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before

U. S. Dept. of Labor, Occupational Safety and Health Hearing on Proposed Occupational Asbestos Standard

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My name is George W. Wright. I am the Head of Medical Research in the Department of Medicine of St. Luke's Hospital, Cleveland, Ohio. In 1932 I received the MD degree from Indiana University School of Medicine. After five years of Post Graduate Training in Internal Medicine with special training in pulmonary disease I spent two additional years in research training in the Department of Physiology at Case-Western Reserve University. From 1939 to 1953 I was a member of the Saranac Laboratories of the Trudeau Foundation engaged in studies of the pneumoconiosis, including asbestosis. Since 1953, I have continued this general field of study in my current position.

My statement will be confined to consideration of a single primary question. This question is: What is the quantity or concentration of asbestos, in the air breathed by those engaged in the production, manufacture or use of asbestos products, which can be tolerated for a normally expected work life without risk that such exposure will cause disease? The answer to this question goes to the heart of these hearings. While the question is rather easy to formulate it is difficult though not impossible to answer.

The obligatory information required for developing an answer to the primary question on an acceptable scientific basis is of two categories. One of these essential categories concerns the dose or quantity of asbestos inhaled and the other essential category concerns the biological response. What we need to learn is the dose-risk relationship.

The dose is estimated in terms of concentration of respirable asbestos in the air multiplied by the duration of the exposure. It is obligatory that we establish a dose, in measurable terms, at which the biological manifestation of disease occurs and then, by observing populations exposed to progressively smaller and smaller doses, determine the level at which the biological reaction to asbestos no longer occurs.

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To make observations with regard to the various dose levels of different working populations without knowledge of the biological response of these same populations is of no use in answering the primary question. To make observations of the biological response in various working populations without valid knowledge of the different levels of asbestos exposure experienced by these same populations is equally of no use for finding an answer to the primary question. Standards for a safe level of asbestos exposure proposed or set in the absence of reliable and valid data in both of these two categories must be considered empirical and to some degree arbitrary and should be recognized as such.

I wish to indicate some of the difficulties encountered in efforts to obtain the information essential for determining what the safe level of asbestos in the occupational environment truly is.

Asbestos is a generic term embracing four varieties that are in common commercial use. Of these, chrysotile is used in largest quantities followed by crocidolite, amosite and anthophylite. Each of these has different chemical and physical attributes. Some working populations are exposed to only one variety of asbestos while others are exposed to two or more simultaneously and in varying proportions. Thus there are several kinds of populations each exposed in quite different ways to one or more kinds of asbestos, each of which might react biologically in a different manner. Moreover, workers exposed to asbestos are further exposed simultaneously to other airborne agents. In production, manufacturing and utilization processes these may differ greatly. For example, the asbestos producer is exposed to asbestos with minimal coexisting agents while, in contrast, the insulation manufacturer inhales asbestos plus silica plus other pneumoconiosis producing dusts. Insulation applicators inhale these materials plus a variety of

A08100

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additional agents present in whatever the environment is in the place where he is working at that time. Attention to such coexisting agents has been ignored in studies of insulation workers. Variations of type of fiber and the circumstances surrounding their use must be taken into account in answering the primary question and would suggest the possibility that different standards might be rational in order to meet different conditions of use of asbestos.

There are four, totally different biological phenomena or diseases thought to be related in some way to the inhalation of asbestos fiber. These are pulmonary fibrosis, bronchogenic cancer, thickening of the pleura, and mesothelioma. Not a single one of these diseases is peculiarly related to or caused solely by the inhalation of asbestos fiber. Each of these four diseases occurs "de novo" or for other reasons in persons who have never been exposed occupationally to asbestos. The fact is, the biological reaction attributable to asbestos is not a specific kind of disease induced solely by asbestos. Instead, the biological reaction to asbestos manifests itself by the fact that in some populations occupationally exposed to asbestos, there is, in contrast to nonexposed populations, an excess occurrence of one or more of the four previously mentioned diseases. To determine the safe level for the use of asbestos, we are required to demonstrate the level of exposure to asbestos at which no excess of disease develops when the exposed population is compared to a nonexposed population. In order to establish a safe level we must examine suitable control populations not exposed to asbestos, but age matched and residence matched, from which population one can learn the frequency of occurrence of the specific diseases in question. In addition, we must examine an exposed population, which can be ranked in various levels of exposure, in order to learn at what level an excess of these diseases occur in the exposed population.

