

Asbestos Exposure Causes Mesothelioma, But Not *This* Asbestos Exposure:

An Amicus Brief to the Michigan Supreme Court

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PLAINTIFF EXHIBIT

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Manufacturers of asbestos brakes, supported by many manufacturing and insurance industry *amicus curie*, requested the Michigan Supreme Court to dismiss testimony of an expert regarding the ability of asbestos dust from brakes to cause mesothelioma as “junk science.” Scientists are concerned with the sweeping and unequivocal claims that any conclusion that asbestos from brakes caused a signature asbestos-related disease in a particular person must be “junk science.” The manufacturers’ sweeping pronouncements are what veer from accepted, reliable mainstream scientific methods and conclusions. This article outlines the evidence supporting the conclusion that asbestos from brakes can and does cause mesothelioma, and describes the defendants’ attempts to fabricate doubt about this conclusion. *Key words:* asbestos; brakes; chrysotile; mechanic; occupation; epidemiology; mesothelioma.

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Companies that made and sold asbestos-containing brakes asked the Supreme Court of Michigan to effectively rule that it is impossible to contract asbestos-related diseases as a result of exposure to asbestos from asbestos brakes.[†] As physicians and scientists, we are concerned about the epidemic of asbestos disease that continues to cause the deaths of thousands of workers each year in the United States. The signers of this paper represent hundreds of years of experience researching, diagnosing, and treating asbestos-related diseases in workers and their families. We have published extensively in this field for more than 30 years and have conducted dozens of epidemiologic and other studies into the issues of asbestos and disease. Many of us have testified before legislative and regulatory bodies regarding asbestos and disease and in court proceedings at the request of individuals suffering from mesothelioma and other asbestos-related diseases.

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We make no claim to know the “correct” answer to disease causation in the specific case under review in the court. Our concern is with the sweeping and unequivocal claim that any conclusion that asbestos from brakes has caused a signature asbestos-related disease in a particular person must be “junk science.” We find that sweeping pronouncement itself is what veers from accepted, reliable mainstream scientific methods and conclusions.

Ample Evidence Supports the Conclusion That Asbestos from Brakes Can and Does Cause Mesothelioma

Chrysotile causes cancer, including mesothelioma. “There is general agreement among scientists and health agencies . . . [e]xposure to any asbestos type (i.e., serpentine [chrysotile] or amphibole) can increase the likelihood of lung cancer, mesothelioma, and nonmalignant lung and pleural disorders.”¹

Many other reviews support this conclusion, such as those from the American Conference of Governmental Industrial Hygienists,² the American Thoracic Society,³ the Environmental Protection Agency,⁴ the International Agency for Research on Cancer,⁵ the National Toxicology Program,⁶ the Occupational Safety and Health Administration,⁷ the Consumer Products Safety Commission (CPSC),⁸ the World Health Organization,⁹⁻¹¹ and the World Trade Organization.¹² This scientific consensus is also reflected in the Consensus Report of the 1997 Helsinki Conference,¹³ and publications from the American Cancer Society¹⁴ and the National Cancer Institute of the National Institutes of Health.¹⁵

Thorough scientific inquiry requires consideration of all available information. Accordingly, in reaching the conclusion that chrysotile asbestos causes mesothelioma, scientists properly consider numerous accepted sources of scientific data, including epidemiologic studies of all varieties, case reports and series of case reports, controlled animal experiments, and toxicologic studies.^{1,16-23}

Asbestos industry arguments to the contrary have not been supported over time. Chrysotile asbestos mining companies and manufacturers have argued for more than 30 years either that their products do not cause disease or that there is insufficient evidence to reach a reliable conclusion. Numerous scientific articles and criticisms have specifically exposed the artificial uncertainty cre-

ated by the proponents of chrysotile asbestos, and their position has been repeatedly and consistently rejected by the mainstream scientific and regulatory communities.^{18-20,24-26}

Like many scientists, we are concerned with the development and expansion of "doubt science."^{27,28} A centerpiece of the "doubt science" model is the assertion that whatever piece of evidence supports the position of the industry in question (or whatever piece of evidence might be as yet undetermined) is *the* critical piece of evidence, to the exclusion of all others. While we acknowledge that industry-sponsored research can and does often provide valuable scientific insight and developments, the efforts of the tobacco and asbestos industries to deny their products cause cancer have become a paradigm for "doubt science."

