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M E M O

To: Participants in Asbestos Industry Response to the Proposed Asbestos Regulation

Subject: Task Force Status Report -- Forwarding of Comments by Hans Weill, M. D. on Proposed Revision to OSHA Asbestos Standard

The Association's Executive Committee will meet with Mr. Guy G. Gabrielson, Jr., Chairman, AIA/NA OSHA Standard Task Force, Thursday, March 25 for review of documents to be submitted to OSHA by the announced comment deadline April 9. On Friday, March 26, the "comment package" will be presented at a meeting of the Association's directors following which the documents, including the comprehensive report of the Roy F. Weston, Inc. technological feasibility and economic impact study, and specific recommendations for changes to the proposed revision to the standard, will be mailed to all participants for endorsement. It is recognized that little time will be available for review of the documents by those not attending the Board of Directors meeting. The time requirements of the task offer no alternative if we are to meet the April 9 deadline.

Because of the significance of the comments prepared by the Association's medical consultant, Dr. Hans Weill, on the medical science issues raised in the proposed revision to the OSHA standard to those participants who are preparing company or trade association response to OSHA in addition to endorsement of the AIA/NA task force presentation, Dr. Weill's statement is forwarded with this memorandum.

We are pleased to advise that the Association continues to increase its membership and a number of non-member companies and associations have contributed to the extraordinary expense of consultant fees and task force administrative costs. We are appreciative of the splendid cooperation of participants in the Association's efforts to assist the Department of Labor in the development of a practicable standard for the protection of asbestos workers.

R. H. Mereness
Executive Director

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Enclosure

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Statement by Hans Weill, M.D., on Rules on Occupational Exposure to Asbestos proposed by OSHA (Docket No. H-033)

By way of introduction, I am Professor of Medicine at Tulane University School of Medicine and direct a large interdisciplinary program in the investigation of occupational lung diseases. These research activities involve all varieties of inhalants including mineral and organic dusts and chemical vapors and gases. The goal of our program is to provide the scientific basis for the prevention of occupational lung disorders. Specific aims are to establish, whenever possible, causal relationships between environmental conditions and a definable biologic response, dose-response relationships, and threshold levels of exposure below which the adverse effect does not occur. As an independent university-based research unit, our work is in large part supported through competitive federal funding mechanisms, including the NIH and NIOSH. Published results of our investigations are available for peer review in the scientific literature. Because of the appropriate interest of responsible industry in protecting the health of their workers, a portion of our research activities has also been funded from industry sources. Our investigative interest in the health effects of asbestos exposure dates back approximately six years with a significant portion of this research having been accomplished with the collaboration of the Medical Research Council Pneumoconiosis Unit in Britain.

I would like to make clear my role in submitting these comments on the proposed new standard for occupational exposure to asbestos. I was asked to prepare an objective, scientific assessment of the literature cited by OSHA in support of its proposed standard change by the Asbestos Information Association of North America. I accepted this consultative task because I firmly believe that industry, in order to meet its occupational health responsibilities, must have available to them outside advice from academic and other sources. It is my objective in the following discussion to provide scientifically-based and dispassionate judgments on the issues raised and in no way assume an advocacy or adversary position.

There should be no doubt or confusion concerning the reasons or indeed justification for the setting of occupational health standards by regulatory agencies: these standards are set to protect workers exposed to environmental hazards in the work place. The standard-setting process should begin with a scientific data base providing quantitative information on the relationship between the environmental exposures and any associated health effects. Without data for both sides of this equation for a particular health hazard, resulting information is incomplete in terms of the important function of setting standards for safe levels of occupational exposures. Although these requirements have now been recognized by both government and non-governmental scientists, the fund of hard data of this type which is

