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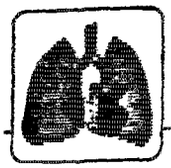

special report

Asbestos-Associated Diseases*

Science, Public Policy, and Litigation

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PLAINTIFF'S EXHIBIT

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The asbestos-associated health effects have, during the past ten years, commanded a unique position in the awareness and concern of individuals and various groups in our society. This unprecedented interest has extended well beyond the medical, public health, and biomedical research professionals, and there is no occupational health issue (a special set of public health concerns) which has had as much potential for interaction between social scientists, research investigators, government officials, industrial management, labor organizations, and members of the legal profession. The effectiveness in utilizing an emerging scientific data base in dealing with the public policy issues has been strikingly variable. Ethical, economic, and other social implications have resulted from occupational exposure to asbestos, and it is likely that in the United States, we are currently at or near the peak effect in terms of the various manifestations of this impact.

Historical Perspective

Commercial use of the naturally occurring fibrous mineral, asbestos, began in the last two decades of the previous century. By the 1930s, it was generally recognized that the use of asbestos fiber in manufacturing processes could lead to pulmonary fibrosis called asbestosis.[1] During the period between the 1930s and the passage of the Occupational Safety and Health Act by the US Congress in 1970 (establishing the Occupational Safety and Health Administration [OSHA] in the Department of Labor with the charge of protecting workers from occupational hazards), there has been a steady increase in the scientific information relating exposure to asbestos with both nonmalignant and malignant effects which occur primarily, but not exclusively, in the chest. A causal link between exposure to asbestos and asbestosis with lung cancer was suspected in the 1930s and 1940s and was established in the mid-1950s.[2] The relationship between exposure to asbestos dust and the development of malignant mesothelioma was demonstrated in 1960.[3] Regulatory efforts in association with industrial action to reduce airborne dust levels paralleled the accumulation of this increasing data base in those decades. An attempt to promulgate "safe" levels of exposure in the workplace began in the 1930s in the United States, the United Kingdom, and Europe. While undoubtedly well-intentioned, the ultimate failure of these past efforts (by both government and industry) to prevent the occupational diseases resulting from exposure to asbestos is, of course, apparent by the continuing occurrences of these adverse health effects; however, it must be recognized that because all of these asbestos-related health effects have latency periods of generally 15 or more years, the burden of these diseases now being diagnosed reflect exposures of the 1960s, 1950s, and before.

Early recognition of the consequences of exposure to asbestos centered on the mining and milling of this mineral and its use in the manufacture of asbestos-containing products. Awareness of the hazard in the use of these products (*eg*, insulators, shipyard workers[4]) came later, the risk of using end products not being widely recognized until the 1960s.[4] Much of the present attention directed toward economic and other social consequences of this exposure indeed relates to the late recognition of the risks associated with the use of asbestos products. This stems in part from the realization that the numbers of exposed workers who have come in contact with asbestos-containing products, both in shipyards and in the course of on-land insulating operations, are substantially greater than in the other workplace opportunities for exposure to asbestos indicated previously.

Past Influence of Science on Decisions

There has been inconstant reliance on scientific knowledge regarding the asbestos-related diseases in the formulation of public policy. Since the recognition of asbestos-related diseases in the 1930s, decision-

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making on public policy has frequently been at best obscurely related to the existing biomedical scientific data base. This disparity has probably increased in recent years, and at times, scientific considerations have been largely ignored in the process of formulating policy.

Society must address two outcomes in regard to the asbestos-related health effects: (1) how to deal equitably with existing disease produced by exposure to asbestos, and (2) what safeguards should be implemented to prevent the continuing occurrence of asbestos-associated health effects. We are dealing with neither of these in a reasonable, efficient, or valid manner at present. In regard to existing disease in the United States, we have relied on inequitable, largely state-structured schemes for compensation and, more recently, on the judicial process, the latter primarily for workers exposed to product dust in application operations (product liability or tort cases which now number in the many thousands). The prevention of disease is primarily, but not exclusively, the responsibility of federal regulatory agencies with the cooperation of both segments of industry, *ie*, management and labor. These efforts must be directed toward establishing levels of exposure sufficiently low in order that they not constitute an unreasonable risk for the development of these diseases.

