Statement of

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I am pleased to have been invited to discuss occupational exposure to asbestos, a subject I have been involved with since 1970. I participated in the investigation that helped to stop exposures in the asbestos insulation plant in Tyler, Texas, where over one fourth of the plant's former employees are expected to contract asbestos-related diseases. I have also conducted industrial hygiene and epidemiologic studies in approximately a dozen other asbestos manufacturing and processing facilities in the United States. I was the primary author of the NIOSH revised recommended standard for asbestos, as well as the monograph on asbestos published by the International Agency for Research on Cancer (IARC).

I would like to begin by describing the functions of the National Institute for Occupational Safety and Health (NIOSH) and then give a brief review of what is known about asbestos-related disease. I will also provide some information about shipboard exposures.

NIOSH was established by the Occupational Safety and Health Act of 1970 to conduct programs of occupational safety and health research, standards development, technical assistance and manpower development. Under the Federal Mine Safety and Health Act NIOSH is also responsible for conducting mine health research, recommending health standards and conducting health hazard evaluations in mines. Under these authorities NIOSH makes recommendations to the Department of Labor for new or revised occupational standards.

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Asbestos

Workplace exposure to asbestos is probably the number one occupational health problem in the United States, both because of the vast number of workers exposed--an estimated 8 million workers since the beginning of World War II--and the potential for very severe and debilitating disease.

Asbestos has been termed "the mineral with a thousand uses", which suggests the degree to which the substance pervades modern life. By actual count there are more than 3,000 uses for asbestos, and this versatility is reflected in the variety as well as the size of the workforce exposed. About 1 million workers are currently thought to be exposed to asbestos.

There are several commercial forms of asbestos used in the manufacture of various products. Amosite, sometimes referred to as "brown" asbestos, and crocidolite, sometimes referred to as "blue" asbestos, and chrysolite, sometimes referred to as "white" asbestos, were the most common fiber types used in the shipbuilding industry. Although their physical characteristics vary somewhat, each of these types of asbestos has been shown to cause cancer and fibrotic lung changes in man.

Workers are exposed to asbestos while mining, milling, and transporting the mineral and while manufacturing and working with products which contain the substance. These products include protective clothing, mailbags, padding, conveyor belts, pipes, roofing shingles and gutters. It is also used in manufacturing special paper products, including millboard, fine quality electrical papers, friction materials, such as gaskets and automotive clutch

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and brake linings, as well as insulation for pipes and boilers and bulkhead linings for ships.

Many workers not directly employed in industrial processes involving asbestos exposure must also be considered susceptible to exposure. Maintenance personnel must often repair machines, pipes, boilers and bulkheads in asbestos-contaminated work spaces or work directly with products containing asbestos.

Asbestos-Related Diseases

Exposure to asbestos significantly increases the risk of contracting three serious diseases: asbestosis, lung cancer and mesothelioma. Other causes of death that have been associated with asbestos exposure include gastro-intestinal cancer and laryngeal cancer. Although asbestos workers who do not smoke can contract lung cancer, the risk is dramatically increased for asbestos workers who also smoke cigarettes.

It is not known how asbestos causes disease. There has been speculation that surface properties, fiber size, and metallic content of the fibers may influence the carcinogenic properties of the substance. The speculation, however, remains to be proven. What is known is that fibers too fine to be seen by the human eye become airborne during various industrial processes, repair or demolition and may be inhaled or swallowed.

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Asbestos comes from a Greek word meaning "inextinguishable". The etymology aptly describes an especially vexing property of asbestos: its ability to remain in the body, to be, in a sense, "inextinguishable" after it has been inhaled or ingested. As much as 50 percent of inhaled asbestos fibers remains lodged in the lungs where it is almost impossible for the body to dispose of it.

Asbestos-related diseases, particularly cancers have long latency periods. Frequently it is from 20 to 40 years after first exposure that the disease becomes apparent.

Our best information about the increased risk associated with asbestos exposure is based on NIOSH and other HEW supported studies of workers heavily exposed to asbestos before the Government began to regulate the substance in the late 1960's and early 1970's. Of the approximately 4 million workers heavily exposed to asbestos in the past, approximately 40 percent can be expected to die of asbestos-related cancer. In the absence of asbestos exposure less than 10 percent would be expected to die from these types of cancer. Workers without high exposures face smaller risks of asbestos-related disease. But it must be noted that scientists have not been able to determine a safe level of exposure to asbestos.

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Exposures to Asbestos Among Shipbuilders and Seamen

During World War II, 4.5 million workers are estimated to have worked in shipyards. Currently there are some 120,000 shipyard workers employed in about 450 establishments in the United States.

Several epidemiologic studies have been conducted of persons involved in the building. maintenance and repair of seagoing vessels. Each of the studies has shown an excess of asbestos-associated diseases, including asbestosis, lung cancer and mesothelioma. In some cases asbestos-induced changes in the lung were detected in the X-rays of over 50 percent of the workforce. Mortality among shipyard workers has been much greater than that expected when compared with control populations. This excess has mainly been accounted for by diseases associated with asbestos exposure.

