\$9,999	32.3	37.9	25.3	35.3	34.8
\$10,000-\$14,999	32.6	46.9	21.8	29.0	*
\$15,000 or more	30.8	38.8	23.7	30.3	*
<u>Place of residence</u>					
SMSA	33.0	39.8	24.0	34.3	42.0
Central city	31.9	35.4	24.0	33.9	45.5
Not central city	33.8	43.1	24.0	34.5	38.2
Outside SMSA:					
Nonfarm	33.0	38.5	22.2	39.0	40.8
Farm	24.9	26.0	*	28.2	*

SOURCE: National Center for Health Statistics: Prevalence of selected chronic respiratory conditions: United States, 1970. *Vital and Health Statistics*. Series 10-No. 84. DHEW Pub. No. (HRA) 74-1511. Health Resources Administration. Washington. U.S. Government Printing Office. Sept. 1973.

Table 8. Prevalence of emphysema reported on the basis of physicians' diagnosis in health interviews, by age, family income, and place of residence: United States, 1970

Family income and place of residence	All ages	Under 45 years	45-64 years	65 years and over
Total	6.6	1.0	13.9	31.7
<u>Family income</u>				
Less than \$3,000	14.5	*	28.0	27.0
\$3,000-\$4,999	12.0	*	18.9	40.4
\$5,000-\$6,999	7.5	*	17.1	43.1
\$7,000-\$9,999	5.1	*	12.9	38.3
\$10,000-\$14,999	3.4	*	10.6	*
\$15,000 or more	3.4	*	8.5	*
<u>Place of residence</u>				
SMSA	5.4	0.7	11.4	28.0
Central city	6.1	*	12.8	26.5
Not central city	4.9	*	10.3	29.3
Outside SMSA:				
Nonfarm	8.8	1.5	19.4	38.7
Farm	7.7	*	*	*

SOURCE: National Center for Health Statistics: Prevalence of selected chronic respiratory conditions: United States, 1970. *Vital and Health Statistics*. Series 10-No. 84. DHEW Pub. No. (HRA) 74-1511. Health Resources Administration. Washington. U.S. Government Printing Office, Sept. 1973.

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APPENDIX I

OCCUPATIONAL DISEASE REPORTING IN CALIFORNIA

Esther Baginsky, *Occupational Health Branch,
California State Department of Health*

Under longstanding provisions of the California Worker's Compensation Law^b all work-incurred injuries and illnesses except first aid cases are reportable to the State. By definition, this includes any injury or disease arising out of employment. It may result from one injury or exposure or be the result of cumulative injury. The law covers about 85 percent of the State's workers, currently more than 7.5 million people, including farm workers and public agency employees. Excluded are self-employed persons and those under Federal, maritime, or railroad compensation systems.

The physician is required to file his report, Doctor's First Report of Occupational Injury or Illness (figure I), after the patient's first visit. In practice, he also forwards a copy to the insurance carrier as documentation of his medical services. The carriers require a report from both employer and physician for their claims procedures. The legal obligation to report applies to the physician whether he performs on a fee-for-services basis or is salaried. In practice, however, there is some underreporting by salaried physicians. Reports are not received from nurses in

inplant medical services for care they give under standing orders of a physician.

This system generates more than a million physicians' reports annually. About 40,000 reports of occupational illness for a selected list of disease conditions are routed for review to the California Department of Health. Included are both lost workday and nonlost workday cases.

Doctors' First Reports and Final Diagnoses

Although the physician usually sees the patient again, no further report to the State is required. A survey in 1959 to determine agreement between the doctor's first report and final diagnosis showed that the final diagnosis generally agreed with the one given on the first report and that the physician did not gain much more information during patients' revisits. This was corroborated as recently as 1972 in a review of medical records that showed that there was seldom any additional information for the doctor to base his diagnosis on other than disease progression. All the information available to the physician regarding the worker's exposure is contained, in a majority of cases, in the doctor's first report.

How accurate is information given by physicians who are not specialists in occupational medicine? Experienced occupational health personnel concluded after an evaluation study that in 8 or 9 out of 10 cases investigated the reporting physician was correct when designating a case as probably occupational in origin.

