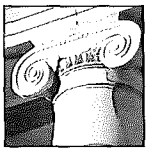

Any Exposure Above Background: Is It Really Causative?

A Commentary by Mark G. Zellmer, Partner, Husch Blackwell LLP
Author bio on page 8

The mantra becomes repetitious in many depositions of plaintiffs' medical experts: any exposure to asbestos in excess of background is enough to cause mesothelioma. This notion, to the extent that it has any basis at all, arose from early efforts to determine how many cases of cancer were caused in whole or in part by asbestos through extrapolation from studies with high exposures. However, without any quantification of exposure and without any expression of the increased level of risk, this statement of causation is more definition than science. The validity of such extrapolation is subject to serious doubt and criticism. A "low dose" exposure that is less than background exposure can hardly cause mesothelioma when background exposure does not cause mesothelioma. Case control studies attempting to prove that low dose exposures cause mesothelioma are subject to bias and reach highly inconsistent results. Finally, epidemiology of cohorts exposed to various types of asbestos gives a clear sense of what sort of exposures are necessary to cause mesothelioma.

Estimates of Asbestos-Associated Cancers

Some statements in the 1964 asbestos conference in New York suggested that the carcinogenicity of asbestos would be considered of "low order."[1] Today, plaintiffs assert that any exposure, no matter how small, will cause mesothelioma or, at least, materially increase the risk of mesothelioma. How did this notion originate? Although the question cannot be answered with certainty, pronouncements of the National Cancer Institute in 1978 provide some insight.[2] Noting that cancer may have more than one cause, the report accounted for latency, duration of exposures and changes in exposure patterns and then used certain epidemiological studies of "heavily exposed workers" to extrapolate to the number of cancers caused by asbestos.[3] The conclusion was that "13-18 percent of all cancer deaths" per year in the United States are associated with asbestos exposure.[4]

The report immediately received press coverage and had an impact on the global opinion regarding the dangers of asbestos exposure, including citation by the U.S. Congress as a significant accomplishment of HEW in 1978.[5] Underlying this estimate in the report, i.e. attributing a high percentage of cancers to asbestos exposure, is the sense that low doses, certainly lower doses than those in the epidemiological studies used to reach the conclusions of the report, cause asbestos-related diseases. In fact, some sources promptly criticized the report on such grounds. In one critique, Stallones and Downs criticized the report for its failure to account "for the fact that not all persons were so heavily exposed."[6]

The critique of Stallones and Downs was prescient. A number of researchers performed calculations of the proportion of cancers due to occupational exposures, particularly asbestos. The subsequent studies, Higginson in 1980,[7] Hogan and Hoel in 1981,[8] Enterline in 1981,[9] McDonald and McDonald in 1981,[10] and Nicholson et al in 1981,[11] found that 1-3 percent of all cancer may be related in some manner to asbestos exposure, a small fraction of the percentage found in the government's NCI report. The weight of the evidence — in fact, some would say a consensus — told the world that, overall, the effect of asbestos exposure was truly less than originally reported; however, the misconception of the carcinogenic potency of asbestos has lived on to this day.

Background Exposure

The thought that each and every exposure, no matter how small, causes mesothelioma can be evaluated in comparison to background exposures sustained in any urban area. Monitoring data has shown that background exposure, particularly in an urban area, approximates at least 0.0001 fibers/cc.[12] Such exposures "have no potential to cause disease."[13] Most experts testifying for plaintiffs have stated a similar position or have claimed that they do not know whether such exposures cause or contribute to cause mesothelioma.[14] Other plaintiff experts have testified that such exposure

might be causative without providing any opinion regarding how much background exposure contributes to risk.