available is clearly limited. This deficiency does not, of course, justify delay in the setting of standards using the best available quantitative information, however incomplete. Nor does our limited knowledge justify abdicating the role of science which is rationally applied to the problems of occupational health. Such a negative approach may be employed to support either extreme of little or no control on the one hand and unrealistically stringent control on the other, and is self-defeating and certainly counter-productive. It is often forgotten that standards, like science, are not written in concrete and as scientific evidence provides more information on dose-response relationships, the standard setting process must be responsive by means of regulatory action in either direction. It is clearly stated in the published OSHA proposal that the lowering of the permissible level of eight-hour time-weighted average exposure to 0.5 asbestos fibers per milliliter is based upon new medical and scientific evidence which has become available since the last asbestos standard was promulgated in 1972. My subsequent remarks will focus on this "new evidence".

There should be little debate concerning the causal association between occupational exposure to asbestos dust and certain adverse health effects, including but perhaps not limited to asbestosis, bronchogenic carcinoma of the lung, mesothelioma of the pleural and peritoneal surfaces, and gastrointestinal neoplasms. Most references cited by OSHA, dating

back to 1907, do not relate to the central issue, that being at what level (if any) of exposure do such effects fail to occur. Documenting that these health effects have occurred in workers who have had years of exposure to asbestos dust without relating measurement or reasonable estimates of past exposures to these effects has been of great importance, but does not materially assist in the standard setting process. It is respectfully suggested that the great majority of the 42 references cited in the proposal must be classified in this way.

Because of the emphasis placed by the writers of this proposal on a few, mainly unpublished, recent reports, these will be reviewed in some detail. Few studies have received as much attention and imputed importance in this standard setting process than the epidemiologic investigation of a cohort of workers employed in an asbestos textile plant in the industrial midlands of England. However, it is with some dismay that our British colleagues view the current use of their data, which is in many ways incomplete, by scientists and regulatory agencies in the U.S. Certain facts seem indisputable. The initial report published by Professor Doll in 1955 demonstrated a clear excess in respiratory cancer and pulmonary fibrosis in workers who had previous exposure for 20 or more years in this plant (1A). Follow-up of mortality data published in 1968 provided evidence that workers having their initial exposure since the asbestos regulations

took effect in 1933 had a decrease in lung cancer and asbestosis mortality and even suggested that the hazard of bronchial carcinoma had been eliminated in this population (13). It was clearly stated, however, that longer follow-up was necessary and "the data are insufficient to estimate the extent of the risk which may remain". While the improved health status of the more recently exposed workers was encouraging, limited dust exposure data presented indicated that this cohort had been exposed to average levels of asbestos dust which were often higher than current U.S. or U.K. standards.

The most recent update on the mortality experience of this cohort was presented at the International Congress on Occupational Health, held in Brighton, England, in September, 1975 (30). This continued follow-up for an appropriately longer period of time revealed a modest excess of respiratory cancer in the working population first employed since 1933. Perhaps of greatest interest was that even in a sub-cohort comprising 255 men and 93 women entering the industry for the first time since 1951 and having more than 10 years exposure, five deaths from respiratory cancer were observed in individuals with more than 15 years since first exposure. The expected number would be 1.86, an excess which was statistically significant at $p = .04$. This risk was found to be greatest in those individuals having more than 20 years' exposure. Importantly, while it was indicated that major dust control measures were completed in this plant in the late 1950's, no specific

dust exposure data were presented nor were claims made in regard to the utility of these results in setting safe standards or in generating dose-response relationships between asbestos exposure and risk of developing respiratory cancer.

