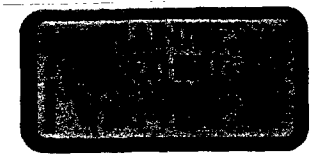

*Malcolm Ross*[1]

Authorized Reprint from
Special Technical Publication 834
Copyright
American Society for Testing and Materials
1916 Race Street, Philadelphia, PA 19103
1984

# 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 Nonoccupational Exposure to Fibrous Minerals

**REFERENCE:** Ross, M., "**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 Nonoccupational Exposure to Fibrous Minerals,**" *Definitions for Asbestos and Other Health-Related Silicates, ASTM STP 834*, Benjamin Levadie, Ed., American Society for Testing and Materials, Philadelphia, 1984, pp. 51–104.

**ABSTRACT:** A review based on 36 published epidemiological studies is given of disease patterns that have developed among industrial workers, miners, and millers who had been exposed to dusts of one or more of the commercial asbestos minerals or to dusts from minerals perceived to be asbestos-like. Health data are also reviewed for individuals exposed to asbestos dusts in nonoccupational settings. From the published reports it is clear that there are very significant differences in the health effects of the several asbestos or asbestos-like minerals

Of the commercial asbestos utilized in the United States, about 95% has been chrysotile or "white" asbestos, about 2% amosite or "brown" asbestos, and about 2% crocidolite or "blue" asbestos. The common white asbestos has had the least effect on those occupationally exposed, whereas blue asbestos has had the most effect. Despite the wide dissemination of white asbestos in our environment—in schools, homes, public buildings, brake lining emissions, and so forth—there is little evidence that the very frequent nonoccupational exposures to this form of asbestos have caused any harm. On the other hand, nonoccupational exposure to blue asbestos has been conclusively proven to have caused significant mortality. The different health effects of the various forms of asbestos require different regulatory responses and remedial actions.

**KEY WORDS:** asbestos, health-related silicates, asbestos-related disease, health hazard, mortality, cancer, asbestosis, mesothelioma, lung cancer

[1] Research mineralogist, U.S. Geological Survey, MS959, Reston, Va. 22092.



10002274

*The Problem*

The widespread use of amphibole and serpentine asbestos[2] by industrial society for such uses 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 that 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 that 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 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 the 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

[2] At present, the most widely used definition of asbestos in the United States is taken from the notice of proposed rule-making for "Occupational Exposure to Asbestos" published in the *Federal Register* on 9 Oct. 1975 (pp. 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: a length greater than 5 μm, a maximum diameter less than 5 μm, and a length-to-diameter ratio of three or greater. Any product containing *any* of these minerals in this size range is also defined as asbestos.

10002275

ne asbestos[2] by industrial society
s, electrical and heat insulation,
filters, packings, and construc-
safety and convenience. Yet,
gible benefits, many asbestos
and mesothelioma.
er certain conditions have been
dinary actions to greatly reduce
h animals demonstrate that the
r fibrous materials, can cause
planted within the pleura. These
sts that asbestos-related diseases
es: the mineral type, according
actor in the etiology of disease,
t enter the human body.
nd regulatory establishments is
benefits. Should the asbestos
be banned from use? Minerals
ly important in this regard, for
ne habits that are considered by

less of many asbestos products,
s-like minerals in the natural
ons, and the uncertainty of the
als, different mineral particle
ombine to present a formidable
lustries, and legal and health
sbestos be stopped? Must all
d to the lowest feasible levels
and, thus, isolated from the
eliminated from our drinking
st we cease to use asbestos in

n the United States is taken from the
to Asbestos" published in the *Federal*
upational Safety and Health Adminis-
hibole minerals amosite, crocidolite,
ral chrysotile are classified as asbestos
llowing dimensions: a length greater
n-to-diameter ratio of three or greater.
ge is also defined as asbestos.



brake linings, cement water pipe, and structural building materials? Must eve low levels of nonoccupational exposure to asbestos or asbestos-like materials b avoided at any cost?

In order to provide information that will aid the reader in addressing thes questions intelligently, the author will review the role of asbestos in the worl economy, list 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, in the mining and milling occupations, and in individuals not occupationally exposed to asbestos or asbestos-like minerals. As we will see, the six asbestos minerals used in commerce are not identical in crystal structure, chemical composition, abundance, or 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 for causing 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 use in human culture goes back to at least 2500 B.C. Archeological studies [*1*][3] show that the inhabitants of the Lake Juojärvi region of East Finland knew how to strengthen earthenware pots and cooking utensils with anthophyllite asbestos. This asbestos probably came from the same areas that have 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 includes such endeavors as the small industry developed in Russia during the rule of Peter the Great, in which chrysotile asbestos from the Urals was used for a short period of time in the production of textiles.

In the 1860s and 1870s, 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 Province, Canada. The supply for the first time was ample, and the market was ready.

[3] The italic numbers in brackets refer to the list of references appended to this paper.

10002276

### *The Modern Industry*

The reopening of the asbestos deposits of northern Italy, deposits that 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 [*2*]; 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 asbestos fiber was probably about 200 000 tonnes (metric), certainly no more than 300 000 tonnes. Of this, 150 000 tonnes came from Quebec Province, Canada. By 1980 more than 100 million tonnes of asbestos had been mined throughout the world; of this, more than 90% was chrysotile and more than 5% 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. The total production of anthophyllite asbestos to date is probably no more than 400 000 tonnes, 350 000 tonnes being produced by Finland alone. The production of tremolite asbestos has been sporadic, and it has been mined in various parts of the world for short periods of time. The total production to date of 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 [*3*]. Russia leads with 46.1% and Canada is second with 28.9% of the world's output. Both countries mine only chrysotile asbestos, and most of it comes from the Ural Mountains and Quebec Province. The third leading asbestos producer is the Republic of South Africa (7.1%); the asbestos ore consists of amosite, crocidolite, and chrysotile. These three countries furnished 82.1% 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. Detailed understanding of the chemistry and crystal structures of these asbestos minerals came later than their discovery; 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 nonfibrous varieties are known. About 90% of the past and about 95% of the present world production of asbestos was or is of the chrysotile variety.

10002277

them Italy, deposits that had
l the beginning of the modern
was full-blown, with hundreds
f the century, the large South
. and the Russian deposits in
ntity. Within a few years, the
ited.

os by Stone Age man to 1900,
os fiber was probably about
) 000 tonnes. Of this, 150 000
1980 more than 100 million
world; of this, more than 90%
l amosite. Nearly 40 million
le mined in Quebec Province
he total production of antho-
an 400 000 tonnes, 350 000
duction of tremolite asbestos
s parts of the world for short
form of asbestos is probably
ial exploitation of actinolite

in Table 1 [3]. Russia leads
of the world's output. Both
t of it comes from the Ural
ng asbestos producer is the
nsists of amosite, crocidolite,
.1% of the world's asbestos
luce mostly chrysotile.

ears usually list six forms of
amosite, crocidolite, antho-
riety is chrysotile. Detailed
s of these asbestos minerals
er literature can be confusing

ommon polymorphs of ser-
varieties are known. About
l production of asbestos was

TABLE 1—*World asbestos production in 1978* [3].

| Fiber | Location | 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 |
| | Russia (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 |

Amosite is the very rare asbestiform variety of grunerite amphibole [$(Fe,Mg)_7Si_8O_{22}(OH)_2$]; the name of this variety is derived from the term *AMOSA*, an acronym for the company Asbestos Mines of South Africa [4]. This valuable commercial asbestos is mined only in the Transvaal Province of South Africa.

Crocidolite is the asbestiform variety of riebeckite amphibole [ideally $Na_2(Fe^{2+},Mg)_3Fe_2^{3+}Si_8O_{22}(OH)_2$] 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.

10002278

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, or prismatic; these terms suggest an elongate habit. Although such minerals are extremely common, only in a relatively few places do they have physical and chemical properties that make them 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:

(*a*) *Type I*—alpine-type ultramafic rocks, including ophiolites (chrysotile, anthophyllite, and tremolite);
(*b*) *Type II*—stratiform ultramafic intrusions (chrysotile and tremolite);
(*c*) *Type III*—serpentinized limestone (chrysotile); and
(*d*) *Type IV*—banded ironstones (amosite and crocidolite).

Type I deposits are by far the most important and probably account for more than 85% of the asbestos ever mined. The most important Type I deposits are those in Quebec Province and in the Ural Mountains.

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, Ariz., and in the Carolina area of the Transvaal Province of South Africa. Type IV deposits are found only in Precambrian banded ironstones located in the Transvaal and Cape Provinces of South Africa and in 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 [5].

**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

10002279

rrences of tremolite asbestos
s of occurrences of actinolite
d actinolite asbestos are now,
mportance.

*ercial Asbestos*

me serpentines, are described
rm, or prismatic; these terms
are extremely common, only
and chemical properties that
ally, amphibole minerals may
ings and in areas of secondary
ient quantity to be profitably

our types of rocks:

ing ophiolites (chrysotile, an-

rysotile and tremolite);
e); and
rocidolite).

l probably account for more
nportant Type I deposits are
ns.
, Swaziland, and Zimbabwe.
deposits are small; the most
in the Carolina area of the
osits are found only in Pre-
aal and Cape Provinces of
uth African deposits are still
occurrences of commercial

o one or more of the com-
r, which includes cancer of
oma, a cancer of the pleural
nd abdominal cavities, re-
fibrosis of the lung tissue,
ng function and respiratory
vorkers is also complicated



by its association with cigarette smoking, which leads to considerable difficulty in assigning the relative risks of asbestos exposure to smokers. Mesothelioma, a disease 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 [*6*]; other studies do not support this conclusion [*7–10*]. Some question still exists then as to the role played by asbestos in the etiology of digestive tract cancers. Becklake [*11*], Selikoff and Lee [*6*], and Simpson [*12*] give reviews of the subject of asbestos and disease.

The 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 that 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 [*13*]. Most particles that enter the upper respiratory tract (the main stem, bronchi, and bronchioles) are quickly and effectively removed by the mucociliary 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 longer than approximately 10 μm are not easily 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. Asbestos fibers can stimulate deposition of excess interstitial collagen and reticulin fibers. This causes the alveolar septa to become thickened, with ensuing impairment of oxygen uptake [*13*]. 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, anthophyllite, 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 the first exposure. A significant increase in the lung cancer death rate appears 10 to 14 years after the first exposure and peaks between 30 and 35 years after. The mesothelioma death rate becomes significant 20 years after the first exposure but continues to climb even after 45 years have elapsed. The asbestosis death rate becomes significant 15 to 20 years after the first exposure and apparently peaks between 40 and 45 years after [*14*].

10002280

*Epidemiology*

Before considering the mortality studies of the various occupational groups exposed to asbestos, we should briefly consider the roles the three important types of asbestos (amosite, crocidolite, and chrysotile) have played in the commerce of North America and Europe, the areas where the major epidemiological studies of asbestos workers have been 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 shipbuilding 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 brought into the United States are given in Tables 2*a* and 2*b*. Large amounts of manufactured goods containing crocidolite were also imported, but tonnage estimates cannot be made. Not until the mid-1930s 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, N.J., in 1920, and in 1924 the operation moved to larger facilities in Millington, N.J. The many advertisements in the trade journal *Asbestos* between 1920 and 1945 indicate that crocidolite was used in many products and particularly for insulation of steam boilers, locomotives, and pipes. For example, a product containing crocidolite asbestos and called "85% magnesian sectional pipe covering" was advertised monthly in *Asbestos* from 1920 to 1945 (see also Ref *15*). Amosite, crocidolite, and chrysotile were almost universally used aboard ships 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 [*16*],[4] railroad cars, buildings, and other structures. 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 American ships; their principal uses were as fireproofing for steel building girders and as acoustical coatings in schools and offices.

*Asbestos Trades Workers*—A very significantly increased incidence, in relation 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, and buildings; and fabrication and installation of

[4] Mesothelioma is prevalent in the shipyard workers of Europe at Walcheren, Wilhelmshaven, Plymouth, Trieste, Hamburg, Nantes, Rotterdam, and Malmö [*16*]. 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.

10002281

he various occupational groups
r the roles the three important
sotile) have played in the com-
here the major epidemiological

ket in large quantities early in
ed in the United States in 1912
til World War I that its use for
particularly in the shipbuilding
idolite fiber had been imported
crocidolite asbestos from South
n in Tables 2*a* and 2*b*. Large
idolite were also imported, but
nid-1930s did amosite asbestos
to replace crocidolite for high-
Bound Brook, N.J., in 1920,
lities in Millington, N.J. The
*s* between 1920 and 1945 in-
and particularly for insulation
xample, a product containing
sectional pipe covering" was
5 (see also Ref *15*). Amosite,
lly used aboard ships during
lers and pipes, crocidolite for
sotile for low-temperature and

in North America, with one
lite asbestos as a sprayed-on
ildings, and other structures.
States after World War II, but
sotile rather than crocidolite.
merican ships; their principal
and as acoustical coatings in

ncreased incidence, in relation
estosis, and mesothelioma is
trades"—insulation of steam
abrication and installation of

rope at Walcheren, Wilhelmshaven,
*16*]. The extensive use of crocidolite
ggested to be an important factor in

TABLE 2*a*—*Minimum estimates of imports of crude crocidolite asbestos from South Africa brought into the United States for the periods 1907–1929,[a] 1940–1945,[b] 1946–1974.[c]*

| Years | Crocidolite Crude, short tons[d] |
|---|---|
| 1907–1908 | probably none |
| 1909–1910 | no data |
| 1911 | little |
| 1912 | 9 |
| 1913 | 1 |
| 1914 | no data |
| 1915 | probably none |
| 1916 | 1 184 |
| 1917 | 2 081 |
| 1918 | 837 |
| 1919 | 1 056 |
| 1920 | 2 979 |
| 1921 | 704 |
| 1922 | 1 684 |
| 1923 | 2 040 |
| 1924 | 1 457 |
| 1925 | 606 |
| 1926 | 4 873 |
| 1927 | 5 587 |
| 1928 | ? |
| 1929 | 9 952 |
| Total (1907–1929) | 35 050 |
| 1940 | 2 708 |
| 1941 | 2 976 |
| 1942 | 4 213 |
| 1943 | 4 808 |
| 1944 | 2 946 |
| 1945 | 3 100 |
| Total (1940–1945) | 20 751 |
| 1946–1974 | 346 796 |

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

[b]Crocidolite composed 19.4% of the South African crude asbestos imported into the United States during this period—which totaled 107 039 short tons; over 80 000 tons was amosite asbestos. Sources: *Minerals Yearbook* (1940 through 1945), U.S. Bureau of Mines, Washington, D.C.; *Mineral Trade Notes*, Confidential Series, Nos. 1–31, 1940–1945, U.S. Bureau of Mines, U.S. Department of the Interior, Washington, D.C.

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

[d]1 short ton = 0.907 tonnes (907 kg).

10002282

TABLE 2*b*—*Imports of all crude asbestos from South Africa for the period 1930–1939.*[a]

| Years | Asbestos Crude, short tons[b] |
|---|---|
| 1930 | 3 635 |
| 1931 | 2 290 |
| 1932–1933 | 1 370 |
| 1934 | 4 269 |
| 1935 | 2 529 |
| 1936 | ? |
| 1937 | 3 025 |
| 1938 | 4 248 |
| 1939 | 6 422 |
| Total | 27 788[c] |

[a]The import statistics do not differentiate between various forms of asbestos. Little chrysotile was imported from South Africa; thus, the figures are mostly for amosite plus crocidolite. The import data for crocidolite during World War II suggest that at least 20% of the imports for 1930 through 1939 were crocidolite. The amosite market was just developing in the United States in the 1930s. Most of the crocidolite was from Cape Province.

[b]1 short ton = 0.907 tonnes (907 kg).

[c]Assuming a ratio of 4:1 for amosite/crocidolite based on World War II import figures, at least 5558 short tons of crocidolite was imported into the United States from 1930 through 1939. Sources: *Mineral Resources of the United States* (1930, 1939), U.S. Geological Survey, Washington, D.C.; *Minerals Yearbook* (1932 through 1939), U.S. Bureau of Mines, Washington, D.C.

asbestos-containing textiles, roofing materials, cement products, tiles, wallboards, brake linings, clutch facings, filters, packings, gaskets, and other products. Those in these "trades" generally worked with several types of asbestos minerals during their working careers; most commonly, these types were chrysotile, crocidolite, and amosite, rarely anthophyllite. Significant exposure by any group of workers, at least for the past 40 years, to tremolite or actinolite asbestos dusts has probably not occurred.