Clearly, achievement of these aims depends primarily upon scientific knowledge. In the assessment and compensation of asbestos-related disease, medical criteria for establishing its presence and severity is a clear, but by no means easily attainable, objective. Determination that a disease is caused by exposure to asbestos may be reasonably simple with a valid diagnosis of mesothelioma (a rare tumor most often associated with exposure to asbestos) but far more difficult when there is a substantial background of the condition in the general population, such as lung cancer, most often due to other causal factors. In the latter circumstances, science may ultimately provide the basis for apportioning causation (*eg*, smoking and occupational dust exposure) based on appropriate population-based epidemiologic studies, but currently, our scientific data base does not allow us to accomplish this with any certainty in any individual case. Since neither all exposed individuals nor all of those who have instituted a claim are, in fact, injured (these are dose-related consequences), it seems unlikely that, particularly with limited economic resources, society will opt for a policy which leads to the provision of economic benefit for all of the millions of workers who have at some time been exposed to asbestos dust, however, the state of our knowledge does provide us with a sufficient basis to make reasonable judgments concerning the ascertainment and probable major contribution to the diseases which have been associated with exposure to asbestos.

The prevention of occupational pulmonary diseases, which result from the inhalation of airborne injurious agents such as asbestos, depends upon the development of dose-response relationships in studies of working populations where estimates of lifetime exposure can be based on measurement of airborne concentrations of the material of interest. In fact, dose-response relationships have been generated through epidemiologic studies of workers exposed to asbestos in mining and milling, as well as in various manufacturing operations. Because of the difficulty in estimating individual exposures in populations who have been engaged in the use of end products, these exposure-disease relationships are not available. Despite the limitations of our data base regarding dose-response relationships, discussed further subsequently, more is known concerning these relations for exposure to asbestos than for any other hazardous occupational inhalant. Those with decision-making responsibilities should use this information to assure that acceptable reduction of risk will be associated with current and future occupational levels of exposure to asbestos.

During the past decade, all branches of government have been actively involved in the consideration of asbestos-associated disease. The legislative branch, by promulgating OSHA, first placed overall responsibility for occupational safety and health squarely under federal jurisdiction. The first health standard was promulgated by OSHA in 1972, setting the permissible limit of exposure for asbestos dust in the workplace. Perhaps the most visible federal governmental activities during the 1970s have been by the regulatory and other agencies in the executive branch. In addition to OSHA, these include the Environmental Protection Agency, the Consumer Products Safety Commission, the Food and Drug Administration, and others with no or limited regulatory power, such as the National Institute of Occupational Safety and Health, the National Cancer Institute, and the National Institute for Environmental Health Sciences. In the last several years, the third branch of government, the judicial, has become increasingly visible (some would say inundated) in dealing with asbestos-related disease. In addition to decisions on compensation in state boards and courts, there has been an exponential increase in the number of product liability, or so-called "third-party," suits filed and, to a far lesser extent, heard in the federal courts. Issues involve primarily the claim of asbestos-associated disease in users of asbestos-containing products and, specifically, when or under what circumstances the manufacturer had the duty to warn of potential hazards to these users. The level of involvement of the legal profession in this litigation is staggering, and the economic and other social consequences can be readily adduced.

Compensation of Existing Disease

In examining current approaches to the compensation and prevention of asbestos-associated diseases, particular emphasis is placed on the utilization (or lack of it) of the biomedical scientific data base in accomplishing these objectives. It should be emphasized that, as with many other occupationally induced disorders, the asbestos-associated diseases are best dealt with by prevention through control of exposures to airborne concentrations of asbestos dust. In addition to the human costs of these diseases, no scheme of compensation can provide adequate disincentive for industry which would lead to the prevention of these conditions, this is due in large part to the long period of latency between relevant exposure and clinical manifestations of these diseases; however, recognizing that past exposures have produced these injuries, society must be committed to the provision of just resolution of claims by injured workers. Disease-specific approaches to federal legislation on compensation are not optimal in reaching the objective of equitable and timely awards for all workers who develop diseases as a consequence of their employment. The inadequacies of the legislation on black lung have received considerable attention and have been the subject of periodic controversy, often focusing on questions of cost effectiveness and equity. Currently, in the US Congress, legislation dealing with asbestos-associated diseases is being debated, undoubtedly related to overwhelming public and economic pressure. In recent months the opinion has often been expressed that the present system is strikingly inadequate in meeting the needs of individuals who have developed asbestos-associated diseases.

