~~Hiccoughs~~. *
Lewinsohn paper

AUG 27 1979

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

Connecticut River Valley Local Section

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Ronald Jones

August 22, 1979

The first meeting of the 1979-80 year of the Connecticut River Valley Section of the AIHA will be held on Wednesday, September 12, 1979, at Valle's Steak House, Airport Road Exit off Route 91 in Hartford, Connecticut.

Menu: Roast Beef $9.25
Stuffed Boneless Breast of Capon 8.50

Speaker: Dr. Hilton C. Lewinsohn
Corporate Medical Director, Raybestos Manhattan
Dr. Lewinsohn is an expert on pneumonoconiosis and asbestos-related disease. Besides his work at Raybestos Manhattan, he is also a lecturer in epidemiology at Yale.

Topic: "Asbestos and Fiber Carcinogenesis"

Time: Cocktails at 6:00 p.m., Dinner at 7:00 p.m.

For reservations contact Elaine Briggs at 446-4771. Please indicate your choice of meal. Reservations must be made by September 7th. Please notify me if you are canceling your reservation as you will be expected to pay for the meal whether you attend or not.

Sincerely,

Elaine S. Briggs

Elaine S. Briggs
Secretary

Dues for 1980 are now payable. Please send check to Mr. Art Abrams, P.O. Box 571, Westport, CT 06880. Deadline is December 31, 1979.

Name: _____

Affiliation: _____

Mailing Address _____

_____ Zip: _____

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Lewinsohn papers *

AUG 17 1979

Olin

CORPORATE HEALTH AFFAIRS
ENVIRONMENTAL HYGIENE AND TOXICOLOGY DEPARTMENT
91 Shelton Avenue, New Haven, Connecticut 06511
(203) 789-5000

August 14, 1979

Hilton C. Lewinsohn, M.D.
Corporate Medical Director
Raybestos Manhattan
100 Oakview Drive
Trumbull, Conn. 06611

Dear Dr. Lewinsohn:

Thank you for agreeing to be our speaker at the September 12 meeting of the Connecticut River Section of the American Industrial Hygiene Association. The location of the meeting has not been decided yet but we usually meet either in the Hartford or New Haven area. I will let you know the exact location as soon as it is available. Our meetings consist of a cocktail hour from 6-7, a dinner from about 7-8:30 and a presentation by a guest speaker from about 8:30-9:15.

The subject you chose to speak on "Asbestos and Fibre Carcinogensis" is of great interest to industrial hygienists. I appreciate your willingness to discuss this topic at our local section meeting.

Sincerely,

Brad Garber

Brad T. Garber, Ph.D.
President Elect
Connecticut River Valley Section
American Industrial Hygiene
Association

BTG/abi

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INTRODUCTION AND DEFINITIONS

1. Asbestos

According to Tibor Zoltai (1978):

> "Until a few years ago there was no problem with the asbestos-related mineralogical terminology. Mineralogists knew exactly what other mineralogists meant when they used terms like asbestos, asbestiform, fibrous, and acicular, even if some of these terms, like asbestiform, are not always defined in textbooks. The last syllable of asbestiform (that is -form) is consistent with several adjectives used for the description of textures or crystallization habits (e.g., reniform, filiform, dentiform, colloform). Consequently, it is understood without question, that asbestiform is a descriptive term for a certain texture of crystallization habit."

The mineralogical calm was shattered when asbestos hit the headlines in the lay and scientific press and environmentalists and scientists in the public health field, as well as engineers and lawyers, "introduced a redefinition of most of the critical expressions, in spite of the objection of leading mineralogists". (Zoltai, 1978)

Asbestos as defined by mineralogists is a generic term for a variety of hydrated silicate minerals which crystallize in the form of long, strong and flexible fibers, aggregated in parallel or radiating bundles from which the fibers can easily be separated.

Asbestos has been defined by the Minnesota Distric Court during the Reserve Mining Company trial as a "generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils". This definition allows all amphiboles and a number of other minerals to become possible candidates for inclusion in the term asbestos. Zoltai points out that on crushing amphiboles always produce acicular fragments because of perfect prismatic cleavage. Acicular fragments are not fibers, are not flexible and are not composed of ~~by~~ fibrils. However, they may be indistinguishable from asbestos fibers in routine electron microscopic examination. For this reason, the Minnesota District Court defines a fiber as a "mineral which is at least three times as long as it is wide" and this redefinition was soon adopted by most environmental and public health scientists.

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The Court's definition of fiber simplified the position and only retained the shape of the particle as a decisive criterion. This meant that all acicular amphibole cleavage fragments became fibers and it was indirectly implied that all amphibole minerals became asbestos.

Mineralogists objected to these oversimplified definitions and two less frequently used terms, "asbestiform" and "asbestos-like", were redefined in line with the new definitions of asbestos and fiber. "The new definitions were introduced in the Minnesota courtroom, and subsequently in the language of the news media and the environmental literature.

