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Geological Survey

A Survey of Asbestos-related Disease in Trades and Mining Occupations and in Factory and Mining Communities as a Means of Predicting Health Risks of Non-Occupational Exposure to Fibrous Minerals*

by

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INTRODUCTION

The Problem

The widespread use of amphibole and serpentine asbestos[1/] by industrial society for such items as service in brake and clutch facings, electrical and heat insulation, fireproofing materials, cement water pipe, tiles, filters, packings, and construction materials, has contributed greatly to human safety and convenience. Yet, while our society was accruing these very tangible benefits, many asbestos workers were dying of asbestosis, lung cancer, and mesothelioma.

The hazards of certain forms of asbestos under certain conditions have been so great that several countries have taken extraordinary actions to greatly reduce or even ban their use. Recent experiments with animals demonstrate that the commercial asbestos minerals as well as other fibrous materials can cause tumors to form when the fibrous particles are implanted within the pleura. These experiments have convinced some health specialists that asbestos-related diseases can be caused by many types of elongate particles; the mineral type according to these health specialists, is not the important factor in the etiology of disease, but rather the size and shape of the particles which enter the human body.

The question now before the World's health and regulatory establishments is whether the hazards of asbestos outweigh the benefits. Should the asbestos minerals and perhaps other asbestos-like minerals be banned from use? Minerals belonging to the amphibole group are particularly important in this regard for they are ubiquitous and commonly have crystalline habits which are considered by some to be asbestos-like.

The Dilemma

The concern for human health, the great usefulness of many asbestos products, the appearance of asbestos minerals or asbestos-like minerals in the natural background and in many kinds of mining operations, and the uncertainty of the exact health effects of different kinds of minerals, different mineral particle sizes, and different mineral dust concentrations combine to present a formidable problem to minerals scientists, the

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1/At present, the most widely used definition of asbestos in the United States, is from the notice of proposed rule-making for "Occupational Exposure to Asbestos" published in the Federal Register (Oct. 9, 1975, p. 47652, 47660) by the U.S. Occupational Safety and Health Administration (OSHA). In this notice, the naturally occurring amphibole minerals amosite, crocidolite, anthophyllite, tremolite, and actinolite and the serpentine mineral chrysotile are classified as asbestos if the individual crystallites or crystal fragments have the following dimensions: length greater than 5 micrometers, maximum diameter less than 5 micrometers, and a length-to-diameter ratio of 3 or greater. Any product containing any of these minerals in this size range is also defined as asbestos.

minerals industries, and legal and health professionals. Must the use of all commercial asbestos be stopped? Must all mine dusts containing such particles be controlled to the lowest feasible levels and wastes from those mines be considered toxic and thus isolated from surrounding air and water? Must all asbestos be eliminated from our drinking water, our schools, and our public buildings? Must we cease to use asbestos in brake linings, cement water pipe, and structural building materials? Must even low levels of non-occupational exposure to asbestos or asbestos-like materials be avoided at any cost?

In order to obtain an insight that will enable us to intelligently address these questions I will review the role of asbestos in the world economy, the important geological occurrences of commercial asbestos, give estimates of asbestos-related mortality in the United States in the recent past, and document the incidence of asbestos-related disease in the trades occupations, the mining and milling occupations, and in those non-occupationally exposed to asbestos or asbestos-like minerals. As we will see, the six asbestos minerals used in commerce are not identical in their crystal structure, chemical composition, abundance, and geologic occurrence; nor do the different asbestos dusts have the same impact on human health. Instead of treating all asbestos minerals as equally potent carcinogens (apparently the prevailing opinion in the United States) each mineral should be examined on its own merits and demerits with regard to its usefulness to society and its potential to cause disease.

ASBESTOS IN THE WORLD ECONOMY

Early Beginnings

Whereas the general use of asbestos in international commerce dates only to the late 19th century, its utility in human culture goes back to at least 2500 B.C. Archeological studies (Europaeus-Äyräpää, 1930) show that the inhabitants of the Lake Juojarvi region of East Finland knew how to strengthen earthenware pots and cooking utensils with anthophyllite asbestos. This asbestos probably came from the same areas where it has been commercially exploited in recent times. Until recently, however, most other uses of asbestos were trivial; such as its fabrication into such curiosities as cremation cloth, tablecloths, lamp wicks, and purses. Even well into the last century, asbestos could not be regarded as a product of commerce unless one included such endeavors as the small industry developed in Russia during the rule of Peter the Great where chrysotile asbestos from the Urals was used for a short period of time in the production of textiles.

In the 1860's and 1870's, the market for asbestos products rapidly changed - probably for three reasons; the need for insulation for the new steam technology, the formation of an international trading company of Italian and English entrepreneurs, and the reopening of the chrysotile asbestos deposits of northern Italy and simultaneous exploitation of the vast chrysotile resources in Quebec. The supply for the first time was ample, the market was ready.

The Modern Industry

The reopening of the asbestos deposits of northern Italy, deposits which had been worked as far back as Roman times, marked the beginning of the modern asbestos industry. By 1890, the asbestos industry was full blown, with hundreds of applications being introduced (Jones, 1890); by the turn of the century the large South African crocidolite deposits had been opened up and the Russian deposits in the Urals were once again producing in large quantity. Within a few years, the amosite deposits of the Transvaal would be exploited.

From the time of the first recorded use of asbestos by Stone Age man to 1900 the total world production of all types of fiber was probably about 200,000 metric tonnes, certainly no more than 300,000 tonnes. Of this 150,000 tonnes came from Quebec. By 1980 more than 100 million tonnes of asbestos had been mined throughout the World; of this more than 90 percent was chrysotile and more than 5 percent crocidolite and amosite. Nearly 40 million tonnes of this total World production was chrysotile mined in Quebec Province near the towns of Thetford Mines and Asbestos. Total production of anthophyllite asbestos to date is probably no more than 400,000 tonnes; 350,000 tonnes being produced by Finland alone. Production of tremolite asbestos has been sporadic and it has been mined in various parts of the World for short periods of time. Total production to date for this form of asbestos is probably no more than a few thousand tonnes. Commercial exploitation of actinolite asbestos is practically unknown.

The World asbestos production for 1978 is given in Table 1. Russia leads with 46.1 percent and Canada is second with 28.9 percent of the world output. Both countries mine only chrysotile asbestos and most of the fiber comes from the Urals and Quebec. The third leading asbestos producer is the Republic of South Africa (7.1 percent); the asbestos ore consists of amosite, crocidolite, and chrysotile. These three countries furnished 82.1 percent of the World's asbestos in 1978. The other countries listed in Table 1 produce mostly chrysotile.

COMMERCIAL ASBESTOS

The Asbestos Minerals

Standard references published over the past 50 years usually list six forms of commercial asbestos; the amphibole varieties are amosite, crocidolite, anthophyllite, tremolite, and actinolite, the serpentine variety is chrysotile. A detailed understanding of the chemistry and crystal structures of these asbestos minerals postdate their discoveries; thus some of the older literature can be confusing with regard to mineral identifications.

Chrysotile, $Mg_3Si_2O_5(OH)_4$, one of the three common polymorphs of serpentine is generally fibrous although non-fibrous varieties are known. About 90 percent of the past and about 95 percent of the present World production of asbestos was or is the chrysotile form.

Amosite is the very rare asbestiform variety of grunerite amphibole, $(Fe,Mg)_7Si_8O_{22}(OH)_2$; this varietal name is derived from the word Amosa - an acronym for the company "Asbestos Mines of South Africa" (Hall, 1918, p. 13-14). This valuable commercial asbestos is mined only in the Transvaal Province of South Africa.

Crocidolite is the asbestiform variety of riebeckite amphibole, ideally $Na_2(Fe^{2+},Mg)_3Fe^{3+}_2Si_8O_{22}(OH)_2$, and has been mined in only four localities; in the Transvaal and Cape Provinces of South Africa, in the Hammersley Range area of Western Australia, and in the Cochabamba area of Bolivia. Only the South African mines are still active.

The only other form of amphibole asbestos that has been mined commercially on a significant scale is anthophyllite, $(Mg,Fe)_7Si_8O_{22}(OH)_2$, from the Paakkila area of East Finland. With the Finnish mines now closed there is now very little anthophyllite asbestos production anywhere in the World.

There are numerous reports of minor occurrences of tremolite asbestos, $Ca_2Mg_5Si_8O_{22}(OH)_2$, and relatively few reports of occurrences of actinolite asbestos, $Ca_2(Fe,Mg)_5Si_8O_{22}(OH)_2$. Tremolite and actinolite asbestos are now, as they have been in the past, of little economic importance.

The Important Geological Occurrences of Commercial Asbestos

Many minerals, including the amphiboles and some serpentines, are described variously as fibrous, asbestiform, acicular, filiform, prismatic; these terms suggest an elongate habit. Although such minerals are extremely

Table 1. World asbestos production in 1978 (Clifton, 1979)

Fiber Locality	Production (in thousands of metric tonnes)
Chrysotile	
North America	
Canada	1620
United States	93
South America	
Argentina	1
Brazil	100
Europe	
Bulgaria	21
Italy	162
U.S.S.R.	2582
Yugoslavia	10
Africa	
Zimbabwe	210
South Africa	118
Swaziland	48
other	1
Asia	
China	210
Cyprus	37
India	21
Japan	7
Korea	7
Taiwan	1
Turkey	10
Oceania	
Australia	58
(World chrysotile total)	5317
Crocidolite	
South Africa	210
Amosite	
South Africa	71

common, in only relatively few places do they have physical and chemical properties suitable to be valuable as commercial asbestos. Locally, amphibole minerals may show an asbestiform habit, for example in vein fillings and in areas of secondary alteration, but usually they do not appear in sufficient quantity to be profitably exploited.

Deposits of commercial asbestos are found in four types of rocks: (I) - alpine-type ultramafic rocks including ophiolites (chrysotile, anthophyllite, and tremolite), (II) - stratiform ultramafic intrusions (chrysotile and tremolite), (III) - serpentinized limestone (chrysotile), and (IV) - banded ironstones (amosite and crocidolite). Type I deposits are by far the most important and probably account for more than 85 percent of the asbestos ever mined. The most important Type I deposits are those of Quebec and the Urals.

Type II deposits are found mostly in South Africa, Swaziland and Zimbabwe. These furnish mostly chrysotile asbestos. Type III deposits are small; the most notable of these are located in Globe, Arizona and in the Carolina area of the Transvaal Province of South Africa. Type IV deposits are found only in the Precambrian banded ironstones of the Transvaal and Cape Provinces of South Africa and of Western Australia. Only the South African deposits are still in production. A complete review of the geological occurrences of commercial asbestos is given by Ross (1981).

HEALTH HAZARDS OF ASBESTOS

Diseases Related to Asbestos Exposure

Three principal diseases are related to exposure to one or more of the commercial asbestos minerals. These are: (1) lung cancer which includes cancer of the trachea, bronchus, and lung proper; (2) mesothelioma, a cancer of the pleural and peritoneal membranes which invest the lung and abdominal cavities, respectively; and (3) asbestosis, a diffuse interstitial fibrosis of the lung tissue often leading after long exposure to severe loss of lung function and respiratory failure. The occurrence of lung cancer in asbestos workers is also complicated by the association with cigarette smoking which leads to considerable difficulty in assigning relative risks of asbestos exposure to smokers. Mesothelioma, a disease which is usually fatal in one to two years after diagnosis, is rare, accounting for less than 300 deaths per year in the United States and Canada.

Some epidemiological studies suggest that asbestos workers may suffer excess cancer of the digestive tract (Selikoff and Lee, 1978); other studies do not support this conclusion (McDonald and McDonald, 1980; Meurman et al., 1974; Rubino et al., 1979; Nicholson et al., 1979). Some question still exists then as to the role played by asbestos in the etiology of digestive tract cancers. Becklake (1976), Selikoff and Lee (1978), and Simpson (1979) give a complete review of the subject of asbestos and disease.

Particle size and shape appear to be the factors controlling whether mineral particles enter and remain in the lung or are removed from the lung after entering. Particles such as asbestos fibers which have diameters

greater than approximately 5 μm cannot enter the bronchial airways, those having smaller diameters do. Particles having diameters less than 1.5 μm are particularly dangerous for they can penetrate to the smaller bronchioles and even to the alveolar sacs (Davis, 1981). Most particles which enter the upper respiratory tract (the mainstem, bronchi, and bronchioles) are quickly and effectively removed by the mucocilliary escalator. A second lung clearance mechanism operates in the lower respiratory tract (the respiratory bronchioles and alveoli). Here, pulmonary macrophages engulf the foreign particles (phagocytosis) and then: (1) move to the upper respiratory tract to where the mucociliary escalator is operative or (2) move through the alveolar wall into the interstitium and eventually to the lymph channels.

Asbestos fibers which are longer than approximately 10 μm are not readily phagocytized by the macrophage cells and thus tend to remain in the lower respiratory tract or they may penetrate the pleural membrane and enter the interpleural space. Asbestosis may occur when such fibers remain in the lung parenchyma for lengthy periods of time. The asbestos fibers can stimulate deposition of excess interstial collagen and reticulin fibers. This causes the alveolar septa to become thickened with ensuing impairment of oxygen uptake (Davis, 1981). Long-term residency of fibers in the lung and pleura may also induce lung cancer and mesothelioma; the mechanisms by which this takes place are far from being understood.