In this context, tort litigation has been shown to be inequitable, costly, and markedly unjust in the distribution of resources, a high proportion of awards going to individuals in whom evidence of disease cannot be demonstrated or toward legal fees. Indeed, one of the main deficiencies in this approach is that it distorts medical evidence, calling, for instance, for "proof" when, in fact, medical diagnosis depends on reasonable judgments as to the best available explanation, given the facts about a case. Other deficiencies are the failure to rely on objective medical evidence in individual cases, as in mass settlements, and the fact that in the assessment of individual cases, medical experts may be chosen not for their expertise and objectivity, but because their testimony will support one side or the other, however, the motivational basis for differing medical opinions need not necessarily be suspect. Given the nature of medical diagnosis, the facts about a case are frequently open to more than one interpretation and uncertainty, a concept with which the legal system deals poorly. One must be mindful of the projections of the future burden of such litigation affecting all segments of society. Similarly, as is widely recognized, the state systems of compensation have generally dealt poorly with asbestos and other workplace-associated diseases, and ideally a uniform federal system of compensation for occupational pulmonary disease should be developed, much as it is in Europe and the United Kingdom.

The known asbestos-associated diseases have all been shown to be dose-related, and one must be persuaded by the evidence that there are differences in biologic activity of asbestos related to fiber type and specific industrial process. It seems reasonable to suggest that the scientific data base can and should be used in the decision-making process as a means of assessing the likelihoood that a given disease is due to the exposure, particularly for conditions which also occur in the general population in the absence of exposure to asbestos. While one readily recognizes that many of the decisions made in the clinical setting concerning individual cases lack precision, informed medical opinions most often lead to reasonable conclusions.

An equitable and workable system of compensating the injured worker who has been exposed to asbestos rests upon certain elements. In the individual case the major medical issues to be decided are diagnosis, causation, and impairment. The process of reaching a diagnosis depends upon sound medical practice regardless of the underlying cause of disease. Determining the most likely cause will usually depend on assessing the levels of exposure to known causative agents; this will be based on epidemiologic studies of occupationally exposed populations. The assessment of impairment due to asbestos-induced fibrotic disease will depend primarily on evaluation of disturbance in pulmonary function. Ultimately, guidelines must be established to facilitate this decision-making process, and these must be the subject of continuing review in light of emerging scientific evidence. The structure of a workable system could include a national panel of experts to develop and periodically modify guidelines and criteria for the adjudication of claims for asbestos-associated diseases, and regional panels, which would include health professionals knowledgeable in occupational pulmonary diseases, which would be responsible for the case-by-case determinations. A mechanism must be developed to make possible an appeal of the medical decisions rendered by the regional panel. The following illustrates how current knowledge can be used in the adjudication of compensation for workers claiming asbestos-associated diseases.

Asbestosis

Asbestosis is a pneumoconiosis defined as diffuse

fibrosis of the lungs caused by exposure to asbestos. Its features include rales (crackles), breathlessness, finger clubbing, pulmonary functional abnormalities (usually reduced volumes and impaired gas exchange), radiographic changes (irregular and linear opacities), and histopathologic demonstration of fibrosis with tissue fiber identification. The first four of these are nonspecific: changes on the x-ray film and pathologic tissue examination have increasing, but by no means absolute, specificity. Individually, these are not sufficient to make a diagnosis; in combination the diagnosis depends upon weighing probabilities and assessing the total evidence, an important component of which is estimation of past exposure. In practice the diagnosis is usually established on radiographic evidence and evidence of exposure. An expert committee of the College of American Pathologists and the National Institute for Occupational Safety and Health has recently developed guidelines for the pathologic diagnosis of asbestos-associated disease.[5] When pulmonary tissue is available for histopathologic examination, a diagnosis of asbestosis depends minimally upon the demonstration of discrete foci of fibrosis in the walls of respiratory bronchioles, associated with accumulations of asbestos bodies (light microscopy). Additional findings are diffuse interstitial pneumonia and fibrosis. Neither fibrosis nor asbestos bodies alone are sufficient for the histopathologic diagnosis of asbestosis.

Nonmalignant Pleural Effects

These include pleural effusions, focal hyaline thickening (plaques), and diffuse pleural fibrosis. Plaques indicate exposure, not disease, because they do not cause symptoms or functional impairment. Attributability is assessed on the basis of the history of exposure and the absence of other causal factors. Careful examination of the scientific evidence leads to the conclusion that there are no definitive data which demonstrate that the presence of benign pleural effects *per se* represents an independent predictor (beyond the effect of exposure) of increased risk for asbestos-associated malignant effects, including lung cancer and mesothelioma. It should also be recognized that the radiographic ascertainment of pleural plaques is fortuitous, and surgical or postmortem examination of the open chest of occupationally exposed workers will demonstrate such focal pleural changes in a very high proportion of such individuals. Finally, the term, "pleural asbestosis," has no validity, and its use should be abandoned.

