IN THE SUPERIOR COURT OF THE STATE OF DELAWARE

IN AND FOR NEW CASTLE COUNTY

IN RE: ASBESTOS LITIGATION) C.A. NO. 77C-ASB-2

Date Submitted: March 1, 2006
Date Decided: May 9, 2006
Revised and Corrected: June 9, 2006

Upon Consideration of Defendant DaimlerChysler Corporation's Motion in Limine to Exclude Expert Testimony that Friction Products Cause Asbestosis, Lung Cancer and Mesothelioma.
DENIED.

MEMORANDUM OPINION

Ian Connor Bifferato, Esquire, BIFFERATO, GENTILOTTI, BIDEN & BALICK, Wilmington, Delaware. Thomas C. Crumplar, Esquire, JACOBS & CRUMPLAR, Wilmington, Delaware. Christopher J. Panatier, Esquire and John J. Spillane, Esquire, BARON & BUDD, P.C., Dallas, Texas. William A. Kohlburn, Esquire and Michael J. Angelides, Esquire, SIMMONS COOPER LLC, East Alton, Illinois. Cameron R. Waddell, Esquire, LEBLANC & WADDELL, LLP, Baton Rouge, Louisiana. Attorneys for plaintiffs.

Somers S. Price, Jr., Esquire and James M. Kron, Esquire, POTTER, ANDERSON & CORROON, LLP, Wilmington, Delaware. Samuel L. Tarry, Jr., Esquire, MCGUIREWOODS, LLP, Richmond, Virginia. Attorneys for Defendant DaimlerChrysler Corporation.

Paul A. Bradley, Esquire, MCCARTER & ENGLISH, LLP, Wilmington, Delaware. Steven T. Johnson, Esquire, PEPPLE, JOHNSON, CANTU & SCHMIDT, PLC, Seattle, Washington. Attorneys for Defendant Owens-Illinois, Inc.

SLIGHTS, J.

I.

By motion *in limine*, Defendant, DaimlerChrysler Corporation ("Chrysler"), invites the Court to weigh in on a debate that has been hosted in state trial courts across the country in which Chrysler or similarly situated defendants have called the question of whether plaintiffs in asbestos litigation can reliably establish a medical or scientific link between exposure to so-called automotive friction products (or "friction products") and asbestos disease. This debate has been led by highly skilled attorneys on both sides who have presented well-credentialed experts to provide the evidentiary platform upon which their legal arguments have rested. In Delaware, the presentation fit the parameters of Delaware's version of the *Daubert* standard,[1] and occurred over the course of a multi-day evidentiary hearing followed by extensive briefing and oral argument. The matter is now ripe for decision.

Chrysler's motion mounts a focused attack on the plaintiffs' causation experts. It has specifically declined to go after the plaintiffs' "specific causation" case -- that is, Chrysler has left for another day the question of whether individual plaintiffs can establish that their exposure to particular friction products has, in fact, proximately caused their injury. Instead, Chrysler has focused on plaintiffs' "general causation"

---

[1] *See Daubert v. Merrell Dow Pharm., Inc.,* 509 U.S. 137 (1999); *M.G. Bancorporation, Inc. v. Le Beau*, 737 A.2d 513, 521 (Del. 1999).

case. Specifically, Chrysler contends that plaintiffs cannot establish that any exposure to asbestos-containing friction products increases the risk of developing any asbestos-related disease. Chrysler challenges the methodology employed by the plaintiffs' experts and is particularly critical of their failure to account for compelling epidemiological evidence that, according to Chrysler, conclusively establishes a negative association between exposure to friction products and asbestos disease. Although plaintiffs have questioned this framing of the issue - - specifically, the extent to which Chrysler can meaningfully challenge general causation given the purportedly conclusive evidence that chrysotile asbestos causes disease - - it was clear during the hearing that both parties were litigating the reliability of plaintiffs' medical and scientific evidence that exposure to *friction products* (not just chrysotile) increases the risk of contracting an asbestos-related disease.[2]

Needless to say, at trial, plaintiffs will bear the burden of proving all of the requisite elements of their claims by a preponderance of the evidence, including

[2]The disagreement with respect to the scope of the motion, as best as the Court can tell, derives from the parties' differing views of Chrysler's concession for purposes of this motion that exposure to chrysotile asbestos can cause disease. Since chrysotile asbestos is a component part of friction products, plaintiffs contend that Chrysler's concession with respect to chrysotile asbestos ends the general causation inquiry. According to Chrysler, the manufacturing process for friction products renders the chrysotile asbestos contained therein harmless. Thus, their concession regarding general causation in the context of chrysotile asbestos does not answer the general causation question in the context of friction products. That question, according to Chrysler, is still very much in dispute. Where appropriate, the Court will address these differing characterizations of the issues as it analyzes the merits of the motion.

proximate causation. *Vel non* plaintiffs can carry that burden is not an issue joined in the motion *sub judice.*[3] For now, the only question before the Court is whether the plaintiffs have carried their burden to establish that their expert opinions on friction products causation are sufficiently reliable to allow them to be presented to the jury. For the reasons that follow, the Court finds plaintiffs' medical and scientific evidence that exposure to friction products increases the risk of contracting an asbestos-related disease is sufficiently reliable to pass through the *Daubert* filter, and that the proper manner by which to challenge the plaintiffs' theories, and to expose their weaknesses, is through vigorous cross examination of the plaintiffs' expert witnesses. Accordingly, Chrysler's motion *in limine* to exclude plaintiffs' friction product

[3]In its pre-hearing brief, Chrysler seemed to suggest that on this motion the Court should decide the degree of "relative risk" (or the quantum of causation proof) the plaintiffs would have to present at trial in order to meet their burden of proving proximate cause. *See* Docket Item ("D.I.") 2168 at 6-7. At the post-hearing oral argument, however, counsel for Chrysler stated: "One of the things, perhaps thankfully, that I don't think the this Court has to grapple with is how much risk equals proximate cause in this proceeding." D.I. 2686, 01/20/06 at 20. After carefully considering the motion, the Court agrees with Chrysler's post-hearing view of the issues *sub judice*. The quantum of evidence required to make a *prima facie* case for proximate cause will, if necessary, be decided on another day.

causation witnesses must be **DENIED.**[4]

II.

Several plaintiffs in Delaware's "asbestos litigation" allege that they have contracted, or in the future will contract, various asbestos-related diseases as a proximate result of exposure to asbestos-containing brake and clutch products associated with Chrysler. Specifically, these plaintiffs allege that as a result of working with Chrysler's "automotive friction products" - - namely brakes and clutches - - they have contracted either asbestosis, mesothelioma or lung cancer.

For purposes of the motion *sub judice*, the parties agree that the automotive friction products at issue contained chrysotile asbestos and that exposure to chrysotile asbestos can cause asbestos-related diseases.[5] According to Chrysler, however, asbestos-containing products are not generic and, as a matter of science (and law), the

---

[4]By order dated September 16, 2005, the Court invited all parties to the asbestos litigation to indicate either their support for or opposition to Chrysler's motion and to indicate their desire to participate in the hearing on the motion. The Court further advised that its decision on Chrysler's motion would be binding upon all parties, plaintiffs and defendants, who may be asserting or defending claims involving automotive friction products or prosecuting cross claims against such parties. All joinders and oppositions have been considered in connection with this opinion and will be mentioned specifically where appropriate. *See* D.I. 2248 (9/16/05 Order at ¶¶ 2, 9); D.I. 2255-2258, 2260-2266, 2268-2270 (joinders in support of motion); D.I. 2271 (joinder in opposition to motion).

[5]As the name would suggest, friction brakes and clutches generate friction while in operation. The friction, in turn, generates heat. The asbestos used in the friction products minimizes the heat that is generated and retards the conduction of the heat to other automotive components. Def.'s Ex. ("DX") 296 at 110-12.

Court cannot assume that the physical and chemical propensities of all asbestos products and asbestos fibers are the same. In the case of automotive friction products, Chrysler contends that the most reliable science suggests that the chrysotile asbestos contained therein, after undergoing the manufacturing process and after use, is not dangerous. Plaintiffs disagree and contend they have presented evidence sufficient to establish for *Daubert* purposes that chrysotile is chrysotile - - its toxic properties are not significantly affected by the use to which it is put. These competing views set the stage for the first, and perhaps most important, factual dispute raised by the motion: what is the product at issue here - chrysotile or automotive friction products that contain chrysotile? Is there a difference and, if so, is the difference meaningful in the *Daubert* context?

Next, the evidentiary presentation focused on the scientific method and whether the plaintiffs' expert opinions withstand scrutiny when measured against settled scientific methodology. While the evidence in this regard was, at times, quite dense and, from a lay perspective, quite technical, in the Court's mind, the fact question boiled down to this: in the face of what Chrysler has characterized as settled epidemiological evidence that exposure to automotive friction products does not increase the risk of disease, can the plaintiffs' non-epidemiology experts reach

reliable conclusions to the contrary and stay true to the scientific method?[6] Stated differently, when considering the link between toxic exposure and human disease, does epidemiological evidence, when it exists, trump all other science for purposes of testing the reliability of a scientific hypothesis and assessing the reliability of a scientific conclusion? Not surprisingly, Chrysler's evidence said yes; plaintiffs' evidence said no.

Before turning to the legal analysis, the Court will review the evidence presented with respect to these factual issues. In doing so, the Court will review briefly the experts who testified and the substance of their opinions. When appropriate, the Court will highlight the exhibits, including the scientific studies and literature upon which the experts relied. The Court notes that the evidentiary record is extensive and contains much more information than will be recounted here. The Court has attempted to cull through the evidence to get to what is, in the Court's view, at the core of this controversy.

A. Chrysotile Asbestos

The Court learned about six different forms of asbestos: amosite, crocididolite, actinolite, tremolite, anthophyllite and chrysotile. Of these forms, chrysotile is the

[6]As discussed below, this factual issue also implicates a legal issue: Is epidemiological evidence of association required as a matter law?

most widely used in commercial applications. Chrysotile is considered a "serpentine" form of the mineral asbestos, while the other five forms are characterized as "amphobiles." Serpentine fibers appear under the microscope as curvy and flexible. They are generally regarded to be more readily broken down once inhaled in the body. Amphobiles, on the other hand, are long thin fibers that are more rigid than chrysotile fibers. They are generally regarded to be more likely to remain in the body once inhaled.[7] Thus, it is generally accepted in the scientific community and among government regulators that amphibole fibers are more carcinogenic than serpentine (chrysotile) fibers.[8] Nevertheless, it is also generally recognized in the scientific community and by government regulators that exposure to *all* forms of asbestos, including chrysotile, can cause serious diseases including asbestosis, mesothelioma and lung cancer.[9]

---

[7] *See generally* Pls. Ex. ("PX") 205 at 48-52; D.I. 2683, 10/17/05 p.m. at 69-70; *Mechanisms of Fibre Carcinogenesis*, IARC PUBLICATIONS SCI, No. 140, at 1-10 (A.B. Kane, *et al.* eds., 1996) (referred to by defense counsel during closing argument without objection but apparently not formally moved into evidence).

[8] *See* DX 22, 10/17/05 p.m. at 75-77. *But see* PX 342 (Dr. Wagner was unable to confirm in animal tissue studies that amphobiles were more carcinogenic than serpentine fibers.).

[9] *See* PX 2, 3.

B. Friction Products

Chrysler contends that the preponderance of the evidence in the record demonstrates that the chrysotile asbestos contained in friction products is significantly different from unrefined chrysotile out of the mill.[10] As part of the manufacturing process for friction products, milled chrysotile asbestos is saturated with resin and then heated. According to Chrysler, this process is meant to bind the asbestos in a way that does not occur with asbestos that is not so refined.[11] Chrysler contends that once manufactured into friction products, chrysotile's mineral properties are altered in a manner that reduces its biological activity in the human body if inhaled.[12] Indeed, Chrysler maintains that, if inhaled, chrysotile from newly manufactured friction products will not cause disease.[13] Moreover, according to Chrysler, after friction products have been used and are ready to be replaced, the chrysotile that once was present has been broken down by intense heat and transformed into an amorphous product called forsterite.[14]

---

[10]For lack of a better term, the Court may refer to chrysotile that has not been subjected to the friction product manufacturing process as "unrefined chysotile."

[11]*See* DX 357; D.I. 2168, Ex. C, at 8.

[12]*See* D.I. 2168 at 14-21; DX 184; DX 372.

[13]*Id.*

[14]*Id.*

Based on the foregoing, Chrysler contends that the commercial or shade tree mechanic who installs or replaces an automotive friction product is exposed to little, if any, respirable asbestos.[15] New friction products are manufactured in a way that binds the asbestos such that it does not become airborne and any fibers that might be inhaled are not harmful. Used friction products yield an impotent, non-fibrous material.[16] Thus, Chrysler contends that friction products are safe when new, and safe after use.