As will be described later, pleural cancer seems to be induced by crocidolite asbestos but not by chrysotile or anthophyllite asbestos. Lung cancer is caused by chrysotile, anthyophyllite, amosite, and crocidolite asbestos; particularly in asbestos workers who smoke cigarettes. Two completely different substances, asbestos and cigarette smoke, combine to produce a very significant risk to those who have been heavily exposed to asbestos dusts.

Generally, asbestos-related diseases appear in asbestos workers only after many years have elapsed since first exposure. A significant increase in the lung cancer death rate appears 10 to 14 years after first exposure and peaks at 30 to 35 years. The mesothelioma death rate becomes significant 20 years after first exposure but continues to climb even after 45 years have elapsed. The asbestosis death rate becomes significant 15 to 20 years after first exposure and apparently peaks at 40 to 45 years (Selikoff et al., 1980a).

Epidemiology

Before considering the mortality studies of the various occupational groups exposed to asbestos, we should briefly consider the role the three important types of asbestos (amosite, crocidolite, and chrysotile) played in the commerce of North America and Europe, the areas where the major epidemilogical studies of asbestos workers were made.

In North America, chrysotile entered the market in large quantities early in this century. Crocidolite was apparently first used in the United States in 1912 when 9 tonnes were imported, but it was not until World War I that its use for high temperature insulation became established - particularly in the ship building industry. By 1930, 35,000 tonnes of crude crocidolite fiber had been imported into the United States. Import statistics for crude crocidolite asbestos from South Africa into the United States are given in Tables 2a and 2b. Large amounts of manufactured goods containing crocidolite were also imported but tonnage estimates cannot be made. Not until the mid 1930's did amosite asbestos gain a market in North America when it began to replace crocidolite for high temperature insulation. Crocidolite was milled in Bound Brook, New Jersey in 1920 and in 1924 the operation moved to larger facilities in Millington, New Jersey. The many advertisements in the trade journal Asbestos from 1920 to 1945 indicate that crocidolite was used in many products and particularly for insulation of steam boilers, locomotives, and pipes. As an example, a product containing crocidolite asbestos and called "85% Magnesian Sectional Pipe Covering" was advertised monthly in Asbestos from 1920 to 1945 (see also, McCullagh, 1980). Amosite, crocidolite, and chrysotile were almost universally used aboard ship during World War II; amosite for high temperature boilers and pipes, crocidolite for packings exposed to acids or salt water, and chrysotile for low temperature and electric insulation.

The use of asbestos in Europe paralleled that in North America, with one notable exception - the extensive use of crocidolite asbestos as a sprayed-on coating to fireproof ships,[1/] railroad cars, buildings, etc. Sprayed-on coatings were also used in the United States after World War II but the coatings contained, with few exceptions, chrysotile rather than crocidolite. Sprayed-on asbestos coatings were not used on U.S. ships; the principal use being to fireproof steel building girders and as acoustical coatings in schools and offices.

Asbestos trades workers. A very significant increased incidence relative to the general male population, of lung cancer, asbestosis and mesothelioma is found in men who were employed in the "asbestos trades" - insulation of steam locomotives, boilers, ships, buildings; fabrication and installation of asbestos-containing textiles, roofing materials, cement products, tiles, wallboards, brake linings, clutch facings, filters, packings, gaskets, etc. Those in the "trades" generally used several types of asbestos minerals during their working careers; most commonly these were chrysotile, crocidolite, and amosite, rarely anthophyllite. Significant exposures by any group of workers, at least for the past 40 years, to tremolite or actinolite asbestos dusts has probably not occurred.

[1/]Mesothelioma is prevalent in the shipyard workers of Europe; at Walcheren, Wilhelmshaven, Plymouth, Trieste, Hamburg, Nantes, Rotterdam, Malmö (McDonald and McDonald, 1977). The extensive use of crocidolite aboard European ships prior to and during World War II is suggested to be an important factor in the etiology of this disease.

Table 2a. Minimum estimates of imports of crude crocidolite asbestos from South Africa into the United States (1907-1929),* (1940-1945)**, (1946-1974).***

Year(s)	Crocidolite crude (short tons)
1907-1908	probably none
1909-1910	no data
1911	little
1912	9
1913	1
1914	no data
1915	probably none
1916	1184
1917	2081
1918	837
1919	1056
1920	2979
1921	704
1922	1684
1923	2040
1924	1457
1925	606
1926	4873
1927	5587
1928	?
1929	9952
Total (1907-1929)	35,050 short tons
1940	2708
1941	2976
1942	4213
1943	4808
1944	2946
1945	3100
Total (1940-1945)	20,751 short tons
1946-1974	346,796 short tons

*Some of the imports were shipped through England. Small amounts of chrysotile may be included in import figures but no amosite. Figures do not include any manufactured asbestos products. Most crocidolite came from the South Cape Prov., some may have come from the Transvaal. Source: Mineral Resources of the United States (1907)...(1929) U.S. Geological Survey, Washington, D.C.

**Crocidolite composed 19.4% of the South African crude asbestos imports into the U.S. during this period - which totaled 107,039 short tons, over 80,000 tons was amosite asbestos. Sources: Minerals Yearbook (1940)...(1945), U.S. Bureau of Mines, Washington, D.C. Mineral Trade Notes, Confidential Series, No. 1-31, U.S. Bureau of Mines, U.S. Dept. Interior, Washington, D.C. (1940-1945).

***Minerals Yearbook (1946)....(1974) U.S. Bureau of Mines, Washington, D.C.

Table 2b. Imports of all crude asbestos from South Africa (1930-1939)*

Year(s)	Asbestos Crude (short tons)
1930	3635
1931	2290
1932-33	1370
1934	4269
1935	2529
1936	?
1937	3025
1938	4248
1939	6422
Total	27,788 short tons**

*Import statistics do not differentiate between various forms of asbestos. Little chrysotile was imported from South Africa thus the figures are for mostly amosite plus crocidolite. Import data for crocidolite during World War II suggests that at least 20% of the 1930-39 imports were crocidolite. The amosite market was just developing in the U.S. in the 1930's. Most of the crocidolite was from the Cape Province.

**Assuming a ratio of 4:1 of amosite: crocidolite based on WWII import figures, at least 5558 short tons of crocidolite was imported into the U.S. from 1930 to 1939.
Sources: Mineral Resources of the United States (1930)(1931). U.S. Geological Survey, Washington, D.C. Minerals Yearbook (1932)... (1939). U.S. Bureau of Mines, Washington, D.C.

Statistical data for 21 mortality studies of defined cohorts of asbestos trades workers (mostly male) are presented in Tables 3a and 3b. Of those that are continuing prospective studies, the most recent update is given. Twelve of the studies are of asbestos factory workers, eight are of asbestos insulation workers, and one is of asbestos construction workers. In all, 50,143 individuals were followed (1,517 were female); of the 7,166 listed deaths 1,198 (16.7 percent) were reported as due to lung cancer and 402 (5.61 percent) were reported as due to mesothelioma. In the 21 studies, the lung cancer mortality accounted for 6.1 to 26.6 percent of all deaths; mesothelioma mortality accounted for 0 to 16.1 percent of all deaths (Tables 3a, 3b, Fig. 1). The workers involved in study No. VI worked only with chrysotile, those involved in Studies X and XXI worked mostly with crocidolite, and those in the remaining studies probably worked with more than one form of asbestos.

Estimates of expected cancer mortality are very difficult to predict, for cancer rates are modified by the individual's "lifestyle" as well as by occupation. The "lifestyle" contribution to lung cancer is cigarette use. To better assess the significance of these health studies it is necessary to examine the cancer-mortality patterns of cigarette smokers who were not exposed to asbestos dusts. Unless prevalence of smoking within the study group is carefully evaluated it is impossible to predict accurately the health effects of occupational exposure to carcinogens such as asbestos, radon gas, and arsenic. Unfortunately, in few of the studies listed in Tables 3a and 3b have adequate assessments been made of the proportion of workers who smoke cigarettes.

The contribution of cigarette smoking to the increased incidence of disease has been evaluated in several studies and has lead to a consensus that this habit produces a very significant increase in risk of dying of lung cancer as well as of the various cardiovascular diseases. The largest study of cigarette smokers is that of E. Cuyler Hammond and colleagues under the auspices of the American Cancer Society. This study is based on questionnaires and mortality follow-up accomplished in the United States between July 1960 and June 1971 for approximately 51,000 men (Hammond et al., 1978). The proportional mortality of lung cancer (percent lung cancer deaths relative to deaths by all causes), based on the Hammond study, is shown graphically in Figure 2. For a group of men who all smoke cigarettes (cohort of 100 percent smokers), lung cancer mortality is approximately 7 percent at age 45, reaches a maximum of approximately 10 percent at age 70, then decreases slightly at older ages. For a cohort of male non-smokers, lung cancer mortality is 2 percent at age 45 and then decreases continuously to approximately 1 percent at age 95.

Smoking is most prevalent in blue-collar occupations relative to professional and managerial occupations (Sterling and Weinkam, 1978). This prevalence also holds true for the asbestos trades, mining, and milling occupations. In a group of 13,722 asbestos insulation workers, whose smoking habits were recorded, 70 percent had a history of cigarette smoking (Selikoff and Hammond, 1975; Saracci, 1977). In a group of 1,015 chrysotile asbestos miners and millers 85 percent were smokers (McDonald et al., 1974). Data given in Figure 2 predicts that the lung cancer mortality for a cohort

Table 3a. cohort mortality studies of asbestos trades workers; excluding miners and millers (see table 4). Most recent update of prospective studies I-XI are given (1964-1979).

Study		I	II	III	IV	V	VI	VII	VIII	IX	X	XI
cause of death[1]	number of deaths	Elwood et al., 1964	Kleinfeld et al., 1967	Mancuso and El-Attar, 1967	Selikoff et al., 1973[4]	Elmes and Simpson, 1977	Weiss, 1977	Peto et al., 1977	Peto et al., 1977 Peto, 1978 [5]	Nicholson et al., 1978	McDonald & McDonald, 1978	Henderson & Enterline, 1979
All causes (000-999)	Obs.(Exp.)	46(57.9)	46	361	446(316.28)	122(54)	66(108.76)	210(191.31)	186(167.62)	274(188.19)	56	781(648.67)
	Obs./Exp.	0.96			1.41	2.26	0.61	1.10	1.11	1.46		1.20
Cancer-all sites (140-239) or (140-209)	Obs.(Exp.)	10(9.1)	23	96	172(56.34)	66	13(17.41)	59(51.32)	50(42.77)	99(39.93)	23	173(108.81)
	Obs./Exp.	1.10			3.05		0.75	1.15	1.17	2.48		1.59
	% all deaths	21.7	50.0	26.6	38.6	54.1	19.7	28.1	18.6	36.1		22.2
Cancer-lung, trachea, bronchus (162)	Obs.(Exp.)	6(3.02)	10(1.42)	41	79(17.47)	35	4(4.32)	31(19.30)	29(18.38)	35(12.53)	8	61(23.30)
	Obs./Exp.	1.99	7.0		4.52		0.93	1.61	1.58	2.79		2.62
	% all deaths	13.0	21.7	11.3	17.7	28.7	6.06	14.8	15.6	12.8	14.3	7.81
Mesothelioma-peritoneum, pleura (158,163)	Obs.(Exp.)[2]	1	3	9	23	13	0	5	4	26	9	2
	% all deaths	2.17	6.52	2.49	5.17	10.7	0	2.38	2.15	9.49	16.1	0.26
Cancer-digestive system (150-159)	Obs.(Exp.)			20								55(39.91)
	Obs./Exp.											1.38
	% all deaths			5.54								7.04
Cancer-G.I. tract (150-154)	Obs.(Exp.)	2	5(1.79)		24(10.57)	13	4(3.82)			15(7.99)	3	
	Obs./Exp.		2.79		2.27		1.04			1.88		
	% all deaths	4.35	10.9		5.38	10.7	6.06			5.47	5.36	
Respiratory disease (460-519)	Obs.(Exp.)					30		35(25.02)	31(23.22)	42(12.16)		68(39.31)
	Obs./Exp.							1.40	1.34	3.45		1.73
	% all deaths					24.6		16.7	16.7	15.3		8.71
Non-infectious respiratory disease (510-519)	Obs.(Exp.)	4									4	
	Obs./Exp.											
	% all deaths	8.70									7.14	
Asbestosis (515.2)	Obs.(Exp.)[3]		2	39	26	19	2			35	2	19
	% all deaths		4.35	10.8	5.83	15.6	3.03			12.8	3.57	2.43
Pulmonary tuberculosis (010-011)	Obs.			11		3					3	
	% all deaths			3.05		2.46					5.36	
Number in cohort		Not given	152	1493	7289	162	264	963	679	689	176	1075
Number of males (females)		No females	152(0)	1265(228)	7289(0)	162(0)	264(0)	679(284)	679(0)	not reported	93(83)	1075(0)
% dead		-	30.3	24.2	6.12	75.3	24.2	21.8	27.4	39.8	31.8	72.7
site		Asbestos works Cardiff, Wales	Insulators, New York State	Asbestos Co., U.S.A.	Insulators,SW MidWest, Central U.S.A	Insulators, Belfast, N. Ireland	Asbestos factory	Asbestos textile factory, UK	Asbestos textile factory, UK	Asbestos products factory, USA	Assemblers, gas mask with asbestos filter pads	Asbestos company, U.S.A.
Observation period		1936-1962	1945-1965	1940-1964	1967-1971	1940-1975	1945-1974	1933-1974	1933-1974	1959-1975	1945-1975	1941-1973
Years exposed		>0.5	>15	varies	not reported	varies	>1	>10	>10	varies	1-2	3-51
Years since first exposure		>15	>15	varies	not reported	varies	varies	>10	>10	varies	varies	varies
Controls		Death rates, SE Wales	U.S. National Rates, 1948	Internal, <2 yrs.	U.S. National death rates	Male rates, N. Ireland	U.S. National death rates	National death rates, UK	National death rates, UK	Not reported	Not reported	U.S. National Rates
Smoking data		Not reported	Minimal	Not reported	Not reported	5 non-smokers	Not reported	Not reported	Not reported	Not reported	Not reported	Not reported
Exposure data		Not reported	Not reported	Not reported	Not reported	Not reported	<2f./cm^3 since 1972	yes(1952-1974)	3 to 15 f./cm^3 (1938-72)	Not reported	Light exposure	yes
Asbestos type		Chrysotile, crocidolite	Not reported	Not reported	chrysotile, amosite	Not reported	chrysotile only	chrysotile, crocidolite	chrysotile, some crocidolite	Not reported	Mostly crocidolite, some chrysotile	chrysotile, amosite, crocidolite

1/International Classification of Diseases, 8th Revision (ICD.8)
2/Within the United States mesothelioma mortality unrelated to asbestos exposure accounted for approximately one death in 10,000 in 1972 (estimated from data given by McDonald and McDonald, 1980)
3/Asbestosis is rarely caused by mineral dusts other than commercial asbestos
4/Sub-cohort of cohort XVII (Table 3b)
5/Sub-cohort of cohort VII

Table 3b. Cohort mortality studies of asbestos trades workers; excluding miners and millers (see Table 4). Most recent update of prospective studies XII-XXI are given (1979-1981).