Lung Cancer

In past studies of populations with generally heavy occupational exposure to asbestos where follow-up has been adequate, increased risk for lung cancer has been demonstrated. This increased relative risk occurs in both smokers and nonsmokers, although the important interaction between exposure to asbestos and smoking is the reason that very few cases of lung cancer occur in nonsmoking asbestos-exposed workers. No specific histologic type of this malignant tumor can be linked to exposure to asbestos. Similarly, the location of the tumor within the lungs cannot be used either to support or exclude causation by asbestos.

The diagnosis of lung cancer in asbestos-exposed individuals is no different than in other clinical settings. In population studies of various occupational groups, evidence of asbestosis has been found when excess risk for lung cancer is demonstrated for comparable levels of exposure. It therefore seems probable that in an occupational setting, when exposures to asbestos have been reduced to levels where evidence of asbestosis is lacking, excess lung cancer is not likely to be detected. In the individual case of lung cancer, the cause cannot be determined precisely. For this reason the judgment must take into account the length, intensity, and character of exposure, evidence of other asbestos-associated diseases, and smoking history.

Malignant Mesothelioma

The primary issue is valid diagnosis. The diagnosis generally can not be established clinically or radiographically or with examination of pleural fluid or with limited tissue for biopsy. Since the potential for misdiagnosis is considerable, adequate histologic specimens should be examined by pathologists experienced and expert in the diagnosis of this tumor (*eg*, mesothelioma panels, which exist on both sides of the Atlantic). When the diagnosis has been established, a history of exposure, even if short, is sufficient for a judgment of causation; however, in essentially all retrospective studies of cases of mesothelioma, a varying proportion of individuals with this malignant neoplasm cannot be demonstrated to have had an occupational or nonoccupational exposure to asbestos. Finally, the limited evidence on dose-response relationships for mesothelioma suggests that the risk for developing this tumor is, as with the other asbestos-associated conditions, dose-related.[6] Smoking does not affect the risk for mesothelioma.

Other Health Issues

As part of the process of determining compensation, assessment of the extent of impairment (which leads to a determination of disability) follows the establishment of an asbestos-associated disease. In cases of extensive asbestosis or extensive pleural fibrosis, reference should be made to recently published criteria for

impairment in restrictive disorders.[7] Primary use is made of tests of pulmonary function, and exercise performance testing is used when necessary.

Substantial controversy exists concerning the possible contribution of exposure to asbestos to the prevalent chronic airways obstruction in the population. The chronic obstructive pulmonary diseases are not the result of a single cause; smoking is by far the most prominent factor in the development of these conditions (chronic bronchitis and emphysema); however, emerging evidence also suggests a contribution from allergic factors, a variety of occupational exposures, infections in childhood, and perhaps general environmental air pollution. How then can we summarize the most reasonable current position on the relationship between exposure to asbestos and chronic obstructive pulmonary disease? While a contribution of exposure to asbestos to both chronic bronchitis and chronic airways obstruction (these do not invariably coexist) has been suggested on the basis of limited evidence,[8,9] these data relate mainly to effects on the small airways early in the course of pulmonary damage, and clinically important chronic airways obstruction is not likely to be primarily the result of this exposure. Typically, a middle-aged smoker previously exposed to asbestos has pleural plaques or minimal evidence of pulmonary fibrosis (asbestosis) (or both) and moderate or severe chronic airways obstruction. His functional impairment is probably attributable to smoking, and it is unlikely that exposure to asbestos measurably contributed to it.

In some studies of mortality in asbestos-exposed populations, excess gastrointestinal cancer and isolated instances of other malignant neoplasms have been demonstrated.[10] These have often not been shown to be dose-related, and in other studies where the risk of lung cancer has been substantially increased, no excess gastrointestinal or other cancer has evolved.[11] The inconstancy of the results of these studies makes it prudent to withhold final judgment concerning the role of asbestos in increasing the risk for these malignant neoplasms. This uncertainty represents the present consensus of investigators in this field.