Asbestiform became a prefix added to the name of any mineral which is known to occur as "asbestos on occasion and/or produce "fibers" when crushed."

"Asbestos-like was defined as any hydrous silicate particle which is at least three times longer than wide, that is, which is a "fiber".

In 1977, the U. S. Bureau of Mines published a circular entitled "Selected Silicate Minerals and Their Asbestiform Varieties: Mineralogical Definitions and Identification-Characterization".

The U. S. Bureau of Mines definition of a mineral fiber is as follows: "The smallest elongated crystalline unit that can be separated from a bundle or appears to have grown individually in that shape, and that exhibits a resemblance to organic fibers."

The occurrence of a mineral in bundles of fibers, resembling organic fibers in texture, from which the fibers can usually be separated, is described by the term "fibrous". Asbestiform refers to a specific type of mineral fibrosity in which the fibers and fibrils possess high tensile strength and flexibility.

Table 1. lists some common silicate minerals and their asbestiform varieties together with their relationships and formulas.

TABLE 1. - Selected silicate minerals and their asbestiform varieties

Mineral	Asbestiform variety
AMPHIBOLE GROUP	
Anthophyllite: $(Mg,Fe^{+2})_7 Si_8O_{22}(OH,F)_2$...	Anthophyllite asbestos
Cummingtonite-grunerite: $(Mg,Fe^{+2})_7 Si_8O_{22}(OH)_2$	Cummingtonite-grunerite asbestos.
Tremolite-actinolite: $Ca_2(Mg,Fe^{+2})_5Si_8O_{22}(OH,F)_2$	Tremolite-actinolite asbestos
Riebeckite: $Na_2Fe_3^{+2} Fe_2^{+3} Si_8O_{22}(OH,F)_2$...	Crocidolite
SERPENTINE GROUP	
Serpentine: $Mg_6Si_4O_{10}(OH)_8$.....	Chrysotile

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2. Man-Made Mineral Fibers

Man-made mineral fibers have been produced for more than 40 years. They comprise glass fibers, rock wool or mineral wool. They are all amorphous silicates in contrast to naturally occurring fibers, such as asbestos, which is crystalline in structure and differs chemically. Glass fibers are composed of either borosilicate or calcio-alumina silicate glass.

Man-made mineral fibers are usually coated with a binder, commonly a thermosetting resin of the phenol-formaldehyde type, which is fully polymerized and therefore chemically inert in the finished product. Rock wool may be produced with only an oil as a lubricant, and for special applications, some glass fibers of very fine diameter are produced without any coating at all.

3. Health effects relative to fiber exposures

Most epidemiologic studies of health affects relative to fiber exposures have been concerned with the asbestos minerals. There is epidemiologic evidence to indicate that all types of commercial asbestos, i.e., chrysotile, crocidolite, amosite, tremolite asbestos and anthophyllite asbestos, when inhaled in sufficient concentrations over a sufficient length of time, can cause fibrosis of the lungs (asbestosis) and increase the risk of lung cancer. All but anthophyllite asbestos have been associated with malignant mesothelial tumors.

The relative health risk posed by exposure to the asbestos minerals may be related to the fibril composition, crystal structure, size, shape and total surface area. The relative chemical reactivity of the fibril surface, according to Ross, is predicted to be:

chrysotile < anthophyllite < amosite < crocidolite,

on the basis of the types of oxidation-reduction and exchange reactions that may occur. The 1972 Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer states that "Evidence for an important difference in risk in different occupations and with the type of asbestos has increased. The risk is greatest with crocidolite, less with amosite and apparently less with chrysotile. With amosite and chrysotile, there appears to be a higher risk in manufacturing than in mining and milling. There is also evidence from population studies that a proportion of cases of mesothelioma have no known association with exposure to asbestos."

10 The Known Facts

SLIDE 1 People exposed to asbestos dust as a result of mining, milling, handling or using the raw material in manufacturing processes are known to have developed certain diseases. Although these diseases are apparently directly associated with such exposure, none of the lesions described is actually specifically and solely attributable to asbestos dust.

TR 2 Asbestosis, the first disease process found to be associated with exposure to asbestos would be virtually indistinguishable from other forms of

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(5) SLIDES

pulmonary fibrosis in the absence of a history of asbestos exposure or other indicators, such as asbestos bodies in the sputum or lungs or pleural thickening, hyaline pleural plaques or calcified pleural plaques. The disease process is one of pulmonary fibrosis in which the lungs become scarred, lose their elasticity, become less compliant and eventually cannot transfer oxygen into the bloodstream. Asbestosis was first described in the 1920's and it has since been clearly demonstrated that its development is dependent upon the dust concentrations to which persons were exposed as well as the length of time during which such exposure occurred. It would appear that, because of differences in dust conditions in industry, the health experience of exposed populations in different sectors of industry differs. Asbestosis affects insulation workers more often than it does workers in friction materials manufacture or mining. Asbestos textile workers are second to insulation workers in the table of prevalence of this condition in different groups of exposed workers.