Because of the importance placed on this study and the recent orally presented (but not published) report, and the requirements outlined above for the scientific basis of rationally promulgated occupational health standards, I undertook a visit to this plant during the week of January 12, 1976, with the hope of obtaining specific dust exposure data during the period since 1951, with the view of correlating level of exposure to asbestos dust with the demonstrated mortality results. Exceptional cooperation by the medical director, industrial hygienist, and management of this plant resulted in their providing extensive exposure information for this cohort, which formed the basis for the following comments. Dust data between 1951 and 1960 are based on sampling using the Casella Thermal Precipitator giving particles per milliliter, and since 1961 are based on static or area sampling using the membrane filter method and providing fibers per milliliter. For the decade prior to 1961 (when fiber counts first became available), the particle counts were converted to fiber levels. Using these data, an individual exposure estimate was reconstructed for each member of the mortality cohort for the period 1951 through 1974, using yearly average data

obtained for each job site in order to provide cumulative exposure during the total work time in this plant for all of those employees entering the industry since 1951 and having ten years or more exposure. While my purpose is to summarize and highlight relevant aspects of this information, fuller exposition of the data can be obtained by requesting direct testimony from plant officials, possibly for the hearings to be held on this proposed standard. It should also be noted that concurrent with my receipt of these data, the identical information was supplied to Dr. John Gilson, Director of the Medical Research Council Pneumoconiosis Unit in South Wales, who is concerned with the British Occupational Hygiene Society (BOHS) standard (see below) and Professor Richard Doll of the Oxford Epidemiology Unit, the author of the mortality papers.

The current U. K. chrysotile-asbestos standard was published by the Department of Employment in 1970 with the permissible continuous exposure level being 2 fibers per milliliter . For intermittent exposure up to 12 fibers per milliliter the action required by the factory inspectorate is dependent upon the level and duration of the exposure. Exposure data from the study plant clearly indicate decreasing fiber levels during the 1960's so that in many job areas in the plant dust levels after 1970 were at or near the standard of 2 fibers per ml. It is fair to state that the goal of reaching this standard is being achieved in this factory. However, fiber counts between 1961 and

1972 reveal mean values in some job areas ranging as high as 26 fibers per ml., with standard deviations close to that number, indicating that some individual counts would have to have been many times the current standard in either country. Again, it must be emphasized that in the main these very high values were obtained prior to the 1969 asbestos regulations (U.K.). However, in carding and several other job sites, more recent sampling data continued to show some average values of 4, 5 and 6 fibers per ml., with standard deviations around the mean again indicating individual fiber counts at higher levels.

Among those individuals first exposed to asbestos dust in this plant since 1951, ten have now been certified by the Pneumoconiosis Medical Panel as having asbestosis. The minimum exposure period of this group was 10 years, maximum 18 years, mean of 14 years; and the cumulative mean group exposure was 170 fiber-years per ml. All of these workers were men and entered the plant between 1951 and 1956. Their individual cumulative exposure calculations ranged from 66 to 280 fiber years per ml.

Six individuals in the cohort first employed since 1951 have died of respiratory cancer, five of these whose time from initial exposure to death was greater than 15 years. These persons were all cigarette smokers whose year of first exposure ranged from 1952 until 1956. In each of these six individuals, the average yearly exposure to asbestos while employed exceeded

4 fibers per ml. Asbestosis was indicated on the death certificate in two of these six respiratory cancer deaths, but obviously this in no way indicates the absence of histologic (or radiographic) evidence of dust-related pulmonary fibrosis in the remaining four.

While the concept of a cumulative exposure calculation is probably deficient in that it ignores the residence time of fiber in the lungs and concentration of airborne fiber, the current U. K. asbestos standard is based on the premise that a cumulative exposure of 100 fiber-years per ml. over a working lifetime constitutes an acceptable risk in terms of adverse health effects. In this regard, it should be noted that approximately one-third of the post-1951 cohort has had cumulative exposures exceeding this amount in what has been considerably less than a "working lifetime". Maintenance workers in this plant (and in the rest of the world) have had intermittent high exposures continuing to the present time. In this plant, this is documented by recent dust data obtained between carding machines grouped within an enclosure where ordinarily production workers are not stationed. Some of these fiber counts are in the 20's and 30's but with mean values generally below 10 fibers per ml.

