FILE NAME: Union Carbide (UC)

DATE: 1983 Feb 17

DOC#: UC163

DOCUMENT DESCRIPTION: Memo RE Asbestos Labeling with Attached Sample Label & Report - Asbestos & Health - Perspectives

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Date February 17, 1983
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Copy to Mr. R. A. Butler
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Dr. W. C. Kuryla
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Mr. W. C. Thurber
Mr. R. F. Wolff

Subject Asbestos Labeling

I have studied your memorandum dated February 15, 1983, addressed to Mr. W. C. Thurber and the proposed label attached hereto. Although asbestos is a carcinogen, there does appear to be a dose-response relationship between the amount inhaled over time and the various forms of asbestos related diseases. There is also a substantial body of expert opinion which believes that fibers of a certain size and shape are the most hazardous and that this theory is manifested epidemiologically by differences in biological effects produced by exposure to different forms of asbestiform minerals. I am concerned that the wording of your proposed label may turn out to be inappropriate for the type of asbestos in the container but unfortunately have no epidemiologic evidence to contradict the statement. I therefore suggest the amended version attached to this note for your consideration.

I realize that the wording may not be exactly what is required but I feel that the proposed label is too brief and does not make all the points necessary to insure safe handling by the employee. I would be happy to discuss this further with you.

The attached notes may be of interest and help place the subject in perspective. I apologize for their length!

H. C. Lewinsohn, MB.BCh.,FCCP
Assistant Corporate Medical Director

HCL/bd

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CHRYSOTILE ASBESTOS

WARNING

- CANCER HAZARD -

BREATHING ASBESTOS DUST CAN CAUSE LUNG DAMAGE AND CANCER . THE RISK OF LUNG CANCER IS GREATLY INCREASED IN SMOKERS.

* HANDLE WITH CARE AND AVOID CREATING DUST.

* WEAR RESPIRATORY PROTECTION DEVICES IF WORK AREA IS NOT PROVIDED WITH ADEQUATE EXHAUST VENTILATION OR WHEN WORKING IN CONFINED UNVENTILATED SPACES. ONLY NIOSH / MSHA APPROVED RESPIRATORS SHOULD BE USED.

FOR INDUSTRIAL USE ONLY.

* DO NOT REUSE THIS PACKAGE. DISPOSE OF IT IN IMPERMEABLE SEALED CONTAINER.

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ASBESTOS AND HEALTH - PERSPECTIVES

A. WHAT IS ASBESTOS?

Asbestos is a generic term used to describe naturally occuring durable mineral silicates of filamentary or fibrous nature.[1] The varieties of asbestos in commerical use, their chemical descriptions, their physical appearance and their main source of origin are shown in Table 1.

The USSR is probably the world's largest consumer of asbestos. U.S. mines produced 15% of our consumption in 1978. Canada is the leading importer of asbestos, with the Republic of South Africa second. South africa supplies all of the U.S. demand for crocidolite and amosite.[2]

It should be appreciated that in the primary and immediate post-primary section of the asbestos industry, i.e., mines and manufacturing industry, relatively few people are involved, whereas millions of people are ultimately exposed to apparently ever decreasing amounts of fiber in the products of the entire industry. Furthermore, asbestos is ubiquitous in the earth's atmosphere and has been since the beginning of recorded time.

Fibers below 0.5 um diameter and less than 5 um in length are not visible with the optical microscope and need to be identified by transmission electron microscopy, scanning electron microscopy or combinations of these instruments and various forms of selected area diffraction or microprobe analysis. X-ray diffraction has been used for the quantitative determination of chrysotile and amphiboles in air and water samples. The latter technique has also been used to analyze asbestos in lung tissue.

Knowledge of the type of asbestos to which a person has been exposed is of value because it may be an important factor in the appreciation of special risks involved.

Differences in the physical characteristics of the various types of asbestos fibers determine their particular commercial usefulness. Chrysotile consists of long, mainly pliable fibers that split progressively into finer fibrils and it may be used in textiles, whereas crocidolite and amosite can be used in marine insulation because of their acid resistant properties. Certain asbestos cement products may be made from blends of chrysotile and amosite and/or crocidolite.

B. WHAT ARE THE PATHOLOGICAL EFFECTS OF EXPOSURE IN MAN?

Exposure to asbestos at work or elsewhere may result in five conditions:

1. The presence of asbestos in tissues without disease - e.g., asbestos bodies in the general population.[3]

2. The presence of asbestos in the tissues causing benign changes - e.g., skin warts, pleural plaques.[4]

3. The presence of asbestos in the tissues and the development of malignant mesothelioma of the pleura or peritoneum.

4. Asbestos in the lungs with tissue damage and the development of lung cancer.

5. Asbestos present with potentially fatal damage to the lungs (pulmonary fibrosis or asbestosis), but no cancer.

There are two other conditions which have been detected in greater numbers among asbestos workers than would be expected from a similar sample of the general population:

a. Cancer of the gastro-intestinal system involving oesophagus, stomach and colon and rectum.

b. Cancer of the larynx.