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It is, however, a fact that for many years asbestos has been used less and less in insulation materials and dust control systems have become more and more efficient in textile plants as well as in all other sectors of industry using asbestos. Unfortunately, very little data exists which can be utilised to derive meaningful dose-response relationships for the establishment of a universally acceptable industrial hygiene standard and at present, the 1968 recommendations of the British Occupational Hygiene Society remain the basis for the level of two fibers per c.c. adopted by OSHA in the U. S. A. It should always be recognised that the standard was five fibers per c.c. from 1972 until 1976 and the present level of two fibers per c.c. only became effective three years ago. Furthermore, the "Two Fiber Standard" was not officially recognized in the U. K. until 1970 after the U. K. Asbestos Regulations, 1969, took effect. No population is available for study in the U. K. or U. S. A. where it can be documented that exposure was never above two fibers per cc. or where such low exposures have existed for twenty years or more. The outcome of studies now being finalised in the U. K. is eagerly awaited.

TR 4

TR 5 6

TR 7

SLIDE 27

Lung cancer is a common complication of asbestosis. [redacted] There are, however, three coexisting facts which need to be considered in order to place this problem into its true perspective. First, it has been clearly demonstrated that cigarette-smoking asbestos workers have a far higher chance of dying from lung cancer than non-smokers. [redacted] Secondly, there is evidence to suggest that asbestosis and

SLIDE 28

other lung conditions, may be commoner in smokers than non-smokers. Thirdly, because disease seen today in asbestos workers was probably induced by first exposure in past dusty working conditions, fifteen to twenty-five years or more ago, modern industrial conditions may (and probably do) have a far lower risk attached to them. There is some evidence to suggest that populations exposed to better condtions are beginning to show an improved health experience pattern, although the excess of lung cancer deaths has not completely disappeared yet.

SLIDE 29

SLIDE 30

TR 8

Diffuse malignant mesothelioma of the pleura or peritoneum has been generally accepted as being associated with asbestos exposure since the early 1960's. This previously very rare tumor has been recognized and diagnosed more frequently in the past ten or fifteen years and in most reported series, approximately 80% are associated with an exposure to asbestos. In the crocidolite mining areas, in shipyards where crocidolite and amosite were used in substantial amounts and in certain selected occupations, such as gas-mask manufacturing using crocidolite, this tumor appears to occur more frequently than in mining areas or operations processing chrysotile asbestos. The International Agency for Research on Cancer in 1972 proposed that the risk of developing mesothelioma appeared to be greater from exposure to crocidolite, less from amosite and least from chrysotile and in many countries the use of crocidolite has now been banned or more stringently controlled. Although anthophyllite has been associated with asbestosis, lung cancer and pleural plaque formation, no cases of mesothelioma have been described in association with this asbestiform mineral. It has recently been shown that mesothelioma can occur in populations not exposed to asbestos, but living in an area where zeolites appear to be the causative factor.

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Two other cancer sites have been identified as being more prone to tumors related to asbestos and they are the gastro-intestinal tract (stomach, colon, rectum) and the larynx. Although there is epidemiological evidence to confirm that an excess number of deaths result from G. I. cancers and laryngeal cancers in heavily exposed persons, animal experiments (by feeding and inhalation) have failed to verify this association. Animal studies to date have failed to demonstrate satisfactorily the ability of asbestos fibers to migrate or be transported through the gastro-intestinal mucosa and feeding experiments have not produced tumors in the animals given asbestos in their diets.

Non-occupational exposure to asbestos, or indirect exposure of workers in the proximity of asbestos workers, was first described by Wagner in 1960 and later positively identified by Newhouse and Thompson, Harries and recently by Anderson, et al. Pleural plaques have been attributed to anthophyllite exposure in Finland affecting persons living near asbestos mines, but not associated with asbestosis, excess cancer deaths or mesothelioma in the non-occupational cases. Pleural plaques have also been described in relatives of amosite asbestos workers in the United States. Both Wagner and Newhouse have described neighborhood cases of mesothelioma and domestic contact cases, largely due to exposure to crocidolite. Although it has been alleged that these cases could possibly have resulted from minimal exposure, there is reason to believe that their exposures were quite heavy and even prolonged, although in terms of occupational exposures in the same era (20 - 40 years ago), they could have been regarded as "minimal". Not all studies of populations living in the vicinity of asbestos plants have positively identified a hazard and in the U. S. A., Hammond was unable

SLIDES 31+32

to demonstrate an excess of cancer deaths in the community which lived near a dusty plant in New Jersey when he compared it with a similar community with no asbestos plant in its midst.