C. The Products At Issue Are Automotive Friction Products

As stated, plaintiffs expended much effort, particularly in their written submissions, to characterize the product at issue here as chrysotile asbestos rather than automotive friction products. Their tactic is understandable. The scientific evidence is well settled, at least for *Daubert* purposes, that chrysotile causes asbestos diseases. According to the plaintiffs, then, the multi-day hearing and hundreds of exhibits that have been submitted to the Court were not necessary. This characterization of the record, however, ignores Chrysler's evidence that automotive friction products are different from unrefined chrysotile.[17] It also ignores the

---

[15] *Id.*

[16] *Id.*

[17] *See e.g.* PX 258.

parameters set for this *Daubert* proceeding in the proposed order submitted by the parties and signed by the Court prior to the hearing: "The issue to be considered by the Court under the Chrysler Motion is not whether chrysotile causes asbestos related diseases, but whether automotive friction products, which contain chrysotile asbestos, can cause Mesothelioma, Lung Cancer or Asbestosis."[18] The Court is satisfied that the products at issue here are automotive friction products, and that the plaintiffs' expert testimony regarding general causation should be considered in that context. Specifically, plaintiffs must demonstrate under the applicable *Daubert* standard the relevance and reliability of their experts' opinions that Chrysler's friction products cause disease.[19]

D. The Experts

The plaintiffs presented live testimony from four expert witnesses of different backgrounds and specialties within the scientific community. Each expert brought a different perspective to the causation question in order to reach the same

[18]D.I. 2248, 9/16/05 Order at ¶12. At the parties' request, this order also recognized that Chrysler did not intend its motion to address general causation as it relates to "asbestos related pleural disease." *Id.* at ¶11. Plaintiffs disagreed. *Id.* This is Chrysler's motion; it gets to determine what issues to raise and what issues not to raise in its own motion. If plaintiffs want an *in limine* ruling regarding asbestos related pleural disease, they can file an appropriate motion.

[19]Of course, as will be discussed below, by framing the issue in this manner, the Court is not foreclosing plaintiffs from demonstrating, through their experts, that the chrysotile in friction products maintains its carcinogenic properties after the manufacturing process such that the evidence of general causation with respect to chrysotile becomes relevant to the question of general causation with respect to friction products.

conclusion: exposure to automotive friction products increases one's risk to contract mesothelioma, lung cancer and asbestosis. Each of the experts had addressed this question in other proceedings. Indeed, given the extensive experience of both counsel and the experts with the issues and with each other, the direct and cross examinations of these witnesses often took on a familiar and congenial tone. There were no surprises.

1. Ronald F. Dodson, Ph.D.

Plaintiffs' first witness was Ronald F. Dodson, Ph.D., a researcher with an advanced degree in life sciences and a specialty in biological electron microscopy. He has studied and written about asbestos and asbestos diseases since 1977. Among his extensive writings is a book entitled "Asbestos Risk Assessment Epidemiology and Health Effects" of which he is the senior editor and the author of a chapter on "tissue and fiber burden analysis." He has been on the faculty of the Baylor College of Medicine and the University of Texas at Tyler. He has been a reviewer for several peer-reviewed journals in the fields of toxicology and occupational and environmental health. His work has evolved into a focus on asbestos and asbestos diseases. At the time of his testimony, he had just returned from Italy where he gave a presentation to the Collegium Ramazzini (the International Occupational Medicine Society) on the measurement of asbestos in tissue and the techniques and

instrumentation used to make such measurements. He has testified for plaintiffs and defendants in asbestos litigation.[20]

In the realm of asbestos, microscopy involves the study of human tissue and cell structure and the analysis of asbestos fibers under sensitive and powerful microscopes. Using his analysis of tissues under these microscopes, Dr. Dodson explained the various asbestos diseases and, particularly, the biological disease process. He also discussed the measurement of asbestos fibers, and chrysotile fibers in particular, including the means by which government regulatory bodies, such as the National Institute for Occupational Safety and Health ("NIOSH"), prescribe that fibers should be measured. He explained how the lung either purges or attempts to destroy harmful fibers and how certain fibers become trapped in the various structures of the lung, or are "relocated" to other structures in the body, and go on to cause disease there. According to Dr. Dodson, smaller asbestos fibers are more likely to be removed from the lung naturally, but even some very small fibers (less than 5 microns) can remain and cause disease (a theory tested and proven by researchers, including Pott and Stanton).[21]

---

[20]D.I. 2682, 10/17/05 a.m. at 43-61.

[21]*See* D.I. 2682, 10/17/05 a.m. at 133-35. Dr. Dodson discussed studies by Dr. Suzuki that supported the notion that short fibers can be carcinogenic. *See* PX 329, 330. It should be noted, however, that Dr. Dodson also acknowledged that there was disagreement in the scientific literature on this subject. D.I. 2683, 10/17/05 p.m. at 80-83. *See e.g.* DX 41, 293.

Dr. Dodson noted that government regulators, e.g., the Occupational Health and Safety Administration ("OSHA") and the Environmental Protection Agency ("EPA"), do not differentiate among the types of asbestos when addressing their propensity to cause disease.[22] Based on his own research and his review of the literature, he testified unequivocally that it is generally accepted within his field of science that chrysotile asbestos causes asbestosis, pleural plaquing (a scarring of the pleura of the lung), lung cancer and mesothelioma.[23]

With respect to automotive friction products, Dr. Dodson testified that the chrysotile contained in brake linings and clutches is no different than the chrysotile he has studied in other types of products.[24] Indeed, Dr. Dodson wrote a peer-reviewed paper in which he explained how he "washed" worn automotive clutches and looked at the surface debris under an electron microscope.[25] He found some short chrysotile fibers and "a considerable number" of long chrysotile fibers.[26] He then studied lung tissue of an individual "whose primary work activity had centered on clutch refabrication" and found asbestos fibers comparable to those he observed from

---

[22]D.I. 2682, 10/17/05 a.m. at 124.

[23]*Id.* at 76-79.

[24]D.I. 2683, 10/17/05 p.m. at 27.

[25]*See* DX 257, 258.

[26]D.I. 2683, 10/17/05 p.m. at 28-29.

the worn clutches.[27] He performed a similar experiment with new friction brakes and, again, reduced his findings to writing in a peer-reviewed paper.[28] In this instance, he was testing a hypothesis that friction products that have been bound in a matrix do not release respirable asbestos fibers.[29] After washing the brakes, among other materials (including the resin binding), he found respirable chrysotile asbestos fibers.[30] He also concluded that for mechanics and others who would grind or sand brakes - - common processes during brake replacement - - more respirable chrysotile fibers would be released.[31] Dr. Dodson testified that his findings were consistent with similar findings he has seen in the peer-reviewed scientific literature.[32]

Based on his research, and his review of the literature, Dr. Dodson opined that there is no reason to conclude that chrysotile asbestos from friction products is any less likely to cause disease than chrysotile asbestos used in other applications.[33] He

---

[27]PX 258.

[28]PX 167.

[29]D.I. 2683, 10/17/05 p.m. at 32. *See e.g.* DX 369, 370 (studies by Francis W. Weir Ph.D. *et al.* concluding that the resin binding in friction brakes substantially reduced the risk of inhaling respirable asbestos fibers).

[30]D.I. 2683, 10/17/05 p.m. at 35-37.

[31]*Id.* at 39.

[32]*Id.* at 39-41*; See e.g.* PX 305, 306.

[33]D.I. 2683, 10/17/05 p.m. at 44-46.

also opined that short chrysotile fibers, like those found in friction products, are capable of causing disease, including lung cancer, asbestosis, and mesothelioma.[34] His views in this regard have been confirmed in studies performed by others and reported in the peer-reviewed literature.[35]

2. Samuel Hammar, M.D.

Plaintiffs' next witness was Samual Hammar, M.D., a pathologist board certified in anatomic and clinical pathology. He explained that anatomic pathology involves the diagnosis of disease by analyzing cells, tissues and organs. He has been involved in the study of asbestos and asbestos diseases since the early 1970's. His practice is in close proximity to a Naval Shipyard where historically a significant number of workers were exposed to asbestos and have contracted disease. Indeed, his home town of Bremerton, Washington has the dubious distinction of maintaining the highest incidence of mesothelioma per capita in the United States. Currently, he sees in his practice between 10 and 20 new cases of mesothelioma per year, which is extraordinary given the rareness of the disease. In total, he has seen over 4,000 cases of mesothelioma and has conducted approximately 475 autopsies on patients with mesothelioma.

---

[34] *Id.* at 103-06.

[35] *Id.*

In addition to his clinical practice of pathology, Dr. Hammar has also conducted extensive research relating to asbestos diseases. This research has included a study of the "morphology" of mesothelioma, meaning the disease process as observed under the microscope. He has worked with Dr. Dodson on such issues as fiber analysis in lung tissue and the concentration of asbestos in various tissues, particularly involving the pleura. He has published extensively in the peer-reviewed literature (in excess of 100 articles, book chapters, etc.). Not surprisingly, in all of his scientific endeavors, he maintains that he stays true to generally accepted scientific and medical principles and methodologies.[36]

Dr. Hammar testified that the methodologies he employs in his practice and in his research are not any different than those he employs when consulted as a forensic expert.[37] The only difference, according to Dr. Hammar, is that he actually can learn more of the clinical history of a patient in litigation cases than he typically learns in his practice with respect to patients he is treating. He likens the process of reaching a diagnosis in the clinical setting to the process of reaching a causation opinion in the litigation setting. In both instances, he looks at the entire picture; he will not consider

[36]D.I. 2342, 10/18/05 a.m. at 3-12.

[37]*Id.* at 18.

one piece of information or source of data to the exclusion of others.[38]

Having said this, Dr. Hammar endorsed the so-called "evidence-based medicine" movement both with respect to making causation determinations and reaching treatment decisions. Under this movement, the physician considers all available evidence, but does so in a hierarchy in recognition that some evidence is more probative of the question under study than other evidence. In this hierarchy, he testified that clinical trials are likely most reliable. Such studies are, for obvious reasons, not available in the asbestos context - - doctors will not introduce asbestos into their patients to monitor the reactions. Next in line would be observational studies, otherwise known as epidemiology, where, in the asbestos context, patients who are believed to have been exposed to asbestos are monitored in a controlled fashion either prospectively or retrospectively. After epidemiology would come the individual observations of a physician, also known as case reports.[39]

After addressing scientific methodologies, Dr. Hammar turned to the disease process that follows after asbestos fibers are inhaled into the lungs. In this regard, he did not hesitate to opine that short asbestos fibers cause disease in the lungs just as

---

[38] *Id.* at 19-21.

[39] *Id.* at 72-75.

long asbestos fibers do.[40] He explained that when an individual is exposed to different asbestos fibers there is no way to determine specifically which specific fibers caused disease. According to Dr. Hammar, in cumulative exposure cases, scientists take the view that all of the exposures contribute to the end result of disease.[41] Each fiber of chrysotile, therefore, if inhaled as part of a cumulative exposure, would contribute to any asbestos related disease that ultimately occurs.[42]

Dr. Hammar testified that the chrysotile in friction products is not unique.[43] Such fibers can cause scarring and other injury to the lung in the same manner that chrysotile fibers from other products cause injury (including asbestosis and cancer).[44] According to Dr. Hammar, "there is a definite dose response relationship to every asbestos related disease," meaning that "the more your exposed to, the higher your risk would be of developing any of these diseases."[45] This applies to all exposures, including exposures to friction products.

---

[40] *Id.* at 25.

[41] *Id.* at 30.

[42] *Id.*

[43] *Id.* at 30, 32.

[44] *Id.* at 34-35.

[45] *Id.* at 36.

Dr. Hammar disputes Chrysler's contention that friction products cannot cause disease on several grounds. First, he contends that friction products contain chrysotile and there is no reason to believe that chrysotile in a friction product is any different from chrysotile used in other applications. Thus, the abundant evidence that chrysotile causes disease must be considered here. Second, he referred to several reports in the peer-reviewed literature that document cases where individuals, whose only known exposure to asbestos was exposure to friction products, went on to contract asbestos-related disease.[46] Third, Dr. Hammar himself has observed in practice approximately ten patients who, by their history, were exposed only to automotive friction products and then contracted asbestos related disease.[47] Finally, he considered the Australian "mesothelioma registry," a government sponsored compilation of data relating to asbestos exposures among various industries and occupations within Australia. In that data, the researchers found that a scientifically significant number of individuals exposed to brakes, either through manufacture or repair, contracted mesothelioma.[48] Indeed, according to Dr. Hammar's interpretation of the data, brake mechanics were "one of the major categories of individuals

---

[46] *Id.* at 38-39. *See e.g.* PX 232, 233, 234.

[47] D.I. 2342, 10/18/05 a.m. at 40-41.

[48] *Id.* at 42-44. *See also* PX 254 (report by Dr. James Leigh summarizing the findings in the Australian Mesothelioma Registry).

[reported in the study] who developed mesothelioma in Australia."[49]

Based on his consideration of each of the factors mentioned above, Dr. Hammar has concluded that exposure to automotive friction products can increase the risk of contracting asbestos-related diseases.[50] This conclusion is shared by others in his scientific community and reported in the peer-reviewed literature.[51] It is also supported by Dr. Henderson's analysis of the Australian data as submitted in his report to the World Trade Organization entitled "European Communities - Measures Affecting Asbestos and Asbestos-Containing Products."[52] In that report, Dr.

