ASBESTOS CONFERENCE ATTENDEES

June 2, 1972

Biochemicals

J. B. Young - Belle Plant

Central Research

D. J. Beatty - Experimental Station
L. A. Wharry - Experimental Station
E. R. Willis - Experimental Station

Elastomer Chemicals

S. Wenzel - Louisville Plant
W. T. Muncaster - Wilmington
A. F. Myers - Wilmington

Employee Relations

C. W. Eddy - Haskell Laboratory
J. R. Haden - Safety & Fire
D. G. Windsor - Safety & Fire

Engineering

J. R. Allen - Louviers Building
F. H. Fuller - Louviers Building
H. A. Moak - Louviers Building
P. A. Palmer - Louviers Building
J. L. Parsons - Louviers Building
W. M. Uhrich - Construction - Louviers Building
I. Zeise - Construction - Louviers Building

Fabrics & Finishes

J. C. Fang - Marshall Laboratory - Philadelphia
G. A. Johnsen - Marshall Laboratory
T. J. Nelson - Marshall Laboratory
A. J. Geib - Wilmington
G. R. McClure - Wilmington
E. E. Swain - Wilmington

Film

C. Franklin - Clinton Plant
A. E. Barton - Spruance Plant
R. Misak - Tecumeseh Plant
W. E. Neff - Wilmington

Industrial Chemicals

G. R. Amery - Niagara Falls Plant
G. F. Reichwein, M.D. - Repauno Works
J. A. Robbe - Repauno Works
J. G. Haverty - Wilmington

Organic Chemicals

W. L. Sprout, M.D. - Chambers Works
J. C. Warner - Chambers Works
W. E. Acquard - Corpus Christi Plant
C. L. Hobbs - Jackson Laboratory

Photo Products

F. Reig - Brevard Plant

Pigments

J. T. Cavanaugh - Edgemoor Plant
R. M. Luckring - Edgemoor Plant

Plastics

Y. L. Power, M.D. - Carney's Point
E. O. Randolph - Carney's Point
A. Schelling - Carney's Point
J. B. Armitage - Experimental Station
H. H. Gibbs - Experimental Station
S. Hatrman - Sabine River Works

Polymer Intermediates

J. P. Hooten - Cape Fear Plant
D. Zippler - Savannah River Plant

Textile Fibers

J. W. Chromy - Chestnut Run
J. M. Martin - Chattanooga Plant
P. V. Nolan, M.D. - Chattanooga Plant
W. S. McMahon - Kinston Plant
B. R. Bolton - Martinsville Plant
S. J. Bright - May Plant
W. O. Cotty - May Plant
C. F. Higgins, M.D. - May Plant
E. C. Shepherd - Old Hickory
C. M. Gardner - Seaford Plant
F. S. Klein - Seaford Plant
R. P. Coon - Waynesboro Plant
W. J. Gatzak, M.D. - Waynesboro Plant
P. F. Philyaw - Waynesboro Plant
R. E. Sayre - Waynesboro Plant
J. G. Bernard - Wilmington
M. L Bradley - Wilmington
S. Danby, Jr. - Wilmington
R. E. Ferrari - Wilmington
R. B. Hayden - Wilmington
J. A. Sigman - Wilmington
H. E. Swink - Wilmington

ASBESTOS SEMINAR

June 2, 1972

8:45 a.m.	Introduction	
8:50 a.m.	Types and Uses of Asbestos	R. J. Hubiak
9:05 a.m.	Health Effects of Exposure to Asbestos	C. F. Reinhardt, M.D.
9:30 a.m.	Changing Requirements under OSHA	J. F. Morgan
10:00 a.m.	COFFEE BREAK	
10:30 a.m.	Medical Surveillance of Asbestos Workers	H. J. Trochimowicz
11:00 a.m.	Surveillance of Working Environment	R. J. Hubiak
11:45 a.m.	LUNCH	
1:00 p.m.	Means of Dust Control	Com. S. Barbee, USN
1:45 p.m.	Potential Substitution of Asbestos in Insulation	G. E. Lang, III
2:00 p.m.	Discussion of Topics	
3:30 p.m.	Adjournment	

INTRODUCTION

by

John A. Zapp, Jr.