Study		XII	XIII	XIV	XV	XVI	XVII	XVIII	XIX	XX	XXI
cause of death[1]	number of deaths	Puntoni et al., 1979	Newhouse and Berry, 1979	Weill et al., 1979	Selikoff et al., 1979a[4]	Selikoff et al., 1979a[4]	Selikoff et al., 1979a	Selikoff et al., 1979b[4]	Selikoff et al., 1980b	Clemmensen and H-Jensen, 1981	Hilt et al., 1981
All causes (000-999)	Obs.(Exp.)	19(11.42)	745(556.0)	601(890.1)	478(328.9)	62(64.6)	2271(1658.9)	79(75.2)	304(158.6)	Not given	13
	Obs./Exp.	1.66	1.34	0.68	1.45	0.96	1.37	1.05	1.92		
Cancer-all sites (140-239) or (140-209)	Obs.(Exp.)	6(3.19)	325(138.41)	120(156.6)	210(57.0)	20(10.1)	995(319.7)	39(16.2)	116(33.4)	167(151.13)	7
	Obs./Exp.	1.88	2.35	0.77	3.68	1.98	3.11	2.41	3.47	1.11	
	% all deaths	31.6	43.6	20.0	43.9	32.3	43.8	49.4	38.2		53.8
Cancer-lung, trachea, bronchus (162)	Obs.(Exp.)	5(0.94)	130(46.41)	51(49.2)	93(13.3)	10(2.9)	486(105.6)	21(5.7)	60(10.1)	44(27.31)[5]	3(0.21)
	Obs./Exp.	5.32	2.80	1.04	6.99	3.45	4.60	3.68	5.94	1.61	14.29
	% all deaths	26.3	17.4	8.49	19.5	16.1	21.4	26.6	19.7		23.1
Mesothelioma peritoneum, pleura (158,163)	Obs.(Exp.)[2]	None reported	67	0	38	3	175	8	14	3	2
	% all deaths		8.99	0	7.95	4.84	7.71	10.1	4.61		15.4
Cancer-digestive system (150-159)	Obs.(Exp.)		60(44.2)	25(50.1)							
	Obs./Exp.		1.36	0.50							
	% all deaths		8.05	4.16							
Cancer-G.I. tract (150-154)	Obs.(Exp.)				43(15.1)	3(1.5)	99(59.4)	3(3.1)	16(8.0)	31(29.87)	1
	Obs./Exp.				2.85	2.00	1.67	0.97	2.00	1.04	
	% all deaths				9.00	4.84	4.36	3.80	5.26		7.69
Respiratory disease (460-519)	Obs.(Exp.)	10(1.24)	102(78.0)								
	Obs./Exp.	8.06	1.31								
	% all deaths	52.6	13.7								
Non-infectious respiratory disease (510-519)	Obs.(Exp.)				45(9.3)	7(1.1)	212(59.0)	14(3.2)	24(4.7)		
	Obs./Exp.				4.84	6.34	3.59	4.38	5.11		
	% all deaths				9.41	11.3	9.34	17.7	7.89		
Asbestosis (515.2)	Obs.(Exp.)[3]				41	6	168	13	18		
	% all deaths				8.58	9.68	7.40	16.4	5.92		
Number in cohort		41	5522	5645	632	833	17,800	440	582	5686	20
Number of males (females)		41(0)	4600(922)	5645(0)	632(0)	833(0)	17,800(0)	440(0)	582(0)	5686(0)	20(0)
% dead		46.3	13.5	10.6	75.6	7.44	12.8	18.0	52.2	-	65.0
site		Insulators, shipyards, Genoa, Italy	Asbestos textile factory, E. London	Asbestos cement company, New Orleans, LA	NY-NJ insulation workers	NY-NJ insulation workers	No. American asbestos workers	Insulators, US shipyards	Amosite textile factory, NJ	Asbestos cement factory, Denmark	Construction, 1928-1929, Norway
Observation period		1960-1975	1933-1975	to 1974	1943-1976 first employed <1943	1943-1976 first employed>1943	1967-1976	1967-1976	1961-1977	1944-1976	1943-1980
Years exposed		varies	>10	varies	varies	varies	varies	varies	varies	varies	1
Years since first exposure		varies	>10	>20	most >20	>20	varies	varies	>20	varies	varies
Controls		male rates, Genoa	not given	death rates, US, Louisiana, matched controls	US death rates	US death rates	US death rates	US death rates	Death rates, NJ, smoking adj.	Death rates, Danish provincial towns	Death rates, Norway
Smoking data		Not reported	yes	None	Not reported	Not reported	Not reported	Not reported	yes	yes	yes
Exposure data		Not reported	yes	<10 to >200 mppcf-yr.	Not reported	Not reported	Not reported	Not reported	heavy	yes	heavy
Asbestos type		Not reported	Crocidolite, amosite, chrysotile	Chrysotile, amosite, crocidolite	Not reported	Not reported	Not reported	Not reported	Amosite, some chrysotile	Chrysotile, amosite, some crocidolite	Crocidolite only

[1] International Classification of Diseases, 8th Revision (ICD.8)
[2] Within the United States mesothelioma mortality unrelated to asbestos exposure accounted for approximately one death in 10,000 in 1972 (estimated from data given by McDonald and McDonald, 1980)
[3] Asbestosis is rarely caused by mineral dusts other than commercial asbestos
[4] Sub-cohort of cohort XVII
[5] Excludes trachea

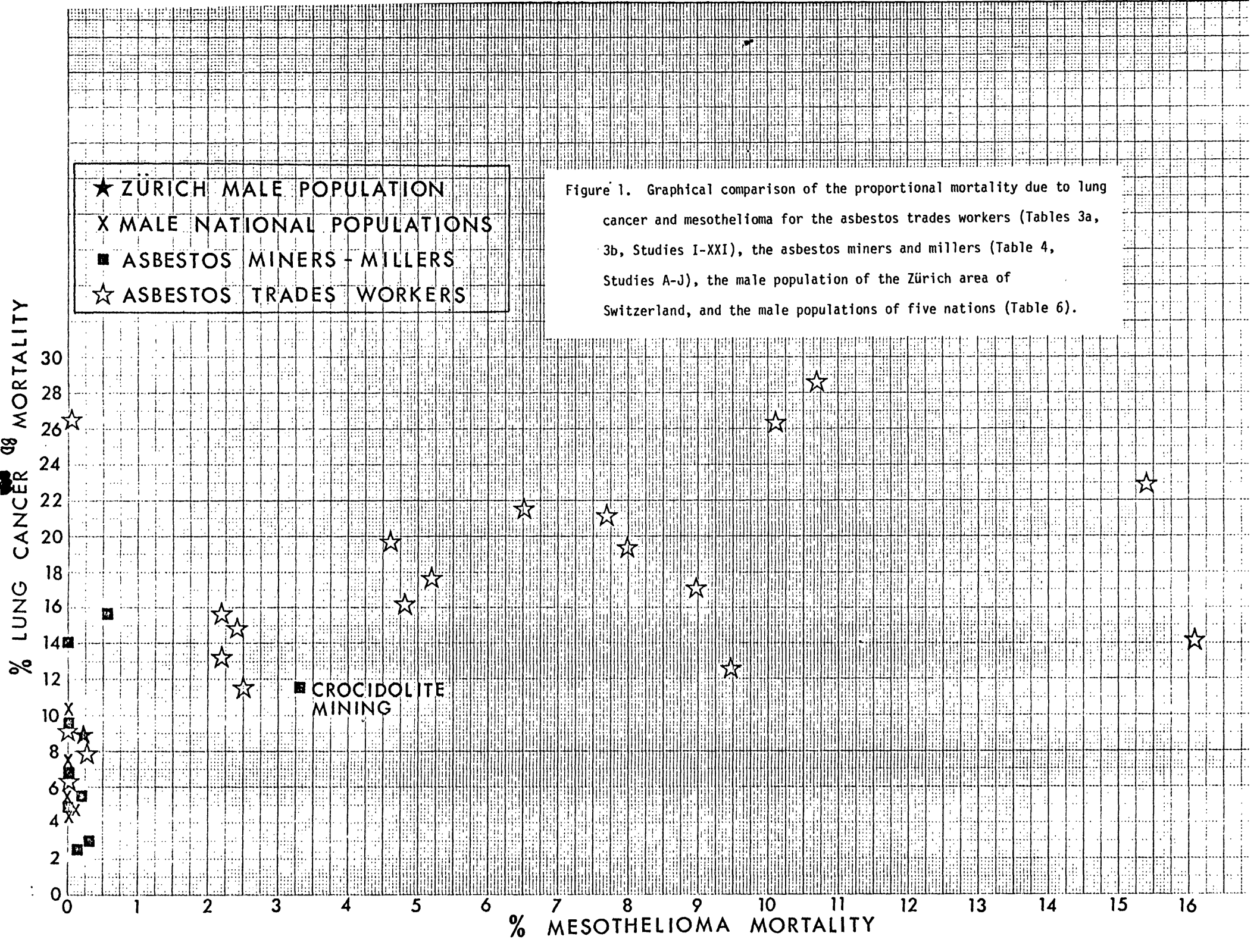

Figure 1. Graphical comparison of the proportional mortality due to lung cancer and mesothelioma for the asbestos trades workers (Tables 3a, 3b, Studies I-XXI), the asbestos miners and millers (Table 4, Studies A-J), the male population of the Zürich area of Switzerland, and the male populations of five nations (Table 6).

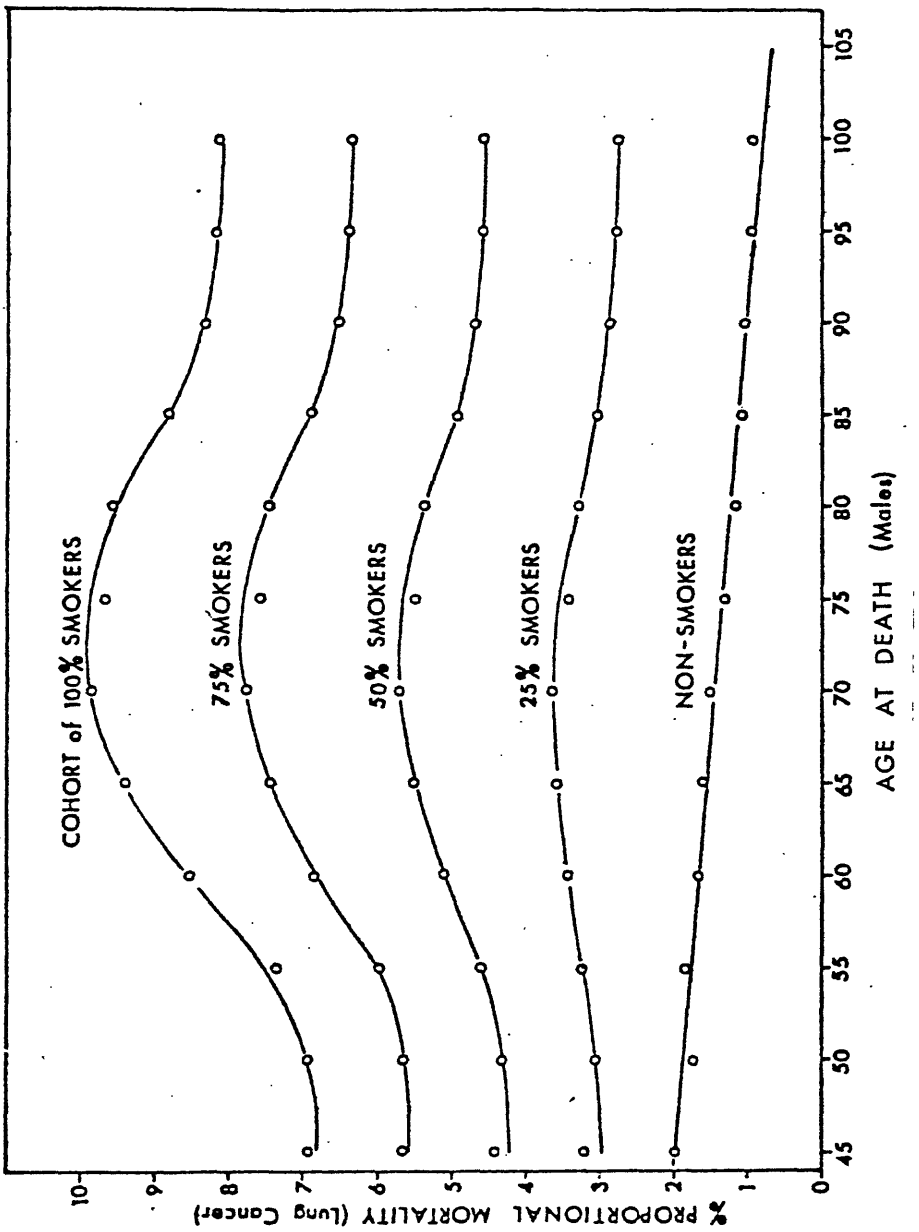

Figure 2. Percent of lung cancer deaths in males relative to deaths by all causes (proportional mortality - lung cancer) plotted with respect to age for four groups (cohorts) having different cigarette-smoking characteristics and for a cohort of non-smokers. For example, for a cohort of 70-year old males which is composed of 75 percent cigarette smokers, 7.8 percent of all deaths at age 70 are predicted to be from lung cancer. Graphical presentation based on data from Hammond et al. (1978).