In this regard, we are cognizant of the fact that the primary articles upon which the asbestos brake manufacturers rely in this matter were paid for by Ford, General Motors, Chrysler and other asbestos brake manufacturers. Publications by Hessel,²⁹ Goodman,³⁰ and Paustenbach^{31,32} were all expressly funded by Ford, General Motors, and Chrysler. Furthermore, the paper by Hessel et al. appeared in a journal funded by the Ford Motor Company and a subsidiary of General Motors. Wong³³ has been reported to have undisclosed origins as an expert witness report for a brake manufacturer.³⁴ Laden acknowledges funding by a law firm that is "national asbestos counsel" for another asbestos brake manufacturer.³⁵

The Scientific Community is in Consensus that Even Brief and Low-level Exposure to Asbestos Can Cause Mesothelioma

The mainstream scientific community has long recognized and continues to recognize today that there is no "safe" level of exposure to asbestos.^{12,13} As noted by NIOSH:

Excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure.³⁶

Attempts to postulate thresholds for exposure have been dismissed as "logical nonsense."³⁷

The lack of a defined "safe" level for exposure to asbestos has been supported by subsequent research. For example, a large French study recently concluded that substantial excess mortality occurs at exposure levels below current regulatory levels.³⁸ A recent study examining the relationship between historical asbestos use and disease rates further supports the conclusion that a linear dose-response relationship exists between exposure to asbestos and disease and that no "safe" level of exposure exists.³⁹

One of the main studies upon which the asbestos brake manufacturers rely⁴⁰ similarly concluded that *all* levels of occupational exposure to asbestos increase the risk of mesothelioma:

Compared to those who never worked or who were never exposed, all levels of probability and intensity [of exposure to asbestos] had an increased significant risk, except subjects with low probability of exposure. For exposure classified as "sure" the OR was 13.2.

Application of this study to the current case under review would result in his placement in the "sure" exposure category, and consequently he would be over 13 times more likely to contract mesothelioma than unexposed individuals. Despite this, the asbestos brake manufacturers assert that the Agudo study proves that no person can ever get mesothelioma from asbestos brakes. That argument is unsound and contrary to the consensus of the scientific community that there is no demonstrable threshold of exposure to asbestos below which adverse health effects do not occur. Accordingly, "an occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated occupationally related" to asbestos exposure.¹³

Mesothelioma Is a Signature Malignancy for Asbestos Exposure

There is no debate that asbestos causes mesothelioma, and that the great majority of mesotheliomas are demonstrably caused by asbestos.⁴¹ Some mesotheliomas are never able to be individually linked to asbestos exposure, and the scientific community has defined these cases as "idiopathic" because information regarding asbestos exposure is unavailable.

However, we know that many individuals do not know that they have been exposed to asbestos.⁴² Many more die before being interviewed regarding potential exposures, forcing researchers to make assumptions about exposure based upon information from next of kin, job titles, or death certificates; these sources often fail to reflect all jobs and exposures.^{43,44} Many epidemiologic studies assess occupational exposure but not para-occupational or environmental exposure, because only occupational information is available from existing records. The fact that a percentage of mesotheliomas are labeled "idiopathic" does not, however, support the conclusion that there are large numbers of spontaneous (i.e., non-asbestos-related) mesotheliomas. To the contrary, a large study of numerous sources of information failed to demonstrate evidence for "spontaneous" mesotheliomas,⁴⁵ and a detailed review of mesothelioma cases in Australia found that over 90% had either a history of exposure or substantial asbestos in lung tissue.⁴²

The asbestos brake manufacturers attempt, without support, to recast the definition of "idiopathic." First,

in an attempt to undercut the indisputable link between asbestos and mesothelioma, they suggest that "idiopathic" mesotheliomas are not caused by asbestos rather than accepting that these are cases where individual exposure has not been identified. Second, they attempt to place mesotheliomas with demonstrable occupational exposures to asbestos—specifically asbestos from brakes—in the "idiopathic" category. There is no scientific support for either position.

Mesothelioma is a signature tumor for asbestos exposure. Individuals with known occupational exposures to asbestos cannot be recast into the "idiopathic" or "unknown exposure" category. When confronted with an individual who has a demonstrated mesothelioma and demonstrated occupational exposure to asbestos, the mainstream scientific community recognizes that the cause of that mesothelioma is the asbestos exposure of the individual even if that exposure was "brief or low-level."

