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AMERICAN THORACIC SOCIETY
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UNION CARBIDE

UNION CARBIDE CORPORATION OLD RIDGEBURY ROAD, DANBURY, CT 06817

January 12, 1987

Dr. Raymond L. H. Murphy
Pulmonary Services, Inc.
1153 Centre Street
Boston, Massachusetts 02130

Dear Dr. Murphy:

Re: Letter to Reuben Cherniak, M.D.
from Drs. Franzlau and Lilis

I have read the above-mentioned letter with interest and reviewed the writers' comments on the ATS statement on "The Diagnosis of Nonmalignant Diseases Related to Asbestos." I would comment as follows on the letter and these comments represent my personal viewpoint and should in no way be construed as an expression of Union Carbide's opinions:

Paragraph 2, Page 1: Franzblau and Lilis' attempt to discredit a perfectly reasonable comment on the state of world production of asbestos by alleging that the decline is due to economic factors rather than health factors. This may be so, but nowhere in the ATS document is there an attempt to "engender a false sense of security." By quoting the statement out of context, Franzblau and Lilis are the ones who are attempting to be misleading! The ATS statement is quite definite that "The cumulative production of asbestos, however, continues to increase." Franzblau and Lilis acknowledge this. It is my opinion that the health hazards of asbestos are probably the best known of all the occupational lung disorders and that throughout the world preventive control measures now exist. If there is no safe exposure level, then Franzblau and Lilis are justified in their comments on the expanded use of asbestos in third world countries or countries with managed economies. It remains to be seen whether the permissible exposure levels in countries other than the United States are more or less effective in preventing the non-malignant diseases related to asbestos and whether, in fact, the current PEL in the U.S. is justified.

I find Franzblau and Lilis' comments on page 2, paragraph 1, confusing. We have stated in the concensus document that: "Microscopically, plaques are seen to be laminated collagenous connective tissue, acellular, with few inflammatory or fibrocytic nuclei; many are covered by a thin layer of regular and well-differentiated mesothelial cells. Capillaries are rare. Elastic staining shows intact lamellae beneath the plaque in continuity with the surrounding normal paretal pleural connective tissue, suggesting that plaques are extrapleural and develop between the latter and its covering layer of mesothelial cells." Franzblau and Lilis imply ulterior motives in separating

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PLAINTIFF'S EXHIBIT
UC-4351

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the description of pleural plaques from that of pleural thickening and thereby display their ignorance of the differences in site and appearances of these two lesions. I can but conclude that they would wish to include three separate pathological processes into a single diagnosis on the basis that they are all due to the fibrogenic properties of asbestos. This is illogical since asbestosis is used specifically to describe bilateral diffuse interstitial pulmonary fibrosis and through common usage is an accepted diagnostic term. By adding the words asbestos-related before pleural plaques or pleural thickening, the etiological diagnosis can still be made and there is no loss of its significance. Franzblau and Lilis are surely aware that pleural plaques can be discovered without evidence of pulmonary fibrosis and pleural thickening usually accompanies pulmonary fibrosis. In the former instance, a diagnosis of asbestosis cannot be made since pulmonary fibrosis has not been demonstrated, whereas in the latter the primary diagnosis is most likely asbestosis with pleural thickening present as a complicating factor.

On the subject of "Exposure History" I can only say that the writers' comments are fatuous and pedantic and if they took the trouble to read the entire section and did not merely attempt to infer double meanings from the choice of certain of our words, they would realize the correctness of our comments. If, as they tell us they are experienced "in evaluating effects of asbestos exposure," they should be familiar with the term "direct contact" as opposed to "casual" or "indirect contact." These terms are referred to in the section.

The issue as to whether a radiographic diagnosis of asbestosis requires a profusion of small opacities of 1/1 or greater is one which cannot presently be resolved. This is certainly the appearance which is radiologically unequivocally acceptable and I suppose the quality of the film and the experience of the X-ray reader are less likely to affect recognition of abnormality at this level of profusion. Nevertheless, Franzblau and Lilis have a point when they suggest that a reading of 1/0 indicates the presence of radiographic abnormalities. It is important that patients be made aware of abnormalities as soon as they are detected so that they can be kept under more intensive surveillance and guard against factors likely to aggravate their condition.