Asbestos is ubiquitous in the earth's environment and can occur naturally in the air we breathe and the water we drink. There is evidence that the amount of asbestos in air in the vicinity of certain asbestos plants or near the toll plazas on busy highways, can be detected at slightly higher levels than in sparsely populated rural areas. When all is said and done, however, these differences can only be expressed in orders of magnitude and measured by using sophisticated electronic microscopes and equipment followed by complicated statistical analytical techniques to evaluate the amount present in terms of 10^{-x} grams per m^3 or y nanagrams per m^3 of air.

Evidence that asbestos was present in ambient air could be obtained by examining human lungs and when this was done in some of the world's major cities, asbestos bodies and asbestos fibers were found in from 50% to 90% of random autopsies, in persons who had died from causes unrelated to asbestos disease. According to Selikoff, the frequency of occurrence of asbestos bodies in New York does not seem to have altered between 1934 and 1967, 53% being found in 1934 and 60% in 1967. Oldham has reported a steady increase in the proportion of asbestos bodies in lung tissue from 100 consecutive autopsies in London at ten year intervals from 1936-1966. Doniach et al. found an association between the distribution of cases with asbestos bodies seen in the East End of London and occupational or neighborhood exposure.

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The Unresolved Items

Long-term exposure to all varieties of asbestos, especially at high concentrations, is known to be a health hazard and results in disability from asbestosis or death from asbestosis, lung cancer, mesothelioma or other forms of cancer. The BOHS recommended standard for chrysotile was based upon a study of a population of asbestos textile workers in the North of England and has subsequently been acknowledged to be founded upon "scanty data", although it was the best available at the time. Other studies which have attempted to correlate dust concentrations over a period of time with disease incidence have also relied upon scanty data or have used different dust-measuring techniques which do not allow for conversion to fiber counts as used in industry today. Whatever the method used, a dose-response relationship has been found to exist for asbestosis, although the lower limit of this response has not been clearly determined. In the determination of the present standard most of the exposure data examined related to persons with more than ten years exposure, but in the upper levels of dustiness. Figure 1. illustrates the TR 9 difficulty encountered in extrapolating from the curve for the most exposed to the bottom end of the curve where virtually no data exists. The line of best fit probably does not pass through 0 and uncertainty as to where it lies will continue to exist until data becomes available about the effects of low level exposure. FIG 2 TR 10

Although an exposure level probably exists below which there is virtually no risk of developing asbestosis, such a level has as yet not been determined. In assessing the effects of long exposure to relatively low levels of asbestos there is a wide range of possiblities for the health effect ~~as shown by Peto~~ (~~Table 1~~.). This does not mean that the present standard should be abandoned. There is no evidence to suggest that it is inadequate to prevent asbestosis.

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whereas, at least one authoritative study has determined that it will protect 99% of persons exposed at this level. Because our knowledge is incomplete, it is argued that it is inappropriate to set this hygiene standard for asbestos, but this is a negative attitude and should not be allowed to set aside a standard which at the present time provides the best guidance available. Having recognized its limitations, we should aim to produce better data for future assessment - and precisely this is now being done.

Mesothelioma, even in heavily exposed groups of persons, is a rare tumor in comparison with lung cancer in the general population. There is of course a low natural incidence of mesothelioma in the general population probably unrelated to asbestos exposure. As mentioned previously, recent epidemiologic studies in Turkey have found that pleural lesions and tumors of the kind seen in asbestos exposed people occur endemically in a certain region without any asbestos mines, factories or users. It has also been shown that pleural tumors can be produced experimentally in laboratory animals using durable mineral fibers, such as glass fiber and aluminum oxide, which have the same dimensions as asbestos fibers known to cause mesothelioma in man. The Turkish cases appear to be associated with a mineral known as eironite zeolite, which has a fibrous form. Two items remain unresolved with regard to mesothelioma and they are: 1. Can mesothelioma develop in humans exposed to fiber substitutes for asbestos? 2. Is there a level of exposure below which mesothelioma will not develop?

The answers to both of these questions are to some extent inter-related and in both cases unconfirmed as yet. At the present time, epidemiologic studies have not demonstrated an excess incidence of lung cancer or

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mesothelioma. Pulmonary fibrosis does not appear to affect these working groups and the only positive finding in any study thus far has been the finding of an excess of respiratory symptoms suggestive of bronchitis in retirees with long service. This could be non-specific bronchitis found in industrial populations and not directly related to any one type of dust exposure. The aerodynamic behavior of non-asbestos fibers (man-made mineral fibers included) needs to be better characterised in order to determine the ability of airborne particles to penetrate the lungs. Study of post-mortem material derived from exposed populations is needed to determine any adverse pathological effects and long-term longitudinal studies of exposed populations need to be done. It is reassuring, however, that thus far no problem appears to have been detected.