Because Mesothelioma Is a Signature Malignancy with Essentially One Cause—Asbestos—the Scientific Community Has Long Considered Individual Cases of Mesothelioma to Be Sentinel Events

It is not necessary to have an epidemiologic study of a specific occupation to be able to conclude that an individual's exposure to a toxic substance in that occupation can be a cause of disease. To the contrary, as noted by Dr. Lemen,

Specific occupations do not need to be studied nor do epidemiological studies need to be performed to show risk of disease before prevention actions are taken or causal connections concluded. To wait for epidemiology studies of each occupational group is not warranted but has been taken by many in the medico-legal profession as the only way to prove causation by occupation. Such misconceived thinking has been very harmful to the future prevention of asbestos-related diseases.⁴⁶

This is particularly so when examining mesothelioma. Repeated studies have shown that all levels of exposure increase the risk of mesothelioma.^{38,40} Moreover, unlike many other cancers, for which there are multiple, well-documented causal factors, mesothelioma is overwhelmingly caused by asbestos. As noted by one of the studies upon which the asbestos brake manufacturers rely:

Mesothelioma is a rare cancer with one major etiologic exposure, therefore surveillance using each case as a sentinel event might seem more reasonable for this disease than for cancers with multifactorial causation.⁴⁷

In 1983 Rutstein developed a list of sentinel health events (SHE-O) that are occupationally related.⁴⁸

Mesothelioma as a sentinel disease for asbestos exposure was on the initial list of SHE-O, and all subsequent revisions. In fact, the worldwide acceptance of mesothelioma as an asbestos-related cancer began with the case series published by Wagner in 1960.⁴⁹

When examining the question of causation of sentinel diseases such as mesothelioma, the scientific community recognizes that case reports and case series reports are useful and valid tools.

Case series are particularly informative in situations where there are identified occurrences of very rare conditions for which there are few, if any, established causal factors. . . . In fact, recognition of even a small number of cases of the "sentinel" diseases—such as liver angiosarcoma and malignant mesothelioma, which is strongly related to asbestos exposure.⁵⁰

The scientific community has concluded that, for sentinel diseases such as mesothelioma, case series reports can be sufficient by themselves to allow reliable conclusions to be drawn regarding causation. Again, as noted by Checkoway:

Case series reports can be virtually conclusive in their own right when the health outcome is a very rare disease or an uncommon manifestation of a relatively common condition.⁵⁰

We do not suggest that such conclusions are indisputable or inviolate; scientific knowledge rarely is. The relevant question is whether reliable and scientifically justifiable conclusions can be drawn based upon such information, when considered in connection with all other available evidence. They can. In fact, proper application of the scientific method requires consideration of all forms of available evidence.

Accepted Method for Evaluating Disease Causation in an Individual: Generally and as Applied to Asbestos Exposure and Mesothelioma

Examining the question of causation of disease in an individual generally involves four questions: 1) was the individual exposed to a toxic agent 2) does the agent cause the disease present in the individual; 3) was the individual exposed to this substance at a level where disease has occurred in other settings; and 4) have other competing explanations for the disease been excluded?

There is no reasonable dispute regarding Question 2—asbestos causes mesothelioma. Additionally, there are no well-accepted competing explanations regarding mesothelioma that must be excluded, resolving Question 4. As a result, when considering the issue of causation of a mesothelioma, once an occupational or para-occupational exposure to asbestos has been established (Question 1), the sole question remaining for

examination is whether the exposure or set of exposures of that individual is similar to exposures that have been documented to cause mesothelioma in others—Question 3.

The mainstream scientific community is in consensus regarding the resolution of Question 3. As discussed above, there is no safe level of exposure to asbestos. Even exposure at current regulatory levels results in excess mesotheliomas.^{7,38} Accordingly, the consensus of the scientific community is that any occupational or para-occupational exposure to asbestos—even “brief or low-level exposures”—must be considered causal in an individual with a mesothelioma.

The Claim of the Asbestos Brake Manufacturers That the Studies upon Which They Rely Trump All Other Scientific Knowledge Is Scientifically Unsupportable.

The asbestos brake manufacturers cite a number of epidemiologic studies as proof that asbestos from brakes cannot cause mesothelioma. The manufacturers claim the fact that these studies did not detect a statistically significant increased risk of mesothelioma in the occupational groups studied is conclusive proof that no person can ever contract disease from working with asbestos brakes.

That claim is simply not scientifically supportable. We need not examine here the individual shortcomings of the studies relied upon by the asbestos brake manufacturers. Others have done so cogently and in detail.^{16,34} While our rejection of the asbestos brake manufacturers’ sweeping claim is supported by these critiques, the fundamental scientific failing of their claim is not based on the obvious limitations of the individual studies. There is a difference between a truly negative result and a non-positive result. A true negative study must be large, sensitive, and contain accurate exposure data. Even then, the study will be negative only with respect to the exposure level studied. Far from proving that no person can ever get sick from asbestos dust released by brakes, the best that can be said for the studies is that they are inconclusive. Instead, such a claim is based on the scientifically unsupportable proposition that one study, or group of studies, trumps all other evidence, no matter how extensive and well-documented that evidence is. Additional discussion of the implication of “negative” epidemiologic studies may be found elsewhere.⁵¹⁻⁵⁴

As noted above, examination of the question of whether a substance is capable of causing disease requires consideration of all scientific disciplines and all available evidence. This is particularly true when asserting that exposure can *not* cause an effect.