Let me say a few words about why we're having this particular Conference on Asbestos. If I were to rank those chemicals made and used by Du Pont in order of concern to me, I would not put asbestos at the top of the list. But, it has the spotlight on it at the present time for other reasons. There are enough workers throughout the U.S. who are exposed to asbestos and there are enough cases of occupational disease resulting from this exposure to have caused labor unions and the Federal Government to give this problem of asbestosis, and associated lung cancer, the first, and primary attention, under the Occupational Safety and Health Act of 1970. This attention occurred two ways. First of all, asbestos is the first chemical substance to have been made the subject of an emergency standard. This was on the motion of the labor unions that insisted that the current level of exposure as permitted under the Occupational Safety and Health Act of 12 fibers/ml was too high, and therefore, they put pressure on the Department of Labor to set a lower emergency standard. This was set at the current recommended level by the American Conference of Governmental Industrial Hygienists at 5 fibers/ml. This number five is not as low as the labor unions would have liked, so that pressure has continued on the Department of Labor to establish a permanent standard lower than 5 fibers/ml. The mechanism for getting at a permanent standard that affects health, is for the Department of Labor to request the National Institute of Occupational Safety and Health to prepare a criteria document

and recommend a standard. So we have another first. This was the first criteria document recommending a permanent standard to be prepared by the National Institute of Occupational Safety and Health and to which they have complied with the requests of the Secretary of Labor by issuing a criteria document, entitled Criteria for a Recommended Standard--Occupational Exposure to Asbestos. A criteria document is not a standard. A criteria document simply describes the effects observed at different levels of exposure. Based upon the criteria document however, one can recommend a standard. Criteria documents are descriptive. They describe. Standards are prescriptive. They say what you can do and what you can't do. This particular document contains a recommended standard and the criteria on which that recommended standard is based. However, it should be pointed out that this document itself does not necessarily reflect the final standard that will be adopted by the Department of Labor. And, I might just quote Dr. Marcus Key, Director of the National Institute of Occupational Safety and Health, in a talk he gave in February where he said, "Please remember, under the Occupational Safety and Health Act, HEW's recommendations are not standards. The Department of Labor will review our recommendations and those of ad hoc review committees and hold public hearings, if necessary, and will promulgate the final standard." Now the public hearings on asbestos have already been held. They lasted about two weeks, and I understand that it accumulated quite a stack of testimony. Now, the next phase of the current phase is the digestion of this testimony by the Department of Labor. Out of that will come their recommendation for a final standard that will be published

in the Federal Register to take effect on a certain time after publication. We have not yet reached that point. But because this document is out, because it recommends a low standard of 2 fibers/ml, this standard itself has achieved maximum exposure conditions. It has excited more of a discussion and a lot of questions were being asked by people who might have to deal with this problem. As a result of this attention focused on asbestos, plus the intended standard which we don't really know too much about yet, that has caused us to feel that the time was right for another Seminar on Asbestos.

What we're going to try to do today is to give you some of the dimensions of the problem, but again I caution you. The final standard has not yet been promulgated so don't go back and say you have to do this right away. It may well be that the Department of Labor will not adopt all the recommendations of NIOSH after they have considered all of the testimony before them, all comments of experts, professional societies, industry, and labor. It may be for political reasons they will adopt it. We don't know. So that part is still an unknown. But, I hope that you will leave here today with a better understanding of some of the reasons for the recommendations of the National Institute of Occupational Safety and Health or NIOSH, as you will hear it referred to, and, perhaps a better understanding of what you may have to do when the final standard is adopted.

RJH/neh/jtd
June 28, 1972

HEALTH EFFECTS OF EXPOSURE TO ASBESTOS

by

Charles F. Reinhardt, M.D.

