

ASBESTOS LITIGATION: A DEFENSE PRIMER FOR MOTOR VEHICLE MANUFACTURERS

INTRODUCTION

For the past several decades, asbestos litigation has been presumed dead many times only to reawaken in a new form with renewed vigor, much to the horror of those industries and companies next up on the target list. In excess of 60 companies have been bankrupted and hundreds of thousands of jobs have been lost as a result of asbestos litigation. While motor vehicle manufacturers and parts suppliers have been targets for decades, given the disappearance of the companies that supplied raw asbestos and insulation, the automotive industry has increasingly found itself in the cross-hairs of the litigation for the past several years and that trend shows no sign of abating.

Asbestos is a naturally occurring mineral to which we are all exposed at various times in our lives and most of us have asbestos fibers in our lungs. There is no doubt that not all exposures to asbestos have the potential to cause disease because a very small portion of the population actually gets sick as a result of asbestos exposures. In fact, science has established that there must be a sufficient dose, i.e., exposure to a sufficient quantity over a sufficient period of time, for there to be the potential for disease causation. The problem, however, is that the current state of the science does not permit us to know precisely what that dose is and therefore, a very vocal minority of scientists contend that every exposure over and above what is found in the ambient air is potentially causative of disease.

Currently, the motor vehicle manufacturers and parts suppliers find themselves under siege despite the existence of scientific evidence overwhelmingly demonstrating that exposures to asbestos-containing automotive parts do not provide a sufficient dose to cause disease. Defense of these cases requires that a jury understand the relevant science as well as the historical context for the use of asbestos and the concern raised regarding *all* types of asbestos exposures and how that concern was addressed regarding motor vehicle repair. Every exposure does not have the potential to cause disease and it is up to defense counsel to make that concept understandable to a lay jury who does not live and breath asbestos litigation.

WHAT IS ASESTOS?

As mentioned, asbestos is a naturally occurring silicate mineral which became immensely popular for industrial use because of its strength and fire resistant properties. The word asbestos comes from the Greek meaning “unquenchable” or “inextinguishable.” It is critical to note that there are different types of this mineral and that each has different chemical properties, looks different, is found in different parts of the world, has different commercial applications and, most importantly, has different levels of toxicity. There are two classes, serpentine¹ and amphibole. Chrysotile, the most prevalent form of asbestos used in the United States falls within the serpentine class and the remaining forms, amosite, crocidilite, tremolite, anthophyllite and actinolite make up the amphibole class. While litigation experts may disagree, it is well accepted

¹ Serpentine rock which contains chrysotile is the California state rock.

in the scientific community that amphibole asbestos is much more toxic than serpentine.

In the United States, chrysotile asbestos was used in so-called “friction products,” i.e. brakes and clutches because of its ability to withstand the intense heat generated in the braking or clutching process as well as its strength and durability. In brakes, asbestos was contained in the brake linings in drum brakes and in the pads in disc brakes. In clutches, the clutch facings contained the asbestos. The linings, pads and facings were not 100% asbestos, rather, the asbestos fibers were contained within a bonding material, typically a phenolic resin, which contained other materials. Generally, chrysotile asbestos made up only 30% to 50% of the entire friction product.² The exact make-up of the bonded material was typically unique and proprietary to the manufacturer of the friction product. In addition to friction materials, motor vehicles contained multiple gaskets, some of which may have contained asbestos.

The advent of fast, heavy cars in the 20th Century required that a safe, reliable way to stop those cars be utilized. Asbestos containing friction materials were the best alternative and when the use of asbestos in all products, including automotive products, began to be questioned, it took several years to find a suitable alternative. A long process of investigation and testing culminated in most asbestos containing friction products being eliminated from automobiles in the 1980's and 1990's. Until that time, virtually every motorized vehicle sold in

² The content could be as low as 20% or as high as 75% but 30% to 50% constitutes the most typical range.

the United States contained some asbestos containing friction components and perhaps some asbestos containing gaskets.