Finally, the exposure analyses reveal an interesting group of 58 workers in the weaving department whose average exposures have usually been 2 fibers per ml. or below. Cumulative individual exposures in this

sub-population have been below 75 fiber years per ml. in workers first exposed since 1951. It is of interest that in this population, there have been no cases of certified asbestosis or lung cancer, encouraging preliminary evidence in regard to the safety of a 2 fiber per ml. average exposure.

There have been no cases of mesothelioma in the post-1951 cohort of this plant.

The conclusions to be drawn from the biologic and exposure data from this asbestos textile plant since 1951 are reasonably straightforward. It is probable that an excess risk for the development of respiratory cancer exists in the post-1951 cohort being studied by the Oxford group. The numbers are small and Professor Doll indicated to me in January, 1976, that this excess has not yet been firmly established. In addition, asbestosis has appeared in members of this cohort. In this population, asbestos exposures during the past 20 or more years have on average clearly exceeded the current 2 fibers per ml. standard and these studies in no way invalidate that standard in regard to its adequacy in protecting from asbestos-related health effects. The use of this report (30) in supporting a change from a standard (2 fiber per ml.) which has not yet become operational in the U.S. to 0.5 fibers per ml. must be considered at best invalid and at worst misleading.

Often not recognized, particularly in the U.S., is the fact that the current British asbestos standard of 2 fibers per ml. is not based on the Doll

mortality studies discussed previously. The standard is the result of a recommendation by the British Occupational Hygiene Society (BOHS) published in 1968 (10). This standard of 2 fibers per ml. for chrysotile asbestos was suggested by the BOHS subcommittee on asbestos after review of morbidity data provided by Dr. John Knox, medical director of this same British asbestos textile plant. While both clinical and radiographic information was considered in assessing the asbestosis risk, the earliest indicator of disease (upon which the standard is based primarily) is the finding of inspiratory rales on auscultation of the lungs. This standard was reviewed by the BOHS committee and their findings published in 1973 (2A). They concluded that no change in the standard should be recommended at that time but there should be continuing review. Approximately one year prior to this published review of the asbestos standard in Britain, an article authored by the current chief medical officer of this British plant, Dr. Lewinsohn, precipitated considerable controversy, primarily emanating from this side of the Atlantic (14). It was suggested that this "new information" indicated a greater prevalence of radiographic changes of asbestosis than the previous work by Dr. Knox had documented. The arguments are complex, if not confusing, but certain points should be mentioned. The experimental classification used in determining radiological change in the Lewinsohn paper results in significant differences from those published in the BOHS report. The interpretation of the observed radiological abnormal-

ities with respect to their significance to asbestosis was not attempted in the Lewinsohn paper. Equating the lowest category of radiographic change with the disease, asbestosis, is obviously of questionable merit and as previously indicated, the x-ray appearance was not the primary basis for the establishment of the BOHS standard. In view of the described dust exposure levels in this plant since 1951, the controversy concerning differences in interpretation of morbidity data is less relevant in assessing the safety of a 2 fiber per ml. average exposure standard. The BOHS subcommittee has for the past several months been reviewing the asbestos standard in the U.K. Updated dust exposure data similar to those described previously are being correlated with the various indicators of a biologic response in exposed workers, including clinical, radiographic and physiologic information. A recent meeting of this subcommittee (mid-January, 1976) failed to result in even a preliminary position concerning the asbestos standard and it appears that it will be some months before a report to the full BOHS standards committee will be completed. It is only after the BOHS committee has approved the final report that it will become generally available.

Considerable attention has also been directed toward the report of an investigation by NIOSH presented at the Conference on Occupational Carcinogenesis, New York Academy of Sciences, in March, 1975 (41). This study entitled "Morbidity and Mortality among Hard Rock Miners exposed to an

Asbestiform Mineral", has been cited by OSHA in support of their proposed change in the standard for occupational exposure to asbestos. The study purports to show that a population of miners who have been exposed to non-commercial amphibole fibers (cummingtonite, grunerite), in low concentrations and with the preponderance of fibers shorter than 5 microns in length, have experienced an excess of respiratory cancer associated with this exposure. Widespread critical comment concerning this study, its design and interpretation of results, has resulted in two major substantive revisions by the authors since the OSHA citation.