Statistical data for 21 mortality studies of defined cohorts of asbestos trades workers (mostly male) are presented in Tables 3*a* and 3*b* [*17–36*]. For those studies that are still continuing, the most recent update is given. Of the studies, 12 are of asbestos factory workers, 8 are of asbestos insulation workers, and 1 is of asbestos construction workers. In all, 50 143 individuals were studied (1517 were female); of the 7166 listed deaths, 1198 (16.7%) were reported as due to lung cancer and 402 (5.61%) were reported as due to mesothelioma. In the 21 studies, the lung cancer mortality accounted for 6.1 to 26.6% of all deaths; mesothelioma mortality accounted for 0 to 16.1% of all deaths (Tables 3*a* and 3*b* and Fig. 1). The workers involved in Study 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 life-style as well as by occupation. The life-style contribution to lung cancer is cigarette use. To assess the significance of these health studies, it is necessary to examine the cancer mortality patterns

10002283

*frica for the period 1930–1939.*

| Crude, short tons[a] |
|---|
| 3 635 |
| 2 290 |
| 1 370 |
| 4 269 |
| 2 529 |
| ? |
| 3 025 |
| 4 248 |
| 6 422 |
| 27 788[c] |

rms of asbestos. Little chrysotile was amosite plus crocidolite. The import 20% of the imports for 1930 through ng in the United States in the 1930s.

World War II import figures, at least tes from 1930 through 1939. Sources: ological Survey, Washington, D.C.; es, Washington, D.C.

ement products, tiles, wall- ngs, gaskets, and other prod- vith several types of asbestos only, these types were chrys- lite. Significant exposure by ars, to tremolite or actinolite

ed cohorts of asbestos trades a and 3*b* [*17–36*]. For those date is given. Of the studies, tos insulation workers, and 1 ndividuals were studied (1517 7%) were reported as due to to mesothelioma. In the 21 6.1 to 26.6% of all deaths; of all deaths (Tables 3*a* and worked only with chrysotile, ly with crocidolite, and those e than one form of asbestos. difficult to predict, for cancer well as by occupation. The e. To assess the significance the cancer mortality patterns



of cigarette smokers who were not exposed to asbestos dusts. Unless the 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 3*a* and 3*b* 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 led to a consensus that this habit produces a very significant increase in the risk of dying of lung cancer as well as of the various cardiovascular diseases. The largest study of cigarette smokers is that of Hammond and his colleagues [*37*] under the auspices of the American Cancer Society. This study is based on questionnaires and mortality follow-up studies done in the United States between July 1960 and June 1971 for approximately 51 000 men. The proportional mortality of lung cancer (percentage of lung cancer deaths in relation to deaths by all causes), based on the Hammond study, is shown graphically in Fig. 2. For a group of men who all smoke cigarettes (cohort of 100% smokers), lung cancer mortality is approximately 7% at age 45, reaches a maximum of approximately 10% at age 70, and then decreases slightly at older ages. For a cohort of male nonsmokers, lung cancer mortality is 2% at age 45 and then decreases continuously to approximately 1% at age 95.

Smoking is more prevalent in blue-collar occupations than in professional and managerial occupations [*38*]. This smoking 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% had a history of cigarette smoking [*39,40*]. In a group of 1015 chrysotile asbestos miners and millers, 85% were smokers [*41*]. Data given in Fig. 2 predict that the lung cancer mortality for a cohort composed of 75% smokers would be at least 6 to 7.5%, regardless of occupation. In Tables 3*a* and 3*b* we see that the lung cancer mortality for the total of 21 cohorts of asbestos trades workers was 16.7%—approximately three times that expected if the mortality predictions were based only on the apparent smoking habits.

The risk of lung cancer due to asbestos exposure is lower in nonsmokers than in smokers [*40,42*]. There appears to be no relationship between smoking habits and the incidence of mesothelioma; the disease is equally prevalent in smokers and nonsmokers alike. Of the studies listed in Tables 3*a* and 3*b*, only Study VI, which is 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

10002284

TABLE 3a—*Cohort mortality studies of asbestos trades workers, excluding miners and millers (see Table 4). The most recent updates of Studies I through XI are given (1964–1982).*

| Cause of Death (Classification)[a] | Number of Deaths | Study I [17] | Study II [18] | Study III [19] | Study IV [20] | Study V [21] |
|---|---|---|---|---|---|---|
| All causes (000–999) | observed (expected) | 351 (343) | 46 | 361 | 446 (316.28) | 122 (54) |
| | ratio observed/expected | 1.02 | ... | ... | 1.41 | 2.26 |
| Cancer—all sites (140–239) or (140–209) | observed (expected) | 74 (80.7) | 23 | 96 | 172 (56.34) | 66 |
| | ratio observed/expected | 0.92 | ... | ... | 3.05 | ... |
| | % of all deaths | 21.1 | 50.0 | 26.6 | 38.6 | 54.1 |
| Cancer—lung, trachea, bronchus (162) | observed (expected) | 28 (33.0) | 10 (1.42) | 41 | 79 (17.47) | 35 |
| | ratio observed/expected | 0.85 | 7.0 | ... | 4.52 | ... |
| | % of all deaths | 8.0 | 21.7 | 11.3 | 17.7 | 28.7 |
| Mesothelioma—peritoneum, pleura (158, 163) | observed (expected)[a] | 2 (<1936) | 3 | 9 | 23 | 13 |
| | % of all deaths | 0.6 | 6.52 | 2.49 | 5.17 | 10.7 |
| Cancer—digestive system (150–159) | observed (expected) | ... | ... | 20 | ... | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | 5.54 | ... | ... |
| Cancer—gastrointestinal tract (150–154) | observed (expected) | 18 (19.6) | 5 (1.79) | ... | 24 (10.57) | 13 |
| | ratio observed/expected | 0.92 | 2.79 | ... | 2.27 | ... |
| | % of all deaths | 5.1 | 10.9 | ... | 5.38 | 10.7 |
| Respiratory disease (460–519) | observed (expected) | ... | ... | ... | ... | 30 |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of deaths | ... | ... | ... | ... | 24.6 |
| Noninfectious respiratory disease (510–519) | observed (expected) | ... | ... | ... | ... | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | ... |

10002285

| | | | | | | |
|---|---|---|---|---|---|---|
| system (150–159) | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | 5.54 | ... | ... |
| Cancer— gastrointestinal tract (150–154) | observed (expected) | 18 (19.6) | 5 (1.79) | ... | 24 (10.57) | 13 |
| | ratio observed/expected | 0.92 | 2.79 | ... | 2.27 | ... |
| | % of all deaths | 5.1 | 10.9 | ... | 5.38 | 10.7 |
| Respiratory disease (460–519) | observed (expected) | ... | ... | ... | ... | 30 |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of deaths | ... | ... | ... | ... | 24.6 |
| Noninfectious respiratory disease (510–519) | observed (expected) | ... | ... | ... | ... | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | ... |
| Asbestosis (515.2) | observed (expected)[f] | 0 | 2 | 39 | 26 | 19 |
| | % of all deaths | 0 | 4.35 | 10.8 | 5.83 | 15.6 |
| Pulmonary tuberculosis (010–011) | observed | ... | ... | 11 | ... | 3 |
| | % of all deaths | ... | ... | 3.05 | ... | 2.46 |
| Number in cohort | | 1540 | 152 | 1493 | 7289 | 162 |
| Number of males (females) | | no females | 152 (0) | 1265 (228) | 7289 (0) | 162 (0) |
| Mortality, % | | ... | 30.3 | 24.2 | 6.12 | 75 3 |
| Product and location | | asbestos works, Cardiff, Wales | insulators, New York State | asbestos company, U.S.A | insulators, southwest, midwest, and central U.S.A. | insulators, Belfast, N Ireland |
| Observation period | | 1936–1977 | 1945–1965 | 1940–1964 | 1967–1971 | 1940–1975 |
| Years exposed | | >0.5 | >15 | varies | not reported | varies |
| Years since first exposure | | >15 | >15 | varies | not reported | varies |
| Controls | | death rates, southeastern Wales | U.S. national rates, 1948 | internal, <2 years | U.S. national death rates | male rates, N Ireland |
| Smoking data | | minimal | minimal | not reported | not reported | 5 nonsmokers |
| Exposure data | | some | not reported | not reported | not reported | not reported |
| Asbestos type | | chrysotile >1936, crocidolite + chrysotile <1936 | not reported | not reported | chrysotile, amosite | not reported |

[a]International Classification of Diseases, 8th Revision (ICD.8).
[b]Subcohort of Cohort XVII (Table 3*b*).
[c]Subcohort of Cohort VII.
[d]Within the United States, mesothelioma mortality unrelated to asbestos exposure accounted for approximately 1 death in 10 000 in 1972 (estimated from data given by McDonald and McDonald [7]).
[f]Asbestosis is rarely caused by mineral dusts other than commercial asbestos.



10002286

TABLE 3a—*Continued.*

| Cause of Death (Classification)[a] | Number of Deaths | Study VI [22] | Study VII [23] | Study VIII[c] [23, 24] | Study IX [26] | Study X [27] | Study XI [28] |
|---|---|---|---|---|---|---|---|
| All causes (000–999) | observed (expected) | 66 (108.76) | 210 (191.31) | 186 (167.62) | 274 (188.19) | 56 | 781 (648.67) |
| | ratio observed/expected | 0.61 | 1.10 | 1.11 | 1.46 | ... | 1.20 |
| Cancer—all sites (140–239) or (140–209) | observed (expected) | 13 (17.41) | 59 (51.32) | 50 (42.77) | 99 (39.93) | 23 | 173 (108.81) |
| | ratio observed/expected | 0.75 | 1.15 | 1.17 | 2.48 | ... | 1.59 |
| | % of all deaths | 19.7 | 28.1 | 18.6 | 36.1 | ... | 22.2 |
| Cancer—lung, trachea, bronchus (162) | observed (expected) | 4 (4.32) | 31 (19.30) | 29 (18.38) | 35 (12.53) | 8 | 61 (23.30) |
| | ratio observed/expected | 0.93 | 1.61 | 1.58 | 2.79 | ... | 2.62 |
| | % of all deaths | 6.06 | 14.8 | 15.6 | 12.8 | 14.3 | 7.81 |
| Mesothelioma—peritoneum, pleura (158, 163) | observed (expected)[d] | 0 | 5 | 4 | 26 | 9 | 2 |
| | % of all deaths | 0 | 2.38 | 2.15 | 9.49 | 16.1 | 0.26 |
| Cancer—digestive system (150–159) | observed (expected) | ... | ... | ... | ... | ... | 55 (39.91) |
| | ratio observed/expected | ... | ... | ... | ... | ... | 1.38 |
| | % of all deaths | ... | ... | ... | ... | ... | 7.04 |
| Cancer—gastrointestinal tract (150–154) | observed (expected) | 4 (3.82) | ... | ... | 15 (7.99) | 3 | ... |
| | ratio observed/expected | 1.04 | ... | ... | 1.88 | ... | ... |
| | % of all deaths | 6.06 | ... | ... | 5.47 | 5.36 | ... |
| Respiratory disease (460–519) | observed (expected) | ... | 35 (25.02) | 31 (23.22) | 42 (12.16) | ... | 68 (39.31) |
| | ratio observed/expected | ... | 1.40 | 1.34 | 3.45 | ... | 1.73 |
| | % of deaths | ... | 16.7 | 16.7 | 15.3 | ..., | 8.71 |
| Noninfectious respiratory disease (510–519) | observed (expected) | ... | ... | ... | ... | 4 | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | 7.14 | ... |
| Asbestosis (515.2) | observed (expected)[f] | 2 | ... | ... | 35 | 2 | 19 |
| | % of all deaths | 3.03 | ... | ... | 12.8 | 3.57 | 2.43 |

Pulmonary observed

10002287

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| (150–154) | % of all deaths | ... | ... | ... | ... | ... | 7.04 |
| Cancer—gastrointestinal tract (150–154) | observed (expected) | 4 (3.82) | ... | ... | 15 (7.99) | 3 | ... |
| | ratio observed/expected | 1.04 | ... | ... | 1.88 | ... | ... |
| | % of all deaths | 6.06 | ... | ... | 5.47 | 5.36 | ... |
| Respiratory disease (460–519) | observed (expected) | ... | 35 (25.02) | 31 (23.22) | 42 (12.16) | ... | 68 (39.31) |
| | ratio observed/expected | ... | 1.40 | 1.34 | 3.45 | ... | 1.73 |
| | % of deaths | ... | 16.7 | 16.7 | 15.3 | ... | 8.71 |
| Noninfectious respiratory disease (510–519) | observed (expected) | ... | ... | ... | ... | 4 | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | 7.14 | ... |
| Asbestosis (515.2) | observed (expected)[f] | 2 | ... | ... | 35 | 2 | 19 |
| | % of all deaths | 3.03 | ... | ... | 12.8 | 3.57 | 2.43 |
| Pulmonary tuberculosis (010–011) | observed | ... | ... | ... | ... | 3 | ... |
| | % of all deaths | ... | ... | ... | ... | 5.36 | ... |
| Number in cohort | | 264 | 963 | 679 | 689 | 176 | 1075 |
| Number of males (females) | | 264 (0) | 679 (284) | 679 (0) | not reported | 93 (83) | 1075 (0) |
| Mortality, % | | 24.2 | 21.8 | 27.4 | 39.8 | 31.8 | 72.7 |
| Product and location | | asbestos factory | asbestos textile factory, U.K. | asbestos textile factory, U.K. | asbestos products factory, U.S.A. | assemblers, gas mask with asbestos filter pads | asbestos company, U.S.A. |
| Observation period | | 1945–1974 | 1933–1974 | 1933–1974 | 1959–1975 | 1945–1975 | 1941–1973 |
| Years exposed | | >1 | >10 | >10 | varies | 1 to 2 | 3 to 51 |
| Years since first exposure | | varies | >10 | >10 | varies | varies | varies |
| Controls | | U.S. national death rates | national death rates, U.K. | national death rates, U.K. | not reported | not reported | U.S. national rates |
| Smoking data | | not reported | not reported | not reported | not reported | not reported | not reported |
| Exposure data | | <2 fibers/cm³ since 1972 | yes (1952–1974) | 3 to 15 fibers/cm³ (1938–1972) | not reported | light exposure | yes |
| Asbestos type | | chrysotile only | chrysotile, crocidolite | chrysotile, some crocidolite | not reported | mostly crocidolite, some chrysotile | chrysotile, amosite, crocidolite |

[a]International Classification of Diseases, 8th Revision (ICD.8).
[b]Subcohort of Cohort XVII (Table 3*b*).
[c]Subcohort of Cohort VII.
[d]Within the United States, mesothelioma mortality unrelated to asbestos exposure accounted for approximately 1 death in 10 000 in 1972 (estimated from data given by McDonald and McDonald [7]).
[e]Asbestosis is rarely caused by mineral dusts other than commercial asbestos.