composed of 75 percent smokers would be at least 6 to 7.5 percent, regardless of occupation. In Tables 3a and 3b we see that the lung cancer mortality for the total of 21 cohorts of asbestos "trades" workers was 16.7 percent - approximately three times that expected if mortality predictions were based only on the apparent smoking habits.

The risk of lung cancer due to asbestos exposure is lower in non-smokers than in smokers (Saracci, 1977, J. C. McDonald, 1980b). There appears to be no relationship between smoking habits and the incidence of mesothelioma, the disease is equally prevalent in smokers and non-smokers alike. Of the studies listed in Tables 3a and 3b, only Study No. VI of chrysotile factory workers shows a lung cancer mortality that would be expected from the smoking habits alone.

Asbestos miners and millers. Men working in the mining and milling of asbestos ore are generally exposed to only one form of fiber. A few exceptions occur in the mining regions of South Africa where some workers have been employed in crocidolite, amosite, and chrysotile mines. Anthophyllite and tremolite asbestos miners may have been exposed to some chrysotile asbestos, for these minerals can coexist in metamorphosed ultramafic rocks, for example, those of Paakkila, Finland.

Epidemiological studies of asbestos miners and millers who were exposed to only one form of asbestos are useful to understand how the different asbestos minerals affect human health. Table 4 gives the mortality data for the five major epidemiological studies of asbestos miners and millers. In addition three studies are given of miners exposed to cummingtonite and grunerite amphibole dusts and one study of tunnel workers exposed to hornblende amphibole dust. Some classify these amphiboles as asbestos even though they do not possess the physical properties requisite to be valuable commercially. Such a classification has been made in the case of taconite mining by the courts (United States District Court for Minnesota, 380 F. Supp. 11) and by the U.S. Environmental Protection Agency (Reserve Mining vs. EPA, U.S. Court of Appeals Eighth Circuit, March 14, 1975); the latter has sued to prevent the Reserve Mining Company from dumping taconite tailings into Lake Superior because of the perception that these tailings contain "amosite asbestos" and thus constitute a threat to public health. For a complete review of the case see 514 Federal Reporter, 2d Series, 492-542, 1975; 256 North Western Reporter, 2nd Series, 808-852, 1977. Of interest regarding this suit are the health studies of the Reserve iron ore miners exposed to cummingtonite and grunerite in the taconite rock (Table 4, Study B) and on the Homestake gold miners exposed to cummingtonite in

Table 4. Mortality from selected causes in the principal epidemiological studies of commercial asbestos miners and millers and other hard rock miners and tunnel workers exposed to rock dust containing minerals sometimes defined as asbestos[1/]

Cause of Death[2/]	Number of Deaths	Study A 900 men 1936-1967	B 5751 men 1952-1976	C 932 men 1955-1972	D 440 men 1960-1973	E 1321 men 1937-1973	F -- 1943-1977	G 933 men 1946-1975	H 544 men 1961-1977	J 10939 men 1910-1975	Totals (excluding Study F)
All causes (000-999)	Observed Expected Obs./Exp.	216	298 344 0.87	294 225 1.30	71 52.9 1.34	631 549.7 1.15	519 600.3 0.86	332 214.4 1.55	178 159.9 1.11	4463	6483
Respiratory cancer (162)	Obs. % all deaths Exp. Obs./Exp.	21 9.7 12.6 1.67	15 5.0 17.9 0.84	21 7.1 13.15 1.60	10 14.1[3/] 2.7 3.0	16 2.5 16.5 0.97	60 11.6 38.9 1.54	10 3.0 10.4 0.96	28 15.7 11.1 2.5	250 5.6	371 5.7
Mesothelioma, peritoneum, pleura (158,163)	Obs. % all deaths	0 0	0 0		0 0	1? 0.16?	17 3.3	1? 0.30?	1 0.56	10[5/] 0.22	11 + 2? 0.17+0.03?
Gastro-intestinal cancer (150-154) or (150-159)	Obs. % all deaths Exp. Obs./Exp.	7 3.2 14.9 0.47	20 6.7 17.6 1.14	10 3.4 11.13 0.90		39 6.2 35.1 1.11		19 5.7 19.3 0.98	10 5.6 9.5 1.05	168 3.8	273 4.2
Pneumoconiosis (500-519)	Obs. % all deaths		4[7/] 1.3	20 6.8	5 7.0	37 5.9	21[4/] 4.0	20 6.0	30 16.9	46[6/] 1.0	
Asbestosis (515.2)	Obs.	13						9	26		
Silicosis (515.0)	Obs.					35					
Respiratory tuberculosis (010-011)	Obs. % all deaths	36 16.7		11 3.7		39 6.2	4 0.77	18 5.4		248 5.6	
Locality		North Savo, Finland	Minnesota, U.S.A.	Manhatten I. N.Y., U.S.A.	Lead, ND, U.S.A.	Lead, ND, U.S.A.	Wittenoom, W. Australia	Balangero, Italy	Quebec, Canada	Quebec, Canada	
Type of Mining		asbestos	iron ore	tunneling	gold	gold	asbestos	asbestos	asbestos	asbestos	
Type of Rock		ultramafic	taconite	schist,gneiss, amphibolite	qtz-cumming-tonite schist	qtz-cumming-tonite schist	banded ironstone	serpentinite	serpentinite	serpentinite	
Suspected mineral pathogen		anthophyllite asbestos	cummingtonite, grunerite, quartz	hornblende, quartz	cummingtonite, hornblende, quartz	cummingtonite, hornblende, quartz	crocidolite asbestos, quartz	chrysotile asbestos	chrysotile asbestos	chrysotile asbestos	
Source		Meurman et. al., 1974	Higgins, 1981	Selikoff, 1978	Gillam et al. 1976	McDonald et. al., 1978	Hobbs et al., 1980	Rubino et al., 1979	Nicholson et al., 1979	McDonald et al., 1980	

1/ Cummingtonite, grunerite,and hornblende (Studies B, C, D, E) may be defined as "asbestos" in U.S. Federal Regulations
2/ International Classification of Diseases, 8th revision (ICD-8)
3/ Includes one carcinoma of the maxillary sinus and one mediastinal carcinoma (unspecified). See footnote 2/ in text.
4/ Pneumoconiosis Board Records (Western Australia) show pneumoconiosis of mixed type, asbestosis, silico-asbestosis, and silicosis.
5/ Two mesothelioma victims worked with crocidolite in addition to chrysotile.
6/ Pneumoconiosis is probably predominantly asbestosis since rock dust contains little crystalline silica (quartz, etc.)
7/ "Selected respiratory disease"

the gold-bearing schists (Table 4, Study D, E)[2/]. Studies B and E show no evidence of asbestos-related diseases appearing in the study groups.

Mortality comparisons, trades vs. mines. The cancer mortality pattern for those in the asbestos trades and mining occupations is graphically presented in Figure 1 where percent lung cancer mortality is plotted with respect to percent mortality due to mesothelioma. The studies of the asbestos trades workers (Tables 3a, 3b; Fig. 1, open stars) show a very significant excess of mortality due to mesothelioma relative to that found in the miners (Table 4, Fig. 1, solid squares) - with one exception, the crocidolite miners of Western Australia (Study F).

In regard to high mesothelioma mortality, it is important to note two health studies of specialized factory workers who, during World War II, were employed at the task of manufacturing asbestos-bearing filter pads and of placing them into gas mask canisters. One study (McDonald and McDonald, 1978) was of Canadian workers who, at three factories, were involved with the manufacture or handling of crocidolite-bearing filter pads. The maximum duration of exposure during the period 1939-1941 was no more than two and a half years. The cohort studied was composed of 93 men and 83 women (Study X, Table 3a). Of the 56 now dead, 8 died of lung cancer (14.3%) and 9 of mesothelioma (16.1%).

The second study (Jones et al., 1976, 1980a) was of a cohort of 951 women who worked either at a factory in Nottingham, England or at a factory in Birmingham, England. The Nottingham group worked for only five months in the years 1939-1940 assembling "civilian filter pads" that contained chrysotile asbestos. The Birmingham group worked for up to four and half years during the period 1940-1944 assembling "military filter pads" that contained crocidolite asbestos. The crocidolite was thought to have come from Wittenoom, Western Australia. The mortality data is given in Table 5. None of those exposed only to chrysotile died of lung cancer or mesothelioma whereas there were 11 lung cancer deaths and 16 mesothelioma deaths among those who worked only with crocidolite. None of the women in either factory, as far as is known, was exposed to asbestos occupationally apart from their wartime work. Dust levels, as with the Canadian gas mask workers, was considered to be very light to moderate.

[2/]Study E (McDonald et al., 1978) discredits Study D (Gillam et al., 1976). Study D was made on a sub-cohort of the Study E cohort. The latter study is much more complete; satistically it is based on 631 deaths whereas Study D is based on 71 deaths. In addition, Study D presents an implausible mortality pattern: (1) No deaths due to silicosis, tuberculosis, or silico-tuberculosis were reported despite the fact that the mining company has been coping with a quartz dust problem for a century and (2) a 14·1 percent respiratory cancer mortality (incorrectly included as respiratory cancer were a sinus and a mediastinal carcinoma) was attributed to "cummingtonite asbestos" yet no mortality due to asbestosis was reported. The cummingtonite found in the quartz-cummingtonite schist host rock is not asbestos; but rather, garden-variety rock-forming amphibole.

Table 5. Mortality Data (Jones et al., 1980a) for a Cohort of 951 Women* (578 traced) who assembled Asbestos-Bearing Filter Pads in English Factories (1939-1944).

Cause of Death	Observed	% Mortality	Asbestos Exposure
All causes	166	100	Crocidolite and chrysotile
G.I. cancer	10	6.0	---
Other cancer	35	21.1	---
Mesothelioma	16	10.2	Crocidolite only
Mesothelioma	1		Crocidolite and chrysotile
Lung cancer	11	7.2	Crocidolite only
Lung cancer	1		Crocidolite and chrysotile

*727 (139 dead) were exposed to only crocidolite, 102 (10 dead) to only chrysotile, 99 (15 dead) to crocidolite plus chrysotile, and 23 (2 dead) unknown exposure.

To make further comparisons it is useful to examine the mortality with respect to lung cancer and mesothelioma in national populations. In Table 6

Table 6. Cancer mortality in men over 24 years of age for 5 nations (McDonald and McDonald, 1977)

Nation	All Deaths (year)	Lung cancer No.(%)*	Mesothelioma No.(%)*
England-Wales	278,617 (1970)	24913 (8.9)	154 (0.06)
Finland	22,332 (1970)	1586 (7.1)	8 (0.04)
Italy	272,795 (1970)	11867 (4.7)	not reported
U.S.A.	988,620 (1969)	50481 (5.1)	250 (0.03)
Canada	82,052 (1970)	4312 (5.3)	25 (0.03)
Totals	1,624,416	93159 (5.7)	437 (0.03)

*percent of all deaths (proportional mortality)

are given the lung cancer and mesothelioma mortality of all males over 24 years of age in five nations. These data are plotted in Figure 1. We find that the average lung cancer mortality of these five national populations is 5.7 percent, a figure identical to the 5.7 percent average mortality of the miners and tunnel workers (Table 4, excluding crocidolite miners). The mesothelioma mortality of the five national populations is 0.03 percent (Table 6) and is probably significantly underreported because of: (1) the great difficulty in diagnosing this disease, even after autopsy (McDonald and McDonald, 1977, 1980; Vejlsted and Hansen, 1980; Kannerstein and Churg, 1980; Legha and Muggia, 1977) and (2) complications arising in properly and consistently coding this disease for later information retrieval.