Among workers having achieved a minimum of five years underground gold mining experience by 1960, ten deaths due to malignant neoplasms of the respiratory system were observed with an expected of 2.7. Two of these tumors did not involve the lung, being classified as carcinoma of the maxillary sinus and mediastinum, locations not previously related to occupational asbestos exposure. In two additional tumors of the lung, it was not specified whether these lesions were primary or secondary in the lung. In the remaining six, a primary malignant neoplasm of the bronchus and lung was specified. The possibility must be considered that a definite excess of primary respiratory cancer in this population has not been demonstrated. Perhaps of greater importance is the fact that this population has been exposed to a number of potentially carcinogenic materials. In the early drafts of their paper, it was stated that arsenic levels of 5 to 6

micrograms per cubic meter were measured in this mine in 1974. Although the authors preclude the possibility that arsenic (a known carcinogenic material) may have played a role in any excess respiratory cancer risk, a NIOSH investigator added to the list of authors in the last draft presented a separate paper on inorganic arsenic at the same New York Academy of Sciences meeting in March with the following statement included in the published abstract of this report: "the only quantitative epidemiological study, reported in 1974, revealed a dose-response demonstrating an increased lung cancer mortality risk at arsenic concentrations above 1 microgram per cubic meter, calculated as the average occupational exposure over a 40-year work life". The conclusions of these NIOSH investigators in this same meeting seemed contradictory, and it is difficult to understand why specific reference to the arsenic levels have been deleted from the final gold mine study draft.

Two of these same NIOSH investigators have previously pointed out the pulmonary carcinogenic effect of radon daughter exposures in the uranium mining industry (3A). Dr. Wagoner also authored a paper entitled "Unusual Cancer Mortality among a Group of Underground Metal Miners" published in the New England Journal of Medicine in 1963 showing a respiratory cancer excess of a magnitude similar to that claimed in the gold mine study (4A). No etiologic factor was firmly established, although trace metals,

arsenic, and radioactivity were mentioned as possible causes for this observed mortality experience. In a 1971 monograph published jointly by the National Institute for Environmental Health Sciences (NIEHS) and NIOSH, reference is made to the underground hard rock mining experience reported in 1963 with the conclusion that although radon daughter exposure levels were low in 1958, previous exposures were probably significantly higher due to poorer ventilation in past years (5A). Certainly, this same explanation for significant radon daughter exposure levels in the past could also have been applied to the gold mine study but for some reason was not. It is also very curious that in none of the three drafts of the gold mine study was there reference to the 1963 metal mining publication although the current study claims to show excess respiratory cancer mortality which the same author had already described in metal mining more than ten years previously. The causal factors may have been different in the two mines, but this is by no means proven.

Additional confusing factors concerning the gold mine study should be noted. The initial draft contained morbidity information concerning the prevalence of radiographic changes thought to be consistent with pneumoconiosis. The final draft has dropped this aspect of the report. In the second draft of this paper, the authors state "These samples indicated an airborne silica concentration of 3.3 to 7.2 milligrams per cubic meter,

both of which exceed the TLV for silica". These airborne silica data are excluded from the last draft. This is of particular interest since the authors claim that a statistically significant excess in non-malignant respiratory disease was noted in this cohort. In the earlier draft, this excess is entirely explained by a diagnosis of "silicosis" in four of these miners. It would be difficult to associate the finding of silicosis at death with past exposures to small asbestos fibers. This subject is further confused when in the last draft the term "pneumoconiotic disease" is substituted for silicosis as the cause of death in the non-malignant respiratory disease category.