The conclusion that some exposure is devoid of harmful effect (e.g. a certain chemical is not carcinogenic) must be based on a synthesis of the

whole available literature: it can never rely on one single study. Hence, all the scientific evidence (i.e. theoretical experimental, and epidemiologic) that exists must be combined.⁵⁵

Substantial insight into this issue is provided by the industry consultants hired by Ford, General Motors, and Chrysler. When hired to represent the auto industry in asbestos-brake litigation, the industry consultants assert that their cited epidemiologic studies trump all other evidence, and conclusively refute the claim that asbestos from brakes can cause mesothelioma.^{29,30} Conversely, when hired by the power industry to provide testimony regarding epidemiologic studies that were damaging to that industry, Dr. Hessel rejected this same position:

Because of such recognized limitations, epidemiology studies by themselves generally do not provide sufficient basis to support conclusions about causation. That is why the assessment of health risk must rely on data from toxicological studies in animals, studies in human cells and tissues and experimental clinical studies.⁵⁶

The opportunistic rejection of whatever evidence exists contrary to the position of the industry being defended is a hallmark of “doubt science.” We disagree with both extremes. Epidemiologic evidence may, in cases, be sufficient to make reasoned and well-founded judgments regarding causation after consideration of other available evidence, even if evidence from one or more other scientific disciplines is absent. Conversely, consideration of other scientific evidence may allow reasoned conclusions regarding causation in the absence of positive epidemiologic studies regarding a specific population.

It is unscientific for the asbestos brake manufacturers to assert that their chosen epidemiologic studies trump all other evidence, just as it was unscientific for the tobacco industry to claim that lack of understanding of the mechanism by which tobacco causes cancer made it impossible to conclude that cigarettes cause cancer. Proper application of the scientific method requires that all available evidence be considered when examining issues of causation.

Evidence Supporting the Conclusion That Asbestos from Brakes Can and Does Cause Disease, Including Mesothelioma

The danger of asbestos in brakes has been recognized for decades. The hazard from exposure to asbestos in friction products has been known and accepted for over 70 years. In 1948, General Motors’ chief industrial hygienist published regarding the hazards created when manipulating asbestos brake materials in the factory.⁵⁷ By 1958, the danger of exposure to asbestos dust from brakes was sufficiently well documented that it was

included in the American Industrial Hygiene Association's Hygienic Guide series.⁵⁸ Additionally, mesotheliomas have been documented repeatedly in workers at friction-product factories.^{34,59-62}

Today, the asbestos brake manufacturers assert that this danger is confined to the friction-product manufacturing facility. However, there is no scientific justification for asserting that dust from an asbestos brake can cause disease when the brake is ground in a factory but cannot cause disease when that same brake is ground in a garage.

Mechanics who work with asbestos brakes without dust-control measures are exposed to asbestos. Numerous studies have demonstrated that mechanics who worked with asbestos-containing brakes without dust-control measures were exposed to asbestos dust. This is particularly true when the mechanic grinds, files, or sands the new asbestos brake and uses compressed air or dry brushing to clean out wear dust from old asbestos brakes.⁶³⁻⁶⁶ Both the EPA and OSHA have issued guidance to reduce the risk of disease from asbestos exposure during brake work.^{7,67,68} OSHA requires the use of dust controls when employees work with asbestos-containing brakes and clutches (for specific details see appendix F of the standard).⁷ The EPA has adopted these standards for municipal employees in jurisdictions not governed by state asbestos-control plans.⁶⁷ Other regulatory agencies have similarly issued guidance to mechanics to reduce exposures to asbestos from brakes and clutches.⁶⁹⁻⁷²

Equally important, it has been proven that use of effective dust-control measures can lower exposure levels during work with asbestos brakes.^{73,74} Accordingly, to provide a reliable basis for the conclusion that asbestos from brakes can never cause disease, a study of brake mechanics would ascertain whether individuals considered "exposed" to asbestos brakes used dust-control practices. None of the studies relied upon by the asbestos brake manufacturers contains such information.

Studies have shown increased incidences of non-malignant asbestos-related diseases among mechanics known to have performed work with asbestos-containing brakes. Excessive non-malignant disease in mechanics occurs in individuals known to have worked with asbestos-containing brakes.^{65,75} It is universally accepted that the amount of asbestos exposure needed to cause asbestosis is greater than the amount needed to cause mesothelioma. Accordingly, studies demonstrating excess asbestosis in asbestos brake-repair workers demonstrate that these workers were historically exposed to quantities of asbestos far in excess of that needed to cause mesothelioma.

