PLAINTIFF'S EXHIBIT
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LOW-LEVEL ASBESTOS EXPOSURE

What are the risks of low-level asbestos exposure? Perhaps it's time policy makers investigated the evidence before committing more time and money to removal efforts.

Introduction By William E. Bailey

The core issue in any discussion of asbestos abatement is its necessity. The Environmental Protection Agency's (EPA) regulations and standards are driving the current flurry of activity among property owners and managers (and their lawyers and bankers) to determine which of their buildings fall within the regulations and must comply.

Larry Cetrulo, a partner in the Boston firm of Burns and Levinson, contends that medical, scientific and epidemiological data do not support the regulations and that we are hell bent to spend billions of dollars removing asbestos in public and private buildings where the actual risk factors for occupants and visitors is so negligible as to be virtually zero. Cetrulo bases his arguments on recent studies comparing the known data on the carcinogenicity of various asbestos fiber types in occupational settings and then attempting to extrapolate backwards the potential risk factor for much lower level exposures.

Cetrulo has been active in asbestos litigation since shortly after he joined Burns and Levinson in 1976. In fact, I believe it was as a result of my suggestion to him that he explore this fast-growing area of legal expertise. For the past several years, he has been the Asbestos Claims Facility's coordinating counsel in Boston. More recently, he has been drawn into the property damage claims area and the new wave of litigation that poses a major threat to asbestos product manufacturers at a time when many are exhausting their available insurance coverages in bodily injury cases.

Cetrulo's article will encourage some and infuriate others. He has struck a sensitive nerve with his arguments and his conclusions. AI will continue to provide a forum for all sides of all issues, including "Is this all much ado about nothing?"

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For example, if a contractor has completed 50 percent of a project, but has been paid 75 percent of the contract price, there would likely be a deficiency in the contract balance if the contractor defaults. However, if the owner has paid the appropriate amount for the performance of the work (50 percent of the contract price, in this case), it's less likely there will be a deficiency. In determining whether a deficiency exists, the owner has to take into account the cost of the bond premium and the added cost charged by the contractor for having posted collateral with the surety company.