For example, in a study conducted by Dr. Irving J. Selikoff, cancer was responsible for one-half of the total deaths observed among shipyard workers, with lung cancer accounting for 27 percent of all deaths, mesothelioma for 10 percent, and asbestosis for 17 percent. In this study, the levels were generally in the range of 4 to 12 fibers/cc for fibers greater than 5 microns in length.

One environmental assessment study conducted for the U.S. Maritime Administration, which was submitted to NIOSH for review, reported on sampling done on two ships in port and one ship at sea.

The samples were taken in various areas of the engine room of the ships but not during routine maintenance or repair activities. Concentrations in the engine rooms of the two ships at port ranged from .001 to .152 fibers/cc greater than 5 microns in length. In the ship at sea the samples were somewhat lower but in the same magnitude range. Samples taken at the Long Beach Naval Shipyard during asbestos removal operations on other ships reported average counts of 40 fibers/cc greater than 5 microns in length and some reported as high as 150 fibers/cc greater than 5 microns in length. (NIOSH currently recommends that workers not be exposed to more than 0.1 asbestos fibers per cubic centimeter.) Based on these reports, maintenance at sea could generate exposures in these higher fiber ranges. Studies of industrial workers in asbestos trades have shown excessive disease and death for workers exposed at much lower levels than are reported during these rip-out operations. To date scientists have been unable to determine a dosage level below which asbestos will not cause disease.

Occupational Standards for Asbestos

As evidence of its importance as an occupational hazard, asbestos was the subject of the first criteria document developed by NIOSH. This document served as the basis for the first new standard promulgated by the Occupational Safety and Health Administration (OSHA) after the Occupational Safety and Health Act was passed.

The standard, promulgated in 1972, was intended primarily to prevent asbestosis and reduce the risk of contracting asbestos-induced cancer. It

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provided for an exposure limit of 5 fibers greater than 5 microns in length per cubic centimeter of air until July 1976, when the limit was to be reduced to 2 fibers per cubic centimeter.

The current standard for occupational exposure to asbestos permits a maximum of 2 asbestos fibers larger than 5 microns in length per cubic centimeter of air. This is not because fibers shorter than 5 microns do not pose a health hazard, but because shorter fibers are difficult to detect by the economically accepted method of analysis, phase contrast optical microscopy. The current limit actually provides for a broad range of exposure to asbestos. This is because the relative proportion of airborne fibers longer than 5 microns may vary from 1 to 50 percent of the total asbestos in a particular air sample. Thus, a work environment may be in compliance with the Federal exposure standard and still offer the possibility of exposure to rather large amounts of asbestos.

Even when one considers only the fibers 5 microns or larger, the total number of fibers a worker can inhale in a permissible occupational environment can be quite large. The average worker inhales 6-8 million cubic centimeters of air per working shift. In an atmosphere which complies with the standard, this could mean inhaling up to 16 million "permissible" fibers per day in addition to the asbestos particles less than 5 microns long.

In 1976, NIOSH evaluated the studies published on asbestos since the 1972 criteria document was transmitted. By then there was evidence that chest x-

rays of British asbestos workers were continuing to show abnormalities despite exposures within a 2 fiber limit.

Based on this and other evidence, NIOSH recommended that OSHA further lower the standard to 0.1 fibers per cubic centimeter, the lowest concentration at which fibers could be reliably monitored by phase contrast microscopy. In transmitting the revised recommendations to OSHA, NIOSH made the following comment:

> "Because it is not possible to specify a safe exposure level for a carcinogen, only a ban on the use of asbestos can ensure complete protection against this mineral's carcinogenic effect. Therefore, emphasis should be placed on prohibiting the occupational use of asbestos in other than completely closed operations and on substituting other products whenever possible. Asbestos should be replaced, where technically feasible, by substitutes with the lowest possible chronic toxicities."

OSHA is presently reviewing the NIOSH recommendations for a new asbestos standard and NIOSH and OSHA are working together to update information on asbestos related-disease.

Control of exposures to seafaring persons should include, as the U.S. Navy has now required, that no new ships contain asbestos and that maintenance, repair and asbestos removal operations be controlled to the greatest extent possible. This would involve assuring appropriate protective clothing and work practices

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for those directly involved and keeping non-protected persons away from areas in the ship where asbestos exposure can occur.

Public Health Service Hospitals

Under interagency agreement with the Bureau of Medical Services, NIOSH is supporting the development of a Cooperative Center for Occupational and Environmental Health located on the PHS Hospital grounds in Baltimore. This center also represents a collaborative effort with the Johns Hopkins Hospital University School of Public Health. It will create an opportunity for clinical research, educational and training activities as well as technical assistance and outreach activities serving the greater Baltimore area. An important part of this program will be the diagnosis and treatment of asbestos-related disease. Secretary Harris has asked the Assistant Secretary of Health to monitor this project to determine whether similar projects could be undertaken elsewhere. It is hoped that the center will serve as a model to involve other units of the PHS hospital system in meeting the occupational health needs of the communities which they serve.

Mr. Chairman, I will be pleased to answer any questions you or members of your subcommittee may have.

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Epidemiology of Asbestos-Related Diseases

by R. A. Lemen,* J. M. Dement,† and J. K. Wagoner‡

This paper is intended to give the reader an overview of the epidemiology of asbestos-related diseases and is restricted to primarily occupational exposure studies. However, some mention of nonoccupational exposures are made because of their direct relationship to a worker or to a secondary occupational source.

