THE FIRST JUDICIAL DISTRICT OF PENNSYLVANIA, PHILADELPHIA COUNTY
IN THE COURT OF COMMON PLEAS

IN RE:	**:**	**TRIAL DIVISION- CIVIL**
ASBESTOS LITIGATION	**:**	**OCTOBER TERM, 1986**
	:	**No. 0001**
	:	
	:	
Certain Asbestos Friction Cases Involving	**:**	
Chrysler LLC	**:**	
	:	**Control # 084682**
Motion to Exclude Plaintiff's Causation Expert	**:**	
Testimony that Relies Upon Novel Scientific	**:**	
Evidence and Request for Evidentiary Hearing	**:**	

F I N D I N G S, M E M O R A N D U M
and O R D E R

This matter comes before the Court as a result of a Motion for a *Frye* Hearing filed by Defendant, Chrysler LLC on August 27, 2007.

The Motion was filed generally in this Court's Asbestos Mass Tort Program, which, because of the Mass Tort status accorded to these cases in the Complex Litigation Center which this Court supervises, is generally captioned: *In Re: Asbestos Litigation* and sub captioned, *Certain Asbestos Friction Cases Involving Chrysler LLC.*

As part of the management protocols for administering the Asbestos Litigation, this Court establishes Trial Groups for the upcoming calendar year which identify the trial listings, including cases subject to this motion. In order to make the anticipated evidentiary issues manageable and present a cross section of the issues which were relevant for purposes of the *Frye* Hearing, this Court directed the parties to identify not more than "six cases wherein the experts' methodology used to support their respective opinions on causation are being challenged." (Order of January 25, 2008, Control #114291).

The following four (4) cases were selected:

Caswell v. A.W. Chesterton, Inc., et al., 0609-0782
Duke v. Chrysler LLC, et al., 0612-3451
Fisher v. A.O. Smith Corp, et al, 0608-2483
Young v. A.W. Chesterton, Inc., et al, 0609-0962

In order to present an accurate and complete history of the Plaintiffs involved

here, certain work histories were produced. The purpose of this was to show the universe of exposures to asbestos products.

The information is from summaries produced by the Defendants and admitted into evidence as:

D-17 *Caswell v. A.W. Chesterton, Inc., et al.*
D-18 *Duke v. Chrysler LLC, et al.*
D-19 *Fisher v. A.O. Smith Corp., et al.*
D-20 *Young v. A. W. Chesterton, Inc., et al.*

Caswell Exposures from D-17 and P-12 (Lab Report) [1]

Mr. Caswell was born 8/31/45. After serving in the military from 1964-1967, he began what would be his primary employment by the Budd Company, from 1968 to 2003. During this employment Mr. Caswell was exposed to raw asbestos as part of the welding process when "he manipuled asbestos gloves, welding rods, firebricks and flux" and "cooling welded steel rods in large metal boxes of raw asbestos, and stated that 'it was, like, loose, like powder.'" Mr. Caswell also remembered working with and around asbestos gaskets in the automotive machine shop and was also present during two (2) asbestos abatements of pipe insulation in the plant. This was prior to 1980 at which time he was transferred to another plant where he was less exposed to asbestos powder but did continue to be near welders as they worked.

Mr. Caswell has other exposures in a non-occupational setting as a result of changing brakes, either disc or drum, between 1968 and 2007. He had specific recall of 28 brake replacements over the 29 year period but stated that he had done 75 to 100 brake replacements in total without any additional level of specificity. The time to replace the brakes ranged from 30 minutes to 60-90 minutes. The tasks performed during the replacement were myriad, ranging from wiping down the drums with gasoline to remove any road dust to pulling the shoes or drums and sanding the discs' leading edges. These tasks were completed in garages or carports.

His asbestos related disease is reported to be pulmonary asbestosis.

His health history includes one and one-half to two packs a day of smoking filtered and non-filtered cigarettes lasting over 46 years (1960-2006).

He also has a history of coronary artery disease, diabetes mellitus, cancer, alcoholism and is significantly overweight at 271 pounds and 70 inches tall.

The asbestos related disease was diagnosed by Dr. Jonathan L. Gelfand, based

1. The Chrysler Defendants were dismissed from the *Caswell* case.

upon his impression of, "bilateral pleural thickening and interstitial lung disease and pulmonary asbestosis." (Exhibit P-12, Letter of 9/14/2004).

Dr. Gelfand offered his opinion as to the causation of Mr. Caswell's asbestos related disease in the following manner:

> In summary, Mr. Caswell has a history of exposure to asbestos in the workplace over a period of many years. He has asbestos pleural disease and pulmonary asbestosis. In my opinion, exposure to asbestos in the workplace is the cause of the asbestos pleural disease and pulmonary asbestosis and is a substantial contributing factor to his pulmonary function abnormality and to his dyspnea on exertion. Each and every exposure to asbestos has been a substantial contributing factor to the asbestos related abnormalities noted. I hold those opinions to a reasonable degree of medical certainty. In addition, Mr. Caswell has a history of cigarette smoking. This is also a contributing factor to his dyspnea on exertion. In addition, Mr. Caswell has diabetes mellitus and coronary artery disease. Notwithstanding the coronary artery disease, diabetes and cigarette smoking, it is exposure to asbestos which has caused the asbestos related abnormalities notes. I hold those opinions to a reasonable degree of medical certainty. In addition, in my opinion, because of cigarette smoking and the compounding factors of air trapping possibly caused by cigarette smoking, the restriction which would otherwise be seen on his pulmonary function test may be obscured. I hold that opinion to a reasonable degree of medical certainty. In addition, Mr. Caswell is significantly overweight. In my opinion, excess weight is a contributing factor to his dyspnea on exertion. I hold that opinion to a reasonable degree of medical certainty.

Exhibit P-17, Letter of October 26, 2007.

It is significant to note that Dr. Gelfand uses the term, *workplace exposure,* as being synonymous with occupational exposure and does not refer to any non-occupational exposure related to changing brakes which for these purposes are categorized as non-occupational exposures. Further, Dr. Gelfand does not distinguish between exposures when he says, "Each and every exposure to asbestos has been a substantial contributing factor to the asbestos related abnormalities noted." Dr. Gelfand does not cite any sources or identify the process he employed to arrive at this causation conclusion.

Duke Exposures from D-18

Mr. Duke was born on March 13, 1948 and was diagnosed with malignant mesothelioma in 2006. His alleged exposures fall into two categories which are both occupational and non-occupational in nature. The occupational exposures are from (1) pipefitting and plumbing work with asbestos materials generally associated with those occupations and (2) employment as an auto mechanic where he may have been exposed to friction by-product of asbestos lined brakes and clutches, at some employment locations.

He also claimed exposures non-occupationally to friction by-product of asbestos by witnessing his father work on various brake jobs while he loitered at his father's places of employment before going to school or at night when his father did brake jobs. He also claims exposure to brake products when he started doing brake jobs on his own.

There were three (3) expert reports produced in the *Duke* case. Richard A. Lemen, Ph.D., MSPH, (December 2007); Dr. William E. Longo, Ph.D. (December 3, 2007); Dr. Eugene J. Mark, M.D., (December 4, 2007). Dr. Lemem did not testify at the *Frye* Hearing. Dr. Longo and Dr. Mark did. Their reports and testimony will be discussed later in this opinion.

Dr. Lemen made a conclusion in his report that "because of the strong association of mesothelioma to asbestos exposure, it is not necessary to have an epidemiological study for every occupation and every type of exposure in order to establish that a particular occupational exposure caused a mesothelioma." This statement, which sounds like an "each and every exposure to asbestos" theory, is relevant to this discussion not withstanding Dr. Lemen's failure to testify at the *Frye* Hearing because of its similarity to other such conclusions reached by the experts that did testify.

Fisher Exposure and Background from D-19 and P-13

Mr. Fisher was born November 17, 1927. He was 80 years old at the time of his death from lung cancer. He was a smoker of non-filtered cigarettes with a 25 pack/year habit until he stopped smoking around the age of 40. He was employed as a plumber from 1939 to 1990. His exposure during the approximately fifty-year period was from a variety of sources which included asbestos pipes, asbestos pipe coverings, asbestos coil packing, asbestos gaskets, asbestos boiler insulation (hard), asbestos insulation (soft), asbestos cement and asbestos putty.

Mr. Fisher also claims exposure in non-occupational settings from doing home

repairs where he was exposed to miscellaneous products such as insulation, siding and roofing products of an unspecified nature which he did on an occasional basis.

He also performed oil changes, brake repairs and lube jobs on multiple automobiles. He could remember approximately 11 brake changes over his lifetime with maybe a couple more to his 1933 and 1940 Plymouth. Jonathan L. Gelfand, M.D., offered an expert report which is marked as Exhibit P-13. Dr. Gelfand reviewed Mr. Fisher's records and related in part that "it was Dr. Joyce's opinion that (Mr. Fisher's) pulmonary fibrosis seen on chest x-ray and chest CT could have been related to asbestos exposure." *Id.* (emphasis supplied). Dr. Gelfand concluded on page 2 of his two-page report:

> In summary, Mr. Fisher had a long history of exposure to asbestos dust as part of his work over a period of thirty years. He had interstitial pulmonary fibrosis and small cell cancer of the lung. In my opinion, asbestos was the cause of the pulmonary fibrosis also known as asbestosis and was a substantial contributing factor to his lung cancer and his death. I hold that opinion to a reasonable degree of medical certainty. Each and every exposure to asbestos was a substantial contributing factor to his pulmonary fibrosis and to his death. In addition, it is my opinion that although he had a cigarette smoking history, he had stopped sufficiently far in the past that cigarette smoking was not a substantial contributing factor to his pulmonary fibrosis, to his lung cancer or to his death. I hold that opinion to a reasonable degree of medical certainty.

Exhibit P-13.

Although friction products were not directly identified as the source of the cancer causing asbestos exposure, Dr. Gelfand seems to have implicitly implicated these exposures when he opines that, "Each and every exposure to asbestos was a substantial contributing factor to his pulmonary fibrosis and to his death." No sources are cited and no methodology was identified in his Report.

Young Exposures from D-20

Plaintiff did not advance any evidence with respect to the *Young* case. Defendants Chrysler and General Motors were sued by Plaintiff (see individual Docket Case Number, August Term, 2005, No. 2690) and both are in the friction product group of defendants. The *Young* matter was incorporated into the *Frye* Motion upon Chrysler's

request (See Motion under Control #104897). Plaintiff argues in his Post Hearing Memoranda of March 18, 2008, that *Young* should not have been included in this *Frye* Group as the expert deadline had not yet run at the time of the hearing. Because of this, there was no evidence presented by Plaintiff. The status of this case is addressed below.

Preliminary Discussion

Before the discussion of the merits of the *Frye* testimony, a brief discussion of certain events and subsequent orders entered by this Court focusing on the prerequisite findings to proceeding with the *Frye* issues is appropriate.

This discussion must necessarily concern the use of the word "novel" and what the definition is of same when used in connection with a *Frye* proceeding.

On December 27, 2007, this Court entered the following Order:

> And now, to wit, this 27th day of December 2007, this Court writes in response to Defendant Chrysler's letter of December 14, 2007, wherein Chrysler's counsel asks this Court to enter an express finding of novelty as a prerequisite to proceeding with the scheduled *Frye* hearing.
>
> This Court declines to enter such a finding because the term novel, in this context, has never been satisfactorily defined.
>
> First, this Court agrees with the majority in *Trach v. Fellin*, 817 A.2d 1102, wherein it held that the word "novel" does not necessarily mean "new." As recognized by the Court in *Trach v. Fellin*:
>
> > We, like the dissent, are aware that ebb and flow are at the heart of the scientific method: the theory of relativity is only valid until someone disproves it. As the *Frye* court so elegantly stated, however, "While courts will go a long way in admitting expert testimony deduced from a well-recognized scientific principle or discovery, the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs." *Frye*, 293 F. 15 1024. In this single, simple sentence, the *Frye* court recognized that the essence of admissibility is general acceptance: that a principle or discovery can fall by the wayside as science advances is just another way of saying it is not generally accepted. We therefore conclude that we are merely stating the law in Pennsylvania when we state that *Frye* applies only to novel science.
>
> Therefore, in keeping with the holding of *Trach v. Fellin*, this Court finds that movant has raised legitimate issues regarding the general acceptability of Plaintiff's expert methodologies, such that a full evidentiary hearing is justified.
>
> This Court further finds that an analysis pursuant to Pa. R.C.P.

207.1 (explanatory note) wherein this Court considered:

> In deciding whether to address prior to trial the admissibility of the testimony of an expert witness, the following factors are among those which the Court should consider; the dispositive nature or significance of the issue to the case, the complexity of the issue involved in the testimony of the expert witness, the degree of novelty of the proposed evidence, the complexity of the case, the anticipated length of trial, the potential for delay of trial, and feasibility of the court evaluating the expert witness's testimony when offered at trial.

See In Re: Asbestos Litigation, Order, 12/27/07.