Proper scientific inquiry cannot ignore the hundreds of reported cases of mesothelioma in mechanics. We reject the contention of the asbestos brake manufacturers that the scientist must close his or her eyes and refuse to consider case reports or case series of mesothelioma in mechanics, irrespective of how many cases are reported. This contention flies in the face of sound sci-

entific reasoning, which *requires* thoughtful consideration of *all* available evidence.

As discussed above, the consideration of case reports is even more critical when examining rare, sentinel diseases such as mesothelioma because of the great difficulty in conducting epidemiologic studies with sufficient power to reliably detect increases in disease.^{53,54} Hundreds of cases of mesothelioma in mechanics have been reported in the medical literature, including dozens of cases in the studies relied upon by the asbestos brake manufacturers.¹⁶

The precise number of cases is not important for purposes of our discussion, nor is the possibility that some cases may have involved exposures to asbestos from sources other than brakes. The important point is that proper scientific inquiry not only can consider these reports, but, in fact, must consider them. Contrary to the suggestion of the asbestos brake manufacturers, these cases cannot be cavalierly dismissed as "unscientific" or "insufficient to support conclusions regarding causation." When considering the important question of whether working with asbestos-containing brakes can cause incurable, inevitably terminal diseases, such as mesothelioma, case series must be considered and evaluated, along with all other available evidence.

There is nothing novel regarding the use of Sir Austin Bradford Hill's viewpoints to arrive at the conclusion that asbestos from brakes can cause disease. Application of his viewpoints has been an accepted and valid method for examination of questions of causation for decades and remains so today.⁷⁶ His own wise words are worth repeating:

Here then are nine different viewpoints from all of which we should study association before we cry causation. What I do not believe—and this has been suggested—that we can usefully lay down some hard-and-fast rules of evidence that must be obeyed before we can accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*. What they can do, with greater or less strength, is to help us make up our minds on the fundamental question—is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?

Contrary to the all-or-nothing position of the asbestos brake manufacturers, there is no single scientific discipline or type of study that takes precedence over others. Thoughtful scientific inquiry requires consideration of all evidence when making determinations regarding causation.

CONCLUSION

Asbestos causes mesothelioma. Mechanics are exposed to asbestos dust during the servicing and replacement

of brakes. While the asbestos brake manufacturers claim that the average amount of asbestos released from brake repair work is comparatively low, there is no reasonable dispute that exposure levels were higher when mechanics routinely ground, filed, and sanded brakes and used compressed air to blow out brake wear debris, and did this work without dust control. It is those historic higher exposures that caused disease appearing now. The scientific community is in consensus that brief and low-level exposures to asbestos can cause mesothelioma. The scientific literature contains hundreds of cases of mesothelioma among brake mechanics; and epidemiologic studies of mechanics known to have performed repair work on asbestos-containing brakes have demonstrated increased levels of nonmalignant diseases.

This combination of evidence, and the vast amount of additional scientific information regarding asbestos and mesothelioma, provides more than sufficient evidence to allow someone to conclude within a reasonable degree of scientific certainty that a mesothelioma in a mechanic who worked with asbestos-containing brakes was caused by that asbestos exposure.

Since 2000, Ford, General Motors, and Chrysler have paid over \$30,000,000 to hire consultants for the purpose of generating the very papers they rely upon, and for testifying regarding those papers in Courts.[†] One of the main industry experts has acknowledged that the papers were conceived and authored for the purpose of buttressing testimony in court cases involving mechanics suffering from mesothelioma.[‡]

The same expert also acknowledged that this business model is a pattern he has also followed with dioxin, benzene, hexavalent chromium, beryllium, formaldehyde, and glycol ethers. Recent revelations regarding undisclosed involvement of the employer of these experts in connection with publication of a paper favorable to the chromium industry have been well publicized and led to the retraction of that paper.^{77,78} It is in no way surprising that the experts and papers financed by these manufacturers conclude that asbestos in brakes can never cause mesothelioma. To the contrary, the exoneration of the sponsoring industry is the *expected* conclusion of doubt science. Despite the best efforts of the asbestos brake manufacturers and their hired experts to fabricate scientific uncertainty where none exists, the mainstream scientific community and regulatory communities have considered the available evidence and concluded that the danger to mechanics from asbestos in brakes is real.