C. HOW DO PHYSICAL FACTORS RELATE TO CAUSATION OF DISEASE?

Whether or not inhaled asbestos fibers will reach the depths of the lung depends on the aerodynamic behavior of the particles, the size of the airways they enter, and the individual's breathing pattern.[5] The larger dust particles are trapped in the nose and throat. Smaller fibers get down into the trachea, bronchi and smaller bronchioles, but because of the turbulent airflow in the large airways, fibers are thrown outward and deposit on the sticky lining mucosa. These fibers are carried back out of the lungs on the muco-ciliary escalator by the beating of the cilia. When they reach the larynx, they are swallowed. Thus three factors are involved:

1. deposition (or internal forces in large airways),

2. gravitational (settling of fibers in smaller airways), and 402769

3. diffusion forces in alveolar spaces.

Other physical factors to consider are itemized:

1. Respirability is dependent upon the size and shape of fibers inhaled.

2. Larger particles are trapped in the upper airways and removed from the lungs by the muco-ciliary escalator.

3. Fibers with an aerodynamic diameter less than 3 um are respirable, although their length may be as much as 100 um - 200 um.

4. Fibers with an aerodynamic diameter less than 3 um and greater than 10 to 20 um in length are thought to be those most likely to cause disease.

The shape of inhaled particles, aside from size per se, determines in part their deposition characteristics. Long, irregular particles such as asbestos fibers settle much less than would be expected from their total fiber mass. Other irregularly shaped particles (e.g., quartz, coal) are

aerodynamically equivalent to spherical particles one-half to three-quarters of their measured diameters. Most models consider particles in terms of unit density spherical shapes (i.e., as aerosols), to reach a reasonable agreement between theoretical predictions and experimental observations. In addition to space, the density of a particle determines its deposition characteristics.

Differences in disease-producing potentials of fibers may arise from the fact that curly, flexible, soft chrysotile fibers are more likely to be caught and filtered out by this system than the straight fibers of the other forms of asbestos in commercial use. To escape this filter mechanism, the fibers must be light enough to remain in suspension and short enough not to be intercepted by the branching of the smaller airways. Examination of human lungs has revealed straight fibers in the lung up to 200 um in length and also coils of chrysotile which may be even longer if stretched out. Once a fiber is carried beyond the ciliated part of the airway, it may still be deposited and stay there or it may be carried out again with the next expiration of air. The proportion of fibers trapped at this stage still depends on size - long fibers are caught, small ones breathed out. Because in a typical dust cloud there are millions of very small fibers, more are retained in the lung than larger ones. Many of these very small fibers are too small to be counted with a light microscope and can only be counted by examining the lung or digests of the lung under the electron microscope. Their biological effects, if any, are not yet known.

In the tissues of the lung, and elsewhere where the fibers may lodge because of transportation in the body by blood, lymphatics and tissue fluid, the fibers may be coated with a brown iron-pigmented material called ferritin, to form 'asbestos bodies'. Asbestos bodies are thought to be innocuous. Not all asbestos fibers are coated in this way and in humans it has been estimated that for every asbestos body in the lung there are 1,000 uncoated asbestos fibers. It is not known whether this is the case in all types of asbestos or in other tissue. It is known that asbestos bodies form rapidly, reside in tissue many years and gradually degenerate over the years releasing their fiber core. It is not known whether these released fibers, after many years, are still capable of causing disease. It is also known that uncoated fibers are capable of causing tissue damage when first inhaled, but it is not known whether they retain this potential indefinitely or are dealt with by some unknown defense mechanism other than the ferritin coating process. Chrysotile fibers have been shown to dissolve in tissue fluids so that it may be impossible to confirm that a person has been exposed by looking for these fibers in the tissues thirty or forty years later, unless exposure was continuous throughout the individual's lifetime up to the time of retirement. Crocidolite and amosite can be identified in tissue even after as long an interval as this, and it has been claimed by one investigator that it is actually possible to identify the geological origin of such fibers by the use of electron microscopic techniques.

The physical factors outlined above may be invoked to explain why asbestos miners seem to be less at risk than primary process workers who in turn seem less at hazard than those who use processed asbestos under dusty

conditions. It is possible that freshly mined asbestos is still aggregated in bundles and less likely to be respirable or retained in the lung and thus less likely to be damaging. The more processing the asbestos receives, the finer the division of the fiber bundles and the more dangerous it becomes. Dust studies to support this physical characteristic have been reported. Chrysotile fibers collected in the carding area of an asbestos textile plant tended to have smaller diameters than fibers collected in the dryer and bagging areas of an asbestos mill.