---

[49]D.I. 2342, 10/18/05 a.m. at 56. During cross examination, Dr. Hammar acknowledged that this epidemiological study was of primary importance to him in reaching his opinion. He also admitted that in previous testimony he has stated that without the Australian study he would not reach the conclusion that exposure to friction products increases the risk of disease. He acknowledged that there were weaknesses in the Australian study, including its failure to consider latency periods and later-discovered errors in assessing other exposures. Finally, he acknowledged that the epidemiological conclusions of Drs. Henderson and Leigh were published in a journal that "was not afraid to publish provocative papers (according to the authors)." *Id.* at 82-121. Nevertheless, he testified that "despite the epidemiology, it would be very difficult for me to conclude from those case reports that asbestos were not [sic] the cause [of the diseases reported there]. I believe in the epidemiology. I think it's very important.... I'm just saying that sometimes you can't always prove things from an epidemiologic studies [sic]." *Id.* at 85, 87. *See also id.* at 144-45 (if presented with a case where an individual's only known exposure to asbestos was automotive friction products, even in the absence of epidemiological support, his "conclusion would have been that the person's mesothelioma was caused by chrysotile asbestos.").

[50]Dr. Hammar appears to have phrased his conclusions in this manner because, according to his testimony, the concept of an increase in the risk of developing a disease is the appropriate characterization of the onset of disease when scientists don't fully comprehend the mechanism of a particular disease, such as cancer and mesothelioma. D.I. 2342, 10/18/05 a.m. at 66-68.

[51]*See* PX 248, 254.

[52]D.I. 2342, 10/18/05 a.m. at 60-61. *See also* PX 223.

Henderson states: "automotive mechanics constitute a large population of workers potentially exposed to chrysotile derived from brake linings."[53] He goes on to state that the Australian data reported several cases of "mesotheliomas among brake mechanics with no other exposures to asbestos," and that the rate of exposure among such individuals was "substantially above the upper limit of the estimated background rate...."[54] Significantly, Dr. Henderson reached his conclusion even after considering the fact the some chrysotile will convert to forsterite during use of the friction product, and that most of the chrysotile fibers released from friction products are "short-length fibers."[55]

Dr. Hammar believes that the epidemiological evidence is in conflict,[56] and that it is not appropriate to place too much weight on the epidemiological studies to the exclusion of other reliable sources of scientific evidence.[57] Indeed, with respect specifically to mesothelioma, where the disease is so rare and the background incidence rate is basically zero, Dr. Hammar is of the view that epidemiological

---

[53]PX 223 at 300.

[54]*Id.* at 303.

[55]*Id.* at 300-301. These considerations are significant, of course, because both of these features of friction products have been offered by Chrysler as bases to contrast friction products from unrefined chrysotile.

[56]D.I. 2342, 10/18/05 a.m. at 85-86.

[57]*Id.* at 128-33.

evidence is of less relevance because the background rate of incidence is so low. In this regard, he testified:

Q. Okay. I'd like to follow up a little bit very briefly on the background incidence. Can you tell the Court what the largest cohort ever studied of asbestos exposed people is?

A. The asbestos insulators, members of the heat and frost insulators union.

Q. And how many people have been in that cohort?

A. 17,800.

Q. What's the expected incidence of mesothelioma going against the general public in a cohort of 17,800 people?

A. Basically, the experience there is that they have 10 percent of the people have died from mesothelioma. The expected rate of death in mesothelioma in generally [sic] is basically zero.

Q. So if it was – the expected rate would be zero in a cohort of 17,800 people?

A. That is correct.

Q. What would the expected rate be in 178,000 people?

A. Zero.

Q. Okay. What would the expected rate be in 500,000 people?

A. Zero.

Q. Does this, in your opinion, in terms of your review of epidemiology, this fact that we're dealing with a disease that is so

rare that the background rate approaches zero, does that have an impact on the use of epidemiology in the evaluation of that disease?

A. Sure, it does.

Q. Okay. And not to belabor the obvious, but could you explain briefly why that is?

A. Because if you think that mesothelioma has a certain rate of existence as a spontaneous tumor in the background population, that's going to really change your data with respect to if there was an increased number of cases versus a lack of increased number of cases in a population of people that are being studied, especially in a case – control type studies where there are a small number of people.

I'm not an epidemiologist, but that's going to markedly skew the data. Because if you say you have a number of three cases per million people as a background, and that's how many cases you find in a certain number of people that you calculate that, then there's going to be no increase in incidence. To me that doesn't mean, based on what I think is correct, that those people with mesothelioma was not caused by asbestos.[58]

When asked specifically why he disagreed with Chrysler's contention that its friction products could not be a source of disease, he stated:

Well, ... I think they are wrong with respect to stating that chrysotile doesn't cause mesothelioma in a setting where people are exposed from brake products. Because we know that chrysotile in generally [sic] does cause mesothelioma. It causes it in most settings. It could can [sic] cause it in different types of situations. There wouldn't had been [sic] any reason to exclude the possibility that chrysotile could not cause

[58]D.I. 2342, 10/18/05 a.m. at 138-40.

> mesothelioma in an individual who was exposed in basically any way, whether brakes, whether it was a clock, whether it's whatever.
>
> * * *
>
> I think there is overwhelming epidemiological proof that chrysotile causes mesothelioma. I don't think it's even a question here. I guess in the brake mechanic issue is that based on the epidemiologic studies there seems to be a question as to whether chrysotile exposure causes an increased incidence of mesothelioma based on what has been published. And that if you – and here – and I'm not an epidemiologist. And here is where my problem comes with that is just what I've said, is that so here I see a case of a person who we have good evidence that was the only exposure they had. They have elevated concentrations of asbestos in their lung. They have elevated concentrations of asbestos in their pleura. You don't find anything else. How can you not say that that was caused by asbestos?[59]

3. Richard A. Lemen, Ph.D.

Plaintiffs' third witness was Richard A. Lemen, Ph.D., an epidemiologist and industrial hygienist who has studied asbestos for more than thirty years. His past experience includes appointments as the Assistant Director and later Deputy Director ("number two person") of NIOSH and former Assistant Surgeon General of the United States. He has taught epidemiology at Emory University and has published extensively in the field in numerous peer-reviewed journals. He currently works as a private consultant in the fields of epidemiology, occupational health and public

[59] *Id.* at 133-34, 159-60.

health.[60]

Dr. Lemen talked in general about the practice of epidemiology and the general scientific concepts employed in this field. First, he explained that epidemiologists deal with concepts of association and risk which could translate into or assist in a general causation analysis. They do not address issues relating to specific causation. In determining issues of general association or increased risk with regard to the toxicity of a particular substance, epidemiologists consider several perspectives including the mechanism of injury, the biological activity of the substance, toxicology and pathology studies, and animal experimentation. They also consider "case reports," which are reports of individual cases. These reports are of more significance in cases of rare "signature" or "sentinel" diseases like mesothelioma. In such instances, the "case report" offers significant guidance because the general association between the substance (e.g. asbestos) and the disease (e.g. mesothelioma) is well established in the scientific community.[61] Indeed, Dr. Lemen does not agree with the notion, at least with respect to the question *sub judice*, that epidemiology trumps the other scientific data.[62] As discussed below, Dr. Lemen is of the view that

---

[60]D.I. 2684, 10/18/05 p.m. at 5-10.

[61]*Id.* at 12-15, 29-31. *See also* D.I. 2343, 10/19/05 a.m. at 14-17.

[62]D.I. 2684, 10/18/05 p.m. at 96-99.

the epidemiological evidence on this subject is equivocal and that other data, including that which is contained in the case reports, offer a more definitive answer to the general causation question.[63]

Dr. Lemen addressed the issue of "relative risk" previously discussed by Dr. Hammar. He confirmed that the relative risk for mesothelioma was zero, meaning there is virtually no background incidence rate for the disease in an unexposed population.[64] He then explained that "attributable risk" is the amount of disease among exposed workers that can be attributed to a particular exposure above the background or relative risk.[65] He explained that "confounders" are factors that can alter the results of a study unless they are properly controlled for, such as other exposures, limited inclusion criteria, and latency of disease.[66]

In the specific context of asbestos, Dr. Lemen discussed certain criteria that have been developed in the scientific community to help identify association. For

---

[63] *Id.*

[64] *Id.* at 22 (referring to studies by Dr. Selikoff). *See e.g.* PX 312, 313, 316-18. He acknowledged that the information available about the background incidence is really just an estimate and that there are reports of mesothelioma occurring without any known exposure to asbestos. *See* D.I. 2343, 10/19/05 a.m. at 85-87; D.I. 2685, 10/19/05 p.m. at 12. *See also* PX 226 (Dr. Hillerdal discussing the low background rate for mesothelioma and the low threshold of exposure needed to acquire the disease).

[65] D.I. 2684, 10/18/05 p.m. at 23.

[66] *Id.* at 16, 55-57.

instance, he discussed the "Helsinki Criteria," a compilation prepared by scientists at the request of the Finish government to look at "attribution criteria" for asbestos that would assist the clinician in making specific causation determinations.[67] These criteria identify occupational history and history of exposure as the most reliable means to make an association between asbestos exposure and asbestos disease.[68] The "Bradford Hill Criteria," promulgated by a noted scientist, Sir Austin Bradford Hill, offer nine factors that epidemiologists should consider when looking at associations, including "plausibility, coherence, strength of association, consistency of observed associations, biological gradient, experiment, analogy, specialty of the association, and temporality."[69] None of these criteria stand alone; they are all important when considering the issues of association and risk.[70] On the question of whether chrystotile causes asbestos-related diseases, Dr. Lemen opined that each of the

---

[67] *Id.* at 33-34.

[68] *Id.*

[69] *Id.* at 34-40. In brief, "plausibility" refers to consistency of one's causation theory with other theories of causation; "coherence" is consistency with known facts; "strength of association" refers to the power and integrity of the study; "consistency" refers to whether the results can be observed repeatedly under different circumstances; "biological gradient" asks whether a dose response relationship exists; "experimental evidence" refers to data from animal, laboratory, and pathology studies and controlled clinical trials; "analogy" looks at other epidemiological and other scientific studies to determine if analogous substances cause analogous effects; "specificity" asks if each cause has a single or logically related similar effect; and "temporality" considers whether the cause precedes the effect. *Id.*

[70] *Id.* at 35.

Bradford Hill criteria support the conclusion that the association is valid.[71] Government agencies, including NIOSH, OSHA, EPA, the United States Consumer Products Safety Commission, the World Trade Organzation, and the World Health Organization, among others, all have issued statements in which they have concluded that chrysotile causes mesothelioma.[72]

As a general matter, Dr. Lemen agreed that asbestos diseases are dose response diseases - - the more exposure, the greater the risk of developing disease.[73] He addressed the issue of the relative carcinogenicity of amphiboles and serpentines, and agreed that amphiboles are generally regarded as being more potent than serpentine fibers.[74] Nevertheless, he opined that there is, in fact, a synergistic interaction that occurs in multiple exposures of different types of fibers which results in a phenomenon where "you get more disease when you have mixed exposure than you do from the individual exposures to each of the fiber types alone."[75] According to

---

[71]*Id.* at 39-43. Dr. Lemen opined that the Bradford Hill criteria would be applied the same for mesothelioma, lung cancer and asbestosis.

[72]*Id.* at 48-50.

[73]*Id.* at 70. *See also* PX 238 (Dr. Iwatsubo *et al.* discuss the dose response relationship in the context of mixed exposures and cumulative doses).

[74]Dr. Lemen made it clear, in his view, that this is so only with respect to mesothelioma. There is, according to Dr. Lemen, no basis to conclude that chrysotile is less toxic with respect to lung cancer and asbestosis. *See* D.I. 2684, 10/18/05 p.m. at 44.

[75]*Id.* at 42-43.

Dr. Lemen, this conclusion is confirmed in the epidemiology literature.[76] Thus, even in cases of mixed exposures, Dr. Lemen is of the opinion that exposure to chrysotile can be separated as a cause of all asbestos diseases. And, once again, he is satisfied that the epidemiological evidence supports this conclusion.[77]

With respect to automotive friction products specifically, Dr. Lemen has reported on more than 165 published cases that support the conclusion that exposure to friction products can cause mesothelioma.[78] He is of the view that exposure to chrysotile from friction products is no different than exposure to other chrysotile-containing products. According to Dr. Lemen, "it is not the job title, it is the exposure."[79] In his direct testimony, Dr. Lemen explained that as he has studied the

---

[76] *Id.* *See also* PX 327 (referencing other studies).

[77] D.I. 2684, 10/18/05 p.m. at 52.

[78] *See* PX 0001.