[†]Ford, General Motors, and Chrysler have admitted in litigation that, since 2000, they have paid over \$30,000,000 to these experts. See, Ford and General Motors, Answers to Interrogatories, *Unden v. General Motors*, Case No. 05:6311, Circuit Court for Hillsborough County, Florida, and Chrysler IRS Form 1099s produced in litigation.

[‡]Deposition of Dennis Paustenbach, July 1, 2005, *Mallia v. Bennett Auto et al.*, Case Number 04-16236 CA 42, Circuit Court in and for Dade County, Florida.

Apparently, the asbestos brake manufacturers hope that these arguments can be used to sway the Supreme Court of Michigan and other courts. As scientists who have devoted substantial portions of our professional lives working to research, prevent, and treat asbestos-related diseases, we reject these attempts to fabricate uncertainty where none exists. Instead, we request that these courts attend to the work of thousands of experts from around the world who have concluded that asbestos, in any form, and through any occupational exposure, can and does cause disease.

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References

1. U.S. Public Health Service, U.S. Department of Health and Human Services. Toxicological profile for asbestos. Atlanta, GA: Agency for Toxic Substances and Disease Registry, 2001.

2. American Conference of Governmental Industrial Hygienists. Asbestos: TLV(r) Chemical Substances 7th Edition Documentation. Publication #7DOC-040. Cincinnati OH: ACGIH, 2001.
3. Diagnosis and initial management of nonmalignant diseases related to asbestos. *Am J Respir Crit Care Med* 2004; 170:691-715.
4. Environmental Protection Agency. Airborne asbestos health assessment update. Washington, DC: U.S. EPA (EPA/600/8-84/003F), 1986.
5. International Agency for Research on Cancer. Asbestos: Monograph on the Evaluation of Carcinogenic Risk to Man. Lyon, France: IARC, 1988.
6. National Toxicology Program. Report on Carcinogens, 11th ed. U.S. Department of Health and Human Services, Public Health Service, 2005.
7. Occupational Safety and Health Administration. Occupational exposure to asbestos; final rule. *Fed Reg*. 1994;59:40964-41162.
8. Consumer Product Safety Commission. CANCER HAZARD! CPSC Warns About Asbestos in Consumer Products: Safety Alert. CPSC Document #5080. 2004.
9. World Health Organization. Elimination of asbestos related diseases. Geneva, Switzerland: WHO, 2006.
10. World Health Organization. Environmental Health Criteria 53: Asbestos and Other Natural Mineral Fibres. Geneva, Switzerland: WHO, 1986.
11. World Health Organization. Environmental Health Criteria 203: Chrysotile Asbestos. Geneva, Switzerland: WHO, 1998.
12. World Trade Organization. European Communities—Measures Affecting Asbestos and Asbestos-containing Products. WT/DS135/R. 2000.
13. Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. *Scand J Work Environ Health* 1997; 23:311-6.
14. Malignant Mesothelioma. American Cancer Society, 2006. 10-19-2006.
15. National Cancer Institute. Factsheet—Asbestos: Questions and Answers. Bethesda, MD: National Institutes of Health, 2003.
16. Lemen RA. Asbestos in brakes: exposure and risk of disease. *Am J Ind Med*. 2004;45:229-37.
17. Frank AL, Dodson RF, Williams MG. Carcinogenic implications of the lack of tremolite in IICG reference chrysotile. *Am J Ind Med*. 1998;34:314-7.
18. Smith AH, Wright CC. Chrysotile asbestos is the main cause of pleural mesothelioma. *Am J Ind Med*. 1996;30:252-66.
19. Cullen MR. Chrysotile asbestos: enough is enough. *Lancet*. 1998;351(9113):1377-8.
20. Landrigan PJ, Nicholson WJ, Suzuki Y, LaDou J. The hazards of chrysotile asbestos: a critical review. *Ind Health*. 1999; 37:271-80.
21. Landrigan PJ, Soffritti M. Collegium Ramazzini call for an international ban on asbestos. *Am J Ind Med*. 2005;47:471-4.
22. Stayner LT, Dankovic DA, Lemen R. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. *Am J Public Health*. 1996;86:179-86.
23. Li L, Sun TD, Zhang X, et al. Cohort studies on cancer mortality among workers exposed only to chrysotile asbestos: a meta-analysis. *Biomed Environ Sci*. 2004;17:459-68.
24. Suzuki Y, Yuen SR. Asbestos fibers contributing to the induction of human malignant mesothelioma. *Ann NY Acad Sci*. 2002; 982:160-76.
25. Landrigan PJ. Asbestos—still a carcinogen. *N Engl J Med*. 1998; 338:1618-9.
26. Egilman D, Fehnel C, Bohme SR. Exposing the “myth” of ABC, “anything but chrysotile”: a critique of the Canadian asbestos mining industry and McGill University chrysotile studies. *Am J Ind Med*. 2003;44:540-57.