10002288

TABLE 3b—*Cohort mortality studies of asbestos trades workers, excluding miners and millers (see Table 4). The most recent update of Studies XII through XXI are given (1979–1981).*

| Cause of Death (Classification)[a] | Number of Deaths | Study XII [29] | Study XIII [30] | Study XIV [31] | Study XV[b] [32] | Study XVI[b] [32] |
|---|---|---|---|---|---|---|
| All causes (000–999) | observed (expected) | 19 (11.42) | 745 (556.0) | 601 (890.1) | 478 (328.9) | 62 (64.6) |
| | ratio observed/expected | 1.66 | 1.34 | 0.68 | 1.45 | 0.96 |
| Cancer—all sites (140–239) or (140–209) | observed (expected) | 6 (3.19) | 325 (138.41) | 120 (156.6) | 210 (57.0) | 20 (10.1) |
| | ratio observed/expected | 1.88 | 2.35 | 0.77 | 3.68 | 1.98 |
| | % of all deaths | 31.6 | 43.6 | 20.0 | 43.9 | 32.3 |
| Cancer—lung, trachea, bronchus (162) | observed (expected) | 5 (0.94) | 130 (46.41) | 51 (49.2) | 93 (13.3) | 10 (2.9) |
| | ratio observed/expected | 5.32 | 2.80 | 1.04 | 6.99 | 3.45 |
| | % of all deaths | 26.3 | 17.4 | 8.49 | 19.5 | 16.1 |
| Mesothelioma—peritoneum, pleura (158, 163) | observed (expected)[c] | none reported | 67 | 0 | 38 | 3 |
| | % of all deaths | none reported | 8.99 | 0 | 7.95 | 4.84 |
| Cancer—digestive system (150–159) | observed (expected) | ... | 60 (44.2) | 25 (50.1) | ... | ... |
| | ratio observed/expected | ... | 1.36 | 0.50 | ... | ... |
| | % of all deaths | ... | 8.05 | 4.16 | ... | ... |
| Cancer—gastrointestinal tract (150–154) | observed (expected) | ... | ... | ... | 43 (15.1) | 3 (1.5) |
| | ratio observed/expected | ... | ... | ... | 2.85 | 2.00 |
| | % of all deaths | ... | ... | ... | 9.00 | 4.84 |
| Respiratory disease (460–519) | observed (expected) | 10 (1.24) | 102 (78.0) | ... | ... | ... |
| | ratio observed/expected | 8.06 | 1.31 | ... | ... | ... |
| | % of all deaths | 52.6 | 13.7 | ... | ... | ... |
| Noninfectious respiratory disease (510–519) | observed (expected) | ... | ... | ... | 45 (9.3) | 7 (1.1) |
| | ratio observed/expected | ... | ... | ... | 4.84 | 6.34 |
| | % of all deaths | ... | ... | ... | 9.41 | 11.3 |
| Asbestosis (515.2) | observed (expected)[c] | ... | ... | ... | 41 | 6 |
| | % of all deaths | ... | ... | ... | 8.58 | 9.68 |

10002289

TABLE 3*b*—*Continued.*

| Cause of Death (Classification)[a] | Number of Deaths | Study XVII [32] | Study XVIII[b] [33] | Study XIX [34] | Study XX [35] | Study XXI [36] |
|---|---|---|---|---|---|---|
| All causes (000–999) | observed (expected) | 2 271 (1 658.9) | 79 (75.2) | 304 (158.6) | not given | 13 |
| | ratio observed/expected | 1.37 | 1.05 | 1.92 | not given | ... |
| Cancer—all sites (140–239) or (140–209) | observed (expected) | 995 (319.7) | 39 (16.2) | 116 (33.4) | 167 (151.13) | 7 |
| | ratio observed/expected | 3.11 | 2.41 | 3.47 | 1.11 | ... |
| | % of all deaths | 43.8 | 49.4 | 38.2 | ... | 53.8 |
| Cancer—lung, trachea, bronchus (162) | observed (expected) | 486 (105.6) | 21 (5.7) | 60 (10.1) | 44 (27.31)[c] | 3 (0.21) |
| | ratio observed/expected | 4.60 | 3.68 | 5.94 | 1.61 | 14.29 |
| | % of all deaths | 21.4 | 26.6 | 19.7 | ... | 23.1 |
| Mesothelioma—peritoneum, pleura (158, 163) | observed (expected)[d] | 175 | 8 | 14 | 3 | 2 |
| | % of all deaths | 7.71 | 10.1 | 4.61 | ... | 15.4 |
| Cancer—digestive system (150–159) | observed (expected) | ... | ... | ... | ... | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | ... |
| Cancer—gastrointestinal tract (150–154) | observed (expected) | 99 (59.4) | 3 (3.1) | 16 (8.0) | 31 (29.87) | 1 |
| | ratio observed/expected | 1.67 | 0.97 | 2.00 | 1.04 | ... |
| | % of all deaths | 4.36 | 3.80 | 5.26 | ... | 7.69 |
| Respiratory disease (460–519) | observed (expected) | ... | ... | ... | ... | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | ... |
| Noninfectious respiratory disease (510–519) | observed (expected) | 212 (59.0) | 14 (3.2) | 24 (4.7) | ... | ... |
| | ratio observed/expected | 3.59 | 4.38 | 5.11 | ... | ... |
| | % of all deaths | 9.34 | 17.7 | 7.89 | ... | ... |

10002290

| | | | | | | |
|---|---|---|---|---|---|---|
| Cancer— digestive system (150–159) | observed (expected) | ... | ... | ... | ... | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | ... |
| Cancer— gastrointestinal tract (150–154) | observed (expected) | 99 (59.4) | 3 (3.1) | 16 (8.0) | 31 (29.87) | 1 |
| | ratio observed/expected | 1.67 | 0.97 | 2.00 | 1.04 | ... |
| | % of all deaths | 4.36 | 3.80 | 5.26 | ... | 7.69 |
| Respiratory disease (460–519) | observed (expected) | ... | ... | ... | ... | ... |
| | ratio observed/expected | ... | ... | ... | ... | ... |
| | % of all deaths | ... | ... | ... | ... | ... |
| Noninfectious respiratory disease (510–519) | observed (expected) | 212 (59.0) | 14 (3.2) | 24 (4.7) | ... | ... |
| | ratio observed/expected | 3.59 | 4.38 | 5.11 | ... | ... |
| | % of all deaths | 9.34 | 17.7 | 7.89 | ... | ... |
| Asbestosis (515.2) | observed (expected)[f] | 168 | 13 | 18 | ... | ... |
| | % of all deaths | 7.40 | 16.4 | 5.92 | ... | .. |
| Number in cohort | | 17 800 | 440 | 582 | 5 686 | 20 |
| Number of males (females) | | 17 800 (0) | 440 (0) | 582 (0) | 5 686 (0) | 20 (0) |
| Mortality, % | | 12.8 | 18.0 | 52.2 | ... | 65.0 |
| Product and location | | North American asbestos workers | insulators, U.S. shipyards | amosite textile factory, N.J. | asbestos cement factory, Denmark | construction, 1928–1929, Norway |
| Observation period | | 1967–1976 | 1967–1976 | 1961–1977 | 1944–1976 | 1943–1980 |
| Years exposed | | varies | varies | varies | varies | 1 |
| Years since first exposure | | varies | varies | >20 | varies | varies |
| Controls | | U.S. death rates | U.S. death rates | death rates, N.J. smoking | death rates, Danish provincial towns | death rates, Norway |
| Smoking data | | not reported | not reported | yes | yes | yes |
| Exposure data | | not reported | not reported | heavy | yes | heavy |
| Asbestos type | | not reported | not reported | amosite, some chrysotile | chrysotile, amosite, some crocidolite | crocidolite only |

[a] International Classification of Diseases, 8th Revision (ICD.8).
[b] Subcohort of Cohort XVII.
[c] Excludes the trachea.
[d] Within the United States, mesothelioma mortality unrelated to asbestos exposure accounted for approximately 1 death in 10 000 in 1972 (estimated from data given by McDonald and McDonald [7]).
[e] Asbestosis is rarely caused by mineral dusts other than commercial asbestos.

10002291

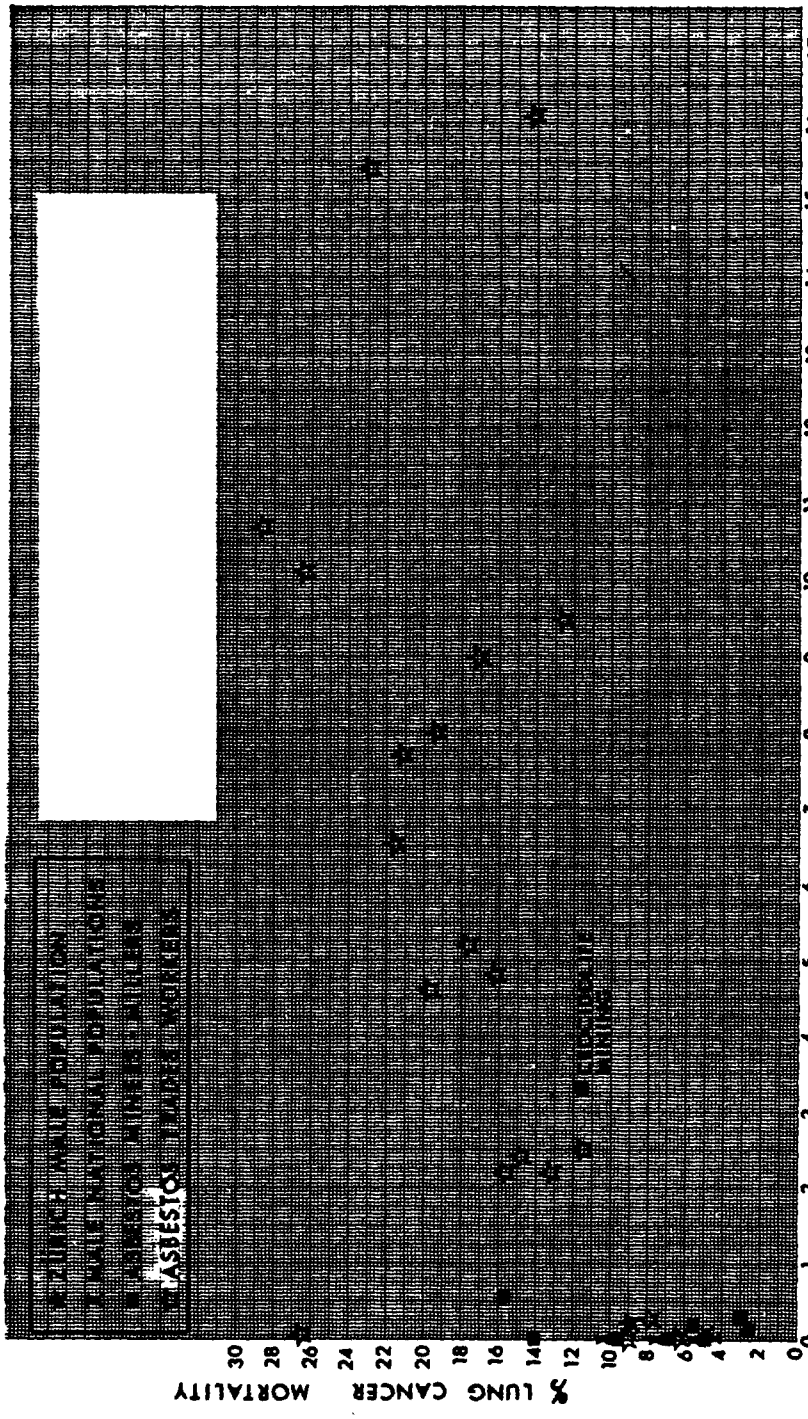


FIG. 1—*Graphical comparison of the proportional mortality due to lung cancer and mesothelioma for asbestos trades workers (Tables 3a and 3b, Studies I through XXI), asbestos miners and millers (Table 4, Studies A through J), the male population of the Zurich area of Switzerland, and the male populations of five nations (during 1969 or 1970) (Table 6).*

10002292

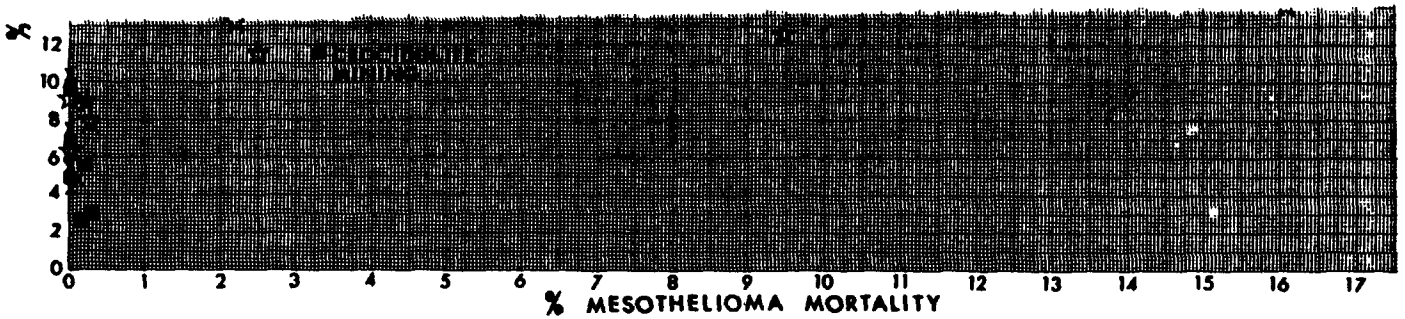


FIG. 1—*Graphical comparison of the proportional mortality due to lung cancer and mesothelioma for asbestos trades workers (Tables 3a and 3b, Studies I through XXI), asbestos miners and millers (Table 4, Studies A through J), the male population of the Zurich area of Switzerland, and the male populations of five nations (during 1969 or 1970) (Table 6).*

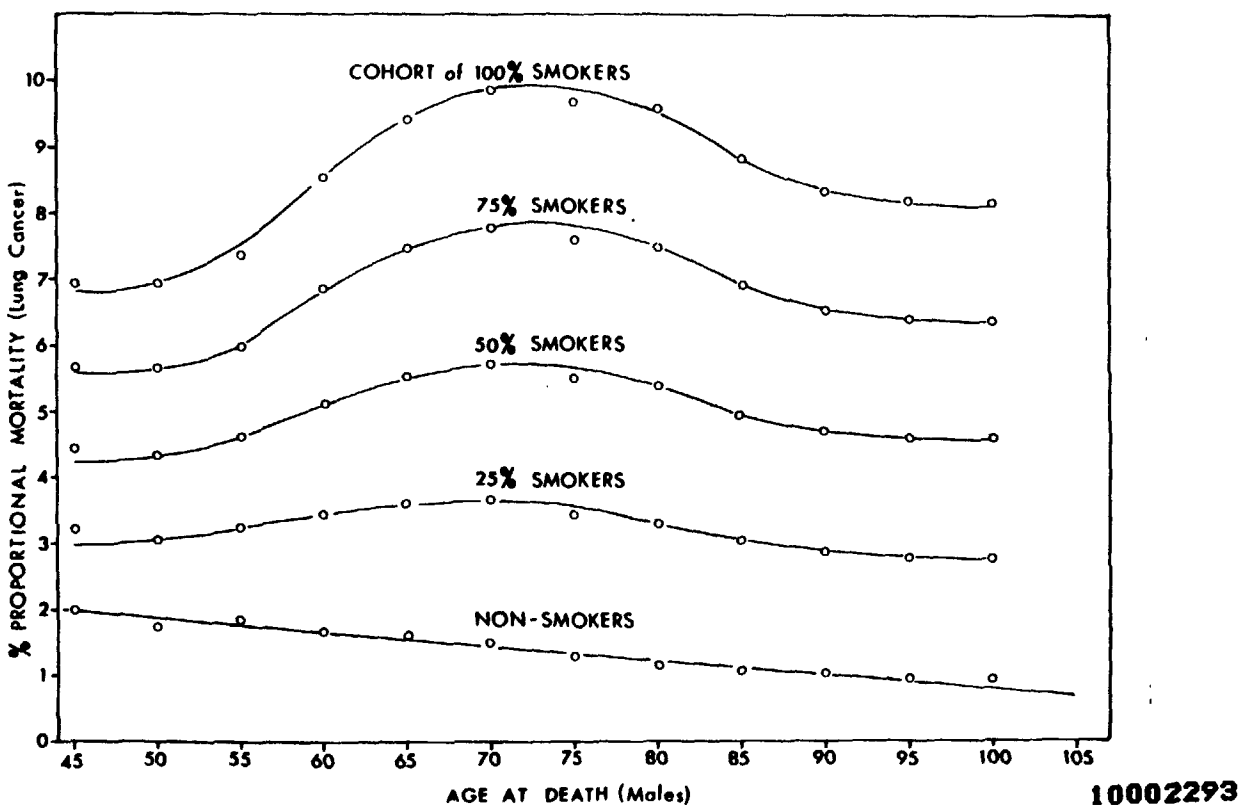


FIG. 2—*Graphical depiction of the percentage of lung cancer deaths in males in relation to male deaths from all causes (proportional mortality for lung cancer), plotted with respect to age for four groups (cohorts) with different cigarette-smoking characteristics and a cohort of nonsmokers. For example, for a cohort of 70-year-old males composed of 75% cigarette smokers, 7.5% of all deaths at age 70 are predicted to be from lung cancer. The graph is based on data from Hammond et al [37].*

these minerals can coexist in metamorphosed ultramafic rocks, for example, those in Paakkila, Finland.