Over 100 epidemiological studies are reviewed, dating back to the first case of asbestos-associated disease reported by Montague Murray in 1906. The studies are divided by specific fiber type and by specific disease outcomes and the interaction of asbestos and cigarette smoking is discussed in great detail.

Introduction

This paper is intended to give the reader an overview of the epidemiology of asbestos-related diseases and is restricted to primarily occupational exposure studies. However, some mention of nonoccupational exposures is made because of their direct relationship to a worker or to a secondary occupational source.

Nonmalignant Respiratory Diseases

Historical Studies

The use of asbestos dates back thousands of years; however, the modern industry dates from about 1880, when it was used to make heat and acid resistant fabrics (*1*, *2*). With the increasing use of asbestos materials, reports of asbestos-related disease emerged.

The first record of a case of asbestosis was reported in England by Montague Murray in 1906. Hoffman (*3*) reported (1918) that it was the practice of American and Canadian insurance companies not to insure asbestos workers due to unhealthful conditions in that industry. Pancoast et al. (*4*) commented on x-ray changes resembling pneumoconiosis in 15 individuals exposed to asbestos. The first complete description of asbestosis and of the "curious bodies" seen in lung tissue appeared in 1927, when Cooke (*5*) reported on a case of asbestosis, and McDonald (*6*) reported on the same and another case. Each author gave reasons for believing that these "curious bodies" originated from asbestos fibers that had reached the lungs. Mills (*7*) reported the first case of asbestosis in the United States in 1930, and in the same year, Lynch and Smith (*8*) reported on "asbestosis bodies" found in the sputum of asbestos workers. ("Ferruginous bodies" is a more descriptive term, as other inhaled fibers, e.g., fibrous glass, may also become iron-coated.) Early studies led many investigators to conclude that people exposed to asbestos dust developed the disease "asbestosis" if the dust concentration was high or their exposure was long (*9-12*).

Epidemiologic Studies

Harries (*13*) reported that, although first impressions would lead one to believe that only workers continuously exposed to asbestos are at risk of developing asbestosis, further consideration of the industry and processes should have suggested that many other workers were also at risk. For example, some trades worked in confined spaces where asbestos was used. Work in shipboard trades was accepted by the Pneumoconiosis Panel of the United Kingdom as associated with asbestosis.

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Murphy et al. (*14*) found that asbestosis was 11 times as common among pipe insulators involved in new ship construction as among a control group. Asbestosis first appeared 13 years after exposure or at about 60 mppcf-years. The prevalence was 38% after 20 years. They also reported a case of extensive pleural calcification in a worker whose only known asbestos exposure was during sanding asphalt and vinyl tile floors (*15*).

Lorimer et al. (*16*), in a study of brake repair and maintenance workers exposed to asbestos, found that 25% of the workers showed evidence of x-ray abnormalities consistent with asbestosis. One quarter also had restrictive pulmonary function test findings. Meurman et al. (*17*) found a three-fold risk of dyspnea and a two-fold risk of cough for asbestos workers as compared with controls, after adjusting for smoking.

Weill et al. (*18*) reported a decreased lung function in relation to increasing cumulative dust exposure in a group of asbestos cement manufacturing workers. Ayer and Burg (*19*) reported a decrease in pulmonary function in asbestos textile workers with less than ten years of exposure.

In a study of 232 former insulation plant employees, Selikoff (*20*) reported positive x-ray findings among individuals having exposures to asbestos known to be as short as one day. More recently, Anderson et al. (*21*) reported x-ray findings consistent with asbestosis in household and family members having no known exposure to asbestos other than residing with a known asbestos worker. These two studies demonstrate the presence of asbestos disease in the absence of continuing new known exposures.

Wagoner et al (*22*) demonstrated a significantly increased risk of death from nonmalignant respiratory disease and for diseases of the heart, which in part were secondary to pulmonary disease, among a cohort of workers in a major manufacturing complex using predominantly chrysotile. Among those workers observed 20 or more years after onset of employment, a fourfold increased risk of death due to nonmalignant respiratory disease was observed. Further evaluation of these deaths revealed that the majority occurred within one year after termination of employment and at an average age of 53.8 years.

Newhouse (*23*) reported an increased risk of death from nonmalignant respiratory disease in male asbestos textile and insulation workers with low to moderate exposure.

Enterline and Henderson (*24*) reported that for all ages, only 18 deaths from asbestosis occurred in several asbestos plants studied from 1941 to 1969. It is significant to note, however, that the state of New Jersey alone, in the years 1969-1970, had awarded workman's compensation for asbestosis to 455 workers from one of the plants in the study (*25, 26*).

Selikoff (*20*) reported a significant excess of deaths due to asbestosis among a group of workers in the U.S. and Canada. Out of 17,000 asbestos insulation workers, there were 119 observed deaths attributed to asbestosis. Although it was not reported, the expected death rates from asbestosis in the general population would be virtually zero.

Description of Asbestosis

Asbestosis is a chronic lung disease due to the inhalation of asbestos fibers and is characterized by diffuse interstitial fibrosis, frequently associated with pleural fibrosis (thickening) or pleural calcification. The characteristic x-ray changes of asbestosis are small irregular opacities in the lower and middle lung fields, often accompanied by pleural thickening and pleural calcifications.