The question regarding the exposure level, if any, below which mesothelioma will not occur, is to some extent bound up with the question of substitutes. It has been postulated that the ability of asbestos fibers to penetrate the lung depends upon their aerodynamic diameter. Fibers which have a diameter greater than 3 um will not be able to pass through the narrowest airways into the alveoli (air sacs) and will be trapped at this level or, if larger in diameter than approximately 7 - 10 um, will be deposited in the large airways and eliminated on the muco-ciliary escalators. The length of the fibers is not important from the point of view of interception by branching small airways except for the fact that chrysotile fibers may be long, flexible and curly and therefore be trapped more readily than shorter, more rigid amphibole fibers. Fibers as long as 200 um have been found in the depth of the lung, but their diameters were below 3 um in

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SLIDE 34
SLIDE 35

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order for them to penetrate. Once in the lung, alveolar macrophages deal with fibers less than 8 um in length, but above this length, the ability of the alveolar defenses to cope diminishes rapidly. The long, thin fibers have been shown experimentally to be the most carcinogenic. The long, thin fibers may also be the ones which are not visible by means of optical light microscopy and can probably only be identified using the electron-microscope. The question, therefore, is how can the dose (exposure level) be determined, unless highly sophisticated measuring techniques are introduced? One means of attempting to do this is from the use of circumstantial evidence derived from mortality studies which suggest that the risk of mesothelioma is dose-related and usually occurs as a result of relatively heavy exposure to asbestos dust. It is not related to cigarette smoking. Figure 3a. and Figure 3b. TR. 11 illustrate the difficulty in determining whether or not a no-effect level of exposure exists for mesothelioma. In Figure 2a., because some cases of mesothelioma probably occur naturally, the line is likely to intercept the horizontal axis at some point above 0. It is uncertain from present evidence whether the line is a straight one as in Figure 3a. or whether it flattens off first, as in 3b.

Although the experimental evidence seems to incriminate crocidolite as being more dangerous than chrysotile because of its suggested greater penetrability of the lung due to its needle-like shape, there is also evidence to suggest that the amphibiles are dustier to handle than chrysotile and that the proportion of fibers in dust clouds of equal mass for the two varieties of asbestos are vastly different, the amphiboles containing more biologically active fibers than chrysotile. If this is the case, adequate dust control should equalize the risks for all varieties of asbestos.

Unfortunately, because crocidolite appears to be more hazardous, whatever the reason, its use has been severely restricted (or even banned) in many countries.

The role of asbestos in the production of lung cancer is compounded by the multiplicative effect of cigarette smoking on the incidence of this disease in asbestos workers. Lung cancer in the general population has become a public health problem of great magnitude and is largely attributed to smoking. There is some disagreement as to whether lung cancer in a non-smoking asbestos worker should be attributed to asbestos exposure, and some authorities have expressed the opinion that non-smoking asbestos workers do not have a greater chance of developing lung cancer than the general population. There is some evidence that lung cancer excess deaths may be slightly increased in non-smoking asbestos workers in populations such as insulation workers. It may be more liable to occur in such heavily exposed persons or in association with asbestosis which is another indicator of substantial exposure. The point of view has been expressed that in view of the slight association with the more heavily exposed groups, atmospheric pollution or general industrial pollution may be a cofactor. Lung cancer probably has a multi-factoral causability and it is not always possible, in an industrial population, to pin-point the cause, unless of course the statistical correlations between a substance and the observed effect are highly significant. This is the case with cigarette-smoking asbestos workers.

One of the major difficulties confronting the establishment of an asbestos standard is the scientific theory which postulates that there is no known

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UCC 010306

safe level for a carcinogen. The human body is able to deal with small doses, or low levels of exposure to cancer-causing materials, with no adverse long-term effect, according to some authorities.

"The prevailing scientific view is that human cells are equipped with mechanisms that prevent unrestrained reproduction. Exposure to carcinogens somehow causes some cells to become asocial and reproduce abnormally, eventually invading other parts of the body. A few scientists are of the opinion that one molecule of a carcinogen, acting on a single cell, could eventually result in cancer. Since it has not been proved positively that this does not happen, they argue that all human contact with carcinogens should be prevented wherever possible.

On the other hand, however, daily experience with naturally occuring carcinogens suggests that there is probably a threshold below which there is no harmful biological effect, at least for most of us. For example, the ultraviolet rays in sunlight are known to be potent carcinogens, but most people do not contract skin cancer from being out in the sun. Moreover, our bodies contain trace amounts of a number of elements which are known carcinogens. Recent experiments have shown that certain hormones, such as the female sex hormone estrogen, are also carcinogenic."

(Monsanto. Chemical Facts Bulletin)

Nothing in life is risk-free and society, weighing the known scientific facts in the balance ultimately will decide what risks to take. It is important that society is made aware of improvements in industry and its products to enable it to compare the risks taken thirty years ago with those of today.

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No unnecessary exposure is justifiable and should be avoided as far as is reasonably practicable. Before condemning useful products or useful materials on the basis of incomplete evidence, the consequences of their disappearance from daily use need to be considered.