Epidemiological studies of asbestos miners and millers exposed to only one form of asbestos are useful in understanding 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 [*8–10,43–48*]. 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 scientists 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, 14 March 1975); in the latter case, the U.S. Environmental Protection Agency (EPA) sued to prevent the Reserve Mining Co. 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; and 256 *Northwestern Reporter*, 2d Series, 808–852, 1977. Of interest in relation to this suit are the health studies of the Reserve Mining Company's iron ore miners exposed to cummingtonite and grunerite in the taconite rock (Table 4, Study B) and of the Homestake, N. Dak., gold miners exposed to cummingtonite in the gold-bearing schists (Table 4, Studies D and E).[5] Studies B and E show no evidence of asbestos-related diseases appearing in the study groups.

*Mortality Comparisons, Trades Versus Mines*—The cancer mortality pattern for those in the asbestos trades and mining occupations is graphically presented in Fig. 1, where the percentage of lung cancer mortality is plotted in relation to the percentage of mortality due to mesothelioma. The studies of the asbestos trades workers (Tables 3*a* and 3*b* and Fig. 1, *open stars*) show a very significant excess of mortality due to mesothelioma in comparison with that found in the miners (Table 4 and 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

[5] Study E [*46*] discredits Study D [*45*]. Study D was made on a subcohort of the Study E cohort. The latter study (Study E) is much more complete; statistically 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 silicotuberculosis 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% 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.

10002294

iltramafic rocks, for example,

id millers exposed to only one
the different asbestos minerals
data for the five major epide-
rs [*8–10,43–48*]. In addition,
nmingtonite and grunerite am-
posed to hornblende amphibole
as asbestos, even though they
to be valuable commercially.
f taconite mining by the courts
F. Supp. 11) and by the U.S.
ning vs. EPA, U.S. Court of
latter case, the U.S. Environ-
the Reserve Mining Co. from
use of the perception that these
stitute a threat to public health.
*ral Reporter*, 2d Series, 492–
d Series, 808–852, 1977. Of
tudies of the Reserve Mining
gtonite and grunerite in the ta-
estake, N. Dak., gold miners
hists (Table 4, Studies D and
stos-related diseases appearing

—The cancer mortality pattern
ations is graphically presented
ortality is plotted in relation to
a. The studies of the asbestos
*stars*) show a very significant
parison with that found in the
one exception, the crocidolite

important to note two health
World War II, were employed

on a subcohort of the Study E cohort.
ly it is based on 631 deaths, whereas
s an implausible mortality pattern: (1)
were reported, despite the fact that the
r a century, and (2) a 14.1% respiratory
e a sinus and a mediastinal carcinoma)
y due to asbestosis was reported. The
ock is not asbestos, but rather, garden-



at the task of manufacturing asbestos-bearing filter pads and placing them into gas mask canisters. One study [*27*] 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 through 1941 was no more than two and a half years. The cohort studied was composed of 93 men and 83 women (Table 3*a*, Study X). Of the 56 now dead, 8 died of lung cancer (14.3%) and 9 of mesothelioma (16.1%).

The second study [*49,50*] 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 through 1940 assembling "civilian filter pads" that contained chrysotile asbestos. The Birmingham group worked for up to four and a half years during the period 1940 through 1944 assembling "military filter pads" that contained crocidolite asbestos. The crocidolite was thought to have come from Wittenoom, Western Australia. The mortality data are 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. The dust levels, as with the Canadian gas mask workers, were considered to be very light to moderate.

To make further comparisons, it is useful to examine the mortality due to lung cancer and mesothelioma in national populations. In Table 6 are given the lung cancer and mesothelioma mortality of all males over 24 years of age in five nations during 1969 or 1970. These data are plotted in Fig. 1. The average lung cancer mortality of these five national populations was 5.7%, a figure identical to the 5.7% average mortality of miners and tunnel workers (Table 4, excluding crocidolite miners). The mesothelioma mortality of the five national populations was 0.03% (Table 6) and was probably significantly underreported because of (1) the great difficulty in diagnosing this disease, even after autopsy [*7,16,51–53*], and (2) complications arising in coding this disease properly and consistently 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 [*54*] of the deaths in the Zurich 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) between 1961 and 1976, 51 deaths were due to mesothelioma (0.18%) and 2466 were due to lung cancer (8.8%). Among female deaths (22 583 in the same time period), 23 were caused by mesothelioma (0.10%) and 368 caused by lung cancer (1.6%). The proportional mesothelioma mortality for hard rock miners, tunnel workers, and asbestos miners (other than crocidolite asbestos) is

10002295

TABLE 4—*Mortality from selected causes in the principal epidemiological and tunnel workers exposed to rock dust containing*

| Cause of Death (Classification)[a] | Number of Deaths | Study A (900 Men, 1936–1967) |
|---|---|---|
| All causes (000–999) | observed | 216 |
| | expected | |
| | ratio observed/expected | |
| Respiratory cancer (162) | observed | 21 |
| | % of all deaths | 9.7 |
| | expected | 12.6 |
| | ratio observed/expected | 1.67 |
| Mesothelioma, peritoneum, pleura (158, 163) | observed | 0 |
| | % of all deaths | 0 |
| Gastrointestinal tract cancer (150–154) or (150-159) | observed | 7 |
| | % of all deaths | 3.2 |
| | expected | 14.9 |
| | ratio observed/expected | 0.47 |
| Pneumoconiosis (500–519) | observed | ... |
| | % of all deaths | ... |
| Asbestosis (515.2) | observed | 13 |
| Silicosis (515.0) | observed | ... |
| Respiratory tuberculosis (010–011) | observed | 36 |
| | % of all deaths | 16.7 |
| Locality | | North Savo, Finland |
| Type of mining | | asbestos |
| Type of rock | | ultramafic |
| Suspected mineral pathogen | | anthophyllite asbestos |
| Source | | [8] |

[a]Cummingtonite, grunerite, and hornblende (Studies B, C, D, and E) may be defined as asbestos in United States federal regulations.

[b]International Classification of Diseases, 8th Revision (ICD.8).

[c]Includes one carcinoma of the maxillary sinus and one mediastinal carcinoma (unspecified). See Footnote 5 in text.

[d]Two mesothelioma victims worked with crocidolite in addition to chrysotile.

10002296

*causes in the principal epidemiological workers exposed to rock dust containing studies of commercial asbestos miners and millers and other hard rock miners minerals sometimes defined as asbestos.*[a]

| | Study A (900 Men, 1936–1967) | Study B (5751 Men, 1952–1976) | Study C (932 Men, 1955–1972) | Study D (440 Men, 1960–1973) | Study E (1321 Men, 1937–1973) |
|---|---|---|---|---|---|
| | 216 | 298 | 294 | 71 | 631 |
| | | 344 | 225 | 52.9 | 549.7 |
| ected | | 0.87 | 1.30 | 1.34 | 1.15 |
| | 21 | 15 | 21 | 10 | 16 |
| | 9.7 | 5.0 | 7.1 | 14.1[c] | 2.5 |
| | 12.6 | 17.9 | 13.15 | 2.7 | 16.5 |
| ected | 1.67 | 0.84 | 1.60 | 3.0 | 0.97 |
| | 0 | 0 | ... | 0 | 1? |
| | 0 | 0 | ... | 0 | 0.16? |
| | 7 | 20 | 10 | ... | 39 |
| | 3.2 | 6.7 | 3.4 | ... | 6.2 |
| | 14.9 | 17.6 | 11.13 | ... | 35.1 |
| ected | 0.47 | 1.14 | 0.90 | ... | 1.11 |
| | ... | 4[d] | 20 | 5 | 37 |
| | ... | 1.3 | 6.8 | 7.0 | 5.9 |
| | 13 | ... | ... | ... | ... |
| | ... | ... | ... | ... | 35 |
| | 36 | ... | 11 | ... | 39 |
| | 16.7 | ... | 3.7 | ... | 6.2 |
| | North Savo, Finland | Minnesota | Manhattan Island, N.Y. | Lead, N.D. | Lead, N.D. |
| | asbestos | iron ore | tunneling | gold | gold |
| | ultramafic | taconite | schist, gneiss, amphibolite | quartz-cummingtonite schist | quartz-cummingtonite schist |
| | anthophyllite asbestos | cummingtonite, grunerite, quartz | hornblende, quartz | cummingtonite, hornblende, quartz | cummingtonite, hornblende, quartz |
| | [8] | [43] | [44] | [45] | [46] |

. D, and E) may be defined as asbestos

:D.8).
ediastinal carcinoma (unspecified). See

ddition to chrysotile.

[c]"Selected respiratory disease."
[d]Pneumoconiosis Board records (Western Australia) show pneumoconiosis of mixed type, asbestosis, silico-asbestosis, and silicosis.
[e]Pneumoconiosis is probably predominantly asbestosis, since rock dust contains little crystalline silica (quartz).

10002297

TABLE 4—

| Cause of Death (Classification)[b] | Number of Deaths | Study F (Number Unknown, 1943–1977) |
|---|---|---|
| All causes (000–999) | observed | 519 |
| | expected | 600.3 |
| | ratio observed/expected | 0.86 |
| Respiratory cancer (162) | observed | 60 |
| | % of all deaths | 11.6 |
| | expected | 38.9 |
| | ratio observed/expected | 1.54 |
| Mesothelioma, peritoneum, pleura (158, 163) | observed | 17 |
| | % of all deaths | 3.3 |
| Gastrointestinal tract cancer (150–154) or (150–159) | observed | ... |
| | % of all deaths | ... |
| | expected | ... |
| | ratio observed/expected | ... |
| Pneumoconiosis (500–519) | observed | 21[f] |
| | % of all deaths | 4.0 |
| Asbestosis (515.2) | observed | ... |
| Silicosis (515.0) | observed | ... |
| Respiratory tuberculosis (010–011) | observed | 4 |
| | % of all deaths | 0.77 |
| Locality | | Wittenoom, Western Australia |
| Type of mining | | asbestos |
| Type of rock | | banded ironstone |
| Suspected mineral pathogen | | crocidolite asbestos, quartz |
| Source | | [47] |

[a]Cummingtonite, grunerite, and hornblende (Studies B, C, D, and E) may be defined as asbestos in United States federal regulations.

[b]International Classification of Diseases, 8th Revision (ICD.8).

[c]Includes one carcinoma of the maxillary sinus and one mediastinal carcinoma (unspecified). See Footnote 5 in text.

[d]Two mesothelioma victims worked with crocidolite in addition to chrysotile.

10002298

TABLE 4—

*Continued.*

| | Study F (Number Unknown, 1943–1977) | Study G (933 Men, 1946–1975) | Study H (544 Men, 1961–1977) | Study J (10 939 Men, 1910–1975) | Totals (Excluding Study F) |
|---|---|---|---|---|---|
| | 519 | 332 | 178 | 4463 | 6483 |
| | 600.3 | 214.4 | 159.9 | ... | ... |
| ected | 0.86 | 1.55 | 1.11 | ... | ... |
| | 60 | 10 | 28 | 250 | 371 |
| | 11.6 | 3.0 | 15.7 | 5.6 | 5.7 |
| | 38.9 | 10.4 | 11.1 | ... | ... |
| ected | 1.54 | 0.96 | 2.5 | ... | ... |
| | 17 | 1? | 1 | 10[e] | 11 + 2? |
| | 3.3 | 0.30? | 0.56 | 0.22 | 0.17 + 0.03? |
| | ... | 19 | 10 | 168 | 273 |
| | ... | 5.7 | 5.6 | 3.8 | 4.2 |
| | ... | 19.3 | 9.5 | ... | ... |
| ected | ... | 0.98 | 1.05 | ... | ... |
| | 21[f] | 20 | 30 | 46[e] | ... |
| | 4.0 | 6.0 | 16.9 | 1.0 | ... |
| | ... | 9 | 26 | ... | ... |
| | ... | ... | ... | ... | ... |
| | 4 | 18 | ... | 248 | ... |
| | 0.77 | 5.4 | ... | 5.6 | ... |
| | Wittenoom, Western Australia | Balangero, Italy | Quebec Province, Canada | Quebec Province, Canada | |
| | asbestos | asbestos | asbestos | asbestos | |
| | banded ironstone | serpentinite | serpentinite | serpentinite | |
| | crocidolite asbestos, quartz | chrysotile asbestos | chrysotile asbestos | chrysotile asbestos | |
| | [47] | [9] | [10] | [48] | |

. D, and E) may be defined as asbestos

D.8).

:diastinal carcinoma (unspecified). See

dition to chrysotile.

"Selected respiratory disease."

[f]Pneumoconiosis Board records (Western Australia) show pneumoconiosis of mixed type, asbestosis, silico-asbestosis, and silicosis.

[e]Pneumoconiosis is probably predominantly asbestosis, since rock dust contains little crystalline silica (quartz).

10002299

TABLE 5—*Mortality data for a cohort of 951 women[a] (578 traced) who assembled asbestos-bearing filter pads in English factories during the years 1939–1944* [50].

| Cause of Death | Observed, No. | Mortality, % | Asbestos Exposure |
|---|---|---|---|
| All causes | 166 | 100 | crocidolite and chrysotile |
| Gastrointestinal tract cancer | 10 | 6.0 | ... |
| Other cancer | 35 | 21.1 | ... |
| Mesothelioma | 16 | 10.2 | crocidolite only |
| | 1 | | crocidolite and chrysotile |
| Lung cancer | 11 | 7.2 | crocidolite only |
| | 1 | | crocidolite and chrysotile |

[a]Of the 951, 727 (139 dead) had been exposed only to crocidolite, 102 (10 dead) only to chrysotile, 99 (15 dead) to crocidolite plus chrysotile, and 23 (2 dead) to unknown exposure.

0.17 to 0.20% (Table 4). The asbestos trades workers, in contrast, have an average mesothelioma mortality of 5.6% (Tables 3*a* and 3*b*).

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 significant excess mortality due to lung cancer and asbestosis—but not to mesothelioma (Table 4, Studies A and H). The most detailed health study of asbestos miners to date is that of the chrysotile asbestos miners of Quebec Province, Canada (Table 4, Study J). Here, McDonald et al [*48*] 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 were low level, 2.5 to 4.2 millions of particles per cubic foot (mp/ft$^3$);[6] medium

TABLE 6—*Cancer mortality in men over 24 years of age for five nations during 1969 or 1970* [16].

| Nation | All Deaths, No. (year) | Lung Cancer, No. (%)[a] | Mesothelioma, No. (%)[a] |
|---|---|---|---|
| England/Wales | 278 617 (1970) | 24 913 (8.9) | 154 (0.06) |
| Finland | 22 332 (1970) | 1 586 (7.1) | 8 (0.04) |
| Italy | 272 795 (1970) | 11 867 (4.7) | not reported |
| United States | 988 620 (1969) | 50 481 (5.1) | 250 (0.03) |
| Canada | 82 052 (1970) | 4 312 (5.3) | 25 (0.03) |
| Total | 1 624 416 | 93 159 (5.7) | 437 (0.03) |

[a]Percentage of all deaths (proportional mortality).

[6] mp/ft$^3$ = millions of particles of rock dust per cubic foot. Conversion of this measurement unit into asbestos fibers per cubic centimetre, the usual measurement for industrial hygiene monitoring, is difficult, but an approximate and conservatively small conversion factor is 1 mp/ft$^3$ = 3 fibers/cm$^3$ [*48,56*].

