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A New Approach to an Old Defense:
Debunking State of the Art for Low Dose Cases

A Commentary by Mark G. Zellmer of Husch Blackwell LLP
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When plaintiffs pursue claims against defendants for low dose exposures, those defendants should consider the state-of-the-art defense specifically as it relates to the knowledge of the alleged hazards of low dose exposures. When it was known that asbestos was hazardous is not the issue in a "low dose" case; rather, the issue is when was there actual, scientific knowledge that low dose asbestos exposures allegedly cause mesothelioma?[1]

This is particularly important when plaintiff's counsel invokes a "bait and switch" argument. Plaintiffs initially offer evidence that the hazards of asbestos have been known since early in the 20th Century and then offer evidence that low doses of exposure cause mesothelioma. Plaintiffs' argument utterly ignores that the alleged "proof" of the effects of low dose exposure has come to light only recently and that the hazards of asbestos for most of the 20th Century were only associated with highly exposed workers.

Certainly the issue has been presented elsewhere that evidence of the effects of low dose exposure to asbestos is scientifically unreliable; however, this article presents a different issue relating to evidence of state of the art. Plaintiffs should not be allowed to claim that knowledge of hazards of high exposure puts a defendant on notice of hazards of low exposure. To that end, the article will discuss:

- The legal issues regarding evidence of state of the art;
- The definition of low dose — then and now;
- The early scientific and medical studies focusing on high exposures;
- The 1976 NIOSH report wrongly suggesting effects of low dose exposure;
- The 1978 NCI report wrongly suggesting extrapolation from high to low dose exposures; and
- The relevance of the studies actually claiming proof of the effects of low dose exposure.

Legal Issues Regarding Evidence of State of the Art

Although the issue of the admissibility of state-of-the-art evidence by a defendant varies from state to state and also varies according to the claims asserted by plaintiffs, such evidence may be admissible on the basis of a variety of arguments.

Plaintiff asserts a claim for failure to warn. Some states have enacted statutes that state of the art is a defense to failure to warn claims.[2] In other states, case law provides a basis for admission of such evidence.[3] Some states refuse to allow such evidence.[4]

Plaintiff asserts a claim for negligence. Although the admissibility of state-of-the-art evidence would seem to be in no doubt in defense of a negligence case, case law does not often address the issue. Likely the matter is simply just too clear cut to be an issue on appeal; however, some cases address the issue holding that the evidence is admissible.[5]

Plaintiff asserts a claim for strict product liability, either for manufacturing or design defect. Evidence of state of the art is often but not always inadmissible.[6] In Illinois, for example, evidence of state of the art is not admissible in strict liability except possibly for proof that a product complies with a safety standard or government regulation.[7]

Plaintiff asserts a claim for punitive damages. There would not seem to be doubt that, if plaintiff looks to prove willful or wanton or similar conduct, defendant should be allowed to negate such proof with evidence of what was or was not known at the time of the alleged tort.[8]

Plaintiff presents evidence on state of the art. This is the classic case in which plaintiff "opens the door" by putting on evidence of the state of the art which defendant is entitled to rebut.[9]

The Definition of Low Dose — Then and Now

What is a low dose of asbestos exposure is a function of time and knowledge. Writing in 1978 about exposures in the 1960s and earlier, Dr. Irving Selikoff referred to "low dosages" but admitted that knowledge that grave consequences could occur was still tenuous.[10] In fact, one year later in 1979, he specifically referred to asbestos exposures of insulators in the 1960s and before as "limited."[11]

Over the decades what has been regarded as a safe dose of asbestos exposure has changed and so have the standards intended to protect the workers possibly confronted with an exposure to asbestos. The following is a summary of how the threshold limit values, also labeled permissible exposure limits, have changed over the years:

- 1946: ACGIH, 5 million particles per cubic foot
- May 1971: OSHA, 12 fibers/cubic centimeter
- Later in 1971: OSHA, 5 fibers/cubic centimeter
- 1976: OSHA, 2 fibers/cubic centimeter
- 1986: OSHA, 0.2 fibers/cubic centimeter
- 1994: OSHA, 0.1 fibers/cubic centimeter

A low dose exposure should not be defined by the hindsight of today's alleged knowledge about the hazards of asbestos, but rather, is best defined by comparison to the standards in effect at the time.