^b“Every employer, insurer, and physician or surgeon who attends any injured employee shall file with the Division of Labor Statistics and Research a complete report of every injury or occupational illness to each employee arising out of or in the course of his employment unless disability resulting from such injury does not last through the day or does not require medical service other than ordinary first aid treatment.” (California Labor Code, Section 6409).

**DOCTOR'S FIRST REPORT
OF
OCCUPATIONAL INJURY OR ILLNESS**

STATE OF CALIFORNIA
AGRICULTURE AND SERVICES AGENCY
DEPARTMENT OF INDUSTRIAL RELATIONS
DIVISION OF LABOR STATISTICS AND RESEARCH
P. O. Box 965, San Francisco, Calif. 94101

Immediately after first examination mail one copy directly to the Division of Labor Statistics and Research. Failure to file a report with the Division is a misdemeanor. (Labor Code Section 6413.5) Answer all questions fully.

A. INSURANCE CARRIER

	Do not write in this space
1. EMPLOYER	
2. Address (No., St. & City)	
3. Business (Manufacturing shoes, building construction, retailing men's clothes, etc.)	
4. EMPLOYEE (First name, middle initial, last name)	Soc. Sec. No.
5. Address (No., St. & City)	
6. Occupation	Age Sex
7. Date injured Hour M. Date last worked	
8. Injured at (No., St. & City)	County
9. Date of your first examination Hour M. Who engaged your services?	
10. Name other doctors who treated employee for this injury	
11. ACCIDENT OR EXPOSURE: Did employee notify employer of this injury? Employee's statement of cause of injury or illness:	
12. NATURE AND EXTENT OF INJURY OR DISEASE (Include all objective findings, subjective complaints, and diagnoses. If occupational disease state date of onset, occupational history, and exposures.)	
13. X-rays: By whom taken? (State if none)	
Findings:	
14. Treatment:	
15. Kind of case (Office, home or hospital) If hospitalized, date..... Estimated stay.....	
Name and address of hospital	
16. Further treatment (Estimated frequency and duration)	
17. Estimated period of disability for: Regular work Modified work	
18. Describe any permanent disability or disfigurement expected (State if none)	
19. If death ensued, give date	
20. REMARKS (Note any pre-existing injuries or diseases, need for special examination or laboratory tests, other pertinent information.)	

Name Degree..... PERSONAL SIGNATURE OF DOCTOR

(Type or print)

Date of report Address (No., St. & City)

Figure 1. Doctor's First Report of Occupational Injury or Illness

Information From Doctors' Reports

The doctor's report identifies the worker, employer, insurance carrier, and physician. While the employee's social security number is supplied on three-fourths of the reports, occupation is often missing or only vaguely described. Age and sex are almost always reported.

The employee's statement concerning the exposure and symptoms and the physician's findings and diagnoses are the basis for regularly produced California statistics on occupational disease. Certain limitations should be noted, however, in the detail contained in physicians' reports. Ten percent of all reports have no "hazard" classification and are coded "unknown." In addition about 30 percent classify exposure hazards in some "other and unspecified" category (e.g., gas, dust, etc.) or as chemically unspecified materials identified by use (e.g., solvent, paint, cleaning compound, agricultural spray, etc.).

One-half of all the reports reviewed in 1973^c described skin diseases or disorders, such as dermatitis, chemical burns, and inflammations. Another 30 percent listed eye conditions, mainly chemical conjunctivitis. Ten percent showed respiratory conditions and systemic effects of toxic materials. The remaining reports included disorders due to physical agents, infective and parasitic diseases, heart conditions, neoplasms, and other infrequently reported conditions.

Chronic or latent diseases are underreported in the California reporting system, as they are in most other occupational injury and illness reporting systems. In 1973, for instance, only 162 cases of noise-induced hearing loss were reported; on the other hand, studies of operating engineers and drillers in construction have shown that more hearing loss exists in these groups than is reported for all workers. There were 13 reports of pneumoconiosis, about the same as in previous years, yet 80 death certifi-

icates were filed in 1973 with mention of pneumoconiosis. Doctors' reports of neoplasms, benign or malignant, numbered 25.

Files of doctors' reports are maintained for 3 years. For a few conditions they have been retained for as long as 10 years. Among the selected cases are poisoning by heavy metals, certain gases, and pesticides. Coded data for all reports are also stored on magnetic tape for data retrieval and statistical use; however, the only identification is the name of the employer.