A08101

The observation of a biological abnormality in only one or two persons who have been exposed to the inhalation of asbestos fibers does not permit a comparison to suitable control groups or afford contrasting exposure values. Such observations are of no use for determining a safe level standard. Isolated case reports of mesothelioma, brochogenic cancer or pulmonary fibrosis in individuals who may also have had an exposure to the inhalation of asbestos fiber are of no use in setting a safe standard for asbestos based upon sound scientific principles. Larger populations in which only the frequency of occurrence of disease is reported, without the necessary data indicating ranges of exposure and in which no attempt to relate variations of exposure to disease is made, fall into the same nonusable category for setting a valid safe level. This is not to say that observations in such groups are of no value for other purposes, but the issue should not be confused by introducing data from such inadequate studies into considerations of safe levels of asbestos exposure. Suitable control populations made up of individuals in sufficient number who were never exposed to the occupational inhalation of asbestos fiber and occupational groups whose exposures are demonstrated to have wide variation are difficult to obtain but absolutely essential for our purpose. The absence of this kind of control data, excused or overlooked on the basis that it cannot be obtained, is an unacceptable condition if the safe level standard is alleged to be set on the basis of universally accepted scientific principles.

There is difficulty also with assessing the frequency of occurrence of the diseases thought to be related under some circumstances to the inhalation of asbestos fiber. There is disagreement as to the criteria for a valid diagnosis of mesothelioma. The criteria for a diagnosis of "asbestosis," especially in its least severe manifestation, are not generally agreed upon. The use of the chest X-ray for diagnosing the early manifestation of asbestosis is a case in point. A recent report by Murphy and his co-workers of a study of employees in a shipyard demonstrated that

A08102

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twenty percent of the control, or nonexposed population gave evidence of X-ray abnormalities which, if observed in the exposed population in the absence of controls, in all probability would have been interpreted as being evidence of the effect of the inhalation of asbestos fibers. In a population which has been exposed to asbestos inhalation, it is very tempting to ascribe any departure from a perfect appearance in the chest roentgenogram to the occupational exposure, and indeed, in some studies this has been done. It is imperative to recognize that some persons who have never had an occupational exposure to asbestos will show the same X-ray shadows that have been interpreted by some as evidences of asbestosis in populations exposed to the inhalations of asbestos fiber. Studies such as those by Murphy et al exemplify the necessity of having suitable controls included in epidemiological studies bearing on the determination of a safe standard for asbestos.

There are still other difficulties having to do with establishing the dose-risk relationship. The health effects of asbestos inhalation are both dose and lapse-time related. Overt evidences of asbestosis do not appear until years after the initiation of the exposure. The interval between onset of exposure and its effect is even greater for the development of bronchogenic cancer or mesothelioma. This interval ranges between twenty and forty years. Because of the long time lapse between the initiation of exposure and the manifestation of injury, the incidence of disease occurring in populations now under study is the result of, and must be related to, exposures which took place twenty to forty years or more ago. To relate exposure measured only in the past five or so years to the current frequency of development of disease which actually was induced by exposure occurring years earlier, is a serious error. During the past twenty-five to thirty years there have been many technicological changes in the production, manufacture and use of asbestos

A08103

containing material. In many situations there is much less asbestos now being incorporated in the material than was true twenty or more years ago. In addition, there has been a progressive dust control effort to reduce the concentration of airborne asbestos in mines, mills and manufacturing establishments in a deliberate attempt to reduce and abolish asbestos related disease. So much has been accomplished in this direction by technicological change and systematic asbestos control efforts, that contemporary measurements of occupational environments cannot be accepted as representative of conditions twenty to forty years ago.

To summarize this part of my statement I strongly urge that when data offered in support of setting a safe level standard is being judged, one should ask the following questions:

1. What were the different kinds and proportions of asbestos used and what were the coexisting agents to which workers were exposed in the different occupations?
2. What were the specific criteria and methods used, and were they adequate for making a diagnosis of the biological reaction attributed to the inhalation of asbestos?
3. Were suitable controls in the sense of nonexposed populations and exposed populations whose exposures varied in intensity and duration utilized?
4. Was the exposure which was actually responsible for the disease properly determined or were contemporary dust estimates inappropriately applied to disease which was in fact the result of exposure years ago?