It may be more meaningful to compare mesothelioma mortality among asbestos workers and miners, in whom this disease is anticipated, with the mortality in a population where the determinations of the causes of death are based on a large number of autopsies and where asbestos exposure is minimal. A review

has been made by Rüttner (1978) of the deaths in the Zürich area of Switzerland where there are no asbestos mines, mills, or industries and where the cause of death is often determined by autopsy. Among the 28,110 male deaths (all autopsied) from 1961 to 1976, 51 deaths were due to mesothelioma (0.18 percent) and 2466 were due to lung cancer (8.8 percent). Among women (22,583 deaths) 23 were caused by mesothelioma (0.10 percent) and 368 caused by lung cancer (1.6 percent). The proportional mesothelioma mortality for hard-rock miners, tunnel workers, and asbestos miners (other than crocidolite) is 0.17 to 0.20 percent (Table 4). The asbestos trades workers, by contrast, have an average mesothelioma mortality of 5.6 percent (Table 3).

Among the asbestos miners and millers there is no question that those exposed to heavy concentrations of chrysotile and anthophyllite dust over long periods of time have suffered a significant excess mortality due to lung cancer and asbestosis - but not to mesothelioma (Studies A, H, Table 4). The most detailed health study of asbestos miners to date is that of the chrysotile asbestos miners of Quebec (Table 4, Study J). Here, McDonald et al. (1980) have carefully documented the relationship between lung cancer incidence and cumulative dust exposure. The average dust concentrations that the miners and millers experienced during the working day were divided into four categories depending on the work tasks performed during their careers in the mines. These are: low level 2.5 to 4.2 mpcf[3/], medium level 4.3 to 9.4, high level 14.4 to 23.6, and very high level 46.8 to 82.6 mpcf. The mean within these four categories in terms of chrysotile fibers per cm^3 is: low 10 fibers/cm^3, medium 21 fibers/cm^3, high 95 fibers/cm^3, and very high 194 fibers/cm^3. For the men exposed for over 20 years (see Table 17, column A) in the low and medium dust categories (averaging 6.6 mpcf or approximately 20 fibers/cm^3) total mortality was less than expected (SMR = 0.94). For these men there was a slight risk of excess lung cancer (SMR = 1.15) and respiratory tuberculosis (SMR = 1.14). As exposures of 20 fibers/cm^3 are an order of magnitude higher than that experienced now (dust levels for the past few years have been maintained at less than 2 fibers/cm^3), miners working a lifetime under the present dust levels are not expected to present any significant health problems relative to those in other mining industries (Liddell, 1981).

McDonald et al. (1980) have also studied the health statistics of a cohort of 440 women who also worked in the Quebec chrysotile asbestos mines and mills. Of the 84 who have died there was one death due to lung cancer and one due to mesothelioma.

3/mpcf = millions of particles of rock dust per cubic foot. Conversions of this figure into asbestos fibers per cubic centimeter, the usual measurement for industrial hygiene monitoring, is difficult but an approximate and conservatively small figure is: 1 mpcf = 3 fibers/cm^3 (McDonald et al., 1980, p. 21, 23; see also McDonald and Becklake 1976).

Crocidolite exposure. There are persuasive data, many already surveyed, which show that crocidolite asbestos is much more hazardous than chrysotile, anthophyllite, and amosite. Of the mining populations of the world, only those in the crocidolite mining areas of the Cape Province of South Africa and at Wittenoon Gorge, Western Australia have a statistically significant increase in mortality due to mesothelioma. Also, mesothelioma deaths have been reported among the residents of these areas who are not employed in the mines or mills. For example, Webster (1978) reports that the South African Asbestos Tumour Reference Panel placed 712 cases of mesothelioma on the register, which included all the known cases since 1956. Of these occupational and environmental background was established for 420 cases. Actual mining exposure accounted for 139 of the 420 cases of which 120 were in connection with Cape crocidolite mining and two with Transvaal crocidolite mining. There were only four mesothelioma cases in those associated with amosite mining and two of these had been exposed to Cape crocidolite as well. In the chrysotile mining industry there was only one case - a miner from Rhodesia. Of the 100 environmental cases (those not employed in any occupation where asbestos is used) 93 had been exposed to Cape crocidolite, two to Transvaal crocidolite, and one possibly to amosite.

Additional prevalence studies in the Cape Province (Talent et al., 1980) discovered 65 active cases of mesothelioma in people who had presented themselves for medical examination. Fifteen of these cases appeared in two groups, numbering 755 and 947 individuals, who were once employed in the crocidolite mines. An additional thirty eight mesothelioma cases appeared in a survey of certain patients at the St. Michael's Hospital in Kuruman, Cape Province, who were not responding to treatment for suspected pulmonary tuberculosis. Fourteen of these mesothelioma patients were known to have worked in the crocidolite asbestos mines. Lastly, 12 of the 65 cases appeared in a medical survey of 53 females who, in the past, hand-cobbed crocidolite asbestos.

In contrast to the prevalence of mesothelioma in the Cape Province, this disease is very rare in the Transvaal where all of the world's amosite is mined. Wagner et al. (1960), in regard to their initial discovery of the association of crocidolite asbestos with mesothelioma, state (p.260) "the tumour (referring to mesothelioma) is rarely encountered elsewhere in South Africa. During the past five years, with the exception of the present series (in Cape Province), no neoplasm of this nature has been diagnosed amongst 10,000 lungs examined at the Pneumoconiosis Bureau in Johannesburg, or in the Pathology Department of the South African Institute for Medical Research."

The incidence of mesothelioma in Zimbabwe (Rhodesia), a country which is a major producer of chrysotile but mines no other form of asbestos, is very low. In a communication to Mostert and Meintjes (1979), the Secretary of the Rhodesia Pneumoconiosis Board stated that no cases of mesothelioma were reported in the mining industry. It is of interest to note that two cases of mesothelioma were reported in the Rhodesian railway industry, a locomotive engineer and a storeman. The locomotives were insulated with crocidolite asbestos to which these two men were exposed (Mostert and Meintjes, 1979). Cochrane and Webster (1978) report 12 cases of mesothelioma in men employed as insulators in the locomotive workshops of the South African Railways.

The prevalence of mesothelioma among the miners of Wittenoon Gorge has been discussed (Table 4, Study F). The town of Wittenoon, the center of crocidolite mining in Western Australia, reached a peak population of about 1,000 in the 1960's. At present the population is down to about 200 and the West Australian State Government has suggested the closing of the town and evacuation of the residents because of continuing risks of airborne asbestos dust (Chemical Week, December 8 , 1978, p. 25). The risk of mesothelioma among the residents of the town who were not employed by mines is demonstrated by the case of a 27-year-old woman who had an environmental childhood exposure to crocidolite (Langlois et al., 1978).

Effects of Non-occupational Exposure to Asbestos

It is difficult to assess the health effects of non-occupational exposure to asbestos, for cohorts are hard to define, exposure levels are usually low, and any excess of lung cancer is disguised by the strong association of this disease with cigarette smoking. To study the non-occupationally exposed, epidemiologists are thus constrained to look for increased incidence of asbestosis and particularly mesothelioma in two types of cohorts; those who live in neighborhoods surrounding asbestos factories, mills, or mines and those who live within the household of an asbestos worker who presumably carried asbestos dust back to the home on his or her clothing.

Background incidence of mesothelioma. There appears to be a definite "background" mortality due to mesothelioma that is not related to asbestos exposure. McDonald and McDonald (1977), reviewed 4539 fatal mesothelioma cases reported from 22 countries between 1959 and 1976. They found that for 923 of the 2453 cases where a history had been recorded a definite or probable exposure to asbestos could not be shown. More recently, Jones and Silver (1979) report eight cases with no environmental exposure, Brenner et al. (1981a) report of 25 patients entering Memorial Hospital, N.Y. since 1950 with no exposure history, Brenner et al. (1981b) report of seven children dying of mesothelioma who had no exposure history to asbestos, and Griffiths et al. (1980) report on 10 mesothelioma patients with no history of exposure to asbestos who entered Austin Hospital, Melbourne, Australia.

A possible genetic basis for some mesothelioma incidence is suggested by Risberg et al. (1980) in their report of five deaths due to mesothelioma within a single family; the father, 3 sons, and a daughter. Four of the five had worked in the building industry where random exposure to asbestos containing products could have occurred. They lived in a town of 100,000 inhabitants which had no asbestos industry in the vicinity.

An estimate of mesothelioma mortality not related to exposure to asbestos can be obtained from the data given by McDonald and McDonald (1980). Ascertainment, through 7,400 pathologists, of all fatal malignant mesothelioma tumors in Canada (1960-1975) and in the U.S.A. (1972) was made. The pathology review panel accepted 73 and 65 percent of the U.S. and Canadian cases, respectively. Occupational histories indicate that 50 percent of the male and five percent of the female deaths could be attributed

to asbestos exposure. Thus, in the United States in 1972, there were 245 cases of mesothelioma reported (189 male, 56 female) and of these, 140 male and 39 female cases are accepted. Assuming a 30 percent underreporting (McDonald and McDonald, 1980, p. 1655), the 1972 incidence in the U.S. is approximately 200 male and 56 female deaths. Of these, about 100 male and 3 female mesothelioma deaths can be attributed to asbestos exposure; the remaining 153 deaths to other causes or background. If these figures are reasonably correct, then the mesothelioma proportional mortality due to "background" in 1972 was 0.008 percent (153 deaths out of 1,963,944). The mesothelioma death rate due to background in 1972 is calculated to be 0.7 deaths per million U.S. population. The study of McDonald and McDonald (1980, see also McDonald, 1979) indicate that the Canadian mesothelioma death rates are very similar to those in the U.S.

Asbestos-related disease in residential areas. The residents of areas where there are asbestos factories, mines or mills may contract asbestos-related diseases even though they are not actually employed in the asbestos industry. The high prevalence of mesothelioma among residents of the crocidolite mining areas of the Cape Province of South Africa has already been discussed. The appearance of 93 mesothelioma deaths among those non-occupationally exposed to Cape crocidolite is in extreme contrast to the rarity of this disease in the amosite mining regions of the Transvaal Province where only one possible case has been reported (Webster, 1978).

Asbestos-related disease among residents of chrysotile mining areas is rare. Theriault and Grand-Bois (1978) report the following mesothelioma mortality in Quebec Province: (1) asbestos-producing regions (observed 2, expected 1.3), (2) areas surrounding the asbestos producing regions (obs. 5, exp. 4.8), (3) other rural regions of Quebec (obs. 12, exp. 32.4), (4) city of Quebec (obs. 7, exp. 4.7), and (5) city of Montreal (obs. 42, exp. 24.4).

McDonald (1980a) has reviewed all known fatal mesothelioma cases in Quebec Province for the period 1960-1978. Of the total of 254 cases registered, 181 were males, 73 females. Occupational and residential histories were obtained from 91% of the men and 86% of the women. About 40% of the male cases and 5.4% of the female cases were attributed to occupational exposure to some form of asbestos. Twenty one cases were individuals who at some time had been employed in the chrysotile mining and milling industry. But 5 of these 21 had been exposed to crocidolite while manufacturing filter pads for gas masks and 2 more possibly exposed to crocidolite in one of the mills, which for about two years processed crocidolite fiber for the gas mask filters. In addition to these 21 cases there were four daughters and two sons of chrysotile miners or millers who died of mesothelioma. Apart from these cases McDonald (1980a) reports of only two persons who died of this disease who lived within 20 miles of the chrysotile mines and mills of Quebec.

Pampalon (1980) reported on the mortality patterns of the Quebec asbestos mining towns of Thetford Mines (population approximately 20,000) and Asbestos (population approximately 10,000). Cancer mortality among the female residents of these towns is particularly informative since very few were employed in the asbestos industry. However, they did receive over

much of their lifetime very heavy non-occupational exposures to chrysotile asbestos contained in the rock dusts emitted from the operation of the nearby mines and mills.[4/] The mortality data for the female residents of Asbestos and Thetford Mines is given in Table 7 (see also Tables 8 and 9).

Table 7. Mortality Data for Women Living in Quebec Asbestos Mining Towns for the Period 1966 to 1977 (Pampalon, 1979).

Cause of death	Observed	Expected	Excess (Deficiency)
all causes	1225	1356	(131)
all cancer	292	321	(29)
lung cancer	23	23	0
G.I. cancer	97	91	6
resp. disease	35	58	(23)

Another cancer mortality survey of various regions in Quebec was made by Graham (1981). His data, summarized in Table 8, show the rates for various cancers that may be associated with exposure to asbestos dusts for five regions; asbestos mining counties, counties surrounding the asbestos counties, other rural counties, and the cities of Quebec and Montreal. The rates for the asbestos mining counties, which contain the large semi-industrialized towns of Thetford Mines and Asbestos, are intermediate between the rural counties ("other rural" and "peripheral") and the cities of Quebec and Montreal. This is to be expected for cancer rates are highly correlated with the interrelated factors, (1) degree of urbanization, (2) socioeconomic class, and (3) "lifestyle" (Doll and Peto, 1981; Goldsmith, 1980; Higginson and Muir, 1979; Higginson, 1980; Weisberger, 1978; Gori, 1979; Wynder, 1980). Graham (1981) states that the rates for cancers of

4/ Even in 1974 and 1975, when rock dust levels at the Quebec asbestos mines and mills had been much reduced from levels as high as 200 million particles per cubic foot (ppcf) in the early 1950's to less than 7 million ppcf in the mid-1970's (McDonald and Becklake, 1976), the rock dust in the ambient air over the town of Thetford mines averaged 80,000 ng/m^3 when the mines and mills were in operation and 40,000 ng/m^3 when the mining operations were shut down during the strike of April to September, 1975 (Brulotte, 1976). When the mines were operating, the average weight of dust falling daily over the town was 377 kg/km^2. Recent measurements by Gibbs, Rowlands, and Brulotte (Air Pollution Control Assoc., 1980) of dust in the ambient air of the towns of Thetford Mines and Black Lake show a chrysotile asbestos content of 160 to 11,000 ng/m^3 - a considerable environmental exposure even in these better times. In this regard, it is pertinent to cite the work of Nicholson et al. (1980) who analyzed the chrysotile fiber content in the air in houses located in two chrysotile mining districts (Copperopolis, California and Baie Verte, Newfoundland). The 13 air samples taken in homes of crysotile miners and millers show the following chrysotile content: 4 (50-100 ng/m^3), 4 (100-200 ng/m^3), 2 (200-500 ng/m^3), 2 (500-1000 ng/m^3), and 1 (2000-5000 ng/m^3). Three samples taken in houses of non-miners in Baie Verte gave concentrations of 32, 45, and 65 ng/m^3.