[79] *Id.* Dr. Lemen reviewed the literature where reports of individuals working with or around friction products have recounted exposure to chrysotile during both installation and removal as a result of grinding the product for fit, chiseling the product during removal, and cleaning out the brake drum after removal and before installation of the new product. *Id.* at 77-78, 84-90. Most fibers released in these processes are short fibers, but some are long fibers. D.I. 2343, 10/19/05 a.m. at 18-37. Both can cause disease. *Id.* He also testified that while some chrysotile used in friction products can be converted into forsterite during use, there is still "quite a bit of chrysotile left." D.I. 2684, 10/18/05 p.m. at 78-79. Finally, he acknowledged that applying heat (such as during the manufacturing process of friction products) may change the biological activity of chrysotile. Nevertheless, he testified that there is no evidence that these changes render the mineral inert or even minimize its toxicity. Some studies show the contrary is true, i.e., heated chrysotile causes a greater cytotoxic reaction than unheated chrysotile. D.I. 2343, 10/19/05 a.m. at 117-120; DX 357 (Valentine suggests that forsterite may be "more cytotoxic than the parent chrysotile."). It should be noted that Dr. Lemen is familiar with the manufacture and use of friction products because he has studied both in the field. *See* D.I. 2343, 10/19/05 a.m. at 103-10; PX 299 (reporting the results of this study).

science relating to asbestos and asbestos related disease over the years, including the epidemiological data, he has reached the conclusion that exposure to friction products causes all of the known asbestos-related diseases, particularly mesothelioma, lung cancer, asbestosis, and other asbestos-related non-malignant diseases. In this regard, he places particular emphasis on the works of Drs. Selikoff and Nicholson, and the conclusions and precautionary recommendations published by NIOSH and distributed widely throughout the automotive industry.[80] He explained that his opinion extends to both single occupation cases, i.e., cases where the only exposure to asbestos was as a brake mechanic, and cumulative exposure cases, i.e., cases where a person is exposed to asbestos not only as a mechanic through work with (or around) friction products but also through other occupations and work with (or around) other types of asbestos-containing products.[81]

---

[80]D.I. 2684, 10/18/05 p.m. at 59-60, 63-66. *See also* D.I. 2343, 10/19/05 a.m. at 39-41 (summarizing his opinions). *See e.g.* PX 284 (Nicholson/Selikoff study); PX 46 (NIOSH recommendations); PX 209, 262 (other studies).

[81]D.I. 2684, 10/18/05 p.m. at 73-74. *See also* PX 283 (study by Drs. Nicholson, Perkel and Selikoff addressing cumulative exposure in those that have worked as automobile mechanics, noting that it is not unusual to find cumulative exposure since automobile mechanics often move on to other trades); PX 223 (Dr. Henderson concludes: "It is not valid to point to one exposure among the others and incriminate it as the sole cause of a mesothelioma, with exoneration of the other exposures."). Dr. Lemen also extrapolated data from two peer-reviewed studies to conclude that, in certain instances, brake mechanics can be exposed to high levels of chrysotile in a manner that "[could] cause a significant amount of disease." D.I. 2684, 10/18/05 p.m. at 90-94. *See also* D.I. 2685, 10/19/05 p.m. at 5-9 (discussing the process on cross examination).

On cross examination, Dr. Lemen acknowledged prior deposition testimony in which he opined that he lacked the scientific evidence to support a conclusion that exposure to friction products causes lung cancer. He then amended his testimony in this proceeding by admitting that "there is [not] enough evidence to convince [him] that ... an increased risk [of] disease [lung cancer] ... [has] been demonstrated."[82]

Finally, Dr. Lemen evaluated the body of epidemiological studies that have considered the association between friction products and asbestos disease. He noted that each of the studies he has reviewed suffer from various confounders or limitations that affect the reliability of the results.[83] Consequently, Dr. Lemen has published a paper in which he concludes that the epidemiological evidence on exposure to friction products and risk of disease is "equivocal."[84] For instance, some of the studies suffer from inadequate follow up of the cohort, others don't focus on brake mechanics or at least don't make that focus clear in the report, others fail to consider the impact of other exposures (or lack thereof), others demonstrate

---

[82]D.I. 2343, 10/19/05 a.m. at 142-43.

[83]This observation applies as well to the Leigh Henderson Australian study upon which Dr. Hammar relies and upon which Dr. Lemen relies in part to support the proposition that epidemiological studies have shown an increased risk of disease upon exposure to friction products. D.I. 2684, 10/18/05 p.m. at 108-09; D.I. 2343, 10/19/05 a.m. at 64-84 (Dr. Lemen addresses some of the shortcomings in the Australian study on cross examination).

[84]D.I. 2684, 10/18/05 p.m. at 96. Importantly, Dr. Lemen does not believe the mere fact that scientists may disagree renders their work "unscientific." Indeed, such disagreements are critical to the evolution of science. D.I. 2343, 10/19/05 a.m. at 47.

inadequate occupational and medical histories of the study participants, others fail to consider non-occupational exposures (e.g. the prolific "shade tree" mechanic), and others fail to consider the latency of asbestos-related diseases.[85]

Dr. Lemen also discussed the limitations of the meta-analysis performed by Chrysler's expert, Dr. Goodman.[86] In particular, Dr. Lemen notes that Dr. Goodman's study does not adequately account for the significant confounders that plague each of the studies that form the bases of his conclusion.[87] He is also critical of Dr. Goodman's failure to attempt to correlate his statistical findings with the contrary findings of clinicians who have studied the question.[88]

In summary, Dr. Lemen does not believe the epidemiological studies that have specifically addressed the issue offer definitive guidance one way or the other on whether exposure to friction products increases the risk of contracting asbestos-related disease. Thus, scientists must look to the entire body of scientific evidence on the issue. In this case, after reviewing the entire body of scientific evidence, he

---

[85] *See* D.I. 2684, 10/18/05 p.m. at 95-110, 116-128; D.I. 2343, 10/19/05 a.m. at 3-13, 126-132.

[86] As will be discussed below, Dr. Goodman performed a meta-analysis (a comparison) of 17 epidemiological studies to conclude that exposure to friction products does not increase the risk of contracting mesothelioma or lung cancer.

[87] D.I. 2684, 10/18/05 p.m. at 113-14; D.I. 2343, 10/19/05 a.m. at 99-100. *See also* PX 168 (Dr. Bailar discusses the problems and shortcomings of meta-analyses generally in the New England Journal of Medicine).

[88] *Id.* *See also* D.I. 2343, 10/19/05 a.m. at 52-63.

has reached the conclusion that a positive association does exist with respect to exposure to friction products and the development of mesothelioma and, perhaps, asbestosis. Not so with lung cancer.[89]

4. Arthur L. Frank, M.D., Ph.D.

Plaintiffs' final witness was Dr. Arthur Frank, an occupational medicine physician with a doctorate degree in biomedical sciences. He has studied and treated asbestos diseases for more than thirty years, including at Mount Sinai Hospital, a hot bed for asbestos research. While there, Dr. Frank worked closely with Dr. Irving Selikoff, a leading asbestos researcher. He has taught at Mount Sinai and at the University of Kentucky and University of Texas at Tyler medical schools. He currently teaches at Drexel University's School of Public Health. Through all of this time, Dr. Frank has seen and evaluated patients both as an occupational medicine physician and as a general internist. He has taught residents. For the past fifteen

[89]The final state of the record is not entirely clear with respect to Dr. Lemen's view on general causation of friction products and asbestosis. *See* D.I. 2343, 10/19/05 a.m. at 142 ("I still don't have enough information on lung cancer. And I think the risk of lung cancer and asbestosis are probably fairly low. I think there is evidence that exposures occurring to brake workers are high enough to produce those diseases, but I think that at this time, we don't have sufficient data on the lung cancer issue, and I think the disease of most concern is the mesothelioma."); *Id.* at 144-48 (admitting that there are no studies indicating that brake workers have received adequate exposure to asbestos to contract asbestosis). He later clarified that because asbestosis is a cumulative injury process, often occurring after mixed exposures, it cannot be said that exposure to friction products does not contribute to the "overall burden that leads to the resultant disease." D.I. 2685, 10/19/05 p.m. at 22-23.

years, he has trained physicians in China in the field of occupational medicine. He has also written extensively on the subjects of asbestos and asbestos diseases.[90]

Dr. Frank testified that different occupations have different exposure levels to asbestos. The fact that brake workers may have a lower exposure level relative to an insulation worker does not mean that the brake worker's exposure is insignificant or incapable of causing disease. Dr. Selikoff made this fundamental but very important observation in 1964 and it continues to hold true today.[91] This is the essence of the so-called "dose response relationship." Asbestosis has a relatively high dose response relationship. But mesothelioma and lung cancer, according to Dr. Frank, have a very low dose response relationship to the point where animal studies have shown that "as [little] as one day of exposure was enough to produce both lung cancers and mesotheliomas."[92] According to Dr. Frank, "each and every exposure contributes to the disease."[93] Indeed, according to Dr. Frank, in the causation analysis, if you left out any one exposure among all of the exposures, you would have to leave out all exposures. There is no way to pinpoint one exposure in the causation

---

[90]*Id.* at 52-65.

[91]*Id.* at 66-67. *See* PX 313, 283 (Dr. Selikoff's writings on this subject).

[92]D.I. 2685, 10/19/05 p.m. at 73.

[93]*Id.* at 77.

analysis to the exclusion of others.[94]

Dr. Frank has worked with brake mechanics throughout his career, both in his clinical practice and in the research he has conducted.[95] He reiterated the results of the Nicholson study. According to Dr. Frank, Nicholson's epidemiological research demonstrated that brake mechanics were exposed, in some instances, to high levels of chrysotile asbestos and that "a greater prevalence of X-ray abnormalities is found among garage mechanics who repaired brakes than among blue collar controls or garage workers who do not engage in brake or auto body work."[96] For his part, Dr. Frank is of the view that brake workers are exposed to "inhalable [asbestos] dust, not as much as an insulator, [but] certainly more than the background levels we all have."[97] And, according to Dr. Frank, "we know" that the fibers contained within that dust are capable of causing injury. This, in Dr. Frank's mind, ends the general causation inquiry.[98]

---

[94]*Id.* at 77-82. *See also* PX 351 (World Trade Organization notes that each exposure must be considered and none excluded as a cause of mesothelioma).

[95]D.I. 2685, 10/19/05 p.m. at 86; PX 262 (although not listed as a contributor to the article, Dr. Frank assisted Lorimer with his research on asbestos exposure of brake mechanics). On cross examination, he acknowledged some of the study's limitations. D.I. 2685, 10/19/05 p.m. at 113-122.

[96]PX 284 at 93. *See also* D.I. 2685, 10/19/05 p.m. at 122-29.

[97]D.I. 2685, 10/19/05 p.m. at 96-97, 104-05.

[98]*Id.* at 97, 100.

5. Michael Goodman, M.D.

Chrysler's first and only witness was Dr. Michael Goodman, a physician and epidemiologist. He is currently on the faculty of the Department of Epidemiology at the Emory University School of Public Health. He is board certified in pediatrics. He has trained in the field of preventive medicine and has earned board certification. In connection with his training in preventive medicine, he developed a specialty as a "physician epidemiologist." Now he works as a private consultant in the field of epidemiology with a concentration on cancer epidemiology. He began asbestos research in 1997 and has since prepared research papers that reflect the results of meta-analysis he has performed in connection with diseases in asbestos-exposed occupational cohorts, including an analysis of the risk of developing lung cancer and mesothelioma in individuals involved in motor-vehicle repair.[99]

Dr. Goodman first addressed, in general terms, the body of scientific research that has been conducted on asbestos and asbestos diseases. He acknowledged that the prevailing scientific data reveals that "asbestos is irrefutably, in many circumstances, causally related to mesothelioma, lung cancer and, of course, asbestosis."[100] This

---

[99]D.I. 2344, 10/20/05 a.m. at 5-17.

[100]*Id.* at 18.

connection was established in human epidemiological studies.[101] He addressed how the "grade" of the asbestos, meaning the length of the fibers, is a factor that impacts the biological effects of asbestos. Friction products contain the highest grade of chrysotile which means that the shortest fibers are used.[102] He discussed a publication of the International Agency For Research on Cancer ("IARC") which provides methodologies to determine if a substance can be classified as a carcinogen. The IARC standards require "sufficient evidence" of carcinogenicity in humans before a substance will fit within IARC's "category" of carcinogens.[103] Under the IARC standards, Dr. Goodman opined that both asbestos generally and chrysotile specifically are carcinogenic.[104]

Dr. Goodman discussed the scientific method and its application in epidemiology. He explained, in general, that the scientific method involves generating a hypothesis, testing the hypothesis through research with appropriate structure and controls for comparison, then reaching a conclusion regarding the validity of the hypothesis. In the context of cancer research, hypothesis often are

---

[101] *Id.*

[102] *Id.* at 20-21.

[103] "Sufficient evidence" is not defined.

[104] D.I. 2344, 10/20/05 a.m. at 26-27.

generated from animal studies or case reports where "observations" of activity are recorded. In a cohort study, the epidemiologist will then test the hypothesis by attempting to establish "frequency;" how common is the activity that forms the basis of the observation? This is tested with a test group and a control group made up of a similar cohort. The researcher then compares the results after a defined period of time. To ensure consistency, Dr. Goodman stated that the test should be repeated or, at least, the researcher should look for similar tests conducted by other researchers.