A08104

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The demands inherent in these questions pose formidable obstacles to arriving at a scientifically valid single number which will indicate the concentration of occupational airborne asbestos that can be tolerated with safety for the customary duration of employment. In spite of this, as I will show later, there are substantial data which will satisfy the requirements posed by these questions and which can be used for the purpose of determining at least a first approximation to the safe level of occupational exposure to airborne asbestos.

We must now examine the various established and proposed standards for a safe level of asbestos in the occupational environment, in the light of the just discussed requirements as to data needed for setting a standard on a sound scientific basis. Systematic efforts to reduce the amount of asbestos in the air of working places by governmental directive was made in the factories of Great Britain beginning in 1931. No numerical standard for this purpose was set, but Statuatory Rules and Orders were promulgated. Later, on the basis of limited data relating impinger dust counts to X-ray abnormalities in several asbestos textile factories of the USA, Sayers and Dreeson suggested a numerical standard of five mp/ft^3 as a tentative safe level. This was the limit adopted by the American Congress of Governmental Industrial Hygienists in 1946. Utilizing the conversion data for textile mills published by Lynch and Ayers of the U. S. Public Health Service, five mp/ft^3 is the equivalent of approximately thirty fibers/cc.

In 1968 the British Occupational Hygiene Society suggested numerical guides aimed at reducing the risk of developing asbestosis. The suggested guides indicate that those concentrations, averaged over a three month period below 2 fiber/cc ought to be considered low, and that over the course of 50 years of employment, 100 fiber years is the level below which there would be less than a 1% chance of developing asbestosis as defined chiefly by the presence of rales.

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If asbestosis is defined by X-ray abnormality, the level becomes 135 fiber years. In 1968 the ACGIH changed its recommended standard to a TWA of 2 mp/ft^3, or twelve fibers/cc. In 1970 this Committee decided to change the standard to 5 fibers/cc, which is equivalent to less than 1.0 mp/ft^3. Several months ago OSHA set an emergency standard of 5 fibers/cc, and within the past few days NIOSH now proposes a standard of 2 fibers/cc.

What is the scientific basis for these various standards? The standard proposed by Sayers and Dreeson did attempt to relate dose of dust containing asbestos as measured by the midget impinger to the risk of developing pulmonary abnormalities in terms of abnormal X-ray patterns. Their study showed a higher frequency of abnormal X-ray patterns in those most heavily exposed as contrasted to those less exposed. They found no excess of abnormal chest X-ray patterns in those workers exposed to less than 5 mp/ft^3. Hence their suggested standard of 5 mp/ft^3. We now know, however, that the population studied was not observed long enough for the full range of disease to develop. The authors appreciated this possibility and proposed the standard as being a tentative one. In retrospect we know that the standard, although it led to marked lessening of the dustiness in some occupations, was not adequately based and the passage of time has demonstrated it to be an inadequate standard.

The numbers proposed by the British Occupational Hygiene Society are based on a study of 290 male employees from a single asbestos textile mill. Their employment began after 1933, all had worked for at least ten years, and the exposure period extended from 1933 until 1966. The indicator of disease was basal rales and X-ray changes. No measurements of dustiness in this mill were available prior to 1950. Membrane fiber counts done in the modern manner were used only in the last year of the study and these served as the factor for converting previous counts using

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ACL Doc 16673 · Page 9 of 22 asbestosclaims.law/library/16673/9

The lowering of the 1938 standard from 5, to 2 mp/ft^3 and 12 fibers per cc, adopted by the ACGIH in 1968 appears to have been based in part upon the data underlying the British proposal and also upon the fact that there was a growing awareness that the old standard of 1938 was not universally effective. The further lowering to 5 fibers/cc was not based upon new scientific data, but appeared to have been made to establish a margin or "factor of safety." In a personal communication from Dr. Herbert Stockinger, Head of the Division of Laboratory and Criteria Development of the National Institute of Occupational Safety and Health and Chairman of the Committee on Threshold Limit Values of the American Conference of Government Industrial Hygienist, I am informed that four months ago, in November of 1971, the Committee of the ACGIH reviewed the standard of 5 fibers/cc and found no evidence to indicate that it should be changed. There are several relatively current studies which support this decision.