Potentials for Use in Studies

The recognition and reporting of work-incurred illness depends on what the patient and physician recognize as being related to occupation based on past experience, specialized training, and alertness to occupational health hazards. There are limited opportunities, therefore, to anticipate and recognize new problems on reports filed by physicians.

The most fruitful uses of doctors' reports of occupational disease would appear to be in providing background to initiate studies. As shown below, the reports could be used to:

Provide clues to initiate industry studies with specific health exposure problems (e.g., electroplating, woodworking, etc.).

Provide followup of workers who have reported certain occupational health problems already recognized (e.g., lead poisoning, chlorinated hydrocarbon solvent exposures, etc.) in order to study possible long-term effects. For workers selected in such a manner, linkup with other sets of data might be useful.

Provide a basis for setting priorities for study.

Provide information useful in setting occupational health standards.

^cCategories of illness reviewed by the Occupational Health Section, California Department of Health, differ in some respects from those defined in Federal and State Occupational Safety and Health recordkeeping regulations.

APPENDIX II

OCCUPATIONAL DISEASE REPORTING AND SURVEY RESULTS

Esther Baginsky, *Occupational Health Branch,*
California State Department of Health

From July to December 1971, as part of a survey of occupational illness in certain establishments in California funded by the National Institute for Occupational Safety and Health, a comparison was made of nonfatal occupational illnesses reported in the survey with those reported on the Doctor's First Report of Occupational Injury or Illness. In general, the ratio of doctors' reports to NIOSH survey cases was 0.74, with a higher ratio for cases with a lost workday (0.84) than for cases with no workday loss (0.71).

A survey involving interview and examination of workers among small industries in Oregon and Washington was reported by Discher, Kleinman, and Foster.²

The study had two goals: (1) to determine the utility of procedures designed for finding occupational disease in a cross-sectional survey, and (2) to ascertain how much new data would be generated by this method.

Some of the findings were:

Over 1,100 medical conditions were found among 908 participants: probable occupational, 31 percent; probable nonoccupational, 45 percent; doubtful occupational or can't evaluate, 14 percent; and suggestive history, 10 percent.

Of 346 cases of probable occupational disease, hearing loss was most frequent, 28 percent; then skin, 18; lower respiratory conditions, 14; toxic and low-grade toxic

effects and nonsymptomatic conditions (mainly elevated blood leads), 14; upper respiratory conditions, 11 percent; and eye conditions, 9 percent. Anemia, diseases of the musculoskeletal and connective tissues and other conditions accounted for the remaining 6 percent.

Of the 908 workers participating in the medical survey, 258 workers were found with 346 cases of probable occupational disease, giving a prevalence rate of 28.4 per 100 workers. Occupational exposures were determined for all workers in the survey. Those known to have been exposed to poorly controlled hazards had a higher prevalence rate (39.0).

The survey morbidity data were checked for duplication with records of compensation claims and injury and illness records employers are now required to keep under OSHA (Employer's Log). Of 451 reports of occupational disease, 89 percent were found on the survey only; 2 percent on the Employer's Log; and 3 percent on compensation claims only; 6 percent were duplications.

A nationwide survey is recommended, based on techniques developed in this study, for an estimate, not available elsewhere, of the extent and kinds of occupational disease. The results would help delineate problem areas, ascertain

Table I. Medical conditions¹ by occupational relationship among workers in small industries in Oregon and Washington

Medical condition ¹	Total	Probable occupational disease ²	Can't be evaluated ³ and doubtful ⁴	Suggestive history ⁵	Probable non-occupational disease ⁶
Total	1,116	346	151	113	506
Anemia and other diseases of blood	49	7	21	1	20
Conjunctivitis and other conditions of the eye	74	31	1	24	18
Refractive errors	84	0	1	0	83
Hearing loss, mild	86	68	12	0	6
Hearing loss, moderate	32	26	4	0	2
Hearing loss, advanced	4	2	2	0	0
Hearing loss, possible threshold	16	0	16	0	0
Hypertension	138	1	0	0	137
Heart disease	14	0	1	0	13
Other diseases of circulatory system	3	0	0	0	3
Respiratory conditions, excluding upper	289	49	76	11	153
Upper respiratory conditions	73	38	2	21	12
Skin conditions	128	64	4	35	25
Disease of musculoskeletal and connective tissues	28	9	2	7	10
Symptoms and ill-defined conditions	17	1	6	1	9
Nonsymptomatic conditions	27	27	0	0	0
Toxic and other adverse effects	23	9	1	13	0
Low grade toxic effects	14	13	1	0	0
Other conditions	17	1	1	0	15

¹Does not include employees who took hearing tests only.