The "Early" Studies of "High Dose" Exposures

The studies that plaintiffs offer as early evidence of the knowledge of risks associated with asbestos exposure almost universally involved high dose exposures; hence, these studies present no real evidence that a defendant should have been aware of any risk from low dose exposures.

In 1938, Waldemar Dreessen presented his seminal work for the U.S. Public Health Service recommending that exposures to asbestos should be kept below 5 million particles per cubic foot (mppcf) to prevent asbestosis because he and his co-investigators were not finding cases of asbestosis below that level after 5-10 years of employment. Although different concentrations of asbestos dust were found for different operations in the plant, maximum concentrations reached 74 mppcf.[12]

"Plaintiffs should not be allowed to claim that knowledge of hazards of high exposure puts a defendant on notice of hazards of low exposure."

In 1955, Richard Doll (later Sir Richard Doll, knighted for his work in epidemiology) undertook to investigate asbestos and lung cancer. He studied 105 deaths of men who had been employees of the asbestos works (later known to be Turner and Newell) for at least nine years before dust reducing regulations were enacted. Of these, 18 men had lung cancer, of which 15 had asbestosis. Both the occurrence of asbestosis and exposure before the "dust" regulations demonstrate the likely substantial amounts of exposure.[13]

In 1960, Christopher Wagner published his now famous study of diffuse malignant pleural mesothelioma from crocidolite mining work in the Cape asbestos field of South Africa. Of his 33 cases of mesothelioma, almost all were described with some sort of heavy exposure to asbestos at work, in the home or from the environment. Among co-workers without mesothelioma, Wagner found any number of cases of asbestosis reflecting the high level of exposure.[14] The children, with unfortunately fatal consequences, played in the tailing piles, debris left from the mining operation. Exposures were so high environmentally that the residents were described as living in a "blue haze."[15]

Shortly thereafter, in the mid-1960s, Irving Selikoff began to publish his findings on mesothelioma among the insulation trade.[16] Asbestosis was a common finding among the insulators of this era once again, demonstrating the level of exposure putting these men at risk.[17] These insulators were found to be the workers at highest risk of mesothelioma among users of end products containing asbestos.[18] However, as late as 1969, Dr. Selikoff could not say that the asbestos dust that caused disease among insulators was sufficient to cause health problems at lower levels of exposure.[19]

In the early 1970s, OSHA summarized the state of knowledge about relatively low dose exposures. The agency noted that "[n]o one has disputed that exposure to asbestos of high enough intensity and long enough duration is causally related to asbestosis and cancer. The dispute is as to the determination of a specific level below which exposure is safe."[20] However, OSHA was comfortable that the present standard of 5 f/cc would protect workers stating that "so long as the ceiling limit is complied with, no harm is reasonably expected to result from exposures during the transitional period."[21]

The 1976 NIOSH Report

The first ever comment that there was no safe dose of asbestos exposure almost certainly came from NIOSH in 1976. At that time NIOSH pronounced that "[e]xcessive cancer risk have been demonstrated at all fiber concentrations studied

to date. Evaluation of all available human data provides no evidence for a threshold or a 'safe' level of asbestos exposure."[22] So, there it is; NIOSH said it, but was it entitled to belief by those in industry and science who chose to look at the basis for the statement? Initially and most importantly, NIOSH did not pronounce that there was no safe dose; rather, the wording was most careful -- only that NIOSH could not determine a safe dose based upon the data. What was the data that truly low doses of asbestos exposure would cause mesothelioma?