10002300

*(578 traced) who assembled asbestos- e the years 1939–1944* [50].

| ility, % | Asbestos Exposure |
|---|---|
| 0 | crocidolite and chrysotile |
| 6.0 | ... |
| 21.1 | ... |
| 0.2 | crocidolite only |
| | crocidolite and chrysotile |
| 7.2 | crocidolite only |
| | crocidolite and chrysotile |

ocidolite, 102 (10 dead) only to chrysotile, d) to unknown exposure.

es workers, in contrast, have an les 3*a* and 3*b*).

is no question that those exposed nophyllite dust over long periods ality due to lung cancer and as- udies A and H). The most detailed of the chrysotile asbestos miners J). Here, McDonald et al [*48*] tween lung cancer incidence and oncentrations that the miners and ere divided into four categories, their careers in the mines. These per cubic foot (mp/ft³);[6] medium

*irs of age for five nations during* ].

| Lung Cancer, No. (%)* | Mesothelioma, No. (%)* |
|---|---|
| 24 913 (8.9) | 154 (0.06) |
| 1 586 (7.1) | 8 (0.04) |
| 11 867 (4.7) | not reported |
| 50 481 (5.1) | 250 (0.03) |
| 4 312 (5.3) | 25 (0.03) |
| 93 159 (5.7) | 437 (0.03) |

oot. Conversion of this measurement unit ement for industrial hygiene monitoring, conversion factor is 1 mp/ft³ = 3 fibers/



level, 4.3 to 9.4; high level, 14.4 to 23.6; and very high level, 46.8 to 82.6 The mean within these four categories in terms of chrysotile fibers per cubic centimetre was low, 10 fibers/cm³; medium, 21 fibers/cm³; high, 95 fibers/cm³, and very high, 194 fibers/cm³. For the men exposed for over 20 years (see Table 16) in the low and medium dust categories (averaging 6.6 mp/ft³ or approximately 20 fibers/cm³), the total mortality was less than expected [standardized mortality ratio (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/cm³ 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/cm³), chrysotile miners working a lifetime under the present dust levels are not expected to show any significant health problems in comparison with those in other mining industries [*55*].

McDonald et al [*48*] 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 1 death due to lung cancer and 1 due to mesothelioma.

*Crocidolite Exposure*—There are persuasive data, many already surveyed, that 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 Cape Province, South Africa, and at Wittenoom Gorge, Western Australia, have a statistically significant increase in mortality due to mesothelioma. Also, mesothelioma deaths have been reported among residents of these areas who are not employed in the mines or mills. For example, Webster [*57*] 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 backgrounds were 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 2 with Transvaal crocidolite mining. There were only 4 mesothelioma cases in those associated with amosite mining, and 2 of these had been exposed to Cape crocidolite as well. In the chrysotile mining industry there was only 1 case—a miner from Rhodesia. Of the 100 environmental cases (those not employed in any occupation in which asbestos is used), 93 of these individuals had been exposed to Cape crocidolite, 2 to Transvaal crocidolite, and 1 possibly to amosite.

Additional prevalence studies in Cape Province [*58*] revealed 65 active cases of mesothelioma in individuals 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 38 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

10002301

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, had hand-cobbed crocidolite asbestos.

In contrast to the prevalence of mesothelioma in Cape Province, this disease is very rare in the Transvaal where all of the world's amosite is mined. Wagner et al [*59*], in regard to their initial discovery of the association of crocidolite asbestos with mesothelioma, state that "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" (Ref *59*, p. 260).

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 [*60*], the Secretary of the Rhodesia Pneumoconiosis Board stated that no cases of mesothelioma had been reported in the mining industry. It is of interest to note that 2 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 had been exposed [*60*]. Cochrane and Webster [*61*] 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 Wittenoom Gorge in Western Australia has been discussed (Table 4, Study F). The town of Wittenoom, the center of crocidolite mining in Western Australia, reached a peak population of about 1000 in the 1960s. 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*, 8 Dec. 1978, p. 25). The risk of mesothelioma among residents of the town who were not employed by mines is demonstrated by the case of a 27-year-old woman who had had an environmental childhood exposure to crocidolite [*62*].

### *Effects of Nonoccupational Exposure to Asbestos*

It is difficult to assess the health effects of nonoccupational 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. In order to study the nonoccupationally 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 has carried asbestos dust back to the home on his or her clothing.

10002302

crocidolite asbestos mines. Lastly,
ey of 53 females who, in the past,

oma in Cape Province, this disease
world's amosite is mined. Wagner
y of the association of crocidolite
umour [referring to mesothelioma]
ca. During the past five years, with
ovince], no neoplasm of this nature
examined at the Pneumoconiosis
Department of the South African
260).
we (Rhodesia), a country which is
ther form of asbestos, is very low.
[60], the Secretary of the Rhodesia
f mesothelioma had been reported
that 2 cases of mesothelioma were
locomotive engineer and a store-
rocidolite asbestos to which these
nd Webster [61] report 12 cases of
s in the locomotive workshops of

he miners of Wittenoom Gorge in
4, Study F). The town of Witten-
estern Australia, reached a peak
present the population is down to
ernment has suggested the closing
ause of continuing risks of airborne
p. 25). The risk of mesothelioma
nployed by mines is demonstrated
d had an environmental childhood

*stos*

f nonoccupational exposure to as-
re levels are usually low, and any
ng association of this disease with
ccupationally exposed, epidemiol-
eased incidence of asbestosis and
horts: those who live in neighbor-
r mines and those who live within
sumably has carried asbestos dust



*Background Incidence of Mesothelioma*—There appears to be a definite "background" mortality due to mesothelioma that is not related to any known asbestos exposure. McDonald and McDonald [16], reviewed 4539 fatal mesothelioma cases reported from 22 countries between 1959 and 1976. They found that, for 923 of the 2453 cases for which a history had been recorded, a definite or probable exposure to asbestos could not be shown. More recently, Jones and Silver [63] reported 8 cases with no known environmental exposure; Brenner et al [64] report on 25 patients entering Memorial Hospital, New York City, since 1950, with no exposure history; Brenner et al [65] report on 7 children dying of mesothelioma who had had no history of exposure to asbestos; and Griffiths et al [66] 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 [25] in their report of 5 deaths due to mesothelioma within a single family: the father, 3 sons, and a daughter. Four of the 5 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 direct exposure to asbestos can be obtained from data given by McDonald and McDonald [7]. Determination, through 7400 pathologists, of all the fatal malignant mesothelioma tumors in Canada between 1960 and 1975 and in the United States in 1972 was made. The pathology review panel accepted 73 and 65% of the cases in the United States and Canada, respectively, as mesothelioma. The occupational histories indicate that 50% of the male and 5% of the female deaths could be attributed to known 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 have been accepted as mesothelioma. Assuming a 30% underreporting (Ref 7, p. 1655), the 1972 incidence in the United States was approximately 200 male and 56 female deaths. Of these, about 100 male and 3 female mesothelioma deaths can be attributed to known 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% (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 for the population of the United States. Other studies indicate that the Canadian mesothelioma death rates are very similar to those in the United States [7,67].

*Asbestos-Related Disease in Residential Areas*—The residents of areas in which 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 Cape Province in South Africa has already been discussed. The appearance of 93 mesothelioma deaths among individuals not occupationally ex-

10002303

posed to Cape crocidolite is in extreme contrast to the rarity of this disease in the amosite mining regions of Transvaal Province, where only 1 possible case has been reported [57].

Asbestos-related disease among residents of chrysotile mining areas is rare. Thériault and Grand-Bois [68] report the following mesothelioma mortalities in Quebec Province for 1969 through 1972: (1) asbestos-producing regions—observed 2, expected 1.3; (2) areas surrounding the asbestos-producing regions—observed 5, expected 4.8; (3) other rural regions of Quebec—observed 12, expected 32.4; (4) the city of Quebec—observed 7, expected 4.7; and (5) the city of Montreal—observed 42, expected 24.4).

McDonald [69] has reviewed all known fatal mesothelioma cases in Quebec Province for the period 1960 through 1978. Of the total of 254 cases registered, 181 were males and 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 had possibly been exposed to crocidolite in one of the mills, which for about two years had processed crocidolite fiber for the gas mask filters. In addition to these 21 cases there were 4 daughters and 2 sons of chrysotile miners or millers who died of mesothelioma. Apart from these cases, McDonald [69] reports only 2 persons who died of this disease who lived within 20 miles of the chrysotile mines and mills of Quebec Province.

Pampalon [70] 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 of them were employed in the asbestos industry. However, they did receive over much of their lives very heavy nonoccupational exposures to chrysotile asbestos contained in the rock dusts emitted from the operation of the nearby mines and mills [71,72].[7] The

[7] Even in 1974 and 1975, when the rock dust levels at the Quebec asbestos mines and mills had been reduced from levels as high as 200 mp/ft$^3$ (see Footnote 6 for metric conversion factor) in the early 1950s to less than 7 mp/ft$^3$ in the mid-1970s [41], 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 from April to September 1975 [71]. 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 Association, 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 [72] who analyzed the *chrysotile fiber* content in the air in houses located in two chrysotile mining districts (Copperopolis, Calif., and Baie Verte, Newfoundland). The 13 air samples taken in homes of crysotile miners and millers show the following chrysotile content: four samples, 50 to 100 ng/m$^3$; four samples, 100 to 200 ng/m$^3$; two samples, 200 to 500 ng/m$^3$; two samples, 500 to 1000 ng/m$^3$; and one sample, 2000 to 5000 ng/m$^3$. Three samples taken in houses of nonminers in Baie Verte gave concentrations of 32, 45, and 65 ng/m$^3$.

10002304

st to the rarity of this disease in
ince, where only 1 possible case

chrysotile mining areas is rare.
ving mesothelioma mortalities in
asbestos-producing regions—ob-
he asbestos-producing regions—
gions of Quebec—observed 12,
ved 7, expected 4.7; and (5) the
).
al mesothelioma cases in Quebec
the total of 254 cases registered,
al and residential histories were
women. About 40% of the male
uted to occupational exposure to
re individuals who at some time
l milling industry. But 5 of these
facturing filter pads for gas masks
idolite in one of the mills, which
fiber for the gas mask filters. In
rs and 2 sons of chrysotile miners
rom these cases, McDonald [69]
se who lived within 20 miles of
ince.
patterns of the Quebec asbestos
proximately 20 000) and Asbestos
rtality among the female residents
very few of them were employed
ive over much of their lives very
e asbestos contained in the rock
y mines and mills [71,72].[7] The

the Quebec asbestos mines and mills had
note 6 for metric conversion factor) in the
the rock dust in the ambient air over the
e mines and mills were in operation and
during the strike from April to September
veight of dust falling daily over the town
nds, and Brulotte (Air Pollution Control
of Thetford Mines and Black Lake show
onsiderable environmental exposure even
e the work of Nicholson et al [72] who
located in two chrysotile mining districts
13 air samples taken in homes of crysotile
it: four samples, 50 to 100 ng/m$^3$; four
m$^3$; two samples, 500 to 1000 ng/m$^3$; and
houses of nonminers in Baie Verte gave



mortality data for the female residents of Asbestos and Thetford Mines are given in Table 7. (See also Tables 8 and 9.)

Another cancer mortality survey of various regions in Quebec Province was made by Graham [73]. 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 those of the rural counties ("other rural" and "peripheral") and those of 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) life-style [74–80]. Graham [73] states that the rates for cancers of the pleura, peritoneum, lip, tongue, salivary gland, and small intestine in males and of the pleura, lip, kidney, salivary gland, and skin (melanoma) in females are in excess[8] in the asbestos mining counties. Graham further states that cancer of the colon, stomach, and lung are "at a level so low as to be unimpressive" (Ref 73, p. 40). These are cancers known to be in excess in many cohorts of asbestos trades workers (Tables 3a and 3b). The low cancer rates found in the asbestos mining localities are not surprising, for McDonald et al [48] report that, during the five decades 1926 through 1975, 4350 male Quebec asbestos miners and millers died in comparison with the 4107 expected on the basis of age-and-year-specific death rates for Quebec Province, giving a standardized mortality ratio (SMR) of 1.06.

It should also be noted that the residents of Thetford Mines and Asbestos used drinking water that contained very high concentrations of chrysotile asbestos, ranging from 172 million to 1.3 billion fibers per litre [81]. No evidence of excess cancer mortality could be attributed to asbestos in the drinking water of these towns [81].[9]

Toft et al [86] expanded on the study of Wigle [81] by comparing mortality data from 71 municipalities with the amount of asbestos in the drinking water. Particularly informative are the death rates of the female residents of Thetford Mines in comparison with the rates found in women who lived in 52 other Canadian 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 nonoccupational exposure to chrysotile asbestos carried in the air and water. The death-rate comparisons (Table 9) indicate that the women of Thetford Mines have not been affected by exposure to chrysotile asbestos.

[8] A total of one to three deaths for these cancers was reported for the five-year period 1969 through 1973. The statistical significance of such small numbers is questioned.

[9] In regard to ingestion of asbestos and cancer incidence, a number of animal studies are now complete [82–85,109,110]. None of these studies shows any evidence that ingestion of asbestos causes tumors in animals. Hallenbeck et al state that "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" (Ref. 83, p. 349).

10002305

TABLE 7—*Mortality data for women living in Quebec asbestos mining towns for the period 1966–1977* [70].

| Cause of Death | Observed, No. | Expected, No. | Excess (Deficiency), No. |
|---|---|---|---|
| All causes | 1225 | 1356 | (131) |
| All cancer | 292 | 321 | (29) |
| Lung cancer | 23 | 23 | 0 |
| Gastrointestinal tract cancer | 97 | 91 | 6 |
| Respiratory disease | 35 | 58 | (23) |

Hammond et al [*87*] have given mortality statistics for the residents in the neighborhood of Riverside, N.J., which surrounds the Patterson, N.J., amosite asbestos factory. The mortality of the workers in this factory is given in Table 3*b*, Study XIX. The mortality data for Riverside are statistically indistinguishable from those 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 has occurred since.

*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 that included an asbestos worker. Epler et al [*88*] summarize 14 published reports of 43 such mesothelioma cases, plus 4 more found in their own study. Antman et al [*89*] mention 3 more, and Vianna and Polen [*90*] 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 caused 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 [*91*] (see also Ref 92) 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 11 Sept. 1978, Joseph A. Califano, then Secretary of the U.S. Department of Health, Education, and Welfare, gave a major speech at the American Federation of Labor/Congress of Industrial Organizations (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% of all cancer deaths in the United States each year for the next 30 to 35 years will be associated with previous exposure

10002306

*asbestos mining towns for the period*

| J. No. | Excess (Deficiency), No. |
|---|---|
| 6 | (131) |
| 1 | (29) |
| 3 | 0 |
| 1 | 6 |
| 58 | (23) |

statistics for the residents in the unds the Patterson, N.J., amosite s in this factory is given in Table le are statistically indistinguishable owa, N.J., situated several miles sothelioma death occurred in Riv-

*of Asbestos Workers*—There are a ig in individuals who, though not ouseholds that included an asbestos ed reports of 43 such mesothelioma Antman et al [*89*] mention 3 more, e patients who lived with husbands cupations. It is difficult, perhaps ds of asbestos caused the mesothe- s most probable that the workers report of Newhouse [*91*] (see also he reports that 11 individuals who d with asbestos nor had a relative he immediate vicinity of a factory

**s Due to Exposure to Asbestos**

n Secretary of the U.S. Department major speech at the American Fed- nizations (AFL-CIO) National Con- which he described how the federal d preventing occupational disease. cancer deaths in the United States associated with previous exposure



TABLE 8—*Cancer mortality rates[a] (deaths per 100,000 residents) for various geographic localities in Quebec Province and for upper New York State; the data are the annual average for the period 1969–1973* [73].

| Cancer Location | Asbestos-Mining Counties | Peripheral rural Counties | Other Rural Counties | Montreal | Quebec City | All Quebec Province | Upper New York 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 intestine | 1.11 | 0.58 | 0.32 | 0.68 | 0.95 | 0.64 | 0.78 |
| Large intestine | 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 |
| Total | 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 intestine | 0.38 | 0.68 | 0.32 | 0.65 | 1.16 | | |
| Large intestine | 18.75 | 17.74 | 15.36 | 22.64 | 21.89 | | |
| Rectum | 10.86 | 8.78 | 8.24 | 9.51 | 11.26 | | |
| Total | 45.11 | 40.36 | 35.58 | 51.17 | 53.2 | | |

[a]Only those cancers known to be associated with excess mortality in asbestos trades workers are listed.

10002307

TABLE 9—*Age-adjusted mortality rates (per 100 000 residents) in Canada for females age 25–69* [86].

| Cause of Death | Thetford Mines[a] | 52 Comparison Localities[b] |
|---|---|---|
| All causes | 420 | 433 |
| All cancer | 138 | 158 |
| Lung cancer | 8.5 | 13.4 |
| Gastrointestinal tract cancer | 42.2 | 41.5 |
| Respiratory system[c] | 8.9 | 16.9 |

[a]Treated water, 110 to 150 million chrysotile fibers per litre.
[b]All are localities that contained less than 5 million chrysotile fibers per litre of water.
[c]Nonneoplastic disease.

to asbestos. This translates into 67 000 cancer deaths per year due to asbestos [National Cancer Institute (NCI), National Institute of Environmental Health Sciences (NIEHS) press release, *Draft Summary*, 11 Sept. 1978]. The Califano speech was based on an unpublished document [*93*] prepared by several medical scientists at three of the National Institutes of Health [NCI, NIEHS, and the National Institute for Occupational Safety and Health (NIOSH)]. Doll and Peto [*74*] have reviewed this document and state, "However, these estimates of total risk were so grossly in error that no arguments based even loosely on them should be taken seriously" (Ref *74*, p. 1240).