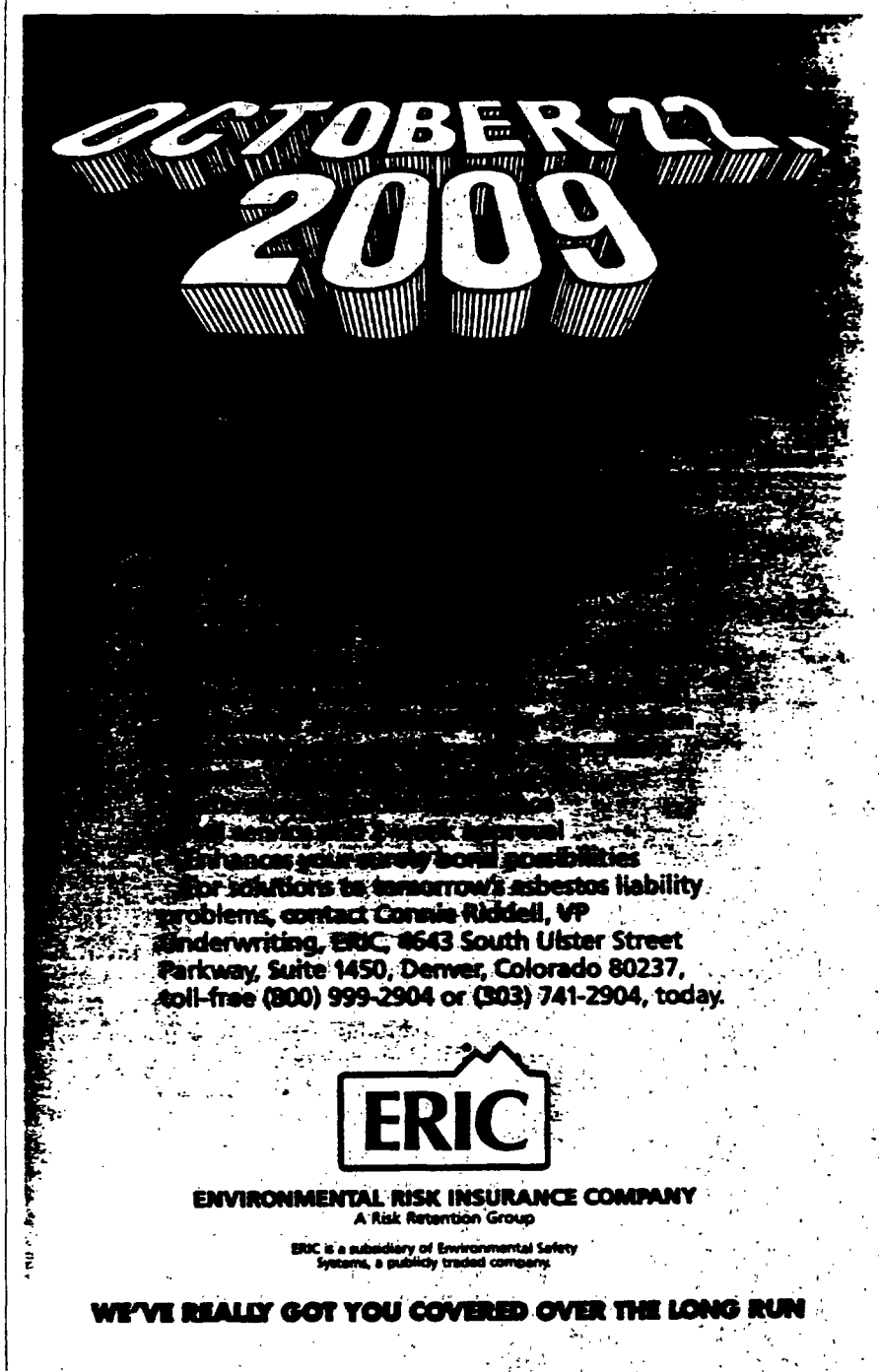
With respect to material and labor payment bonds, the principles of analysis are similar. If the contractor has performed work but has not paid his laborers or material suppliers, a deficiency could exist which would require response by the surety company. However, there are methods of protecting against such problems. In many states, if a project owner obtains a sworn statement from the contractor with each pay request that all bills for labor and materials have been paid, he may be protected against paying the contractor and then later having to pay for materials and labor for which the contractor has not made payment. The owner could also require the contractor and any suppliers to execute lien waivers in return for progress payments. Since materials do not comprise a large part of an asbestos abatement project contract price, the likelihood of unpaid material claims is reduced. Also, laborers will generally not work for extended periods without pay.

There is another factor involved with asbestos abatement projects that can assist in the analysis of whether bonds are required by an owner or consultant. While many general construction projects are of long duration, asbestos abatement projects are typically of a shorter duration, meaning that performance will be completed more quickly. This can have several important effects. First, the short duration of the project means that a contractor has less chance of "getting into trouble" financially on the project. Furthermore, the short duration of the project means that there is less time for price escalations in labor and/or materials. Often, the increase in the cost to complete a project upon a contractor's defaulting is attributable to price increases that have occurred within the prolonged period of time that elapsed from the original signing of the contract and beginning of performance of the work to the date of default.

In addition, it must be recognized that many asbestos abatement projects are performed on a time and materials basis. Since payment is only made for work that has been performed and materials that have been purchased and placed upon the job, the utility of a bond in such circumstances is questionable. Where there is no fixed contract price other than the cost of labor and materials per unit of work performed, the penal sum of the bond cannot be readily determined. It would be difficult in many circumstances to obtain a bond on such a project. Although the unit price of work can increase, the loss to the owner cannot be calculated, since the number of units has not been agreed upon.

Traditions die slowly. The requirement of performance and payment

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By Lawrence G. Cetrulo, John B. Savoca and Randolph L. Smith

The question of whether low-level asbestos exposure, such as the exposure encountered by occupants of asbestos-containing buildings, presents a material risk of causing asbestos-related disease has become a major legal and public policy issue. The view that there is no known threshold level for asbestos exposure below which disease will not occur has, to date, dominated public debate and resulted in a wave of litigation, regulations and fear. Concern over the lack of an informed public policy in this area is now being expressed by the business community ("Scary Asbestos," *The Wall Street Journal*, Oct. 12, 1987, and "Why Throw Money at Asbestos?" *Fortune*, June 6, 1988).