I would now like to discuss five of these supporting studies. Murphy et al, in December of 1971 published data on workers exposed over a period of approximately thirty-five years as pipe coverers in new ship construction. The authors demonstrate that no evidence of asbestosis was found until at least 60 million particles per cubic foot years of exposure had been exceeded. This would indicate that for a forty year period of employment, 1.5 million particles per cubic foot would be the value below which no asbestosis occurred. The report indicated that the actual occurrence of asbestosis was restricted almost solely to those exposed to more than 75 million particles per cubic foot years, which would be close to a value of 2.0 million particles per cubic foot for those employed for forty years. Utilizing a conservative number to convert particles to fibers per cc, one arrives at a level of 5 or more as being that below which no asbestosis was observed. If one utilizes their konimeter data, one can, by suitable calculations,

A08108

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estimate that the average exposure for these workmen was in the neighborhood of 8 fibers per cc, and this too would suggest that a number of 5 or more fibers per cc would have been the level below which no evidence of asbestosis developed. This study indicates, not only that the old standard of 5 mp/ft^3 is unsafe but also that a standard of 5 fibers/cc will prevent asbestosis in employees working for forty years or more.

An extensive and detailed study of a population comprising thousands of workers in the mining and milling of chrysotile in eastern Canada conducted over the past few years by Dr. Corbett McDonald and his associates is of great importance in our considerations because this study has dust counts going back many years and chest roentgenograms available from approximately 1935 to date. Dr. McDonald will report on these studies at these hearings. It is my understanding that these studies support the conclusion that those individuals exposed in these occupations for forty or more years would have to be exposed to higher than 6 fibers/cc in order to reveal any evidences of asbestosis.

Becklake and her coworkers, utilizing a segment of the population from McDonald's larger study, have demonstrated that in those individuals who have been employed for long periods of time in the mining and milling of chrysotile asbestos and who do not even show evidence of X-ray changes, the accumulated exposure must have been more than 110 mp/ft^3 years before the most sophisticated techniques can detect evidence of those physiologic changes thought to develop in response to inhalation of asbestos. These data suggest that something larger than 2.5 million particles per cubic foot over a forty year period can be tolerated without even the most subtle manifestations of asbestos exposure. This number would translate into something between five and 10 fibers per cubic centimeter.

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ACL Doc 16673 · Page 11 of 22 asbestosclaims.law/library/16673/11

In the fall of 1971 at the Fourth International Congress on Pneumoconiosis, held in Bucharest, Dr. McDonald updated his previously reported large study and reaffirmed that an excess of bronchogenic cancer did not occur in the workers in mining and milling of chrysotile asbestos until an exposure equivalent to 200 million particles per cubic foot years was exceeded. This translates into 5 million particles per cubic foot for forty years. It indicates that in the mining and milling of chrysotile an excess of bronchogenic cancer would not be demonstrable in those individuals who were exposed to less than 5 million particles per cubic foot. This is equivalent to 10 or more fibers per cc. This most important finding supports the view held by Steward and others that an excess of bronchogenic cancer occurs only in those who have manifestations of asbestosis and that the standard adequate to protect against bronchogenic cancer is a larger number than that required to protect against asbestosis.

Recently, Enterline and his coworkers have reported the results of a study of retirees employed in various asbestos-using manufacturing processes including textiles and where the exposure was different from that in mining and milling because of the inclusion of exposure to crocidolite and amosite as well as other agents which might coexist. This study shows, and I quote "there appeared to be no direct relationship between asbestos dust exposure and respiratory cancer below 125 million particles per cubic foot years. Important increments in respiratory cancer mortality apparently occurred somewhere between 100 and 200 million particles per cubic foot years' exposure." This would indicate that exposure to 3 million particles per cubic foot would be tolerated for a forty year period without a risk of excess bronchogenic cancer in these types of occupations. Using the conversion factor suggested by Lynch et al, this is equivalent to 18 fibers/cc.

A08110

This series of studies indicates that a lower standard is required to protect against pulmonary fibrosis or asbestosis than to protect against bronchogenic cancer. These five studies offer strong support for the position taken by the American Conference of Governmental Industrial Hygienists that there is no need to lower the standard below 5 fibers/cc in order to protect against pulmonary fibrosis or bronchogenic cancer.