The diseases shown on the first slide are the ones which may be associated with exposure to asbestos. First, is the primary one which is asbestosis--a form of fibrosis of the lung. Then there are at least two types, and possibly more, of cancer. There is bronchogenic cancer--a cancer of the bronchial tubes of the lung. This is the common type of cancer associated with exposure to asbestos. Then there is mesothelioma, a rare form of cancer. It is cancer of the pleura and peritoneum. The pleura is the thin membrane which covers the surface of the lung and lines the thoracic cavity. The peritoneum is the same type membrane which lines the abdominal cavity and covers the surface of the organs within the abdomen. In addition, although not listed here, there may be other types of tumors associated with exposure to asbestos since there is an increased incidence of cancer of the gastrointestinal tract in asbestos workers.

(Next Slide) Asbestosis was first described about the turn of this century in London at a post-mortem examination by Dr. Montague Murray. After that, for the next 20 years or so, there were few deaths attributed to asbestosis. However, in the 1930's, asbestos was shown to be a major cause of disability in workers in the asbestos textile industry in England and in the United States. The latent period for this disease is 10 to 20 years. This is the period of time which elapses between the first

exposure and the onset of the disease. This is an average which is shown here and it may be shorter or it may be longer, it could be as short as 5 years under very dusty conditions.

Now to move on the symptoms and signs of asbestosis. First, let me mention that the onset of this disease is incideous, that is, it may creep up on an individual and he may be unaware that it is present. One cause of this is that the early symptoms, that is, shortness of breath and chest pain, are rather common types of symptoms that are not particularly characteristic of this disease and therefore, diagnosis at this stage is somewhat uncertain. However, later in the disease, certain signs will occur which are more diagnostic. The first of these is basal râles. This is a physical finding of the lungs which is due to a collection of fluid in the base of the lungs and is detected upon examination of the lungs. There may be clubbing of the fingers, which means an enlargement of the ends of the fingers. Clubbing may also involve the toes. There are usually characteristic x-ray changes of the lungs; typically, there would be a ground-glass appearance. This is due to the diffuse interstitial fibrosis which is characteristic of asbestosis. This type of fibrosis is in contrast to the nodular type seen in silicosis. There may be pleural thickening which may be detected on the chest x-ray. Other findings may include abnormal lung function. I will not describe what each of these is in detail because they are somewhat technical in nature. However, in general, there is a restrictive functional pattern. In asbestosis, the lungs become stiff and lose their elasticity or compliance. One of the characteristic findings is impaired diffusion. This refers to diffusion of gases across the alveolar-capillary membrane in the lungs.

Lungs from urban areas contain typical asbestos bodies in small numbers. Depending on whose figures you read, asbestos bodies may be found in from 50 percent to 100 percent of the lungs which are examined. One complicating factor here is that other mineral fibers can become coated in the same manner and maybe this is why the term ferruginous bodies is preferred by some.

(Next Slide) I thought you might be interested in some of the theories of the pathogenesis of asbestosis, i.e. how it occurs or why it occurs. Originally there was the theory of physical irritation and this means simply what it says. That is, the presence of asbestos fibers in the terminal air spaces causes irritation and damage to the walls of the alveoli of the lungs leading to fibrosis. However, this theory is not generally accepted today. Then there is the solubility theory which says that fibrosis is due to the effects of the silic acid and metal ions leaching out from the asbestos fibers. Again this is not a widely accepted theory on the pathogenesis of asbestosis.

Then there is the autoimmune theory. There are two concepts here. First, the presence of asbestos in lungs and its reaction with phagocytes or fibroblasts produces or localizes abnormal globulin leading to fibrosis. Phagocytes and fibroblasts are kinds of cells which may be found in the lungs. Phagocytes are of particular interest because they are the cells that engulf or scavenge foreign materials that enter the lungs.