The data actually proved nothing regarding the effect of low dose asbestos exposure. NIOSH cited certain studies on the association between asbestos and mesothelioma. For example, NIOSH cited Gobbato and Ferri from 1973; however, those authors studied insulation workers in the Italian shipyards, not typically identified as a low exposure occupation.[23]

Hain et al from 1974, also a NIOSH reference, studied workers in occupations such as asbestos textile, insulating industries, navy and shipbuilding, again, not typically identified as a low exposure occupation.[24] They concluded that exposure to asbestos dust of at least two years duration was required to precede the occurrence of mesothelioma.[25] This conclusion flatly contradicts the finding suggested by NIOSH that no safe threshold is known and that low dose exposures are a concern as a cause of mesothelioma.

Another reference from the 1976 NIOSH report was Zielhuis et al in 1975.[26] The authors noted a single case of short duration exposure; however, short duration exposure was defined as less than a year, which could be a substantial exposure depending on the nature of the job. The authors found that their cohort on the whole experienced intensive exposures that were often intermittent and often over a duration of 10 years.

The NIOSH report also cites Greenberg et al published 1975. The authors did not presume to quantify exposures, but found the following for those definitely diagnosed with mesothelioma:

Exposure Classification	# of Cases
Definite occupational exposure	167
Possible occupational exposure	16
Neighborhood, domestic or hobby	13
None	38
Not obtained	12

The authors found an instance of an individual with mesothelioma who had one day of asbestos exposure from sawing asbestos cement sheets.[27] It is of course known that even if an exposure is "possible" or "neighborhood, domestic or hobby" the exposure on a cumulative basis may still be substantial.[28]

Zielhuis et al mentioned an individual case of exposure less than one year in duration. Greenberg et al mentioned an individual case of one day of exposure. Relevant to such exposures, the NIOSH report stated that there was "an association between asbestos and mesothelioma from exposures of as brief as 1 day." Notably, NIOSH used the term "association." Rather than regard such instances as proof of causation, NIOSH appropriately cautioned that approximately 15 percent of the mesotheliomas occurred without any known exposure to asbestos.[29] In simple terms, the short or low dose asbestos exposure associated with the occurrence of mesothelioma may have been an artifact. The mesothelioma could have occurred without asbestos exposure. This is notable. Case reports or individual cases cannot prove causation. They have no control group for comparison. They have no manner or method to calculate whether the occurrence of the disease reflects an increased risk of disease from the exposure. The number of subjects, often as few as one, leave no possibility that any statistically significant risk can be found.

The entire 1976 NIOSH report cited only one study that compared mortality from malignancy with airborne concentrations of asbestos exposure.[30] Despite exposures averaging 0.36 f/cc >5 microns, the authors reported no occurrence of mesothelioma.[31]

In sum, the 1976 NIOSH report provided no scientific support for the proposition that "low dose" exposures would cause mesothelioma; rather, NIOSH engaged in fear mongering that there could be risk from "low dose" exposures. NIOSH used careful but misleading language about "all fiber concentrations studied to date."[32] In fact NIOSH had no scientific study of low concentration exposures from which to draw any supportable conclusions. Now, if our government wished to provide protection against exceedingly low risks, risks that may not and likely did not even exist, then that is a policy decision. It is not a basis to put any company on notice that low dose exposures in fact cause mesothelioma or materially increase the risk of mesothelioma.

The 1978 Report of the National Cancer Institute

In 1978 the National Cancer Institute provided a report on the number of cancers attributable to asbestos exposure.[33] 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.[34] The conclusion was that "13-18 percent of all cancer deaths" per year in the United States are associated with asbestos exposure.[35] Through this extrapolation, the National Cancer Institute assumed that lower doses than those in the epi-

demiological studies caused asbestos related diseases and then attempted to calculate cancers occurring from such low doses of exposure.

Some sources promptly criticized the report for exactly that reason. In fact, Stallones and Downs stated in their critique that, among other criticisms, the report fails to account "for the fact that not all persons were so heavily exposed."[36] Other criticisms immediately followed. A number of researchers performed calculations of the proportion of cancers due to occupational exposures, particularly asbestos.