The pulmonary fibrotic changes develop slowly over the years — often progressively even without further exposure — and their radiographic detection is a direct correlate of their extent and profusion. In some cases, minor fibrosis with considerable respiratory impairment and disability can be present without equivalent x-ray changes. Conversely, extensive radiographic findings may be present with little functional impairment.

Commonly found in asbestosis are pulmonary rales, dyspnea, finger clubbing, and cyanosis, but any or all can be absent in any one case. Pulmonary hypertension is frequently associated with advanced asbestosis, and the resultant cor pulmonale (right-sided heart failure) may be the cause of death.

Carcinogenicity

Occupational Exposure: Historical Studies

In 1935, 55 years after the start of large-scale usage of asbestos in industry, suspicion of an association between asbestosis and lung cancer was reported by Lynch and Smith (*27*) in the U.S. and by Gloyne (*28*) in the U.K. About 10 years later, case reports of pleural and peritoneal tumors associated with asbestos appeared (*29-31*). Epidemiologic evidence from Doll (*32*) showed a tenfold excess risk of lung cancers in those U.K. asbestos textile workers who had been employed before 1930, before regulations produced improved dust conditions in factories. Similar findings were reported in the U.S. in 1961. Mesotheliomas were also detected, but this fact was not published until later (*33, 34*). Possible variations in risk with different types of fiber were rarely considered in the early reports. Since 1964, following the

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recommendations of the UICC Working Group on Asbestos Cancers (UICC 1965) (*35*) for new studies, there has been an expansion of epidemiologic studies in many parts of the world.

Epidemiologic Studies: Lung Cancer, Pleural and Peritoneal Mesotheliomas

Mixed Types of Fiber. In most industrial processes different types of fiber are mixed, so that pure exposures to a single asbestos type are rare. Mortality studies of defined populations of asbestos-manufacturing, insulating and shipyard workers have provided the most concrete evidence concerning the association between bronchial cancer, pleural, and peritoneal mesotheliomas and exposure to asbestos. Reports have come from several countries: England (*36, 37*), Germany (*38*), the United States (*39*), The Netherlands (*40*), and Italy (*41*).

Elmes and Simpson (*42*) have extended their earlier report (*37*) to include deaths occurring since 1965 through 1975. The mortality trend has shifted from a preponderance of asbestosis and gastrointestinal deaths to malignancies from lung cancer and mesothelioma, diseases associated with longer latent periods. These authors report their findings would suggest any standard based "on the prevention of asbestosis may not provide adequate protection against neoplasia."

A sevenfold excess of lung cancer was found in a group of insulation workers whose exposures had been to chrysotile and amosite, but not crocidolite (*43*). Enterline and Henderson (*24*) reported a 4.4 times increased risk of respiratory cancer mortality among retired men who had worked as production or maintenance employees in the asbestos industry and who had been exposed to mixed fibers. Among men with mixed exposure to crocidolite and chrysotile in the asbestos cement industry, the rate was 6.1 times the expected rate. In a British naval dockyard population, Harries (*44*) showed that there had been a steep rise in mesotheliomas since 1964. However, the full biologic effects of asbestos in shipyard workers would not have been expected to be detected until the 1970's and thereafter (*20*).

Edge (*45*) reported that shipyard workers with mixed asbestos exposure and pleural plaques (without evidence of pulmonary fibrosis) had a 2.5 times increased risk of developing carcinoma of the bronchus, when compared with matched controls without plaques. In a study of sheet metal workers (*46*) with measurable and mixed asbestos exposure, an excess of deaths from malignant neoplasms (24.7% of deaths for two cohorts selected for 5 or more years worked in the trade, 19.1% of deaths for a group with death claims where 14.5% was expected) was largely attributed to an excess of malignant tumors of the respiratory tract. Of the 307 deaths in the first cohort, 32 lung cancer deaths were significantly in excess (1.7 times the expected). One pleural mesothelioma was observed.

Additional confirmatory evidence of the association between mesotheliomas and past exposure to asbestos comes from many institutes and departments of pathology and cancer registers (*47-54*). These studies have shown an association between asbestos and mesothelioma even with exposures as brief as one day; however, approximately 15% of the mesotheliomas are not known to be related to exposure to asbestos. Three studies (*50, 55, 56*) showed a poor correlation between certified cause of death and histologic diagnosis of mesothelioma. There is still a need to reduce the inter-observer variation in the diagnosis of these rare and pleomorphic tumors (*57*).

The ratio of pleural to peritoneal tumors appears to be associated with heavier exposures (*58*). Among a number of occupationally exposed groups studied, approximately 5-7% of deaths have been from mesotheliomas (*20, 59, 60*). More recently, however, an estimate has projected that 11% of asbestos workers' deaths in England will be from mesotheliomas (*61*).

Weill et al. (*62*) reported on the mortality experience of a cohort of 5,645 men employed in production of asbestos cement products and who had at least 20 years since first exposure. These workers were exposed largely to chrysotile with some crocidolite and amosite. Among this group, 601 persons were identified as deceased by the Social Security Administration. Those with unknown vital status (25%) by this source were assumed alive, thus likely resulting in underestimation of the true risk. Death certificates were obtained for 91% of the known deaths. Dust exposures were estimated using each worker's employment history in conjunction with historical industrial hygiene data.