Relatively, simple arithmetic allows the calculation of the approximate, cumulative, background exposure to asbestos that a person may have. For a 70-year-old, the cumulative background exposure would be 0.0252 f/cc-years. [15] The cumulative exposure from a specific, one-time task may also be calculated and then compared to the cumulative, background exposure. For example, the cumulative lifetime exposure from an hour-long task with an exposure level of 2 f/cc would be 0.0006893 f/cc-years.[16] Such amount is clearly less than the cumulative background exposure of 0.0252 f/cc-years. In fact, cumulative background exposure is almost 36 times greater than the exposure from this hypothetical, one-time task.

If background exposure cannot cause mesothelioma, a lesser exposure of 0.0006893 also should not cause mesothelioma. If it cannot be known whether a background exposure of 0.0252 f/cc-years causes mesothelioma, no one can know if a lesser exposure of 0.0006893 f/cc-years can cause mesothelioma. Even if background exposure may contribute to the risk of mesothelioma, certainly a lesser exposure contributes less to the risk of contracting the disease. As a result, it is more likely than not that background exposure caused the disease instead of that lesser exposure.

The logic that each and every exposure above background contributes to cause the disease is no logic at all. That each and every exposure contributes to causing mesothelioma cannot be proven. It is not science. It is merely an arbitrary definition of exposure to escape any need to justify an opinion scientifically that an exposure caused the disease. This can be particularly important when considering the defense of cases involving relatively short, low level or doubtful exposures. Some courts have held that plaintiff must prove that the exposure was sufficient to cause the disease.[17] Short, low level or doubtful exposures will not meet plaintiff's burden of proof.

The "Low Dose" Studies

Some plaintiff advocates will cite certain studies to demonstrate that low dose exposures will allegedly cause mesothelioma. [18] The following chart summarizes the findings of the principal studies:

Lead Author	Cumulative Exposure	Odds Ratio/Hazard Ratio
Iwatsubo	0.5-0.99 f/cc-years	4.2
Rodelsperger	>0-0.15 f/cc-years	7.9
Lacourt/Study B	>0-0.1 f/cc-years	1.9
Lacourt/Study A	>0-0.1 f/cc-years	Not statistically significant
Lacourt II	>0.-0.1 f/cc-years	4.0
Offermans/DOMJEM	4 years duration	Not statistically significant
Offermans/FINJEM	0.20=mean exposure	2.69

The reliability of these studies is subject to criticism.[19] However, all of those criticisms will not be repeated here.

The law that governs the admissibility of expert testimony requires either or both that (1) the methods and results should be generally accepted in the relevant scientific community[20] and (2) the methodology used as a basis for opinions should provide reliably consistent results.[21] Often the evidence that was thought to support a principle derived from earlier scientific studies will fade in later studies.[22] This is occurring with these "low dose studies." Under such circumstances these studies should not be considered as "generally accepted" or as providing reliably consistent results. For example, comparing the results in the Lacourt study with the earlier Rodelsperger study, for almost the same range of cumulative exposure, Lacourt found only an odds ratio of 1.9 while Rodelsperger had earlier found an odds ratio of 7.9. [23]

In addition, the Lacourt article is actually two epidemiological studies in the same article: study A and study B. While the results for study B show an odds ratio of 1.9, the results of study A for exposures of 0-0.1 f/cc-years were not statistically significant. In study A, the authors could not prove statistically that exposure up to 0.1 f/cc-years would increase the risk of mesothelioma. [24]

The 2014 Offermans studies undertook retrospective exposure assessments using two "job exposure matrices" (JEM). [25] DOMJEM assessed duration of exposure. FINJEM estimated cumulative exposure. Although a mean exposure of 0.2 f/cc-years under FINJEM led to a hazards ratio of 2.69, DOMJEM, despite the mean duration of 4 years of exposure, could not statistically prove an increased risk. [26]

Some of the same authors who undertook the efforts in the Lacourt studies

published further work on occupations and industries with a risk of mesothelioma.[27] *See the following Chart:*

Occupation	Odds ratio
Plumbers and pipefitters	5.57
Sheet-metal workers	5.00
Construction workers	3.46
Laborers	2.50
Carpenters, joiners, parquetry workers	1.94

The authors repeatedly refer to these and other occupations as "high risk" of mesothelioma.[28] The odds ratios for these "high risk" occupations, occupations at high risk of mesothelioma, are less than the odds ratio for "low dose" exposure in Rodelsperger and comparable to the odds ratios for "low dose" exposure in Iwatsubo and Lacourt. It is just not sensible that low dose exposures have a risk similar to, or even higher than, the risk of mesothelioma in "high risk" occupations.[29] This difference is substantial and raises questions about the reliability of the risk estimates in the low dose studies.