In the case control study, the research begins with the activity (e.g. the disease) in question. The subjects with the disease are interviewed to determine how many of them fit the profile of the hypothesis (e.g. how many were exposed to the suspected carcinogen). A control group is established of healthy subjects to determine if they were exposed to the suspected carcinogen. The results are then compared. The researchers analyze the data to determine the relative risk, and they calculate a confidence interval to confirm that the results did not occur by chance. If there is a "positive association" (e.g. the suspected substance does cause the suspected disease) or a "negative association" (e.g. the suspected substance does not cause disease), then the hypothesis is either proven or not proven.[105] If the hypothesis embodied in the case report is not proven by the epidemiological study, then the "case report

[105] *Id.* at 29-41.

become[s] virtually useless."[106]

Dr. Goodman discussed the epidemiological evidence that has been developed regarding the association between asbestos exposure and mesothelioma. He identified insulators, shipyard workers, boiler makers, and plumbers and pipe fitters as occupations where the epidemiological evidence reveals that there is an association between exposure to asbestos in these cohorts and the onset of mesothelioma. These studies reveal consistent results and are reliable.[107] As to automobile mechanics, however, Dr. Goodman testified that no epidemiologic study "found an increase in -- a significant increase in risk of mesothelioma among people engaged in motor vehicle repair."[108] This does not mean that auto mechanics will not contract mesothelioma. According to Dr. Goodman, no occupation as large as the population of auto mechanics will have zero risk of contracting the disease even though the disease is very rare. Dr. Goodman's opinion is that there is no statistically significant

---

[106]D.I. 2345, 10/20/05 p.m. at 4. Dr. Goodman is referring here to the hierarchy of evidence to be considered in making a causation determination. He opines that epidemiology is the "main tool" to assess causation in human beings and that when such evidence is conclusive, other evidence further down the reliability chain becomes less relevant if not irrelevant. *Id.* at 14-16. If, however, epidemiology studies are not available or are inadequate, then "it is reasonable to use evidence from circumstances that are as close as possible, because you don't have any better data." *Id.* at 27. *See also id.* at 92 (referring to IARC standards regarding the hierarchy of scientific evidence in cancer research).

[107]D.I. 2344, 10/20/05 a.m. at 57-62.

[108]*Id.* at 62.

increase in the relative risk of contracting mesothelioma while working as an auto mechanic.[109]

Dr. Goodman reviewed eighteen different epidemiologic studies to reach his conclusion. He explained the methodology employed in each study, explained the confounders and the manner in which the researchers attempted to control for confounders, highlighted the strengths and acknowledged the weaknesses of each study, and then explained the results.[110] In one study, for instance, the researchers had earlier prepared a case study in which they concluded, from four auto mechanic patients they had seen in their practice, that exposure to friction products increases the risk of mesothelioma. Each mechanic had contracted mesothelioma with no other known exposures. The researchers then went on to test that hypothesis with an epidemiological control study that revealed "there is no evidence that car mechanics are exposed to an increased risk of mesothelioma even if they do brake repairs."[111] This result was consistent throughout all of the studies that Dr. Goodman reviewed.[112]

---

[109] *Id.* at 62-64. This is so even though Dr. Goodman acknowledges that friction fibers release respirable asbestos fibers. D.I. 2345, 10/20/05 p.m. at 17. According to Dr. Goodman, exposure to friction product chrysotile fibers is quantitatively and qualitatively different than the types of exposures reported in the epidemiology literature demonstrating a positive association between occupational exposure and disease. *Id.* at 82.

[110] D.I. 2344, 10/20/05 a.m. at 75-122.

[111] *Id.* at 96. *See also* DX 374 (epidemiology study); DX 375 (case report).

[112] D.I. 2344, 10/20/05 a.m. at 122.

The studies also revealed no support for the proposition that people who worked around friction products longer were at an increased risk of disease (i.e. no dose response relationship),[113] nor was there evidence of a synergistic relationship between chrysotile released from friction products and other types of fibers that might be inhaled during other exposures.[114]

Dr. Goodman also explained the means by which he assembled these studies in his meta-analysis. Specifically, he explained how he separated the studies by tiers in which he placed similar studies (based on methodologies, cohorts, results, etc.) and then weighed them based on their reliability. His goal was to combine the studies in a manner that would, in essence, create a single study from which a single conclusion could be drawn - in this instance, the single conclusion was that exposure to friction products does not increase the risk of mesothelioma.[115]

The meta-analysis also addressed the association between friction products and lung cancer. Here again, Dr. Goodman's study revealed that the epidemiological evidence available in the literature consistently shows no significant increase in the

---

[113]*Id.* at 128.

[114]*Id.* at 128-29. *See also* DX 133 (Hessel report addressing, *inter alia*, the purported "synergistic" relationship between chrysotile from friction products and asbestos from other exposures).

[115]D.I. 2344, 10/20/05 a.m. at 130-35.

relative risk of developing lung cancer by working with friction products. The research reveals that auto mechanics are among the most prolific smokers of any studied occupation. Accordingly, the studies controlled for smoking and then found no increased risk of lung cancer from exposure to friction products alone.[116] Stated differently, if a smoker works in a garage, "the garage work [will not] add to that person's risk of lung cancer."[117]

Dr. Goodman acknowledged that none of the cohort or case control studies that he reviewed addressed the relative risk of contracting asbestosis from exposure to friction products.[118] Nevertheless, he determined that it was reasonable to conclude that epidemiology would not support this association because the dose required to induce disease was much higher with asbestosis than with mesothelioma or lung cancer.[119]

At the end of the day, Dr. Goodman's opinion is perhaps best summarized by his answers to the final questions posed to him (in the form of a hypothetical) during

---

[116] *Id.* at 137-44. *See also* DX 118 (Gustavsson cohort study addressing association between friction products and both mesothelioma and lung cancer).

[117] D.I. 2344, 10/20/05 a.m. at 149.

[118] *Cf.* DX 60 (PMR analysis of proportion mortality from asbestosis revealing no increase). According to Dr. Goodman, this conclusion does find support in cross sectional studies reported in the literature. D.I. 2344, 10/20/05 a.m. at 151.

[119] D.I. 2344, 10/20/05 a.m. at 150.

re-cross examination:

> Q. Dr. Goodman, I want you to assume a case of a person whose testimony taken during their lifetime is, I worked as a brake mechanic for several decades. I did several brake jobs pretty much every day. I blew out all the dust with an air hose every time I did a brake job. I wore no respiratory protection. That person is subsequently diagnosed, unequivocally, with mesothelioma - -
>
> A. Right.
>
> Q. His pleural tissue is sent to Dr. Dodson, who does tissue studies and finds greatly elevated levels of chrysotile asbestos and the person was questioned also during their lifetime and they said, this is my only known asbestos exposure, that's all I did my whole life, okay?
>
> A. Right
>
> Q. In your opinion, under those circumstances, what would be - would asbestos be a cause of that person's mesothelioma?
>
> A. No, I cannot accept that in the presence of existing data.
>
> Q. And that's because of your opinion that the epidemiological data in this situation trumps whatever the findings and circumstances are?
>
> A. It's not my opinion. You know, that's how you do [it].[120]

[120]D.I. 2345, 10/20/05 p.m. at 95-96.

III.

"No one will deny that the law should in some way effectively use expert knowledge wherever it will aid in settling disputes. The only question is as to how it can do so best."[121] Judge Quillen, in *Minner v. Amer. Mort. & Guar. Co.,*[122] used this basic yet sage observation from Learned Hand as a springboard to launch a thorough and thoughtful review of the use of experts in the courtroom and the evolution of the legal standards by which the admissibility of expert testimony has been measured. The Court will not attempt to recreate Judge Quillen's celebrated exposition on expert testimony here but, instead, will commend it as required reading to all who wish to understand the perspective from which this Court will view so-called *Daubert* motions, and the process by which such motions will be addressed. Suffice it to say, the import of Judge Quillen's review is that, despite a history of skepticism, trial courts now encourage the use of expert testimony if it will be of assistance to the trier of fact and if the opinions of the expert are reliable and rest on "good grounds."[123] But the expert's access to the courtroom is not unfettered. "The polestar must always be scientific or other validity and the evidentiary relevance and

---

[121]Learned Hand, *Historical and Practical Considerations Regarding Expert Testimony*, 15 Harv. L. Rev. 40 (1901).

[122]791 A.2d 826, 833 (Del. Super. Ct. 2000).

[123]*Id.* at 841.

reliability of the principles that underlie a proposed submission."[124]

A prominent feature of modern civil litigation is the central role that science and other technical disciplines play in the adversarial search for the truth.[125] In recognition of this phenomenon, the Federal Rules of Evidence, and now Delaware's Uniform Rules of Evidence, provide:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, training or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.[126]

Prior to *Daubert,* the Delaware Supreme Court recognized the importance of the Rules of Evidence in determining the admissibility of expert testimony, and identified several factors to guide the trial courts in determining when to allow an expert opinion to reach the jury:

1) The expert witness is qualified (D.R.E. 702);
2) The evidence is otherwise admissible, relevant, and reliable (D.R.E. 401 and 402);
3) The bases for the opinion are those reasonably relied upon by experts in the field (D.R.E. 703);

---

[124]*Id.* at 843.

[125]Steven J. Breyer, *Introduction to Reference Manual on Scientific Evidence*, Fed. Jud. Ctr. 2d ed., at 2 (2000)(hereinafter "Reference Manual").

[126]*See* D.R.E. 702 ("Rule 702").

4) The specialized knowledge being offered will assist the trier of fact to understand the evidence or determine a fact in issue (D.R.E. 702); and
5) The evidence does not create unfair prejudice, confuse the issues, or mislead the jury (D.R.E. 403).[127]

Then, in 1999, the Delaware Supreme Court explicitly adopted *Daubert* as the law of this state in recognition that our rules of evidence mirrored the federal counterparts upon which *Daubert* was decided.[128] Thus, "under *Daubert*, *Kumho*, and *M.G. Bancorporation*,[129] the Trial Judge acts as the gatekeeper to ensure that the scientific testimony is not only relevant but reliable."[130] As the trial court performs this function, it must be mindful not only of the factors offered by *Nelson,* but also of the similar guidance offered by *Daubert* in the form of non-exclusive factors for consideration, including: (1) whether the technique or scientific knowledge has been tested or can be tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) the known or potential rate of error and the control standards for the technique's operation; and (4) whether the technique has gained

[127]*Minner*, 791 A.2d at 842, 843 (citing *Nelson v. State*, 628 A.2d 69, 74 (Del. 1993)).

[128]*M.G. Bancorporation*, 737 A.2d 513 at 521.

[129]*See Daubert,* 509 U.S. 579 (1993); *Kumho Tire Co., Ltd. v. Carmichael,* 526 U.S. 137 (1999); *M.G. Bancorporation, Inc.*, 737 A.2d 513 (Del. 1999).

[130]*Minner*, 791 A.2d at 843.

general acceptance.[131] These factors do not function as a "definitive checklist or test."[132] Rather, courts should apply the factors, as set forth in both *Nelson* and *Daubert*, in a flexible manner that takes into account the particular specialty of the expert under review and the particular facts of the underlying case.[133]

At its core, *Daubert* dictates that Rule 702 is the governing standard for the admissibility of scientific evidence by specifying that "*if scientific*, technical, or other specialized knowledge *will assist the trier of fact* to understand the evidence or to determine a fact in issue," then the expert "may testify thereto."[134] The *Daubert* interpretation of the phrase "scientific knowledge" in Rule 702 is the genesis of the so-called "reliability" requirement. The adjective "scientific" linked with "knowledge" "implies a grounding in the methods and procedures of science."[135] And "knowledge" is more than unsupported beliefs; it must be derived from supportable facts.[136] Although scientific opinions need not be "[held] to a certainty" to be offered

---

[131] *Daubert*, 509 U.S. at 593-594.

[132] *Kumho,* 526 U.S. at 150 (quoting *Daubert*, 509 U.S. at 593).

[133] *Id.* at 152.

[134] *Id.* (emphasis in original)

[135] *Daubert*, 509 U.S. at 590.

[136] *Id.* (The Court quoted the definition of "knowledge" from WEBSTER'S THIRD NEW INTERNATIONAL DICTIONARY 1252 (1986) noting that the term "applies to any body of ideas inferred from such facts or accepted as truths on good grounds.").

at trial, they must be grounded in the scientific method to qualify as "scientific knowledge."[137]

Rule 702 also requires that expert testimony be relevant by requiring that it "assist the trier of fact to understand the evidence or to determine a fact in issue."[138] If proffered testimony is not related to the case, then it will not aid in clarifying a contested fact and is, therefore, not relevant.[139] Accordingly, the "helpfulness" standard requires that evidence have "a valid scientific connection to the pertinent inquiry as a precondition to admissibility."[140] *Daubert* characterized this requirement as one of "fit."[141]

In conducting the *Daubert* analysis, the court cannot lose sight of the fact that competing interests are at stake. As Judge Quillen observed:

> *Daubert* is a two-sided coin. On the one side, it is expansive, rejecting the exclusivity of the "general acceptance" requirement; on the other side, it is restrictive, with a focus on the Trial Judge's responsibility as a gatekeeper on reliability.[142]

---

[137] *Id.*

[138] *Id.* at 591.

[139] *Id.*

[140] *Id.* at 592.

[141] *Id.* at 591.

[142] *Minner,* 791 A.2d at 841.

