Latest word on asbestos won't be the last

Scientific evidence against asbestos continues to accumulate, and OSHA's much-delayed exposure standard goes on bringing criticism from all concerned parties. Meanwhile, millions of workers remain exposed to asbestos dust in plants throughout the world.

☐ Bad news about asbestos continues to make headlines. In September, for instance, Joseph A. Califano, Secretary of Health, Education, and Welfare (HEW), drew attention to an alarming statistic in a new study by the National Cancer Institute (NCI) and the National Institute of Environmental Health Sciences (NIEHS): An astonishing 17% of all cancer deaths in the U.S. over the next few decades will be linked to some previous exposure to asbestos.

Speaking at the AFL-CIO National Conference on Occupational Safety and Health, Califano said that the NCI/NIEHS report definitely rejects the conventional estimate that 1 to 5% of all U.S. cancer incidence is related to occupational exposure to carcinogenic agents. Estimates of 20% to as high as 38% are not unreasonable, he added.

EXPOSURE THAT KILLS—The joint study estimates that between 8 and 11 million U.S. workers have been exposed to asbestos since the beginning of World War II. Of these, about 4 million have been "heavily exposed,"—i.e., worked directly with the material most of the time—and 4 to 7 million have received "peripheral" exposure—e.g., of the sort that a maintenance worker would get in a plant where asbestos was being installed.

The NCI/NIEHS report assumes that 20 to 25% of those in the first group will die of lung cancer, 7 to 10% of pleural or peritoneal mesothelioma (a particularly lethal cancer), and 8 to 9% of gastrointestinal cancer. Assuming that the risk to the second group is 25% that of the first one, the report concludes that a total of 2.15 million workers will die over the next 30 to 35 years, or about 67,000 per year. This represents a whopping 17% of all cancers detected annually in the U.S.

The joint study's figures on exposure for workers are taken from HEW estimates (the department believes that 1.5 to 2.5 million U.S. workers are still being exposed to asbestos), while the fatality percentages are borrowed directly from a World Health Organization (WHO) position paper issued in early 1977. This link with the WHO document is confirmed by Joseph Wagoner, special assistant for carcinogenesis with the Occupational Safety and Health Administration (OSHA), who was a member of the group of health experts that authored the WHO study.

Meanwhile, a spokesman for NCI/NIEHS says that the joint report is basically an update of epidemiological data on asbestos, in the light of new information uncovered in the past year.

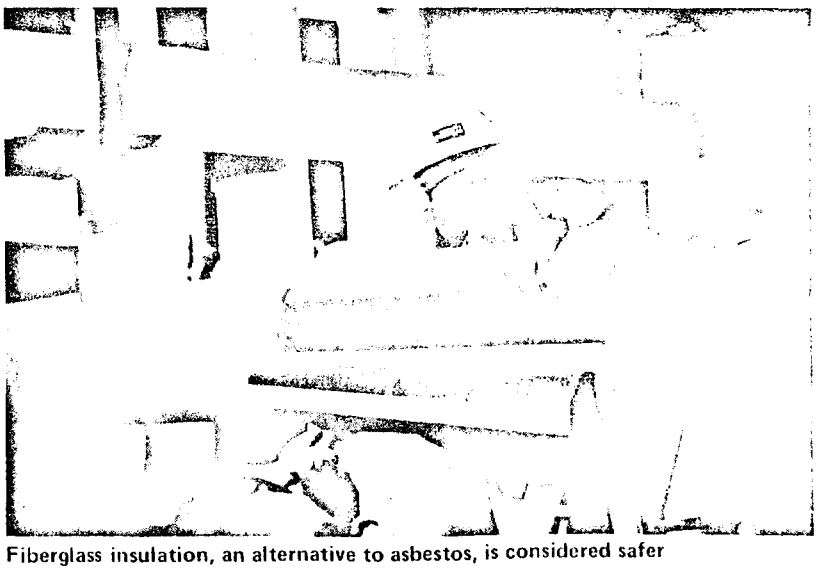
Whatever its origin, the NCI/NIEHS work is attracting its share of criticism by way of an attack on Califano's recent statement. Some claim that the HEW secretary is unnecessarily alarming the public. Asbestos no longer causes widespread sickness because working conditions have changed, they say.

Dr. Irving J. Selikoff, a well-known authority on asbestos, and director of Mt. Sinai Medical Center's Environmental Health Laboratory (New York City), notes that "Califano's remarks are projections based on limited data, and probably represent an upper limit." But he warns that even if Califano's figures are off by a factor of two, the problem is enormous.

However, a spokesman for the American Cancer Soc. (ACS), New York City, feels that the NCI/NIEHS report does overstate the problem.

INDUSTRY RESPONSE—The Asbestos Information Assn. (AIA), Arlington, Va., an organization of asbestos processors, says that the NCI/NIEHS data are being analyzed by the group's scientific advisors, and that a statement will be issued when the study is complete. Meanwhile, an AIA press release calls the report "simply another extrapolation in statistical terms from the same data which have been available to the international scientific community for some time."

Another AIA press release, issued in response to a Califano statement (made in April) regarding a large



Fiberglass insulation, an alternative to asbestos, is considered safer

U.S. firms still use it, despite its checkered career

The term "asbestos" comprises a group of impure magnesium silicate minerals—e.g., chrysotile, amosite—that occur in fibrous form. Reports of the toxic properties of asbestos date back to the 1920s in the U.S. However, it wasn't until the war broke out, when millions of workers were employed in shipyards insulating warships, that mass exposure to asbestos dust occurred in this country.

The material enjoyed wide popularity as an insulator in plants and buildings until 1970, when, in response to mounting evidence of its toxic properties, manufacturers stopped producing insulation made of loosely bound asbestos fiber.

The Asbestos Information Assn. says that the 3,000 asbestos products made today in the U.S. contain chemically and physically bound fibers that do not flake off. Current end-use breakdown: asbestos cement products (pipe, sheets), 22.4%; roofing products, 34.9%; asbestos paper, 4.3%; flooring products, 15.6%; packing and gaskets, 2.8%; thermal and electrical insulation, 1.2%; friction products (brake linings), 8.8%; coatings and compounds, 2.7%; plastics, 3.0%; textiles, 1.0%; and miscellaneous, 3.3%.

outbreak of asbestos-related diseases among World War II shipyard workers, explains that such a heavy exposure to asbestos existed before medical information revealed the dangers involved. Since then, millions of dollars have been spent in dust-control equipment by miners, millers, processors and manufacturers of asbestos.

In addition, note other asbestos-industry spokesmen, the type of products made in the past—e.g., asbestos thermal insulation—are no longer produced. They were phased out in the early 1970s, when the inherent hazards became apparent (see box).

Nevertheless, asbestos is still used as an insulator or fire retardant in more than 3,000 products ranging from water pipes to brake linings. And an estimated 500 million tons of the material are still around in plants and buildings throughout the U.S., according to Roy Steinfurth, administrator of the insulation health-hazards programs for the International Assn. of Heat and Frost Insulators and Asbestos Workers (Washington). Workers still exposed to asbestos are not adequately protected, charges Selikoff.

CIGARETTE LINK—Industry spokesmen also point out that exposure to asbestos alone may not be enough to cause lung cancer—the disease most commonly associated with the breathing of asbestos fibers—and that the connection between exposure and cigarette smoking has not been stressed enough. Says John A. McKinney, president of Johns-Manville Corp. (a leading maker of asbestos products): "Clinical evidence shows that lung cancer in asbestos workers is virtually limited to those who smoke cigarettes, and that for practical purposes, lung cancer as an asbestos-related disease would not be a problem were it not for cigarette smokers."

AIA is currently on a vigorous campaign to persuade industry workers to stop smoking.