ASBESTOS RELATED DISEASES³

There are several diseases which have been associated with asbestos exposures: mesothelioma, lung cancer, asbestosis and benign asbestos-related pleural disease. Mesothelioma, a cancer of the mesothelium associated with asbestos exposure, is typically the most serious of these diseases and the type of cancer which most often gives rise to litigation that ultimately results in a jury verdict. The association of lung cancer with multiple causes besides asbestos, including cigarette smoking, make these cases more difficult to prove from a causation standpoint than cases in which the plaintiff complains of an asbestos-related mesothelioma. Cases in which the plaintiff asserts asbestosis or benign asbestos-related disease such as parietal pleural plaques are typically referred to as non-malignancy cases.

Mesothelioma, otherwise known as malignant diffuse mesothelioma, is a malignant cancer of the mesothelium or serosal linings of the pleural, pericardial or peritoneal cavities. It is a rare cancer which has a very strong association with asbestos exposures. There is a period of time between exposure and the onset of the disease which is known as the latency period, The typical latency period is 30 to 40 years but it may be as short as 15 years or as long as 70 years. The disease is most often the result of occupational exposures in trades involving prolonged and intensive exposures to asbestos and affects men primarily. The

³ Information for this section taken from: Roggli, VL, Oury, TD, and Sporn, TA, *Pathology of Asbestos-Associated Diseases*, Boston: Little, Brown and Company (2004).

rates for mesothelioma have increased for men since the industrial revolution concomitant with increased occupational uses of asbestos, while the rates for women have remained relatively the same. This observation is consistent with the generally accepted notion that there is a background rate of mesothelioma which is not necessarily caused by asbestos exposures. When there is no known cause for the disease, it is referred to as idiopathic i.e., arising spontaneously or from an unknown cause. While some experts who testify for plaintiffs assert that *all* mesotheliomas are caused by asbestos exposure, most experts in the field agree that some percentages are idiopathic, and this number increases with females and with peritoneal mesothelioma.

It is generally accepted that amphibole asbestos fibers have much greater potential to cause mesothelioma than do serpentine fibers. In particular, crocidolite is considered the most mesotheliogenic, followed by amosite which was the most commercially used amphibole in the United States. There is some controversy as to whether chrysotile fibers can cause mesothelioma at all but it is generally agreed that chrysotile is, at the very least, much less carcinogenic than the commercial amphiboles. In addition, shorter fibers, less than 5 microns in length, are considered unlikely to cause mesothelioma. Peritoneal mesothelioma is most commonly associated with very high level exposures to commercial amphiboles.

ARE MECHANICS AT GREATER RISK FOR ASBESTOS RELATED DISEASES?

With respect to mesothelioma, at least at this time, science does not permit us to go to the first cancer cell and determine exactly which exposure

caused the disease to start. It is generally accepted that once the disease process starts, subsequent exposures are irrelevant as to causation but it is not currently possible to know exactly when the disease starts. Often a mesothelioma victim may have multiple exposures over the course of a lifetime. For instance, there may be intense exposure to highly toxic amosite asbestos insulation while on ships in the US Navy as well as a period of time working as a mechanic. Which exposure caused the disease?

Epidemiology is the study of the incidence, distribution and causation of disease in a human population. Epidemiologists study groups and determine which groups are at greater risk for disease. For instance, epidemiological studies were useful in determining that people who smoke have a greater risk for cancer and cardiovascular disease. These types of studies can be quite complex as the scientists try to account for confounding factors, or factors which can confuse the data. Epidemiological studies were important in determining the correlation between asbestos exposures and disease, including mesothelioma. These types of studies are often relied upon in litigation to determine whether exposure to a particular agent was the cause of the plaintiff's particular disease and asbestos litigation is no exception.

It is undisputed that although numerous epidemiological studies have been conducted over the past thirty years or so, no such study has found that professional vehicle mechanics are at greater risk for mesothelioma than the general population. Contrary to the assertion that these studies were funded by the automotive industry, they are the product of a broad array of sources

including the National Cancer Institute, NIOSH, other governmental health agencies, universities, and unions. Epidemiological studies *do* establish a causal connection between mesothelioma and occupations such as shipyard workers, insulators and plumbers where exposure is high, especially to commercial amphiboles.