In this regard, commentators have noted that "[a]ny effort to bring better science into the courtroom must respect the jury's constitutionally specified role - even if doing so means that, from a scientific perspective, an incorrect result is sometimes produced."[143] *Daubert* emphasized that the filtering effect of the adversarial trial system should not be discounted. "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence."[144]

As the judge performs his role as gatekeeper, and attempts in that process to test the expert testimony against the standards of reliability and relevance as directed by *Daubert*, it cannot be forgotten at the end of the day that "[the] judge is not a scientist, and the courtroom is not a scientific laboratory."[145] Thus, although judges are expected to perform the role of "gatekeeper" under *Daubert*, there is no

---

[143]Reference Manual at 4-5. *See also M.G. Bancorporation*, 737 A.2d at 522 (emphasizing the "liberal thrust" of the Federal Rules of Evidence generally favoring the admissibility of evidence).

[144]*Daubert*, 509 U.S. at 596.

[145]Reference Manual at 2.

expectation that they will do so with scientific precision.[146]

Finally, it is appropriate to close the discussion of the *Daubert* standard of review by emphasizing that the impact of the court's *Daubert* analysis may well reach beyond the case in controversy. In the products liability context, an incorrect decision can either deprive a plaintiff of warranted compensation while discouraging other similarly situated individuals from trying to obtain compensation, or it can improperly impose liability in a manner that will cause the abandonment of an important product or technology.[147] Either result is unacceptable. The Court must tread carefully.

IV.

A. The Burden of Proof

There was some debate among the parties prior to the *Daubert* hearing as to which party would bear the burden of proof. Plaintiffs acknowledged that their experts were under scrutiny and that, in the typical case, they would bear the burden of proving the admissibility of their expert's testimony under *Daubert* standards by

---

[146]*Id*. at 4. *See also Bowen v. E.I. duPont DeNemours & Co.,* 2005 WL 1952859, at *8-9 (Del. Super. Ct. June 23, 2005)(citation omitted)("[I]t is not necessary that the judge decide the admissibility of scientific evidence with the degree of certainty required in scientific circles."); DAVID L. FAIGMAN ET AL., SCIENCE IN THE LAW, Preface at v (2002)("Judges and lawyers, in general, are not known for expertise in science and mathematics. ... Indeed, law students, as a group, seem peculiarly averse to math and science.").

[147]Reference Manual at 3.

a preponderance of the evidence.[148] They urged the Court to shift that burden to Chrysler, however, because Chrysler's motion, in essence, sought summary judgment on the question of causation. In addition, plaintiffs argued that since Chrysler acknowledged that chrysotile caused asbestos diseases, and that its friction products contained chrysotile, the burden of proving that friction products did not cause asbestos diseases should fall to Chrysler.[149] The Court disagreed.

Prior to the hearing, the Court advised the parties that plaintiffs would bear the burden of establishing the reliability of its expert testimony in this case just as it would in any other products liability case. There is simply no basis that the Court can discern, under these facts, that would justify requiring Chrysler to prove that plaintiffs' experts are unreliable.[150] *Daubert* confirmed that the burden was on the proffering party and Delaware case law has reiterated this point time and time

---

[148] *Minner*, 791 A.2d at 843 citing *National Bank of Commerce v. Dow Chem.* Co., 965 F. Supp. 1490, 1497 (E.D. Ark. 1996) *aff'd*, 133 F.3d 1132 (8th Cir. 1998)(The proponent of the proffered expert testimony bears the burden of establishing by a preponderance of the evidence the relevance and reliability of the evidence); *Schmaltz v. Norfolk & W. Ry. Co.*, 878 F. Supp. 1119, 1120 (N.D. Ill. 1995). *See also* Harvey Brown, *Procedural Issues Under Daubert*, 36 Hous. L. Rev. 1133, 1136 (1999)(citing cases).

[149] D.I. 2307 at 9-10.

[150] *See* FAIGMAN ET AL., *supra*, §1-3.1.2 at 16 (Acknowledging some confusion in the case law, the authors conclude: "[T]he question of who should have the burden of proof to show that proffered expert testimony is relevant and reliable is obvious: the proponent of evidence always bears the burden of persuading the court that the conditions for its admission are met.").

again.[151]

In establishing the scientific validity of expert testimony, the proponent's focus should be on the methodology applied by the expert rather than the conclusions he generates.[152] "Proponents do not need to demonstrate to the judge by a preponderance of the evidence that the assessments of their experts are correct, they only have to demonstrate by a preponderance of the evidence that their opinions are reliable."[153] When assessing whether the proponent has met its burden, the trial court does not choose between competing scientific theories, nor is it empowered to determine which theory is stronger.[154] *Daubert* requires only that the trial court determine whether the proponent of the evidence has demonstrated that scientific conclusions have been generated using sound and reliable approaches.[155] When a trial court determines that an expert's testimony is reliable, this does not mean that contradictory expert testimony by default is unreliable. *Daubert* permits testimony that is the

---

[151]*Daubert*, 509 U.S. at 592; *Minner*, 791 A.2d at 843 (citations ommitted).

[152]*Daubert*, 509 U.S. at 595.

[153]*In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 744 (3d Cir. 1994), *cert den., Gen. Elec. Co., et al. V. Ingram, et al.*, 513 U.S. 1190 (1995).

[154]*Minner*, 791 A.2d at 848 citing *Ruiz-Troche v. Pepsi Cola of Puerto Rico Bottling Co.*, 161 F.3d 77, 85 (1st Cir. 1998).

[155]*In re Paoli,* 35 F.3d at 744.

product of competing principles or methods in the same field of expertise.[156]

Chrysler's motion is narrowly drawn. It has not challenged the credentials of plaintiffs' experts, and for good reason. Their credentials are impeccable. It also has not challenged the relevance of the plaintiffs' experts' opinions. Accordingly, this is not a motion that requires the Court to determine if the experts' opinions "fit," as that term is used in *Daubert.*[157] The only issue to be decided by the Court is whether plaintiffs' experts have employed a reliable methodology in reaching the conclusions that they propose to offer to the jury at trial.[158]

B. Plaintiffs' Experts Survive the Daubert Challenge

After carefully reviewing the evidence, the Court is satisfied that the plaintiffs have carried their burden of proving the admissibility of their proffered expert testimony on general causation by a preponderance of the evidence under either of two analytical approaches.[159] Both are addressed below.

---

[156]*Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 160 (3d Cir. 1999)(expert testimony cannot be excluded simply because the expert uses one test rather than another, when both tests are accepted in the field and produce reliable results.).

[157]*Daubert*, 509 U.S. at 591.

[158]*See* D.I. 2686, 1/20/06 at 50-52.

[159]The Court notes that it has assessed the admissibility of expert testimony, not the admissibility of a theory. Plaintiffs may present their general causation evidence through the witnesses evaluated by the Court. This does not mean, however, that they may present their general causation theory through any expert witness of their choosing. The admissibility of expert testimony depends upon the qualifications and methodologies employed by the witness giving it.

1. The Similarity of Unrefined Chrysotile and Friction Products

As previously mentioned, the plaintiffs have taken the position in this proceeding that the Court need not evaluate the *Daubert* evidence that has been presented because we already know that friction products contain chrysotile, chrysotile causes disease and, therefore, friction products cause disease. In other words, Chrysler's admission that its products contain a known carcinogen ends the inquiry. The Court has rejected this approach and has found that plaintiffs must establish that their experts can reliably conclude that exposure to friction products increases the risk of contracting an asbestos-related disease. This does not, however, preclude the plaintiffs from attempting to carry this burden by presenting competent evidence that friction products, in certain circumstances, release respirable chrysotile fibers that are indistinguishable in size and other characteristics from unrefined chrysotile fibers. With this evidentiary predicate in hand, plaintiffs' experts can then turn to the scientific evidence, undisputed for purposes of this hearing, that chrysotile causes asbestosis, lung cancer, and mesothelioma. In the Court's view, this approach, if sufficiently supported by the evidence, would satisfy the reliability component of *Daubert*.[160]

---

[160] *See Minner*, 791 A.2d at 848 ("where [a] known cause exist[s], and the opinion is voiced that a similar factor present in a given case probably caused the illness, one must at least be tempted to let the injured party have his or her day in Court.").

On the question of the similarity of friction products to unrefined chrysotile, Dr. Dodson offered compelling and persuasive testimony. He wrote a peer-reviewed paper in which he described in detail his comparison of new and worn friction fibers with unrefined chrysotile under an electron microscope.[161] In the friction fibers, he found both short and long chrysotile fibers.[162] He also studied lung tissue of an individual "whose primary work activity had centered on clutch refabrication" and found chrysotile fibers comparable to those he observed from the worn clutches.[163] According to Dr. Dodson, his findings were consistent with similar findings he has seen in the peer-reviewed scientific literature.[164]

Dr. Dodson also concluded that a significant amount of respirable chrysotile would be relased during typical functions performed by the mechanic during a brake

---

[161] *See* PX 257, 258.

[162] D.I. 2683, 10/17/05 p.m. at 28-29. As to the so-called "Stanton hypothesis" that short fibers do not cause disease, Dr. Dodson testified that short chrysotile fibers have been found at the sites where tumors have developed. *Id.* at 99-100, 103-04. *See also* D.I. 2685 10/19/05 p.m. at 105-06 (Dr. Frank testified: "There is nothing that says that small fibers should be exonerated, that once it gets into the lungs it acts like a fiber that could come from any other product.").

[163] PX 258. *See also* PX 210 (Falgout study showing that chrysotile fibers are released from grinded brake shoes).

[164] D.I. 2683, 10/17/05 p.m. at 39-41. *See e.g.* PX 305, 306. The conclusion also appears, at first glance, to be consistent with some of Chrysler's own evidence. *See e.g.* D.I. 2390, Ex. B (Patent showing that friction brakes are not subject to heat over 350ºC in the curing that occurs during manufacturing process); DX 184 (Langer notes that temperatures between 810-820ºC "completely dehydroxylate chrysotile" and also referencing temperatures greater than 350ºC when other changes were detected to occur).

or clutch installation or replacement. He referred to studies by Langer and Rohl which found that a significant volume of fibers would be released during sanding, grinding and beveling of the brakes for installation, and "blowing out" brake drums during replacement.[165] From this peer-reviewed evidence, Dr. Dodson was able to opine that work with friction products in the garage setting would release sufficient chrysotile fibers to create a dose response for asbestosis and lung cancer. Since mesothelioma is less dependent upon a dose response, it follows that sufficient fibers would be released to increase the risk for contracting that disease as well.[166]

In addition to looking at the size and amount of chrysotile fibers released from friction products, Dr. Dodson also considered the surface characteristics of the fibers and concluded that there is no basis to distinguish the surface characteristics of

---

[165]D.I. 2683, 10/17/05 p.m. at 39-40. *See also* PX 252 (Langer study); PX 305, 306 (Rohl studies); PX 283 (Nicholson study); PX 23, 24, 28, 29 (Chrysler documents reflecting at least some appreciation that its friction products release sufficient quantities of respirable asbestos fibers potentially to cause a "hazard."). Even Dr. Goodman agrees that friction products can release respirable asbestos fibers. D.I. 2345, 10/20/05 p.m. at 17.

[166]D.I. 2673, 10/17/05 p.m. at 76, 79. *See also* PX 001 at 231-32 (Dr. Lemen's study reveals sufficient release of chrysotile during brake installation or repair to increase risk for mesothelioma); D.I. 2684, 10/18/05 p.m. at 60-61 (discussing NIOSH warning to brake workers regarding friction products after Dr. Selikoff reported troublesome exposure levels); PX 284 (Nicholson Selikoff study of health hazards in brake lining repair). This evidence reveals that work on friction products can release in excess of the 0.15 fibers per cc reported to be permissible background exposure levels by NIOSH in the early 1970's. In 1986, EPA followed with a bulletin warning mechanics that working on friction products can release "millions of asbestos fibers" and noted the risk of contracting mesothelioma from exposure. *See* PX 006.

friction fibers from those of other chrysotile fibers.[167] And, although Dr. Dodson acknowledged that he could not confirm what occurs biologically or chemically (i.e. surface charge or surface chemistry) when lung or pleura tissue comes into contact with a friction fiber,[168] even Chrysler concedes that "no one can describe the factors that make any fiber carcinogenic."[169] Thus, it is difficult to accept Chrysler's argument that the plaintiffs' experts' opinions are somehow unreliable simply because they are unable to do with friction product exposure that which the scientific community has been unable to do with respect to most other toxic exposures: that is, determine exactly how (biologically, chemically or otherwise) exposure to the toxic

[167] *See* D.I. 2682, 10/17/05 a.m. at 29 (Fibers inspected were "identifiable as chrysotile fibers"); *Id.* at 36-37 (chrysotile fibrils inspected from washing friction products were "evident morphologically as unaltered tubes."); *Id.* at 59 (he would have detected changes in surface characteristics under TEM microscopy).

[168] *See* D.I. 2683, 10/17/05 p.m. at 96.