²Manifestations of disease are consistent with those known to result from excessive exposure to a given injurious agent; this injurious agent is present in the patient's working environment and significant contact in course of usual duties is likely.

³An association between disease and occupational factors has never been clearly established; e.g., cardiovascular disease, respiratory infections, mental and psychoneurotic episodes.

⁴(1) Manifestations of disease are not entirely consistent with those known to result from excessive exposure to an injurious agent or (2) manifestations of illness are consistent with those known but no significant contact with alleged injurious agent can be established.

⁵Manifestations of disease as given in history are consistent with those known to result from excessive exposure to a given injurious agent; this exposure is or has taken place; there is no objective evidence of disease at the time of the medical examination.

⁶Manifestations of disease but no relationship to occupational exposures.

adequacy of standards, and might provide new insights into the relationship of occupational exposures and diseases.

Table I shows the definitions of "Probable

occupational disease," etc., and the frequency of relevant categories of medical conditions according to the likelihood of an occupational relationship.



APPENDIX III

MORTALITY SURVEILLANCE USING CORONER'S REPORTS

Margaret Deane, *Epidemiological Studies Laboratory,*
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Background

Since the early 1950's the California Health Department has been interested in providing to the general community a warning of excess mortality or morbidity through a mechanism which would respond rapidly to significant changes. Such a system meets the definition of surveillance, which implies continuing measurement and examination of some index in a manner allowing prompt action to alter the forces causing departures from expected values.

In 1954, the department began operating a mortality surveillance system using weekly reports of deaths and admissions to hospitals from about 100 nursing homes in the Los Angeles area.

The study was based on the idea that the nursing home population largely represented old and chronically ill people who would be expected to be relatively sensitive to the effect of the environment. When initiated, it was planned as a system which, on a few hours' notice, would alert the community to the occurrence of excessive mortality. Although the regular weekly reports were received by mail, during periods of high air pollution, data were obtained by telephone with a lag time of only a few hours.

The nursing home study was discontinued in 1967 for two reasons. First, many of the homes had acquired air conditioning so that the patients were no longer exposed to environmental conditions characteristic of those impinging on the general noninstitutional population. Second, the possibility arose of using mortality data

from the Los Angeles Coroner's Office, which had indications of being a more manageable and effective source of data.

Data Currently Available

Since 1968, the Los Angeles Coroner's Office has sent weekly reports of all cases recorded during the preceding week. The reports are based on the daily log, which is the most quickly available source of information and which includes name, age, sex, date (or presumed date) of death, and an indication of whether the death was presumptively classified as "suicide," "homicide," "motor vehicle accident," "other accident," or "due to natural causes." Data are edited and transferred to computer tape for subsequent processing.

Coroner's reports have several advantages over general daily mortality for use in a surveillance system. First, they are available on a current basis, while data based on total vital statistics are not. Furthermore, since the reports originate in coroners' offices rather than in numerous hospitals and other institutions, it should be relatively easy to establish a system of collection and transmission that would provide data even faster than that presently available, reducing the lag time from between 1 and 2 weeks to between 1 and 2 days. This might be done by entering the data through a remote computer terminal located in the coroner's office. Second, since coroner's cases represent a selected group of deaths, that is, those resulting from violence or suicide, those of individuals not

in attendance by a physician, those in which the cause of death is unknown, and those following an injury or accident, they may be more sensitive than general mortality to environmental causes and effects of drugs and pesticides. For example, rates for accidents and other sudden deaths may be more affected by air pollution and weather; and homicides, suicides, and poisoning may be more closely related to drug use, especially among young adults. By obtaining additional medical data following autopsy or by record linkage to obtain cause-of-death information from death certificates, it should also be possible to examine some specific causes of death that are particularly apt to be related to environmental factors. These could include myocardial infarctions, deaths of early infancy, childhood cancer, anemia, leukemia, and blood dyscrasia.