Finally, it should be emphasized that the scientific evidence is fully concordant in the finding of substantial "latency" or lag time in the development of the asbestos-related health effects. While the period of latency is variable within diseases and between them, perhaps ranging from 15 to 40 or more years, the implications for the societal burden of these diseases are clear. With dust levels generally declining since the 1950s and more markedly since the 1960s, we are likely to soon reach a plateau, followed by a decline in the incidence of the asbestos-associated diseases; however, the risk extends well beyond the termination of exposure, and the period of latency for the risk of lung cancer is probably not influenced by cumulative exposure-dose.

In recent years, it has been popular to "project" the burden of occupationally induced cancer, particularly that due to exposure to asbestos, in relation to the overall experience of cancer in the population. The most visible document, not published but widely distributed under the imprimatur of various government agencies and their senior officials, has commonly been referred to as the "estimates document" and contained grossly exaggerated (some would say irresponsible) claims regarding the relative contribution of occupational exposures to malignant disease.[12] In a recent review by highly respected British cancer epidemiologists, it was suggested that this document was written for "political rather than scientific purposes."[12(p134)] It is obvious that such distortions can lead to irrational public health policies.

Dose-Response Data and Setting Standards

As previously indicated, the setting of permissible limits of exposure to asbestos, primarily a workplace issue, depends upon the establishment of dose-response relationships, with subsequent determination of an acceptable level of risk. For the asbestos-associated diseases, this task would be immeasurably simpler if dose-response relationships were similar for all industrial processes, all fiber types, and all asbestos-induced diseases. Unfortunately, the evidence indicates substantial variability of relationships within these categories.

It has already been suggested that the cumulative dose of exposure to asbestos which will produce early indicators of asbestosis may very well be lower than that which has been shown to result in excess risk of lung cancer.[16-18] It is likely that even lower, and almost certainly short, exposures to asbestos dust will lead to cases of mesothelioma, which fortunately remain rare. In general, from a regulatory standpoint, a standard can reasonably be based on epidemiologic evidence on either asbestosis or lung cancer, assuming that the population has been followed adequately and taking into account the period of latency. Contrary to some assertions, evidence based on asbestosis will probably generate a more restrictive standard than that based on lung cancer. It should be comforting to know that there are recent studies of mortality from exposure to asbestos in which the population[17] or subgroups[14] show no excess risk of lung cancer. It does not seem likely that additional dose-response data for mesothelioma will make it possible to set limits on exposure which can predictably be expected to prevent the occurrence of this effect.

Variability in the risk of lung cancer related to the segment of the industry studied is substantial. Some

studies of occupationally exposed populations cannot be used for comparing dose-response relationships because appropriate individual estimates of exposure over a working lifetime are not possible. This problem has been particularly notable in studies of insulators and other workers exposed to the dust of product use. Where exposure and effect have been adequately measured or estimated, it is clear that there is a gradient of risk of lung cancer, the lowest for a given dose being found in miners and millers[10] and the highest in asbestos textile manufacturing workers,[11] with intermediate slopes among others engaged in the manufacture of asbestos products, such as asbestos cement building materials.[14] On the basis of limited information on exposure, one may infer that a steep dose-response curve also exists for asbestos-exposed insulators. The basis for these differing risks by process, while not fully understood, has a number of biologically plausible hypotheses, some of which are based on studies in animals. Varying physical dimensions of asbestos fibers are likely to be encountered in these different segments of the industry. Specifically, the diameter or length of the fiber (or both) have pathogenetic importance, the thinner, longer fibers being most likely to possess enhanced potency in producing both the fibrogenic[18] and malignant effects.[19] Limited data suggest that retained fibers in the lungs of asbestos textile workers have greater length than fibers in other asbestos manufacturing workers.[20] Variations in biologic potency in specific portions of the industry have obvious regulatory implications, and the setting of different standards for discrete portions of an industry or even a plant is not without precedent in the United States.[21]

For many years, evidence has been evolving which indicates substantial differences in the risks of adverse health effects for various types of asbestos fibers. Limited data suggest that for equivalent doses, the risks of asbestosis and lung cancer are increased for exposures to amphibole fiber, particularly crocidolite, in contrast to populations exposed only to chrysotile fiber,[14,22] however, the weight of the evidence on differential risks in relation to the type of fiber relates to mesothelioma. In mining (chrysotile,[16] in contrast to crocidolite)[23] and manufacturing (friction materials,[17] textiles,[11,24] and gas masks[25,26]), the experience with mesothelioma has been strikingly less favorable in those populations exposed to crocidolite than chrysotile. Most European and UK standards[27] have taken this differential risk into account based on these recent and increasingly convincing research findings. In the United States, we have to date not yet accepted this evidence. Clearly, this is an important example of nonconcordance between this scientific data base and decisions on public policy.