[169] *See* D.I. 2526 at 1. *See also* D.I. 2683, 10/17/05 p.m. at 49-51 (Dr. Dodson explains the difficulties in reaching definitive conclusions regarding the biological and chemical mechanisms involved in the development of cancer related diseases, including lung cancer and mesothelioma); D.I. 2334, 10/20/05 a.m. at 20-21 (Dr. Goodman unable to say what morphologic characteristics of asbestos fibers contribute to their ability to cause disease); D.I. 2342, 10/18/05 a.m. at 33-34 (Dr. Hammar concurs that the exact biological activity that occurs when asbestos causes cancer is unknown).

substance causes disease.[170]

After reviewing Dr. Dodson's testimony, and the evidence to which he cites in support of his conclusions, the Court is satisfied that plaintiffs have demonstrated a sufficient basis to allow their experts to rely upon the body of scientific data that has been developed regarding the link between exposure to unrefined chrysotile and an increased risk to develop mesothelioma, lung cancer and asbestosis. Specifically, plaintiffs' experts may rely upon this body of evidence to support their conclusions that exposure to friction products increases the risk of developing these diseases. Chrysler, of course, may present its evidence to the contrary. Nothing here should be taken as a determination that one side's science on this point - - the similarity between friction fibers and chrysotile fibers - - is superior.[171]

Having concluded that Dr. Dodson has provided a bridge, grounded in reliable science, between the scientific data regarding the association between unrefined chrysotile and asbestos-related diseases and the association between friction products

---

[170]*See In Re Asbestos Litig.,* C.A. No. 2004-03964, Davis, J., at 10 (Tex. Dist. Ct. June 30, 2005)(in the now infamous "baseball opinion" in which Judge Davis heard a challenge to plaintiffs' causation experts in a chrysotile asbestos case and offered his opinion in baseball parlance, the court described the defendant's argument regarding the plaintiff's failure to describe how chrysotile causes disease as a "suicide squeeze" and, in rejecting the argument, noted the lack of a similar explanation with regard to "all other known carcinogens.").

[171]*See Minner*, 791 A.2d at 848 citing *Ruiz-Troche*, 161 F.3d at 85 (in a *Daubert* analysis the court does not determine if the proffered science is correct or which of competing scientific views is superior).

and asbestos-related diseases, the question still remains whether plaintiffs' other experts have appropriately relied upon this data and have otherwise practiced reliable science in reaching their opinions.

Dr. Hammar wasted little time in his testimony before he turned to the extensive scientific evidence that supports the hypothesis that chrysotile causes asbestos-related disease.[172] He then discussed how his own experience in his clinical pathology practice is consistent with this data by describing approximately ten cases of patients he has treated who were exposed to friction products and went on to contract an asbestos-related disease.[173] His clinical experiences have been confirmed by other clinicians who have reported such cases in the literature.[174] He also discussed animal studies where chrysotile was injected into rats and provoked an increase in the incidence of mesothelioma.[175] He discussed *in vitro* studies that suggest a link between chrysotile and asbestos diseases. In his view, his clinical

---

[172]D.I. 2342, 10/18/05 a.m. at 37-39, 133-34. The terms "asbestos-related disease" and "asbestos disease" were used throughout the proceedings without definition or precision. It was clear, however, that Dr. Hammar's reference to the term included asbestosis, lung cancer and mesothelioma. *Id.* at 23-36 (discussing each disease when describing the mechanism of injury after inhalation of chrysotile fibers).

[173]*Id.* at 40.

[174]*See e.g.* PX 232-34.

[175]D.I. 2342, 10/18/05 a.m. at 141-42.

experiences and research results find support in the epidemiology literature as well.[176]

Dr. Hammar's conclusions are soundly based in the scientific method, find support in the peer-reviewed literature, and appear to be sufficiently accepted by others in his field to carry an air of reliability. While they certainly offer room for challenge on cross examination, they pass through the *Daubert* filter and will be admissible at trial.

Dr. Lemen's opinions in this litigation have been summarized in a peer-reviewed article published in the American Journal of Industrial Medicine.[177] His use of the Bradford Hill criteria to reach his conclusion that exposure to friction products increases the risk of asbestos disease reflects an appreciation for and adherence to a sound scientific methodology. The scientific literature Dr. Lemen reviewed consistently reveals that exposure to chrysotile causes asbestosis, lung cancer, and mesothelioma.[178] This body of evidence includes epidemiology, case reports, including his own compilation of more than 165 cases of mesothelioma in end-product users of friction products, human tissue studies, and animal studies.[179] He is

---

[176] *Id.* at 42-44, 159-60.

[177] *See* PX 001.

[178] D.I. 2684, 10/18/05 p.m. at 40-50.

[179] *Id. See also* PX 353 (Yano epidemiological study showing association between exposure to chrysotile and mesothelioma); PX 295 (Piolatto and Silvestri study reaching similar conclusions); PX 186 (Cullen study reaching similar conclusions).

also of the opinion, based on Dr. Dodson's research and other peer-reviewed research, that there is no scientifically justifiable reason to exclude exposure to friction products from this comprehensive body of scientific evidence.[180] Thus, in his opinion, the scientific data that are available with respect to chrysotile's disease-causing propensities apply equally to friction products.

Based on Dr. Dodson's studies, and Dr. Lemen's own observations, the Court is satisfied that Dr. Lemen's reliance upon the substantial scientific evidence of the association between chrysotile and asbestos disease is sufficiently reliable to withstand scrutiny under *Daubert.*[181] There is, again, ample room for cross examination and the Court has little doubt that this will occur given the vigorous cross examination that was conducted during the *Daubert* hearing.

---

[180]D.I. 2684, 10/18/05 p.m. at 78-80; PX 001.

[181]The Court acknowledges Dr. Lemen's equivocal testimony regarding the association between friction products and lung cancer and asbestosis. D.I. 2343, 10/19/05 a.m. at 141-42. His reservations appeared to be based on some question in his mind whether the auto mechanic would be exposed to a sufficient amount of chrysotile to cause disease. *Id.* In other testimony, he stated that "there is evidence that exposures occurring to brake workers are high enough to produce those diseases." *Id.* at 142. He then concluded that in a "mixed exposure" context, where the brake mechanic has been exposed to asbestos in other occupational settings, the cumulative nature of the mixed exposure could lead to asbestosis and lung cancer. *See* D.I. 2684, 10/18/05 p.m. at 42-43; D.I. 2685, 10/19/05 p.m. at 22-23. The discrepancies in his testimony provide fodder for cross examination. They do not, however, mandate a wholesale striking of his testimony under *Daubert.* Assuming the Rule 26(b)(4) disclosure of his testimony addresses a general causal link between exposure to friction products and asbestosis, lung cancer and mesothelioma, he may address all three diseases in his testimony on behalf of the various plaintiffs who retain him.

To reach his general causation opinion, Dr. Frank first referred to the literature which, in his view, establishes that "there is nothing different about the asbestos in brake products."[182] He then relied upon his own observations as an occupational medicine physician, trained by one of the foremost experts in asbestos diseases, Irving Selikoff, M.D., and recounted his experience treating patients who were exposed to asbestos as brake mechanics and then went on to develop asbestosis. He opined that if these patients were exposed to enough asbestos to develop asbestosis, they were certainly at an increased risk for cancer (including lung cancer and mesothelioma).[183] Some of his observations in this regard were reported in a peer-reviewed study in which he participated.[184] Finally, Dr. Frank relied upon the extensive scientific data reporting the association between exposure to chrysotile and all asbestos related diseases.[185] Dr. Frank's approach reflects a sound methodology consistent with *Daubert's* "reliability" component.

---

[182]D.I. 2685, 10/19/05 p.m. at 100. *See also id.* at 96-97, 104-05; PX 284.

[183]D.I. 2685, 10/19/05 p.m. at 86-91.

[184]*See* PX 262.

[185]D.I. 2685, 10/19/05 p.m. at 97, 100. The Court notes that some of Dr. Frank's testimony crossed over into more specific causation testimony, including his testimony regarding multiple exposures to asbestos and his discussion of a single fiber/no-threshold theory of contracting disease. *See e.g. id.* at 82 ("So it's whatever asbestos you can identify as being above background that people get exposed to that allows you, I think, at the end of the day to say it is more likely than not that *in this case* asbestos caused the disease....")(emphasis supplied). This does not, however, diminish the admissibility of his general causation testimony.

The Court has concluded that Dr. Dodson has adequately established for *Daubert* purposes that the chrysotile contained in friction products is the same as chrysotile that has not been subjected to the friction product manufacturing processes. Accordingly, each of the plaintiffs' experts have appropriately relied upon the settled data generated by multiple scientific disciplines, including epidemiology, that establishes a scientifically significant positive association between exposure to chrysotile and asbestosis, lung cancer and mesothelioma. Their methodology was sound and their resulting conclusions sufficiently reliable to pass by Chrysler's *Daubert* challenge and through the gates of the courtroom.

2. Chrysler's Epidemiology Does Not "Trump" Plaintiffs' Proffered Science

a. Epidemiology Is Not Required As A Matter Of Law

Before turning to the evidentiary record on the question of whether plaintiffs must support their general causation case with epidemiology, it is appropriate first to consider the legal landscape in which the question must be considered. Courts from several jurisdictions have considered the role of epidemiology in the courtroom with mixed results. Some courts require epidemiology; others do not.[186] Under the

[186] *Compare Ferebee v. Chevron Chem. Co.*, 736 F.2d 1529 (D.C. Cir. 1984)(epidemiology not required) with *Brock v. Merrell Dow Pharm., Inc.*, 874 F.2d 307 (5th Cir.), *modified,* 884 F.2d 166 (5th Cir. 1989)(epidemiology required).

circumstances presented here, the Court is satisfied that the jurisdictions that have declined to adopt a hard and fast rule have endorsed the better and more practical view on the subject.

Both parties appear to like this court's decision in *Long v. Weider Nutrition Group, Inc.,*[187] so the Court will begin its legal analysis here. In *Long*, the court considered the admissibility of causation testimony in the context of plaintiff's claim that her father's use of an "ephedrine-alkaloid product" caused a sudden cardiac arrest and subsequent death. Plaintiff presented the testimony of an expert in toxicology and pathology to make the general causation connection. This expert had published case reports in peer-reviewed literature in which she concluded that taking ephedra was linked to cardiovascular stimulant effects resulting in serious injury or death. Defendants challenged this testimony under *Daubert* principally on the ground that the opinion was not supported by epidemiological studies.[188] The court rejected the *Daubert* challenge, holding:

> Although there is a split of authority, other jurisdictions have found that epidemiological studies are not necessary as a threshold for admitting an expert's opinion on causation. As a matter of public policy, courts should not be hampered in the search for the truth by the rigid proposition that no expert, however qualified, can reliably opine on the

---

[187]2004 WL 1543226 (Del. Super. Ct. June 25, 2004).

[188]*Id.* at *5.

> causal link between a toxic substance and injury without epidemiological studies conducted according to strict guidelines.... If a properly qualified medical expert performs a reliable differential diagnosis through which, to a reasonable degree of medical certainty, all other possible causes of the victims' condition can be eliminated, leaving only the toxic substance as the cause, a causation opinion based on that differential diagnosis should be admitted. In determining the reliability of expert testimony, it is within the discretion of the trial court to exclude evidence of causation based solely on the expert's evaluation of the case reports and differential diagnosis. Nevertheless, courts have admitted expert testimony when a differential diagnosis is supported by scientific and clinical studies linking the allegedly dangerous substance to harmful effects.[189]

Chrysler cites *Long* for the proposition that a medical expert may offer a differential diagnosis regarding causation only if all other possible causes have been ruled out to a reasonable degree of medical certainty. Otherwise, epidemiological evidence is required. Plaintiffs cite *Long* for the proposition that epidemiological studies are not, under any circumstance, *required* to establish general causation. Both parties have accurately quoted the opinion. Nevertheless, the Court reads *Long* as readily supporting the admission of plaintiffs' proffered expert testimony in this case. This is so not only because *Long* recognizes that epidemiology is not required, as a

---

[189] *Id.* at *6 (extensive citations omitted). *See also Ferebee*, 736 F.2d at 1535 ("Thus, a cause-effect relationship need not be clearly established by animal or epidemiological studies before a doctor can testify that, in his opinion, such a relationship exists."); *Bloomquist v. Wapello County,* 500 N.W.2d 1 (Iowa 1993)(court recognized as sufficient in toxic tort cases proof of causation based on traditional cause-and-effect testimony, such as by treating doctors; epidemiological evidence is helpful but not an absolute requirement in establishing causation). *Cf. Lee v. A.C. & S. Co., Inc.,* 542 A.2d 352 (Del. Super. Ct. 1987)(holding that epidemiology cannot address specific causation; a medical doctor must provide the causal link).

matter of law, to establish general causation in every case, but also because Chrysler's interpretation of *Long* is entirely inconsistent with Delaware's settled law on proximate cause.