In the context of what goes on in the real world, the results of these epidemiology studies make sense. First, mechanic work involving asbestos-containing products in motor vehicles is intermittent and limited. Accessing a clutch requires a significant amount of work and expense and does not need to be done very often. Although some gaskets may contain asbestos, the ones that do, do not need to be handled very often. Changing brake pads and linings involves very little time actually handling the asbestos containing components. Moreover, professional mechanics spend their time doing many other things besides brake, clutch and gasket work. Second, the type of asbestos used in automotive products in the United States is chrysotile, the least carcinogenic fiber. Third, for a number of reasons the actual dose of asbestos which a mechanic may experience is extremely low and mainly consists of short fibers. Viewed in conjunction with the reality of what happens in a garage, the epidemiology studies make sense and establish that professional mechanics, never mind amateur, shade tree mechanics and bystanders, are not at greater risk for mesothelioma than the general public.

WHY THE LOW DOSE?

Mechanics spend their time in large bays which open to the outside throughout the course of the day. Cars are driven in and out and the outside breeze carries in fresh air while it carries out dust and debris. As stated above, mechanics do many things during the course of their day and even if they are in contact with asbestos containing components, the exposure is limited. Time is money and mechanics do not spend needless time handling used or new parts. Motor vehicles have thousands of parts and those that contain asbestos are but a very small fraction.

With respect to asbestos containing gaskets, clutch plates and brake linings, the asbestos fibers which only make up a percentage of these parts, typically less than or close to 50%, are contained in a resin which affects their ability to be respirable. Thus even if new parts are subject to grinding, sanding or drilling, which is not typically necessary, the fibers are not free floating. Rather, studies have shown that the fibers remain attached to the resin and thus are much less likely to be breathed in and much less likely to be retained in the lung if they are inhaled.

When removing an old brake, mechanics would typically "blow out" the old dust which remained in the drum. This dust which is referred to as brake dust contains very little asbestos. Of course, disc brakes are open to the outside air flowing through and so there is little if any brake dust with which to contend where they are concerned.

Many studies have been conducted on the contents of brake dust. Since the components start out at 30% to 50% chrysotile asbestos, one would expect that the content of brake dust would be similar but that is not the case. The vast majority of studies have concluded that brake dust contains less than 1% asbestos fibers and most of those fibers are short fibers. While a couple of earlier studies got slightly higher concentrations, those studies did not use the currently accepted NIOSH method for identifying fibers.

What happens to the 30% to 50% chrysotile asbestos which was contained in the original brake lining? In order to slow a motor vehicle, the kinetic energy of the vehicle must be dissipated and that is done by converting the energy to friction and heat during the braking process. The heating and shearing forces created during the braking process not only break the fibers into smaller pieces but they also convert the fibers chemically into another mineral: forsterite, a non-carcinogenic olivine. A question has been raised as to whether even the small amount of chrysotile left in brake dust retains its carcinogenicity because, although not completely converted to forsterite, the surface properties of the remaining chrysotile fibers undergo significant changes as a result of the heat involved in braking.⁴

Not surprisingly, given the low levels of respirable fibers to which a mechanic is actually exposed doing brake work, studies have shown that mechanic's exposures consistently fell below the Permissible Exposure Limit ("PEL") set by OSHA at the relevant time period when the work was done. While

⁴ Langer, AM, *Reduction of the Biological Potential of Chrysotile Asbestos Arising from Conditions of Service on Brake Pads*, Regul Toxicol Pharmacol 38(1):71-77 (2003).

some peak exposures may fall above this level, the PEL is determined by looking at the Time Weighted Average ("TWA") over an 8-hour work day to determine the maximum permissible exposure. Thus, while at certain times of day a mechanic might experience a peak exposure concentration, because of the intermittent nature of the work, the total exposure seen is well within permissible limits.

Heavy truck and bus mechanics experience slightly higher exposures than do car and light truck mechanics. Nonetheless, the various studies performed in the US and in other countries show that exposures to asbestos are extremely low for professional mechanics.

WHY THE CONTROVERSY?