The most recent study by Lacourt et al in 2014 examined 988 cases of mesothelioma matched with 1125 controls and fails to provide any significant support for the concept that "low doses" materially increase the risk of mesothelioma.[30] *See Chart Below.*

The odds ratios for cumulative exposure of 0.1 and 1.0 f/cc-years are not statistically significant and thereby suggest no statistically proven increased risk. Notably, the odds ratios for cumulative exposure are not in excess of 2.0 until the cumulative exposure increases to 75 f/cc-years. All of these values belie the risk calculations in the earlier Rodelsperger, Iwatsubo and Lacourt studies.

No Observable Effect

Since these studies do not provide a basis to determine that low level exposures can materially increase the risk of mesothelioma, other studies may provide the data needed to determine what is the dose of asbestos necessary to increase the risk of mesothelioma materially. In fact, there are studies for each of the various commercially used asbestos fiber types.

Chrysotile

Research has offered proof for the proposition that chrysotile will not cause mesothelioma.[31] However, that point will not be subject to debate or analysis here. If chrysotile is causative, even weakly causative of mesothelioma, at what amount of exposure might be observed a material risk of mesothelioma? Pierce et al reviewed a number of epidemiological studies to discover the "no effect" level, meaning the "highest estimated cumulative exposure at which no increased risk was reported."[32] In each study chrysotile was almost the exclusive fiber type. The studies that they reviewed and relied upon to reach their conclusions were the following:

- Lacquet in 1980 of a Belgian cement factory;
- Albin in 1990 and 1996 of a Swedish cement factory;
- McDonald in 1984 of a friction products and packing manufacturing facility; and
- Piolatto in 1990 and Rubino in 1979 of the Balangero mine.[33]

Although the quality of the data in the studies varied somewhat, the authors were able to conclude that the "no observable effect level" from exposure to chrysotile asbestos for the causation of mesothelioma may be as high as 500 f/cc-years though maybe as low as 15 f/cc-years .[34] This method should be applicable to other types of asbestos mined, milled and used commercially.

Crocidolite

Generally regarded as the most potent carcinogen of the various commercial

Cumulative exposure f/cc-year	Odds ratio	95 percent confidence interval
0.1	1.0	1.0-1.0
1	1.1	1.0-1.1
10	1.9	1.4-2.5
25	1.9	1.5-2.4
50	1.9	1.4-2.5
75	2.6	1.9-3.4

forms of asbestos, miners and millers at Wittenoom Gorge in Western Australian mined and processed blue crocidolite asbestos for a number of years until 1966. Pictures of the Wittenoom mining and milling operation show the conditions: cramped underground mining; piles of crocidolite dust in the mill; a pervasive blue in the rocks and in the dust in the air; and all confronted without any respiratory protection. Fiber concentrations for the workers ranged from 100 f/cc in the bagging room of the mill to 20 f/cc in the mine.[35] Dust emanating from the mine and mill also exposed the workers as well as their families. The mine company piled three million tons of tailings, mine waste still containing asbestos, around the mine and used the tailings for roads and other construction around the town.[36] Exposure from residence alone at Wittenoom was on the average 5.5 f/cc/year.[37]