Long finds ample support in the case law for its conclusion that epidemiology is not a prerequisite to establishing general causation in a toxic tort case, as evidenced by its lengthy string citation to supporting federal authority.[190] *Long* is also in tune with the practical realities of the court's role as gatekeeper. "Judges, both trial and appellate, have no special competence to resolve the complex and refractory causal issues raised by the attempt to link low level exposure to toxic chemicals with human disease."[191] This observation is all the more insightful when considered in the context of a case, like this one, where the sufficiency of the epidemiological evidence is hotly contested by competent scientists on both sides.[192] The Court "cannot dismiss plaintiffs' experts as poseurs or witnesses for hire. They are serious scientists...."[193] Thus, even if the Court may agree with Chrysler that its analysis of the state of the

---

[190] 2004 WL 1543226, at *6.

[191] *Ferebee*, 736 F.2d at 1534.

[192] *See Callahan v. Cardinal Glennon Hosp.*, 863 S.W.2d 852 (Mo. 1993)(en banc)(allowing experts to testify whether polio vaccine can develop into polio virus under certain circumstances without the support of epidemiological studies, noting that the studies that did exist were not definitive and the incidence of polio is very rare).

[193] *Smith v. Gen. Elec. Co.*, 2004 WL 870832, at *4 (D. Mass. Apr. 23, 2004).

epidemiological evidence is correct, the Court does not "have the authority [under *Daubert*] to conclude a case [as a matter of law] simply because [it] is convinced that one sides's science is superior to the other's."[194]

This Court's legal conclusion on this point is consistent with the only known decision that has considered the precise question before the Court here - - the admissibility of plaintiffs' general causation evidence in the face of contrary epidemiology - - under a *Daubert* standard. In *Chapin v. A & L Auto Parts, Inc.*,[195] the trial court conducted a lengthy hearing during which much of the same evidence, and most of the same legal arguments, were presented by the parties on both sides of the issue. In response to the argument that plaintiffs needed epidemiological evidence to support the association between friction products and disease, the court concluded: "It is not really important to have an epidemiological study to determine whether the risk of cancer is increased by asbestos exposure in every occupation."[196] The court in *Breidenstein v. Allied Signal, Inc.* reached the identical conclusion under

---

[194] *Id.*

[195] Case No. 03-3247775-NP (Mich. 3d Cir. Ct., May 28, 2004)(bench ruling).

[196] *Id.* at 24.

a *Frye* standard.[197]

Chrysler attempts to bolster its argument that epidemiology is required here by citing *Long* for the proposition that plaintiffs must first establish that their injuries were caused only by friction products before they may be excused from presenting supporting epidemiology in their causation case. The argument misses the mark. Delaware law is well settled, even in the asbestos context, that "there may be more than one proximate cause of an injury."[198] This is horn book law.[199] In asbestos cases where plaintiffs allege negligence against multiple defendants for multiple exposures, "the liability of a particular defendant is not dependent upon a showing that the

---

[197]Case No. 2004-11581, at 5 (N.Y. Sup. Ct. Sept. 2005)(in a *Frye* jurisdiction, the court quoted *Chapin* and concluded that occupation specific epidemiology was not required). *But see In Re Asbestos Litig.*, No. 2004-03964, Davis, J. (Tex. Dist. Ct. June 30, 2005) (granting motion to exclude plaintiffs' epidemiology expert upon concluding that a Texas Supreme Court decision required "in mass tort cases that epidemiological evidence be presented establishing causation to be at least twice as likely as a control group within a ninety-five percent certainty."); *In Re: Toxic Substance Cases*, Admin. Docket No. 03-319, Colville, J. (Pa. Ct. Com. Pleas, Allegheny Cty, Feb. 27, 2006)(applying *Frye,* and without explanation, granting defendants' motion to exclude plaintiffs' general causation experts in an automotive friction products case).

[198]*Money v. Manville Corp. Asbestos Disease Comp. Trust Fund*, 596 A.2d 1372, 1375 (Del. 1991)(citing *Culver v. Bennett*, 588 A.2d 1094, 1097 (Del. 1991)).

[199]*See e.g.* W. PAGE KEETON ET AL., PROSSER & KEETON ON TORTS, §41 at 266 (5th ed. 1984)("instructions to the jury that they must find the defendant's conduct to be 'the sole cause,' or 'the dominant cause,' ... of the injury are rightly condemned as misleading error."); J.D. LEE ET AL., MODERN TORT LAW, §28:6.10 at 28-23 (2d ed. 2002)("In an occupational-exposure toxic tort case, medical causation may be established by (1) factual proof of the plaintiff's frequent, regular and proximate exposure to a defendants' product and (2) medical and/or scientific proof of nexus between the exposure and the plaintiff's condition.").

defendant's conduct was the exclusive cause of the plaintiff's injuries."[200] The plaintiff must show that "there was a causal relationship between [each of] the defendants' product and the plaintiff's physical injury, i.e., that but for the plaintiff's exposure to the defendant's asbestos product, the plaintiff's injury would not have occurred."[201]

Moreover, the question of whether an injury has occurred as a result of a minimal exposure to asbestos as part of an ongoing, cumulative exposure is for the jury to decide, assuming the supporting evidence is competent and reliable.[202] Nothing in Delaware law *requires* that evidence to take the form of, or to be comprised (even in part) of, epidemiological evidence. This is true regardless of

---

[200] *Money*, 596 A.2d at 1375.

[201] *Id.* at 1377.

[202] *See Nutt v. A.C. & S. Co., Inc.*, 517 A.2d 690, 694 (Del. Super. Ct. 1986). *See also Borel v. Fibreboard Paper Prod. Corp.,* 493 F.2d 1076 (5th Cir. 1973), *cert. den.,* 419 U.S. 869 (1974)(in a friction products asbestos case, the court noted that it was impossible as a practical matter to determine which of multiple exposures resulted in injury, and allowed plaintiff to present a cumulative exposure causation theory); *Migues v. Fibreboard Corp.*, 662 F.2d 1182 (5th Cir. 1981)(allowing cumulative exposure causation theory). The Court notes that Chrysler's attack on plaintiffs' so-called "single fiber or no threshold theory" - - that is, that a single fiber of asbestos is sufficient to cause mesothelioma - - is beyond the scope of these proceedings. To be clear, the Court has been asked to determine whether plaintiffs' experts have reliably reached conclusions that exposure to friction products can cause asbestos-related diseases. These experts have testified that use of such products *can* release respirable fibers in a volume above background levels and in a size and configuration capable of causing disease. Chrysler disputes this testimony. If, in a given case, a plaintiff must rely upon a no threshold theory to establish causation, the Court can determine the reliability of that testimony on a separate *in limine* motion. Suffice it to say, the testimony will be scrutinized carefully. *See Bartel v. John Crane, Inc.*, 316 F. Supp. 2d 603, 611 (N.D. Ohio 2004)(finding Dr. Frank's single fiber theory to be inconsistent with prevailing scientific evidence, including the testimony of Drs. Lemen and Hammar).

whether plaintiff is able to exclude all other potential causes beyond the toxic substance at issue.

Plaintiffs need not support their general causation case with epidemiological evidence as a matter of law. Other scientific evidence, if sufficiently relevant and reliable, may suffice. *Vel non* occupation specific epidemiological evidence is required in this case, as a matter of science, is a factual issue that will be addressed below.

b. Plaintiffs' Evidence "Stands Up" To Chrysler's Epidemiology

At the end of the day, it was clear that Dr. Goodman was of the view that occupation-specific epidemiology trumps all, even when undisputed evidence of substantial, unprotected, and uninterrupted exposure to friction products, coupled with positive tissue burden studies, indicate that such exposure caused disease.[203] If the epidemiological data exists, Dr. Goodman is of the view that this data is dispositive of the general causation issue and that all other conflicting data is inherently unreliable.[204] Plaintiffs' epidemiologist, Dr. Lemen, disagrees.[205] According to Dr. Lemen, a scientist must "look at the reality of the situation as it

---

[203]D.I. 2345, 10/20/05 p.m. at 95-96.

[204]*Id.* at 27.

[205]D.I. 2344, 10/19/05 a.m. at 96-99.

exists in the real world" when determining what scientific evidence will inform the determination of whether an association exists between a toxic substance and disease.[206] Dr. Lemen believes that "the reality of the situation" with respect to the association between friction products and asbestos diseases is that the epidemiology is "equivocal" and that other sources of scientific data offer meaningful guidance on the question.[207] Drs. Hammar and Frank agree.[208] Dr. Lemen was critical of each of the epidemioligical studies relied upon by Dr. Goodman and, as to each study, he identified either structural defects or "confounders" in the study that weakened the reliability of the study's conclusions.[209] Moreover, according to Dr. Lemen, epidemiology is less informative when the disease under study is rare. Finally, both Dr. Lemen and Dr. Hammar rely upon the epidemiological data out of Australia that, in their view, notwithstanding admitted shortcomings, supports an association between exposure to friction products and asbestos diseases.[210]

---

[206] *Id.* at 98.

[207] *Id.* at 96. *See also* Reference Manual at 414, n. 40 (difficulties in conducting epidemiological research have led "some researchers to conclude that 'many negative epidemiological studies must be considered inconclusive' for exposure to low dose or weak carcinogens.").

[208] D.I. 2342, 10/18/05 a.m. 85-87 (Dr. Hammar); D.I. 2685, 10/19/05 p.m. at 75 (Dr. Frank).

[209] D.I. 2684, 10/18/05 p.m. at 95- 128; D.I. 2343, 10/19/05 a.m. at 3-13, 126-32.

[210] D.I. 2684, 10/18/05 p.m. at 107-09 (Dr. Lemen); D.I. 2342, 10/18/05 a.m. at 42-44, 56 (Dr. Hammar).

Dr. Goodman responded to Dr. Lemen's criticisms by stating that he accounted for any weaknesses in the epidemiological studies in the design of his meta-analysis.[211] In response, Dr. Lemen questioned the efficacy of the meta-analysis approach as a means to evaluate the reliability of epidemiological data given that such studies are primarily designed to address controlled clinical trials and also given the extensive data linking chrysotile to disease.[212] Dr. Lemen's criticism finds support in the scientific literature and in the Federal Reference Manual on Scientific Evidence.[213]

Equipped with an advanced degree in science and years of training and experience in the field of epidemiology, perhaps the Court could act as arbiter of this dispute between well-credentialed camps of scientists and conclusively proclaim whether or not Chrysler's epidemiological evidence is reliable and definitive. An undergraduate political science degree coupled with a law degree, however, hardly qualifies the Court to undertake this exercise.[214] If the epidemiology was settled, Chrysler's claim of scientific preeminence on behalf of its evidence may have more

---

[211]D.I. 2344, 10/20/05 a.m. at 130-35.

[212]D.I. 2684, 10/18/05 p.m. at 112-16.

[213]*See* PX 168 (Bailar reports on the shortcomings of meta-analysis); Reference Manual at 361-381, 414 (addressing multiple concerns relating to metal-analysis).

[214]Reference Manual at 2 ("[the] judge is not a scientist and the courtroom is not a scientific laboratory.").

appeal at this stage of the proceedings. But the epidemiology is not settled. Meaningful challenges to the data have been mounted and must be considered. Under these circumstances, the Court cannot and will not pick one side's reliable science and cast off the other's.[215] This scientific dispute will be good and appropriate grist for the jury.[216]

Once the notion that "epidemiology trumps all" is put to rest, the Court's conclusion regarding the reliability of the testimony of each of the plaintiffs' witnesses, as discussed above, is dispositive. For the reasons stated above, each of the plaintiffs' witnesses are qualified, offer conclusions that have been tested and subjected to peer review, rely upon information reasonably relied upon by others in their fields, and will testify in a manner that will assist the trier of fact to understand the evidence and determine a fact in issue (causation).[217] Accordingly, each witness passes the *Daubert* test.

---

[215] *See Minner*, 791 A.2d at 848 (the trial court does not choose between competing reliable opinions, nor is it empowered to determine which theory is stronger).

[216] *See e.g. Ford Motor Co. v. Wood*, 703 A.2d 1315 (Md. Ct. Spec. App.), *cert. den.*, 709 A.2d 139 (Md. 1998) (appeal after jury trial of friction product asbestos case complete with varied science supporting plaintiffs' claims and purportedly definitive contrary epidemiological evidence in support of the defense); *Becker v. Baron Bros.*, 649 A.2d 613 (N.J. 1994)(same).

[217] *See generally Daubert,* 509 U.S. at 593-94; *Nelson*, 628 A.2d at 74.

V.

The Court has conducted the required analysis under *Daubert* as to each of plaintiffs' proffered causation experts. While there certainly is room for competent scientists to disagree, each of plaintiffs' experts have employed appropriate methodologies to reach reliable conclusions that exposure to friction products increases the risk of contracting asbestosis, lung cancer and mesothelioma. Occupation specific epidemiology is not required to support these conclusions. Chrysler may present its scientific evidence to the contrary and may cross examine plaintiffs' experts as vigorously at trial as it did during the *Daubert* hearing. Chrysler may also mount a challenge to each individual plaintiff's specific causation case. The jury will not be mislead; reliable expert testimony on both sides, facilitated by the adversarial process, will bring clarity to the issue. Chrysler's Motion *In Limine* To Exclude Expert Testimony That Automotive Friction Products Cause Asbestosis, Lung Cancer and Mesothelioma is **DENIED.**

IT IS SO ORDERED.

/s/ Joseph R. Slights, III
Judge Joseph R. Slights, III

Original to Prothonotary