Weill et al. (*62*) observed increased respiratory cancer mortality only among those with exposure in excess of 100 mppcf-yr, where 23 cases were observed versus 9.3 expected. The unusually low SMR for all causes in the low-exposure groups suggest thus the possibility of a selection bias and any interpretation of risks at low exposures should be done with caution. Two pleural mesotheliomas were reported. Separating the cohort by type of fiber exposure, the authors concluded that the addition of crocidolite to chrysotile enhanced the risk for respiratory malignancy; however, an excess risk was observed among those not exposed to crocidolite with cumulative exposures in excess of 200 mppcf-months. Both average concentration of exposure

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and duration of exposure were found to be related to cancer risk.

Berry et al. (*63*) extended the 1968 observations by including persons completing 10 or more years employment by 1972. Persons who left after June 30, 1966, were also contacted and encouraged to participate with 68 of 113 persons eventually participating. Outcome measures studied included chest radiographs, medical examination including assessment of basal crepitations and pulmonary function (FEV, FVC, FRC, TLC RF, TL, $PaCO_2$).

Chest films were read by four readers by the ILO/UC 1971 classification system with readings being averaged. Dust exposures were estimated for each person using available hygiene data and estimates of control effectiveness. In this study, "possible asbestosis" was diagnosed based on one or more combinations of basal rales or crepitations, radiological changes, a falling transfer factor, and restrictive lung function changes. Among these 379 men, 60 cases of possible asbestosis were diagnosed by the factory medical officer, whereas 85 cases were diagnosed by an independent clinician. Collaboration by these investigators subsequently resulted in 82 men with crepitations, 58 "possible asbestosis," and 34 certified asbestosis. Using the exposure data these authors estimated the cumulative dose necessary for a 1% incidence for crepitations, possible asbestosis, and certified asbestosis to be 43 fiber/cm^3-yr, 55 fiber/cm^3-yr, and 72 fiber/cm^3-yr, respectively. These authors point out limitations of the cumulative dose concept and acknowledge the imprecision of their exposure estimates. Two cases of certified asbestosis were observed among nonsmokers and nine among ex-smokers. There were, in general, fewer respiratory symptoms and signs in nonsmokers and light smokers than in heavy and ex-smokers.

Baselga-Monte and Francis' examination (*64*) of 1262 workers employed in four factories in the Barcelona area demonstrated a dose-response relationship based upon radiologic images. The authors demonstrate a quick response for pleural radiological changes at individual cumulative doses as low as 5 fiber-year/cm^3 while the pulmonary and pleuropulmonary responses tend to appear later but not at statistically different doses. The authors are reluctant to draw conclusions because of the design of the epidemiologic evaluation which considered only active employees. Other epidemiologic studies of worker populations would indicate that evaluation of only active employees would tend to underestimate the health risk since diseased workers oftentimes tend to self-select out of the active workerforce (*65-67*). Baselga-Monte and Francis conclude that "the present worldwide trend to establish more exigent hygienic criteria for exposure to asbest(os) is confirmed". Based on their working model this level for a 50-year working life should be 0.07-0.10 fiber/cm^3, "(taking into account protection levels of 99 and 95%)."

Crocidolite. In 1956, Wagner started investigating the occurrence of pleural and peritoneal mesotheliomas in the crocidolite mining areas of the Northwest Cape Province in South Africa. It was shown that these tumors occurred in the non-mining population living in the vicinity, as well as among men working in the mines and mills and in the transportation and handling of the fiber (*68*). Asbestosis was not invariably present. The latent period between first exposure and clinical recognition of the tumor was long — a mean of 40 years. Subsequent surveillance of the mining population in all the asbestos-producing areas in South Africa has added support for a major difference in the incidence of mesotheliomas within the crocidolite mining areas of that country (*69, 70*). The mining of crocidolite in northwest Australia has been associated with mesotheliomas (*71*). Jones et al. (*72*) have reported a high incidence of mesotheliomas among women who worked with crocidolite in a factory producing gas mask canisters during World War II.

McDonald and McDonald (*73*) have also studied the mortality of 199 workers exposed to crocidolite during gas mask manufacture in Canada during 1939 to 1942. This cohort was followed through 1975 and 56 deaths occurred. Out of these 56 deaths, four (7%) were from mesothelioma and eight (14%) from lung cancer. This was compared to 0.26% mesothelioma deaths among chrysotile miners and millers in Canada. It should be pointed out that an additional five mesotheliomas not reported on death certificates were diagnosed on review of pathology or autopsy material.

Chrysotile. McDonald et al. (*55, 74*) reported an increased risk of lung cancer among men employed in Quebec chrysotile mines and mills. The risk of lung cancer among those workers most heavily exposed was five times greater than those least exposed. These same chrysotile miners and millers of Quebec as of 1977 had experienced nine confirmed and two suspected mesotheliomas (*75*). The author concluded (*75*) for the seven cases observed at Thetford mines that, "There is therefore no good reason to doubt chrysotile exposures as the cause."