The issue as this Court perceives it is that there seems to be an argument that a court is required to find a methodology "novel" as a pre-requisite for proceeding with a *Frye* hearing. This may be to avoid what the Superior Court identified as an issue when it said, "Clearly, however, our Supreme Court did not intend that trial courts be required to apply the *Frye* standard every time scientific experts are called to render an opinion at trial, a result that is nothing short of Kafkaesque to contemplate." *Trach v. Fellin*, 817 A.2d at 1110. The problem with this approach is that the *Frye* test requires a conclusion that the proffered scientific methodology is novel as a basis for precluding its admission. To avoid such confusion that would be inherent where a court would be required to find that the evidence is novel before the hearing and then be asked to find on the issue of novelty after hearing the evidence, this Court opted to find that the movant has raised a legitimate issue regarding the general acceptability of Plaintiff's expert methodologies within the body of the expert reports offered in support of causation.

In order to understand the evidence that is being offered by Plaintiffs, it is necessary to review same and attempt to categorize it since some of it is supported by expert reports which were supported by testimony of the authors, and some of it was solely the subject of certain testimony, not supported by expert reports.

Plaintiffs Experts

Plaintiffs offered the opinions of the following experts, either by expert report or by presentation at the *Frye* Hearing:

Dr. Eugene Mark

Dr. Mark offered Expert Reports in the *Duke* case. Exhibit P-2, November 4, 2007 Report and Exhibit P-3, December 4, 2007 Report. He also testified at the *Frye* Hearing. Dr. Mark is a physician and pathologist. He is a graduate of Harvard University and Harvard Medical School and completed his residency at Massachusetts General Hospital. He has been a senior lung pathologist, teaching about and diagnosing lung diseases since 1974. Dr. Mark has published "ten or twenty articles having to do with asbestos" and numerous case records in the New England Journal of Medicine. He has written two books "which deal with asbestos and diffuse malignant mesothelioma and other mesotheliomas." He also lectures frequently. (N.T. 2/11/08, A.M., pp. 86-90).

Dr. William Longo

Dr. Longo submitted a Report, Exhibit P-5, also in the *Duke* case and he testified in support of his Report.

Dr. Jonathan Gelfand

Dr. Gelfand issued two (2) Expert Reports, Exhibit P-12 in the *Caswell* case and Exhibit P-13 in the *Fisher* case. He did not testify at the *Frye* Hearing.

Dr. Arthur L. Frank

Dr. Frank did not issue Reports in any of the four (4) cases identified as part of this *Frye* Hearing. He was deposed in the *Fisher* case, (N.T. 2/12/08, p. 64), and he testified in the *Frye* Hearing regarding the *Fisher* and *Caswell* cases. Dr. Frank has a medical degree and a Ph.D. He is employed at Drexel University School of Public Health and is Chair of the Department of Environmental and Occupational Health. He is a Professor of Public Health and Internal Medicine. Dr. Frank serves on the editorial boards of a number of journals, including, the American Journal of Industrial Medicine, the International Journal of Toxicology, the Journal of the National Cancer Institute, JAMA and Science. He is engaged in active peer review in this area of science. He has always been an "academic physician at a university—in a university setting." (N.T., 2/12/08, P.M., pp. 6-15).

Analysis of Plaintiffs' Expert Reports

Having now identified the written Reports introduced in the *Frye* Hearing by Plaintiff as P-2, P-3, P-5, P-12 and P-13, it is quite clear on the face of these documents that the methodology employed by the experts in determining causation is absent.[2]

The experts offered by Plaintiffs render their conclusions on causation in a manner which evidences consistent uniformity:

Dr. Mark says this in the *Duke* case in P-3, which is his final Report:

> Asbestos is the only established cause of diffuse malignant mesothelioma in patients in the United States who have not received prior radiotherapy at the site of the tumor. All of the exposures to asbestos which occur prior to the development of a diffuse malignant mesothelioma contribute to be pathogenesis. All of the types of asbestos can cause diffuse malignant mesothelioma.
>
> I conclude that the patient has developed a diffuse malignant mesothelioma of the pleura. I conclude that the asbestos to which he reportedly was exposed caused the diffuse malignant mesothelioma. *I conclude that all of the exposures which occurred prior to the occurrence of the malignancy together contributed to cause the diffuse malignant mesothelioma. I conclude that each of the exposures which occurred prior to the occurrence of the malignancy was a substantial contributing factor in the causation of the diffuse malignant mesothelioma.*

(Emphasis supplied). Dr. Gelfand came to a similar conclusion in the *Caswell* case:

> In summary, Mr. Caswell has a history of exposure to asbestos in the workplace over a period of many years. He has asbestos pleural disease and pulmonary asbestosis. *In my opinion, exposure to asbestos in the workplace is the cause of the asbestos pleural disease and pulmonary asbestosis and is a substantial contributing factor to his pulmonary function abnormality and to his dyspnea on exertion. Each and every exposure to asbestos has been a substantial contributing factor to the asbestos related abnormalities noted.* I hold those opinions to a reasonable degree of medical certainty. In addition, Mr. Caswell has a history of cigarette smoking. This is also a contributing factor to his dyspnea on exertion. In addition, Mr. Caswell has diabetes mellitus and coronary artery disease. Notwithstanding the coronary artery disease, diabetes and cigarette smoking, it is exposure to asbestos which has

2 This, in and of itself, is problematic since an Expert Report with no explanation of the Expert's methodology is almost guaranteed to require a *Frye* hearing and this triggers the Kafkaesque scenario previously referred to in *Trach v. Fellin.* Id.

> caused the asbestos related abnormalities noted. I hold those opinions to a reasonable degree of medical certainty. In addition, in my opinion, because of cigarette smoking and the compounding factors of air trapping possibly caused by cigarette smoking, the restriction which would otherwise be seen on his pulmonary function test may be obscured. I hold that opinion to a reasonable degree of medical certainty. In addition, Mr. Caswell is significantly overweight. In my opinion, excess weight is a contributing factor to his dyspnea on exertion. I hold that opinion to a reasonable degree of medical certainty.

(Exhibit P-12) (emphasis supplied). Dr. Gelfand also came to this conclusion in the Fisher case:

> In summary, Mr. Fisher had a long history of exposure to asbestos dust as part of his work over a period of thirty years. *He had interstitial pulmonary fibrosis also known as asbestosis and was a substantial contributing factor to his lung cancer and to his death. I hold that opinion to a reasonable degree of medical certainty. Each and every exposure to asbestos was a substantial contributing factor to his pulmonary fibrosis and to his death.* In addition, it is my opinion that although he had a cigarette smoking history, he had stopped sufficiently far in the past, that cigarette smoking was not a substantial contributing factor to his pulmonary fibrosis to his lung cancer or to his death. I hold that opinion to a reasonable degree of medical certainty.

(Exhibit P-13) (emphasis supplied).

Dr. Frank did not issue an Expert Report in these cases. His expert opinion, relevant to this *Frye* hearing, was developed in separate depositions taken in the *Fisher* and *Caswell* cases. He testified regarding his opinion in these cases at the instant hearing.

> Q. I want to talk to you some about two specific cases that are before this Court. The first is the Fisher case. What was Mr. Fisher's diagnosis?
> A. Small cell carcinoma of the lung.
> Q. Is that a type of lung cancer?
> A. It is a type of lung cancer, and specifically it is a type of lung cancer caused by exposure to asbestos.
> Q. You gave a deposition in the Fisher case, do you recall?
> A. Yes.
> Q. And in that deposition, you were provided—you provided a conclusion regarding Mr. Fisher's exposures—and correct me if I'm wrong on these exposures—they

were to asbestos-containing gaskets, packing, tile, board and brakes?
A. That is my recollection.
Q. It was presented to you in a hypothetical?
A. Yes.
Q. The only information that you reviewed was the diagnosis and a single piece of paper that was provided to you by Mr. Fisher's lawyers?
A. Yes.
Q. And on that piece of paper, it provided you the occupational and para-occupational exposures to asbestos products?
A. And the latency information.
Q. And I want to show you your testimony and ask you, first, if that is your conclusion. Do you believe that the dust, if there was any dust given off from any particular asbestos products that Mr. Fisher worked around, do you believe that that dust, if it was done in an occupational setting that he would testify was made of asbestos, do you believe that each and every breath, or do you have an opinion as to whether or not each and every breath of that material would have been a factual cause of his lung cancer?

And your response was, I have an opinion. And each and every breath containing asbestos in an occupational setting that exposed him above background would have contributed to his disease. Do you recall that testimony?
A. Yes.
Q. Is that your conclusion in the Fisher case?
A. Yes.
Q. Was that conclusion based upon the methodology that we've discussed here today?
A. Yes.

MR. LANGDOC: Could we mark for identification purposes and admit into evidence Plaintiff's Exhibits 12 and 13.

MR. SAMMS: What are they?

MR. PAUL: They are the medical reports, the diagnosing reports in the two cases.

MR. SAMMS: No objection.

BY MR. LANGDOC:
Q. That conclusion that you derive in the Fisher deposition -- and I'm sorry -- was my last question whether or not it was based upon the methodology?
A. Yes.
Q. Okay. And you said yes.

I want to now move on to the Caswell case. Are you familiar with the Caswell case?
A. I have some familiarity with it.

Q. Is that something you've discussed with Mr. Caswell's attorneys over the last few days?
A. Yes.
Q. What's your understanding as to what Mr. Caswell's diagnosis of disease was?
A. Asbestosis was the diagnosis. He had an appropriate latency period, and he had various exposures.
Q. And correct me if I'm wrong, is this your understanding from discussions you had with attorneys: That he had bystander exposure to asbestos insulation from abatement, that he worked with raw asbestos from a box that contained welding rods, that he wore asbestos gloves, and that he changed asbestos-containing brakes on his own cars 70-100 times.
A. That is my understanding.
Q. And in that case, sir, you have not rendered a report yet: is that correct?
A. Correct.
Q. What would your conclusion be as to whether or not these exposures would have been substantial contributing causes to his disease, asbestosis?
A. If, in fact, there would be evidence, and I suspect there is, that he has appropriate either radiologic or pathologic changes in his tissue, my opinion would be that those exposures would have contributed in a substantial way to his developing his asbestosis.
Q. Is there anything other than asbestos that causes the disease asbestosis?
A. Nothing that I know of. That's why the name is asbestosis. There are other diseases that can look like it, which is why it goes back to my earlier comments: You have to document the exposure.

But, no, the only disease that will cause asbestosis is asbestos. And all of the different fiber types of asbestos have the ability to produce asbestosis.
Q. Have you reviewed medical scientific literature showing that work with asbestos-containing brake products can cause the disease, asbestosis, which Mr. Caswell has?
A. I have.

THE COURT: Go back for a moment.

Do all of the sources of asbestos have the same type of fiber?

THE WITNESS: Probably not. My guess would be that certainly the likelihood of the raw asbestos being chrysotile. The only thing I don't know is the nature of the asbestos insulation, which depending on its nature may have been chrysotile or may have also contained amphibole.

BY MR. LANGDOC:
Q. Is this conclusion based upon the methodology

that you've discussed with this Court today?
A. All of those things we discussed are what I take into consideration when I render such a judgment.
Q. Exposures that contribute to the aggregate dose are not always the same as exposures that are substantial contributing factors: Agree or disagree?
A. I would agree with that.
Q. When you make a conclusion, such as you did in the Fisher case, that each and every occupational exposure above the background level contributed or was a substantial contributing factor to the disease, what does that mean?
A. What it means is faced with the diagnosis of a disease that I know to be caused by asbestos, that when I have documentation that the exposure is above background, I can relate with a reasonable degree of medical certainty in that individual that their disease was substantially contributed to by their exposures.

N.T. 2/12/08, pp.63-69, P.M. Session

Dr. Frank's testimony was clarified on examination:

BY MR. SAMMS:
Q. Hi, Dr. Frank.
A. Hello, Mr. Samms.
Q. Dr. Frank, would I be correct if I characterized your testimony and your opinion in asbestos cases that each and every breath substantially contributes to asbestos disease?
A. Each and every breath that has levels above background would be contributory to someone -- someone's disease if they developed an asbestos-related disease.
Q. So as long as they have an asbestos-related disease and exposure, if I came to you and I asked you an hypothesis: To assume that somebody was diagnosed with a lung cancer that could be related to asbestos, and they had just background exposure and one brake change, that would be enough for you to say that the brake change was a substantial contributing factor.
A. There is no way to say that all of the exposures that they had wouldn't have contributed to their disease. What I have always testified is that if I only had just background exposure, I could not testify in a given case with a reasonable degree of medical certainty that that case was caused by asbestos.
But when you provide me evidence of exposure above background, then it seems reasonable and logical, understanding how carcinogens work, to say that that was a substantial contributing cause.

> Q. So in my hypo, your answer would be, yes, that that one brake change was a substantial contributing factor?
> A. Understanding carcinogenesis theory as I do, I would have to say yes.
> Q. Okay. And it would be fair to say in the past, you've testified that's the case regardless of intensity of exposure, type of exposure or type of fiber?
> A. If it fits the hypothetical you just gave me where the intensity is unknown, the type of fiber certainly doesn't matter, and what was the third one? Frequency? One brake job theoretically could do it. The likelihood would be extremely small, but it's not zero.
> Q. Well, that's where I'm confused a little bit, sir. You say the likelihood is very small. But in that exact scenario, you would use the wording that it was a substantial contributing factor?
> A. If they developed -- if that individual developed a lung cancer and that was part of their known exposure with the proper latency, yes.