About 40 coroner's cases per day are reported from Los Angeles County, or between 14,000 and 15,000 per year. Thus for the 5-year period 1968-72, we have about 75,000 records available for analysis. Coroners' cases should therefore be applicable as a surveillance index for other large metropolitan areas, and can provide guidance for evaluating medical service needs.

Long-Term Objectives

One objective of mortality surveillance is to develop a system that can be used to detect departures from "expected" numbers of deaths to alert officials to seek the cause, whether it be an increase in communicable disease, effects of some social factors such as alcohol or drugs, or an effect of such environmental factors as pollution or weather. To be effective, such a system must use methods of collection, transmission, updating, editing, and analysis that give results promptly enough so that effective action can be taken to alter the force of mortality.

Another aim is to carry out analyses designed to investigate the relationship between daily mortality as reflected in coroner's reports and causative factors that are amenable to control; for example, air or water pollution, weather, pesticides, drug abuse, prenatal effects. These analyses would have two results. First, to

support hypothesis that such relationships do, in fact, exist, and second, to provide a basis for establishing expected values based on past experience.

Present Status

Data for 1968-74 have been edited and are available on computer tape in a format suitable for analysis. Several models are being tested to establish expected values based on these 5 years of data. The years are treated as replicates of a 52-week period and Fourier analysis is being used to fit curves with up to nine harmonics. Departure of weekly values for 1973 from expected values predicted from these curves will then be examined in relationship to statistical significance and coincidence with environmental factors such as elevated levels of air pollution and ambient temperature, taking into account also the effect of epidemics. Alternatively, expected values have been calculated using moving averages of the 5-year means for each week.

Future Plans

Depending upon the availability of resources, further work will be done in developing and testing the predictive models and in examining departures of current data from predicted values. These also may include multiple regression models including pollution levels, humidity, time of year, trend, and various functions of these including rapidity of change over time. The use of discriminative analysis is also being considered. Cu-sum methods may be used to test for significant departures from predicted values.

Explanation of the Coroner's Study Coding

Cause of death is the only item to be coded. However, there are other items on the form that must be edited.

1. Age is probably the most frequent problem to arise. As the age of infants is often given in months, this should be the first item coded on the form since we wish to convert age into years in a three-column field.

All ages under 1 year should be coded 000.

All ages between 12 months and 23 months should be coded 001.

Any age unknown should be coded 999.

Stillborns should be lined out completely and not coded.

2. Frequently, entries will appear that are not coroners' cases. These can be determined by the letters "NCC" usually found in the column labeled "Disposition." These cases should also be lined out and not coded.

3. Usually, there will appear on the green sheet a list of numbers or items labeled "Hold-overs." These holdovers should be lined out and not coded. They will appear in a later report.

4. There are seven cause-of-death codes (see table II). The cause of death should be coded in red pencil just to the right of the cause.

The name of the doctor is no longer being coded. On the white sheet, the doctor's name appears in the extreme right-hand column labeled "Disposition." On the green sheet, the doctor's name appears in the left-hand column above the case numbers assigned to the doctor. A list of code numbers is attached. The code number should always be coded on the extreme left margin of both forms. If a doctor's name is not on the list of code numbers, add the name to the list and assign it the next number.

Surveillance of Daily Coroner's Reports

<u>Col.</u>	<u>Explanation</u>
1-2	Year of report
3-7	Case number
8-11	Date of death
8-9	Month
10-11	Day
12-16	Time of death
12-13	Hour
14-15	Minute
16	AM: 1; PM: 2; UNK: 9

If 2 dates appear here, the first date is the day of death; the second date is the day the death was reported to the coroner's office. In coding, the second date can be ignored.

Table II. The seven cause-of-death codes

<u>Code</u>	<u>Cause</u>
1. Motor vehicle accidents . .	Collisions involving motor vehicles (cars, buses, trucks, motorcycles, tractors)
2. Nonmotor vehicle accidents	Airplanes, trains, industrial accidents, fires, and "accident," "possible accident," "accident?"
3. Suicide	"Suicide," "possible suicide," "suicide?"
4. Homicide	"Homicide," "possible homicide," "homicide?"
5. Natural ¹	"Natural"
6. Possibly natural ¹	"Possibly natural," "apparently natural," "natural?"
7. Undetermined	"Undetermined," or a combination of any two of the above causes; e.g., "natural?-accident?"