Table 8. Cancer mortality rates (deaths per 100,000 residents) for various geographic localities in Quebec Province and for Upper New York State. Annual average for the period 1969-1973 (Graham, 1981).

Region	Asbestos-mining counties	Peripheral rural counties	Other rural counties	Montreal	Quebec City	All Quebec Province	Upper NY State
			MALES				
Lung	33.59	23.71	27.29	48.67	50.53	43.70	59.02
Pleura	1.02	0.28	0.13	0.51	0.20	Not given	Not given
Peritoneum	0.64	0.37	0.32	0.80	0.60	1.48	1.30
Stomach	16.00	12.54	12.61	12.38	18.01	17.74	11.99
Esophagus	2.42	1.38	1.94	3.93	3.56	3.17	4.71
Small Int.	1.11	0.58	0.32	0.68	0.95	0.64	0.78
Large Int.	15.05	13.43	10.92	18.69	20.79	18.11	27.11
Rectum	7.48	10.58	9.99	12.84	14.30	13.55	16.28
	77.31	62.87	63.52	98.5	108.94	98.39	120.41
			FEMALES				
Lung	4.39	4.64	3.87	8.70	6.96		
Pleura	0.35	0.20	0.10	0.18	0.18		
Peritoneum	0	0.78	0.53	0.75	1.51		
Stomach	9.34	7.14	6.40	7.51	8.98		
Esophagus	1.04	0.40	0.76	1.23	1.26		
Small Int.	0.38	0.68	0.32	0.65	1.16		
Large Int.	18.75	17.74	15.36	22.64	21.89		
Rectum	10.86	8.78	8.24	9.51	11.26		
	45.11	40.36	35.58	51.17	53.2		

1/Only those cancers known to be associated with excess mortality in asbestos trades workers are listed.

the pleura[5/], peritoneum[5/], lip, tongue, salivery gland and small intestine[5/] in males and of the pleura[5/], lip[5/], kidney, salivery gland and melanoma in females, are in excess[5/] in the asbestos mining counties. Graham (1981, p. 40) further states that cancer of the colon, stomach, and lung were "at a level so low as to be unimpressive." These are cancers known to be excess in many cohorts of asbestos trades workers (Table 3a, 3b). The low cancer rates found in the asbestos mining localities are not surprising for McDonald et al. (1980, p. 12) report that during the five decades, 1926-1975, 4350 male Quebec asbestos miners and millers died compared to 4107 expected on the basis of Quebec age-and-year specific death rates, giving a standardized mortality ratio (SMR) of 1.06.

It should also be noted that the residents of Thetford Mines and Asbestos, Quebec used drinking water that contained very high concentrations of chrysotile asbestos, ranging from 172 million to 1.3 billion fibers per liter (Wigle, 1977). No evidence of excess cancer mortality could be attributed to asbestos in the drinking water of these towns (Wigle, 1977).[6/]

Toft et al. (1981) expanded on the study of Wigle (1977) by comparing mortality data from 71 municipalities to the amount of asbestos in the drinking water. Particularly informative are the death rates of the female residents of Thetford Mines relative to the rates found in women who lived in 52 other localities that had very little asbestos in the drinking water. Most of the women of Thetford Mines did not work in the asbestos industry, but they did receive heavy non-occupational exposure to chrysotile asbestos carried in the air and water. Death-rate comparisons (Table 9) indicate that the women of Thetford Mines have not been effected by exposure to chrysotile asbestos.

[5/]A total of 1 to 3 deaths for these cancers were reported for the five year period 1969-1973. The statistical significance of such small numbers is questioned.

[6/]In regard to ingestion of asbestos and cancer incidence, a number of animal studies are now complete (Donham et al., 1980); Hallenbeck et al., 1981; Hilding et al., 1981; Smith et al., 1980; DHHS, 1981a; DHHS, 1981b). None of these studies show any evidence that ingestion of asbestos causes tumors in animals. Hallenbeck et al. (1981, p. 349) state, "the results of this study (a baboon gavaged with commercial asbestos) indicate that asbestos fibers do not penetrate the gastrointestinal tract of the baboon and migrate to various tissue."

Table 9. Age-adjusted Mortality Rates (per 100,000 Residents) for Females (age 25-69), Toft et al. (1981).

Cause of Death	Thetford Mines[1]	52 Comparison Localities[2]
all causes	420	433
all cancer	138	158
lung cancer	8.5	13.4
G.I. tract cancer	42.2	41.5
respiratory system[3]	8.9	16.9

[1] Treated water, 110-150 million chrysotile fibers per liter.
[2] All localities contained less than 5 million fibers per liter of water.
[3] non-neoplastic

Hammond et al. (1979) have given mortality statistics for the residents in the neighborhood of Riverside which surrounds the Patterson, New Jersey amosite asbestos factory. The mortality experience of the workers in this factory is given in Table 3b, Study XIX. The mortality data of Riverside is statistically indistinguishable from that of the control community of Totowa, N.J., situated several miles from the amosite factory (Table 10). One mesothelioma death occurred in Riverside in 1966 but none since.

Table 10. Cancer Mortality Data for Riverside and Totowa, New Jersey for the Period 1962-1976 (Hammond et al., 1979)

Cause of Death	Riverside[1]		Totowa[2]	
	No.	% Mortality	No.	% Mortality
all causes	780	100.00	1735	100.00
all cancer	163	20.90	353	20.35
lung cancer	41	5.26	98	5.65
colon-rectal cancer	24	3.07	74	4.27
stomach cancer	9	1.15	22	1.27
esophageal cancer	4	0.51	12	0.69

[1] Neighborhood near amosite asbestos factory
[2] Control neighborhood

Asbestos-related diseases in households of asbestos workers. There are a number of reports of mesothelioma occurring in individuals who, though not occupationally exposed to asbestos, lived in households which included an asbestos worker. Epler et al. (1980, p. 236) summarizes 14 published reports of 43 such mesothelioma cases plus four more found in their own study. Antman et al., (1980) mention 3 more and Vianna and Polen (1978) report on 10 female patients who lived with husbands or fathers employed in asbestos-utilizing occupations. It is difficult, perhaps impossible, to define retrospectively what kinds of asbestos was causing the mesothelioma

in these household contacts, but it is most probable that the workers handled more than one kind of asbestos. The report of Newhouse (1981, see also Newhouse and Thompson, 1965) may enlighten us on this subject. She reports that 11 individuals who died of mesothelioma and had neither worked with asbestos nor had a relative who worked with asbestos all had lived in the immediate vicinity of a factory that was a heavy user of crocidolite asbestos.

ESTIMATES OF MORTALITY IN THE UNITED STATES DUE TO EXPOSURE TO ASBESTOS

Previous Estimates

On September 11, 1978, Joseph A. Califano, then Secretary of the U. S. Department of Health, Education and Welfare, gave a major speech at the AFL-CIO National Conference on Occupational Safety and Health in which he described how the Federal Government was assisting in discovering and preventing occupational disease. One of his statements was that 17 percent of all cancer deaths in the United States each year for the next 30 to 35 years will be associated with previous exposure to asbestos. This translated into 67,000 cancer deaths per year due to asbestos (NCI, NIEHS Press Release, Draft Summary, Sept. 11, 1978). The Califano speech was based upon an unpublished document (Bridbord et al., 1978) prepared by several medical scientists at three of the National Institutes of Health (NCI, NIOSH, NIEHS). Doll and Peto (1981) have reviewed this document and state (p. 1240) "However, these estimates of total risk were so grossly in error that no arguments based even loosely on them should be taken seriously;" see also, Doll and Peto (1981, p. 1241, 1305-1308).

On January 15, 1980, Dr. Irving Selikoff of the Mt. Sinai School of Medicine, New York City, stated at a press conference in conjunction with the annual AMA meeting in Chicago, that 20,000 U.S. asbestos workers will die each year for the next 40 years of "excess disease" (see J. Am. Med. Assoc., vol. 243, p. 211, Jan. 18, 1980). On September 27, 1981, through a press release to Robert Locke (Associated Press Wire Service), Dr. Selikoff stated that 10,000 American workers are dying each year because of asbestos exposure. Dr. Selikoff does not state upon what data he bases these two estimates.

Hogan and Hoel (1981) estimate (p. 74) that future excess cancer deaths among U.S. workers exposed to asbestos could constitute as much as 3.0 percent (range 1.4-4.4%) of an estimated annual cancer death toll of 400,000 persons - or 12,000 asbestos-related cancer deaths per year. These authors based their estimates on an analysis of the number of people possibly exposed to asbestos, possible exposure levels, and estimated exposure-associated cancer risk.

In the above paragraphs, future mortality projections of 67,000, 20,000, 10,000 and 12,000 deaths per year due to asbestos exposure are quoted. Are any of these predictions correct? In the following, a method is proposed of estimating past asbestos-related mortality which can be used to predict future mortality.

Past Mortality Based on Asbestosis Incidence

The number of deaths due to asbestosis reported in the Vital Statistics of the United States (vol. II - Mortality, U.S. Dept. of Health and Human Services, National Center for Health Statistics) for the whole nation for the 11 year period 1967-1977 is: 1967(36 deaths), 1968(29), 1969(34), 1970(26), 1971(33), 1972(58), 1973(42), 1974(35), 1975(45), 1976 (54), and 1977 (55). The average number of asbestosis deaths per year for the period 1967-1977 is 41 with a high of 58 in 1972 and a low of 26 in 1970.

If it is known how many die from asbestosis each year then the total yearly asbestos-related deaths can be estimated from major epidemiological studies of asbestos workers. One of the largest such studies is of the 17,800 North American insulation workers (Selikoff et al., 1979a). This group, of which 2271 are now dead is one of the most severely effected by asbestos dusts. The mortality data and estimates of "excess" death due to asbestos is given in Table 11 (see also Table 3b, Study XVII).

Table 11. Mortality Among 17,800 Insulation Workers (Selikoff et al., 1979a)

Cause of death	Expected	Observed(BE)*	Excess
all causes	1659	2271	612
all cancer	320	995	675
mesothelioma	-	175	175
lung cancer	106	486	380
G.I. cancer	59	99	40
other cancer	155	235	80
Asbestosis	-	168	168
Non-infectious Respiratory disease	59	212	153

*BE-Best Estimate

If the asbestosis deaths given in the Vital Statistics are underreported by the same amount as observed by Selikoff et al. (1979, p. 103, Best Estimate (168)/Death Certificate (78) = 2.15), the average number of asbestosis deaths each year during the period 1967 to 1977 is 41x2.15 or 88. Using disease ratios taken from Table 11 (asbestosis/excess cancer, asbestosis/excess respiratory disease), the average annual asbestos-related mortality in the United States for the period 1967 to 1977 is estimated to be 522 deaths, including 354 cancer deaths (Table 12).

Table 12. Average Yearly Mortality due to Asbestos (1967-1977) Based on U.S. Recorded Asbestosis Deaths

Mesothelioma	92
Lung cancer	199
G.I. cancer	21
Other cancer	42
Asbestosis	88
Non-infectious resp. disease	80
Total asbestos-related deaths per year	522

Past Mortality Based on the 1972 Mesothelioma Incidence

It is not possible to obtain accurate mortality data for mesothelioma from the Vital Statistics of the United States for this disease is coded (ICD.8) with a number of other neoplasms under the headings: malignant neoplasms of peritoneum and retroperitoneal tissue (158.0, 158.9) and malignant neoplasms of other and unspecified respiratory organs; pleura (163.0), mediastinum (163.1), and site unspecified (163.9). Also, as pointed out previously, mesothelioma is a difficult disease to diagnose. These difficulties in coding and diagnosis make it necessary that national mortality estimates be made on decisions of mesothelioma review panels such as those described by McDonald (1979), Kannerstein et al. (1979), McDonald and McDonald (1980), and Jones et al. (1980b).

The mesothelioma study of McDonald and McDonald (1980) can be used to estimate the excess asbestos-related mortality. Their data, as described above, indicates that approximately 103 mesothelioma deaths in the United States in 1972 can be attributed to exposure to asbestos. Again, using the mortality data of Selikoff et al. (1979a) listed in Table 11 to calculate disease ratios (mesothelioma/excess other cancers, mesothelioma/asbestosis, mesothelioma/excess respiratory disease), the number of asbestos-related deaths in the United States in 1972 is estimated to be 587 of which 398 are due to cancer (Table 13).