Smoking analysis, which was included in earlier drafts, is no longer present in the pre-publication or last draft. Additionally, the single paper which was presented orally at the conference was divided into two papers after the second draft, the additional paper now entitled "Asbestos Fiber Exposures in a Hard Rock Gold Mine". It describes the amphibole fiber measurements indicating an average concentration of 0.36 fibers per ml. greater than 5 micron in length, and an average total fiber concentration of 4.82 fibers per ml., with 94% of the fibers being less than 5 microns in length. The conclusions of this second paper, however, include broad statements concerning the health effects of this exposure, none of which are supported or even dealt with in the results reported in the manuscript.

It can be appreciated from the above that there is little or no evidence presented to support an association between the fibrous dust exposure in this gold mine and an excess risk for the development of respiratory cancer in a cohort of past mining employees. These workers have been exposed to a number of potential carcinogenic agents and indeed an excess mortality risk for respiratory cancer in metal mining had previously been demonstrated by the NIOSH group. The exposures to these other potential inhalants, particularly radon daughters and arsenic, have been inadequately characterized in this cohort, particularly in the last draft. Information from previous drafts suggests that at least for arsenic, the exposures may have exceeded those levels suggested separately by one of the authors to be associated with an excess respiratory cancer risk. In view of these comments, it is difficult to understand OSHA's justification for citing this study in support of its proposed asbestos standard change.

I would like to turn now to a briefer summary of the additional "new evidence" cited by OSHA in support of the proposed standard change. A cancer risk was again noted in a cohort of insulation workers being followed by Selikoff (16). This review is essentially an update of previously presented data but does not relate the mortality experience with information concerning past asbestos dust exposure. As with most of the other studies available, no information upon which numerical standards of asbestos exposure can be

based is forthcoming in this latest report presented at the International Conference on Biological Effects of Asbestos, in Lyon, France, in 1972. At this same meeting, Dr. Selikoff's group again confirmed the interaction between asbestos exposure and cigarette smoking and the risk of developing carcinoma of the lung (20). It was suggested that this risk may also extend to asbestosis but again in the absence of exposure information, it is difficult to justify the inclusion of this report in the literature purporting to support the asbestos standard change. Also made available in the recent past is a paper reporting the presence of asbestosis, lung cancer, and mesothelioma in a cohort of workers who have had past exposure in an amosite insulation manufacturing operation (18). It is clearly stated that "no information is available concerning dust levels in this plant" and this interesting study is hardly useful in the setting of safe asbestos standards. A study of insulation workers in Belfast, published in 1971, confirmed the New York insulation experience and found excess mortality in asbestos-exposed insulators for the specific causes of lung fibrosis, lung cancer, mesothelioma, and gastro-intestinal malignancy (26). Of interest is that the lung cancer cases had associated pulmonary fibrosis (asbestosis) while those individuals who died of mesothelioma did not. This result speaks to the controversy concerning whether asbestos-related lung cancer is associated with a level of exposure which has also resulted in pulmonary fibrosis. Again, in the absence of

exposure information, this report does not help in the standard-setting process. A report by Enterline in 1972 (17) reveals that in workers engaged in the manufacture of asbestos products who have had mixed fiber exposures that maintenance men had a greater risk of developing respiratory cancer than production workers, presumably because of high intermittent exposures. It was also pointed out that men in maintenance jobs may have had a higher crocidolite exposure and that this type fiber may be more carcinogenic than chrysotile. That crocidolite exposure at similar total asbestos dust levels may be more hazardous in regard to the development of asbestosis was recently reported at the Fourth International Symposium on Particles and Vapors, in Edinburgh, in September, 1975 (6A). Little relevance to occupational standard setting is gleaned from these reports except that they suggest the possibility that, as in the U.K., there should perhaps be a different occupational standard for crocidolite fiber than there is for chrysotile.

Three reports are cited in the OSHA proposal which draw attention to the health effects associated with past employment in dockyard and shipyard workers (35, 39, 40). No dust exposure information is available in these studies and the comments made above in regard to requirements for standard-setting apply equally to these reports. Fletcher (40) suggested that a better association existed between malignancy and pleural plaques than with pulmonary fibrosis.