With regard to the comments on the omission of pleural abnormalities from the final summary, I do not find justification for these remarks. Pleural lesions are commented on and to describe them would not serve any real purpose since the ILO U/C classification which we refer to provides adequate guidance on the diagnostic features. It would appear that the writers of the letter reserve the real motives for their concerns to the last sentence of their letter and that is that by differentiating between the pulmonary fibrotic effects of asbestos exposure and the extra-pulmonary effects they will have difficulty in justifying their expert evidence in lawsuits based mainly on pleural lesions (the commonest asbestos-related lesion) rather than on disabling pulmonary fibrosis (or asbestosis). Perhaps we were remiss in our

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document in not defining asbestosis as a disabling form of pulmonary fibrosis resulting from exposure to asbestos, defining pleural plaques as benign and non-disabling lesions and indicating that diffuse pleural thickening could aggravate either of the former conditions.

I hope my impassioned comments are of some use to you if you decide to draft a response to Dr. Franzlau and Dr. Lilis.

Best wishes for 1987.

Sincerely,

H. C. Lewinsohn

Hilton C. Lewinsohn, M.D

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RECEIVED
JAN 7 1987
H. C. LEWINSOHN, M.D.

RAYMOND L. H. MURPHY, JR., M. D.

December 31, 1986

To: Committee Members

Prom: Raymond L. H. Murphy, M.D.

Re: Attached

I received this letter from Dr. Reuben Cherniack. If you have any comments, please let me know.

Sincerely,

Raymond L.H. Murphy, M.D.

RLHM/dt

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November 22, 1986

Reuben Cherniack, M.D.
Editor
American Review of Respiratory Disease
National Jewish Center for Immunology
and Respiratory Medicine
1400 Jackson Street
Denver, Colorado 80206

Dear Sir:

The recent consensus statement by the Ad Hoc Committee of Scientific Assembly on Environmental and Occupational Health[1] (the Committee) regarding the diagnosis of nonmalignant asbestos-related disease had the stated purposes of summarizing "the current state of knowledge while pointing out areas where additional information is necessary", and to "summarize [the] present knowledge on the diagnosis of nonmalignant asbestos-related pulmonary disease". Unfortunately, the desired clarification of this complex issue was not achieved, and in some instances additional confusion was generated. The following are comments on some of the important issues raised.

The statement, "World production of asbestos has dropped markedly since the mid 1970's" is misleading, and engenders a false sense of security regarding the trend of asbestos impact on worker health. The decrease in world production of asbestos since the all-time peak of 1973 is probably attributable to the world-wide recession of the mid-1970's[2]. The impact of environmental and health concerns on asbestos utilization is only a phenomenon of Western industrialized countries, specifically the United States. Third world countries, countries with managed economies (the Soviet Union, eastern Europe, Peoples Republic of China), and many industrialized nations have maintained or expanded their production and use of asbestos from 1978 through 1985[2,3]. More significantly, the U.S. Bureau of Mines predicts a 4% annual <u>increase</u> in use of asbestos in the United States from 1985 through 1990[3]. As the authors of the consensus document suggest, the asbestos materials currently in the environment will present hazards as they deteriorate and are disturbed during repair, replacment, and removal. The cumulative burden is not only growing, but at an increasing rate.

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The section entitled "Benign Pleural Abnormalities Associated with Asbestos" is confusing. The term 'pleural plaques' has been used to describe circumscribed pleural fibrosis. Diffuse pleural fibrosis, which by definition is extensive and involves the costophrenic angles, does not fit the term 'pleural plaques'. Throughout this section 'pleural fibrosis', which is the pathologic process underlying both the circumscribed pleural abnormalities (pleural plaques) and the diffuse pleural thickening, is only mentioned once. This is a significant choice of terminology since it obscures the relationship between the fibrotic changes which can occur in both the pleura and parenchyma. Once it is recognized that asbestos is fibrogenic for the pleura, the reason to separate the fibrogenicity for the pleura from that for the pulmonary parenchyma, and to include only the latter in the definition of asbestosis, disappears.

Thus, the definition of asbestosis chosen by the Committee is artificially restrictive. The justification offered for the exclusion of pleural abnormalities is that "there are differences between pleural and parenchymal fibrosis in epidemiology, clinical features, and prognosis". Although such differences exist, it is an arbitrary and illogical distinction. Numerous examples could be given of diseases in which manifestations with different clinical features and prognosis are included within the usually accepted definition. Some examples are tuberculosis, rheumatic fever, silicosis, and rheumatoid arthritis.