N.T. 2/12/08, pp.79-81 P.M. Session

It is quite clear that the above experts' opinions on the issue of causation are based upon their assumptions that each and every breath of asbestos is a substantial contributing factor in the causation of any asbestos disease. As it is used by these experts, the term "each and every exposure to asbestos" is also a substantial contributing factor to the causation of asbestos disease. These conclusions are made without any apparent consideration of frequency, regularity and proximity. Their written and oral opinions, as to causation are not informed with the methodology by which they arrived at their conclusion.

In an attempt to remedy the deficits in their experts' opinions, Plaintiffs offered Dr. Mark as a witness at the *Frye* hearing:

> Q. Dr. Mark, today's topic, what we're going to be asking you about is what method do you, as a medical doctor, use to determine what is a significant contributing factor in causing a disease? And specifically, we are going to be asking you what methodology you are going to be applying in Mr. Duke's case to analyze whether or not his exposure to friction brake products was a significant contributing factor towards causing Mr. Duke's mesothelioma.
>
> Are you prepared to offer your opinion on that today, and to provide an explanation of the methodology that you used to derive that opinion?
> A. Yes, I am, to the best of my ability.

N.T. 2/11/08, pp. 83-84 A.M. Session

Dr. Mark is a physician and pathologist specializing in lung disease. (N.T. 2/11/08, at 86, A.M. Session). He is a diagnostician having diagnosed thousands of diffuse malignant mesothelioma (hereinafter "DMM") which is extricably linked to asbestos. (N.T. 2/11/08, pp. 88-89, A.M. Session). Dr. Mark testified as to being qualified as a pathologist to determine what contributes to a patient's DMM as a result of studying the disease and its development over several decades and by understanding of physics, chemistry, toxicology, industrial health, occupational medicine, and molecular physics. (N.T. 2/11/08, pp. 100-101, A.M. Session).

Dr. Mark begins his approach to the causation of DMM with the assumption that the background rate of DMM is virtually zero and, "that there are no idiopathic mesotheliomas."

Dr. Mark then goes on to lay out his methodology:

> Q. Can you describe for the court some of the steps that-- the initial steps that you would go through under your methodology with respect to answering this question? Were the friction brake exposures that Mr. Duke experienced a substantial contributing factor toward his developing diffuse malignant mesothelioma? And so, when I say methodology, I mean start from the very beginning, what is the first step in your process when you are giving [sic] Mr. Duke's medical information?
> A. My first step, as I started to say earlier in response to a different question, is to confirm the diagnosis. And in this area the diagnosis is more important than in other cases, because this diagnosis is a signal tumor, or a signal diagnosis. That is the diagnosis, itself, indicates essentially its causation.
>
> So, as far as what caused the disease in the usual sense, the chances are overwhelming that it was caused by asbestos. Now -- and that's the most clear use of causation in such a case. Asbestos caused the disease.
>
> Now, if the question is where did the asbestos come from, and how did it get into the person's lung? Those are less concrete, but they can be approached. And if you are going to use less precise areas, some of these are listed here, particularly the Bradford-Hill criteria, nine criteria that can be used in a more general sense for deciding on causation. But if you would go back to the prior slide just a moment.
> Q. Okay.

A. The middle part of that chart is the modern scientific approach. Molecular studies, genetic studies, tissue culture studies, animal studies, that is science. When you pick up the Scientific Journal now, like Nature or like Science, or like the New England Journal of Medicine, that's what you see, that's the science. At the bottom there's epidemiology, that is a science, it's defined as the study of epidemics. It doesn't fit in with the way we use methodology in the clearest sense. A methodology, in its clearest sense, is something where you can repeat it. But you can't repeat what happened to a person thirty years ago. On the other hand you can repeat a study tomorrow but –

N.T. 2/11/08, pp. 106-108, A.M. Session.

Dr. Mark was asked to differentiate the various exposures to asbestos.

Q. Okay. And so, can you explain what you were saying when you said that all of the exposures which occurred prior to the occurrence of the malignancy together contributed to cause the diffuse malignant mesothelioma, and how that differentiates from the analysis that you would do to determine whether or not the friction exposure was a substantial contributing factor towards the development of the disease?

A. As I hear the question there are three subtle different questions embedded in that one question. So, let me try to sort it out as best I can.

One function of that sentence was to differentiate the exposures prior to the occurrence of the malignancy from exposures after the malignancy. And although the sentence doesn't talk about after, it confines itself to prior. So, it was meant to say that after the tumor was developed, other exposure are moot, because the tumor is now out of the -- the horse is out of the barn, and it's over as far as that.

So, all of the exposures prior to the occurrence of the malignancy. And then it is meant to indicate that there's a totality, because the word together is used. So, it's the totality of all of the exposures that caused his malignancy.

N.T. 2/11/08, pp. 112-113, A.M. Session.

Once again, Dr. Mark repeated his methodology as it pertained to Mr. Duke:

Q. So, just to clarify here, Dr. Mark, are you saying that the total exposures, all of the exposures that occurred that Mr. Duke had, to asbestos, were enough to cause him to have diffuse malignant mesothelioma, that his dose

was enough, all of the exposure?
A. Now, as I hear the question, there's two possible questions in there.
Q. Sorry.
A. If you mean, Mr. Duke, yes. If you mean, in general. No. So --
Q. I mean Mr. Duke.
A. Mr. Duke, the answer is yes.
Q. Okay. And here's the question, and here's the critical distinction. Are you, in anyway, saying that Mr. Duke having a breath of exposure to friction products would be a substantial contributing factor toward his development of mesothelioma?
A. If that's the whole story. No, it wouldn't.
Q. And are you -- when you look at analyzing whether or not in Mr. Duke's case, the friction products were a substantial contributing factor toward the development of Mr. Duke's mesothelioma are you going to analyze the frequency and proximity and duration of his exposure to those friction products prior to opining with respect to the significance of that exposure toward his development of mesothelioma?
A. If asked the question on it, yes. To the degree I am supplied with that information. Yes. And I would say as follows, the greater the duration, the greater the frequency, the greater intensity. To that degree all of those exposures with that knowledge has a greater contribution than some other exposure that has a lesser frequency, a lesser intensity, a lesser duration.

N.T. 2/11/08, pp.116-120, A.M. Session.

As part of his approach to causation, Dr. Mark testified as to his reliance on the *Bradford-Hill* analysis. (N.T. 2/11/08, p. 127, A.M. Session). He describes the *Bradford-Hill* analysis this way:

> And the Bradford-Hill criteria were directed mainly at medicine, but not entirely, and they certainly are worthy of thought of the language and philosophy. But he broke them down into these nine areas which one could somewhat segregate one from another, and then his suggestion was to run through this checklist, if you will, and see whether there was a link between the two areas of concern, and if there was a link that would be a positive, and if there wasn't a link, it would be neutral or negative. And that's the method by which the Bradford-Hill criteria can be used.

N.T. 2/11/08, p. 128, A.M. Session.

As part of his approach, Dr. Mark says he considered strength of association. (N.T. 2/11/08, p. 129, A.M. Session). In determining strength of association, he believed that case reports were very important in establishing the link between asbestos and DMM. (N.T. 2/11/08, pp. 13-14, P.M. Session). He also admitted to the use of epidemiology as part of his Bradford-Hill analysis. (N.T. 2/11/08, pp. 38-39, P.M. Session). His appreciation of the use of epidemiology in this area of friction products was described by him in connection with a study that he relied upon:

> Q. And are you also aware of strength of association of epidemiological studies, even recent epidemiological studies, that study friction workers--excuse me--those workers exposed to friction products and mesothelioma?
> A. Repeat that, please.
> Q. Well, we had been talking about strength of association?
> A. Yes.
> Q. And that was one of the factors that you went through in your analysis. We had talked about case series reports that you looked at with respect to workers exposed to friction products who had been reported to have developed mesothelioma. And then I wanted to move to epidemiological studies?
> A. Yes. Well, I had -- I think I had already stated the ones that I was -- Dr. Leigh, Greenberg, Jarvholm, Hansen, for example.
> Q. And can you flesh out for us a little bit what Dr. Leigh's study was?
> A. Dr. Leigh in Australia reported -- I think it was more than a thousand cases of mesothelioma, diffuse malignant mesothelioma. And it was an epidemic. It is an epidemic in Australia, which has a high incidence of the disease.
>
> And he broke out the occupations, essentially. And there were maybe 30 possibilities or more, 30 occupations. And then he gave an expected incidence of the disease in that particular occupation.
>
> And in the occupation that was called brake workers or brake mechanics or auto mechanics, he had, I think, about 60 such cases.
>
> This article was entered into litigation. And, as I understand, under oath he said, Well, it wasn't -- we shouldn't count 60; but we should really count 40, which is still a large number.
>
> He was asked or I think he said it wasn't an epidemiologic study; but it satisfies the criteria for an epidemic and the study of an epidemic. So it was an epidemiologic study, in my opinion, even though he said under oath it wasn't.

Q. Dr. Mark, is 40 -- the 40 mesotheliomas that Dr. Leigh testified that he found, what significance does that have to you in terms of -- excuse me-- that Dr. Leigh testified that he found among those exposed to brake friction products, what significance does that have to you in formulating your opinion with respect to whether or not exposure to brake friction products could lead to the development of malignant mesothelioma?

A. It influenced me. They do.

Q. And how so?

A. They do so, as did other products, through asbestos. Brakes, per se, don't cause mesothelioma. Work with brakes doesn't cause mesothelioma. Work with brakes that doesn't release asbestos doesn't cause mesothelioma.

The methodology is not about brakes, per se, or a company, per se. Methodology, as we understand it today scientifically, is asbestos; asbestos, and the disease is the methodology. And then to go beyond that, you can use history from the patient; but it shows that the brake work that released asbestos caused the disease.

Q. So just to clarify, when a worker is -- like Mr.-- like the Plaintiff at issue here is doing work, like Leslie Duke, is doing work with brakes, and he is then caused to be exposed to respirable asbestos fibers by means of that work, that, in your opinion, would lead to exposures that could be significant such that they would be a significant contributing factor towards the development of mesothelioma?

A. Yes. And beyond that, a criticism-- another criticism of Dr. Leigh's study was -- well, many occupations, as I said before, they are so listed. So how do we know that there weren't exposures in some other occupation? And the answer is, We don't.

On the other hand, you could use the same argument with Dr. Selikoff's original work about the insulation union or take Mr. Mancuso's work about mesothelioma, that cohort being particularly high incidence of malignant mesothelioma.

Dr. Mancuso didn't take a history saying, Did you ever work with brakes or not? He never took that history. It was never reported. But these were poor individuals doing very dirty work on the railroads in West Virginia. And it would be surprising if some of them, being mechanically inclined, weren't working with brakes.

So you can say, Okay, Dr. Leigh didn't exclude other exposures; but you could also say Mr. Mancuso didn't exclude other exposures. It works both ways. And I bring that out because it's a criticism that's raised, but

you have to think about it in that way.

N.T. 2/11/08, pp. 21-23, P.M. Session

Dr. Mark, in concluding his direct examination by Plaintiffs, related how he believed the Branford-Hill criteria as reflected in *Epidemiology for Public Health Practice* by Friss and Sellers, allowed him his conclusion:

> Q. I want to wrap up, then, with the book you referenced earlier, the book, Epidemiology For Public Health Practice by Robert Friss, F-r-i-s-s, and Thomas Sellers, the third edition, published in 2004, Jones and Bartlet, publishers.
>
> You had referenced this as also recommending the application of the Bradford-Hill Criteria as a methodological approach to see if a certain agent was a causative factor with respect to disease; is that correct?
>
> A. Yes.

N.T. 2/11/08, p. 37, P.M. Session

> Q. And then going back to the book, did you, like the authors of this book, use the Bradford-Hill Criteria in assisting you in the methodological analysis of Mr. Duke's causative relationship between his friction asbestos exposure and mesothelioma?
>
> A. I used it, and the authors talk about it. I don't know that I could say they used it, but they advocated it.
>
> Q. Okay. And I want to show you what the author's conclusion was with respect to selected lists of environmental disease agents and examples of links. See, here the authors of this book say, Type of agent, asbestos. Examples of exposure, brake linings. Health effect studied, mesothelioma.
>
> Do you agree with the conclusions of these authors of this 2004 textbook with respect to asbestos deriving from brake linings and the resulting health effect being mesothelioma?
>
> A. Yes. Now, what's unusual about that table is that usually an author would say for the first example, Insulation work or construction materials, which they use as a second; and brake linings might or might not appear.
>
> Here the authors put number one, brake linings. So I'm not saying that brake linings are the major cause of diffuse malignant mesotheliomas. They are not. But in this author's idea, it was so important or let's say it was important enough that it was the number one listed occupation or product.
>
> Q. And in your medical opinion, Dr. Mark, was Mr.

> Duke's exposure to brake linings and friction products and asbestos derived there from a substantial contributing factor in Mr. Duke's case towards the development of his diffuse malignant mesothelioma?
> A. Yes. His total exposure to asbestos caused his disease. And among those total exposures, the exposures to brake dust was part of it. And so they were a significant contributing factor.

N.T. 2/11/08, pp. 39-40, P.M. Session

The above excerpts from the direct testimony of the expert, Dr. Mark, are presented to demonstrate that the claimed methodology simply does not exist or is so convoluted and inherently contradictory so as to defy any comprehension.