Table 13. Mortality due to Asbestos in 1972 Based on Estimated Mesothelioma Deaths of McDonald and McDonald, 1980

Mesothelioma	103
Lung cancer	224
G.I. cancer	24
Other cancer	47
Asbestosis	99
Non-infectious resp. disease	90
Total asbestos-related deaths in 1972	587

In order to obtain a sense of perspective, it is useful to compare the death estimates obtained above for asbestos-related disease (522 and 587 deaths per year) to mortality from other types of industrial dusts, such as those of silica and coal. The Vital Statistics of the U.S. report that there were 215 silicosis deaths (ICD.8, 515.0) and 92 silicotuberculosis deaths (ICD.8, 010) in 1976. If it is assumed that mortality due to silicosis and asbestosis are equally underreported by a factor of 2.15 (Selikoff et al., 1979a, p. 103) and that the ratio of silicosis to excess non-infectious respiratory disease mortality is the same as the ratio of asbestosis to excess non-infectious respiratory disease, then the silica-related mortality for the United States in 1976 is estimated to total 975 deaths (Table 14).

Table 14. Estimated Mortality in 1976 due to Silica Dust

Silicosis	462
Silicotuberculosis	92
Non-infectious resp. disease	421
Total silica-related deaths in 1976	975

In 1976, there were 879 reported deaths due to anthracosilicosis (ICD.8, 515.1, Vital Statistics of the U.S.). Underreporting and other excess respiratory disease may have also occurred in coal miners who have suffered the "black lung" diseases.

These estimates of past asbestos-related mortality indicate that there will be nowhere near 10,000 deaths per year in the future. Asbestos-related mortality should peak between 1980 and 1985, 35 to 40 years after the large World War II shipyard employment. Linear regression analysis of past mortality (Vital Statistics of the U.S.) due to asbestosis (515.2, males and females, 1967-1977) and "pleural neoplasms" (163.0, males[7], 1968-1977) suggests that there has been an increase in the number of deaths with time. Calculated mortality for various years is given in Table 15. The variance (r^2) of the regression line is 0.49 for asbestosis and 0.31 for "pleural neoplasms." If this apparent increase in mortality is real and continues, then the calculated mortality for asbestosis given in Table 15 predicts that a total of 1337 asbestos-related deaths will occur in the year 2000 (calculation by the method given previously). Hopefully, asbestos-related mortality is now peaking and will soon decline.

[7]/A significant number of male mesothelioma cases will probably be reported under this code (163.0) thus a trend in this mortality may reflect a trend in total asbestos-related mortality.

Table 15. Calculated mortality based on linear regression analysis of past reported mortality - asbestosis (males + females, 1967-1977) and "pleural neoplasms" (males, 1968-1977) - Vital Statistics of the U.S., 1967-1977.

Year	Asbestosis (males & females)	"Pleural Neoplasms"(males)
1967	27 deaths	184 deaths
1977	52	222
1982	64	241
2000	105	308

In regard to mesothelioma incidence, it is pertinent to note the number of deaths from this disease in the four hospitals of the New York University Medical Center. The reported deaths for the period 1967 to 1976 are (Demopoulos, 1980a): 1967(3 deaths), 1968(2), 1969(3), 1970(4), 1971(1), 1972(2), 1973(1), 1974(3), 1975(4), and 1976(3). No significant trend with time is noted (r^2 = 0.01). Sprayed-on chrysotile asbestos was extensively used in building construction in New York City until recently. Also, chrysotile asbestos emissions from brake linings give measurable fiber counts in the ambient air of the New York City streets. For example, Nicholson et al. (1980) report that 43 of the 89 air samples collected in New York City exceeded 50 ng of chrysotile asbestos per m^3. Samples taken in New York City public schools ranged from 9 to 1950 ng/m^3 with 15 out of 27 samples exceeding 100 ng of chrysotile per m^3. Despite the long presence of chrysotile asbestos in the New York City air, Demopoulos (1980b) found no evidence of an increase in the number of mesothelioma deaths in this city over the past 12 years (1967-1978). Vianna et al. (1981) in a study of malignant mesothelioma in New York State (excluding New York City) found that there was no increase in the incidence of disease from 1973 through 1978.

SUMMARY

Asbestos Production

Of the six forms of asbestos, only four have been used to any significant extent in commerce. These are amosite, crocidolite, anthophyllite, and chrysotile. Although asbestos was used by Stone Age man it was not until the latter part of the 19th century that it came into widespread use in the industrialized world. The modern industry began in Italy and England after 1860, with Quebec being the main supplier of the crude fiber. By 1980, more than 100 million tonnes of asbestos had been mined worldwide of which more than 90 million tonnes was the chrysotile variety, about 2.7 million tonnes the crocidolite variety, about 2.2 million tonnes the amosite variety, and about 0.4 million tonnes the anthophyllite variety. Approximately 75 percent of all asbestos ever mined has come from just three chrysotile mining localities, Quebec, Canada and the central and southern Urals of the Soviet Union. The chrysotile-producing countries in order of importance are: the Soviet Union (46.1 percent of the world's total asbestos production in 1978), Canada (28.9%), Zimbabwe (3.8%), China (3.8%), Italy (2.9%), South Africa (2.1%), Brazil (1.8%), U.S.A. (1.7%), and Australia (1.0%).

Comparative Epidemiology

The three principal diseases which are related to asbestos exposure are: (1) lung cancer, (2) cancer of the pleural and peritoneal membranes (mesothelioma), and (3) asbestosis, a condition in which the lung tissue becomes fibrous and thus loses its ability to function. These diseases, however, are not equally prevalent in the various groups of asbestos workers that have been studied; the amount and type of disease depends on the duration of exposure, on the intensity of exposure, and particularly on the type or types of asbestos to which the individual was in contact.

Chrysotile or "white" asbestos. Chrysotile asbestos, sometimes referred to in the trade as "white" asbestos, is the form that is usually used in the United States - as wall coatings, in brake linings, as pipe insulation, etc. About 95 percent of the asbestos in place in the U.S. is the chrysotile variety and a large percentage of this was mined and milled in Quebec. Epidemiological studies of the chrysotile asbestos miners and millers of Quebec undertaken by medical researchers in Canada show that for men exposed for more than 20 years to chrysotile dust averaging 20 fibers/cm^3 the total mortality was less than expected (620 observed deaths, 659 expected deaths). Risk to lung cancer was slightly increased; 48 deaths observed, 42 deaths expected (Table 17, column A). Exposures to 20 fibers/cm^3 are an order of magnitude greater than those experienced now (generally less than 2 fibers/cm^3); thus chrysotile miners working a lifetime under these present dust levels should not be expected to suffer any measurable excess cancer. A similar mortality picture is reported for Italian chrysotile miners and millers (Study G, Table 4).

The results of only one epidemiological study of a cohort of trades workers known to be exposed only to chrysotile asbestos been published (Study VI, Table 3a). This study reports 2 deaths due to asbestosis but no excess of any cancer was detected.

Mesothelioma incidence among those working only with chrysotile asbestos is very low. Thus far, about 16 deaths due to this disease have been reported among chrysotile asbestos miners and millers and none among chrysotile trades workers.[8/] In addition, 6 deaths among sons and daughters of chrysotile miners and millers and two among others living in chrysotile asbestos mining localities have been reported as being due to mesothelioma.

Four epidemiological studies of the female residents of the Quebec chrysotile mining localities show no statistically significant evidence that their life-long exposure to asbestos dust from the nearby mines and mills has caused excess disease.

Crocidolite or "blue" asbestos. Crocidolite, usually referred to in the trade as "blue" asbestos, was first imported into the United States in 1911 or 1912. By 1930, 35,000 short tons of crude blue fiber had entered the country and by 1946 an additional 21,000 tons were imported. In addition to these imports, much crocidolite came into the United States as manufactured products such as yarns, tapes, and pipe coverings. Almost all of the imported crocidolite came from South Africa.

Epidemiological studies of groups who worked only with crocidolite asbestos show that rather short periods of exposure, or even relatively light exposure, causes a large excess of mortality due to lung cancer, mesothelioma, and asbestosis. This is evident not only in those exposed to crocidolite during gas mask fabrication and building construction but in those employed in the crocidolite mines.

There are only two mining regions in the world where mesothelioma is a statistically significant cause of death. These are the crocidolite mining districts of the Cape Province, South Africa and at Wittenoom, Western Australia. Prevalence studies in the Cape Province report that at least 278 people have died of mesothelioma as a result of exposure to crocidolite; 161 of these people worked in the mines and mills and 117 others lived in the vicinity of the mines.

Thirty-one men who worked in the crocidolite industry at Wittenoom, Western Australia have died of mesothelioma. Of these, 13 had worked for less than 12 months and 9 had light to medium exposure to blue asbestos. Sixty miners and millers at Wittenoom have died of lung cancer; 34 of these men had worked in the industry for less than 12 months and 19 had light to medium exposure to the crocidolite dust. In addition to this occupationally related mortality, 6 others who lived near but did not work in the mines or mills have died of mesothelioma.

Amosite or "brown" asbestos. All amosite asbestos comes from the Transvaal Province of South Africa where between 1917 and 1979 approximately

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8/Acheson et al. (1981, p. 1405) cite two new studies (in press) of asbestos trades workers thought to have been exposed to only chrysotile which confirm the rarity of mesothelioma associated with chrysotile.

2.2 million tonnes have been mined. Importation of amosite into the U.S. started in the 1930's.

One complete epidemiological study of trades workers exposed mainly to amosite asbestos has been published. The incidence of asbestos-associated disease in this group of men formerly employed at a factory in Patterson, N.J. was excessive; there being a 19.7% lung cancer mortality (60 cases), a 4.6% mesothelioma mortality (14 cases), and a 5.9% asbestosis mortality (18 cases). An additional study, only partially published, reports on a group of workers exposed mostly to amosite in a London insulation board factory. Here 5 men have thus far died of mesothelioma (Acheson et al., 1981). Only prevalence studies have been made of amosite miners and millers. Two have died of mesothelioma. One resident of an amosite mining district has been reported as having died of this disease.

The rock-forming amphibole minerals grunerite and cummingtonite, which are isostructural and chemically similar to amosite, are considered (incorrectly) by some to be forms of asbestos. Health studies of miners working ores which contain these minerals as gangue do not show any indication of asbestos-related mortality (studies B,E, Table 4).

Anthophyllite asbestos. This form of asbestos has been mined sporadically in many localities but the only major production was at Paakkila, Finland where approximately 350,000 tonnes was mined between 1918 and 1975. The only health study of individuals exposed predominantly to anthophyllite asbestos is that of the Paakkila miners (Study A, Table 4). This group showed a 67% excess of lung cancer and a large mortality due to tuberculosis and asbestosis. None died of mesothelioma. Because anthophyllite was and is used so little in commerce no additional health studies appear possible except for follow-up of the Paakkila miners.

Comparison of health effects of white, blue, and brown asbestos. There is a large contrast in the incidence of mesothelioma among those who were exposed to only one form of the three commonly used asbestos minerals-chrysotile, crocidolite, and amosite. This is demonstrated by comparing the total asbestos production to the number of mesothelioma deaths reported in the literature for miners, millers, and residents of four major asbestos mining localities. The pertinent data are given in Table 16. The difference between mesothelioma mortality reported in the chrysotile mining district and that reported in the crocidolite and amosite mining districts may be even greater than indicated in Table 16. This is because asbestos-related mortality is probably much underreported in Western Australia and South Africa due to the transient nature of the mining populations where many are lost from view. Quebec, on the other hand, has a very stable mining and residential population, the medical surveillance of which has been excellent.

Table 16. Mesothelioma Mortality[1] in Four Asbestos Mining Districts Relative to Asbestos Production

	Quebec Canada	Western Australia	Cape Province South Africa	Transvaal Prov. South Africa
Asbestos type	chrysotile	crocidolite	crocidolite	amosite
Tonnes mined	40,000,000	155,000	2,700,000	2,200,000
Years mined	1878-Pres.	1938-1966	1893-Pres.	1917-Pres.
No. deaths (mesothelioma)	22[2]	37	278	3[3]
deaths/100,000 tonnes mined	0.06	23.9	10.3	0.14

[1] Reported in the scientific literature up to 1979 for miners, millers and other residents of the mining districts
[2] Excludes 7 mesothelioma cases that were exposed to crocidolite.
[3] Excludes 4 mesothelioma cases that were exposed to crocidolite.

Several studies (Jones et al., 1980a, 1980b; McDonald, 1980b; McDonald, 1980c) have been made on the types and amounts of asbestos fiber in the lung tissue of asbestos workers who died of mesothelioma and in "controls" who died of other diseases and who were not occupationally exposed to asbestos. For example, Jones et al. (1980b) found that chrysotile was present no more frequently nor in greater amounts, in the mesothelioma cases than in the control cases. They further state (p. 197) that "this study therefore provides no evidence to indict chrysotile in the etiology of mesothelioma." Similar findings are reported by McDonald (1980b) and McDonald (1980c). The above cited studies also show that amphibole fibers (crocidolite and amosite) were more prevalent in the lung tissues of the mesothelioma cases than in the controls.