27. Michaels D. Manufactured uncertainty: protecting public health in the age of contested science and product defense. *Ann NY Acad Sci*. 2006;1076:149-62.
28. Michaels D. Doubt is their product. *Sci Am*. 2005;292(6):96-101.
29. Hessel PA, Teta MJ, Goodman M, Lau E. Mesothelioma among brake mechanics: an expanded analysis of a case-control study. *Risk Anal*. 2004;24:547-52.
30. Goodman M, Teta MJ, Hessel PA, et al. Mesothelioma and lung cancer among motor vehicle mechanics: a meta-analysis. *Ann Occup Hyg*. 2004;48:309-26.
31. Paustenbach DJ, Finley BL, Lu ET, Brorby GP, Sheehan PJ. Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to present): a “state-of-the-art” review. *J Toxicol Environ Health B Crit Rev*. 2004;7:25-80.
32. Paustenbach DJ, Richter RO, Finley BL, Sheehan PJ. An evaluation of the historical exposures of mechanics to asbestos in brake dust. *Appl Occup Environ Hyg*. 2003;18:786-804.
33. Wong O. Malignant mesothelioma and asbestos exposure among auto mechanics: appraisal of scientific evidence. *Regul Toxicol Pharmacol*. 2001;34:170-7.
34. Egilman DS, Billings MA. Abuse of epidemiology: automobile manufacturers manufacture a defense to asbestos liability. *Int J Occup Environ Health*. 2005;11:360-71.
35. Laden F, Stampfer MJ, Walker AM. Lung cancer and mesothelioma among male automobile mechanics: a review. *Rev Environ Health*. 2004;19:39-61.
36. National Institute for Occupational Safety and Health. Workplace Exposure to Asbestos: Review and Recommendations: NIOSH/OSHA Asbestos Work Group Recommendations. Department of Health and Human Services, 1980: 81-103.
37. Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann Occup Hyg*. 2000;44:565-601.
38. Iwatsubo Y, Pairon JC, Boutin C, et al. Pleural mesothelioma: dose-response relation at low levels of asbestos exposure in a French population-based case-control study. *Am J Epidemiol*. 1998;148(2):133-42.
39. Lin RT, Takahashi K, Karjalainen A, et al. Ecological association between asbestos-related diseases and historical asbestos consumption: an international analysis. *Lancet*. 2007;369(9564): 844-9.
40. Agudo A, Gonzalez CA, Bleda MJ, et al. Occupation and risk of malignant pleural mesothelioma: a case-control study in Spain. *Am J Ind Med*. 2000;37:159-68.
41. Checkoway H, Pearce NE, Crawford-Brown DJ. Research Methods in Occupational Epidemiology. New York: Oxford University Press, 1989.
42. Leigh J, Driscoll T. Malignant mesothelioma in Australia 1945-2002. *Int J Occup Environ Health*. 2003;9:206-17.
43. Coggon D, Pippard EC, Acheson ED. Accuracy of occupational histories obtained from wives. *Br J Ind Med*. 1985;42:563-4.
44. Lerchen ML, Samet JM. An assessment of the validity of questionnaire responses provided by a surviving spouse. *Am J Epidemiol*. 1986;123:481.
45. Mark EJ, Yokoi T. Absence of evidence for a significant background incidence of diffuse malignant mesothelioma apart from asbestos exposure. *Ann NY Acad Sci*. 1991;643:196-204.
46. Lemen RA. Asbestos: Risk Assessment, Epidemiology, and Health Effects. Boca Raton, FL: Taylor and Francis, 2006.
47. Teschke K, Morgan MS, Checkoway H, et al. Mesothelioma surveillance to locate sources of exposure to asbestos. *Can J Public Health*. 1997; 88:163-8.
48. Rutstein DD, Mullan RJ, Frazier TM, Halperin WE, Melius JM, Sestito JP. Sentinel health events (occupational): a basis for physician recognition and public health surveillance. *Am J Public Health*. 1983;73:1054-61.
49. Wagner JC, Sleggs CA, Marchand P. Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. *Br J Ind Med*. 1960;17:260-71.
50. Checkoway H, Pearce N, Crawford-Brown DJ. Research Methods in Occupational Epidemiology. 2nd ed. London, U.K.: Oxford University Press, 2004.
51. Ahlbom A, Axelson O, Stottrup Hansen ES, Hogstedt C, Jensen UJ, Olsen J. Interpretation of “negative” studies in occupational epidemiology. *Scand J Work Environ Health*. 1990;16:153-7.
52. Altman DG, Bland JM. Absence of evidence is not evidence of absence. *BMJ*. 1995;311(7003):485.
53. Bailar JC. How to distort the scientific record without actually lying: truth, and the arts of science. *Eur J Oncol*. 2006;11:217-24.
54. Hernberg S. “Negative” results in cohort studies—how to recognize fallacies. *Scand J Work Environ Health*, 1981; 7 suppl 4:121-6.
55. Hernberg S. Some guidelines for interpreting epidemiologic studies. In: Introduction to Occupational Epidemiology. Chelsea, MI: Lewis Publishing, 1992: 201-23.