But, assuming for argument's sake, the witness put forth some vague attempts at establishing a methodology, it is clear that whatever was offered was merely an attempt to create an illusion of methodology which, in the end, lacked any substance that could be considered as generally accepted in the relevant scientific community.

Initially, the witness' own words reveal that he comes to the "right" conclusion first and then decides which "methodology" will support the conclusion.

> I don't know that experts are required to do anything. But in particular, I don't see a methodology as being in the singular, answering the question, because methodology, the beauty of science is that you can apply different methodologies. Although the word is used in different manners, you use different methodology's [sic] and you see which of them fit best, *which of them work best, and which of them in total come up to the right conclusion.*
>
> So, to say the methodology, I wouldn't agree with that, and I wouldn't agree with required, and I wouldn't agree with epidemiological as the methodology.

N.T. 2/11/08, p. 105, A.M. Session (emphasis supplied).

When asked about his "methodology" of analyzing the frequency, proximity and duration of Mr. Duke's exposure to Defendant's friction products, the witness disclosed this:

> Q. So, just to clarify here, Dr. Mark, are you saying that the total exposures, all of the exposures that occurred that Mr. Duke had, to asbestos, were enough to cause him to have diffuse malignant mesothelioma, that his dose was enough, all of the exposure?
> A. Now, as I hear the question, there's two possible questions in there.

Q. Sorry.
A. If you mean, Mr. Duke, No. So—
Q. I mean Mr. Duke.
A. Mr. Duke, the answer is yes.
Q. Okay. And here's the question, and here's the critical distinction. Are you, in any way, saying by means of that sentence, or in any of your opinions, are you in anyway saying that Mr. Duke having a breath of exposure to friction products would be a substantial contributing factor toward his development of mesothelioma?
A. If that's the whole story. No, it wouldn't.
Q. And are you -- when you look at analyzing whether or not in Mr. Duke's case, the friction products were a substantial contributing factor toward the development of Mr. Duke's mesothelioma are you going to analyze the frequency and proximity and duration of his exposure to those friction products prior to opining with respect to the significance of that exposure toward his development of mesothelioma?
A. If asked the question on it, yes. To the degree I am supplied with that information. Yes. And I would say as follows, the greater the duration, the greater the frequency, the greater intensity. To that degree, all of those exposures with that knowledge have a greater contribution than some other exposure that has a lesser frequency, a lesser intensity, a lesser duration.

However, since the exposures have occurred years ago, it's difficult to apportion. But to say it's difficult to apportion doesn't mean it's a black box, or that it's a hole, or that it's hopeless. You can usually have numbers of exposure either in terms [sic] have beginning in 1960 and extending ten years, compared to an exposure beginning in 1970, and extending one year. That's a way to quantitate them that's usually accurate. Another way, is there an exposure each day, once a week, once a month? So, once a week is more than once a month. You can consider that.

Thirdly, is it dusty or not? Usually that's all we have going back many years. But we have dust or not dust as observed. And those are the direct evidences that we have. Then the indirect evidence is all from the literature. The literature says, this was a fiber that we think would have occurred under those conditions, but that's not. That's part of the understanding, but it's not -- can't be necessarily applied to an individual patient usually, because rarely do we have those figures. But we do have the figures of duration, intensity, and as I have explained.

N.T. 2/11/08, pp. 116-118, A.M. Session.

Although the witness referred to the frequency, regularity and duration issues, he offered no explanation of how he used his "methodology" to arrive at his conclusion and it was clear that he was merely reiterating his version of each and every exposure in his testimony and nothing more.

In discussing his methodology regarding the different fiber characteristics, the witness offered his observation but failed to include any methodology that he employed in arriving at any conclusion which considered the admitted differences.

> THE COURT: Doctor, with respect to the middle part, which you call the hard science part, molecular, genetic, and tissue culture alternative annals studies, has it been your experience that a fiber, a specific type of asbestos fiber retains it's characteristics and integrity throughout the process of exposure through development of pre malignancy and then into malignancy, such that if one looked at a tissue sample, could one determine whether or not there were certain types of fibers present in the tissue, and to the extent and degree how much of the fiber retained is concentrated in a separate--in the tissue under study.
>
> THE WITNESS: Yes, Your Honor. There's a--much work has been done in this field, so let me just make sure that I cover everything I want to in your answer.
>
> First of all, asbestos is relatively impervious to heat, and to acid, but fibers are cleared and dissolved from the lung over time.
>
> Secondly, fibers tend to be coated in the lung as a sort of self protective mechanism, and that's when an asbestos fiber becomes an asbestos body.
>
> Thirdly, and most germane to your question is, the disappearing rate of the various fiber types has been studied, and they disappear at quite remarkably different rates. So, if you do a fiber digestion burden, you can analyze, to keep it simple, the three major fiber types, chrysolite [sic-crocidolite], amosite and chrysotile. But chrysotile has a half life in tissue in animals, and presumably in humans measured in weeks, six weeks, twelve weeks. You can find this literature in the annals of the New York Academy of Sciences in 1964.
>
> Six to twelve weeks, the half life of the other fibers, amosite and crocidolite are measured in months or years. And I wouldn't explain half life. Or should I explain half life?

N.T. 2/11/08, pp. 119-120, A.M. Session.

The Doctor was asked about how animal studies figured into his methodology and

again he failed to relate any understandable process by which such studies informed his conclusion regarding friction product asbestos other than "asbestos in all of its forms cause cellular changes in animals" which is another version of his underlying opinion that all asbestos in all of its forms causes mesothelioma.

> Q. Okay. And what about animal studies? How did those influence your opinion? Did you use those as part of your methodological process in providing your opinion today with respect to Mr. Duke?
> A. Animal studies, as opposed to tissue culture studies where you are looking at an intact animal, we know how to induce tumors. There is contradictory evidence there because not all animals will develop mesotheliomas. Is the mesothelioma that develops in an animal the same as one in a human and how does it differ? Can other substances injected in animals cause tumors? Do substances inhaled cause as much tumor as substances injected?
>
> So there are books written on this, hundreds of articles. But the underlining theme is asbestos in all of its forms causes cellular changes in animals, and sometimes not just cellular changes, but tumors that are similar to the human tumor.
> Q. And that would include asbestos, chrysotile asbestos, arising from friction products such as brake product; is that right?
> A. A chrysotile fiber doesn't know where it came from, either in the air or in the body; and it doesn't care. If it came from a brake, fine. If it came from a gasket, fine. You can't find and identify the chrysotile.
>
> One, you have to know that chrysotile causes the disease. Two, you have to know that brakes at one time contained chrysotile. Three, you have to know something about the mechanics, the industry of auto mechanics and brake repair. You have to know-- those are the three spheres that you have to know to make a conclusion.
>
> Do they all meld into one? No. They are different. As I said, we're using the language of these different spheres. And we're trying to say causation as in Socrates, A to B to C. Therefore, A to C. That's philosophy. That's applicable to nuclear physics. Methodology is very applicable to nuclear physics. But methodology in this is more complicated. We're not dealing with nuclear physics. We're dealing with humans. We're dealing with memory. We're dealing with industry. We're dealing with products, and with all the variables.
>
> So, I do the best that I can; but, of course, there is methodology. I mean, it's not that someone would guess. That's the answer.

N.T. 2/11/08, pp. 31-34, P.M. Session.

Dr. Frank was offered as an expert by Plaintiffs here, although he did not issue a written report.

Initially, it is noted that Dr. Frank disagreed with Dr. Mark on the issue of whether mesothelioma is generally accepted as idiopathic. (N.T. 2/12/08, pp. 25-28, P.M. Session). Critical to Dr. Mark's methodology was his belief that mesothelioma was not idiopathic, which in turn supported his belief that once mesothelioma was diagnosed, no further inquiry as to causation was necessary.

Dr. Frank signed on to the same methodology employed by Dr. Marks.

> Q. And when you say the basic methodology as outlined, Dr. Marks' methodology was consider diagnosis, consider latency, consider the Bradford-Hill Criteria, consider molecular studies, consider what he termed genetic studies, consider tissue culture studies, animal studies, case reports, experiences as a diagnosing doctor and epidemiology? Did I get that list right?
> A. You got the list right. And there is something about every one of those that I've been involved with.

N.T. 2/12/08, p. 33, P.M. Session

In addition to the above criteria, Dr. Frank included his life experiences which taught him that every asbestos exposure could cause an asbestos disease.

> Q. Let's talk some about how you consider and if you consider your life experiences as an occupational medicine physician. Do you consider that?
> A. Yes. I think of all the cases that I've seen. There have been so many different ways in which I have learned that people have been exposed to asbestos. And what I've learned is that no matter the variety of exposures, *there is no exposure that I have heard about that I wouldn't think could have the potential to cause disease.*

N.T. 2/12/08, pp. 42-43, P.M. Session (emphasis supplied). Although stated somewhat differently, this is the each and every breath theory of causation.

Dr. Frank, in response to the first question by counsel on cross-examination, repeats the each and every breath opinion but adds the qualification that the level of exposure must be above background.

> Q. Dr. Frank, would I be correct if I characterized

your testimony and your opinion in asbestos cases that each and every breath substantially contributes to asbestos disease?

A. Each and every breath that has levels above background would be contributory to someone--someone's disease if they developed an asbestos-related disease.

Q. So as long as they have an asbestos-related disease and exposure, if I came to you and I asked you an hypothesis: To assume that somebody was diagnosed with a lung cancer that could be related to asbestos, and they had just background exposure and one brake change, that would be enough for you to say that the brake change was a substantial contributing factor?

A. There is no way to say that all of the exposures that they had wouldn't have contributed to their disease. What I have always testified is that if I only had just background exposure, I could not testify in a given case with a reasonable degree of medical certainty that that case was caused by asbestos.

But when you provide me evidence of exposure above background, then it seems reasonable and logical, understanding how carcinogens work, to say that that was a substantial contributing cause.

N.T. 2/12/08, pp. 79-80, P.M. Session.

This answer proves to be illusory because the witness then goes on to state that:

THE COURT: Once the disease is contracted, then all of the exposures would play a role in causing that disease?

THE WITNESS: All of the units of the material that caused the disease would have to be said to cause or have the potential to cause an equal role.

N.T. 2/12/08, p. 90, P.M., Session.

The witness then goes on to repudiate the notion of a threshold in no uncertain terms:

THE COURT: But according to your theory, one exposure to one fiber causes a disease, there is no threshold; correct?

THE WITNESS: For cancer-causing agents. All the examples I've given you with a threshold are not for cancer. Cancer is a different type of disease. And when a material is a carcinogen, has the ability to cause cancer--there is no evidence for any carcinogen that I am aware--for any carcinogen--I have published fairly widely on a wide range of carcinogens and I've studied dozens of

> carcinogens--there is no evidence of a threshold.
>
> It's a different kind of disease because what you are doing is altering the DNA of a cell, which may or may not end up fully maturing into a cancer.
>
> And, theoretically, a small amount can do the genetic change that ends up with cancer.
>
> THE COURT: Such as background exposure?
>
> THE WITNESS: There is nothing different about a fiber in the background than a fiber from any asbestos-containing product.
>
> I would--if I was in front of a medical audience, I would answer that there is no difference, as I'm answering to you.
>
> What I understand--and again, I'm not a lawyer or an attorney. But when I come to court and have to testify with a reasonable degree of medical certainty in a specific case, allowing for other possibilities, unless I have evidence of something above background, since we all have background exposure, I would never say that only background caused the disease, even though as a medical person I recognized that it could. And some of those cases said to be idiopathic may, in fact, have been caused by asbestos; but I have no way to prove that. And so, therefore, would never testify to that.

N.T. 2/12/08, pp. 92-94, P.M. Session.

Dr. Frank also rejected any consideration that the type of asbestos fiber played any role in his analysis of causation.

> Q. So in my hypo, your answer would be, yes, that that one brake change was a substantial contributing factor?
>
> A. Understanding carcinogenesis theory as I do, I would have to say yes.
>
> Q. Okay. And it would be fair to say in the past, you've testified that's the case regardless of intensity of exposure, type of exposure or type of fiber?
>
> A. If it fits the hypothetical you just gave me where the intensity is unknown, the type of fiber certainly doesn't matter, and what was the third one? Frequency? One brake job theoretically could do it. The likelihood would be extremely small, but it's not zero.

N.T. 2/12/08, pp. 80-81, P.M. Session.

The Plaintiffs also offered William E. Longo, Ph.D. Dr. Longo is the President of Materials Analytical Services, Inc., and holds a Ph.D. in Materials Science and Engineering from the University of Florida.

Subsequent to the filing of the Motion requesting a *Frye* Hearing on August 17, 2007, the witness submitted an expert report on December 3, 2007, in the *Duke* matter. The report is identified as P-5. Initially, there was an objection to Dr. Longo's report and subsequent testimony based upon relevancy to the issue before this Court and his qualifications to opine therein.

> Ms. Johnston: Your Honor, I am sorry, we also have a motion this morning, copies have been provided to opposing counsel, to preclude the testimony of Dr. Longo today. I indicated yesterday in opening statements that Dr. Longo's qualifications, and his area of expertise seems inappropriate here. There was an indication yesterday that Dr. Mark relied on Dr. Longo's opinions, but in fact, Dr. Mark's testimony was that he read the opinion for the first time on Sunday. His expert opinions were authored in November and December of last year.
>
> But Dr. Longo is a material and science engineering expert, and the subject here is the general acceptance of the methodologies of causation experts in the field of pathology, occupational medicine, epidemiology, and so forth. It seems that Dr. Longo is not a member of the relevant scientific community, the general acceptance which is at question here.