The constrast in mortality holds also for lung cancer and asbestosis. All three forms cause significant excess of these two diseases in those exposed for long periods of time to high dust levels. However, short term exposure to moderate levels of crocidolite dust appears to be more dangerous than long term exposure to high levels of chrysotile dust. This can be seen by comparing the mortality data for the Quebec chrysotile asbestos miners and millers who were exposed to high to very high dust levels for 20 or more years (Table 17, column B) to the data for the Canadian gas mask assemblers who were exposed to moderate levels of crocidolite dust for no more than 2.5 years (Table 17, column C). For further comparison, mortality data is given for Quebec miners and millers who were exposed to low to medium dust levels (column A).

Table 17. Mortality Data for Quebec Chrysotile Miners and Millers (columns A,B) with 20 or more years service and for Canadian Gas Mask Assemblers (column C).

	A[1]			B[1]			C[2]	
Exposure	Low-medium(10-21 f./cm^3)			High-V.high(95-194 f./cm^3)			Moderate	
Cause of death	Obs.	Exp.	%[3]	Obs.	Exp.	%[3]	Obs.	%[3]
All causes	620	658.8	100	478	367.7	100	56	100
All cancer	147	150.6	23.7	132	82.8	27.6	23	41.1
Lung cancer	48	41.6	7.7	56	23	11.7	8	14.3
G.I. cancer	47	58.7	7.6	45	32.6	9.4	3	5.4
Asbestosis	11	0.7	1.8	25	0.4	5.2	2	3.6
Other Resp.	28	34.3	4.5	31	18.4	6.5	4	7.1
Mesothelioma	-			-			9	16.1

[1] Data from McDonald et al. (1980, Table 7d).
[2] Exposed to moderate levels of crocidolite dust for no more than 2.5 years (McDonald and McDonald, 1978). See also Study X, Table 3a.
[3] Percent of all observed deaths.

The contrast between cancer mortality in the mining-milling cohorts (Table 4) and the trades cohorts (Table 3a,b) is clearly seen in Figure 1 where the proportional mortality due to lung cancer is plotted with respect to mesothelioma proportional mortality. The average mortality for the 8 mining-milling cohorts (excluding the crocidolite miners, Study F, Table 4) is: mesothelioma (0.2%), lung cancer (5.7%). For the 21 trades cohorts, the average mortality is: mesothelioma (5.6%), lung cancer (16.7%). The cancer mortality pattern of the crocidolite mining cohort is very similar to that of the trades cohorts; 3.3% of this group having died of mesothelioma and 11.6% of lung cancer.

Many of the men in the trades cohorts were probably exposed to crocidolite asbestos at some time during their working career. They also were probably often exposed to very high concentrations of asbestos dust, particularly during installation or removal of asbestos in closed spaces such as heating conduits, ship compartments, etc.

Peto (1978, 1980) and Peto et al. (1982) suggest that chrysotile asbestos contributes significantly to mesothelioma mortality. Peto bases his conclusions on a study of workers in an asbestos textile factory in the UK (Peto et al., 1977; Peto 1978, Peto 1980). Exposure was mainly to chrysotile asbestos but crocidolite fiber was processed in this factory at various times since 1933 (Peto, 1978, p. 487). Peto does not cite the work of Jones et al. (1980a) and McDonald and McDonald (1978) on the gas mask assemblers, nor the work of Hilt et al. (1981) on construction workers; studies which clearly show the extreme hazard of crocidolite. Only brief exposure to crocidolite can set up a disease pattern very similar to that

found in trades workers heavily exposed to asbestos for many years. From this we can conclude that mortality studies of asbestos workers who are exposed to crocidolite, even for short periods of time, are not valid in predicting the health effects of other forms of asbestos.

Estimates of Asbestos-related Mortality in the United States

In various press releases and scientific publications, it has been suggested that asbestos exposure in the United States will cause from 10,000 to 67,000 deaths per year for the next 20 to 30 years. These estimates do not appear to be valid when compared to estimates of past mortality that are based on reported asbestos-related death due to mesothelioma or asbestosis. For example: (1) utilizing the mortality pattern of excess disease in 17,800 North American asbestos insulation workers and the incidence of mesothelioma in 1972 given by a pathology review panel, it is estimated that 587 individuals died in that year because of exposure to asbestos; or (2) taking the reported number of asbestosis deaths as given in the Vital Statistics of the United States and again utilizing the mortality data of the North American insulation workers, it is estimated that the average yearly asbestos-related mortality in the United States during the period 1967-1977 was 522 deaths. There is some suggestion from the mortality data given in the Vital Statistics that the incidence of asbestos-related disease has increased somewhat during this same period.

COMMENTARY

The Relative Hazards of the Asbestos Minerals

It is pertinent to repeat one of the questions asked in the introduction of this review: Must the use of all commercial asbestos be stopped? The answer is an emphatic no - but with qualifications presented below.

Non-occupational exposure to chrysotile asbestos, despite its wide dissemination in urban environments throughout the world, has been shown by epidemiological studies to be of no health significance whatsoever. If it were, the women of Thetford Mines, Quebec, where over 20 million tonnes of chrysotile asbestos has been mined, would be dying of asbestos-related diseases. They are not. The health studies accomplished in Canada show that populations can safely breath air and drink water that contain significant amounts of chrysotile fiber. These studies also show that there is a "threshold" value for chrysotile asbestos exposure below which no measurable health effect will occur.

The same fiber dose-disease response relationships observed for chyrsotile asbestos do not hold for crocidolite asbestos. Health studies of those exposed to only crocidolite show it to be much more hazardous than chrysotile; with respect to mesothelioma perhaps 100 to 200 times more hazardous. No study has been reported comparable to that made for chrysotile which indicate what a safe level of exposure to crocidolite would be. The danger of crocidolite dust is particularly emphasized by the many mesothelioma deaths occurring among the residents of the crocidolite mining districts of the Cape Province, South Africa whose only exposure was in a non-occupational setting. Such mortality is practically unknown among residents of the chrysotile mining localities of Quebec. Control of crocidolite dust, particularly in the mines and mills, presents a considerable engineering problem in that dust levels at or below the 1969 British Standard of 0.2 fibers/cm^3 can be achieved hardly anywhere (Simpson, 1979, p. 74).

The hazards of amosite asbestos are more difficult to access. The amosite factory employees of Patterson N.J., who worked under very dusty conditions during World War II, have experienced a great deal excess mortality due to lung cancer, asbestosis, and mesothelioma. In contrast to these factory workers, amosite miners and millers, at least with regard to mesothelioma, do not appear to be at much risk. This suggests that dust controls are possible which can much reduce or prevent the occurrence of asbestos-related diseases in amosite workers.

The fear caused by heavy handed statements such as "one fiber can kill you" and by the much exaggerated predictions of the amount of asbestos-related mortality expected in the next 20 or 30 years has generated great political pressure to remove asbestos from our environment and to greatly reduce or even stop its use. An example of this is the concerted effort in several industrial nations including the United States to remove asbestos from schools, public buildings, homes, ships, appliances, etc. This is being done, even though most asbestos in the U.S. is of the chrysotile variety, and even though asbestos dust levels in schools, public

buildings and city streets is much lower than found in chrysotile asbestos mining communities where no asbestos-related disease appears in the non-occupationally exposed residents. The impetus for these costly removals and appliance recalls (hair dryers, for example) apparently comes from propagandizing the "one fiber can kill you" concept. Not only is this program costly - it could be dangerous if the removal of blue asbestos is not accomplished with great care. In most cases, asbestos coatings and insulation, where necessary, can be repaired at no risk and at a fraction of the cost of complete removal.

Substitutes for Asbestos

If all use of asbestos were to be discontinued, substitutes would have to be developed to meet many diverse requirements such as non-flammability, high strength, flexibility, reasonable cost, and safety. With respect to safety, the substitutes must not induce disease in those exposed to them and also must not endanger lives in other ways because of inferior strength and durability, increased flammability, etc. A high cost for a good substitute must not force instead the use of an inadequate replacement. Possible problems with substitutes may occur, for example, with the replacement of chrysotile asbestos in drum brake linings. The chance of increased automobile accidents due to a possibly inferior substitute material must be weighed against the probability of anyone being harmed by the small amounts of chrysotile asbestos that are emitted from drum brakes. Also, the health effects of emissions from substitute brake linings must be considered.

The requirements of strength and flexibility necessitate that asbestos substitutes be fibrous. Generally, the thinner and longer the fibers, the stronger, more flexible, and useful they are. However, fibers longer than 4 microns and less than 1.5 microns in diameter are capable of producing malignant neoplasms when implanted into the pleura of rats (Stanton et al., 1981). The test fibers in these studies included aluminum oxide, fiber glass, wollastonite ($CaSiO_3$), silicon carbide, dawsonite ($NaAlCO_3OH$), and potassium octatitanate.

Lee et al. (1981a, 1981b) studied the effects on rats, hamsters, and guinea pigs of inhalation of different concentrations of "Fybex", a commercially made potassium octatitanate fiber used to strengthen materials. They found that in addition to the development of pulmonary fibrosis in many animals, 3 hamsters developed pleural mesothelioma, a rare disease in the control animals.

In the report of the Advisory Committee on Asbestos, Health and Safety Commission of Great Britain, the following statement is made in regard to substitutes for asbestos (Simpson, 1979, vol. 1, p. 69).

> "As a general principle we take the view that control of any useful but hazardous material is preferable to the ultimate sanction of prohibition. It is very easy to say that a dangerous substance or process should be banned and to hope that that will solve the problem. In our view this is a gross

> over-simplification of a complex equation of interlinked factors. It ignores the possibility that prohibition of a particular substance may directly result in an increase in health or safety risks, for example from fire, which the use of that substance currently prevents or reduces. It also ignores the implications of statutorily enforcing substitution by materials or substances which at present appear to be suitable but may at a later date be found to constitute a risk to health. The social and economic consequences of the possible closure of factories using the original material or process need to be taken into account."

The recent animals experiments such as those cited above make the Advisory Committee's statement particularly meaningful.

The cost of asbestos substitutes is of particular importance to the "Third World" countries whose developing economies are very dependent upon making the maximum use of cheap, domestically produced materials whereever possible. Asbestos cement is such a material and large quantities of it are vital to the industrialization of these nations. Importation of possible substitutes, for example, plastic and metal water pipe and construction materials, is not an economic choice for many nations. It is significant that several countries are greatly expanding their chrysotile mining and milling operations; the U.S.S.R., Zimbabwe, Greece, Mexico, Yugoslavia (Asbestos, vol. 63, January 1982).

The Ubiquitous Fibrous Minerals and Future Health and Regulatory Policy

Those outside the mining and geoscience professions probably do not appreciate how common fibrous minerals are. Most hard rock mines contain some gangue minerals that are considered by some to be asbestos or asbestos-like. For example, the common rock-forming mineral cummingtonite found in the Reserve Mining Company's iron ore deposits near Lake Superior, Minnesota is considered to be asbestos by the U.S. Environmental Protection Agency and the Courts of Minnesota, although I know of no geologist who would call this mineral asbestos. However, be it as it may, if cummingtonite and other amphiboles are considered to be asbestos for regulatory purposes then a great many mining operations will also be considered asbestos mining operations. The mining and milling of gold and iron ore, talc, vermiculite, and crushed stone have already been effected by asbestos regulations.

In addition to the fibrous minerals found in numerous ore deposits, they are also found in many water supplies, in soils and sediments, in certain sand and gravel deposits, in drilling muds, in portland cement, in ceramic materials, and in large areas overlain by volcanic ash. Should the public be told that even low doses of these mineral fibers can possibly cause cancer? Should human exposure to these fibers be regulated to the lowest feasible limit? Should extreme measures be taken such as moving people out of regions where fibrous minerals are endemic? Such action

was proposed by P. C. Elmes, Director of the MRC Pneumoconiosis Unit, Llandough Hospital, Penarth, Wales. He writes (Elmes, 1980, p. 529),

> "Populations living on soils contaminated with the fibers under dry climatic conditions need to be moved."

In regard to his proposal, it is noted that fibrous zeolite minerals occur in many areas of the southwestern United States and that fibrous clay minerals are common in the coastal plane sediments of the eastern United States.

One does not have to consider the above questions very long before coming to realize that if answered in the affirmative they would present a regulatory, legal, and economic nightmare.

Instead of overreacting to every perceived health risk (this seems to occur particularly in regard to suspected carcinogens) we must allocate our scientific and economic resources to our environmental health problems in proportion to their seriousness. Billions of dollars have been spent directly and indirectly, in understanding and mitigating asbestos-related cancers. Many billions more may be awarded to those filing claims against asbestos companies. In contrast, relatively little has been spent on understanding and mitigating the more serious non-neoplastic lung diseases such as the pneumoconioses caused by inhalation of crystalline silica and coal dusts.

A great deal has been accomplished in understanding the relationships between the intensity of exposure to the several forms of commercial asbestos and the incidence of the asbestos-related diseases. Epidemiological studies have shown that modern dust control methods now in effect can prevent most morbidity and mortality related to exposure to chrysotile asbestos. Similar studies should be completed in order to set dust levels to protect the anthophyllite and amosite asbestos workers (in this regard see the Simpson Report, Simpson, 1979).

A panel of the U. S. National Academy of Sciences is now reviewing the health problems associated with human exposure to fibrous materials including commercial asbestos. The panel is composed of equal numbers of medical and geoscientists and hopefully their cooperative efforts will give us additional information on the present and potential hazards of exposure to the various mineral, inorganic, and organic fibers to which humans may be exposed and also give us guidelines for permissible levels of exposure for each particular type of fiber.

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