Study of the workers at Wittenoom found an excess incidence of mesothelioma per 10,000 man-years only among those who worked more than three months.[38] Those who worked up to three months would have cumulative exposure approximating 5/cc-years. In 2005, further follow-up of those in Wittenoom work force who died of mesothelioma between 1990 and 2002 revealed the occurrence or non-occurrence of mesothelioma at following asbestos exposure levels:[39]

Occurrence of mesothelioma by cumulative exposure	Pleural mesothelioma n=56	Peritoneal mesothelioma n=20	No mesothelioma n=1912
Mean exposure, f/cc-yrs	13	38	5
Cumulative exposure, f/cc-yrs	6-29	16-135	2-13

Mesothelioma was not occurring at cumulative exposures below 6 f/cc-years.

Amosite

In this study, the authors looked at a plant in Paterson, New Jersey, that manufactured insulation for the U.S. Navy and other customers. The fibers were largely, if not almost exclusively, amosite. The median level of exposure at the plant was 50 f/cc, ranging from a high of 120 f/cc for disintegrator operators to 15 f/cc for inspectors, maintenance, firemen, guards, receiving, foremen and supervisors. Even office workers had exposure of 5 f/cc.[40] The authors found that pleural, as well as peritoneal mesothelioma, appeared only among employees who worked six months or more at the Paterson plant.[41]

Fibers per Gram of Lung Tissue

Lung digestion and lung fiber counts are used to determine the number of asbestos fibers or asbestos bodies per gram of lung tissue. When the number of either fibers or bodies exceeds those expected from only background exposure, some experts suggest that occupational exposure has been proven and that the excess can be taken as proof that the exposure caused the mesothelioma. This requires an assumption that any exposure above background causes mesothelioma. The better course is not to assume anything and, rather, to determine what level of fibers in the lung are causative of mesothelioma. Christopher Wagner examined lung fiber counts in persons with and without mesothelioma. He found that lung fiber counts only demonstrate causation of mesothelioma by asbestos when the count exceeds one million amphibole fibers per gram of dry lung tissue.[42]

Case Reports of Mesothelioma Induced by Low Dose Exposure

The literature contains any number of case reports of asbestos exposure and disease. Some of these case reports even mention persons who have had mesothelioma and just minor exposures to asbestos. Case reports are merely anecdotal. They cannot prove causation.[43] If a person contracts cancer after an exposure to a carcinogen known to cause the cancer, the exposure still has not been proven to have caused the cancer. The exposure may be artifact. The cancer may have occurred without the exposure. Even assuming that the case report became part of an epidemiological study, there is no way to know if a study with the case report included would statistically prove a substantially increased risk.

Even the authors of the "low dose" studies have recognized that mesothelioma will occur without asbestos exposure. Lacourt et al found that 12.7 percent of male cases and 35.2 percent of female cases were not attributable to asbestos

exposure.[44] If mesothelioma occurs without exposure to asbestos, some cases of mesothelioma, particularly those with only small reported exposure, should certainly occur without that exposure causing the disease.

Conclusion

Determination of risk from the alleged exposure is the key element. If the exposure creates a great risk, that exposure is more likely a substantial factor in causation of the disease. The reverse is also true. If the risk from exposure is small, the exposure should not be considered causative.[45]

For defense counsel the lessons are rather clear:

- Any dose above background is not proof of causation, merely a definition of the exposure.
- Small or low dose exposures are not guarantees of liability.
- Plaintiff's experts should be cross-examined about the increased risk from exposure.
- Plaintiff's experts should be asked for their basis to testify that low doses cause mesothelioma.
- When cited, plaintiff's experts should be asked about the reliability of low dose studies.
- Case reports should be rejected as proof of causation.

Footnotes

[1] Schepers, G. Comments in "Biological Effects of Asbestos" *Annals of the New York Academy of Sciences* 132: 595 (New York Academy 1965).

[2] Bridford, K. et al. *Estimates of the Fraction of Cancer in the United States Related to Occupational Factors* (National Cancer Institute, September 15, 1978).

[3] Id. at 2-9.