"There is no doubt that exposure and cigarette smoking have a synergistic effect," says an ACS spokesman, "but since so many workers included in past studies were smokers or ex-smokers, it's difficult to assess the risk to non-smokers."

The synergism is acknowledged in the WHO document. "Both cigarette smoking and exposure to asbestos independently cause lung cancer. However, when present together, they act in a multiplicative fashion." The NCI/NIEHS study agrees, and admits that it would be an error to attempt to blame each type of cancer mentioned in the report on an exclusive cause.

A NEW STANDARD?—The current flareup of the asbestos controversy is of more than marginal interest to OSHA, which has been trying to modify present rules governing exposure to the material (the agency is studying the NCI/NIEHS report).

For more than three years, OSHA has been pushing a proposal to reduce the permissible level of airborne fibers in the workplace from a present value of 2 fibers/cm^3 to 0.5 fiber/cm^3 on an 8-h, time-weighted basis, with a 15-min ceiling of 5 fibers/cm^3. All limits apply only to fibers longer than 5 μm. (NIOSH, which serves in an advisory capacity, has recommended a limit of 0.1 fiber/cm^3.)

OSHA is currently evaluating the economic impact of the proposed standard, which is bound to affect both processing and use of the material.

Predictably, industry reponse to the idea has not been favorable. AIA calls the 0.5-fiber/cm^3 limit "not technically feasible," and recommends instead the adoption of 2 fibers/cm^3 as a permanent standard—one that can be realized at a reasonable cost for dust control.

On the other side of the spectrum, Roy Steinfurth, the asbestos workers' spokesman, feels that the only acceptable level is no asbestos exposure at all. The union official says that, even if the exposure level is reduced to 0.5 fiber/cm^3, a worker would breathe in 4 million fibers in an 8-h workday. He estimates that as many as 50% of the members of his organization will eventually die of asbestos-related diseases.

In Europe, there is also a move to cut down on the exposure level, which has been 2 fibers/cm^3 in the past few years. Italy, which lacks asbestos deposits, is attempting to phase out the material completely. Sweden moved to a 1-fiber/cm^3 standard a year ago, and U.K. authorities are clamoring for a reduction of the present limit.

Critics who complain that the OSHA proposal does not go far enough in protecting workers also question why the standard should apply only to fibers longer than 5 μm. Both Selikoff and Wagoner, for example, believe that the smaller fibers are even more active in producing lung tumors. And, according to Selikoff, "For each fiber longer than 5 μm, there may be as many as 100 shorter ones."

OSHA's Health Standards Project Officer William Warren says that the 5-μm length was the most practical alternative because detection equipment does not pick up anything smaller. Warren notes that available monitoring units detect only "from a few percent to 50%" of the actual number of fibers in a sample.

INSULATION DANGER—Spot checks with a number of U.S. insulation manufacturers turned up no one still making the asbestos-containing version. However, there are still tons of it

installed in chemical plants all over the world. Since the asbestos is loosely bound, the cutting, handling and tearing down of such insulation poses a health hazard.

"If the insulation is intact, there's no problem," says an engineer with Britain's ICI, Ltd., "Whenever maintenance requires stripping down and removal, we replace with a substitute material." He adds that the firm does not specify asbestos for any new plants.

"We stopped using asbestos for insulation about seven or eight years ago," claims the safety manager of a U.S. refinery. "Although it is cheaper and easier to apply, the health risks persuaded us to switch to fiberglass and calcium carbide." He further notes that although some asbestos is still used, it is of the bonded type, which does not present a potential dust hazard.

The indirect risks of asbestos insulation were highlighted in a recent study of maintenance workers at American Cyanamid's facility in Bound Brook, N.J. The survey, commissioned by the International Chemical Workers Union (ICWU), Washington, and carried out by the Mt. Sinai Medical Center, identifies the risks faced by maintenance workers (other than insulation installers) when working in the vicinity of an insulation job.

According to Larry Ahern, director of health and safety for ICWU, the results show that 44% of all workers, regardless of job function, had some form of asbestos-related lung abnormality. One group of workers exposed for less than one month experienced a significant increase in the incidence of lung disease.

Worse, the researchers discovered that many of the workers' relatives, who had not been in direct contact with the material, were affected. "About 123 wives were sick," says Steinfurth.

HOW ABOUT SUBSTITUTES?—Such popular asbestos replacements as fiberglass, mineral wool and calcium silicate may have health hazards of their own, but these have yet to be identified.

In a criteria document issued in April 1977, NIOSH said: "Fibrous glass seems to be considerably less hazardous than asbestos . . . and, until more information is available, the recommended standard can also be applied to other manmade mineral fibers." The NIOSH paper suggests a standard of 3 fibers/cm³ for fibers of less than 3.5-μm dia. Larger fibers are not thought to be respirable.

Ralph Zumwalde, an industrial hygienist at NIOSH, points out that most of the exposure to fiberglass involves fibers with diameters greater than 3.5 μm, even though some special products contain thinner ones.

"Fiberglass is an irritant, and can cause skin irritation or upper respiratory irritation," says Dr. Donald J. Billmaier, assistant medical director of Owens-Corning Fiberglas Corp. (Toledo, Ohio, "but our studies show that there are no chronic adverse health effects from using the material."

Calcium silicate is classified as a nuisance dust, not a fiber. The OSHA standard is 15 particles/cm³ for the respirable fraction.

Vincent Cavaseno

Gulf Coast buildup may ease olefin glut

☐ A more hopeful outlook for ethylene derivatives is triggering major expansions in the Gulf Coast area, and these may help alleviate, at least locally, the ethylene surplus that is predicted through the early 1980s (*Chem. Eng.*, Mar. 27, p. 80).

Consider, for example, the current buildup at the Raporte, Tex., complex of the USI Div. of National Distillers and Chemical Corp. The company, which last year increased its high-density polyethylene (HDPE) capacity to 500 million lb/yr, boosted its low-density polyethylene (LDPE) capacity by 200 million lb/yr this spring. Another ethylene-consuming addition is under way—USI will complete early next year a vinyl acetate monomer expansion. This will raise monomer capacity from 375 million lb/yr to 600 million lb/yr. The three expansions will consume a total of just under 500 million lb/yr of ethylene.

Shell has marked the tenth anniversary of its complex at Geismar, La., by inaugurating ethylene oxide and ethylene glycol trains, now about 60% completed. About a year ago, the company began work to increase oxide production from 300 to 700 million lb/yr, and glycol output from 150 to 350 million lb/yr. "This expansion will be ready for startup in early 1979," says plant manager Fred Foster. The ethylene oxide boost will require 340 million lb/yr of ethylene raw material.

Shell will also add flexibility to the Gulf Coast ethylene distribution system with a new 254-mi ethylene pipeline that will run from Mont Belvieu (just east of Houston) to Napoleonville, La., near Shell's petrochemical plant at Norco. The company is building a 1.5-billion-lb/yr ethylene plant there, which is expected to go onstream in 1981. Says A. R. Flora, Shell's olefins business manager, "The pipeline will help to eliminate spot shortages in Texas and Louisiana complexes, which are the heart of the petrochemical industry."

USI executives now speak more optimistically about the future of downstream products, and predict a better supply/demand balance. According to George Kappas, vice-president of National Petrochemicals Corp. (jointly owned by National Distillers and Owens-Illinois), HDPE is currently moving away from oversupply.

"At present," says Kappas, "there is a slight surplus of HDPE. However, producers are not building up year-end inventories, and demand is growing steadily. Consequently, in 1980 and 1981, supply and demand will be somewhat tighter than they are today." Kappas notes that domestic need for the resin has grown at an average annual rate of more than 11% since 1971. LDPE will be in balance by 1980, according to USI estimates.