In my judgment we have no specific information of a suitable scientific nature expressing the dose-risk relationship for mesothelioma. Isolated case reports of mesothelioma are of no value in establishing a dose-risk relationship. Equally useless for this purpose are the data offered in studies of insulation workers, asbestos miners and millers, workers in asbestos using manufacturing, domestic exposures and neighborhood exposures. Not a single one of these studies offers data comparing variable exposures to the incidence of mesothelioma and they are therefore of no use for establishing a safe level of asbestos in the occupational environment. These studies do indicate that in some circumstances the risk is present but none speak to the question of the safe level on scientifically acceptable grounds. Nevertheless, these studies of larger numbers of cases of mesothelioma suggest that, in some circumstances the exposure to asbestos which may be related to the development of mesothelioma might be less than that required to produce asbestosis or an excess of bronchogenic cancer. One factor, which has been demonstrated with respect to the relationship between mesothelioma and inhalation of asbestos is of the utmost importance. In all of those circumstances where a strong relationship between mesothelioma and the inhalation of asbestos is suspected, two varieties of asbestos, namely crocidolite and amosite, invariably have been implicated either alone or combined. This is the case in the excess occurrence of mesothelioma among crocidolite miners, asbestos using insulation workers, workers in asbestos textile and insulation manufacturing and in a large

A08111

multiproduct asbestos using manufacturing plant in the USA. In most of these circumstances there has been a mixed exposure and chrysotile has also been present. In striking contrast, however, when exposure has been solely to chrysotile, as in mining and milling of chrysotile, the frequency of occurrence of mesothelioma has either not been increased or increased only to the very slightest degree. The virtual absence of an excess of mesothelioma in populations exposed solely to chrysotile has been reported by Gilson, Vigliani, Wagner and McDonald on the basis of separate studies. With respect to anthophylite, no excess of mesothelioma has ever been reported. On the basis of information currently in hand, therefore, mesothelioma appears to be predominantly linked with exposures to crocidolite or amosite but the dose relationships are unknown.

The studies referred to thus far are the only ones that I am aware of which in my judgment have a meaningful bearing on the setting of a standard for control of asbestos exposure. Other data does exist but in my judgment it does not conform to those criteria, universally agreed upon, for a sound scientific basis upon which to predicate a safe standard.

There is an abundant other body of data which indicates that the prolonged inhalation of asbestos fibers above some level is hazardous, that the biological effect is dose related but that many of the workers exposed for a life-time never do develop asbestos related disease. This data indicates clearly that past exposures in some occupations, as for example the mining and milling of chrysotile were far less hazardous than exposures in asbestos textile manufacture or the insulation workers' trade and possibly in other occupations as well. The reasons for these differences are not entirely resolved at this time. Until recently the insulation trade uses much more amosite than chrysotile and in some work, especially in England, large amounts of crocidolite have been used. There

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are coexisting agents in the insulation trade which are not present in mining and milling of chrysotile. The peak exposures appear to be much higher in the insulation trades than in mining, milling or manufacturing. Peak exposures may be much more hazardous than lower levels of prolonged exposure. Fiber counts in the insulation trade in shipbuilding in Great Britain and in construction in Finland ranged from twenty to hundreds of fibers/cc depending upon the kind of work being done. Fiber counts in the construction industry in this country currently indicate somewhat lower TWA levels but intermittent high peaks of exposure. For example, during the mixing of asbestos cement, dust counts commonly as high as 75 fibers/cc during a 5 to 10 minute period are reached as often as 5 times or more a day. Unfortunately, there are no data in the insulation worker trade that compare degrees of exposure over a period of many years to the biological manifestations of excess disease. Until this is available, there is no way of utilizing data from these trades to establish a standard for the control of asbestos inhalation.

There is every indication that the levels of exposure should be lowered, especially the peak levels, but whether or not a lower TWA standard is needed in the insulation trade than in the other trades remains unsettled. There is substantial reason to believe that crocidolite and amosite should be controlled more stringently than is chrysotile. There is also reason to believe that the geometric conformation of the fibers in the mining and milling of chrysotile may be different from those fibers generated subsequently in manufacture and use of asbestos. Moreover, the fibers of crocidolite and amosite even in mining have a different geometric configuration, as actually observed by Timbrell in airborne specimens, from that of some forms of chrysotile. For these reasons the pulmonary retention of chrysotile may be substantially less than for other varieties of asbestos or for the kinds of fibers generated in other occupations than mining and milling. Animal experiments have demonstrated this to be true.