N.T. 2/12/08, pp. 4-5, A.M. Session

Plaintiff responded by explaining that Dr. Mark had relied upon Dr. Longo's report in his testimony:

> MS. CLANCY: As Dr. Mark testified yesterday his methodology entailed analyzing the amount entailed first taking into consideration that asbestos particles were emitted upon work with friction products. As a basis for that methodology he was, in part, relying on what Dr. Longo's analysis of the friction products, thus--
>
> THE COURT: And what part, given that he didn't read the report, and when he issued his report.
>
> MISS CLANCY: But his opinion was two parts. Remember, his report was only one part of his opinion, i.e., did the aggregate dose that Mr. Duke received through the lifetime of his career lead to the development of his mesothelioma? He had a second and absolutely critical part of his opinion which was, were the exposures to the friction products a substantial contributing factor toward the development of the mesothelioma? It is that opinion which is at issue in these Frye hearings, because the issue is, is he using acceptable methodology to analyze whether or not the exposure to the friction products was a substantial contributing factor toward the development of

> mesothelioma? As the court may recall, a central theme of defendants' motion was the, quote unquote, each and every breath theory.
>
> Dr. Mark came here yesterday, testified that part of his methodology was an understanding of the fact that asbestos dust particles were emitted from these friction products, not just in the particle by particle level, but by the millions of particles. And that those millions of asbestos particles were a substantial contributing factor toward the development of the disease.

N.T. 2/12/08, pp. 6-7, A.M. Session

The Court allowed the testimony to go forward reserving on the issue of relevancy and the doctor's qualification. At the beginning of the next day of the hearing, the Motion to Preclude on Qualification was denied. (N.T. 2/13/08, p.4, A.M.Session). The issue of relevancy to the *Frye* Motion is now discussed.

A review of the Report shows that it goes extensively into Mr. Duke's work history in an apparent attempt to demonstrate some comparative level of exposure between friction and non-friction products during Mr. Duke's work life history. The end point to this history discussion is missing since Dr. Longo never makes any connection between Mr. Duke's exposure and his disease. The reason for this is clear and was admitted to by Dr. Longo in his testimony:

> Q. Let me ask you a few background questions, Dr. Longo.
>
> You have a Ph.D., correct?
>
> A. I do.
>
> Q. The Ph.D. is in material science, is that correct?
>
> A. That is correct.
>
> Q. You're not a medical doctor?
>
> A. I am not.
>
> Q. You're not qualified to give medical opinions regarding the effects of exposures?
>
> A. That is correct.
>
> Q. Your area of expertise is not epidemiology?
>
> A. That is correct.
>
> Q. And you don't provide opinions about the health effect of asbestos, is that a fair statement?
>
> A. That is fair.
>
> Q. And that's because you don't consider yourself to be an expert in assessing the health risks from exposure to asbestos?
>
> A. It's not an area that I testify about in court.

N.T., 2/13/08, pp. 93-94, A.M. Session.

The testimony was offered in anticipation of other expert testimony which was perceived as presenting evidence related to there being, "no barely miniscule amount of asbestos fibers emitted from working with asbestos products." (N.T., 2/12/08, p. 8, A.M. Session).

The substance of Dr. Longo's testimony was that under specific conditions, certain activities create "significant levels of airborne asbestos fiber." (Exhibit P-5). Dr. Longo further opined that Mr. Duke was exposed to these fibers. "In my opinion, within a reasonable degree of scientific certainty, that Leslie Duke had significant exposure to airborne asbestos fibers from asbestos-containing friction products." (Exhibit P-5).

Dr. Mark admitted to not reading this report prior to issuing either of his expert reports and, therefore does not rely upon Dr. Longo in arriving at his conclusions as to causation.

> Q. You testified at length about use of the Bradford-Hill Criteria.
>
> Let me --the slide that was used, is that a slide that you prepared or was that one the lawyers prepared on Bradford-Hill?
>
> A. I didn't--the slides that were used today, I didn't prepare any of them.
>
> Q. And, again, the report that you issued first in November and then again in December, did you reference Bradford-Hill in either one of those?
>
> A. No.
>
> Q. Did you reference Dr. Longo's work in either one of those reports?
>
> A. No. As I indicated earlier, I hadn't seen Dr. Longo's report until last night.

N.T. 2/11/08, pp. 73-74, P.M. Session.

Dr. Longo had prepared his Report on the amount of asbestos fibers, based upon his experiments conducted in his own laboratory, Materials Analytic Services (MAS). Dr. Longo is President of MAS. (N.T. 2/12/08, pp. 11-12, A.M. Session). The experiments were conducted in a test chamber, "roughly 20' x 15' x 8'" located at the MAS facility. (N.T. 2/12/08, p. 95, A.M. Session). The experiment was not an actual workplace simulation. (N.T. 2/12/08, pp. 96-98, A.M. Session)[3].

When asked about Dr. Longo's studies, Dr. Marks replied in part:

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3. In addition to not being workplace simulations, Dr. Longo's work suffers from other defects which for a number of reasons, will not be further discussed here, because as will be discussed below, Dr. Longo's conclusions are not relevant, not relied upon.

> THE COURT: I didn't--you don't have to answer it if you don't understand what the methodology was. I just asked if you could tell by reading the report what methodology he employed in arriving at his conclusions.
>
> THE WITNESS: Yes, Your Honor, I can tell. It's just that he may use different words, of course.
>
> So his methodology was, first, he was using the methodology of an occupational or industrial hygienist because he had a history of the various occupations and the exposures. And he described them and quantitated them.
>
> So, methodology, first of all, would be an industrial health physician in that regard. And he used as his methodology, knowing about the composition of brakes. And that would be methodology having to do with fiber studies. These would be experimental studies. His experimental studies, in particular, being measuring fiber release under various conditions and manners.
>
> And he attaches what he calls reliance documents. And these are documents that are quantitative analyses. There are experimental in one regard because they are not in live situations. So let's say they are experimental in that regard.

N.T. 2/11/08, pp. 42-43, P.M. Session.

After an exhaustive direct examination by Plaintiffs, Dr. Mark failed to show how Dr. Longo's testimony, experiments and conclusions were relevant to his opinion on causation and, in the final analyses, could do no more than equate Mr. Duke's exposure to friction products to all of his other exposures to asbestos products generally.

> Q. And in your medical opinion, Dr. Mark, was Mr. Duke's exposure to brake linings and friction products and asbestos derived therefrom a substantial contributing factor in Mr. Duke's case towards the development of his diffuse malignant mesothelioma?
>
> A. Yes. His total exposure to asbestos caused his disease. And among those total exposures, the exposure to brake dust was part of it. And so they were a significant contributing factor.

N.T. 2/11/08, p. 40, P.M. Session.

Therefore, Dr. Longo's Reports and testimony are precluded and stricken and have not been considered in this *Frye* analysis.

Defendant's Experts

Pursuant to the Order of this Court of December 27, 2007, scheduling this *Frye* Hearing, the Defendant presented certain experts who opined on the issues of the

Plaintiff's expert's methodology in arriving at the conclusions as to causation.

Defendant presented Dr. Dennis Paustenbach, whose professional accomplishments included being a toxicologist, an industrial hygienist, a risk assessor and a chemical engineer. His professional degrees and certifications include, but are not limited to, a Master in Science Degree in Industrial Hygiene from the University of Michigan, Ann Arbor and a Ph.D. in Toxicology from Purdue. He has a Board Certification in Toxicology. He has a Specialty in Industrial and Environmental Toxicology, Occupational Health and Risk Assessment. He is an Adjunct Professor at the University of Michigan and the University of Massachusetts. (N.T. 2/13/08, pp. 55-59, P.M. Session). Dr. Paustenbach has been a Peer Reviewer[4] and has published and contributed to the textbooks used in the Risk Assessment academic field. (N.T. 2/13/08, pp. 62-64, P.M. Session). Dr. Paustenbach was qualified as an expert in Industrial Hygiene, Toxicology and Risk Assessment without objection. (N.T. 2/13/08, p. 66, P.M. Session). He was asked to opine on a number of issues regarding the methodology of Plaintiff's experts.

The opinion with which we concern ourselves now, focuses on the "each and every breath, each and every exposure" theory postulated by Plaintiff's experts. Dr. Paustenbach initially identified four (4) primary flaws in these opinions:

1. The opinions are not supported by known empirical data and none were identified in their opinions.

2. There is no generally accepted scientific data that demonstrates that background exposure to asbestos increases the rate of cancer.

3. The "every dose contributes" theory is a version of downward extrapolation which is not generally accepted in the relevant scientific community.

4. Use of regulatory agency statements about no safe level of exposure is not generally accepted in the relevant scientific community as a basis for supporting causation opinions. (N.T. 2/13/08, pp. 136-138, P.M. Session).

Dr. Paustenbach also found methodological flaws in the Plaintiffs' experts' conclusions that, "low levels of asbestos fiber are capable of producing mesothelioma and other malignancies" and the use of lab experiments using animal and human data and case reports to support these conclusions. (N.T. 2/13/08, p. 149, P.M. Session).

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4 A person knowledgeable in a subject field who is asked by the Editor of a journal or document literature to read a specific article for publication and identify delivery, shortcoming in design, shortcoming in presentation or analysis. (N.T. 2/13/2008, p.62, P.M. Session).

The methodological flaws in the process of arriving at these conclusions are identified as the failure to define the meaning of "low levels;" the failure to distinguish between the various types of asbestos fibers; the failure to distinguish between studies based upon doses in animals which are not relevant to human causation issues and the failure to account for or explain negative studies showing little or no risk of mesothelioma from chrysotile exposure. (N.T. 2/13/08, pp. 149-150, P.M. Session).

The issue of asbestos fiber type and the failure to address it in the experts' opinions on causation is a fatal flaw in their methodology. To understand why this is so, a review of the types and their respective qualities is necessary.

For our purposes, there are three (3) types of asbestos fibers. The first is white asbestos or chrysotile, which is soft, flexible and curly; second is amosite, which is brown with straight spike-like fibers which are chemically different; third is crocidolite, which is blue and a straight needle-like structure. (N.T. 2/14/08, pp. 10-11, A.M. Session). The amosite and crocidolite are generally considered to be commercial asbestos because of their use in applications like pipe and boiler insulation and pipe and gasket construction. (N.T. 2/14/08, pp. 11-12, A.M.Session).

The need to account for the different asbestos fibers is because of the differences in potency in causing mesothelioma specifically. One study relied upon by Dr. Paustenbach, (*Hodgeson 2005*), assessed potency[5] of 1 for chrysotile, 100-250 for amosite and 500 for crocidolite. (N.T. 2/14/08, pp. 14-16, A.M. Sessions).

As a result of the Court's question, the witness went on to explain the biological significance of the different characteristics:

> Q. Now, let's go back to this. Is it a fair characterization then to say, when either Dr. Mark or Dr. Frank opines that each and every exposure causes or contributes, that there's a flaw, a fundamental flaw in that methodology.
> A. If they don't discuss fiber type, it's a gross generalization.
> THE COURT: Can we just go back for a moment? I think I want this area maybe flushed out a little bit more. And that is, what in the different characteristics of the relative fibers would biologically be responsible for the difference if we accept the difference, in potency?
> THE WITNESS: That's an excellent question.
> It's not fully resolved. I can tell you that the

5. For our purposes, potency is defined as the differences in risk of developing mesothelioma given the same airborne concentration of chrysotile, amosite or crocidolite fibers.

various hypotheses. The first one is solubility. That's many people think that's the first line as to why chrysotile is less potent. And when you --it's biologic half life in the lung is much less than for either amosite or crocidolite. In other words, it's just more chemically inert or -- yes, it's less chemically inert. In other words, it will break down quickly.

Okay, its biologic half life is in days in rats. Whereas it's months or years for amosite and crocidolite. So, it stays longer. The second is it's chemically less difficult to break down by the macrophages. You have heard about the macrophages in the lung engulfing these fibers because they are seen as foreign bodies, they release acid to try to destroy it. And they are less able to destroy amosite and crocidolite. They're able to -- less able to break it down just as in a chemical plant, that's why we select amosite and crocidolite, sometimes.

So, they're known to be less resistant to destruction. Some have said its clearance time. And then the other theories are less clear. In fact, the others are that if it penetrates the cell it's more likely to cause a mutagenic event based on -- for chemical reasons.

Those are probably the three primary hypotheses as to why they act differently in the body.

* * *

Q. But in terms of this fiber potency --

* * *

A. The importance of resistance to breakdown and time in the lung means that there's a possibility for repeated irritation of that fiber in the cell, and repeated irritations as a cause of cancer. Okay? And that's called a non genotoxic mechanism. Even though it causes the tumor, it's not necessarily initially due to an interaction with DNA that would replicate and cause a cancerous cell.

N.T. 2/14/08, pp. 16-19, A.M. Session.

Another methodological flaw of Plaintiff's experts is the failure to distinguish the various fibers according to length.