DRAFT SUMMARY

ESTIMATES OF THE FRACTION OF CANCER INCIDENCE IN THE UNITED STATES ATTRIBUTABLE TO OCCUPATIONAL FACTORS

NATIONAL CANCER INSTITUTE

AND

NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES

Not for use before 3:30 p.m. EDT September 11, 1978.

This statement will address the question: "What fraction of the cancer incidence in the United States is attributable in whole or part to occupational exposure to carcinogens in the workplace?" The conventional estimates have been that this fraction is quite small, and figures of between one percent and five percent are often quoted. However, as we will show below, these estimates were admittedly speculative and were incomplete or deficient in several respects. If the full consequences of occupational exposures in the present and the recent past are taken into account, estimates of at least 20 percent appear much more reasonable and may even be conservative.

Basis of the Estimation that 20 Percent of Cancer Deaths will be Associated with Occupational Exposure to Chemicals

I. Asbestos as a Well-Studied Example

The consequences of occupational exposure to asbestos in the United States have only been fully recognized in the past year. According to estimates made by the U.S. Department of Health, Education, and Welfare, between eight and eleven million workers have been exposed to asbestos in the U.S. since the beginning of World War II. Of that total, approximately 1.5 to 2.5 million are presently employed, while the remainder -- between 6.5 and 8.5 million workers -- were formerly employed in environments with significant asbestos exposure, including 4.5 million who worked in shipyards during World War II. Of these workers, approximately four million are believed to have had heavy exposure to asbestos. Based on epidemiological studies of workers, it is estimated that 20-25 percent of heavily exposed workers die of lung cancer, 7-10 percent of pleural or peritoneal mesothelioma, and 8-9 percent of gastrointestinal cancers. These figures are probably underestimates of lifetime risks, because relatively few workers have yet been followed to the end of their normal lifespan. The total fraction of heavily exposed workers likely to die of these cancers is probably between 35-44 percent.

Of the four million heavily exposed workers, approximately 1.6 million are thus expected to die of asbestos-related cancers. Assuming that the excess risk to the 4-7 million less heavily exposed workers is one-quarter of that to the heavily exposed workers, the total number of cancers associated with asbestos in the less-heavily exposed group would be expected to be about 0.55 million, raising the total to about 2.15 million. Since most of these cancers will be manifested in the next 30-35 years, the expected average number of cancers attributable to asbestos per year in that period will average about 67,000. Such numbers would represent about 17 percent of all cancers detected annually in the United States.

II. Other Less Well Studied Examples

Arsenic

Number of workers potentially exposed: about 1,500,000.
Risk ratios*: 3-8 for lung cancer.
Estimated number of excess cancers per year: 2,100 - 7,300.

Benzene

Number of workers potentially exposed: about 2,000,000.
Risk ratios: 2-3 to 7 for leukemia.
Estimated number of excess cancers per year: 240 - 1,400.

Coal Tar Pitch Volatiles and Coke Oven Emissions

Number of workers potentially exposed: about 60,000.
Risk ratios: 2-6 for cancer of the lung, larynx, skin, and scrotum.
Estimated number of excess cancers per year: 160-800.

* Risk Ratio:

The ratio of cancers to the number expected in a normal population. A risk ratio of two means a doubling of the risk.

Vinyl Chloride

Number of workers potentially exposed: about 2,260,000.
Risk ratios; 200,4 and 1.9 respectively for hemangiosarcoma, brain and lung cancer.
Estimated number of excess cancers: 1,940.

A total of 1-3 percent of the cancers occurring in a year will be associated with these four substances..

III. Substances for which excess cancer incidence has been recorded but for which estimates of the number of workers exposed may be less accurate.

Chromium

Number of workers potentially exposed: about 1,500,000.
Risk ratios: 3-40 for nasal cavity and sinus, lung and larynx.

Estimated number of excess cancers per year: 2,400-46,000.

Iron Oxide

Number of workers potentially exposed: about 1,600,000.
Risk ratios: 2-5 for lung and larynx.
Estimated number of excess cancers per year: 1,300-5,000.

Nickel

Number of workers potentially exposed: about 1,370,000.
Risk ratio: 5-10 for lung.
Estimated number of excess cancers per year: 3,800-5,000.

Petroleum Distillates

Number of workers potentially exposed about 3,000,000.
Risk ratios: 2-6 for lung and larynx.
Estimated number of excess cancers per year: 2,400-12,000.

A total of 3-18 percent of the cancers occurring in a year will be associated with these four substances.

IV. Totals.
Thus the total excess incidence would be from 21 to 38 percent. We choose to use the figure 20 percent in order to be conservative.

Summary and Conclusions

1. The oft-quoted estimatess that only 1 percent to 5 percent of total cancers in the United States are attributable to occupational factors have not been scientifically documented.

2. Most cancers have multiple causes: it is an error to attempt to assign each cancer to an exclusive single cause.

3. Because cancer incidence is strongly dependent on age and upon duration of exposure, most cancers resulting from exposure to carcinogens will occur late in life: many epidemiological studies detect only a small fraction of early-developing cancers.

4. Past exposure to asbestos is expected to result in over 2 million premature cancer deaths in the next three decades: this corresponds to roughly 17 percent of the total cancer incidence expected in that period.

5. Reasonable projections of the future consequences of past exposure to established carcinogens suggests that at least 8 other substances may contribute substantially to cancer incidence comparable in their total effect to asbestos.

6. The projections suggest that occupationally-related cancers may comprise 20 percent or more of total cancer incidence in forth-coming decades. (This does not include cancers attributable to ionizing radiation.)

7. Although exposure to some of the more important occupational carcinogens has been reduced in recent years, there are still many unregulated carcinogens in the U.S. workplaces; a number of occupations are characterized by excess cancer risks which cannot yet be attributed to specific agents.

8. There is no sound reason to assume that the future consequences of present-day exposure to carcinogens in the workplace will be less than those of exposure in the recent past.

9. Patterns and trends in total cancer incidence (and mortality) in the U.S. are consistent with the hypothesis that occupationally-related cancers comprise a substantial and increasing fraction of total cancer incidence.

OSHA's Asbestos Standard— A Status Report

OSHA's current standard on asbestos was promulgated on June 7, 1972. It defined "asbestos" as chrysotile, amosite, crocidolite, tremolite, anthophyllite, and actinolite and included every product containing any of these minerals. For regulatory purposes, an asbestos fiber was defined as a particulate form of asbestos, longer than 5 microns, with a length-to-diameter ratio of at least 3-to-1 and a maximum diameter of 5 microns.

The 1972 standard originally established a maximum 8-hour time-weighted-average (TWA) concentration of 5 asbestos fibers per cubic centimeter of air, and a ceiling exposure limit of 10 fibers per cubic centimeter. On July 1, 1976, a further provision of the standard took effect, lowering the permissible 8-hour TWA to two fibers per cubic centimeter of air. The two-fiber limit remains in effect today.

On October 9, 1975, OSHA proposed a new regulation for asbestos. Among other things, the proposal would lower the permissible 8-hour TWA exposure level to 0.5 fibers per cubic centimeter and would reduce the permissible ceiling exposure level to 5 fibers per cubic centimeter for any 15-minute period. This proposal would not apply to the construction industry, which would be required to follow a different standard to be proposed later.

A separate asbestos standard for the construction industry has not yet been proposed, nor have hearings on the 1975 proposal been scheduled. An economic impact statement concerning the proposed revision is nearing completion.