Kogan et al. (*76*) investigated the cancer mortality among workers in asbestos mining and milling industries between 1948 and 1967. The total cancer mortality rate among workers was 1.6 times higher than that found in the general male population; for female workers the rates were 0.8 for those in mines and 1.3 for those in mills. The lung cancer risk for male miners and millers was twice that of the general

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male population. For females in mines and mills, the risks were 2.1 and 1.4 times that of the general female population, respectively. For those workers over 50 years of age, the risk of lung cancer was greater: for men in mining, 4.0; those in milling, 5.9; for women in mining, 9.5; and for those women in milling, 39.8 times that found in the general population. No mesotheliomas were found, but Kogan et al. (*76*) indicate that this might be explained by the insufficient experience of pathologists with this rare type of cancer in that geographical area. Also, the number of people in the study populations was not reported.

Selikoff (*77*) surveyed 485 current employees of a chrysotile mine in Baire Verte, Newfoundland, which had been in operation since 1963. Fifty employees (10%) had one or more radiographic abnormalities of the type commonly associated with asbestos exposure. Parenchymal abnormalities were most common while pleural changes were detected in only 3% of the individuals surveyed. For those individuals with less than 5 years employment, the prevalence of abnormalities was 5% and this increased with duration of employment. Changes occurred most commonly in those with the most intense exposures. This study was designed only to assess asbestos-related disease under more modern conditions than have previous studies (*76, 78*), and thus short duration of exposure and long latency could not be studied. The interpretation of these data is further complicated by the lack of a cohort population and environmental measurements. The study does demonstrate the prevalence of chest x-ray changes in an appreciable proportion of employed workers despite a short period from onset of initial exposure.

Wagoner et al. (*22*) reported on the cancer risk among a cohort of workers in a major manufacturing complex utilizing predominately chrysotile asbestos in textile, friction, and packaging products. An excess of respiratory cancer occurred among asbestos workers in each duration-of-employment category down to and including 1-9 years. They observed statistically significant standard mortality ratios of 122 for all malignant neoplasms and 244 for malignant neoplasms of the respiratory system. The asbestos workers in this study were located in an area of predominately Amish Dutch population with known low frequencies of smoking. The authors, nevertheless, used the general white male U.S. population as a control group, which would tend to underestimate the degree of risk.

Robinson et al. (*79*) reported an additional 8 years of observation and 385 deaths to the Wagoner et al. (*22*) study of mortality patterns of workers among one facility manufacturing asbestos textile, friction and packing exposed predominately to chrysotile. Chrysotile constituted over 99% per year of the total quantity of asbestos processed, except for 3 years during World War II. During these 3 years, amosite was selectively used to a limited extent because of Naval specifications and accounted for approximately 5% of the total asbestos used per year. Crocidolite and amosite for the other years accounted for less than 1% of total usage in very selected areas. Exposures to these other two types may have played a role in the etiology of disease; however, due to the overwhelming exposure of the cohort to chrysotile it is likely that the other exposures played a minor role in the overall mortality patterns. Robinson et al. confirmed the observations of Wagoner et al. of statistically significant excess deaths due to bronchogenic cancer, suicide, heart disease and nonmalignant respiratory disease, including asbestosis and nonstatistically significant excesses of digestive cancer and lymphoma. Robinson et al. described 17 mesotheliomas, whereas no mesotheliomas were detected in the Wagoner et al. study where observation of mortality ceased in 1967. The appearance of 17 mesotheliomas in the updated study reflect latency periods of 24 to 53 years since onset of first exposure. Further analysis indicated 3 of the 17 mesothelioma deaths occurred during the original study period but were undetected until later analysis. This observation confirms earlier findings by Mancuso and El-Attar (*80*) that mesotheliomas are characterized by very long latency periods.

Weiss et al. (*81*) reported no unusual mortality experience over a 30-year period for a cohort of workers employed in a paper and millboard plant reported to be using only chrysotile. The author concluded that the study results were suggestive of a minimal hazard from chrysotile. This conclusion must be viewed in light of the limitations inherent in the study. First, the study population was small (n = 264) and only 66 workers had died at the time of analyses. Moreover, the unusually low SMR for many of the contrasts in the Weiss et al paper suggests the possibility of a selective bias greater than usually seen in contrasting industrial populations with the general population.

Enterline and Henderson (*24*) found that for retired men who had worked as production or maintenance employees in the asbestos industry and who had reached 65 years of age, those who had been exposed only to chrysotile had a respiratory cancer risk 2-4 times greater than that expected. Among men within the asbestos cement industry exposed only to chrysotile, a one- to fourfold excess of respiratory cancer was found. Of 802 deaths, only one mesothelioma had been recorded in the several plants investigated. In contrast, a subsequent investigation by Borow et al. (*67*) found 70 cases of meso-

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thelioma from only one of these plants. The discrepancy was due to methodologic variations, for example, Enterline and Henderson (*24*) had limited their investigation to men age 65 or over, while many of the mesothelioma cases reported by Borow et al. (*67*) had died before that age.

Acheson et al. (*82*) estimated a 6-fold risk of mesothelioma for persons with chrysotile in the lung tissue.