Central to the current debate are questions of whether all asbestos fiber types are capable of causing disease at low exposure levels and whether risk of disease at low exposure levels can be extrapolated validly from epidemiologic data for occupationally exposed populations.

The genesis of the debate is found in the 1985 EPA report "Airborne Asbestos Health Assessment Update," by William J. Nicholson, Ph.D. The Update contained two critical findings including, "Animal studies confirm the human epidemiological results and indicate that all major asbestos varieties produce lung cancer and mesothelioma, with only limited differences in carcinogenic potency," and "Extrapolations of risks of asbestos cancers for occupational circumstances can be made, although numerical estimates in a specific exposure circumstance have a large (approximately tenfold) uncertainty."[1]

Relying in part on the Update, the EPA states in the preamble to its final Asbestos Hazard Emergency Act (AHERA) rule that its analysis of risk "...shows that asbestos in schools could present a risk of concern and that the measures required by this rule are necessary to protect the public and the environment."[2] The EPA also stated, "With respect to the potency of asbestos, EPA has decided that for the purposes of this rule, there is no need to resolve the divergence of opinion."[3]

The EPA's position on risk assessment has recently been summarized in the "EPA Study of Asbestos-Containing Materials in Public Buildings—A Report to Congress," US EPA (February 1988):

"No safe threshold has been established for asbestos. Effects at low levels of non-occupational exposure have been estimated by extrapolation from higher levels although the validity of this approach has not been empirically demonstrated."[4]

The EPA's efforts to protect the public and the environment have been understandably focused on public health issues rather than on the complex scientific issues involved. However, since the Asbestos Health Update was released, a great deal of literature has been published on the medical, scientific and public policy issues involved. It is important to identify and highlight some of this recent scientific and medical information to hopefully encourage further study and debate on these crucial issues.

"It may be impossible to prove conclusively that at low levels asbestos exposure does not create any increased risk of cancer."

SIGNIFICANCE OF FIBER SIZE AND TYPE

Prior to the introduction of stringent safety standards for occupational exposure to asbestos dust, asbestos workers in some industries and job categories were at times exposed to very high levels of airborne asbestos fibers. It is now known that this type of high-level occupational exposure can cause asbestosis, lung cancer (in smokers) and mesothelioma. It is widely accepted, however, that low-level exposure to asbestos, such as building occupant exposure, does not cause asbestosis. The current issue of scientific and legal debate is whether such low-level asbestos exposure presents a material risk of producing lung cancer or mesothelioma. There is now a growing body of medical literature that suggests that different asbestos fiber sizes and fiber types have vastly different potentials to cause lung cancer or mesothelioma.

Numerous studies have been undertaken in an effort to determine the relative carcinogenicity of different asbestos fiber sizes. There is now scientific evidence to suggest that long, thin asbestos fibers present the greatest risk of producing cancer, with shorter fibers being less, or not at all, carcinogenic.

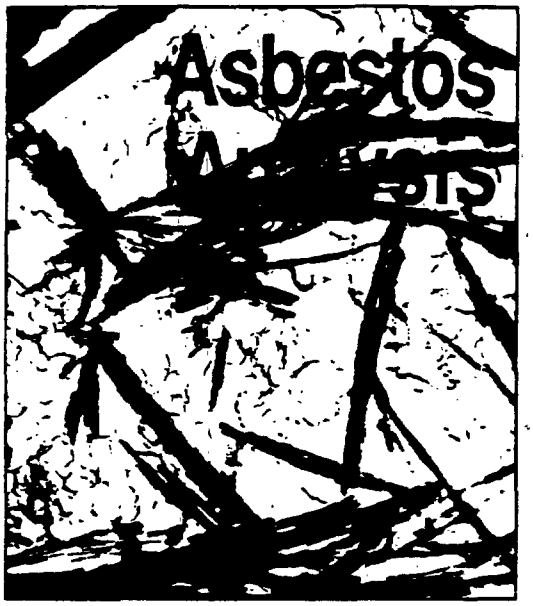
A recent editorial by J.M.G Davis, Ph.D., and Dr. J. Corbett McDonald, (*British Journal of Industrial Medicine*, August 1988) reviews the scientific literature and suggests that the most dangerous fibers are those with a length greater than eight microns and a diameter of less than .25 microns. Davis reports that fibers less than five microns in length may be innocuous in lung tissue. This finding is significant, since the great majority of fibers normally found in ambient air, both indoors and outdoors, are less than five microns in length.