Meanwhile, on December 2, 1975, OSHA asked NIOSH to reevaluate available information on the health effects of occupational exposure to asbestos. Completed in December of 1976, the NIOSH reexamination was forwarded to OSHA in May. Finding no evidence of a "safe" level of asbestos exposure, NIOSH recommended treating asbestos like other carcinogens by allowing only the lowest exposure level detectable by available analytical techniques. NIOSH recommended a 0.1 fiber TWA and a 0.5 fiber ceiling limit for occupational exposures to asbestos.

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Polycyclic Aromatic Hydrocarbons in Soils of a Mountain Valley: Correlation with Highway Traffic and Cancer Incidence

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■ Analyses of soils in the vicinity of a Swiss mountain town show a correlation between the content of polycyclic aromatic hydrocarbons (PAH) and the proximity to a highway. PAH contents range from 300 mg/kg dry soil near the highway to 4–8 mg/kg in the surrounding higher alps. The PAH composition ranges from three- to eight-membered rings and to heavily alkyl-substituted derivatives. The PAH mixtures are far more complex than was assumed in the past and resemble that of automobile exhaust. The low values in town close to industry but remote from the highway, and high PAH values outside of town but near the highway suggest a correlation between automobile traffic and PAH content of soils. These results indirectly suggest also a correlation between the automobile traffic and the observed mortality from cancer in this area.

An epidemological study of a Swiss mountain town has demonstrated a strong correlation between cancer incidence among the residents and the proximity of their residences to the highway (*1*). The town of 3000 inhabitants is located within the 1-km-wide base of a deep valley with predominant winds along its axis and with frequent thermal inversions. It is divided by a 40-m-tall alluvial cone into the older main section with residential housing immediately adjacent to the heavily traveled highway (4000–5000 vehicles per day) and a newer section, about 400 m from the highway and shaded from it by the alluvial cone. Until recently, this section of town was serviced only by a dead-end road.

During the period of the original study (1958–1970), death from cancer was nine times as frequent for residents near the highway. A total of 72 persons died in the old part of town of various forms of cancer, whereas only three cases occurred in the traffic-free area. Cancer mortality near the highway is higher for all groups of residents, without correlation with age, sex, occupation, and smoking habits. Thus, 32 women died; none of them had smoked.

This suggested a link between cancer incidence and environmental carcinogens, associated with the highway traffic, among them petroleum- and coal tar-based road asphalt, tire particles, lubricants, asbestos, and the components of automobile exhaust (lead compounds, polycyclic hydrocarbons, and other reactive chemicals).

We have now surveyed the soils of this valley and of the surrounding mountains for their content of polycyclic aromatic hydrocarbons (PAH), as a possible group of traffic-linked carcinogens.

Samples were taken immediately below the grass within the uppermost humus layer; those representative of the soils near the highways were collected within 1 m from the edge of the road. A dust sample was gathered from a high windowsill in the town church, 50 m from the main highway, and a soot sample was taken from the exhaust pipe of a small car and from the chimney of a residence heated with fuel oil. Most samples were handcarried to the analytical laboratory; they were kept under refrigeration in clean glass containers until extraction. Isolation of the PAH fraction and its final analysis followed the procedure of Giger and Blumer (*2*), except for the more efficient distillation into the mass spectrometer source from a glass capillary with restricted opening (*3*). During each distillation 40 spectra were obtained at 12 eV and inspected on a Finnigan 3200 mass spectrometer with a 6000 data system; the total spectrum for each sample was reconstructed by the summation of all spectra containing appreciable intensities (*3, 4*) of the PAH molecular ions.

Table I presents the total weights of the purified PAH fractions. These values are reproducible (*2*), but they may include the weights of some non-PAH impurities that were not rejected during the separation. Lower values are obtained by UV analysis, since they neglect the presence of alkylated PAH series and of still unidentified PAH components that are evident from the mass spectra.

In our interpretation we consider the 12-eV mass spectra, the UV spectra, chromatographic mobilities, and the relative volatilities as observed in the probe distillation. We believe that our structural assignments (Table II) are sound, since they rest on the correlation of these different and independent analytical parameters. The mass spectra of the PAH fraction demonstrate a nearly uniform composition in all of the soil samples. Unsubstituted hydrocarbons predominate and range from phenanthrene to seven- and eight-membered aromatic ring systems that have not been identified before in environmental samples. Each unsubstituted hydrocarbon is accompanied by alkyl-homologs in gradually decreasing concentrations to C_5 and beyond, with nearly identical abundance patterns in every series. Many isomers, differing in ring arrangement and in position and structure of the side chains, may be present. Some well-known carcinogens (benzo[a]pyrene, benzanthracene) occur together with other carcinogens and cancer initiators, whose presence in environmental samples is rarely considered (methylchrysenes). In addition, vast numbers of aromatic hydrocarbons are present whose structures are not sufficiently well known to assess their biological effect. Among these there may be many still unknown carcinogens and mutagens.

The composition of natural PAH assemblages is influenced by the processes involved in their formation (*4–6*), and chemical analysis can therefore distinguish between contributions from different sources. For instance, the relative abundance of alkylated PAH derivatives is influenced by the formation temperatures of pyrolytic PAH mixtures. At high temperatures, such as in the coking of coal, only the unsubstituted hydrocarbons are formed or survive, while at lower temperatures, for instance during petroleum formation, highly alkylated products predominate. These compositional fea-

[1] Deceased.

tures survive the processing to road asphalt and pitch (7). The mass spectra of the soil hydrocarbons demonstrate an alkylation pattern different from those of coal tar or petroleum (4, 5). The predominance of the unsubstituted hydrocarbons and the gradual decrease in concentration toward the more alkylated members speak for a pyrolytic origin at intermediate temperatures and enable us to rule out a major contribution from petroleum- or coal tar-based road asphalt or from lubricants.

In spite of the general compositional uniformity, we note some subtle differences between samples. The soot from the exhaust pipe of a car contains the same extended PAH series as the soils (a finding in disagreement with earlier analyses obtained with less highly resolving methods). The PAH mixture is depleted in the lowest boiling hydrocarbon series. Apparently, this series is not retained within the hot soot deposits in the exhaust system of the engine. Rather, these compounds pass into the atmosphere, which is consistent with the observation that the phenanthrene and pyrene series are considerably more abundant in the dust from the church, even if that sample has a similar overall composition in terms of ring systems and alkyl-derivatives. Correspondingly, the content of low boiling hydrocarbons in the soils near the highway is intermediate between the soot from the exhaust system of the car and the material collected in the church.

The soot from the chimney of a residence heated with fuel oil has a very different PAH composition. There, alkylated members of lower molecular weight PAH series predominate, and the unsubstituted hydrocarbons are in the minority. Higher ring-number series are present at low concentration or altogether absent. Thus, this soot sample reflects the compositional features that are characteristic for crude oil and its distillates, rather than for high-temperature pyrolysis products, as is the case for car exhaust.

The aromatic hydrocarbon composition of these Swiss soils is very similar to that of recent marine sediments and soils from the U.S. northeast coast. We believe that two different processes, but operating at similar temperatures, have produced a similar set of hydrocarbons. Those in the U.S. samples, and possibly also in the high Swiss Alps, originate in natural fires; they are transported through the troposphere on soot particles and enter the sediments with fallout (4, 8). Extended air transport would result in depletion of the low boiling hydrocarbons. This is observed both in the U.S. samples and those from the high alps. These hydrocarbon assemblages at concentrations near 5 ppm may well represent a worldwide PAH background. The high PAH levels at the bottom of the Swiss valley, on the other hand, cannot be attributed to the same source, especially since their concentration far exceeds the levels in the immediately surrounding alps. The association of such high PAH levels with the proximity to the highway (Table I) suggests that they are produced in internal combustion engines.