Given what the science shows, why is there any controversy at all regarding whether automotive exposures are causative of disease? A number of factors play into the continuing assault on the automobile industry. Although it is axiomatic that not all types of exposures can cause disease, science has been unable to draw a definitive line below which lack of carcinogenicity can be established. This fact leads to the assertion by the experts who testify for plaintiffs in this litigation that since no exposure can be ruled out definitively by science as not being causative, then all exposures must be considered to be causative including the most *de minimis* exposures. Given that the major asbestos manufacturers have by now all gone bankrupt, this type of testimony is crucial to keeping the litigation alive against the so-called "low-dose" defendants such as the motor vehicle manufacturers.

Those in the field which refuse to acknowledge the growing body of science which establishes the lack of causation between work by mechanics and cancer point to case reports of mechanics with supposedly no other asbestos exposure who have contracted mesothelioma. Although, in the absence of other science, case reports can raise the question of a correlation between agent and disease, alone, they cannot establish causation. This approach ignores the science which shows no correlation here and ignores that case reports do not deal well with confounding factors. Case reports do not account for the background level of mesothelioma, nor do they typically verify no other exposure through fiber burden studies, which look to see if there are any commercial amphiboles in the lungs that would establish other exposure besides mechanic work. One fiber burden study which looked at mesotheliomas in brake workers found either normal levels of asbestos or elevated levels of commercial amphiboles in their lungs.⁵ The presence of commercial amphiboles in the lungs of these mechanics is indicative of exposures to asbestos through work other than as a mechanic.

To establish a case against the motor vehicle manufacturers and their parts suppliers, plaintiffs' attorneys must ignore the science and lump all types of asbestos exposures together as bad. Often they are helped by the manufacturers themselves who over the years have done the same thing with respect to Material Safety Data Sheets and other types of documents on the

⁵ Butnor, KJ, A Sharma, TA Sporn, and VL Roggli, *Malignant Mesothelioma and Occupational Exposure to Asbestos: An analysis of 1445 cases*, Ann Occup Hyg 46 (Suppl 1):150-153 (2002); Roggli, VL, A Sharma, KJ Butnor, T Sporn, and RT Vollmer, *Malignant Mesothelioma and Occupational Exposure to Asbestos: A Clinicopathological Correlation of 1445 Cases*, Ultrastructural Pathology 26:55-65 (2002).

subject as well as the warnings on their products. The question is raised, why include a warning if there is no danger? The answer of course is that the history of how asbestos has been dealt with by government and industry is not a simple one. Questions were raised in the mid-1970's as to whether there was any hazard associated with the use of asbestos-containing motor vehicle parts and the topic has been the focus of much study since. While today we can look back and say that the science is clear that such exposures do not present a risk of disease, in the intervening years, at various times, various manufacturers have provided warnings and information regarding the asbestos in their products. They did so not because they were receiving reports of disease attributable to their products. On the contrary, unlike with plumbers, pipefitters and insulators, the questions raised about automotive exposures came about not because of an increased level of disease, but rather because the asbestos was simply there. Taking a few documents out of context now does not give the entire picture with respect to the history of asbestos use and how scientists, government and industry became aware of the hazards to workers with high levels of exposure and how today we have a better understanding of disease causation.

CONCLUSION

The conclusion of the scientific studies in the fields applicable to asbestos exposures, i.e., pathology, epidemiology, industrial hygiene, mineralogy and risk assessment, without exception, lead to the conclusion that motor vehicle mechanics are not at risk for asbestos related diseases by virtue of doing this work. However, given the reduction in deep pockets through bankruptcies, the

motor vehicle manufacturers can continue to expect to be the subject of asbestos lawsuits for the foreseeable future. The plaintiffs' approach to these lawsuits is to ignore the science, lump all exposures together as harmful, use the manufacturer's documents against them and appeal to the emotional reaction expected at the mere mention of the word asbestos. Defense counsel faces the tough assignment of having to explain very complicated science in an exciting and understandable way to a lay jury. Only with a thorough understanding of this science can that task be accomplished.

BIBLIOGRAPHY OF TREATISES RELEVANT TO ALLEGED MOTOR VEHICLE RELATED ASBESTOS EXPOSURES

Paustenbach, D.J., R.O. Richter, B.L. Finley, and P.J. Sheehan. 2003. An evaluation of the historical exposures of mechanics to asbestos in brake dust. *App Occup Environ Hyg.* 18(10):786–804.