[4] Id. at 10.

[5] Enterline, P.E. "Cancer Produced by Non-occupational Asbestos Exposure in the United States." Journal of the Air Pollution Control Association 33: 318-319 (April, 1989).

[6] Stallones, R. and Downs, T, A Critical Review of: Estimates of the Fraction of Cancer in the United States Related to Occupational Factors, Summary Statement (American Industrial Health Council, 1978).

[7] Higginson, J. "Proportion of Cancer Due to Occupation" *Preventive Medicine* 9: 180-188 (1980). Starting with the number of cancers by the anatomical site of origin of the cancer, Higginson determined the percent of cancers due to asbestos and other causes. He concluded that 1 percent of all cancers were due to asbestos.

[8] Hogan, M.D. and Hoel, D.G. "Estimated Cancer Risk Associated with Occupational Asbestos" *Risk Analysis: an Official Publication of the Society for Risk Analysis* 1: 67-76 (1981). Somewhat like the NCI report, by determining the number of exposed workers and the percentage of expected cancers among those workers, the authors concluded that 3 percent of all cancers may be asbestos related.

[9] Enterline, P.E. "Proportion of Cancer Due to Exposure to Asbestos" *Banbury Report 9 Quantification of Occupational Cancer* (Cold Spring Harbor Laboratory 1981) at 19-31. By analyzing the number of cancers to be expected in particular exposed occupations, Enterline suggested that 1 percent of all cancer was due to asbestos, but noted that elimination of asbestos would have an "imperceptible" effect on the reduction of all cancer.

[10] McDonald, J.C. and McDonald, A.D. Mesothelioma as an Index of Asbestos Impact" *Banbury Report 9 Quantification of Occupational Cancer* (Cold Spring Harbor Laboratory 1981) at 73-81. The authors took the number of expected mesothelial tumors (for which they thought there was good numeric information) and calculated the ratio of other

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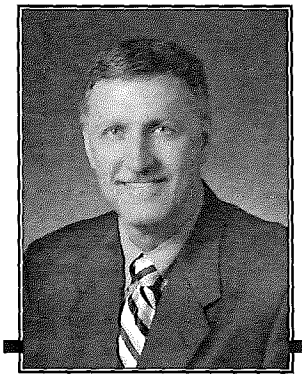
Zellmer speaks and writes extensively on products liability law and asbestos litigation. Such writing and presentation on asbestos litigation include subjects such as premises liability, application of statutes of limitations, medical causation, the litigation in historical context, and exclusivity of remedies under workers' compensation.



Zellmer defends toxic tort cases, particularly relating to asbestos, as well as other substances and chemicals. Zellmer also defends various defendants in product liability regarding vehicular and industrial equipment. Zellmer's trial practice is active in state and federal courts from coast to coast, and he has acted as national counsel in asbestos litigation.

malignant tumors to be expected relative to the number of mesothelial tumors. Their result was that 1.4 percent of all tumors were due to asbestos exposure.

[11] Nicholson W. et al "Cancer from Occupational Asbestos Exposure Projections 1980-2000" *Banbury Report 9 Quantification of Occupational Cancer* (Cold Spring Harbor Laboratory 1981) at 88-107. The authors determined the dose response relationship and the populations at risk as well as the relative risk by industry and then calculated the asbestos related mortality. Under their method they found that 2 percent of all cancer was asbestos related.

[12] Churg, A. and Green, F.H.Y. Pathology of Occupational Lung Disease 2d ed. (Williams & Wilkins: 1998) at 294.

[13] Id.

[14] Some such experts have even opined that, although they do not know (and really have no basis to say so), maybe background exposure is what accounts for the occurrence of spontaneous or idiopathic mesothelioma.