A major contribution from other PAH sources linked to the town or the highway can be excluded. Industrial and domestic heating produces some PAH. However, the low levels in town close to industry but remote from the highway, and the high level at the highway outside the town, speak for the association of the PAH production with the traffic. Aromatic hydrocarbons are also associated with the carbon used as filler in automobile tires. A comparison between tire life and gasoline consumption of automobiles suggests that even a minor conversion of the fuel into carbon can produce soot much more rapidly than it would be released by tire wear.

In combination, the geographic distribution of the hydrocarbons, the correlation between structure and processes of formation, and the chemical agreement in the PAH composition of soot in automobile exhaust with the soil hydrocarbons provide a powerful argument that car exhaust is responsible for the observed PAH accumulation in the Swiss Valley.

This work has many consequences. Our new analytical data,

Table I. Total Polycyclic Aromatic Hydrocarbons in Soils and Sediments

	PAH, mg/kg dry wt
Within town	
Center of town (470 m elev.), at highway	110
North end of town, at highway	220
South end of town, at highway	85
South end of neighboring town, at highway	300
Dust from church, center of town, 50 m from highway	100
Outlying section of town, light traffic, 400 m from main highway	21
Outlying section of town, dead-end road, 250 m from main highway, 100 m from foundry	6
Secondary road in village, at road	18
Open country	
At main highway, 750 m south of town	120
300 m from main highway in valley	15
700 m from main highway in valley	5
1000 m from main highway, alluvial plain	5
Alpine soils, side valleys	
Camp ground, 850 m elev., no through traffic	8
Mountain pass, no road, 1200 m elev.	6
Alpine meadow, 1600 m elev.	4
Soils, marshes, and sediments, USA	
Maine, forest, 115 m from secondary road	7
Cape Cod, forest, 750 m from highway, 400 m from secondary road	13
Air base, Cape Cod, sandy soil, 2 m from highway	2
Marsh, Cape Cod, 750 m from highway	19
Buzzards Bay, Mass., marine sediments, surface	4–5

Table II. Aromatic Hydrocarbon Series in Soils

Initial mass	Extent of series	Representative compounds[a]
178	To C_6	Phenanthrene (UV)
202	To C_6	Pyrene (UV), fluoranthene (UV)
228	To C_6	Benzanthracene (UV), chrysene (UV), triphenylene
252	To C_6	Benzo[a]pyrene (UV), benzo[e]pyrene (UV), perylene (UV)
276	To C_9	Anthanthrene (UV), benzo[ghi]perylene (UV)
278	To C_9	Picene, dibenzanthracene, dibenzophenanthrene
300	To C_7	Coronene (UV)
302	To C_7	Dibenzofluoranthene
326	To C_7	Heptacyclic PAH, e.g., dibenzoperylene
350	To C_7	Octacyclic PAH, e.g., benzocoronene
352	To C_6	Tribenzofluoranthene (MS, chrom., dist.)
376	To C_5	Octacyclic PAH, e.g., tribenzoperylene

Relative abundance of series, at bottom of valley: 202 > 178 ≳ 228 > 252 > 276 > 278 > 302 > 300; for other samples, see text

[a] Structural evidence was derived in all instances from mass spectra, from the chromatographic position, and the relative volatility; "UV" indicates further confirmation from ultraviolet spectra.

obtained with much improved resolution, demonstrate that automobile exhaust and environmental PAH mixtures are far more complex than was assumed in the past. Therefore, earlier analyses now appear much more limited in their power to correlate with, or to predict, public health effects. Numerous additional components of exhaust and of environmental samples must now be considered in their possible roles as carcinogens, tumor inducers or promoters, and mutagens. The demonstrated correlation between highway traffic and the production of carcinogens strengthens indirectly also the correlation between highway traffic and the observed mortality from cancer. The implications for public health, for city and highway planning, and for efforts to control engine exhaust are considerable.

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Determination of Elemental Sulfur by Gas Chromatography

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■ Elemental sulfur was determined by combining electron capture detection with cyclohexane extractions of coal, particulate, and soil samples and with resin sorption of water samples. The sensitivity for sulfur permitted its determination in environmental samples at sub parts per billion levels. The extraction procedures allowed for a minimum of cleanup prior to the rapid and selective gas chromatography.

The usual procedures for the determination of elemental sulfur are reduction to the sulfide or oxidation to the sulfate. These techniques generally lack the selectivity and sensitivity of reported gas-liquid (*1–7*), thin-layer (*8*), and liquid chromatographic (*9*) procedures. These are apparently useful for sulfur determinations, but none has been applied to the quantitation of elemental sulfur in environmental samples.

This paper describes the methodology for the determination of elemental sulfur in stack particulate, soil, coal, and water samples using gas-liquid chromatography for the separation from other components present in the sample and electron capture for the selective and sensitive detection.

Experimental

Apparatus. A Tracor Model 550 equipped with a Ni 63 electron capture detector (ECD) and a Beckman Model GC-5 equipped with a helium discharge ECD were used for the gas chromatography.

Glass columns, 2 m × 4 mm i.d., were packed with the solid supports and liquid phases listed in Table I. These columns were silanized with four injections of 25 μL each Silyl 8 (Pierce Chemical Co.) before use.

A Du Pont 21-490-1 gas chromatograph–mass spectrometer (GC–MS) was used for positive identifications of the elemental sulfur extracted from various environmental samples.

Reagents. Cyclohexane (J. T. Baker Chemical Co.), 98% grade, was further purified by distillation. Sulfur standards used for quantitation were prepared by volumetric dilution of a solution having 10 mg of 99.999% sulfur in 50 mL of cyclohexane.

The 60–100 mesh Florisil (Floridin Co.) used to clean up the sample extracts was calcined at 540 °C by the manufacturer and activated for 5 h at 130 °C prior to use.

Analytical Procedures

Coal, Particulate, and Soil Extractions. One-gram samples of crushed coal which had passed a 60 mesh sieve were Soxhlet extracted for 24 h. Large 35 × 90 mm glass thimbles were used to prevent plugging of the Soxhlet device by the fines from the coal samples. The 90 mL of cyclohexane used for the extraction were then quantitatively transferred to volumetric flasks and diluted to 100 mL. Five-μL aliquots of this cyclohexane solution were subjected to gas chromatography without further cleanup.

Particulate samples were collected from 4-in. sampling ports located approximately half-way up the stack of a local power plant. Three types of samples were collected. Particulates #1 were from the accumulation in the ports. Particulates #2 were collected by drawing the atmosphere from inside the stack through a glass tube containing a glass wool plug. Particulates #3 represented that portion which settled onto horizontal trays placed inside the stack.

Ten grams of particulates were extracted in a Soxhlet for 24 h in 25 × 85 mm glass thimbles using approximately 50 mL of cyclohexane. The cyclohexane was transferred to volumetric flasks and diluted to 50 mL with cyclohexane. Five-μL aliquots of this solution were gas chromatographed without further cleanup.