Dr. Paustenbach reviewed some of the scientific literature which identified the importance of fiber length and the conclusions therein which demonstrates that fiber length is directly related to the potential to cause asbestos related cancer. (N.T. 2/14/08, pp. 21-32, A.M. Session). The witness also identified the epidemiological information supporting the position and referred to animal studies which concluded that the longer fibers were less easily engulfed by the macrophages whereas the shorter fibers (chrysotile) were more easily engulfed and therefore not a factor or a significantly

diminished factor in causing cancer. (N.T. 2/14/08, pp. 22-23, A.M. Session).

The witness also identified the methodological flaws resulting from Plaintiff's experts' failure to account for background exposures to asbestos. (N.T. 2/14/08, p. 33, A.M. Session). Background exposure was identified as the amount and type of asbestos fiber in the ambient air to which all persons are exposed; the concentration of fiber varied according to geographical location. (N.T. 2/14/08, pp. 34-35, A.M. Session). In certain areas of the country, the source could be fresh outcroppings of serpentine rock which is a natural source of asbestos fiber or the industrial or construction activity in cities where airborne asbestos fibers were caused by the use of sprayed insulation. (N.T. 2/14/08, pp. 34-35, A.M. Session). Non-airborne asbestos can also be found in drinking water which picks up the fibers from the pipes through which it flows and from the rocks over which the water courses on its way to becoming drinking water. (N.T. 2/14/08, p. 36, A.M. Session).

Although Plaintiffs' experts at times appeared to make reference to the concept of background levels, their methodology failed to explain how such levels were considered in arriving at their conclusions as to causation. The presence of asbestos in the ambient environment takes on added importance in light of certain lung tissue fiber burden analysis which demonstrates the presence of significant fibers in the non-occupationally exposed general populations. (N.T. 2/14/08, pp. 36-38, A.M. Session). The opinions proffered by Plaintiffs' experts that each and every exposure to asbestos causes or contributes to the disease without an explanation of the contribution background exposure is flawed necessarily because of this omission.

In the Plaintiffs' Expert's opinions, both written and testimonial, there were references to the use of epidemiology as part of their respective methodologies in arriving at their conclusions that chrysotile fibers somehow generated by the use of friction products were a substantial cause in the asbestos disease or cancer acquired by Plaintiffs herein. Dr. Mark initially placed this at the bottom of his *Bradford-Hill* analysis but later admitted that it should be at the top of the hierarchy of evidence considered. Likewise, Dr. Frank gave it some secondary consideration which he felt could not compare to his lifetime experience when considering the relative weight of evidence considered. Dr. Longo flatly refused to consider certain epidemiological evidence and he arbitrarily dismissed studies without considering the merits thereof where he deemed them not worthy of consideration. The use of epidemiological studies as part of one's methodology in determining causation in the area under scrutiny must be understood in

the context of the physical and medical limitations inherent in the human evidence available in these cases.

It is undisputed that asbestos fibers are ubiquitous and fungible. It is conservatively estimated that an adult male in the United States may have hundreds of thousands of asbestos fibers in each gram[6] of lung tissue. (N.T. 2/14/08, pp. 36-38, A.M. Session).

These asbestos fibers include chrysotile, amosite, crocidolite and tremolite. (N.T. 2/14/08, pp. 37-38, A.M. Session). Because of the volume and diversity of asbestos fiber found in the environment and in non-occupationally exposed individuals, it must follow, *a fortiori*, that there can be no direct causal chain of evidence as to the identity of any or all of the asbestos fibers which were responsible for the occurrence of the disease. In these circumstances the science of epidemiology is particularly effective in determining causal relationships.

To define the role of epidemiology in the methodology of causation, Defendants presented Dr. Mary Jane Teta, a chronic disease epidemiologist specializing in occupational epidemiology and cancer epidemiology for approximately thirty (30) years. (N.T. 2/14/08, p. 72, P.M. Session). Dr. Teta has a Master's Degree in Public Health and a Doctorate in Chronic Disease in Epidemiology from Yale University. She has worked extensively in the industry of her field and has published widely. She is well qualified to opine in the area of occupational epidemiology.

In the relevant scientific community, it is understood that epidemiology is divided into two (2) disciplines. One is the descriptive study of the patterns of occurrence of diseases; the other is the analytic study of the causation of diseases, also considered as the science of causation of diseases in human populations. (N.T. 2/14/08, pp. 91-92, P.M. Session).

Within the framework of analytic epidemiology, the ultimate object is to determine if there is a statistically significant association between exposure to the hypothesized causal agent and the occurrence of the disease. (N.T. 2/14/08, pp. 96-97, P.M. Session)

In order to determine statistical significance, you must find the rate of disease in the exposed group and divide it by the rate of disease in the unexposed group.

In epidemiology, a relative risk of one (1.0) means that there is no statistical

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6. For comparison sake, there are about 28.35 grams in an ounce (avoirdupois). *Wikpedia.org.*

significance to the association. A relative risk greater than one (1.0<) indicates a positive association, while a relative risk less than one (>1.0) indicates a negative association. In other words, when the relative risk is equal to one (1.0), the disease occurs at the same rate in the unexposed population as it does in the exposed population. (N.T. 2/14/08, pp. 96-97, P.M. Session)

Using these concepts, this court offers the following hypothetical to explain. Let's assume that we are trying to find out if there is a statistically significant association between lung cancer and drinking three (3) 16oz. bottles of grape soda per day. From the public record we determine the population that drinks grape soda (exposed group). Followed over time, it is determined that this population gets lung cancer at the rate of 4%. We next find the population that does not drink grape soda (unexposed group) and determine over time that this population gets lung cancer at the same rate of 4%. The calculation is 4% ÷ 4% = 1. (A relative risk of 1.0 shows no association between lung cancer and drinking grape soda.) If we change the assumption in this hypothetical to show that the non-grape soda drinking population get lung cancer at the rate of 2%, the calculation is 4% ÷ 2% = 2. (A relative risk of 2.0 indicates that there is a positive association between lung cancer and drinking grape soda). In one final example we change the assumption to show that the non-grape soda drinking population gets lung cancer at the rate of 6%. The calculation is then 4% ÷ 6%= .667. (A relative risk of less than 1.0 indicates that there is a negative association between lung cancer and drinking grape soda).

Interpreting these results shows that in the first scenario there is no difference in the rate of lung cancer between grape soda drinkers and non-grape soda drinkers in the rate of lung cancer. In the second scenario, it appears that grape soda drinkers are twice as likely to develop lung cancer than the general population and in the third scenario, drinking grape soda appears to actually decrease the likelihood of contracting lung cancer.

The above hypothetical is an example of what is known in the world of analytical epidemiology as a cohort study. (N.T. 2/14/08, pp.93-98; 101-105, P.M. Session). In the science of epidemiology, it is generally accepted that there is a hierarchy of studies used to demonstrate a causative relationship between exposure to an agent and development of a disease. Because of the inherent characteristics of the design of the studies, some methods are stronger (more reliable) that others in predicting causation.

The cohort study is the strongest of the methods, followed by the case control

method.[7] (N.T. 2/14/08, pp. 93-100, P.M. Session). The cohort study design essentially determines a group which is exposed to the agent and a group not exposed to the agent and then determines the incidence of the disease at issue in both populations. (N.T. 2/14/08, pp. 96-96, P.M. Session).

Plaintiffs' experts testified about relying upon case reports for concluding causation. Case reports are observations or descriptions of an occurrence of a disease. Because case reports do not include a comparison or control group, they cannot be used to conclude cause and effect or causation. (N.T. 2/14/08, pp. 114-116, P.M. Session).

However, case reports do provide an observation which may be the basis for an hypothesis. This hypothesis is the beginning point for application of the scientific method which is the generally accepted method for determining causation.

The scientific method in this context begins with the observation of the disease, then the formation of a hypothesis as to causation. According to the generally accepted scientific method, the next requirement is to gather all the information that exists which contains epidemiological studies designed to test the causal connection between the exposure and the disease.[8] (N.T. 2/14/08, pp. 115-117, 119, P.M. Session).

Once this information is collected further analysis is required to determine if the studies provide sufficient, reliable information. This analysis includes determining the range of variability in design, the study base, statistical approach, choice of controls, geographic location and number of investigations. Required analysis also includes identifying the strengths and weaknesses of the studies, the confounding selection bias, information bias, precision and confidences intervals. (N.T. 2/14/08, pp. 120-121, P.M. Session). Another critical requirement is that the studies being relied upon have been published and peer reviewed. (N.T. 2/14/08, pp. 124, 147, 149, 158, P.M. Session; N.T. 2/15/08, pp. 36, 53, 54, 74-77, 80, A.M. Session).

Once a positive association is established, the relationship is analyzed in the context of other scientific disciplines such as toxicology, pathology, mineralogy and biologic activity. (N.T. 2/14/08, pp. 141-142, P.M. Session).

According to what is generally accepted in the scientific community, it is at this stage of the investigation where it is appropriate to apply the *Bradford-Hill* criteria. This

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7 Other study designs include proportion mortality and some others not at issue here. (N.T. 2/14/08, pp.126-128, P.M. Session).
8. In the classic application of the scientific method, this step is generally identified as conducting repeated experiments to test cause and effect. As previously shown, this cannot be done; primarily because of the human subject matter.

is in contradiction to Plaintiffs' Experts who testified that they used the *Bradford-Hill* criteria to determine association and causation. The Defendant's experts were consistent in their testimony that *Bradford-Hill* cannot be used to determine whether there is a positive association between exposure and disease. As Dr. Teta testified:

> A. Let's move on.
>
> Q. Dr. Mark, did you see his opinion that friction products exposures are a cause of mesothelioma based in part on the Bradford-Hill methodology?
> A. Yes, I saw that.
> Q. Okay.
> Is that a correct application of the scientific method?
> A. No, the Bradford-Hill, sometimes we call them criteria or considerations, were developed for --
> THE COURT: Initially, let me ask you a question. Is Bradford-Hill a medical diagnosis?
> THE WITNESS: No, Sir Austin --
> THE COURT: Just answer my question. Is it a medical diagnosis? Is it a method of diagnosing a medical condition?
> THE WITNESS: It is not.
> THE COURT: Very well. Go Ahead.
>
> BY MR. LOPEZ:
>
> Q. Okay. What is the problem with who is Bradford-Hill?
> A. Sir Austin Bradford-Hill was a biostatistician and epidemiologist who was asked to give a presentation to help scientists understand how to make a judgment when they have a clear cut positive association in a study, a human study, how to know whether that association implies causation or not. And clear cut, he means the relative risk was significant because he says it's so clear cut you don't expect it to be due to chance. Now, you have an association and a study, or a group of studies. The question is how do I make a judgment whether it's a causative relationship? And he provides considerations for considering that, and coming to interpretation.
> Q. First of all, you have an association, statistically significant, then the question is it a causal association?
> A. And what circumstances can we base from this observed association to a verdict of causation? And this is what I was just explaining, he said clear cut.
> Q. Okay. And that's Bradford-Hill criteria 1965 in defendants Exhibit 37.
> And the Surgeon General has also essentially-- what has the Surgeon General said?
> A. The Surgeon General's report on smoking used the Bradford-Hill criteria to come to a conclusion about

> causation, and says they are meant to be applied to an already established statistical association. If no association has been observed, then these criteria are not relevant. I mean, we know that.
> Q. Now Doctor, is it a correct methodology to use the Bradford-Hill criteria to evaluate whether or not motor vehicle mechanics are at an increased risk based on those epidemiology studies you showed us?
> A. Absolutely no, I wouldn't begin to use Bradford-Hill, and I don't find an association, it's simple.
> Q. So, you don't get over the initial hurtles to use it?
> A. You don't.

(N.T. 2/15/08, pp. 43-45, A.M. Session).

Dr. Teta also testified about issues that arise when a person has contracted a disease and there is a statistical illusion to the cause of the disease, masking the real cause of the disease. This is the epidemiological principal of confounding. (N.T. 2/14/08, pp. 111-113, P.M. Session). The example offered by the witness is illustrative:

> * * *
> But if suppose you want to know—again, back to coffee drinking, does coffee drinking cause heart disease? So you go ahead and you do a study. And you find a high relative risk that appears to have drinking coffee causes heart disease; but you didn't think about smoking. Because what do we know? We know that smoking causes heart disease. It's a risk factor for heart disease. We also know that smokers and coffee go together, don't they? People who smoke like to smoke with their coffee.
>
> * * *
> So, in any case, if you don't take smoking into account, it may appear that coffee drinking causes heart disease when, in fact, you have a confounder; and it's the smoking that is the problem. That's an example of confounding.

(N.T. 2/14/08, pp. 112-113, P.M. Session).

Defendant also presented Dr. Alfred Franzblau, who is an "esteemed medical doctor", researcher and a specialist in occupational medicine. He is a member of the faculty at the University of Michigan, a Professor in the School of Public Health, (also University of Michigan) where be teaches classes on diagnosing occupational environmental disease. There were no objections to Dr. Franzblau being accepted as an expert qualified in his field. (N.T. 2/15/08, pp. 48-51, P.M. Session).

On the issue of whether case reports, as used by Plaintiffs' experts, to support causation conclusions, Dr. Franzblau said that case reports cannot be used to support

cause and effect conclusions and that their importance lies in identifying potential new risks and conditions and by generating hypotheses for further study. (N.T. 2/15/08, pp. 68-69, P.M. Session). This reinforces what Defendants' other expert witnesses testified regarding the generally accepted scientific role of case reports in the study of the cause and effect of disease.