Amosite and Amphibole Fibers in the Cummingtonite – Grunerite Ore Series. Exposures to amosite alone in a factory making insulation material were reported by Selikoff (*20*) and Selikoff et al. (*83*). Ten mesotheliomas were found in addition to an increased risk of lung cancer in workers who were observed 20 years or longer. The excess lung cancer risk in the amosite workers was shown to increase with duration of employment. There was a three-fold increase in lung cancer among those with less than 3 months employment and among those with less than 1 month employment there was a 2.25-fold increase.

In a retrospective study of 914 men who had worked periodically during World War II in a plant manufacturing insulating materials from amosite for the U.S. Navy, Seidman et al. (*84*) concluded that the group of 65 men who had worked for less than 1 month had experienced excess mortality, on the age-specific basis, from lung cancer during the 30 years since the beginning of their exposures, but not from all cancers or all causes of death. Men who had worked for a full month or longer had excess mortalities from all three causations examined, the risk of death from lung cancer increasing with duration of exposure.

Seidman et al. (*85*) have extended their study of amosite asbestos workers with short-term exposures. The study group consisted of 820 men first employed between June 1941 and December 1945 in the production of asbestos insulation. Follow-up was through 1977 with expected deaths adjusted for age and calendar time estimated using death rates for white males in the general population of New Jersey.

Among the cohort studied by Seidman et al., 83 lung cancers were observed according to death certificate information whereas 23.1 were expected. Among 61 men employed less than 1 month, three lung cancers were observed versus 1.3 expected. Although based on small numbers, excess mortality from lung cancer showed an increasing trend with employment duration. Cancer latency periods were progressively shortened with increasing employment duration. Four mesotheliomas were reported on death certificates in contrast to 14 which were identified on autopsy and other tissue diagnosis. Three in the group had less than 1 year of exposure. Although no environmental data are available for this plant, dust counts were made in another plant of the same company using the same fiber type and production process. Seidman et al. reported average exposure at this plant to be 23 fiber/cm^3. Further data available for this comparison plant were published by NIOSH (*86*) showing mean exposures to range from 14 to 75 fibers/cc. At such concentrations, a lung cancer relative risk of 2.3 could be calculated with employment less than one month.

Johnson et al. (*87*) have documented the hazards of asbestosis encountered among workers at a plant manufacturing amosite insulation products in Tyler, Texas. Seven of 18 workers with ten or more years employment at the plant had asbestosis, established by the presence of at least three of five criteria including restrictive pulmonary function, chest x-ray abnormalities, rales, clubbing, and significant dyspnea.

Murphy et al. (*88*) reported a follow-up to their first report (*14*) of shipyard pipe covers exposed predominately to amosite asbestos. Workers in the original Murphy report of 1971 with "asbestosis" diagnosed by multiple criteria had a poor prognosis reported in the 1978 longitudinal survey. The studies of Johnson et al. and Murphy et al. demonstrate the respiratory morbidity associated with manufacture and use respectively, of amosite insulation products.

Gilliam et al. (*89*) reported a threefold excess risk of mortality from respiratory cancer and a twofold excess of nonmalignant respiratory disease (excluding influenza and pneumonia) among miners exposed to amphibole fibers in the cummingtonite-grunerite ore series at concentrations less than 2.0 fibers/cm^3 and a large majority shorter than 5 μm in length. McDonald et al. (*90*) in a subsequent study of the same mine examined the mortality experience of persons with at least 21 years of employment at the mine which showed significant excesses of pneumoconiosis (mainly silicosis), tuberculosis, and heart disease. No overall excess of malignant diseases were found; however, when the population was broken down by exposure, respiratory cancer was in excess. The contrast between the cancer findings of the two studies are not surprising since McDonald et al. looked only at workers surviving at least 21 years of employment. Gilliam et al., on the other hand, did not use such a rigid survival criterion. They included all miners with 5 years employment underground who had never mined underground elsewhere. The findings of McDonald et al. do not negate those reported by Gilliam et al. but rather tend to strengthen them, in that McDonald used rigid survival criteria and further diluted the underground exposed effect by including persons never exposed underground thus any excesses may be underestimated.

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Anthophyllite. In Finland, anthophyllite mining has been associated with an excess bronchial cancer risk of 1-4 times the expectation overall, and about double this figure for those with more than 10 years' exposure (*91*). There was also a higher prevalence of dyspnea and cough in the miners. However, no mesotheliomas were found despite the presence in Finland of an unusually high incidence of pleural thickening and calcification as detected by radiographic and pathologic surveys (*92, 93*).

Other Types of Cancer

Epidemiologic studies of the already defined populations have consistently shown an excess risk of other cancers, especially of the gastrointestinal tract (*22, 37, 58, 74, 76, 80, 94*); however, it has been less than that of lung cancers.

Schneiderman (*95*), in a literature review with an emphasis on dose response, concluded that "good dose-response data, with quantitative estimates of dose are uncommon; however, in all the literature reviewed, only one paper did not support the conclusion that increased exposure to inhaled asbestos particles leads to increased digestive system cancer."

Robinson et al. (*79*) reported an excess of deaths due to lymphosarcoma and malignant lymphoma among white males employed in an asbestos textile, friction and packing products manufacturing facility. There were seven deaths due to cancer of these sites, while 3.28 cases were expected.