Ten-gram amounts of local soils were Soxhlet extracted with cyclohexane as above. The extracts were concentrated

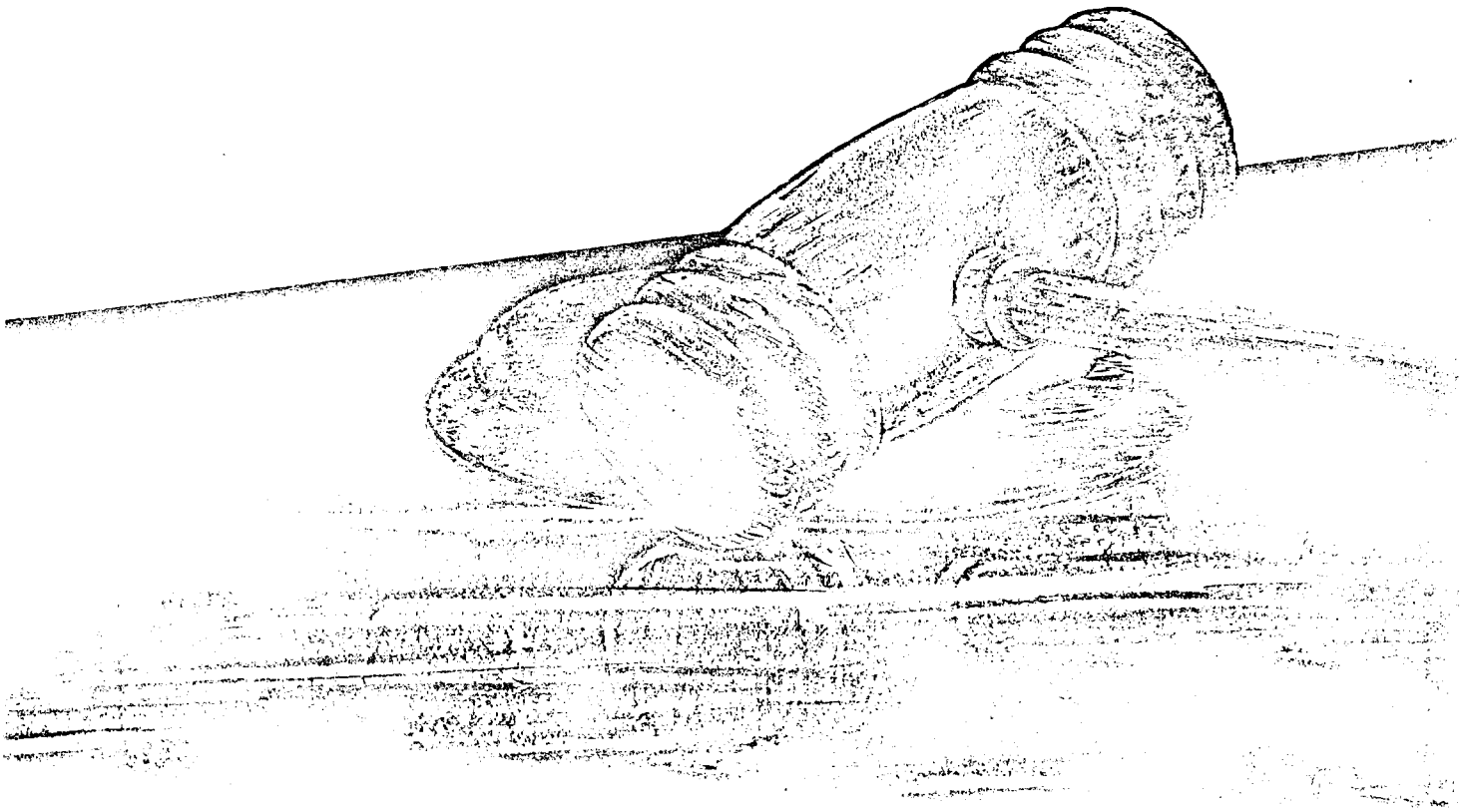
Table I. Gas Chromatographic Data for Elemental Sulfur

Liquid phase	Solid support[a]	t_R, min	Column temp, °C	Flow, mL/min
5% OV-210[b]	C	2.3	180	75
4% SE-30/ 6% OV-210[b]	G	5.2	200	75
3% OV-1[b]	C	1.7	120	75
5% OV-1[b]	C	1.8	120	75
1.5% OV-17/ 1.95% OV-210[c]	G	2.5	200	160
10% DC-200[c]	G	3.6	200	160

[a] C is Chromosorb W HP, 80–100 mesh; G is Gas Chrom Q, 100–120 mesh. [b] Tracor Model 550; detector, 340 °C; injector, 220 °C. [c] Beckman GC-5; detector, 310 °C; injector, 240 °C.

A GUIDE TO THE WORK-RELATEDNESS OF DISEASE

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health



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A GUIDE TO

THE WORK-RELATEDNESS OF DISEASE

Marilyn K. Hutchison, M.D., Editor

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
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PREFACE

The goal of the National Institute for Occupational Safety and Health (NIOSH) is to protect the health and safety of working men and women. Within the context of this program are NIOSH efforts that are directed toward the identification of those disease conditions that are causally related to occupation, as necessary prerequisite to their prevention.

This guide is presented primarily as an aid to State agencies and others concerned with occupational disease compensation. The Guide presents one method for assembling and evaluating evidence that may be relevant in determining the work-relatedness of a disease in an individual. Information on five disease-producing agents is presented to illustrate the decision-making process. It should be noted that such information may not be complete and does not necessarily reflect the most recent data regarding health standards and epidemiologic studies.

NIOSH will welcome suggestions for improvement of the Guide based upon experience with its use.

ABSTRACT

This Guide discusses various factors associated with establishing the relationship between disease and occupation. Prepared as an aid to State agencies, physicians, and others concerned with workers' compensation for occupational disease, the publication describes a method for collecting, organizing, and appraising medical, occupational, and other evidence with the aim of determining the probable work-relatedness of a given disease. Illustrative material on five disease-producing agents is included. The Guide also contains a list of occupations with potential exposure to selected agents, and other information that may be useful to those with decision-making responsibility in cases of occupational disease.

ASBESTOS

Introduction

Asbestos is a mineral fiber, and is the name given to about thirty silicate compounds. Of these, only the following 5 are of significance in industry:

Chrysotile (white asbestos)
Amosite
Tremolite
Crocidolite (blue asbestos)
Anthophyllite

Chrysotile accounts for about 97 percent of all the asbestos used in this country.

Asbestos is widespread in the environment because of its extensive use in industry and the home. Over 3,000 products contain asbestos.

Because of this wide usage, it may be difficult at times to determine if a disease arising from asbestos is occupational in origin. For example, the air of some relatively new apartment buildings has been found to contain more asbestos fibers than the maximum recommended levels in industry. The source of the fibers in the apartment buildings is the insulating materials used in the ventilating system.

Exposure to asbestos can produce a lung fibrosis called asbestosis. The onset of asbestosis is usually gradual, developing over a period of 10 to 30 years of exposure to significant concentrations of asbestos. Occasionally, from very massive exposures, it may develop more quickly.

Asbestos is also a cancer producing agent (bronchogenic carcinoma, mesothelioma) and can cause certain specific skin diseases (asbestotic subcutaneous granulomatosis and asbestotic cutaneous verruca). Heavy exposure to dust containing asbestos can cause skin irritation. Epidemiologic studies (experience with groups of people) and animal studies have shown that increased exposure to any of the types of asbestos increases the risk of lung cancer (bronchial carcinoma). This carcinoma appears to be related to the degree of exposure to asbestos, the type of asbestos and cigarette smoking. It is also significant that cigarette smoking in men and women greatly increase the risk of lunk cancer in those who are exposed to asbestos. Smoking is a factor that should be considered when determining whether lung cancer is caused, wholly or in part, by an occupational exposure to asbestos.

Mesothelioma, a rare malignant tumor of the membrane which lines the chest cavity and the abdominal cavity, is occurring with increasing frequency in workers with exposure to asbestos. The development of this tumor apparently is not related to the amount of asbestos inhaled and it is found in persons not having asbestosis. Levels of exposure which are within accepted standards for protection against asbestosis, may not

protect against mescthelicma.

An increased incidence of malignancy of the stomach and colon has been reported among insulation workers using asbestos.