Paustenbach, D.J., B.L. Finley, E. Lu, G.P. Brorby, and P. Sheehan. 2004. 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.* 7(1):33-110.

Goodman M, Teta MJ, Hessel PA, Garabrandt DH, Craven VA, Scrafford CG, Kelsh MA. Mesothelioma and lung cancer among motor vehicle mechanics: A meta-analysis. *Ann Occup Hyg* 2004; 48(4):309–326.

Hessel PA, Teta MJ, Goodman M, Lau E. Mesothelioma among brake mechanics: An expanded analysis of a case-control study. *Risk Anal* 2004; 24(3):547–552.

Butnor K J., Sporn T A., Roggli V. L. Exposure to Brake Dust and Malignant Mesothelioma: A Study of 10 Cases with Mineral Fiber Analyses *Ann. Hyg.*, June 1, 2003; 47(4): 325 - 330.

Langer AM. Reduction of the biological potential of chrysotile asbestos arising from conditions of service on brake pads. *Regul Toxicol Pharmacol.* 2003 Aug;38(1):71-7.

Anderson, A. E. (1973) Asbestos Emissions from Brake Dynamometer Tests. Document SAE 7305-49, Society of Automotive Engineers , Warrendale, PA

Williams RL, Muhlbaier JL. 1982. Asbestos brake emissions. *Environ Res* 29:70–82.

Hickish, D.E.; Knight, K.L.: Exposure to Asbestos During Brake Maintenance. *Ann Occup Hyg* 13:17-21 (1970)

Finley BL, Richter RO, Mowat FS, Mlynarek S, Paustenbach DJ, Warmerdam JL, Sheehan PJ. Cumulative asbestos exposure for U.S. automobile mechanics involved in brake repair (circa 1950s–2000). *J Exp Sci Environ Epidemiol* 2007; 17:644–655.

Agudo A, González CA, Bleda MJ, Ramírez J, Hernández S, López F, Calleja A, Panadès R, Turuguet D, Escolar A, Beltrán M, González-Moya JE. 2000. Occupation and risk of malignant pleural mesothelioma: a case-control study in Spain. *Am J Ind Med* 37:159-68.

Blake CL, Dotson GS, Harbison RD. 2006. Assessment of airborne asbestos exposure during the servicing and handling of automobile asbestos-containing gaskets. *Regul Toxicol Pharmacol.* 45(2):214-22.

Coggon D, Inskip H, Winter P, Pannett B. 1995. Differences in occupational mortality from pleural cancer, peritoneal cancer, and asbestosis. *Occup Environ Med* 52:775-7.

Darby SC, Muirhead CR, Doll R, Kendall GM, Thakrar B. 1990. Mortality among United Kingdom servicemen who served abroad in the 1950s and 1960s. *Br J Ind Med* 47(12):793-804.

Eastern Research Group Inc. 2003. Report of the Expert Panel on health effects of asbestos and synthetic vitreous fibers: influence of fiber length, 17 March 2003. Available at: <http://www.atsdr.cdc.gov/hac/asbestospanel/index.html>.

Appendix A: List of Expert Panelists

Appendix B: Premeeting Comments, Alphabetized by Author

Appendix C: List of Registered Observers of the Expert Panel Meeting

Appendix D: Agenda for the Expert Panel Meeting

Appendix E: Panelists' Comments Submitted After the Meeting

Hemminki K and Li X. 2003. Time trends and occupational risk factors for pleural Mesothelioma in Sweden. *J Occup Environ Med* 45(4):456-61

Hessel PA, Teta MJ, Goodman M, Lau E. 2004. Mesothelioma among brake mechanics: an expanded analysis of a case-control study. *Risk Analysis* 24(3):547-552

Hodgson JT, Peto J, Jones JR, Matthews FE. 1997. Mesothelioma mortality in Britain: patterns by birth cohort and occupation. *Ann Occup Hyg* 41(suppl 1):129-33.

Hodgson JT, Darnton A. 2000. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann Occup Hyg* 44:565-601.

Langer AM. 2003. Reduction of the biological potential of chrysotile asbestos arising from conditions of service on brake pads. *Regulatory Toxicology and Pharmacology* 38:71-77

McDonald AD, McDonald JC. 1980. Malignant Mesothelioma in North America. *Cancer* 46:1650-6.