A major unresolved issue is whether asbestos poses a threat to the public health. A number of events have recently brought this matter to the forefront of the public scene. It has been shown that the mesothelioma rate in Connecticut has increased rapidly in the past ten years and this has been attributed to the increased usage in parallel periods allowing for the time lag for development of the disease. The discovery of asbestos in waste dumps, asbestos in dry wall spackling compounds, asbestos in ember ashes and recently, asbestos in hair dryers, has all been publicized and proclaimed as a public risk. It is at present impossible to determine whether, in the case of consumer products, any hazard whatever exists, but because of the "no safe level for carcinogen theory" the general public must be protected from any unnecessary exposure.

Waste dumps, asbestos emissions from asbestos manufacturing plants, brake lining and clutch-facing wear products and asbestos in public buildings and schools are all examples of sources of low-level dust exposure of varying degree. It is virtually impossible, by means of the best available technology, to reach inter-observer agreement on ambient air levels of asbestos in differing circumstances and even duplicative sampling by two laboratories produces widely differing results. The one exception is

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the measurement of asbestos in schools with sprayed walls or ceilings. No definite epidemiological studies have been done, and are unlikely to be done, which correlate the minute levels of asbestos in the general environment with the incidence of asbestos-related disease and those which have been attempted are either incomplete in detail or inconclusive in their findings. Even the Connecticut data can be readily faulted because the diagnostic confirmation of mesotheliomas in the Tumor Registry is weak and because occupational history details are unknown for the cases, thus making it impossible at this stage to determine whether they were primarily of occupational or environmental origin.

Summary and Conclusion

A number of known facts have been discussed. Asbestosis, lung cancer and malignant mesothelioma of pleura or peritoneum are all associated with exposure to asbestos. G. I. cancers and cancer of the larynx occur more frequently in heavily exposed asbestos workers than in the general population. There is evidence to suggest that improvements in dust control can eventually eliminate asbestosis almost completely and strongly influence a reduction in the incidence of lung cancer as well. The single most important factor in the reduction of lung cancer is abolition of the cigarette-smoking habit.

Mesothelioma can occur in neighborhood or domestic contact cases which probably had relatively high exposures in comparison with the general population as a whole. Crocidolite appears to play a major role in this disease process.

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UCC 010309

Many issues remain unresolved including the level at which to set a hygiene standard below which everybody will be safe. The general public, exposed to trace amounts of asbestos in the general environment, does not appear to be at risk, but the added effect of unnecessary exposure from asbestos-containing products in daily use has prompted vigorous demands for the abolition of those products and the banning of asbestos in some instances. This is not a scientific issue as no data exists upon which to form a reasoned opinion and will be decided by the mechanisms which society has devised in the form of government bodies and regulatory agencies. Asbestos will continue to be present in the air we breathe and water we drink as it has been since time immemorial. Nobody breathes pure air.

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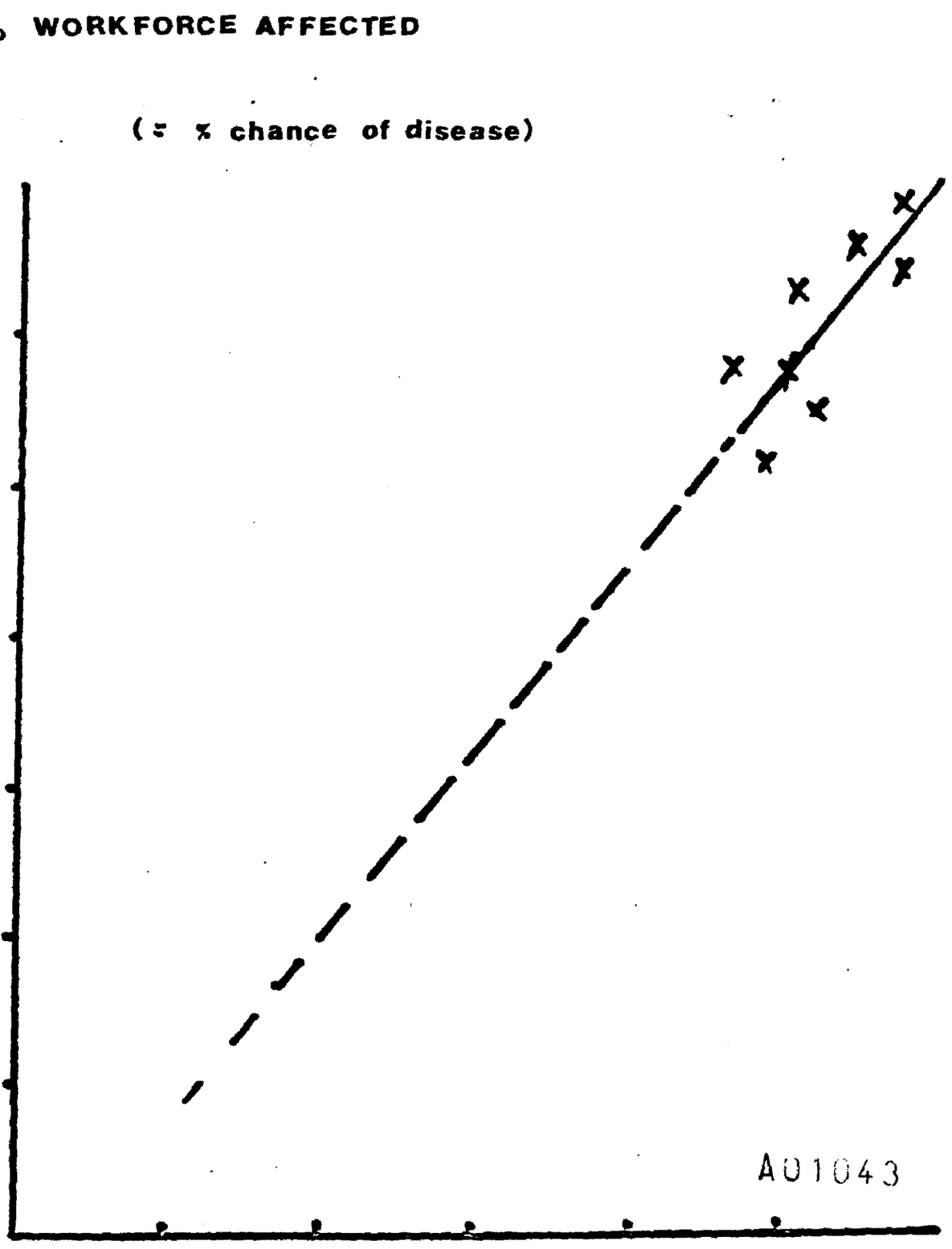
UCC 010310