On 15 Jan. 1980, Dr. Irving Selikoff of the Mt. Sinai School of Medicine, New York City, stated at a press conference in conjunction with the annual American Medical Association meeting in Chicago, that 20 000 asbestos workers in the United States will die each year for the next 40 years of "excess disease" (*Journal of the American Medical Association*, Vol. 243, 18 Jan. 1980, p. 211). On 27 Sept. 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.

Hogan and Hoel [*94*] estimate that future excess cancer deaths among workers in the United States exposed to asbestos could constitute as much as 3.0% (range,

*TABLE 10—Cancer mortality data for Riverside and Totowa, New Jersey, for the period 1962–1976* [87].

| | Riverside Deaths[a] | | Totowa Deaths[b] | |
|---|---|---|---|---|
| Cause of Death | Number | % | Number | % |
| 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 |

[a]Neighborhood near the amosite asbestos factory.
[b]Control neighborhood.

10002308

*esidents) in Canada for females age*

| 52 Comparison Localities[a] |
|---|
| 433 |
| 158 |
| 13.4 |
| 4?.5 |
| 16.9 |

litre.
sotile fibers per litre of water.

deaths per year due to asbestos
stitute of Environmental Health
y, 11 Sept. 1978]. The Califano
93] prepared by several medical
Health [NCI, NIEHS, and the
Health (NIOSH)]. Doll and Peto
lowever, these estimates of total
its based even loosely on them

Mt. Sinai School of Medicine,
in conjunction with the annual
go, that 20 000 asbestos workers
ext 40 years of "excess disease"
Vol. 243, 18 Jan. 1980, p. 211).
Robert Locke (Associated Press
American workers are dying each

ess cancer deaths among workers
nstitute as much as 3.0% (range,

*towa, New Jersey, for the period 1962–*

| | Totowa Deaths[a] | |
|---|---|---|
| – | Number | % |
| 0 | 1735 | 100.00 |
| 0 | 353 | 20.35 |
| 26 | 98 | 5.65 |
| 7 | 74 | 4.27 |
| 15 | 22 | 1.27 |
| 51 | 12 | 0.69 |



1.4 to 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 the estimates on an analysis of the number of people possibly exposed to asbesto the possible exposure levels, and the estimated exposure-associated cancer ris

In the previous paragraphs, future mortality projections of 67 000, 20 00 10 000, and 12 000 deaths per year due to asbestos exposure are quoted. A any of these predictions correct? In the following section, a method is propose for estimating *past* asbestos-related mortality, which can then be used to predi future mortality.

*Past Mortality Based on Asbestosis Incidence*

The number of deaths due to asbestosis reported in *Vital Statistics of th United States* (Vol. II—*Mortality*, U.S. Department of Health and Human Ser ices, National Center for Health Statistics) for the whole nation for the eleven year period 1967 through 1977 is as follows:

| Year | Number of Deaths | Year | Number of Death |
|---|---|---|---|
| 1967 | 36 | 1973 | 42 |
| 1968 | 29 | 1974 | 35 |
| 1969 | 34 | 1975 | 45 |
| 1970 | 26 | 1976 | 54 |
| 1971 | 33 | 1977 | 55 |
| 1972 | 58 | | |

The average number of asbestosis deaths per year for the period 1967 through 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 number of 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 [*32*]. This group, of which 2271 are now dead, is one of the most severely affected by asbestos dusts. The mortality data and estimates of "excess" death due to asbestos are given in Table 11. (See also Table 3*b*, Study XVII.)

If the asbestosis deaths given in *Vital Statistics of the United States* are underreported by the same amount as was observed by Selikoff et al [best estimate (168)/death certificate (78) = 2.15] [*32*], the average number of asbestosis deaths each year during the period 1967 through 1977 is 41 × 2.15 or 88. Using disease ratios taken from Table 11 (asbestosis/excess cancer and asbestosis/excess respiratory disease), the average annual asbestos-related mortality in the United States for the period 1967 through 1977 is estimated to be 522 deaths, including 354 cancer deaths (Table 12).

10002309

TABLE 11—*Mortality among 17 800 insulation workers* [32].

| Cause of Death | Expected, No. | Observed, No.* | Excess, No. |
|---|---|---|---|
| All causes | 1659 | 2271 | 612 |
| All cancer | 320 | 995 | 675 |
| Mesothelioma | ... | 175 | 175 |
| Lung cancer | 106 | 486 | 380 |
| Gastrointestinal tract cancer | 59 | 99 | 40 |
| Other cancer | 155 | 235 | 80 |
| Asbestosis | ... | 168 | 168 |
| Noninfectious respiratory disease | 59 | 212 | 153 |

*Best estimate.

### *Past Mortality Based on the 1972 Mesothelioma Incidence*

It is not possible to obtain accurate mortality data for mesothelioma from *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 [*67*], Kannerstein et al [*95*], McDonald and McDonald [*7*], and Jones et al [*96*].

The mesothelioma study of McDonald and McDonald [*7*] can be used to estimate the excess asbestos-related mortality. Their data, as described previously, indicate 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 [*32*] listed in Table 11 to calculate disease ratios (mesothelioma/excess other cancers, mesothelioma/asbestosis, and mesothelioma/ex-

TABLE 12—*Estimated average yearly mortality due to asbestos for the period 1967–1977.*[a]

| Cause of Death | Estimated Deaths, No. |
|---|---|
| Mesothelioma | 92 |
| Lung cancer | 199 |
| Gastrointestinal tract cancer | 21 |
| Other cancer | 42 |
| Asbestosis | 88 |
| Noninfectious respiratory disease | 80 |
| Total asbestos-related deaths per year | 522 |

[a]Estimated based on recorded asbestosis deaths in *Vital Statistics of the United States*.

10002310

*nsulation workers* [32].

| Observed, No.* | Excess, No. |
|---|---|
| 2271 | 612 |
| 995 | 675 |
| 175 | 175 |
| 486 | 380 |
| 99 | 40 |
| 235 | 80 |
| 168 | 168 |
| 212 | 153 |

### *ıa Incidence*

data for mesothelioma from *Vital* is coded (ICD.8) with a number *ınt neoplasms of peritoneum and lignant neoplasms of other and* ) mediastinum (163.1), and site ɔusly, mesothelioma is a difficult ; and diagnosis make it necessary ɔcisions of mesothelioma review d [67], Kannerstein et al [95], [96].

McDonald [7] can be used to Their data, as described previ- lioma deaths in the United States stos. Again, using the mortality alculate disease ratios (mesothe- ɔestosis, and mesothelioma/ex-

*asbestos for the period 1967–1977.*

| Estimated Deaths, No. |
|---|
| 92 |
| 199 |
| 21 |
| 42 |
| 88 |
| 80 |
| 522 |

*Statistics of the United States.*



TABLE 13—*Estimated mortality due to asbestos in 1972, based on estimates of mesothelioma deaths by McDonald and McDonald* [7].

| Cause of Death | Estimated Deaths, No. |
|---|---|
| Mesothelioma | 103 |
| Lung cancer | 224 |
| Gastrointestinal tract cancer | 24 |
| Other cancer | 47 |
| Asbestosis | 99 |
| Noninfectious respiratory disease | 90 |
| Total asbestos-related deaths in 1972 | 587 |

cess 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).

In order to obtain a sense of perspective, it is useful to compare the death estimates obtained here 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. *Vital Statistics of the United States* reports that there were 215 silicosis deaths (ICD.8, 515.0) and 92 silicotuberculosis deaths (ICD.8, 010) in 1976. If it can be assumed that mortality due to silicosis and asbestosis are equally underreported by a factor of 2.15 and that the ratio of silicosis to excess noninfectious respiratory disease mortality is the same as the ratio of asbestosis to excess noninfectious respiratory disease, then the silica-related mortality for the United States in 1976 is estimated to total 975 deaths (Table 14).

In 1976, there were 879 reported deaths due to anthracosilicosis (ICD.8, 515.1, *Vital Statistics of the United States*). Underreporting and other excess respiratory disease may have also occurred for coal miners who have suffered 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 United States*) due to asbestosis (Code 515.2, males and females,

TABLE 14—*Estimated mortality in 1976 due to silica dust.*

| Cause of Death | Estimated Deaths, No. |
|---|---|
| Silicosis | 462 |
| Silicotuberculosis | 92 |
| Noninfectious respiratory disease | 421 |
| Total silica-related deaths in 1976 | 975 |

10002311

1967–1977) and "pleural neoplasms" (Code 163.0, males,[10] 1968–1977) suggests that there has been an increase in the number of deaths with time. The 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 (calculated by the method given previously). Hopefully, asbestos-related mortality is now peaking and will soon decline.

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, New York City. The reported deaths for the period 1967 through 1976 are as follows [*97*]:

| Year | Number of Deaths | Year | Number of Deaths |
|---|---|---|---|
| 1967 | 3 | 1972 | 2 |
| 1968 | 2 | 1973 | 1 |
| 1969 | 3 | 1974 | 3 |
| 1970 | 4 | 1975 | 4 |
| 1971 | 1 | 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 have produced measurable fiber counts in the ambient air of New York City streets. For example, Nicholson et al [*72*] report that 43 of the 89 air samples collected in New York City exceeded a count of 50 ng of chrysotile asbestos per cubic metre. Samples taken in New York City public schools have ranged from 9 to 1950 ng/m$^3$, with 15 out of 27 samples exceeding 100 ng of chrysotile per cubic metre. Despite the long presence of chrysotile asbestos in the New York City air, Demopoulos

TABLE 15—*Calculated mortality based on linear regression analysis of past reported mortality for asbestosis (males + females, 1967–1977) and pleural neoplasms (males, 1968–1977).*[a]

| Year | Asbestosis (males + females), No. | Pleural Neoplasms (males), No. |
|---|---|---|
| 1967 | 27 deaths | 184 deaths |
| 1977 | 52 | 222 |
| 1982 | 64 | 241 |
| 2000 | 105 | 308 |

[a]From *Vital Statistics of the United States*, 1967–1977.

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

10002312

e 163.0, males,[10] 1968–1977) sug-
number of deaths with time. The
n in Table 15. The variance, $r^2$, of
0.31 for pleural neoplasms. If this
inues, then the calculated mortality
at a total of 1337 asbestos-related
d by the method given previously).
peaking and will soon decline.
is pertinent to note the number of
of the New York University Medical
s for the period 1967 through 1976

| Year | Number of Deaths |
|---|---|
| 1972 | 2 |
| 1973 | 1 |
| 1974 | 3 |
| 1975 | 4 |
| 1976 | 3 |

= 0.01). Sprayed-on chrysotile as-
nstruction in New York City until
from brake linings have produced
New York City streets. For example,
air samples collected in New York
asbestos per cubic metre. Samples
ranged from 9 to 1950 ng/m$^3$, with
chrysotile per cubic metre. Despite
e New York City air, Demopoulos

*ression analysis of past reported mortality pleural neoplasms (males, 1968–1977).*[a]

| Pleural Neoplasms (males), No. |
|---|
| 184 deaths |
| 222 |
| 241 |
| 308 |

7.

will probably be reported under this code
ct a trend in total asbestos-related mortality.



[98] found no evidence of an increase in the number of mesothelioma deaths i this city over the past twelve years (1967 through 1978). Vianna et al [99] i a study of malignant mesothelioma in New York State (excluding New Yor City) found that there was no increase in the incidence of disease between 197 and 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 Province in Canada 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% of all asbestos ever mined has come from just three chrysotile mining localities, Quebec Province in Canada and the central and southern Ural Mountains of the Soviet Union. The chrysotile-producing countries in order of importance (in 1978 figures) are the Soviet Union (46.1% of the world's total asbestos production), Canada (28.9%), Zimbabwe (3.8%), China (3.8%), Italy (2.9%), South Africa (2.1%), Brazil (1.8%), the United States (1.7%), and Australia (1.0%).

### *Comparative Epidemiology*

The three principal diseases 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 has been exposed.

*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 break linings, as pipe insulation, and in other uses. About 95% of the asbestos in place in the United States is the chrysotile variety, and a large percentage of this was mined and milled in Quebec Province, Canada. 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

10002313

was less than expected (620 observed deaths, 659 expected deaths). The risk of lung cancer was slightly increased: 48 deaths observed, 42 deaths expected (Table 16, low to medium exposure). Exposures to 20 fibers/cm$^3$ are an order of magnitude greater than those experienced now (which are 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 (Table 4, Study G).

The results of only one epidemiological study of a cohort of trades workers known to be exposed only to chrysotile asbestos have been published (Table 3*a*, Study VI). 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.[11] In addition, 6 deaths among sons and daughters of chrysotile miners and millers and 2 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 lifelong 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[12] of crude blue fiber had entered the country, and by 1946, an additional 21 000 tons[12] had been imported. In addition to these imports, much crocidolite has come into the United States as manufactured products, such as yarns, tapes, and pipe coverings. Almost all of the imported crocidolite has come from South Africa.

Epidemiological studies of groups that 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 Cape Province, South Africa, and of Wittenoom, Western Australia. Prevalence studies in Cape Province report that at least 278 people have died of

[11] Acheson et al [*100*] cite two new studies of asbestos trades workers thought to have been exposed only to chrysotile which confirm the rarity of mesothelioma associated with chrysotile.

[12] 1 short ton = 0.907 tonnes (907 kg).

10002314

659 expected deaths). The risk of served, 42 deaths expected (Table 20 fibers/cm³ are an order of (which are generally less than 2 lifetime under these present dust surable excess cancer. A similar tile miners and millers (Table 4,

dy of a cohort of trades workers s have been published (Table 3*a*, asbestosis, but no excess of any

ng *only* with chrysotile asbestos this disease have been reported nd none among chrysotile trades d daughters of chrysotile miners sotile asbestos mining localities ma.

sidents of the Quebec chrysotile ant evidence that their lifelong nes and mills has caused excess

, usually referred to in the trade United States in 1911 or 1912. er had entered the country, and imported. In addition to these United States as manufactured ngs. Almost all of the imported

d only with crocidolite asbestos even relatively light exposure, cancer, mesothelioma, and as- d to crocidolite during gas mask ose employed in the crocidolite

world where mesothelioma is a e the crocidolite mining districts oom, Western Australia. Preva- least 278 people have died of

s trades workers thought to have been othelioma associated with chrysotile.



TABLE 16—*Mortality data for Quebec chrysotile miners and millers (low to medium and high to very high exposure) with 20 or more years' service and for Canadian gas mask assemblers (moderate exposure).*

| | Low to Medium Exposure[a] (10 to 21 fibers/cm³) | | | High to Very High Exposure[a] (95 to 194 fibers/cm³) | | | Moderate Exposure[b] | |
|---|---|---|---|---|---|---|---|---|
| Cause of Death | Observed Deaths | Expected Deaths | % of Observed Deaths | Observed Deaths | Expected Deaths | % of Observed Deaths | Observed Deaths | % of Observed Deaths |
| 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 |
| Gastrointestinal tract 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 respiratory disease | 28 | 34.3 | 4.5 | 31 | 18.4 | 6.5 | 4 | 7.1 |
| Mesothelioma | ... | ... | ... | ... | ... | ... | 9 | 16.1 |

[a]The data are from McDonald et al (Table 7*d* of Ref 48).
[b]Exposed to moderate levels of crocidolite dust for no more than 2.5 years [27]. See also Table 3*a*, Study X.

10002315

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 had worked in the crocidolite industry at Wittenoom, Western Australia, have died of mesothelioma. Of these, 13 had worked for less than twelve months and 9 had 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 twelve months and 19 had 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 2.2 million tonnes have been mined. Importation of amosite into the United States started in the 1930s.

One complete epidemiological study of asbestos trades workers exposed mainly to amosite asbestos had 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 [*100*]. Only prevalence studies have been made of amosite miners and millers; 2 individuals 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 that contain these minerals as gangue do not show any indication of asbestos-related mortality (Table 4, Studies B and E).