Occupations with Potential Exposure to Asbestos

Acoustical Product Makers
Acoustical Product Installers
Air filter makers
Asbestos-cement products makers
Asbestos-cement products users
Asbestos-coatings makers
Asbestos-coatings users
Asbestos-grout makers
Asbestos-grout users
Asbestos-millboard makers
Asbestos-millboard users
Asbestos-mortar makers
Asbestos-mortar users
Asbestos millers
Asbestos miners
Asbestos-paper makers
Asbestos-paper users
Asbestos-plaster makers
Asbestos-plaster users
Asbestos sprayers
Asbestos workers
Asphalt mixers
Automobile repair garage workers
Brake lining makers
Building demolition workers
Carders (asbestos)
Caulking compound makers
Caulking compound users
Clutch facing makers
Cobbers (asbestos)
Construction workers
Crushers (Asbestos)
Fiberizers (Asbestos)
Fireproofers
Firemen
Furnace filter makers
Gasket makers
Heal resistant clothing makers
Insulation workers
Inert filter media workers
Ironing board cover makers
Laboratory hood installers
Laggers
Paint makers
Pipe insulators
Plastics makers
Pump packing makers
Roofers
Roofing materials makers
Rubber compounders
Shingle makers
Ship builders
Ship demolition workers
Spinners (Asbestos)
Talc miners
Talc workers
Textile flameproofers
Textile workers
Undercoaters
Vinyl-asbestos tile makers
Vinyl-asbestos tile installers
Weavers (asbestos)

Medical Evaluation

(Also, See Decision-Making Process)

In addition to the usual medical history, the following should be considered:

1. Any history of diseases of the heart or lung or abnormal tissue growth should be carefully evaluated to determine the relationship between the previous disease and the claimant's present condition.

2. A respiratory questionnaire, a sample of which is shown in Appendix, can be useful in evaluating the extent and importance of respiratory symptoms such as:

   - breathlessness
   - phlegm (sputum) production
   - chest pain
   - cough
   - wheezing

Asbestosis

Shortness of breath upon exertion is usually the first symptom, frequently accompanied by a dry cough. This symptom develops after several years of progressive pulmonary fibrosis. As asbestosis progresses, the following signs and symptoms are observed:

- cough with production of sputum
- anorexia (loss of appetite)
- secondary respiratory infections that are difficult to control
- rapid breathing
- repetitive end-inspiratory crackles (crackling sounds heard in the lower part of the lungs through stethoscope when employee completes each of a series of inhaled breaths)
- orthopnea (breathing difficulty in a recumbent position)
- cyanosis (change in skin color to bluish, grayish, slatelike or dark purple)
- decrease of chest expansion
- digital clubbing (rounding of the ends, and swelling of the fingers and/or toes)
- sequelae (other resultant diseases) including cor pulmonale (right heart failure), bronch ogenic carcinoma (lung cancer), stomach or intestinal cancer, or pleural carcinoma (cancer of the membrane lining the chest)

Fibrosis results in alveolo-capillary block (impaired ability of the lungs to transfer oxygen into the blood). This impairment is often more severe than is indicated by chest x-rays.

Mesothelioma

In cases of mesothelioma, the rare malignancy noted above, there may be a long latent period, as much as 40 years, between initial exposure to asbestos and the development of the tumor.

Mesothelioma of the peritoneum (membrane surrounding the abdominal organs) is usually accompanied by abdominal swelling and pain that is not concentrated in a particular area. Signs and symptoms of this type of tumor (which may be associated with asbestos exposure) include:

- weight loss
- obstruction of the bowel
- excessive accumulation of fluid in the abdominal cavity (ascites) is almost always present

This malignant tumor of the peritoneum may spread to the chest cavity.

With mesothelioma of the pleura, complaints include chest pain and breathlessness. Signs and symptoms of pleural mesothelioma include:

- pleural effusion (accumulation of fluid in the space around the lungs)
- the tumor may grow outward through the chest wall in the form of a lump beneath the skin (subcutaneous lump)
- the tumor may spread to involve bone, lymph glands (nodes) mediastinum (area between the right and left lungs), and pericardium (the sac enclosing the heart). As a result, the supraclavicular nodes may become enlarged, ribs may develop tumors, and obstruction of the superior vena cava (major vein draining the upper portion of the body) may occur.
- in addition, pericardial effusion (fluid in the heart cavity) may occur, causing tamponade.

Laboratory

(See Decision-Making Process)

Additional tests which will assist in arriving at a correct diagnosis are:

Chest X-rays

Findings should be classified according to the ILO/UC 1971 Classification of the Radiographs of the Pneumoconioses. (Appendix B)

Findings for asbestosis vary, but the usual picture shows a density in both lungs, with the lower one-third of the lungs involved. In the affected area there is a "ground glass" appearance.

As asbestosis progresses, more and more of the lung is involved, except the apices (tips of the lungs). The X-rays will show gradual obscuring of the border between the lungs and the diaphragm. It may show shadows from the presence of nodules.

X-ray findings usually will show the following as the asbestosis progresses:

- reduced radiographic volume
- formation of cysts combined with increased size of the heart, dilation (enlargement) of the proximal pulmonary arteries (arteries which lead from the heart to the lungs)

Lung Function Tests

Reduced lung capacities and other lung changes do not differ from those resulting from other forms of lung fibrosis, both occupational and nonoccupational. Therefore, the results of lung function tests alone or chest X-ray findings alone do not lead to diagnosis of asbestosis. Asbestos bodies in lymph nodes indicate exposure, but not necessarily asbestosis.

- Asbestosis causes a reduction in the vital capacity (VC) of the lungs and a reduction in total lung capacity (TLC). These capacities are further reduced as the disease progresses.
- The residual volume (RV)of the lungs will be normal or slightly increased.
- The lungs' diffusing capacity for carbon monoxide (D_L) will be reduced.

Other lung function test results which are found in asbestosis include:

- Increased minute ventilation (amount of air breathed in one minute)
- Reduced oxygenation of the arterial blood (arterial hypoxemia)
- Increased static transpulmonary pressures
- Decreased lung compliance

An exercise test will result in an increased amount of air required during physical effort, decreased oxygen in the blood, leading to cyanosis.

Sputum Examination

Asbestos fibers or bodies may be found in the sputum. These indicate asbestos exposure, but not necessarily asbestosis. Where cancer cells are present in the sputum, and chest X-ray findings are normal, bronchoscopy may be necessary to confirm and locate the lung tumor.

Skin Tests--The following tests should be performed by the physician to exclude possible infectious diseases:

1. PPD (tuberculin test)
2. blastomycin
3. histoplasmin
4. coccidioidin

Epidemiological Data

Various epidemiologic studies have demonstrated the relationship between asbestos and lung disease, including mesothelioma, in such trades and occupations as mining, insulation installation, textiles, paint, electrical industries, and many other occupations as a result of the widespread use of this substance.

The available information indicates evidence of a dose-response relationship for asbestos exposure and the risk of asbestosis and/or bronchogenic carcinoma. However, much of this information is epidemiological in nature and there is little correlation between epidemiologic data and environmental exposure data. For this reason and others, including the long latent period for the development of carcinomas, it is difficult to develop a specific dose-response relationship. This should be taken into consideration when referring to the following material:

Enterline[1] has reported an exposure-response relationship between asbestos exposure (evaluated as millions of particles per cubic foot years) and the risk of malignant and nonmalignant respiratory disease. Enterline's data indicates that the risk of respiratory cancer increased from 166.7 (standardized mortality ratio) at minimum exposure to 555.6 at cumulative exposures exceeding 750 million particles per cubic foot years. Enterline's data is summarized in a table by NIOSH[2].