FIG. 1.



HCL 027403

FIG. 2A

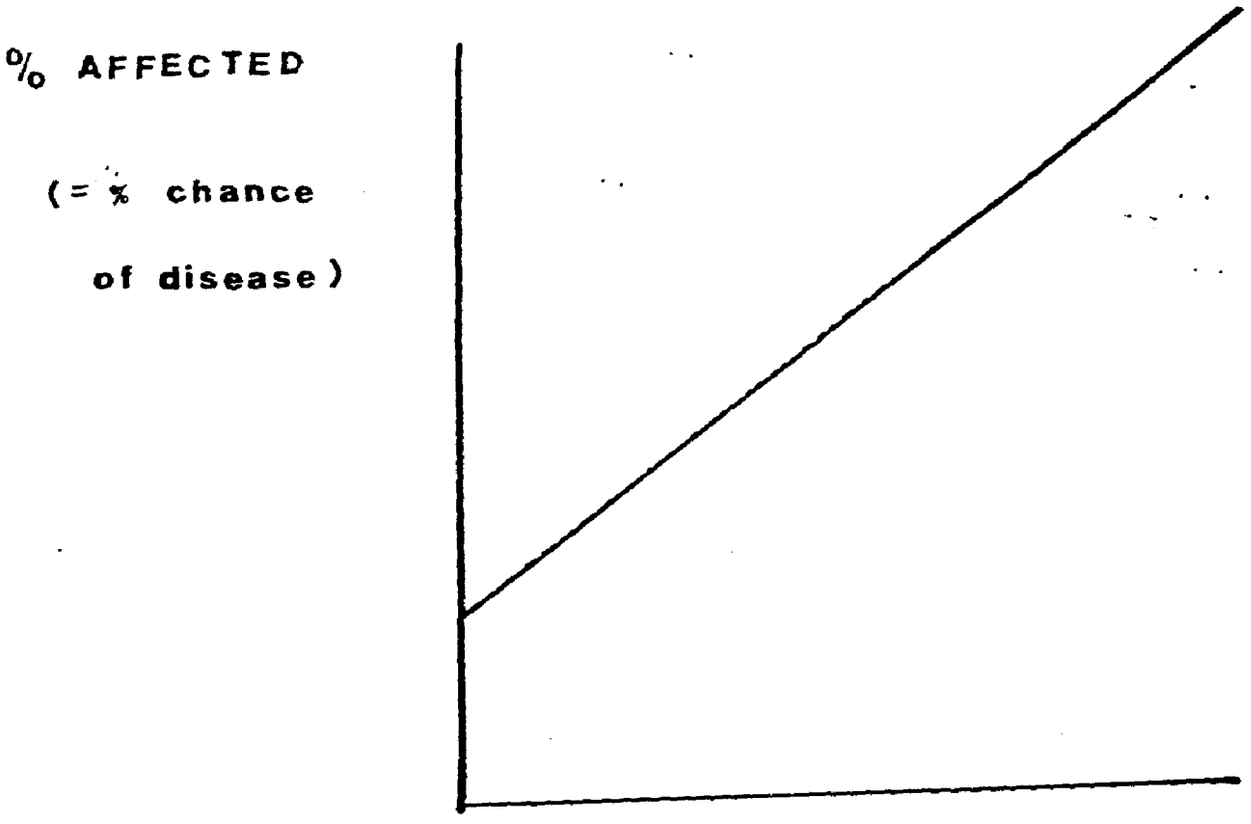
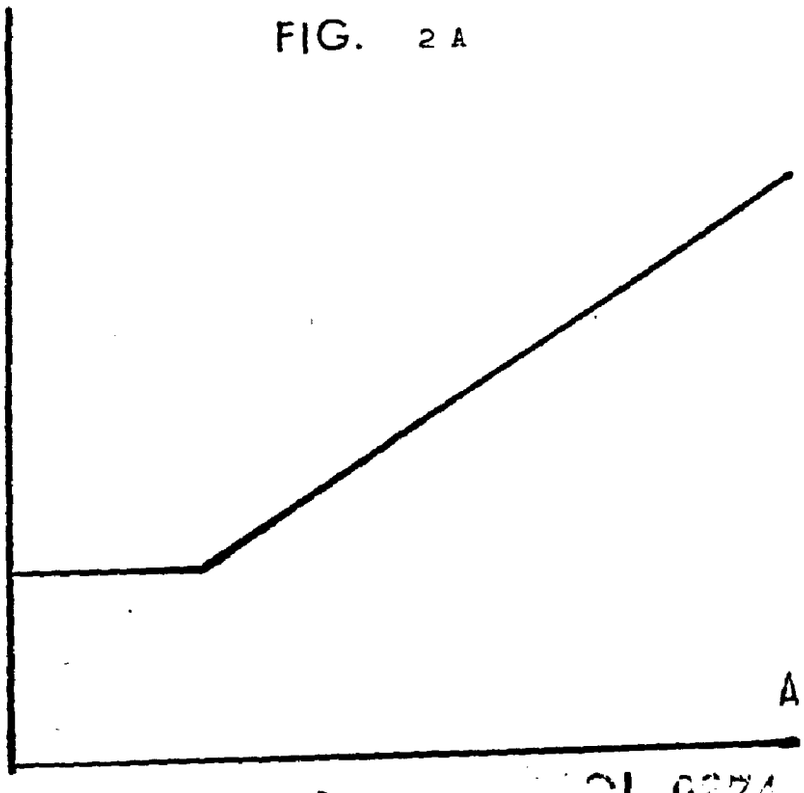

