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DATE: 1969 Oct 2

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DOCUMENT DESCRIPTION: Memo - Personal and Confidential - to J.P. Morgan, Haskel Laboratory from Charles D. Crook, RE Method of Reporting Asbestos Dust Levels with Attachments 1-5

E. I. DU PONT DE NEMOURS & COMPANY
INCORPORATED
OLD HICKORY, TENNESSEE 37138

TEXTILE FIBERS DEPARTMENT

CC: J. E. Crawley
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Old Hickory, Tennessee
October 2, 1969

RECEIVED
OCT 31 1969
HASKELL LABORATORY

PERSONAL AND CONFIDENTIAL

J. P. Morgan
Haskel Laboratory
WILMINGTON

EXHIBIT # P-Morgan-5
DATE 10-23-86
MASTROIANNI & FORMAROLI, INC.
CERTIFIED SHORTHAND REPORTERS

METHOD OF REPORTING ASBESTOS DUST LEVELS

Ref.: (1) Telecon: C. D. Crook and J. P. Morgan, same subject, 9/11/69

(2) Letter: D. D. Drummond to P. J. Tracey, "Asbestos Dust Analysis," 8/21/69

(3) Private communication with Jeremiah R. Lynch, Bureau of Occupational Safety and Health, U. S. Public Health Service, 1014 Broadway, Cincinnati, Ohio

(4) Threshold Limit Values for 1968, American Conference of Industrial Hygients, St. Louis, Missouri

(5) R. J. Blair to O. W. Hess, "Asbestos Exposure Hazards," 8/16/68

Per reference (1), I have formalized what I believe is true about reporting asbestos dust level and request your opinion by return letter. Our eventual agreement will determine how asbestos dust level will be reported within the Textile Fibers Department.

Please comment concerning the following two items:

1. The best method to report asbestos dust level is fibers per milliliter longer than 5 microns in length.

2. Construction Division's adoption (Ref. 2) of an asbestos dust level of 0.5 million fibers per cubic foot less than 5 microns in length. This may be questionable for two reasons:

   - It ignores the long fibers which are known to enter the lung and cause asbestosis.

   - It applies a numerical tolerance value intended for overall dustiness (fibers plus grains) as measured by the impinger method to fiber dustiness (grains not counted) as measured by the membrane filter method. As a result their tolerance value is above that recommended by the U. S. Public Health Service (Ref. 3) and the American Conference of Governmental Industrial Hygients (Ref. 4).

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DISCUSSION

It has been established that long fiber do enter the lung and cause asbestosis. Therefore, the concentration of long airborne fibers must be controlled (see Attachments I, II, and III).

It is not known if short fibers cause asbestosis. Some studies show short fibers to be inert (Attachment IV); others indicate they could be as harmful as long fibers (Attachment V). The U. S. Public Health Service does not consider short fibers inert but maintains that if the long fiber level is low, then the short fiber level will be low also (Ref. 3).

Reference 5 specifies that 0.5 million particles per cubic foot to be used as a tentative tolerance. This was obtained by taking one-tenth of official governmental standard. But the governmental standard is for overall dustiness (fiber plus grains). Obviously, applying the same numerical value to fibers only gives a high tolerance value: 0.5 million fibers per cubic foot is equivalent to 18 fibers per milliliter; the governmental standard for fibers is 12 per milliliter. To avoid confusion in the future, I recommend that we always talk in terms of fibers per milliliter.

DACRON® MANUFACTURING DIVISION

J. K. SNEED
PROCESS SUPERINTENDENT

BY: Charles D. Crook
CHARLES D. CROOK

CDC:dd

Attachments

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ATTACHMENT I

INDUSTRIAL HYGIENE AND TOXICOLOGY
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INTERSCIENCE PUB.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures described first by McDonald.[87] These bodies are 20 to 100 μ in length and are bulbous on one or both ends so that they appear club- or dumbbell-shaped. They are brownish in color, do not stain, and give a Prussian-blue reaction for iron. Simson[88] has produced these bodies in guinea pigs by experimental exposure to atmosphere containing asbestos. Lynch[89] concludes that these "curious bodies" signify exposure to asbestos dust but do not necessarily indicate asbestosis. In other words, they are more properly referred to as "asbestos bodies" and not "asbestosis bodies."