Murphy[3] reported that asbestosis was 11 times more common among pipe coverers in new ship construction than in a control group. The first asbestosis was found after 13 years of exposure to an estimated cumulative dose of about 60 million particles per cubic foot years. After 20 years, asbestosis prevalence was 38%. Murphy reported no asbestosis for men exposed to 60 mppcf years but 20% asbestosis in men exposed to 75-100 mppcf years. Murphy reports atmospheric dust concentrations ranged from 0.8-10.0 mppcf depending on the different operations evaluated. Asbestosis was considered present if the worker had at least three of the following: vascular rales in two or more sites, clubbing of the fingers, vital capacity of less than 80% predicted, roentgenography consistent with moderately advanced or advanced asbestosis, shortness of breath on climbing one flight of stairs.

The Pennsylvania Department of Health[4] reported a study of asbestos dust concentrations in two plants (one studies from 1930-1967 and the other from 1948-1968). 64 cases of asbestosis were reported. In the two plants, the study indicates that the air concentrations of particulates were generally less than five mppcf and in many cases less than two mppcf.

Epidemiological evidence is also available relating the development of mesothelioma with exposure to asbestos. Selikoff[5,6] reported 14 deaths

from mesothelioma in 532 abestos insulation workers from 1943-1968. No deaths from mesothelioma would be expected from the same number of individuals in the general population.

Evidence of Exposure

Historically, there have been two air sampling and analysis methods to determine the quantity of asbestos in the workplace environment. The earlier light field impinger count method allowed only a measure of the overall dust level in the air rather than focusing on the amount of asbestos fibers in the air. The current fiber count method satisfactorily determines the amount of asbestos fibers in the air. It is performed by collecting airborne materials on a membrane filter and then counting the fibers using a phase contrast microscope at a 400 to 450 times magnification ratio (400X-450X).

Asbestos fibers occur in varying lengths and diameters. As of the publication of the guide, the Occupational Safety and Health Act (OSHA) establishes maximum allowable limits for asbestos fibers greater than five micrometers (um) in length. OSHA limits such asbestos fibers to no more than five fibers per cubic centimeter of air (based on an eight hour time-weighted average exposure).

OSHA further requires that no workers be exposed to more than 10 asbestos fibers (greater than five um in length) during any one 15 minute period of time.

For samples collected by the field impinger count method, results may be compared to the pre-1970 limit (TLV) of five million particles per cubic foot of air.

Occupational exposure to asbestos fibers five um in length or greater, at quantities averaging more than five fibers per cubic centimeter of air or frequent exposures to more than 10 such fibers during a 15-minute period of time is evidence of a possible causal relationship between disease and occupation.

Toxicological

(See References 1-6, Appendix A)

Conclusion

The diagnosis of occupational asbestosis is based on meeting the following criteria:

1. Confirmed history of occupational exposure to asbestos.

2. X-ray finding;s compatible with those indicating asbestosis according to ILO/UC 1971 "Classification of Radiographs of the Pneumoconioses."

3. Pulmonary impairment, particularly a decrease in lung diffusing capacity and an increase in alveolar-arterial oxygen difference,

as demonstrated by lung function tests.

The diagnosis of occupational mesothelioma is based on meeting the following criteria:

1. Confirmed history of occupational exposure to asbestos.

2. Pathological evidence of mesothelioma.

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C. SAMPLE RESPIRATORY QUESTIONNAIRE

Use the actual wording of each question. Put X in the appropriate space after each question. When in doubt, record "NO."

PREAMBLE: I am going to ask you some questions mainly about your chest. I should like you to answer 'YES' or 'NO' whenever possible.

	YES	NO	N/A
1. Do you usually cough first thing in the morning or on getting up? (Count a cough with first smoke or on first going out of doors. Exclude throat clearing or a single cough.)	___	___	
2. Do you cough like this on most days for as much as three months each year?	___	___	___
3. Do you cough at work?	___	___	
4. Do you usually bring up some phlegm from your chest first thing in the morning or on getting up? (Count phlegm with the first smoke or on first going out of doors. Exclude phlegm from the nose. Count swallowed phlegm.)	___	___	

5. Do you bring up phlegm like this on most days for as much as three months each year? ___ ___ ___

6. In the past three years, have you had a period of (increased) cough and phlegm lasting 3 weeks or more? ___ ___

7. Have you had more than one such period? ___ ___

8. Does your chest ever feel tight or your breathing become difficult? ___ ___

9. Do you get this apart from colds? ___ ___

(If YES: specify...(Interviewer to code)

(a) With Exercise ___ ___

(b) At Work ___ ___

(c) Any Other Time ___ ___

If disabled from walking by skeletal or other physical disability put 'X' here. ___

10. Are you troubled by shortness of breath, when hurrying on the levels or walking up a slight hill? ___ ___

(If 'NO' omit questions 11 and 12)

11. Do you get short of breath walking with other people of your own age on level ground? ___ ___

(If 'NO' omit question 12)

12. Do you have to stop for breath when walking at your own pace on level ground? ___ ___

13. Do you usually have a stuffy nose or catarrh at the back of your nose in the winter? ___ ___

14. Do you have this in the summer? ___ ___

(If "NO" to both questions 13 and 14, go to question 16)

15. Do you have this on most days for as much as three months each year? ___ ___

16. During the past 3 years have you had any chest illness which has kept you off work or from your usual activities for as much as a week? ___ ___

17. Did you bring up more phlegm than usual in any of these illnesses? ___ ___

18. Have you had more than one illness with phlegm like this in the last 3 years? ___ ___

HAVE YOU EVER HAD:

(Give relevant details after each positive answer.)

19. An injury or operation affecting your chest? _____ ___ ___

_____

20. Heart trouble? _____ ___ ___

_____

21. Bronchitis? _____ ___ ___

_____

22. Pneumonia? _____ ___ ___

_____

23. Pleurisy? _____ ___ ___

_____

24. Pulmonary Tuberculosis? _____ ___ ___

_____

25. Bronchial Asthma? _____ ___ ___

_____

26. Eczema? _____ ___ ___

_____

27. Dermatitis? _____ ___ ___

_____

28. Pneumoconiosis? _____ ___ ___

_____

29. Byssinosis? _____ ___ ___

_____

30. Other chest troubles? _____ ___ ___

_____

31. Have you ever smoked? _____ ___ ___

(Record 'NO' if subject has never smoked as much as one cigarette a day, or 1 oz. tobacco a month, for as long as one year)

32. Age when stopped_____years. Was this in the last month? ___ ___

If 'YES' to 31 and 32, fill in figures below:

	AMOUNT SMOKED NOW	AMOUNT SMOKED BEFORE STOPPING
Cigarettes/day (Average including weekends)	___	_____
Oz. tobacco/week (handrolled)	___	_____
Cigars/week (large)	___	_____
Cigars/week (small)	___	_____

OCCUPATION (1st Interview Only)

(Record on lines the years in which subject has worked in any of these industries, e.g., 1960-1963)

		YES	NO
33.	Have you ever worked in a dusty job? _____	___	___
34.	In a coal mine _____	___	___
35.	In any other mine? _____	___	___
36.	In a quarry? _____	___	___
37.	In a foundry? _____	___	___
38.	In a pottery? _____	___	___
39.	In a cotton, flax or hemp mill? _____	___	___
40.	With asbestos? _____	___	___

41. In any other dusty job? _____ ___ ___

If 'YES', specify _____

_____

42. Have you been exposed regularly to irritating gas or chemical fumes? ___ ___

_____

If 'YES', give details of nature and duration _____

_____

OCCUPATION (Follow-Up only)

43. What is your present job? _____

44. How long have you been doing it? _____

45. What was your previous job in the factory? _____

Taken with minor changes from Operating and Medical Codes of Practice for Safe Working with Toulene Diisocyanate, Health Advisory Committee, British Rubber Manufacturers' Association Ltd.