[15] (1) Multiply the background exposure level by a factor of 4.2 because occupational exposures are calculated over eight hours on a five day work week while background exposure occurs throughout the twenty four hours of each day of a seven day week, i.e. 0.00042 f/cc. (2) Determine the age of the exposed person minus a reasonable period after which the tumor has already occurred and, hence, further exposure may not contribute to causation of the tumor. Although this number is uncertain, it can rather arbitrarily be set at ten years. If plaintiff is age 70, 60 years is the length of time used for calculation of background exposure. (3) Multiply the years by the level of background exposure to determine the cumulative, background exposure, i.e. 0.0252 f/cc-years.

[16] (1) Determine the level of exposure from the task. In this instance we will assume that the task causes exposure of 2 f/cc. (2) Determine the length of time over which the task and, hence, the exposure occurs. In this instance we will assume one hour. (3) Determine the cumulative exposure per day, i.e. 2 f/cc/8=0.25 f/cc. (4) Determine the cumulative dose for the year, i.e. 0.25 f/cc/365=0.0006893 f/cc.

"Case control studies attempting to prove that low dose exposures cause mesothelioma are subject to bias and reach highly inconsistent results."

[17] *Mueller v. Garlock Sealing Technologies, LLC*, 660 F.3d 950, 952 (6th Cir. 2011).

[18] Iwatsubo, Y. et al, *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study,* 148 American Journal of Epidemiology 2: 133 (1998). Rodelsperger, K. et al, *Asbestos and Man Made Viteous Fibers as Risk Factors for Diffuse Malignant Mesothelioma: Results from a German Hospital-Based Case-Control Study,* 39 American Journal of Industrial Medicine 262 (2001). Lacourt, A. et al, *Attributable Risk in Men in Two French Case-Control Studies on Mesothelioma and Asbestos.* European Journal of Epidemiology. (September 7, 2010). Lacourt, A. et al "Occupational and Non-occupational Attributable Risk of Asbestos Exposure for Malignant Pleural Mesothelioma" doi:10.1136/*thoraxjnl*-2013-203744 (2/11/2014) at 1, 5. Offermans, N.S.M. et al "Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in Prospective Netherlands Cohort Study" *Journal of Occupational and Environmental Medicine* 56:6, 11 (2014). All studies except Lacourt II use a 95 percent confidence interval. Lacourt II uses a 99 percent confidence interval. The authors of Lacourt II fail to explain that with a similar size population in each study why, at the lowest exposure doses, the odds ratio more than doubles from 1.9 to 4.0 when the confidence interval is raised from 95 percent in Lacourt study B to 99 percent in Lacourt II. All studies except Offermans determine an odds ratio. Offermans cites a hazards ratio.

[19] Zellmer, M. "Are Low Dose Asbestos Exposure Studies Unreliable?" *Asbestos Columns* (March, 2011) at 4. For instance, these studies are subject to measurement bias. Flecther, R.H. et al *Clinical Epidemiology the Essentials* 5th ed. (Lippincott Williams & Wilkins: 2014) at 8. Without admitting measurement bias, some of the authors recognize the mathematical uncertainty of their exposure measurements. There is "no definition of low level exposure." JEM-based exposure assessment possibly entails "non-differential exposure misclassification" ("non-differential" meaning unrelated to the occurrence of the disease in question). As a result the data does not demonstrate the existence or non-existence of a threshold. Offermans, *supra.*, at 15.

[20] *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923).

[21] *Kumho Tire Company, Limited v. Carmichael*, 526 U.S. 137 (1999).

[22] Lehrer, J. *The Truth Wears Off,* The New Yorker Magazine 52, 54 (December 13, 2010).

Determination of risk from the alleged exposure is the key element. If the exposure creates a great risk, that exposure is more likely a substantial factor in causation of the disease. The reverse is also true."

[23] Rodelsperger, *supra.*;Lacourt, *supra.*

[24] Lacourt, *supra.*

[25] Offermans, *supra.*, at 6.

[26] Id. at 11.