Under "Exposure History" it is stated that "particular attention should be paid to occupations in which direct contact with asbestos has occurred". No clarification of the meaning of "direct contact" is provided, and many readers may infer that the Committee intended to imply personal handling of asbestos during a worker's occupation. If this is the case, this recommendation is not only confusing but also misleading. It is well known to those who have experience in evaluating effects of asbestos exposure that many, and possibly a majority, of patients recently and currently presenting with asbestosis have had exposure by virtue of working in areas where only a small fraction of employees personally handled asbestos. This applies to the shipbuilding and ship repair industry and the construction industry with its many trades. The spraying of asbestos on steel beams is mentioned in the next paragraph, and is an excellent example of a hazardous exposure for workers who had no "direct contact" with asbestos, i.e. who did not spray, but who worked (as electricians, carpenters, welders, etc.) in such areas.

In the summary section it is stated that a number of "necessary" conditions have to be present for a diagnosis of asbestosis to be considered. Specifically, in the absence of a pathologic specimen, a patient must have an appropriate history of exposure and latency interval. We agree with these criteria. However, the subsequent "clinical criteria" raise a number of significant questions. First, the Committee has chosen a radiographic appearance of small opacities corresponding to an ILO classification of 1/1 or greater as a cut-off for a diagnosis of asbestosis. The ILO guidelines do not specify a threshold of parenchymal opacities for the diagnosis of

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asbestosis, and do "not imply legal definitions of pneumoconiosis for compensation purposes, nor set nor imply a level at which compensation is payable"(4). The choice of the above mentioned cut-off is therefore somewhat arbitrary. In our opinion the Committee has decided upon a threshold which is too restrictive. We believe that an ILO classification of 1/0 is more appropriate. By the choice of "1" for the numerator, a clinician or radiologist is indicating that he/she has interpreted a particular film to be abnormal, although "0", or normal, was a consideration in the deliberations. With a level of 1/1 as a cut-off, the Committee appears to encourage practitioners to tell patients with a suitable exposure history and latency, small opacities with profusion 1/0, and no pleural disease, that they have no evidence of asbestos-related disease. From the perspective of patient care, and also, we believe, for the purposes of workers' compensation and liability, 1/1 is too restrictive. It is well known that the PA chest x-ray is not the most sensitive test for interstitial fibrosis, and that as many as 10% of pathologically proven cases of asbestosis will have "normal" chest x-rays (0/0 or 0/1 ILO classification)(5).

An important omission in the consensus document is the complete absence of pleural abnormalities from the final summary of diagnostic criteria for asbestosis. From its title ("The Diagnosis of Nonmalignant Diseases Related to Asbestos"), one would anticipate that the guidelines would help the reader in the diagnosis of both pleural and parenchymal asbestos-related disease. As noted above, the omission of pleural disease from the diagnostic criteria of asbestosis was by design; the definition of asbestosis was chosen to exclude pleural abnormalities. The impression given is that such findings are irrelevant to the diagnosis of nonmalignant asbestos-related disease. Although benign pleural disease results in significant functional impairment only when extensive(6), the goal is the definition of criteria for the diagnosis of nonmalignant asbestos-related disease, and not the degree (or lack thereof) of functional impairment. In addition, the well accepted concept that pleural plaques are a marker of asbestos exposure(7) is not given the focus and attention it deserves. Should a patient with an appropriate exposure and latency, calcified pleural plaques, and "normal" parenchyma (0/0 or 0/1 ILO classification) be told that he has no evidence of biologic effects of asbestos exposure?

In the development of diagnostic criteria one must begin by outlining the goals of such an endeavour, particularly since all nonmalignant asbestos-related conditions are untreatable, except for symptomatic relief. Of utmost importance to clinicians should be their responsibility to their patients, and in this case the advice, prognostic information, palliative therapy, and emotional support which can be offered. As noted in the consensus document, asbestos and asbestos-related disease have become significant public health and public policy issues. Obviously, physicians have an important role to play in these situations. Numerous physicians are, and will continue to be, enmeshed in the legal conflicts resulting from workers' compensation claims and third-party liability suits. Finally, the epidemiologic study of diseases, including asbestos-related conditions, requires that investigators have valid and reproducible

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criteria for defining and diagnosing the disease under study. Although a restrictive definition of a disease may have an attraction for some, we believe that the Committee has formulated diagnostic criteria for nonmalignant asbestos-related disease which are too restrictive from the perspectives of patient care, workers' compensation, third-party liability, public health and public policy.

Alfred Franzblau, M.D.
Ruth Lilis, M.D.

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10 East 102 Street
New York, New York 10029

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