The assessment of risk for potential hazards of exposure to asbestos in schools and other public buildings, or as the result of contact with consumer products containing asbestos, has received recent attention, both in regulatory and judicial arenas. Such assessment generally involves low-dose extrapolation from existing dose-response relationships obtained in occupational settings. It will be obvious from earlier comments that the selection of dose-response relationships to be used for this assessment will importantly influence the estimates of risk at doses which are orders of magnitude lower than in the workplace. The shape of the dose-response curve at such low doses is generally assumed to be linear through the origin, based on two considerations. First, it is considered that the existing data do not allow the conclusion that the shape is nonlinear, which is, of course, not proof that it is indeed linear. Secondly, the assumption of linearity is widely considered to be consistent with prudent policy on public health, since it is likely to err on the side of overestimating the risk. It will, of course, become obvious that low-dose extrapolation assuming linearity in mathematic modelling will result in an anticipated excess of lung cancers for any dose, no matter how small.[28] When such calculations are made for lifetime risk in a large population (*eg*, the United States), any numbers will seem to some to be unacceptable, and only by putting such estimates into perspective by comparison with everyday commonly accepted risks will rational policy emerge.

Additional Economic Issues

The causes and implications of the recent chapter-11 bankruptcy filing of the largest miner and manufacturer of asbestos and asbestos-containing products in North America is understandably receiving wide review. The public statements of the company indicate that its decision was based on the potential economic loss created by present and future product-liability suits, and the data presented by them were generated by epidemiologic consultants using available scientific information.[29] The appropriateness of the selection of data for this purpose, as well as the analysis and interpretation of these data, in addition to future proposals made to the bankruptcy court, must be examined carefully. There seems to be a surfeit of discussion regarding a possible role for government in compensating those with claims and a curious lack of interest in the question of how many claimants are really ill and which among these have asbestos-attributable diseases. Current proposals for legislation on federal compensation for asbestos-associated diseases, their effect on the litigation, and how it has been influenced by the bankruptcy proceedings are clearly areas in which the interface between scientific data and social policy is central.

Another issue has recently arisen in regard to the

distribution of liability among primary and excess coverage insurers of companies faced with litigation on asbestos-associated disease. The economic implications are obviously enormous, and a number of potentially scientifically answerable issues have been raised in an effort to resolve these questions of financial liability. Among these issues is determining which carriers are liable: those whose policies were active throughout the period of exposure of the claimant (the exposure theory), those whose policy covered only the period when the disease became manifest or was diagnosed (manifestation theory), or all carriers who had active policies at any time during exposure or subsequently? The question has been raised whether a major study of the asbestos industry by a large insurance company in the 1930s was adequate in pointing out health hazards;[30] those who have believed that such responsibilities were not adequately met have made this carrier a codefendant. There are instances in which it is important to determine how long, prior to the establishment of a diagnosis of an asbestos-attributable disease, might that diagnosis have been made in order to ascertain the relevant insurance coverage. The probability of progression of asbestosis, given estimates of exposure and level of disease, will often be taken into account in reaching a legal settlement or judgment; these questions can be answered by carefully conducted longitudinal studies of asbestos-exposed and injured workers.[31]

Implications for Future Policy Making

Inevitably, what we have learned concerning the interaction of science and decision-making on public policy in the area of asbestos-associated diseases should have broad implications in how our society and its scientific community can best deal with other occupational and public health problems. It is anticipated that examination of the issues outlined will point to alternative approaches, perhaps in part as practiced in other countries, such as the United Kingdom, Europe, and Canada. Strategies for other potential occupational health problems utilizing nonadversarial methods should be examined, for example, recent studies and timely reporting of results of possible health effects in the man-made mineral fiber industry (fibrous glass and other synthetic fibrous minerals). Consideration of the wider issue of whether responsible and informed scientific input is sufficiently broad and varied in US decision-making processes leads to the present conclusion that it is not. Specific suggestions for an approach to these problems in the future, which will lead to a responsible and effective balance between the health needs of segments of our population and more general societal needs and considerations, many of them economic, should be forthcoming.

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