Dr. Franzblau outlined the generally accepted scientific methodology in determining causation in cases where an asbestos related disease is suspected.

The analysis begins with:

1. Diagnosis
2. Exposure
3. Dose
4. Mitigating Factors
5. Alternative Explanations
6. Conclusion

The witness evaluated the methodology used by Dr. Frank under the above criteria:

> Q. You told the Court earlier that you were able to review Dr. Frank's testimony about this case?
> A. I believe so, yes.
> Q. All right. And I think you said-- how did you characterize the methodology? I don't want to put words in your mouth. How did you characterize the methodology that you saw?
> A. Well, he apparently started with the diagnosis of asbestosis and then inferred, well, he must have had exposure. And it's kind of like putting the cart before the horse, rather than saying, okay, he might have fibrosis; and then let's see what may be the differential diagnoses of that lung fibrosis.
> Q. Did you--let's go to the next slide. Did you attempt to review what Dr. Frank's methodology would have reflected within the generally accepted framework?
> A. Well, I tried to. And this is how I came up with it: That in terms of the diagnosis, he started with asbestosis rather than fibrosis and plaques. He focused on exposure to asbestos from brakes. He certainly said asbestos can cause this disease, which I don't agree with. And then in terms of whether this patient was exposed sufficiently for this health effect to occur as a result--well, he opined that any dose above background is a cause. And --
> Q. Do you agree with that statement?
> A. No, I don't.
> Q. Why not?
> A. Because the published data don't support it. He says that all exposures can contribute to cause. And,

again, I don't agree with that.

He apparently never addressed item number 6, which is the idea of alternative explanation. And then he concluded that his exposure from brakes was an important contributing factor in his disease.

Q. In reviewing what Dr. Frank did, did you develop some opinions about his methodology that would inform this issue in the manner in which he went through the process or the lack thereof.

A. I didn't get the sense that there was an appropriate differential diagnosis and that he started with a diagnosis of asbestos.

He explicitly said at one point that dose is unimportant to him, which is sort of unbelievable. Dose is critical. Dose is everything.

He referred often to the Lorimer study from 1976, which is a case series. It has no control group; and, therefore, was incapable of demonstrating or evaluating risk.

And, in fact, if we go eight years later to the Nicholson study in 1984, Lorimer was, in fact, a co-author of the Nicholson study. And, as we pointed out earlier in the Nicholson study, they found that almost 19 percent of people had radiographic abnormalities that were consistent with pneumoconiosis; but, in fact, those people had no history of exposure to asbestos.

So it just points up the problems with relying on case series for which there is no control group to evaluate or assess the findings in that case series.

I think he misinterprets the Nicholson study. He says it's positive for automotive brake repair; and, in fact, it's not. It's negative, and it says so explicitly within the study.

And, finally, I had the impression that he cherry picks his data because I could not find where he referred to the other epidemiological literature which is available at this point in time: The Elliehausen study, the Marcus study and the Plato study.

Q. Dr. Franzblau, in conclusion, in your opinion as an occupational medicine specialist, when a doctor sees fibrosis and has a history of asbestos exposure and nothing else, is it appropriate to opine the diagnosis of asbestosis?

A. Well, it depends on the whole patient. You have to look at everything. And I've certainly had patients where I have come to that conclusion, and I feel very strongly about it. And I've actually had patients where I have come to the opposite conclusion where they've had a history of exposure, they have lung fibrosis; but I felt that their lung fibrosis was not due to asbestos.

Q. And are you aware of any appropriate

> methodology that would allow attribution as of --of brake work as a cause of Mr. Caswell's fibrosis given the facts presented to you?
> A. Well, if you are asking about clinical tests--is that what you are asking?
> Q. I'm asking about another--is there another methodology than the one you walked through that's generally accepted?
> A. Well, I suspect there are others that essentially have the same steps as mine. But if you don't consider dose, if you don't consider alternative causation, if you don't consider mitigating factors, then I don't see how you can come to the right conclusion.

N.T. 2/15/08, pp. 81-86, P.M. Session.[9]

Defendants offered their last expert witness, Dr. Patrick Hessel, who has a Ph.D. in epidemiology and taught epidemiology to medical students at the University of Alberta.

Dr. Hessel is also a researcher and has studied occupational lung disorders. He was accepted as an expert in epidemiology with a specialty in occupational lung disorders. (N.T. 2/15/08, pp. 136-139, P.M. Session).

The focus of the Dr. Hessel's testimony was the opinions of Dr. Gelfand and Dr. Frank, Plaintiffs' experts in the *Fisher* case, and the methodology for arriving at their conclusion. He reviewed Dr. Frank's affidavit and deposition testimony and Dr. Gelfand's expert report on causation, opining that Mr. Fisher's small cell lung cancer was caused by his exposure to Defendants' friction products. (N.T. 2/15/08, p. 140, P.M. Session).

Plaintiff, Mr. Fisher, was 77 years old when he died in 2005 from small cell lung cancer.[10] He had a smoking history of 30-40 packs per year which ended when he stopped smoking at age 42. He had begun smoking at age 17-18. (N.T. 2/15/08, p. 162, P.M. Session). His primary employment was as a plumber from 1941-1995. His sister died from lung cancer (non-smoker). He was a "shade tree" mechanic who did his own

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9 In evaluating this testimony, the Court notes that at times the witness offered some testimony which was not about the Plaintiffs' Experts' methodology but was about the Experts' conclusions with which the witness disagreed. Where the testimony did not focus on methodology, the Court did not consider it.

10. Small cell lung cancer is not the form of cancer known as mesothelioma. (N.T. 2/15/08, p. 151, P.M. Session). Small cell lung cancer is the type most strongly associated with smoking. (N.T. 2/15/08, pp. 163-164, P.M. Session).

brake work. (N.T. 2/15/08, pp. 141-142, P.M. Session).

Dr. Hessel faulted the methodology of both of Plaintiff's experts in the categories of confounding and attributable risk. Pursuant to Plaintiff's expert theory of causation, exposure to Defendants' brakes caused his lung cancer under the theory that all exposures contributed to his disease.

What was missing from both of Plaintiff's experts' methodology is the consideration of the role that the deceased Plaintiff's smoking played in his cancer. As Dr. Hessel testified:

> Q. Did you offer some critiques of Dr. Frank's affidavit in that regard?
> A. Yes. There was no scientific support for the idea of equating all exposures with causation. He cited no epidemiologic evidence as it related to vehicle repair and lung cancer. There are a number of studies that we just saw, epidemiologic studies. Those were not addressed.
> There was not really a scientific methodology that I could discern in his affidavit, and no consideration--importantly now, no consideration of other exposures or exposures to other carcinogens or other factors that might have confounded.

N.T. 2/15/08, pp.160-161, P.M.Session.

According to Dr. Hessel, a person with Mr. Fisher's smoking history is 17 times more likely to get small cell lung cancer than a non-smoker. (N.T. 2/15/08, pp. 164-165, P.M. Session). Further, occupational plumbers are 15 times more likely to get cancer than non-plumbers. Family history of cancer doubles the likelihood. Brake repair does not show any increase in risk of lung cancer.[11]

The failure to identify these other potential causes of the deceased Plaintiff's lung cancer and present a scientific discussion of why other factors presenting significantly higher probabilities of causing the lung cancer could not have caused same was, according to this expert not a generally accepted methodology in the relevant scientific community. (N.T. 2/15/08, pp.161-169, P.M. Session).

ANALYSIS

11. The above statistics are used to illustrate the principles of confounding and attributable risk. This Court is not making a factual finding of their accuracy because that would be a ratification of Defendants' experts' conclusions regarding the challenged causation theories which would be inappropriate in this hearing on Plaintiff's Expert methodology.

Our analysis must begin by recognizing that Pa.R.E. 702, which governs the "Testimony by Experts," sets the initial standard for admission of such testimony:

> If scientific, technical or other specialized knowledge beyond that possessed by a layperson 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, experience, training or education may testify thereto in the form of an opinion or otherwise.

Pa.R.E. 702.

The specific procedures for challenging an expert's testimony under these rules are presented in Pa.R.C.P. 207.1. Without repeating the discussion in our preliminary analysis above, the moving party here satisfied all of the requirements of this rule and the matter was properly raised and is properly before the court in this pretrial proceeding generally referred to as a *Frye* hearing.

The *Frye* test was first announced in a short and citation free discussion by the Court of Appeals for the District of Columbia regarding the admissibility of evidence arising out of a "systolic blood pressure deception test" which was a "crude precursor to the polygraph machine." *Daubert v. Merrell Dow*, 509 U.S. 579, 112 S.Ct. 2786, 125 L.Ed. 2d 469 (1993). The core concept of the *Frye* test is laid out in a passage from the D.C. Court opinion which has been called "famous (perhaps infamous)" by the United States Supreme Court:

> Just when a scientific principle or discovery crosses the line between the experimental and demonstrable stages is difficult to define. Somewhere in this twilight zone the evidential force of the principle must be recognized, and while courts will go a long way in admitting expert testimony deduced from a well-recognized scientific principle or discovery, *the thing from which the deduction is made must be sufficiently established to have gained general acceptance in the particular field in which it belongs*.[12]

Daubert at 585-586 (emphasis in original) (internal citations omitted).

Any case involving a *Frye* analysis in Pennsylvania must begin with *Grady v.*

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12 The issue of deductive versus inductive logic is discussed below.

Frito Lay, Inc., 576 Pa. 546, 839 A.2d 1038 (2003). In *Grady*, our Supreme Court had before it a case involving a tortilla chip which was alleged to have caused an esophageal tear when eaten by Plaintiff. *Id.* The action was initiated in the Allegheny Court of Common Pleas, which pursuant to defendant's motion in limine conducted a *Frye* analysis and concluded:

> It was the finding of this member of the Court, after taking into account the claimed expertise of the Plaintiffs' experts, and the methodology of Beroes, that Beroes' methodology was not based upon scientific data, or utilizing a methodology that was generally accepted in the community of scientists who evaluate food safety. Indeed, it was the impression of this member of the Court that Beroes' methodology smacked of a high school science fair project and did not bear any relationship to the reality of the mastication and consumption of food-stuffs. Beroes approached the characteristics of the Dorito chips as if it were a static evaluation of a material, rather than a consumable. Accordingly, this member of the Court determined that Beroes' methodology was akin to "junk science," did not meet the test of *Frye v. US.*, 54 App.D.C. 46, 293 F.1013 (D.C.1923) and its progeny, and that Beroes' methodology and opinion would only mislead the jury.

2000 WL 33436367 (Pa.Com.Pl. Allegheny County) (Not reported in A.2d).

The Superior Court at 2001 Pa. Super. 382, 789 A.2d 737 (2001), reversed the trial court. In so doing, the Superior Court held in relevant part:

> Nor do we find that the trial court properly precluded that part of the expert testimony of Dr. Beroes relating to the results of tests he had conducted on the Doritos chips, specifically, three series of compressive strength tests, and four sets of saliva tests conducted on whole chips. Rather, we are of the mind that Dr. Beroes was competent to testify as to the physical characteristics of the chips as revealed by the standard tests he had conducted upon the products of appellee.
>
> The *Frye* test makes the admission of expert testimony dependent "upon the general acceptance of its validity by those scientists active in the field to which the evidence belongs." *Commonwealth v. Topa*, 471 Pa. 223, 231, 369 A.2d 1277, 1281 (1977). In short, the gatekeeping responsibility of the trial court is not to weigh the correctness of an expert's opinion, or to choose between conflicting opinions, or to analyze and study the science in question in order to reach its own conclusions

> from materials in the field. Ultimately, it is the role of the trial court as gatekeeper to ensure the reliability and relevancy of expert testimony. It is to make certain that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field. *Kumho Tire*, 526 U.S. at 152, 119 S.Ct. 1167. *Travelers Property & Casualty Co. v. General Electric Co.*, 150 F.Supp.2d 360, 364 (D.C.Conn.2001).

Id.

The Supreme Court reversed the Superior Court in part and remanded the matter to the Trial Court. There are two parts of the Supreme Court's holding that are relevant to the instant matter.

The first and foremost part of the holding clarified the law as to the central tenet of the *Frye* test and eliminated any confusion on the issue of whether *Frye* requires that the conclusion of the proffered expert must also be generally accepted:[13]

> [I]n applying the *Frye* rule, we have required and continue to require that the proponent of the evidence prove that the methodology an expert used is generally accepted by scientists in the relevant field as a method for arriving at the conclusion the expert will testify to at trial. See, e.g. Blasioli, 713 A.2d at 1119.
>
> This does not mean, however, that the proponent must prove that the scientific community has also generally accepted the expert's conclusion. We have never required and do not require such a showing. This, in our view, is the sensible approach, for it imposes appropriate restrictions on the admission of scientific evidence, without stifling creativity and innovative thought.

Grady, 839 A.2d at 1045.

The second element also made clear who had the burden of establishing that the *Frye* requirements were satisfied:

> [W]e emphasize that the proponent of expert scientific evidence bears the burden of establishing all of the elements for its admission under Pa. R.E. 702, which includes showing that the *Frye* rule is satisfied.

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13. To the extent that the Superior Court wrestled with this issue in *Blum* (see below), this Court has been relieved of this burden as a result of the *Grady* opinion.

Id. The *Frye* analysis conducted by the Court was accomplished with these principles in mind.