[27] Rolland, P. et al "Occupations and Industries in France at High Risk for Pleural Mesothelioma: A Population-Based Case-Control Study (1998-2002)" *American Journal of Industrial Medicine* 53:1207, 1212 (2010).

[28] Id. at 1209, 1215, 1217.

[29] Although this article relates mainly to the effects of "low dose" exposures, it is instructive to compare the results of these studies at higher doses. For example, in Lacourt II, the authors find that a cumulative dose greater than 1 f/cc-years and up to 10 f/cc-years has an odds ratio of 22.5 while Offermans/FINJEM finds an odds ratio of 3.36 for a mean cumulative exposure of 6.57 f/cc-years. Lacourt (2014), *supra.*, at 5. Offermans, *supra.*, at 6.

[30] Lacourt, A. et al "Co-exposure to Refractory Ceramic Fibres and Asbestos and Risk of Pleural Mesothelioma" *European Respiratory Journal* 44: 725-733 (2014).

[31] Yarborough, C.M. "Chrysotile as a Cause of Mesothelioma: An Assessment Based on Epidemiology" *Critical Review of Toxicology* 36(2): 165-187 (2006) (his review of 71 cohort studies did not support any hypothesis that chrysotile without amphibole exposure will cause mesothelioma).

[32] Pierce, J.S. et al "An Evaluation of Reported No-Effect Chrysotile Asbestos Exposures for Lung Cancer and Mesothelioma" *Critical Reviews in Toxicology* 38:191, 193 (2008).

[33] Id. at 194-204.

[34] Id. at 207.

[35] Reid, A. et al "Cancer Incidence among Women and Girls Environmentally and Occupationally Exposed to Blue Asbestos at Wittenoom, Western Australia" *International Journal of Cancer* 122: 2337, 2339 (2007).

[36] Rogers, A. et al "Occupational and Environmental Mesothelioma due to Crocidolite Mining Activities in Wittenoom, Autralia" *Scandinavian Journal of Worker and Environmental Health* 21:259 (1995).

[37] Reid, A. et al "Age and Sex Difference in Malignant Mesothelioma After Residential Exposure to Blue Asbestos (Crocidolite)" *Chest* 131: 376-382 (2007).

[38] Hobbs, M.S.T. et al "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia" *Biological Effects of Mineral Fibers* 92: 615, 619 (1980).

[39] Reid, A. et al "The Additional Risk of Malignant Mesothelioma in Former Workers and Residents of Wittenoom with Benign Pleural Disease or Asbestosis" *Occupational and Environmental Medicine* 62:665, 666 (2005).

[40] Seidman, H. et al "Mortality Experience of Amosite Asbestos Factory Workers: Dose-Response Relationships 5 to 40 Years After Onset of Short-Term Work Exposure" *American journal of Industrial Medicine* 10: 479, 495-498 (1986).

[41] Seidman H. et al "Short-Term Asbestos Work Exposure and Long-Term Observation" Annals of the New York Academy of Sciences 61 (1979). These Seidman studies find an excess of lung cancer which, although not surprising, should be viewed cautiously. The authors used New Jersey males as the control group. If the Paterson work force smoked more and started smoking earlier than the control group, which is likely, the extent of an excess of lung cancer from exposure at the plant is overstated.

[42] Wagner, J.C. "Historical Background and Perspectives on Mesothelioma" in *The Mesothelial Cell and Mesothelioma* 78: 1, 10 (1994).

[43] Wong, O. "Malignant Mesothelioma and Asbestos Exposure among Auto Mechanics: An Appraisal of Scientific Evidence" *Regulatory Toxicology and Pharmacology* 34:170-177 (2001).

[44] Lacourt (2014), *supra.*, at 4.

[45] *Bostic v.Georgia-Pacific Corporation*, 439 S.W.3d 332 (Tex 2014); *Betz v. PneumoAbex LLC*, 44 A.3d 27 (Pa. 2012); *Dixon v. Ford Motor Company*, 47 A.3d 1038 (Md.App. 2012).