*Anthophyllite Asbestos*—This form of asbestos has been mined sporadically in many localities, but the only major production has been 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 (Table 4, Study A). This group showed a 67% excess of lung cancer and a large mortality due to tuberculosis and asbestosis. There were no deaths from mesothelioma. Because anthophyllite was and is used so little in commerce, no additional health studies appear to be possible except for follow-up studies 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 exposed to *only one form* of the three commonly used asbestos minerals—chrysotile, crocidolite,

10002316

olite; 161 of these people worked
the vicinity of the mines.
ocidolite industry at Wittenoom.
Of these, 13 had worked for less
dium exposure to blue asbestos.
died of lung cancer: 34 of these
twelve months and 19 had had
. In addition to this occupationally
lid not work in the mines or mills

e asbestos comes from the Trans-
n 1917 and 1979 approximately
ition of amosite into the United

os trades workers exposed mainly
incidence of asbestos-associated
d at a factory in Patterson, N.J.,
er mortality (60 cases), a 4.6%
asbestosis mortality (18 cases).
. reports on a group of workers
ition board factory. Here 5 men
ly prevalence studies have been
uals have died of mesothelioma.
been reported as having died of

erite and cummingtonite, which
site, are considered (incorrectly)
ies of miners working ores that
ny indication of asbestos-related

os has been mined sporadically
n has been at Paakkila, Finland,
d between 1918 and 1975. The
inantly to anthophyllite asbestos
A). This group showed a 67%
to tuberculosis and asbestosis.
cause anthophyllite was and is
th studies appear to be possible
ners.

*and Brown Asbestos*—There is
na among those exposed to *only*
inerals—chrysotile, crocidolite,



or 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 17. The difference between the 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 17. This is because asbestos-related mortality is probably much underreported in Western Australia and in South Africa because of the transient nature of the mining populations, of which many individuals are lost from view. Quebec Province, on the other hand, has a very stable mining and residential population, the medical surveillance of which has been excellent.

Several studies [*42,50,96,101*] have been made on the types and amounts of asbestos fiber in lung tissues of asbestos workers who died of mesothelioma and in those of "controls" who died of other diseases and who had not been occupationally exposed to asbestos. For example, Jones et al [*96*] found that chrysotile was present no more frequently nor in any greater amounts, in the mesothelioma cases than in the controls. They further state (Ref. *96*, p. 197) that "this study therefore provides no evidence to indict chrysotile in the etiology of mesothelioma." Similar findings are reported by McDonald [*42*] and McDonald [*101*]. These two studies also show that amphibole fibers (crocidolite and amosite) were more prevalent in the lung tissues of the mesothelioma cases than in those of the controls.

The contrast in mortality holds also for lung cancer and asbestosis. All three forms of asbestos (chrysotile, crocidolite, and amosite) 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 in Table 16 for the Quebec chrysotile asbestos miners and millers who had been exposed to high to very high dust levels for 20 or more years with the data for the Canadian gas mask

TABLE 17—*Mesothelioma mortality[a] in four asbestos mining districts in relation to asbestos production.*

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

[a]Reported in the scientific literature up to 1979 for miners, millers, and other residents of the mining districts.
[b]Excludes 7 mesothelioma cases that had been exposed to crocidolite.
[c]Excludes 4 mesothelioma cases that had been exposed to crocidolite.

10002317

assemblers, who had been exposed to moderate levels of crocidolite dust for no more than 2.5 years. For further comparison, mortality data are given for Quebec miners and millers who had been exposed to low to medium dust levels (Table 16).

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

Many of the men in the asbestos trades cohorts were probably exposed to crocidolite asbestos at some time during their working careers. 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, and so forth.

Peto [*24,102*] and Peto et al [*103*] 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 United Kingdom [*23,24,102*]. Their exposure was mainly to chrysotile asbestos, but crocidolite fiber has been processed in this factory at various times since 1933 [*24*]. Peto does not cite the work of Jones et al [*50*] or that of McDonald and McDonald [*27*] on the gas mask assemblers, nor the work of Hilt et al [*36*] on construction workers, studies that clearly show the extreme hazard of crocidolite. Brief exposure only to crocidolite can set up a disease pattern very similar to that found in asbestos trades workers heavily exposed to asbestos for many years. From this we can conclude that mortality studies of asbestos workers who have been 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 between 10 000 and 67 000 deaths per year for the next 20 to 30 years. These estimates do not appear to be valid when compared with estimates of *past* mortality that are based on reported asbestos-related deaths 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, the author estimates that 587 individuals died in that year because of exposure to asbestos; or (2) taking the reported number of

10002318

e levels of crocidolite dust for no
ortality data are given for Quebec
ow to medium dust levels (Table

n the mining and milling cohorts
rts (Tables 3*a* and 3*b*) is clearly
y due to lung cancer is plotted in
othelioma. The average mortality
g the crocidolite miners (Table 4,
r lung cancer. For the 21 asbestos
for mesothelioma and 16.7% for
the crocidolite mining cohort is
orts, 3.3% of this group having
er.
horts were probably exposed to
working careers. They also were
ons of asbestos dust, particularly
closed spaces, such as heating

at chrysotile asbestos contributes
ases his conclusions on a study
United Kingdom [*23,24,102*].
s, but crocidolite fiber has been
1933 [*24*]. Peto does not cite
ald and McDonald [*27*] on the
l [*36*] on construction workers,
crocidolite. Brief exposure only
similar to that found in asbestos
many years. From this we can
kers who have been exposed to
ot valid in predicting the health

*United States*

tions, it has been suggested that
se between 10 000 and 67 000
e estimates do not appear to be
tality that are based on reported
r asbestosis. For example, (1)
in 17 800 North American as-
nesothelioma in 1972 given by
hat 587 individuals died in that
aking the reported number of



asbestosis deaths given in *Vital Statistics of the United States* and again utilizing the mortality data of the North American insulation workers, the author estimates that the average yearly asbestos-related mortality in the United States during the period 1967 through 1977 was 522 deaths. There is some suggestion from the mortality data given in *Vital Statistics of the United States* that the incidence of asbestos-related disease has increased 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 that are presented here.

Nonoccupational exposure to chrysotile asbestos, despite its wide dissemination in urban environments throughout the world, has not been shown by epidemiological studies to be a significant health hazard. 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. Health studies accomplished in Canada show that populations can safely breathe air and drink water that contains significant amounts of chrysotile fiber. These studies also show that there is a "threshold" value for chrysotile asbestos exposure below which no measurable health effects will occur.

The same fiber dose–disease response relationships observed for chrysotile asbestos do not hold for crocidolite asbestos. Health studies of those exposed to crocidolite only 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 would 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 Cape Province, South Africa, whose only exposure was in a nonoccupational setting. Such mortality is practically unknown among residents of the chrysotile mining localities of Quebec Province. 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 [*12*].

The hazards of amosite asbestos are more difficult to assess. The amosite factory employees of Patterson, N.J., who worked under very dusty conditions during World War II, have experienced a great deal of 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 it is possible to have dust controls that can much reduce or prevent the occurrence of asbestos-related diseases in amosite workers.

10002319

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 reduce greatly 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, and so forth. This is being done, even though most asbestos in the United States is of the chrysotile variety and even though asbestos dust levels in schools, public buildings, and city streets are much lower than those found in chrysotile asbestos mining communities, where little asbestos-related disease appears in the nonoccupationally exposed residents. The impetus for these costly removals and appliance recalls (hair dryers, for example) apparently comes from propagandizing of 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 for materials, such as nonflammability, 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 by having inferior strength or durability, increased flammability, or other undesirable characteristics. A good substitute must not have so high a cost that it forces the use of an inadequate replacement. Possible problems can occur with substitutes, 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 emitted from drum brakes. Also, the health effects of emissions from substitute brake linings must be considered.

The requirements of strength and flexibility make it necessary 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 μm and less than 1.5 μm in diameter are capable of producing malignant neoplasms when implanted into the pleura of rats [*104*]. Test fibers used in these studies have included aluminum oxide, fiberglass, wollastonite ($CaSiO_3$), silicon carbide, dawsonite ($NaAlCO_3OH$), and potassium octatitanate.

Lee et al [*105,106*] 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, three hamsters developed pleural mesothelioma, a rare disease in the control animals.

10002320

such as "one fiber can kill you"
the amount of asbestos-related
generated great political pressure
to reduce greatly or even stop its
ort in several industrial nations,
s from schools, public buildings,
is being done, even though most
variety and even though asbestos
city streets are much lower than
nmunities, where little asbestos-
y exposed residents. The impetus
ls (hair dryers, for example) ap-
one fiber can kill you" concept.
langerous if the removal of blue
In most cases, asbestos coatings
ed at no risk and at a fraction of

d, substitutes would have to be
for materials, such as nonflam-
cost, and safety. With respect to
those exposed to them and also
g inferior strength or durability,
aracteristics. A good substitute
e of an inadequate replacement.
r example, with the replacement
chance of increased automobile
aterial must be weighed against
e small amounts of chrysotile
ealth effects of emissions from

make it necessary that asbestos
longer the fibers, the stronger,
bers longer than 4 μm and less
ing malignant neoplasms when
bers used in these studies have
nite ($CaSiO_3$), silicon carbide,
anate.
s, hamsters, and guinea pigs of
a commercially made potassium
They found that, in addition to
animals, three hamsters devel-
control animals.



In the report of the Advisory Committee on Asbestos, a committee of the Health and Safety Commission of Great Britain, the following statement is made in regard to substitutes for asbestos (Ref *12*, 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 oversimplification 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.

Recent animal experiments such as those just cited make the Advisory Committee's statement particularly meaningful.

The cost of asbestos substitutes is of particular importance to Third World countries, whose developing economies are very dependent on making the maximum use of cheap, domestically produced materials wherever 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: Russia, Zimbabwe, Greece, Mexico, and 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, in Minnesota, is considered to be asbestos by the U.S. Environmental Protection Agency and the Courts of Minnesota, although the author knows of no geologist who would call this mineral asbestos. However, be that 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 affected by asbestos regulations.

In addition to the fibrous minerals found in numerous ore deposits, there are also such minerals 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 with volcanic ash. Should the public be

10002321

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 an action was proposed by P. C. Elmes, director of the Medical Research Council Pneumoconiosis Unit of Llandough Hospital, Penarth, Wales [*107*]. He writes, "Populations living on soils contaminated with the fibers under dry climatic conditions need to be moved." In regard to his proposal, it should be noted that fibrous zeolite minerals occur in many areas of southwestern United States and that fibrous clay minerals are common in the coastal plain sediments of eastern United States.

One does not have to consider these 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 with 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 the effort to understand and mitigate asbestos-related cancers. Many billions more may be awarded to those filing claims against asbestos companies. In contrast, relatively little has been spent on study and mitigation of the more serious nonneoplastic lung diseases, such as the pneumoconioses caused by inhalation of crystalline silica and coal dusts.

A great deal has been accomplished toward understanding the relationships between the intensity of exposure to the several forms of commercial asbestos and the incidence of asbestos-related disease. Epidemiological studies have shown that modern dust control methods now in effect may prevent most morbidity and mortality related to exposure to chrysotile asbestos. Similar studies should be completed in order to set dust levels to protect anthophyllite and amosite asbestos workers (in this regard see the Simpson report [*12*].

## References


[*1*] Europaeus-Äyräpää, A., *Acta Archaeology*, Vol. 1, 1930, pp. 169–190.

[*2*] Jones, R. H., *Asbestos, Its Properties, Occurrence and Uses*, Crosby, Lockwood and Son, London, 1890.

[*3*] Clifton, R. A., *Asbestos MCP Mineral Commodity Profiles*, U.S. Bureau of Mines, Washington, D.C., July 1979.

[*4*] Hall, A. L., *Asbestos in the Union of South Africa, Memoirs of the Geological Survey of South Africa*, No. 12, 1918, pp. 13–14.

[*5*] Ross, M., in *Amphiboles and Other Hydrous Pyriboles—Mineralogy*, D. R. Veblen, Ed., Vol. 9a of *Reviews in Mineralogy*, Mineralogical Society of America, Washington, D.C., 1981, pp. 279–323.

[*6*] Selikoff, I. J. and Lee, D. H. K., *Asbestos and Disease*, Academic Press, New York, 1978.

[*7*] McDonald, A. D. and McDonald, J. C., *Cancer*, Vol. 46, 1980, pp. 1650–1656.

[*8*] Meurman, L. O., Kiviluoto, R., and Hakama, M., *British Journal of Industrial Medicine*, Vol. 31, 1974, pp. 105–112.

[*9*] Rubino, G. F., Piolatto, G., Newhouse, M. L., Scansetti, G., Aresini, G. A., and Murray, R., *British Journal of Industrial Medicine*, Vol. 36, 1979, pp. 187–194.


10002322

ibers can possibly cause cancer?
ılated to the lowest feasible limit?
oving people out of regions where
n was proposed by P. C. Elmes,
eumoconiosis Unit of Llandough
'Populations living on soils con-
:onditions need to be moved." In
. fibrous zeolite minerals occur in
nd that fibrous clay minerals are
tern United States.
tions very long before coming to
they would present a regulatory,

. health risk (this seems to occur
ns), we must allocate our scientific
. health problems in proportion to
ı spent, directly and indirectly, in
)s-related cancers. Many billions
; against asbestos companies. In
tudy and mitigation of the more
; the pneumoconioses caused by

d understanding the relationships
ral forms of commercial asbestos
pidemiological studies have shown
t may prevent most morbidity and
)estos. Similar studies should be
ınthophyllite and amosite asbestos
t [12].


. 1930, pp. 169–190.
*e and Uses*, Crosby, Lockwood and Son,

*Profiles*, U.S. Bureau of Mines, Wash-

*a, Memoirs of the Geological Survey of*

*iboles—Mineralogy*, D. R. Veblen, Ed.,
Society of America, Washington, D.C.,

*ease*, Academic Press, New York, 1978.
Vol. 46, 1980, pp. 1650–1656.
, *British Journal of Industrial Medicine*,

ansetti, G., Aresini, G. A., and Murray,
, 1979, pp. 187–194.