The analysis here continues by scrutinizing the underlying basis of Plaintiffs' causation theory which although sometimes expressed in slightly different variations, is that "each and every breath of asbestos fiber is a substantial contributing factor to Plaintiff's disease." The use of this phrase is not unknown in asbestos litigation and has found its way into numerous appellate opinions. *See*, e.g. *Lonasco v. A-Best Products Co*., 757 A.2d 367 (Pa.Super. 2000).

> When asked about the role played by asbestos fibers and the causation of asbestos-related ailments and conditions, Doctor Epstein testified that "it's my professional opinion that all of those [asbestos] fibers can cause all of these changes in the body." (N.T. 10/28/96, at 82). Doctor Epstein expounded upon this opinion to a reasonable degree of medical certainty in the following manner:
>
> In my professional opinion, each exposure to asbestos that has taken place before the latency period that we've talked about; in other words, anything before the recognized latency period, has, in my professional opinion, been a substantial, contributing cause of each of these conditions, whether they be diseases or conditions, as we've discussed them.

Id. at 375. *See also, Cauthorn v. Owens Corning Fiberglass Corp*., 840 A.2d 1028, 1038-39 (Pa.Super. 2004).

> **…**[T]he Plaintiff's medical expert testified as to the effect each fiber of asbestos causes in the human body. In the current case, Mr. Cauthorn's medical expert testified to a "reasonable degree of medical certainty" that "[e]ach breath of air that contained asbestos fibers substantially contributed to the development of 'Mr. Cauthorn's] diseases." Further, he testified: "[b]ecause any asbestos fiber will cause some degree of injury…each fiber will have some small effect and it's the cumulative effect of all the different fibers."

Id.

This phrase recently came under scrutiny by our Supreme Court in *Gregg v. V.J. Auto Parts Company*, 2007 Pa. LEXIS 2935 (Pa. 2007). In *Gregg*, the Court acknowledged Judge Klein's lead opinion in *Summers v. Certainteed Corp.*, 886 A.2d

240 (Pa.Super. 2005) (equally divided court), which discussed the validity of such phrases when used as a basis for causation by an expert:

> Just because a hired expert makes a legal conclusion does not mean that a trial judge has to adopt it if it is not supported by the record and is devoid of common sense. For example, [the Plaintiff's liability expert] used the phrase, "Each and every exposure to asbestos has been a substantial contributing factor to the abnormalities noted." However, suppose an expert said that if one took a bucket of water and dumped it into the ocean, that was a substantial contributing factor" to the size of the ocean. [The expert's] statement saying every breath is a "substantial contributing factor" is not accurate. If someone walks past a mechanic changing brakes, he or she is exposed to asbestos. If that person worked for a factory making lagging, it can hardly be said that one whiff of the asbestos from the brakes is a "substantial factor" in causing disease.

Id. at 244 (emphasis in original).

In *Gregg,* the Supreme Court assessed the value of the "any exposure" equals causation conclusion.

> We recognize that it is common for Plaintiffs to submit expert affidavits attesting that any exposure to asbestos, no matter how minimal, is a substantial contributing factor in asbestos disease. *Id* at 27-28.
>
> …[W]e do not believe that it is a viable solution to indulge in a fiction that each and every exposure to asbestos, no matter how minimal in relation to other exposures, implicates a fact issue concerning substantial factor causation . . .

Id. at 29-30.

The phrase, "each and every breath of asbestos is a substantial factor in Plaintiff's disease" appears to be a form of inductive logic. This form of logic is one where a specific observation is made and then such is used to form a generalized conclusion. A parallel way of stating this might be, "If one breath of asbestos can cause a disease then every breath causes it." This form of logic has been criticized as being an invalid method of concluding that an association exists between cause and effect.[14]

An example of such faulty logic is demonstrated by the following: Assume that an observation of a white swan occurs. From this observation a conclusion is made that

14 *See generally* Legal Theory: Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation. The Legacy of Agent Orange and Bendectin Litigation. 86 Nw. U.L.Rev. 643 (1992).

"all swans are white." The conclusion is valid until the observation of a black swan occurs. The conclusion that all swans are white then becomes invalid.

The opposite of inductive logic is deductive logic which holds that general observations are first made and from these observations a conclusion is then formed about a specific relationship. Using the above example, an observation of the general class of swans is made and it reveals that most are white but some are black. The specific conclusion is that some swans are white and some are not.

The parallel for our purposes is that the general population is exposed to asbestos in one form or another whether it is a background rate or a discrete exposure, some get an asbestos related disease, some do not. Therefore, not all asbestos exposures cause disease. *See* Legal Theory: Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation, *supra; see also,* http://en.wikipedia.org/wiki/inductive_reasoning, for a fully annotated article discussing the relative pros and cons of inductive versus deductive reasoning.

This is where the scientific epidemiology has a role. As a preface to the proceeding discussion, it bears repeating that there can be no direct evidence of causation. The best that can be accomplished is some predictive value as to which type of exposure is more likely to cause the disease. *See id.*

The concept of relative risk has been discussed. The essentials of this are an observation of an exposed group and a calculation of the rate of disease therein. This is compared with an observation of an unexposed group and a calculation of the rate of disease therein. The math is elementary. Divide the exposed by unexposed and if the result is one, no association (11/11=1)[15].

During the course of Plaintiffs' experts' testimony, it appeared that there was an attempt to minimize or limit the role of epidemiology in determining causation. However, after pressing the witnesses on cross-examination, the witnesses acknowledged that epidemiology was the primary scientific method of determining causation in these cases.

In *Blum v. Merril Dow,* 705 A.2d 1314 (Pa.Super. 1997), the role of epidemiology was discussed in Judge Beck's majority opinion. In *Blum*, the disease was birth defects which presented as malformed limbs. The suspected agent was the drug Bendectin,

15. This example is used for simplicity sake only and is not meant to represent an actual epidemiologic calculation since the many variables and formulae actually used in a well founded and properly adjusted epidemiologic calculation will affect the predictive outcome.

which was ingested by the birth mother during pregnancy. The plaintiffs presented Dr. Alan K. Done, M.D., Dr. Gross and Dr. Stewart Newman, Ph.D., as their experts on causation *Id.* at 1319-1320. Each of the experts opined on the issue of causation between Bendectin and the birth defects.

Dr. Done relied upon four different sources of information in reaching his conclusion:

> First he considered "chemical structure analysis," and stated that the molecular structure of doxylamine (an antihistamine which he described as the part of Bendectin that is "harmful and teratogenic") makes the drug "suspect" as a "possible" teratogen.[16] However, even if the science leading to these statements were valid, such statements would lack the certainty necessary to establish causation. Smail v. Flock, 407 Pa. 148, 180 A.2d 59 (1962) (it is not enough for an expert to say something could have happened or to guess; expert testimony must assert that the result came from the cause alleged).
>
> Dr. Done then analyzed the effects of Bendectin on animal cells in in vitro studies, and testified about live animal (in vivo) studies, while conceding that such studies do not prove that a drug will have the same effect on humans, or on any individual. Dr. Done was "unsure" whether one could extrapolate the results from animals to humans, and acknowledged that based on animal studies alone, he could not determine to a reasonable degree of scientific certainty that Bendectin was teratogenic in humans.
>
> Dr. Done finally testified about human, or epidemiological, [4] studies. Despite the fact that no published epidemiological studies demonstrated a statistically significant association between Bendectin and limb defects, Dr. Done found evidence that Bendectin causes clubfeet when he recalculated some data in one published study, the Heinonen study, even though the authors of the published study had reached the contrary conclusion. As will be explained later, Dr. Done's "recalculation" was based on a methodology that was not generally accepted. Other human data used by Dr. Done in reaching his conclusions were derived from unreported preliminary data generated by Dr. Jick. Dr. Jick himself criticized this data as "biased, outdated, or premature and preliminary." Dr. Done admitted that it would be inappropriate for a scientist to rely upon such data.

16 "A 'teratogen' is an agent that causes the production of physical defects in the developing embryo." *Blum, supra*, at 1317, footnote 1.

> R.5290a-91a. Finally, Dr. Done agreed that the FDA, after complete review in 1980, found that available evidence showed no basis for a conclusion that Bendectin causes or increases the risk of birth defects in humans. It is important to note that Dr. Done has never published his conclusions in any scientific journal so as to enable his peers to evaluate them scientifically.
>
> > [4] Epidemiology is the study of the distribution and determinants of disease in human populations. Blum I, 385 Pa. Super. at 155, 560 A.2d at 214-15. Epidemiologists consider whether causation may be inferred by comparing the incidence of a disease in a group of humans who have been exposed to the substance in question with the incidence in a group of humans who have not been exposed to the substance. This ratio is described as an "odds-ratio" or "relative risk." R. 2097a, 2098a, 2123a.

Id.

Dr. Gross based his conclusions upon animal studies which suffered from the same extrapolation flaws identified by Dr. Done above. *Id.* at 1320.

Dr. Newman based his conclusions upon his studies of "Bendectin through the literature" and in vitro animal studies "and his belief that Bendectin had a certain chemical solubility and structural similarities similar to antihistamines which he identified as teratogenic." *Id.* at 1320-21.

After reviewing such methodologies the court concluded that the experts' methodologies failed to meet the *Frye* test and in so doing established the need for well conducted epidemiological studies in determining cause and effect:

> The methodology used to assess the teratogenicity of drugs is more complex than simply collecting certain types of data, *i.e.,* from chemical structure analysis, *in vitro* and *in vivo* studies, and reanalysis of epidemiological studies. Replicated epidemiological studies consistently finding a strong association are necessary to establish causation. Chemical structure analysis and *in vitro* testing can confirm the biological plausibility of a causal relationship suggested by epidemiology, but without an epidemiologically demonstrated association, they contribute nothing to the demonstration of causation. Animal studies can also provide evidence suggestive of causation. However, animal studies without epidemiological studies cannot

> prove causation in humans because drugs to not have the same effect on humans as they do on animals; the doses given to animals in animal studies are very different from those given to humans. Even Dr. Done admitted that animal studies would not be sufficient to prove that Bendectin is teratogenic in human beings. N.T. 12/12/86 at 178. The fact that a few of the animal species tested in studies discussed in this case developed some kinds of birth defects after being given many times the human dose of Bendectin cannot substitute for the lack of epidemiological evidence that Bendectin causes clubfeet in humans. No epidemiological study of Bendectin concludes that there is a statistically significant relative risk high enough to support a claim of general causation of clubfeet.

Id. at 1323.

The Court then went on to reject the Plaintiff's Experts concluding that:

> Although the general types of studies relied on by the Blums' experts are universally accepted as good science, the way they have utilized them to draw conclusions is not. Results derived from chemical analysis, *in vitro* and *in vivo* studies do not yield sufficiently reliable conclusions as to causation unless supported by epidemiological evidence.

Id. at 1324.

Plaintiffs here presented a maze of evidence in an attempt to support their experts' opinions. Within this maze, no recognizable methodology was found. The written reports were bald conclusions which contained no process or procedure detailing how the conclusions were reached or what supporting material or analyses were employed in the process. The testimonial evidence, although more lengthy and complicated, failed to establish that there was any methodology employed and how such (if it existed) was used to arrive at the respective conclusions. The mere mention of methodologies, *i.e.* chemical structure analysis, animal studies without a detailed explanation of how such was used in arriving at certain conclusions, produces scientifically incoherent opinions based upon scientifically incoherent methodologies and such are not generally accepted in the relevant scientific community.

Plaintiffs' experts rely upon the conclusion that each and every exposure to asbestos is a substantial contributing factor in causing Plaintiffs' disease. They have not demonstrated any methodology for arriving at such conclusions. It must follow that this failure cannot meet the *Frye* requirements.[17] Therefore, considering the above and the

17 As the court concluded in Blum, so does this Court:

record as a whole, Plaintiffs' experts are precluded from offering their opinion in *Caswell v. A.W. Chesterton, Inc., et al., Duke v. Chrysler LLC, et al., and Fisher v. A.O. Smith Corp., et al.* No decision is made regarding *Young v. A.W. Chesterton, Inc., et al.*, as this

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It is true that effective cross-examination is a powerful tool, and suffices to reveal the weaknesses in a witness's testimony where the lay jury is faced with common-sense questions of credibility or abilities of observation. However, the complex, confusing and possibly misleading details of scientific testimony do not so readily lend themselves to accurate assessment by even the most discerning jury. Much of such testimony is sophisticated and difficult to comprehend, and an analysis of the scientific validity of the methodologies underlying the testimony is simply beyond the capabilities of most lay persons. Therefore, the gatekeeping role of the court, far from detracting from the jury's function, is in fact essential to it: Scientific methodology and conclusions must initially be scrutinized by the court to ensure that what might appear to the jury to be science is not in fact speculation in disguise. Properly supported scientific evidence, however complex, can then reach the jury for its consideration, while material whose complexity merely hides its unreliability is winnowed out. This is, in essence, the teaching of Frye, and that teaching remains valid. *Blum, supra*, at 1325.

matter was prematurely included in this motion as discovery was not completed and the deadline for expert reports had not arrived at the time of this *Frye* hearing.

BY THE COURT:

_____
ALLAN L. TERESHKO, J.
COORDINATING JUDGE
COMPLEX LITIGATION CENTER

9-24-2008
DATE