X-ray examination. For an excellent review of roentgenographic findings in both silicosis and asbestosis the reader might well refer to Pendergrass.[90] Characteristic differences in the roentgenographic findings in silicosis and asbestosis are tabulated below:

Asbestosis	Silicosis
Fibrosis diffuse (film may have ground glass appearance from pleural involvement).	Fibrosis nodular.
Findings may be either bilateral or unilateral.	Findings characteristically bilateral.
Lesions largely in the lower one half or two thirds of the lung fields.	Lesions predominantly in the upper two-thirds of the lung fields.
Emphysema in upper portion of lung fields.	Emphysema in lower portion of lung fields.
Borderline degree difficult to recognize.	Borderline degree more easily recognized.

Control. Prevention of asbestosis depends largely on preventing exposure to sufficient[illegible] dust. Also, if only long fibers cause the disease, fine dust respirators are not necessary. The more comfortable gauze respirators appear to be perfectly adequate filters for the long fibers. Up to this time, however, they have not been approved by the United States Bureau of Mines for this purpose.

Allowable concentrations. Dreessen, DallaValle, Edwards, Miller, and Sayers[91] have found evidence to indicate that 5 million particles per cubic foot of air is a satisfactory figure for the maximum permissible atmospheric concentration of asbestos to which workers may be exposed. However, in setting up this figure, only fibers less than 10 μ in length were counted and the longer ones were disregarded. It now appears that the shorter fibers may have to be disregarded and permissible concentrations of long fibers established, which has not been done.

The prognosis with early to moderately advanced asbestosis is good, provided the inhalation of asbestos fibers is materially decreased. Unlike silicosis,

[87] E. McDonald, *Brit. Med. J.*, 2, 1025 (1927).
[88] F. W. Simson, *Brit. Med. J.*, 1, 885 (1928).
[89] K. M. Lynch, *J. Am. Med. Assoc.*, 109, 1947 (1936).
[90] E. P. Pendergrass, "Roentgen-Ray Diagnosis in Silicosis and Asbestosis," in A. J. Lanza, ed., *Silicosis and Asbestosis*. Oxford Univ. Press, New York, 1938.
[91] W. C. Dreessen, J. M. DallaValle, T. I. Edwards, J. W. Miller, and R. R. Sayers, *U. S. Pub. Health Bull.* No. 241, 1938.

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2. Nau, C.A.: South. Med. J. 41, 341 (1948).
3. Elkins, H.B.: Chemistry of Industrial Toxicology, Wiley & Sons, N.Y., (1959).
4. Bulmer, F.M.R., et al.: J. Ind. Hyg. & Tox. 22, 111 (1940).

ASBESTOS

5 mppcf

Asbestos is a generic term applying to a number of mineral silicates that are incombustible in air and can be separated into filaments. The most widely used in industry is chrysotile, a magnesium silicate from serpentine. Other types include amosite (an iron magnesium silicate), crocidolite (a sodium iron silicate), tremolite (a calcium magnesium silicate) and anthophyllite (also an iron magnesium silicate).

Miller and Sayers (1) showed that intraperitoneal injection of amosite, chrysotile and crocidolite in guinea pigs produced the reaction of an inert dust. Vorwald et al. (2) confirmed this for short fibers (under 3μ) but observed that long fibers produced a fibrous reaction. Continuing unpublished work by Gardner, these workers demonstrated that long fibers (20 microns and above) produced peribronchiolar fibrosis in lower animals, and developed evidence that this resulted from mechanical rather than chemical action. Asbestos dust containing 0.6 per cent fibers longer than 10 microns, in concentrations of 138 mppcf (or 0.8 million "long" fibers per cu. ft.), was capable of producing experimental asbestosis in guinea pigs. When the concentration was 6.7 per cent fibers over 10 microns, 40 mppcf (equivalent to 2.7 million "long" fibers per cu. ft.), the reaction developed in approximately half the time.