[10] Nicholson, W. J., Selikoff, I. J., Seidman, H., Lilis, R., and Formby, P., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 11–21.
[11] Becklake, M. R., *American Review of Respiratory Disease*, Vol. 114, 1979, pp. 187–227.
[12] Simpson, W., *Asbestos: Final Report of the Advisory Committee*, Vol. 1, and *Papers Commissioned by the Committee*, Vol. 2, Health and Safety Commission, London, U.K., 1979.
[13] Davis, J. M. G., *Annals of Occupational Hygiene*, Vol. 24, 1981, pp. 227–234.
[14] Selikoff, I. J., Hammond, E. C., and Seidman, H., *Cancer*, Vol. 46, 1980, pp. 2736–2740.
[15] McCullagh, S. F., *Journal of the Society of Occupational Medicine*, Vol. 30, 1980, pp. 153–156.
[16] McDonald, J. C. and McDonald, A. D., *Preventive Medicine*, Vol. 6, 1977, pp. 426–446.
[17] Thomas, H. F., Benjamin, I. T., Elwood, P. C., and Sweetnam, P. M., *British Journal of Industrial Medicine*, Vol. 39, 1982, pp. 273–276. See also Ref *108*.
[18] Kleinfeld, M., Messite, J., and Kooyman, O., *Archives of Environmental Health*, Vol. 15, 1967, pp. 177–180.
[19] Mancuso, T. F. and El-Attar, A. A., *Journal of Occupational Medicine*, Vol. 9, 1967, pp. 147–162.
[20] Selikoff, I. J., Hammond, E. C., and Seidman, H. in *Biological Effects of Asbestos, IARC Scientific Publications*, No. 8, International Agency for Research on Cancer, Lyon, France, 1973, pp. 209–216.
[21] Elmes, P. C. and Simpson, J. C., *British Journal of Industrial Medicine*, Vol. 34, 1977, pp. 174–180.
[22] Weiss, W., *Journal of Occupational Medicine*, Vol. 19, 1977, pp. 737–740.
[23] Peto, J., Doll, R., Howard, S. V., Kinlen, L. J., and Lewinsohn, H. C., *British Journal of Industrial Medicine*, Vol. 34, 1977, pp. 169–173.
[24] Peto, J., *Lancet*, Vol. 4, 1978, pp. 484–489.
[25] Risberg, B., Nickels, J., and Wagermark, J., *Cancer*, Vol. 45, 1980, pp. 2422–2427.
[26] Nicholson, W. J., Langer, A. M., and Selikoff, I. J. in *Workshop on Asbestos: Definitions and Measurement Methods, National Bureau of Standards Special Publication 506*, C. C. Gravatt, P. D. LaFleur, and K. F. J. Heinrich, Eds., National Bureau of Standards, Washington, D.C., 1978, pp. 71–93.
[27] McDonald, A. D. and McDonald, J. C., *Environmental Research*, Vol. 17, 1978, pp. 340–346.
[28] Henderson, V. L. and Enterline, P. E., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 117–126.
[29] Puntoni, R., Vercelli, M., Merlo, F., Valerio, F., and Santi, L., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 353–377.
[30] Newhouse, M. L. and Berry, G., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 53–60.
[31] Weill, H., Hughes, J., and Waggenspack, C., *American Review of Respiratory Disease*, Vol. 120, 1979, pp. 345–354.
[32] Selikoff, I. J., Hammond, E. C., and Seidman, H., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 91–116.
[33] Selikoff, I. J., Lilis, R., and Nicholson, W. J., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 295–311.
[34] Selikoff, I. J., Seidman, H., and Hammond, E. C., *Journal of the National Cancer Institute*, Vol. 65, 1980, pp. 507–513.
[35] Clemmesen, J. and Hjalgrim-Jensen, S., *Ecotoxicology and Environmental Safety*, Vol. 5, 1981, pp. 15–23.
[36] Hilt, B., Rosenberg, J., and Langard, S., *Scandinavian Journal of Work and Environmental Health*, Vol. 7, 1981, pp. 185–189.
[37] Hammond, E. C., Garfinkel, L., and Lew, E. A., *Environmental Research*, Vol. 16, 1978, pp. 153–173.
[38] Sterling, T. D. and Weinkam, J. J., *Archives of Environmental Health*, Nov./Dec. 1978, pp. 313–317.
[39] Selikoff, I. J. and Hammond, E. D. in *Persons at High Risk of Cancer*, J. Fraumeni, Ed., Academic Press, New York, 1975.
[40] Saracci, R., *International Journal of Cancer*, Vol. 20, 1977, pp. 323–331.
[41] McDonald, J. C., Becklake, M. R., Gibbs, G. W., McDonald, A. D., and Rossiter, C. E., *Archives of Environmental Health*, Vol. 28, 1974, pp. 61–68.


10002323

[42] McDonald, J. C. in *Biological Effects of Mineral Fibres*, Vol 2, J C Wagner, Ed , *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 587–601.
[43] Higgins, I. T. T., School of Public Health, University of Michigan, personal communication re a mortality study of employees of the Reserve Mining Co., 12 May 1981.
[44] Selikoff, I. J. in *Biochemistry of Silicon and Related Problems*, G. Bendz and I. Lindquist, Eds., Plenum Press, 1978, pp. 311–335.
[45] Gillam, J. D., Dement, J. M., Lemen, R. A., Wagoner, J. K., Archer, V. E., and Blejer, H. P., *Annals of the New York Academy of Sciences*, Vol. 271, 1976, pp. 336–344.
[46] McDonald, J. C., Gibbs, G. W., Liddell, F. D. K., and McDonald, A. D., *American Review of Respiratory Disease*, Vol. 118, 1978, pp. 271–277.
[47] Hobbs, M. S. T., Woodward, S., Murphy, B., Musk, A. W., and Elder, J. E. in *Biological Effects of Mineral Fibres*, Vol. 2, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 615–625.
[48] McDonald, J. C., Liddell, F. D. K., Gibbs, G. W., Eyssen, G. E., and McDonald, A. D., *British Journal of Industrial Medicine*, Vol. 37, 1980, pp. 11–24.
[49] Jones, J. S. P., Pooley, F. D., and Smith, P. G., *INSERM Symposium Series*, Vol. 52, *IARC Scientific Publications*, No. 13, International Agency for Research on Cancer, Lyon, France, 1976, pp. 117–120.
[50] Jones, J. S. P., Pooley, F. D., Sawle, G. W., Madeley, R. J., Smith, P. G., Berry, G., Wignall, B. K., and Aggarwal, A. in *Biological Effects of Mineral Fibres*, Vol. 2, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 637–653.
[51] Vejlsted, H. and Hansen, B. F., *Scandinavian Journal of Thoracic and Cardiovascular Surgery*, Vol. 14, 1980, pp. 119–122.
[52] Kannerstein, M. and Churg, J., *Environmental Health Perspectives*, Vol. 34, 1980, pp. 31–36.
[53] Legha, S. S. and Muggia, F. M., *Annals of Internal Medicine*, Vol. 87, 1977, pp. 613–621.
[54] Rüttner, J. R. in *Proceedings*, Asbestos Symposium, Johannesburg, South Africa, 3–7 Oct. 1977, H. H. Glen, Ed., Department of Mines, Capetown, South Africa, 1978, pp. 86–89.
[55] Liddell, F. D. K., *Canadian Medical Association Journal*, Vol. 125, 1981, pp. 237–239.
[56] McDonald, J. C. and Becklake, M. R., *Hefte Zeitschrift für Unfallheilkunde (Deutsch-Osterreichisch-Schweizerische Unfalltagung in Berlin 1975*, Springer-Verlag, Berlin, 1975), Vol. 126, No. 2, 1976, pp. 521–535.
[57] Webster, I. in *Proceedings*, Asbestos Symposium, Johannesburg, South Africa, 3–7 Oct. 1977, H. H. Glen, Ed., Department of Mines, Capetown, South Africa, 1978, p. 79.
[58] Talent, J. M., Harrison, W. O., Solomon, A., and Webster, I., in *Biological Effects of Mineral Fibres*, Vol. 2, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 723–729.
[59] Wagner, J. C., Sleggs, C. A., and Marchand, P., *British Journal of Industrial Medicine*, Vol. 17, 1960, pp. 260–271.
[60] Mostert, C. and Meintjes, R., *Central African Journal of Medicine*, Vol. 25, 1979, pp. 72–74.
[61] Cochrane, J. C. and Webster, I., *South African Medical Journal*, Vol. 54, 1978, pp. 279–281.
[62] Langlois, S. Le P., Glancy, J. J., and Henderson, D. W., *Australian Radiology*, Vol. 22, 1978, pp. 305–314.
[63] Jones, C. D. E. and Silver, D., *Surgery*, Vol. 86, 1979, pp. 556–560.
[64] Brenner, J., Sordillo, P. P., and Magill, G. B., *Medical Pediatrics and Oncology*, Vol. 9, 1981, pp. 367–373.
[65] Brenner, J., Sordillo, P. P., Magill, G. B., and Golbey, R. B., *American Journal of Gastroenterology*, Vol. 75, 1981, pp. 311–313.
[66] Griffiths, M. H., Riddell, R. J., and Xipell, J. M., *Pathology*, Vol. 12, 1980, pp. 591–603.
[67] McDonald, A. D., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 441–454.
[68] Thériault, G. P., and Grand-Bois, L., *Archives of Environmental Health*, Vol. 33, 1978, pp. 15–19.

10002324

*ibres*, Vol. 2, J. C. Wagner, Ed., *IARC*
y for Research on Cancer, Lyon, France.

y of Michigan, personal communication
ining Co., 12 May 1981
*l Problems*, G. Bendz and I. Lindquist,

oner, J. K., Archer, V. E., and Blejer,
, Vol. 271, 1976, pp. 336–344.
nd McDonald, A. D., *American Review*
77.
k, A. W., and Elder, J. E. in *Biological*
., *IARC Scientific Publications*, No. 30,
., France, 1980, pp. 615–625.
Eyssen, G. E., and McDonald, A. D.,
30, pp. 11–24.
*SERM Symposium Series*, Vol. 52, *IARC*
y for Research on Cancer, Lyon, France,

deley, R. J., Smith, P. G., Berry, G.,
*ffects of Mineral Fibres*, Vol. 2, J. C.
., International Agency for Research on

*urnal of Thoracic and Cardiovascular*

*alth Perspectives*, Vol. 34, 1980, pp.

*Medicine*, Vol. 87, 1977, pp. 613–621.
., Johannesburg, South Africa, 3–7 Oct.
Capetown, South Africa, 1978, pp.

*urnal*, Vol. 125, 1981, pp. 237–239.
*itschrift für Unfallheilkunde (Deutsch-*
*n 1975*, Springer-Verlag, Berlin, 1975),

Johannesburg, South Africa, 3–7 Oct.
town, South Africa, 1978, p. 79.
d Webster, I., in *Biological Effects of*
*ntific Publications*, No. 30, International
0, pp. 723–729.
*British Journal of Industrial Medicine*,

*urnal of Medicine*, Vol. 25, 1979, pp.

*Medical Journal*, Vol. 54, 1978, pp.

D. W., *Australian Radiology*, Vol. 22,

979, pp. 556–560.
*dical Pediatrics and Oncology*, Vol. 9,

lbey, R. B., *American Journal of Gas-*

*Pathology*, Vol. 12, 1980, pp. 591–603.
*v of Sciences*, Vol. 330, 1979, pp. 441–

*nvironmental Health*, Vol. 33, 1978, pp.





[69] McDonald, A. D. in *Biological Effects of Mineral Fibres*, Vol. 2, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 673–680.

[70] Pampalon, R., unpublished work document (Document No. 5596-79c), Asbestos-Heal Project, Division of Epidemiological Studies, Ministry of Social Affairs, Quebec, Canad 1979.

[71] Brulotte, R., *Atmospheric Pollution, Proceedings*, 12th International Colloquium, Paris France, May 1976, M. M. Benarie, Ed., 1976, pp. 447–458.

[72] Nicholson, W. J., Rohl, A. N., Weisman, I., and Selikoff, I. J. in *Biological Effects Mineral Fibres*, Vol. 2, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, Internation Agency for Research on Cancer, Lyon, France, 1980, pp. 823–827.

[73] Graham, S., *Environmental Research*, Vol. 25, 1981, pp. 35–49.

[74] Doll, R. and Peto, R., *Journal of the National Cancer Institute*, Vol. 66, 1981, pp. 1193 1308.

[75] Goldsmith, J. R., *Journal of Environmental Pathology and Toxicology*, Vol. 3, 1980, pp 205–217.

[76] Higginson, J. and Muir, C. S., *Journal of the National Cancer Institute*, Vol. 63, 1979, pp 1291–1298.

[77] Higginson, J., *Journal of Environmental Pathology and Toxicology*, Vol. 3, 1980, pp 113–125.

[78] Weisberger, J. H., *Texas Reports on Biology and Medicine*, Vol. 37, 1978, pp. 1–18.

[79] Gori, G. B., *Cancer*, Vol. 43, 1979, pp. 2151–2161.

[80] Wynder, E. L., *Journal of Environmental Pathology and Toxicology*, Vol. 3, 1980, pp. 171–192.

[81] Wigle, D. T., *Archives of Environmental Health*, Vol. 32, 1977, pp. 185–190.

[82] Donham, K. J., Berg, J. W., Will, L. A., and Leininger, J. R., *Cancer*, Vol. 45, 1980, pp. 1073–1084.

[83] Hallenback, W. H., Markey, D. R., and Dolan, D. G., *Environmental Research*, Vol. 25, 1981, pp. 349–360.

[84] Hilding, A. C., Hilding, D. A., Larson, D. M., and Aufderheide, A. C., *Archives of Environmental Health*, Vol. 36, 1981, pp. 298–303.

[85] Smith, W. E., Hubert, D. D., Sobel, H. J., Peters, E. T., and Doerfler, T. E., *Journal of Environmental Pathology and Toxicology*, Vol. 3, 1980, pp. 277–300.

[86] Toft, P., Wigle, D., Meranger, J. C., and Mao, Y., *Science of the Total Environment*, Vol. 18, 1981, pp. 77–89.

[87] Hammond, E. C., Garfinkel, L., Selikoff, I. J., and Nicholson, W. J., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 417–422.

[88] Epler, G. R., FitzGerald, M. X., Gaensler, E. A., and Carrington, C. B., *Respiration*, Vol. 39, 1980, pp. 229–240.

[89] Antman, K. H., Blum, R. H., Greenberger, J. S., Flowerdew, G., Skarin, A. T., and Canellos, G. P., *American Journal of Medicine*, Vol. 68, 1980, pp. 356–362.

[90] Vianna, N. J. and Polan, A. K., *Lancet*, May 20, 1978, pp. 1061–1063.

[91] Newhouse, M., *Seminars in Oncology*, Vol. 8, 1981, pp. 250–257.

[92] Newhouse, M. L. and Thompson, H., *British Journal of Industrial Medicine*, Vol. 22, 1965, pp. 261–269.

[93] Bridbord, K., Decoufle, P., Fraumeni, J. F., Jr., Hoel, D. G., Hoover, R. N., Rall, D. P., Saffiotti, U., Schneiderman, M. A., and Upton, A. C., "Estimates of the Fraction of Cancer in the United States Related to Occupational Factors," unpublished document from the National Cancer Institute, National Institute of Environmental Safety and Health, and the National Institute of Occupational Safety and Health, Washington, D.C., 15 Sept. 1978, pp. 1–45.

[94] Hogan, M. D. and Hoel, D. G., *Risk Analysis*, Vol. 1, 1981, pp. 67–76.

[95] Kannerstein, M., Churg, J., and McCaughey, W. T. E., *Annals of the New York Academy of Sciences*, Vol. 330, 1979, pp. 433–439.

[96] Jones, J. S. P., Pooley, F. D., Clark, N. J., Owen, W. G., Roberts, G. H., Smith, P. G., Wagner, J. C., Berry, G., and Pollock, D. J. in *Biological Effects of Mineral Fibres*, Vol. 1, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 187–199.


10002325

[97] Demopoulos, H. B., New York University Medical Center, New York City, personal communication, 1980.
[98] Demopoulos, H. B., *Bulletin de l'Association des Mines d'Amiante du Québec*, Vol. 4, 1980, pp. 1–7.
[99] Vianna, N. J., Maslowsky, J., Roberts, S., Spellman, G., and Patton, R. B., *New York State Journal of Medicine*, April 1981, pp. 735–738.
[100] Acheson, E. D., Gardner, M. J., Bennett, C., and Winter, P. D., *Lancet*, 19–26 Dec. 1981, pp. 1403–1406.
[101] McDonald, A. D. in *Biological Effects of Mineral Fibres*, Vol. 2, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 681–685.
[102] Peto, J. in *Biological Effects of Mineral Fibres*, Vol. 2, J. C. Wagner, Ed., *IARC Scientific Publications*, No. 30, International Agency for Research on Cancer, Lyon, France, 1980, pp. 703–711.
[103] Peto, J., Seidman, H., and Selikoff, I. J., *British Journal of Cancer*, Vol. 45, 1982, pp. 124–135.
[104] Stanton, M. F., Layard, M., Tegeris, A., Miller, E., May, M., Morgan, E., and Smith, A., *Journal of the National Cancer Institute*, Vol. 65, 1981, pp. 965–975.
[105] Lee, K. P., Barras, C. E., Griffith, F. D., and Waritz, R. S., *American Journal of Pathology*, Vol. 102, 1981, pp. 314–323.
[106] Lee, K. F., Barras, C. E. Griffith, F. D., Waritz, R. S., and Lapin, C. A., *Environmental Research*, Vol. 24, 1981, pp. 167–191.
[107] Elmes, P. C., *Journal of the Geological Society of London*, Vol. 137, 1980, pp. 525–535.
[108] Elwood, P. C. and Cochrane, A. L., *British Journal of Industrial Medicine*, Vol. 21, 1964, pp. 304–307.
[109] "Carcinogenesis Bioassay of Amosite Asbestos in Syrian Golden Hamsters," Public Health Service, Department of Health and Human Services, Publication No. (NIH) 81-, 23 June 1981, NTP-81-58, Washington, D.C., pp. 1–87.
[110] "Carcinogenesis Bioassay of Chrysotile Asbestos in Syrian Golden Hamsters," Public Health Service, Department of Health and Human Services Publication No. (NIH) 81-, 23 June 1981, NTP-81-51, Washington, D.C., pp. 1–191.

10002326