It should not be forgotten that primary and secondary use of asbestos usually takes place in highly polluted urban environments by people exposed to many additional non-respiratory toxic agents. Cigarette smoking may be a co-factor in the production of occupational disease - it is not usually permitted underground in mines. There may be a synergy between cigarette smoke and asbestos dust only when they are inhaled simultaneously, but this is unlikely and difficult to deduce from epidemiologic studies.

D. WHAT IS THE EVIDENCE FOR STATING THAT ASBESTOS MAY BE PRESENT IN TISSUE WITHOUT DISEASE?

Examination of material from random autopsy series in several cities has revealed the presence of asbestos in lung tissue.[6] The frequency of this finding depends upon the diligence of the search. When digested lung tissue is examined, prevalence approaches 100%. These findings can occur in the absence of any asbestos associated diseases.

E. DOES A DOSE-RESPONSE RELATIONSHIP EXIST IN ASBESTOS-RELATED DISEASES?

The concept of a dose relationship of response to stimulus is a familiar one in pharmacology. This same concept has been invoked in an effort to explain the biologic response to inhaled dust.[5]

An important question immediately arises - Why is one person affected and not the person working alongside? A third factor that has to be introduced into the concept is that a given dose-response curve can be developed for a given population (or person), but that it will be applicable only to another population (or person) of the same "susceptibility." Susceptibility may depend upon several biological factors such as the efficiency of pulmonary clearance mechanisms, the anatomic characteristics of the lung/airway system, or the physical fitness of the person. Susceptibility can also be related to immunogenetic factors. Another important variable, not biological, is the differences in work practices and habits of individuals doing essentially the same job.

Although asbestos dose-response relationships are evident to a greater or lesser extent for all responses, the degree of correlation is difficult to ascertain precisely because of inadequate records of past exposure in all situations studied. The observed response is usually the result of past, rather than current exposure. This poor correlation has led to the current interest in "susceptibility," i.e., factors accounting for between-subject differences in response.

F. IS THERE A SAFE STANDARD TO PROTECT AGAINST THESE DISEASES?

There is a scarcity of adequate data from which to derive a safety standard which would give a 100% assurance of preventing the diseases associated with asbestos.

The present standard is based upon evidence presented in a 1968 report published by the British Occupational Hygiene Society.[7] The data upon which this report was founded was obtained from an asbestos textile factory which had personnel and medical records available for study, as well as dust measurements, from 1951 onwards. The standard assumed that a combination of two variables, namely length of exposure and concentration of fibers during the exposure period, could be statistically analyzed and correlated with the earliest signs of the effects of asbestos exposure recognizable by the plant physician. As a result, it was postulated on this evidence that a cumulative exposure of 100 fibers per cubic centimeter would result in only 1% of persons exposed developing these early signs of asbestosis. The committee speculated that a worker could work for 50 years in dust conentrations of 2 fibers/cc and only run a 1% risk of developing asbestosis. The committee did not propose the standard for protection against lung cancer or mesothelioma.

The crucial issue at stake is whether exposure to dust levels of 2 fibers/cc will also prevent lung cancer and mesothelioma. Furthermore, should the same standard apply to all types of asbestos fibers or should there be an even tighter control on the use of crocidolite?

There is circumstantial evidence from the same factory that the high excess incidence of lung cancer deaths in the heavily exposed groups of workers who were employed before the regulations were introduced in 1931 and became effective in 1933 has been much reduced in the more recently exposed groups, i.e., the post 1933 cohort, although a slight excess may still be detected even in the cohort first exposed after 1950.[8] This slight excess is not highly statistically significant and might be drastically influenced in the future by increasing the follow-up population. Furthermore, it can be explained by the fact that conditions in the factory were by no means all in compliance with the standard requirements of today, and not until this is achieved will this small excess number of deaths abate. There is no numerical data with regard to mesothelioma upon which to build a dose-response curve, although some authorities do believe that a dose-response has been demonstrated based upon historical descriptions of conditions allowing jobs to be classified as severe, moderate, light and negligible exposures.[5]

The present situation is that in the United States no data is available for study, which enables asbestos fiber counts to be correlated with morbidity or mortality. The best data still comes from the factory in the U.K. mentioned previously and it is currently under review by the BOHS.

Cancer is an emotional word. NIOSH and OSHA, both under criticism and charged with being inefficient, respond to pressure groups readily and over-react regularly. Nobody seems to know what to do.

Time will tell. Even if the uses of asbestos decline, the deposits in the earth's crust will be there to exploit again when the dust of past misuse settles and the dose-response can be more clearly determined based upon adequate dust measurement records now being compiled and better recordkeeping of morbidity and mortality statistics.

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TABLE 1

CHARACTERISTICS OF MAIN TYPES OF ASBESTOS FIBER

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/cm^3	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 fiber as taken from rock samples:						
Tensile strength 10^3 kg/cm^2	31	35	17	(<7)	5	5
(Average)(10^3 psi)	(440)	(495)	(250)	(<100)	(<70)	(<70)
Young's Modulus 10^3 kg/cm^2	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	

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

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