That exposure to asbestos is associated with development of a potentially disabling pneumoconiosis in man has been amply demonstrated by industrial experience, (3,4,5,6,7,8,9,10,11). The present threshold limit was recommended by Dreessen et al. (8), after study of 541 employees in three asbestos textile plants using chrysotile. Only three doubtful cases of pneumoconiosis were found in those exposed to dust concentrations under 5 mppcf, whereas numerous well-marked cases were found above 5 mppcf. Counts were from impinger-collected samples in ethyl alcohol and distilled water. Both fibrous and nonfibrous particles were counted, but the latter greatly predominated. While chemical analyses of collected samples of air-borne dust corresponded to those of settled dust samples, it is believed that dust counts of particulates by conventional methods can be expected to give only an inadequate measure of the risk of asbestosis because of the great relative importance of long fibers.

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2. Vorwald, A.J., Durkan, T.M., Pratt, P.C.: Arch. Ind. Hyg. 3, 1 (1951).
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5. Fulton, W.B., Dooley, A., Matthews, J.L., Houtz, R.L.: Penn. Dept. Labor and Ind. Bull. 42 (1935).
6. Lanza, A.J., McConnell, W.J., Fehnel, J.W.: Pub. Health Rep. 50, 1 (1935).
7. Donnelly, J.: J. Ind. Hyg. & Tox., 18, 222 (1936).
8. Dreessen, W.C., DallaValle, J.W., Edwards, T.I., Miller, J.W., Sayers, R.R.: Pub. Health Bull. No. 241, Washington, D.C., 1938.
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ATTACHMENT III
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SECTION V. HUMAN EXPOSURE TO ASBESTOS: DUST CONTROLS AND STANDARDS

THE INHALATION OF FIBROUS DUSTS

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Particles are deposited in the respiratory system by the action of four different mechanisms:

(1) Particles are removed from the inhaled air by settlement under gravity, the magnitude of deposition being proportional to particle free-falling speed and the time available for sedimentation.

(2) When a change in the direction of air flow occurs, particles owing to their inertia tend to continue on their original paths. Some may be deposited in this way on the nasal hairs and on the walls of airways. The efficiency of inertial deposition depends on the relative magnitudes of the drag and inertial forces. The magnitude of inertial deposition is thus proportional to particle free-falling speed.

(3) Particles suspended in the air are under ceaseless bombardment by gas molecules which constantly displaces them and may cause their deposition on surfaces in the respiratory system. Since particle removal by this mechanism is significant only in the small pulmonary air spaces and for particles one-half micron and smaller in size it needs no further consideration in this paper.

(4) There is a fourth mechanism. For a compact particle to remain suspended in the air its center must be separated from all surfaces by distances greater than the particle radius. Deposition occurs when the distance from a surface reduces to equality with the particle radius. The magnitude of deposition by this interception effect in airways increases with increase in the ratio of particle size to tube diameter. For compact particles 10 microns and smaller in size the effect is of no real significance even in the finest airways which are several hundred microns in diameter.

The compact particles found in significant numbers in alveoli are 10 microns and smaller in size. This is true even when men are shown to have been exposed to aerosols containing larger compact particles in appreciable concentrations. In contrast, asbestos fibers 50 microns and even 200 microns and longer have been found in alveoli. Since asbestos fibers are subjected to the same deposition mechanisms as compact particles, and since sedimentation and inertial precipitation, the two mechanisms mainly responsible for particle deposition, have deposition parameters which are proportional to free-falling speed, an investigation of the relationships between the free-falling speeds of fibers and their dimensions was clearly

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ATTACHMENT IV

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bined with cotton and woven as a textile for fireproof and heat-resistant clothing and other substances. Lanza[82] estimates that there are about 10,000 persons exposed to asbestos in the United States. Most observers feel that the incidence of asbestosis in American asbestos workers is quite low. Asbestosis has, however, been reported more frequently in England and Canada.

Lanza, McConnell, and Fehnel[83] concluded from their study of asbestosis that prolonged exposure to asbestos dust causes pulmonary fibrosis different from that produced in silicosis and demonstrable by roentgenogram. Clinically it resembles silicosis in that it is not disabling in the early stages, but it may be markedly disabling when advanced, and frequently leads to death by right heart failure. Although tuberculosis has been shown to be no more prevalent in persons with asbestosis than in the general population, lung cancer is under suspicion as occurring more frequently with asbestosis, especially in England.