Fiber type is also thought by some experts to be a significant factor in the carcinogenicity of asbestos. It has been suggested that the propensity of a particular fiber type to be inhaled and retained in the lung is related to the ability of the fiber to produce cancer. The two major asbestos fiber types, serpentine and amphibole, may differ markedly in their propensity to be inhaled and retained in the lung.

Serpentine asbestos, such as chrysotile, is characterized by long, silky, curly fibers. The aerodynamic properties of these fibers may prevent them from being inhaled deep into the periphery of the lung. Furthermore, the failure of chrysotile to persist in the lung may be due to its chemical properties which allow the fibers to be readily broken down into short fibrils and removed from the lung by the bronchial cilia or by enzymatic digestion by the macrophages.

In contrast, amphibole asbestos,

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such as crocidolite, is characterized by straight, rigid and pointy fibers. The aerodynamic properties of these fibers permit them to be inhaled deep into the periphery of the lung. The rigid pointy structure of amphibole fibers may enable them to penetrate the lung tissue and then pass to the pleura, or lining, of the lung. Finally, the chemical properties of the amphiboles may enable the fibers to withstand decomposition and be retained in the lung for long periods of time.

"It has been suggested that the propensity of a particular fiber type to be inhaled and retained in the lung is related to the ability of the fiber to produce cancer."

There is now growing evidence that pure chrysotile does not cause mesothelioma unless at extremely high levels of exposure.[5] This evidence includes studies that have found a very low incidence of mesothelioma in workers occupationally exposed only to chrysotile. The results of tissue analysis have shown that the lungs of people who have died of mesothelioma contain no more chrysotile than the lungs of people who have died of non-asbestos associated diseases.

The data, with respect to fiber type, is significant since the vast majority of asbestos used in buildings in the United States has been chrysotile asbestos. Concern about low-level exposure to asbestos in buildings may be misplaced if low-level exposure to chrysotile does not cause mesothelioma and the asbestos fibers normally found in the ambient air in buildings are too short to cause this disease.

RISK ASSESSMENT

Presently, the risk of contracting lung cancer or mesothelioma associated with low-level exposure to asbestos can only be assessed indirectly, since low-level exposure is far below the exposure levels at which it has been possible to detect such risks in occupational settings. Attempts have been made to estimate the risks associated with low-level, non-occupational exposures by extrapolating epidemiological data from studies of high-level occupational exposures. This technique of extrapolation is fraught with difficulties and the validity of theoretical low-level asbestos exposure risk assessments derived from linear extrapolation is a matter of dispute.

Extrapolation is a two-step process. First, a dose/response relationship is estimated based upon existing epidemiological data derived from high-level exposure groups. A dose-response relationship can be established by demonstrating that increasingly high levels of asbestos exposure are statistically associated with an increased incidence of cancer. Second, using the dose/response relationship estimated from high-level exposures, the risk for lower exposures is established by linear extrapolation down to the origin. In theory, these extrapolations should be supported by existing scientific and medical evidence.

There are two arguments against the use of linear extrapolation for determining low-level exposure risk. First, there may be deficiencies in the exposure data used to derive a dose/response relationship for asbestos at high exposure levels. Second, and perhaps more importantly, it is argued that there is no generally accepted theory to justify extrapolation of any dose/response relationship established at high exposure levels back to low exposure levels.