¹There is no discernible difference between number 5 and number 6.

17-36	Last name
37-51	First name
52	Middle initial
53-55	Age (1-12 mos: 000; 13-23 mos: 001)
56	Blank
57-60	Date brought in (if different from first date)
57-58	Month
59-60	Day
61-65	Time brought in
61-62	Hour
63-64	Minute
65	AM: 1; PM: 2; UNK: 9
66	Cause of death
67-68	SO (death signed out) or A (autopsy performed)
69-70	Doctor
71	Outside: 1; Inside: 2

Daily Mortality Data

In large populations, the frequency of deaths is such that variations in daily mortality can be used as a sensitive index of health effects which are influenced by changes in environmental

factors. In New York City, for example, daily mortality is sensitive to moderate fluctuations of temperature, and when suitable controls are made for temperature, season of year and other factors, the daily mortality varies with atmospheric sulfur dioxide levels. This valuable

approach should be exploited in other large cities for similar purposes. These investigations have been handicapped by the NCHS decision in 1967 to discontinue punching the day of death on death records. This information must now be obtained less efficiently from local sources.



APPENDIX IV

THE CONTINUOUS WORK-HISTORY SAMPLE^d

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INTRODUCTION

In administering the social security system, an unprecedented volume of economic and demographic data is generated. Much of this basic information is summarized in the monthly Social Security Bulletin, and its Annual Statistical Supplement; and in special releases and reports.

Because the basic administrative records have great value for economic and social research, it is the general policy of the Social Security Administration (SSA) to make its data resources available for this purpose. Such research must meet two conditions: it must provide for safeguarding the confidentiality of information for individuals, firms and reporting units; and it must be feasible without impairing the administration of the social security program.

Our publication, *Some Statistical Research Resources Available at the Social Security Administration*, provides a detailed description of the available data files, the procedures used in their compilation, and how they may be obtained. The overall research program of the Social Security Administration is detailed in the

annual Work Plan of the Office of Research and Statistics.

AVAILABILITY OF RESEARCH FILES

Much of the analytical data drawn from our administrative records are most conveniently handled by the use of samples. In order to provide, at modest cost, outside users with a general research file, SSA makes available an Annual One-Percent Continuous Work-History Sample. The nature of this data file may best be understood by seeing how the various sources of data come together.

When a person applies for a social security number he provides data on his sex, race, and date of birth, enabling us to maintain a file of individuals by social security number (SSN), sex, race, and age. When an employer requests an employer identification number (EIN) he provides data on his geographic location and industry activity, enabling us to maintain a file of employers by EIN coded to State and county, and industry (4-digit SIC^f in manufacturing and 3-digit SIC in nonmanufacturing). Each quarter, covered employers report the wages of their employees up to the taxable limit. (Agricultural and self-employed data are reported annually.) By matching the earnings data first to the SSN file, and then to the EIN file, we obtain for each job quarter, data on sex, race, and age, State and county, industry, and wages.

^fStandard industrial classification.

^dThis paper was developed for presentation at the August 1974 American Statistical Association meeting.

^ePosition at time paper was prepared. Mr. Hirschberg is currently with the U.S. Department of Labor.

Most importantly, the selection of a fixed sampling pattern of social security numbers for inclusion each year permits the establishment of a work-history file for tracing employment, migration, and earnings status of all those who worked in covered industries and for determining their socioeconomic characteristics from 1957 to date. As new workers enter the workforce, those with the specified digits enter the annual sample; as others drop out of the covered workforce those with the specified digits no longer appear.

More recently, claims data have been introduced into the file. These, of course, are obtained from the application for benefits and the Master Beneficiary Record, a comprehensive record for all benefits in force.

Six Available Files

Six files, all of which contain data for sex, race and age, are available to outside users. So that the files may be utilized for longitudinal studies, and the confidentiality of individuals and firms be maintained, the employee and employer identification is included in scrambled form.

One-percent annual employee-employer file.—This includes wage and salary employment reported in the reference year, with one record for each employee-employer combination. Basic data elements include annual and quarterly taxable wages, total estimated wages, State and county, industry, and coverage group (for example, farm, military, and household). This file becomes available approximately 2 years following the year of reference. Currently the file is available for each year from 1957 to 1971.