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ACL Doc 16673 · Page 15 of 22 asbestosclaims.law/library/16673/15

On the basis of data now available and adequate for the purpose, I wish to indicate that in my judgment a standard of five fibers/cc TWA with peaks not to exceed 10 fibers/cc, such as recommended by OSHA in its present standard and by the ACGIH constitutes a level which will protect against the development of asbestosis and bronchogenic cancer and includes a safety factor since it is a level substantially below that which has been demonstrated to be associated unequivocally with an excess of these two abnormalities. There is evidence relating crocidolite and possibly amosite to excess mesothelioma which suggest that a more rigorous control of amosite or crocidolite would be justified. For this reason it may be rational to have different standards covering different occupations depending upon the variety of asbestos used.

Since I am in disagreement with the recommendation of the Criteria Document that two fibers/cc be established as the safe level of occupational airborne asbestos, it is incumbent upon me to indicate the important points of my disagreement.

In my opinion it is incumbent upon the authors of the Criteria Document to present evidence, which will meet the requirements for scientific validity, in support of their proposal to change the standard and to establish a new one. In the entire document I do not find reference to a single study containing data which will, on the one hand meet the criteria of scientific validity for setting standards and, on the other, support the need to change the standard from 5 fibers/cc or to set it at 2 fibers/cc. In fact, the Criteria Document explicitly says on page 10 of Section 5 "The number of studies that have collected both environmental and medical data and with a significant number of exposed workers, is not sufficient to establish a meaningful standard based upon firm scientific data."

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In other words, the authors of the Criteria Document do not provide any scientific data upon which to base their proposal to change the standard from 5 to 2 fibers/cc or to support the choice of a standard of 2 fibers/cc. The failure to provide this support, in my judgment casts the most serious doubts upon the validity of the proposals.

I am in complete disagreement with the Criteria Document with respect to its expressed opinion that the data relating exposure to biological reaction is inadequate to establish a meaningful standard at this time. While the evidence may not be as far reaching as we would like, it is scientifically valid and adequate to support as a first approximation the opinion that the present standard, established by OSHA several months ago at 5 fibers/cc TWA and peaks not to exceed 10 fibers/cc should not be lowered but left as it is. I have cited the studies supporting this opinion.

Lacking dose-risk relationship data adequate to support the proposal of 2 fibers/cc, the Criteria Document appears to promote an aura of extreme toxicity of asbestos fibers in order to justify the new level. This is done chiefly by citing isolated case reports. In my judgment an inordinate amount of weight is given to isolated case reports which, though of interest and some importance, have no possible utility in setting a numerical standard. Their use in the Criteria Document is doubly difficult to accept in view of the fact that on page 24 of Section III there is the following statement "Isolated clinical case reports are difficult to interpret in terms of dose-time response relationship and can only be used to indicate other possible problem areas and to highlight what may prove to be practicable areas for further study."

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ACL Doc 16673 · Page 17 of 22 asbestosclaims.law/library/16673/17

require discussion in a document intended to be a fully developed presentation of the evidence supporting recommendations of such great importance.

It is difficult to understand why McDonald's report is criticized, as stated at the top of page 20 of Section 3, because of the conversion of numbers of particles to numbers of fibers. Particles are converted to fibers by the British Occupational Hygiene Society in developing their standard and at no place in the Criteria Document, pertaining to the British recommendation, does one see a questioning of that procedure. If it is acceptable in the one circumstance it should be acceptable in the other.

It is even more difficult to understand the criticism expressed in the last sentence of the 1st paragraph on page 20 of Section 3 to the effect that the McDonald study lacks the evidence necessary to make general comparisons with the data of other reported work. The published paper by McDonalds' group contains the complete biological data and the required exposure data expressed in mp/ft^3. This is substantially more data than that furnished by others since it relates exposure to biological effect in the same population whereas other studies such as those of insulation workers do not in any way provide this corelated data. The index of dustiness used in the McDonald report is the same as in those of Murphy et al, Decoufle-Enterline and Williams, all of which are quoted in this document. The criticism that McDonald did not indicate the details of the method used for converting $particles/ft^3$ to fibers/cc could easily have been answered by a letter or a telephone call, if the authors of the Criteria Document had been desirous of making the comparisons.