The second concept under the autoimmune theory is that there is lysis of trapped phagocytes. They would be phagocytes which have picked up asbestos fibers and they release a substance that is not accepted by the

tissue as self. That is, it is a foreign material in the lungs and therefore the body reacts to it by producing fibrosis. Again, these two theories, the autoimmune theories, are not widely accepted. However, the fourth theory, the stagnation of phagocytes, which is somewhat similar to the last one I mentioned, says that the phagocytes disintegrate in situ or in place in the lungs and in doing so release sclerosing agents which are probably lipids or lipoproteins. This is the theory which is currently favored as explaining the development of asbestosis.

(Next Slide) Moving on from asbestosis, we come to bronchogenic cancer. This is the type of cancer of the lung which one normally refers to when speaking of lung cancer; that is, the same type of cancer that occurs in cigarette smokers. It is cancer of the bronchial tubes. In the 1930's, an association was discovered between asbestosis and bronchial cancer. This was especially true in asbestos textile workers in whom an excess risk was firmly established in the 1950's. I mention this because this group of workers apparently has a high exposure to asbestos dust. In other groups of workers this relationship has not been found to be as strong. The association appears to be with asbestosis rather than simply exposure to the asbestos dust. Cigarette smoking is an important additional and possible synergistic factor. In fact, Dr. Selikoff estimates an increased risk factor of 90. That is, the risk of developing cancer in an asbestos worker who smokes would be 90 times greater than an asbestos worker who doesn't smoke. This is quite something and I think it emphasizes the importance that if one is an asbestos worker, he should not smoke. The latent period here is 20 to 30 years; if you remember, this contrast with 10 to 20 years for the onset of asbestosis.

(Next Slide) Mesothelioma, which as I mentioned on the first slide, is a rare type of cancer. It is a diffuse cancer spreading over the surface of the lungs and pleura and abdominal organs and peritoneum. One of the interesting things regarding mesothelioma of the abdominal cavity is how the asbestos fibers reach the peritoneal surface. In fact they have been found not only on the peritoneal surface but in other organs in the abdominal cavity. No one is sure how they get there. Perhaps they migrate.

The first large group of cases of mesothelioma was reported in South Africa in the late 1950's and it was found that this form of cancer is associated particularly with exposure to crocidolite fibers. However, apparently it is not limited to this type of asbestos. In the case of mesothelioma, asbestosis is often absent as contrasted to bronchogenic cancer. The latent period here is 40 years and you can see that the latent period is stretching out for each one of these conditions. It is rarely less than 20 years for mesothelioma. Smoking has not been shown to be a contributory factor as in the case of the bronchogenic cancer. Exposures are not always occupational. Several cases have been found in persons living near a source of asbestos and even in members of families of asbestos workers. That is, the workers come home with contaminated clothing and apparently there is enough asbestos on this clothing to cause this type of disease in members of the family.

(Next Slide) What are the possible mechanisms for the carcinogenic behavior of asbestos? Several theories have been put forward. The first of these suggests that metal complexes found in asbestos naturally, or they can get there from processing the fibers may be responsible. Such

metals as iron, chromium, or nickel may be involved. Particularly the latter two, in certain forms, are known to be carcinogenic. Second, oils associated with asbestos may be a factor. These may be naturally occuring or they may be picked up from sacks or they may occur in emulsions added for processing. It has been found that these oils contain benzapyrene and related polycyclic aromatic compounds which are known to be carcinogenic. Another theory is that asbestos may act as a co-carcinogen, that is, by itself it may not be a carcinogen but in the presence of other materials, it may produce cancer. Lastly there is the prolonged residence in the tissue of a chemically inert material which is incapable of being removed by phagocytosis. This is referred to as the Oppenheimer effect, but it is generally not thought of significance in this case because it seems to apply to larger inert bodies which have been placed in tissues. Also, asbestos cannot be regarded as a chemically inert substance.

In summary, asbestos may produce several types of disease, namely, the characteristic asbestosis or fibrosis of the lungs and certain forms of cancer--the bronchogenic cancer or a rare type of cancer, mesothelioma.

CFR/neh
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