Milham S, Ossiander E. 2001. Occupational mortality in Washington State 1950-1999. Washington State Department of Health.

Pukkala E, Saarni H. 1996. Cancer incidence among Finnish seafarers, 1967-92. *Cancer Causes Control* 7(2):231-9.

Saarni H, Pentti J, Pukkala E. 2002. Cancer at sea: a case-control study among male Finnish seafarers. *Occup Environ Med* 59(9):613-9.

Sakai K, Hisanaga N, Shibata E, Ono Y, Takeuchi Y. 2006. Asbestos exposures during reprocessing of automobile brakes and clutches. *Int J Occup Environ Health*. 12(2):95-105.

Teschke K, Morgan MS, Checkoway H, Franklin G, Spinelli JJ, van Belle G, Weiss NS. 1997. Mesothelioma surveillance to locate sources of exposure to asbestos. *Can J Public Health* 88:163-8.

Teta MJ, Lewinsohn HC, Meigs JW, Vidone RA, Mowad LZ, Flannery JT. 1983. Mesothelioma in Connecticut, 1955-1977. Occupational and geographic associations. *J Occup Med* 25:749-56.

Woitowitz HJ., Rödelsperger K. 1994. Mesothelioma among car mechanics? *Ann Occup Hyg* 38:635-8.

Yeung P., Patience K., Apthorpe L., Willcocks D. 1999. An Australian study to evaluate worker exposure to chrysotile in the automotive service industry. *Appl Occup Environ Hyg*. 14(7):448-57.

Sheehy JW, Cooper TC, O'Brien DM, McGlothlin JD, Froehlich PA. 1989. Control of asbestos exposure during brake drum service. National Institute for Occupational Safety and Health, Public Health Service, Centers for Disease Control. US Department of Health and Human Services, August.

Rodelsperger K, Jahn B, Bruckel J, Manke J, Paur R, Woitowitz H-J. 1986. Asbestos dust exposure during brake repair. *Am J Ind Med* 10(1):63-72.

Roberts DR, Zumwalde RD. 1982. Industrial hygiene summary report of asbestos exposure assessment for brake mechanics, Report no. IWS-32-4. National Institute for Occupational Safety and Health, Public Health Service, Centers for Disease Control. US Department of Health and Human Services.

Plato N, Tornling G, Hogsted C et al. (1995) An index of past asbestos exposure as applied to car and bus mechanics. *Ann Occup Hyg*; 39: 441-54.

Nicholson WJ, Daum SM, Lorimer WV, et al. (1984) Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos. Cincinnati, OH: U.S. Department of Commerce, National

Institute for Occupational Safety and Health; 1984. Contract 210-77-0199 PB83-220897

Roberts DR. Industrial Hygiene Report: Asbestos at Allied Brake Shop, Cincinnati, Ohio. Cincinnati, OH: Industrial Health Section, Division of Surveillance, Hazard Occupations, and Field Studies, National Institute of Occupational Safety and Health; 1980.

Roberts DR. Industrial Hygiene Report—Asbestos: Reading Brake and Alignment Service, Reading, Ohio. Cincinnati, OH: Industrial Hygiene Section, Industry-wide Studies Branch, Division of Surveillance, Hazard Occupations, and Field Studies, National Institute of Occupational Safety and Health; 1980.

Johnson P, Zumwalde RD, Roberts D. Industrial Hygiene Assessment of Seven Brake Servicing Facilities. Cincinnati, OH: National Institute of Occupational Safety and Health, Division of Surveillance, Hazard Evaluations, and Field Studies, Industry-wide Studies Branch, Industrial Hygiene Section; 1979.
Kauppinen T, Korhonen K. 1987. Exposure to asbestos during brake maintenance of automotive vehicles by different methods. *Am Ind Hyg Assoc J* 48(5):499–504.

Moore LL. Asbestos exposure associated with automotive brake repair in Pennsylvania. *Am Ind Hyg Assoc J* 1988;49:A12-A13.