A number of cited references concern themselves with the association between asbestos exposure and the risk for the development of pleural or peritoneal mesothelioma (21, 22, 23, 25, 32, 33, 34, 37, 38). As no environmental dust data are reported in any of these studies, dose-response relationships can to date not be established for these malignant tumors. Considerable credit should be given to Wagner (21) for recognizing and reporting this association in 1960. These tumors have been reported in cases where the exposure had been occupational but also where contact had been in the household or in the vicinity of an industrial or mining asbestos source. It should be recognized, however, that these non-occupational sources of exposure are not necessarily low but probably of the "intermittent high level" type, perhaps similar to those exposures experienced by individuals in factory maintenance jobs. In the absence of more precise information, the demonstration of these associations does not help in setting safe levels of asbestos exposure. Certainly, there is no scientific basis for concluding that household or other non-occupational exposures have been in the range of 2 fibers per ml. or less. Newhouse and Berry (25) reported on a statistical model designed to predict future mesothelioma rates in a cohort of workers previously employed in an asbestos textile factory near London. Past exposures had been admittedly high prior to the closing of the plant in 1968. Dust levels, however, were not available and this

interesting paper presented at the Brighton meeting in late 1975 again does not help us in setting standards. Webster (32) reported a differing risk for the development of mesothelioma in South African residents in regard to two deposits of crocidolite asbestos (Cape and Transvaal). He suggests the possibility of an additional mineral in the Cape crocidolite area which had the higher mesothelioma rate but other evidence suggests that the differing structure of fibers from these two sources may prove to be the explanation for these differing biologic effects. Greenberg and Lloyd Davies (33) point out that two-thirds of their cases of mesothelioma registered by the Employment Medical Advisory Service in Britain had a recognizable past exposure to asbestos.

Two reports are cited in the proposal suggesting an association between asbestos exposure and laryngeal carcinoma (27, 28). This association, while requiring confirmation, is certainly not surprising in view of the potential for fiber deposition in the upper respiratory tract. Its relevance to the changing of an asbestos occupational standard is unclear.

Edge (42) emphasizes the presence of pleural plaques in asbestos-exposed men who ultimately developed the described health effects associated with this exposure. No quantitative environmental information is available. Anderson (15) reports radiographic abnormalities consistent with asbestos-associated effects in a group of household members of workers in the

amosite plant previously discussed. Both pleural and parenchymal changes were found but the association of these radiographic abnormalities with asbestos exposure must be studied further and the x-ray readings should be confirmed. Assuming the changes to be present, the past level and intermittency of exposure in these households is completely unknown. Considerable asbestos dust must have been brought to the homes by workers returning from a factory without significant dust control, since the same New York group of investigators have recently found evidence of significant asbestos fiber accumulation in these homes up to the present time.

An interesting and provocative paper presented at Brighton by Nicholson (19) reviews the factors involved in arriving at a threshold limit value (TLV), particularly the limitations of this approach. While this review or editorial provides stimulus for further scientific and philosophical discussion concerning the standard setting process, it does not claim to present evidence supporting the proposed change in the asbestos standard to an average of 0.5 fibers per ml. I agree with the statement in Dr. Nicholson's summary: "in the case of asbestos, current exposures can only be described crudely at any level of exposure, and health effects are only known for past high, but ill-defined, exposures." Finally, in another thoughtful paper cited by OSHA, Berry (11) stresses the importance of acceptable risk as balanced by the benefits of using the particular material

for which the standard is being considered. He uses as an illustration the promulgation of the 1968 BOHS standard of 2 fibers per ml. (10).

The remainder of the references cited in the OSHA proposal including an article by Jane Brody in the New York Times must be considered irrelevant to the current standard setting procedure and hardly requires further discussion.