% AFFECTED
(= % chance
of disease)

FIG. 2A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CL 0274.0
CUMULATIVE EXPOSURE – f yrs/cc

Characteristics of main types of asbestos fibre

Characteristic	Chrysotile	Crocidolite	Amosite	Anthophyllite	Tremolite	Actinolite
Theoretical Formula	$Mg_3[Si_2O_5](OH)_4$	$Na_2Fe^{II}_3Fe^{III}_2[Si_8O_{22}](OH)_2$	$(Fe,Mg)_7[Si_8O_{22}](OH)_2$	$(Mg,Fe)_7[Si_8O_{22}](OH)_2$	$Ca_2Mg_5[Si_8O_{22}](OH)_2$	$Ca_2(Mg,Fe)_5[Si_8O_{22}](OH)_2$
Colour	Usually white to pale green, yellow[1], pink[1]	Blue	Light grey to pale brown	White to grey, pale brown	White to grey	Pale to dark green
Decomposition temperature* (°C)	450–700	400–600	600–800	600–850	950–1040	620–960
Fusion temperature of residual material (°C)	1500	1200	1400	1450	1315	1400
Density g/cm³	2·55	3·3–3·4	3·4–3·5	2·85–3·1	2·9–3·1	3·0–3·2
Resistance to acids	Undergoes fairly rapid attack	Good	Attacked slowly	Very good	Very good	Attacked slowly
Resistance to alkalis	Very good	Good	Good	Very good	Good	Good
Mechanical properties of fibre as taken from rock samples:						
Tensile strength 10^3 kg/cm²	31	35	17	(<7)	5	5
(Average) (10^3 psi)	(440)	(495)	(250)	(<100)	(<70)	(<70)
Young's Modulus 10^3 kg/cm²	1,620	1,860	1,620	—	—	—
(Average) (10^6 psi)	(23)	(27)	(23)			
Texture	Usually flexible, silky and tough	Flexible to brittle and tough	Usually brittle	Usually brittle	Usually brittle	
Producing countries	USSR Canada China Rhodesia USA Italy South Africa Swaziland	South Africa	South Africa	Finland USA Mozambique	USA Italy	

Notes *Dehydroxylation or dehydrogenation accompanied by disruption of crystal lattice and major loss of strength. [1] From serpentinised dolomite deposits.

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