Azari, Mansour R & Nasermoaddeli, Ali & Movahadi, Mohammad & Mehrabi, Yadollah & Hatami, Hossein & Soori, Hamid & Moshfegh, Elaheh & Ramazni, Behnam. (2010). Risk assessment of lung cancer and asbestosis in workers exposed to asbestos fibers in brake shoe factory in Iran. *Industrial health*, 48.

Berman DW. (2010). Reply to Letter to the Editor: "Comparing milled fiber, Quebec ore, and textile factory dust: Has another piece of the asbestos puzzle fallen into place? By D. Wayne Berman." *Crit Rev Toxicol* 40(8): 752-757.

Berman DW. (2010). Comparing milled fiber, Quebec ore, and textile factory dust: Has another piece of the asbestos puzzle fallen into place? *Crit Rev Toxicol* 42:151–188.

Rolland, P., Gramond, C., Lacourt, A., Astoul, P., Chamming's, S., Ducamp, S., Frenay, C., Galateau-Salle, F., Ilg, A. G. S., Imbernon, E., Le Stang, N., Pairon, J. C., Goldberg, M., Brochard, P. and for the PNSM Study Group (2010), Occupations and industries in France at high risk for pleural mesothelioma: A population-based case–control study (1998–2002).

Roggli, VL., Gibbs, AR., Attanoos, R., Churg, A., Popper, H., Cagle, P., Corrin, B., Franks, TJ., Galateau-Salle, F., Galvin, J., Hasleton, PS., Henderson, DW. and Honma, K. (2010) Pathology of Asbestosis—An Update of the Diagnostic

Criteria: Report of the Asbestosis Committee of the College of American Pathologists and Pulmonary Pathology Society. Archives of Pathology & Laboratory Medicine: March 2010, Vol. 134, No. 3, pp. 462-480.

McDonald, J.C. Epidemiology of Malignant Mesothelioma—An Outline. Ann Occup Hyg (2010) 54(8): 851-857 first published online November 8, 2010

Marinaccio A, Binazzi A, Di Marzio D, Scarselli A, Verardo M, Mirabelli D, Gennaro V, Mensi C, Merler E, De Zotti R, Mangone L, Chellini E, Pascucci C, Ascoli V, Menegozzo S, Cavone D, Cauzillo G, Nicita C, Melis M, Iavicoli S. Incidence of extrapleural malignant mesothelioma and asbestos exposure, from the Italian national register. Occup Environ Med. 2010 Nov;67(11):760-5. Epub 2010 Aug 25. PubMed PMID: 20798014.

Lemen, RA., Anderson, H., Bailar, JC, Bingham, E., Castleman, B. Exposure science will not increase protection of workers from asbestos-caused diseases: NIOSH fails to provide needed public health action and leadership. (2011) Journal of Exposure Science and Environmental Epidemiology (2011) 21, 114–115

Lemen RA., Chrysotile asbestos and mesothelioma. Environ Health Perspect. 2010 Jul;118(7):A282.

Donaldson K, Oberdörster G. Continued controversy on chrysotile biopersistence. Int J Occup Environ Health. 2011 Jan-Mar;17(1):98-9

Finkelstein, MM. and Meisenkothen C. Malignant Mesothelioma Among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos Ann Occup Hyg (2010) 54(6): 692-696

Dement, JM., Stayner, LT., Letter to the Editor: "Comparing milled fiber, Quebec ore, and textile factory dust: Has another piece of the asbestos puzzle fallen into place?" by D. Wayne Berman. Crit Rev Toxicol 2010 Sep; 40(8):749-51.

Coggon D, Harris EC, Brown T, Rice S, Palmer KT. Work-related mortality in England and Wales, 1979-2000. Occup Environ Med. 2010 Dec;67(12):816-22.

Rushton, L & Bagga, S & Bevan, R & Brown, T P & Cherrie, J W & Holmes, P & Fortunato, L & Slack, R & Van Tongeren, M & Young, C & Hutchings, S J. (2010). Occupation and cancer in Britain. British journal of cancer, 102. 1428-1437.

Berman, DW. Apples to Apples: The Origin and Magnitude of Differences in Asbestos Cancer Risk Estimates Derived Using Varying Protocols. (2011) Risk Analysis: an official publication of the Society for Risk Analysis. 2011. 1-19.