Stell and McGill (*96*) found that out of 100 men with squamous carcinoma of the larynx, 31 had known exposure to asbestos compared with only three in matched controls. Similar associations have been reported by Shettigara and Morgan (*97*). Newhouse and Berry (*98*) found two cases of cancer of the larynx (ICD 161) in their cohort of over 4000 workers compared with an expected 0.4.

Nonoccupational Exposure

Household contact with asbestos is associated with an increased mesothelioma risk. Anderson et al. (*21*) have reviewed 34 such cases of mesothelioma from nine countries and reported four new cases among the traced family members of 1664 asbestos workers.

Anderson et al. (*99*) (1979) have continued to evaluate the risks of nonmalignant and malignant disease associated with household exposure to work-derived amosite dust. Four mesothelioma cases were reported among household contacts of former workers at a plant manufacturing amosite insulation products in Paterson, New Jersey. Anderson et al. also reported a 35.9% prevalence of radiographic abnormalities among household contacts of former employees at this same amosite plant compared to a 4.6% prevalence among a control group. These radiographic abnormalities included pleural thickening, pleural calcification, pleural plaques, and irregular opacities. These studies raise the spectre of nonoccupational hazards associated with casual or low-level exposures to amosite. Cases of mesotheliomas have also occurred in nonoccupationally exposed individuals living in the neighborhood of industrial sources of asbestos (*36, 100, 101*). Studies of the geographical distribution of cases of mesothelioma in the UK over a 10-year period indicate that the new cases are nearly all from areas in which there has been a recognized industrial source of asbestos (*50, 102*).

Lesions among nonoccupationally exposed persons in Finland have been reported where anthophyllite asbestos is mined. In this study, 118 cases of the total 126 cases of roentgenologically-diagnosed pleural calcification studied, excluding those individuals with hemothorax, emphysema, and tuberculosis, lived or have lived in areas immediately adjacent to asbestos mines (*92*). The results of this study suggest a health hazard from community exposure to ambient asbestos.

Synergism

There is marked enhancement of the risk of lung carcinoma in those workers exposed to asbestos who also smoke cigarettes (*103, 104*). Hammond and Selikoff (*60*) interpret the excess lung carcinoma risk from asbestos in nonsmokers to be small. No link between cigarette smoking and mesotheliomas has been observed in a prospective study by Hammond and Selikoff (*60*). A preliminary study (*105*) on female workers employed between January 1940 and December 1967 in a predominately chrysotile asbestos textile plant revealed seven lung cancer deaths among 580 women when only 0.63 deaths were expected ($p < 0.01$). One lung cancer death was observed in a smoker, two in women of undetermined smoking history, and four in "never" smokers as determined from hospital admission charts.

It is important to note that the historic documentation of cigarette consumption patterns is lacking for most retrospective cohort studies of asbestos workers. It is further important to note that a sizable portion of the general population, the group usually selected for comparison in these studies, are cigarette smokers. Therefore, the risk of lung cancer demonstrated for these industrial groups exposed to asbestos is of such magnitude as to preclude the identification of an independent etiologic role for cigarette smoking.

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Hammond et al. (*106*) have attempted to correct this methodological problem by comparing 12,051 asbestos insulation workers having complete smoking histories to a control population also with no smoking histories. Hammond et al. used as a control population 73,763 men from the American Cancer Society's prospective cancer prevention study who were similar to the asbestos workers in that they were white males; nonfarmers; had no more than high school education; had a history of occupational exposure to dust fumes, vapors, gases, chemicals, or radiation; and were alive as of January 1, 1967. Nonsmoking asbestos workers showed a five times greater risk of dying from lung cancer than their nonsmoking controls, while smoking asbestos workers also had a five times greater risk of dying of lung cancer than their smoking controls. Both smokers and nonsmokers exhibited a fivefold relative risk; however, the attributable risk was greater among the smokers. This higher attributable risk can be accounted for by the large number of smokers in the asbestos-exposed population and the comparison population.

Liddell et al. (*107*) has also looked at smoking among asbestos workers through administering a questionnaire to living workers or relatives of deceased workers dying after 1951. The authors report SMR's of 48 and 46 for nonsmokers and ex-smokers, increasing to 206 for heavy smokers. This study, however, suffers since smoking specific death rates were not used for calculation of expected lung cancer deaths underestimating the risks among nonsmokers.

None of the epidemiologic studies have shown an increase of pleural or peritoneal mesothelioma among smokers as compared to nonsmokers.

Conclusion

In addition to the nonmalignant effects of asbestos, all commercial forms of asbestos have been shown to be carcinogenic in man, and occupational exposure during mining where asbestiform fibers occur also accounted for an excess of cancer in miners. In addition, household contact with asbestos workers has demonstrated a risk for nonmalignant and carcinogenic effects.

It has been estimated that of the 4 million workers heavily exposed to asbestos, at least 1.6 million (35-44%) are thus expected to die of asbestos-related cancers, while in the absence of asbestos exposure only about 0.35 million (8-9%) would have been expected to die of cancer. Broken down more specifically, these cancer deaths include 20-25% from lung cancer, 7-10% of pleural or peritoneal mesothelioma, and 8-9% of gastrointestinal cancer. It is estimated that between 58,000 and 75,000 asbestos-associated deaths will occur each year, which will account for between 13 and 18% of the total cancer deaths in the United States (*108*).

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