56. Hessel PA. Testimony before Public Service Commission of Wisconsin. Docket 05-CE-130. 2007.
57. Castrop. Fume and dust exposure. *National Safety News*. 1948;20, 53, 72-80.
58. American Industrial Hygiene Association. Hygienic standards: asbestos. *J Am Ind Hyg Assoc*. 1958;19:161-2.
59. Godwin MC, Jagatic G. Asbestos and mesothelioma. *JAMA*. 1968;204:1009.
60. Teta MJ, Lewinsohn HC, Meigs JW, Vidone RA, Mowad LZ, Flannery JT. Mesothelioma in Connecticut, 1955-1977. Occupational and geographic associations. *J Occup Med*. 1983;25:749-56.
61. Robinson CF, Lemen RA, Wagoner JK. Mortality patterns, 1940-1974 among workers employed in an asbestos textile friction and packing product manufacturing facility. In: Lemen RA, Dement J (eds). *Dust and Disease*. Park Forest, IL: Pathotax Publishers, 1979: 131-40.
62. McDonald AD, Fry JS, Woolley AJ, McDonald J. Dust exposure and mortality in an American chrysotile textile plant. *Br J Ind Med*. 1983;40:361-7.
63. Sakai K, Hisanaga N, Shibata E, Ono Y, Takeuchi Y. Asbestos exposures during reprocessing of automobile brakes and clutches. *Int J Occup Environ Health*. 2006;12:95-105.
64. Rohl AN, Langer AM, Wolff MS, Weisman I. Asbestos exposure during brake lining maintenance and repair. *Environ Res*. 1976; 12:110-28.
65. Lorimer WV, Rohl AN, Miller A, Nicholson WJ, Selikoff IJ. Asbestos exposure of brake repair workers in the United States. *Mt Sinai J Med*. 1976;43:207-18.
66. Hickish DE, Knight KL. Exposure to asbestos during brake maintenance. *Ann Occup Hyg*. 1970;13:17-21.
67. Environmental Protection Agency. Current Best Practices for Preventing Asbestos Exposure Among Brake and Clutch Repair Workers. EPA 747-F-04-004. Environmental Protection Agency, 2007.
68. Occupational Safety and Health Administration. Asbestos—Automotive Brake and Clutch Repair Work. SHIB 07-26-2006. U. S. Department of Labor; Occupational Safety and Health Administration, 2006.
69. Washington State Department of Labor and Industries. Working Safely with Asbestos in Clutch and Brake Linings. F413-049-000. Olympia WA, Washington State Department of Labor and Industries, 2001.
70. New Hampshire Pollution Prevention Program. Pitstops Manual: Best Management Practices for Automobile Service Facilities. New Hampshire Department of Environmental Services, 2001.
71. Worksafe Alberta. Control of asbestos during brake maintenance and repair. Edmonton, AB: Department of Human Resources and Employment, Government of Alberta, 2004.
72. Minnesota Pollution Control Agency. Facts about Controlling Brake Dust to Protect Your Health . . . What Every Mechanic Should Know. St. Paul, MN: Minnesota Pollution Control Agency, 1998.
73. Johnson P, Zumwalde RD, Roberts D. Industrial Hygiene Assessment of Seven Brake Servicing Facilities—Asbestos. Cincinnati, OH: National Institute for Occupational Safety and Health, 1979.
74. Roberts D. Industrial Hygiene Report—Asbestos, Allied Brake Shop. Cincinnati, OH: National Institute for Occupational Safety and Health, 1980.
75. Nicholson WJ. Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos. 210-77-0119. Cincinnati, OH: National Institute for Occupational Safety and Health, 1984.
76. Hill AB. The environment and disease: association or causation? *Proc R Soc Med*. 1995;58:295-300.
77. Waldman P. Publication to retract an influential water study. *Wall Street Journal*, June 2, 2006.
78. Waldman P. Study tied pollutant to cancer; then consultants got hold of it. *Wall Street Journal*. 2005.