One-percent first-quarter employee-employer file.—This file contains the same data elements as the annual file, except that it becomes available about 15 months after the quarter of reference. Because an effort is made to obtain the file as quickly as possible, late reports are excluded and coding problems which may exist are not resolved. Excluded are agriculture and self-employment data, which are reported on an annual basis.

One-percent sample longitudinal employee-employer data (LEED) file.—The basic data elements are the same as in the annual file,

except that the records are skeletonized, are currently available from 1957 to 1970, and are sequenced so that all records associated with an employee appear together.

One-percent sample annual self-employed file.—This file includes the same basic data elements as the employer-employee files, but covers net and taxable earnings for those who are self-employed. The basic source is the IRS Schedule SE. The earliest date of availability is 1960.

One-percent 1937-to-date continuous work-history sample (CWHS).—This file provides various data indications from 1937 including years employed, first and last years employed, pattern of quarters employed for the last two years, number and quarters of coverage beginning with 1937, patterns of coverage beginning in 1957, farm or nonfarm wage or self-employment indicators, taxable and self-employed earnings each year beginning in 1951, and insurance status and benefit information.

One-tenth percent 1937-to-date continuous work-history sample (CWHS) file.—This file provides the same data as the one-percent CWHS, but includes a greater level of detailed earnings information beginning in 1937. There is no geographic or industrial detail.

LIMITATION OF CWHS

When administrative data are used for analytical purposes, the researcher must be aware of some problems and limitations. These occur because the entire labor force is not covered and the employer reports only wages up to the taxable maximum. Moreover there are problems of timing, improving the geographic and industry coding, and of sampling and nonsampling errors in utilizing the data.

Coverage

No major changes in the coverage provisions of the social security system have taken place since 1954. Currently the sample covers well over 90 percent of workers in paid employment. Two types of Old-Age, Survivors, Disability, Hospital Insurance coverage exist: mandatory and elective. On a mandatory basis are all employees in nonfarm industries (except rail-

road workers), most farm and domestic employees who meet minimum earnings provisions, and Federal employees not covered by the Federal retirement system. Groups covered on an elective basis are ministers, nonprofit establishments, and State and local government workers. All self-employment is covered if earnings exceed \$400 per year.

Essentially for those with more than just marginal earnings, excluded are 3 million Federal workers, 3 of the 10 million State and local government workers, and a small number of nonprofit employees, since most elect coverage.

Wages

Several problems arise when administrative wage data are used for analytical purposes. The major limitation is that the employer reports for each worker the wages to the taxable limit. The taxable limit has risen steadily since 1957 when it was \$4,200; currently it is \$13,200. Because nonfarm wage data are reported by quarter, an estimate of total wages is possible. This procedure estimates the total wage by substituting the last full quarter wage for the quarter in which the taxable limit was reached, and for each subsequent quarter.

For those workers who reached the taxable limit in the first quarter, separate annual estimates for males and females are prepared for each year based on the Pareto method.

Industry and Geographic Coding

As mentioned previously, industry and geographic coding data are obtained when a firm applies for an EIN. On the same form is the question asking if this is a multiestablishment firm. If the answer is "Yes," the firm is asked to participate in our Establishment Reporting Plan covering multiestablishment firms. Ideally, we would like to obtain from each firm an individual report for each establishment. However, reporting by establishment is voluntary, and because of other priorities, only a limited number of technicians are assigned to deal with establishment reporting problems. Simply put, the problem is one of editing, reviewing, and correcting, if necessary, several million firm reports received each quarter. We are planning studies to determine the effect of the reporting

difficulty and how its impact can be minimized. The file does contain internal coding, each record indicating how the industry and geographic assignment has been made. This is a great help in editing the file so that spurious changes can be identified; the nature of the edits depend upon the research undertaken.

Although researchers using the file should be aware of these limitations, the CWHS is still a powerful analytical tool. It permits extensive disaggregation by sex, race, age, industry, geography, workforce participation, and earnings levels. It follows the same individual workers over time so that quarter-to-quarter or year-to-year changes for specific individuals can be observed. Most important, as Nancy and Richard Ruggles⁵⁹ have pointed out, it can be successfully disaggregated to show the anatomy of the total wage bill as it relates to the national economy.