Symptoms. The onset of symptoms of asbestosis, as of silicosis, is slow although symptoms in advanced cases are apt to be somewhat more marked than in silicosis. As in silicosis, dyspnea is the cardinal symptom. Anorexia occurs frequently in advanced stages, and cyanosis and clubbing of the fingers are apt to appear with greater constancy in asbestosis.

Pathology. The fibrosis in asbestosis is diffuse and tends to predominate in the basal portions of the lungs in contrast to the generalized nodular fibrosis of silicosis with a predominance in the upper portions of the lung. Bronchiectasis and bronchiolectasis are frequent, especially in the more fibrous portions. Whereas the proliferation of fibrous tissue is caused by chemical action in silica exposure, it is induced by mechanical action in asbestos exposures. Gardner[84] found that the fibrosis-producing character of asbestos could be almost eliminated by grinding the fibers so that no particles more than 20 μ in length were present. As previously mentioned, asbestos is classified among the inert dusts. Vorwald, Dalton, and Drinker[85] found [illegible] ranging from 20 to 50 μ in length but not by fibers shorter than 20 μ. The significance of this finding on prevention of the disease will be discussed below.

Microscopic anatomy. Johnstone[86] described the microscopic appearance of the lungs somewhat as follows: In the early phases of the disease there is thickening of the alveolar septa which results from fibroblastic proliferation. The alveolar spaces contain numerous phagocytes. With progression of the disease fibrosis becomes more marked; the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few alveoli that remain in the area of fibrosis are lined with low cuboidal epithelium giving them an almost glandular appearance.

[82] A. J. Lanza, *J. Am. Med. Assoc.*, 106, 393 (1936).
[83] A. J. Lanza, W. J. McConnell, and J. W. Fehnel, *U. S. Pub. Health Repts.*, 50, 1 (1935).
[84] L. U. Gardner, *Ind. Med.*, 9, 45 (1940).
[85] A. J. Vorwald, Experimental studies of asbestosis, *Arch. Ind. Hyg. and Occupational Med.*, 3, 1 (1951).
[86] R. T. Johnstone, *Occupational Diseases*. Saunders, Philadelphia, 1942.

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ported from guinea pigs, and they consist of irregular masses of ferritin granules in which are embedded haphazardly arranged particles of asbestos dust together with any other foreign body inclusions present in the macrophages (FIGURE 9). The needlelike particles described in the previous paragraph can be found in these irregular bodies as well as in normal ones.

In the study of guinea pig asbestos bodies it was reported that segmented bodies were only rarely found and that when they were present they were formed by the uneven deposition of ferritin material rather than the erosion or fracture of a previously smooth layer of body coating. In the human material the picture is much more complicated. In some cases the segmentation still appears to be produced by uneven ferritin deposition, but often the segmentation results from the splitting of a previously smooth coat and does seem to be part of a process by which the asbestos bodies break up.

In summary the electron-microscope examination of lung material containing asbestos dust has produced the following interesting or promising information. Some workers have in the past suggested that only relatively large fibers of [illegible] concluded that very small dust particles were comparatively unimportant. [illegible] that is able to [illegible] particle size and indeed much of it is [illegible] large and small particles.

From these electron-microscope studies, it seems that asbestosis is basically an intracellular process and that the alveolar macrophages and their derivatives are the only cells directly involved. In conditions of continuous dust exposure, of course, there always will be free dust in the alveoli, but the animal experiments indicate that all dust will have been phagocytosed within a few weeks of the cessation of dusting. There is no reason to suggest that this does not apply to humans as well. Indeed in the human biopsy material very little free dust was found, although in both cases the patients had been exposed to dust to within a few weeks of the biopsy. It is therefore the reaction of asbestos dust upon the lung macrophages that needs to be studied if an understanding of the pathogenesis of asbestosis is to be obtained. Conversely the reaction of macrophages to asbestos dust is important, and in this respect the most obvious reaction is the formation of asbestos bodies. It was originally thought that only long fiber dust became coated, but the electron-microscope has shown that the ferritin

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