These arguments have been used to cast doubt on low exposure level risk assessments derived from extrapolation. In any event, to the extent that any credibility can be given to low-level risk assessments derived from extrapolation, some argue that based upon the most recent data, such risks are extraordinarily low. For example, a recent report by Sir Richard Doll and University of London Professor Julian Peto estimates that a theoretical risk of only one excess death from environmental asbestos exposure occurs per year in the entire United Kingdom.[6 7 8]

The concept of an exposure "threshold" is very important in low-level exposure risk assessment. Linear extrapolation of low-level risk assumes that there is no safe threshold of asbestos exposure below which there will not be an associated increased risk of cancer. Scientific data has recently been published that suggests there may be a safe threshold for asbestos exposure.

With respect to lung cancer, it has long been argued that excess lung cancer associated with asbestos exposure occurs only when the level of asbestos exposure is high enough to produce asbestosis. While this view is still a matter of controversy among experts, a recent study supports an asbestos exposure threshold for lung cancer.[9]

Although mesothelioma is generally believed to be more likely than lung cancer to be associated with relatively low levels of asbestos exposure, there is some evidence of a threshold of asbestos exposure below which there will not be an increased incidence of mesothelioma. For example, Dr. Andrew Churg has reported that examination of lung tissues of people from the general population and of residents of asbestos mining towns in Canada reveal the presence of significant quantities of asbestos fibers without the production of disease.[10]

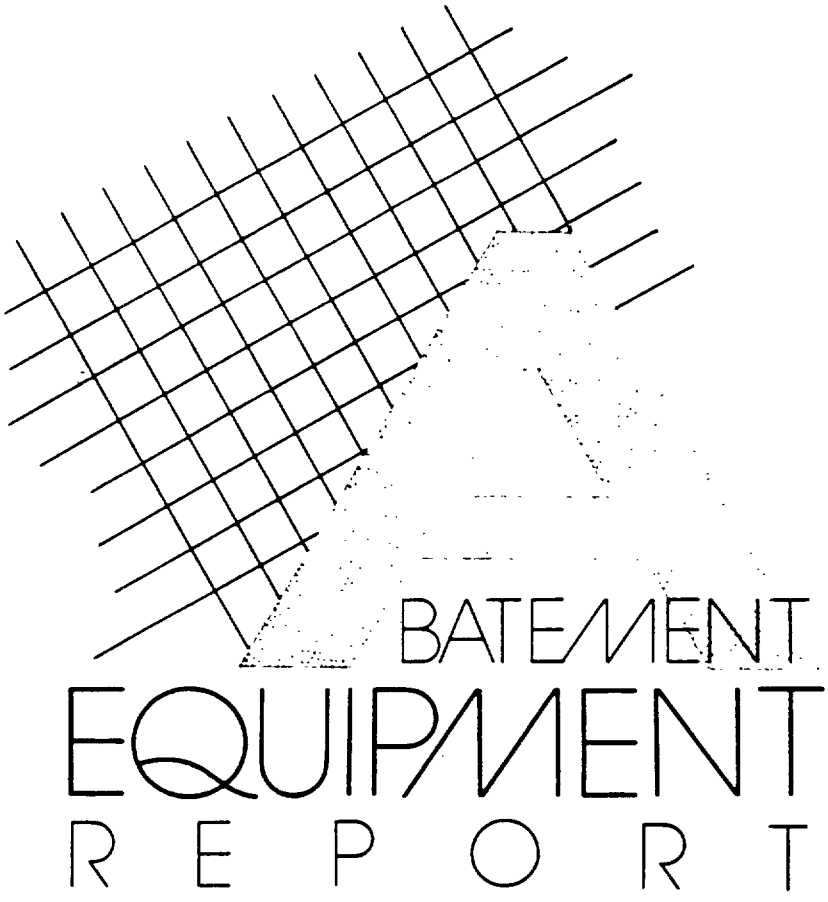
Moreover, a comparison of mesothelioma rates in men and women is revealing on the issue of an exposure threshold for mesothelioma. Although mesothelioma rates have increased in men due to occupational exposure, there has been no corresponding increase in women, suggesting that the background level of mesothelioma has remained constant despite environmental exposure.[11] This finding suggests that there is no linear dose/response relationship for mesothelioma at low-level asbestos exposures and that a "safe" threshold for asbestos exposure may exist.