When used to examine migration, the file provides for the development of area data on gross flows, as compared with net flows, because the net may be an average that masks important characteristics of two very different gross flows.

Cross-sectional data on earnings and mobility status can at best be only partly informative; generally they are misleading. For example, workers who move from one area to another (migrants) earn less than nonmigrants. However, migrants increase their earnings at a faster rate but start from a lower base. If we examine census data, the nonmigrants appear to be the higher paid group, and some economists have suggested, therefore, that mobility does not improve the economic status of migrants.

MEETING OF CWHS USERS

A CWHS users conference is held each year. Invitations are extended to anyone interested in the problems of handling the data files, to those who wish to report on recent research findings, and to those involved in preparing the file in SSA. These meetings over the last several years have provided users of the data with a better understanding of the work currently under way, and have enabled the producers to discuss several of the new developments relating to the file, and methods to improve it.

DATA DEVELOPMENT

The 10-Percent Sample

The one-percent CWHS currently available has important limitations when used for studying small standard metropolitan statistical areas (SMSA's) and rural areas. The initial impetus for the development of a ten-percent sample file came from OMB (Office of Manpower and Budgeting) as a result of urgent needs for intercensal population estimates for revenue sharing and other programs, and because of the decision not to conduct a mid-decade census. At present, the Department of Housing and Urban Affairs is providing the bulk of the funding and other Federal agencies make significant contributions.

The data base for this file will be similar to the first quarter files described earlier, and will include those working in the first quarters of 1971 and 1973. In the United States during the first quarter of 1971, approximately 73 million workers held 80 million jobs. Therefore, the 1971 file will contain records for 7.3 million workers and 8.0 million jobs. The 1973 file will be approximately 6 percent larger.

For each year, the records will be summarized so that the industry and place of work is available for the major job. The file will be merged, indicating for each individual the sex, race, age, if his employer changed, and (for 1971 and 1973) geography, industry, and wages. The file will be sorted so that tabulations by State and county for both years will be available at modest cost.

The ten-percent sample would constitute a significant asset for regional analysis. No other source of data could provide insight into the structure of a local area labor force, so that employment distributions by sex, race, age, wages and wage changes, workforce participation, industry, and regional migration patterns could be analyzed systematically.

Occupational Data

In addition, a detailed proposal has been prepared to test the feasibility of adding occupation as a standard data item to the CWHS. It is contingent on Internal Revenue Service (IRS) cooperation. The approach is to use the IRS

1040 occupation information supplemented by followups when necessary with employees, and/or employers. The proposal is under serious consideration with other statistical agencies and is planned in two stages. The first stage will be a feasibility and quality analysis based on a sample of 15,000 CWHS wage earners. It will be designed to examine the cost and operational feasibility of this approach and to explore issues of quality—in particular, the extent to which occupation entries on the IRS 1040 can be used for classification beyond the major group level.

If the pilot project indicates that this approach to the collection of occupational data appears feasible and produces data of acceptable quality, the project would go into a second phase, expanded to another 60,000 workers, or a one-in-a-thousand sample.

Place of Residence Data

In another project, a modified version of the Census Bureau's address reference file is being used to automatically assign geographic codes to the 1972 CWHS. This operation will provide for place of work and place of residence comparisons, and also facilitate the editing of the file.

CONCLUSION

Ideally a research file of this scope should contain additional information. Occupation and place of residence have already been mentioned. Timeliness of the data has been improved with the availability of first quarter files. In terms of coverage, efforts will be explored to provide data for the noncovered portion of the CWHS. There is a need to incorporate data from the Railroad Retirement System and the Civil Service Commission. (In addition, educational attainment, hours of work, marital status, unemployment, and noncovered wages are other important variables to consider.)

In conclusion, the operation of the Social Security System produces a vast and unique body of longitudinal data on earnings, and on retirement and disability claims and benefits for persons classified by age, race, and sex. It has been our policy to make the data available to

social scientists. Over the past year, administrative and research agencies of government have been extremely helpful in moving some of these research efforts forward and we are

grateful. In undertaking these projects, we will always be careful to safeguard and protect the confidentiality of information relating to individuals.



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