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It is not possible to evaluate fully the exposures from the data shown in some of the Tables of this document. For this we need to know not only the TWA's but also the peaks, then number of observations and the conditions of the environment during the time of sampling. This criticism is especially pertinent to the exposures of insulation workers shown in Table 14. The insulation worker experiences great variability with respect to job conditions, to a large degree dictated by the volume of the space in which he works and by natural ventilation as well as the kind of work he is doing at the time. Dust counts under all sorts of conditions, and peak as well as TWA exposures must be measured before the insulation worker's exposure can be portrayed. With respect to Table 14, it is surprising that the job of "cement mixing," notoriously characterized by peaks of exposure as high as 75 fibers/cc for periods of 5 to 10 minutes several times a day, is omitted.

The summary, on page 16 of Section 5, describing the basis for the new standard recommended by the Criteria Document is confusing and inconsistent. I quote from the second paragraph, "evidence indicates that past and current standards for fiber concentrations in the working places where asbestos fibers occur, though undoubtedly contributing to reduction of the severity and frequency of asbestosis, have not provided complete protection from exposure to asbestos, necessitating development of a new standard." There is evidence that asbestosis developed under some circumstances, in places thought to have an average exposure of five million particles per cubic foot, or in modern parlance, thirty fibers/cc. This was the standard set in 1938. The ACGIH created a new lower standard in 1968 and another still lower, introducing a safety factor in 1970.

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The British Occupational Hygiene Society suggested numbers that might serve as a standard in 1968. The current standard in the USA set by OSHA is five fibers/cc, and was set only a few months ago. If there is to be a new standard it will be one set since a few months ago, or at most, set since three years ago. The statement in the Criteria Document, to which I have just referred, indicates that the authors have evidence that the standard recommended by ACGIH in 1968 has been inadequate and has not provided complete protection and hence a new standard is needed. The authors of the Criteria Document should be required to give that evidence and it is obvious that they have not. Could it be that they are referring to the fact that the standard set in 1938 is inadequate and that a better standard is needed? If so, a lower standard was provided in 1968 and still lower in 1970, and it is this standard which was promulgated as an "emergency standard" by OSHA. It is this OSHA standard for which I believe there is substantial factual evidence of a supportive nature.

An even more serious inconsistency is demonstrated on page 16 of Section 5. Close examination of Items a, b, c, and d reveals in unequivocal terms, that in the judgment of the authors the Criteria Document there are no scientifically usable data on which to base a standard. Nevertheless, without such data they decide that the current standard must be changed and moreover recommend precisely what the new one should be. There is a strong inference from the second paragraph of page 17, Section 5 that one and perhaps the chief reason for setting the recommended standard at two fibers/cc is that the British have recommended such a standard. I disagree with the concept, if actually intended, that this represents a scientifically valid reason for recommending such a standard. Moreover, it is my understanding that the British did not set a standard in the same sense that it is intended to be used under the new Occupational Safety and Health Act. The British Standard is a series of numbers

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ACL Doc 16673 · Page 20 of 22 asbestosclaims.law/library/16673/20

proposed to furnish a guideline with a wide range of personal judgment on the part of their inspectors as to the need for corrective measures when these numbers are exceeded. Under our act it is my interpretation that a standard is to be set which, if not complied with, places the operator under certain penalties. To my mind the two conditions are utterly different.

A08121

standard. It should be emphasized that prior to 1940 and for some years thereafter, when many of those persons now developing disease were actually being exposed, the dust counts in mining, milling and manufacturing commonly were in the range of 10 to 100 mp/ft^3. This was most likely equivalent to 30 to 600 fibers/cc. There is no evidence that exposures during that period for insulation workers was substantially lower and, on the basis of British and Finnish data, it was in that range. It is clear then, that the curent OSHA temporary standard represents a tremendous reduction from the dust levels of previous years and, in my opinion, includes a substantial safety factor. I believe this level will protect workers, using asbestos over a forty year or more span of employment, from developing asbestosis or experiencing an excess of bronchogenic cancer. If crocidolite and perhaps amosite are more stringently controlled, the liability of experiencing mesothelioma will be markedly reduced and probably abolished.

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ACL Doc 16673 · Page 22 of 22 asbestosclaims.law/library/16673/22