PUBLIC POLICY

As the focus of public policy regarding asbestos exposure turns from occupational exposure to exposures in schools and commercial buildings, policy decisions should be made with regard to scientific evidence. The EPA's risk analysis for low-level asbestos exposure has led to a wave of legislative and administrative findings and regulations pertaining to asbestos in buildings. It is important that this risk analysis consider the most recent scientific studies concerning exposure

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FOCUS: AIR MONITORING EQUIPMENT, METHODS AND REGULATIONS

By William R. Gates

Air monitoring for asbestos is only as good as the organization of resulting data and the communication of the results to those affected. This important aspect of the air monitoring program is mandated by OSHA 29 CFR 1926.58, and is the most misunderstood and neglected section of the regulation.

This article is the result of my writing many air monitoring manuals for various companies involved in the asbestos abatement industry. A description of the proper use of monitoring equipment is followed by an explanation of proper reporting and recordkeeping.

Knowing the amount of harmful airborne asbestos in a given area is vital when abatement procedures are undertaken. The accepted method for measuring these airborne fibers is to draw contaminated air through a filter that traps the asbestos on its surface. The average fiber concentration is then calculated by dividing the number of trapped fibers by the volume of air pulled through the filter.

THE CASSETTE

The accepted National Institute for Occupational Safety and Health (NIOSH), the research arm of the Occupational Safety and Health Administration, method of measuring the asbestos concentration in air is the NIOSH 7400 method. This requires the use of a 25 millimeter diameter filter housed in a three-piece polypropylene holder or cassette.

The filter is made of cellulose, the same material that forms the cell walls in plants. Asbestos fibers are impacted on and collected from this cellulose filter. To view the fibers in the laboratory, a liquid that dissolves the cellulose material is applied to the filter, then placed under the microscope.

The design of the present-day cassette is the result of many years of development with successive improvements brought about by addressing problems found during field use. The addition of the 2-inch extension cowl to help prevent filter damage in open-face sampling, and the addition of carbon-treated plastic to help ensure even distribution of the fibers are but two of the many improvements. The cowl, though seemingly troublesome, protects the filter from the bumps, moisture and generally hostile environment of an asbestos abatement area. Carbon is added to render the cassette, especially the cowl, conductive so as to eliminate the buildup of static electricity. Otherwise, static charges would attract asbestos fibers and other airborne debris, causing uneven airflow through the cowl and uneven fiber distribution on the filter.

The actual sampling is done "open-faced," meaning that the cassette's end cap is removed, exposing the entire filter surface so that air can be drawn uniformly through it.

Uniform fiber distribution is important because only 100 small areas, called "fields," are usually

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LOW-LEVEL EXPOSURE

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threshold, fiber size and type and low-dose risk assessment.

As is well known in the science of epidemiology, it is often difficult, and sometimes impossible, to prove a negative. It may be impossible to prove conclusively that at low levels asbestos exposure does *not* create *any* increased risk of cancer. However, a failure to conclusively prove an exact threshold level of asbestos exposure below which there is no increased risk of cancer may not constitute a rational basis for public policy decisions with such significant economic impact. In their article in the *British Journal of Industrial Medicine*, Davis and McDonald concluded, "We cannot say, and it may be impossible to prove or disprove that at very low levels of asbestos exposure the cancer risk is zero. . . . Rational decisions will depend on questions of cost, benefit and the appreciation of risk. It will be for society, not science, to decide what priority should be given to the reduction of *unsubstantiated and undetectable risks*, even from such dreaded effects as mesothelioma and lung cancer."[12]

Current scientific and medical evidence suggests that the hazards of low-level asbestos exposure may have been overstated. Rational public policy should take into account the new evidence that short asbestos fibers may not be carcinogenic, that pure chrysotile exposure may not produce mesothelioma and that the risk associated with low-level exposure, if any, may be so low as to be undetectable. ■

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