Finally, it seems to me that there are two major alternatives presently available in the setting of an occupational standard for asbestos exposure. The first depends upon the premise that the adverse health effects demonstrated in workers have resulted from high but poorly quantitated levels of asbestos dust. Where such information is available, dose-response relationships have indicated that for mortality from malignant disease (7A) and for asbestosis (8A, 9A), these risks were associated with levels of exposure considerably higher than the current or proposed asbestos standards. That a working population has not had long-term exposure to even current standard levels of exposure has already been emphasized. In the absence of such epidemiologic data, one can hardly find convincing evidence on which to base present further lowering of the asbestos standard. Nor can these non-existent data invalidate the 1976 standard of 2 fibers per ml. Because no population has been available whose working lifetime exposure has averaged 2 fibers per ml., one

cannot finally conclude at this time that this level is "safe" in regard to all health effects recognized to be associated with asbestos exposure, in all exposed individuals. However, it is also impossible to state that the proposed average exposure of 0.5 fibers per ml. for a working lifetime is free of these health hazards. However, the limited information available from studies on both sides of the Atlantic, which attempt to define dose-response relationships, is encouraging in regard to a 2 fiber per ml. standard.

The other alternative depends upon the following argument. Occupational exposure to asbestos at some level has been shown to be associated with a carcinogenic risk. Since a safe threshold level of exposure cannot be scientifically proven at this time, the standard must require that all exposures be at or below the lowest technologically feasible level. The implications of this approach, vis a vis the multitude of carcinogenic materials in our environment, are far-reaching and must be faced. Those who favor this alternative must vigorously support a uniform approach and defend the resulting consequences on life in our society. I favor the first alternative as being prudent and protective of the worker's health in light of the best available current scientific information. The 2 fiber per ml. standard, which is to take effect in this country in mid-1976, has not yet become a reality in either the U.S. or U.K. This standard should be given

a reasonable trial while further epidemiologic investigation establishes its safety or lack of it. In conclusion, I wish to indicate that I am pleased to have had the opportunity to comment on the OSHA proposal for a new asbestos occupational exposure. In view of the importance attached to the most recent report on the British studies, I would like to emphasize and again draw attention to the new exposure information which I have summarized earlier in this statement. It is in light of these past exposures that the biologic data must be interpreted.

References

References with numerals only (1-42) are those cited in the OSHA proposal.

1A through 9A follow:

1A Doll, R. Mortality from lung cancer in asbestos workers, Brit. J. Indust. Med., 12:81, 1955

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3A Wagoner, J.K., Archer, V.E., Lundin, F.E., Holaday, D.A., and Lloyd, J.W., Radiation as the cause of lung cancer among uranium miners, N.E.J.M. 273:181, 1965

4A Wagoner, J.K., Miller, R.W., Lundin, F.E., Franmen, J.F. and Haij, M.E., Unusual cancer mortality among a group of underground metal miners, N.E.J.M. 269:284, 1963

5A Lundin, F.E., Wagoner, J.K., and Archer, V.E., Radon daughter exposure and respiratory cancer; quantitative and temporal aspects, NIOSH and NIEHS Joint Monograph No. 1, 1971

6A Weill, H., Rossiter, C.E., Waggenspack, C., Jones, R.N. and Ziskind, M.M.: Differences in lung effects resulting from chrysotile and crocidolite exposure, Fourth International Symposium on Inhaled Particles and Vapours, In press, 1975

7A McDonald, J.C., McDonald, A.D., Gibbs, G.W., Siemiatycki, J. and Rossiter, C.E., Mortality in the chrysotile asbestos mines and mills of Quebec, Arch. Environ. Health, 22:677, 1971

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9A Weill, H., Waggenspack, C., Rossiter, C., Bailey, W., Ziskind, M. Radiographic and physiologic patterns among workers engaged in the manufacture of asbestos cement products: a preliminary report. J. of Occupational Med., 15:248, 1973.