Higginson in 1980 started with the number of cancers occurring at each anatomical site of origin and then determined the percent of cancers due to asbestos versus other causes for each site of origin. After totaling his numbers, he concluded that 1 percent of all cancers were due to asbestos exposure.[37] Several other researchers followed in 1981, calculating much lower numbers than the National Cancer Institute.

Hogan and Hoel[38] used a methodology similar to the NCI report. They determined 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. Enterline[39] suggested that likely only 1 percent of all cancer was due to asbestos exposure. He analyzed the number of cancers to be expected in particular exposed occupations and then noted that elimination of asbestos would have an "imperceptible" effect on the reduction of all cancer. McDonald and McDonald[40] took the number of expected mesothelial tumors (for which they thought there was good numeric information) and calculated the ratio of other 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. Finally Nicholson et al[41] 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.

"Defense experts should be prepared to differentiate between the scientific knowledge of the hazards of asbestos exposure generally and the lack of knowledge that there was a risk from low dose exposures."

All of these later reports found that only 1-3 percent of all cancer may be related in some manner to asbestos exposure. This suggested that criticisms of the government's NCI report were well founded, including criticism of the implicit effort to claim that extrapolation of the effects of high dose exposures to low dose exposures proved causation of mesothelioma and other asbestos related diseases. The weight of the evidence told the world that overall the effect of asbestos exposure was truly less than originally reported by the National Cancer Institute; however, apparently ignoring the justified criticism, the U.S. Congress cited the report as a significant accomplishment of HEW in 1978.[42] Unfortunately, the misconception of the carcinogenic potency of asbestos has lived on through this day.

The "Low Dose" Epidemiology

The articles that provide plaintiffs with their claims that there is epidemiological evidence that low doses of exposure cause mesothelioma were not published in the time frame when asbestos exposures were occurring.[43] The earliest of these epidemiological studies was published in 1998. No one except plaintiffs and their experts would blame a defendant for wondering why it should be liable when the exposure was so low that there was no alleged "science" proving any risk until the late 1990s.

Furthermore, these "low dose" studies are not uniformly accepted either for their findings or methodology.[44] Commentaries on plaintiffs' "low dose" studies level any number of criticisms. The studies use the term "semi-quantitative" which really means subjective. The studies reach inconsistent results belying any reliability of the methods. The studies are affected by all sorts of bias which serves to inflate the risk estimates. If the methods and results of these studies are a matter of dispute, how can the studies, even now, be considered appropriate notice of a risk from exposure?

Conclusion

These thoughts suggest a clear and simple path for "low dose" cases.

- Plaintiffs' experts who address state of the art should be questioned, not about the risks of asbestos exposure, but when it was known that low doses, e.g. below 1 f/cc-yrs, could in fact cause mesothelioma.

• Defense experts should be prepared to differentiate between the scientific knowledge of the hazards of asbestos exposure generally and the lack of knowledge that there was a risk from low dose exposures.

• Plaintiff experts who testify about state of the art in low dose cases should be the subject of motions in limine and objections when they conflate testimony about hazards of asbestos generally and hazards of low dose exposures.

Endnotes

[1] I am obliged to thank my partner, Randall K. Mullendore, for his assistance in preparation of parts of this article. His help was most appreciated.

[2] Mo. Rev. Stat. 537.764; *see also Tretter v. Johns-Manville Corp.*, 88 F.R.D. 329, 333 (E.D. Mo. 1980)(asbestosis case, applying Missouri law, holding that state of the art evidence is relevant to show the extent of the defendant's knowledge).

[3] *Woodill v. Parke Davis & Co.*, 79 Ill. 2d 26, 29, 402 N.E.2d 194, 195 (1980)(plaintiff asserting a failure to warn claim must plead that the defendant knew or should have known of the dangerous propensity of the product); *Anderson v. Owens-Corning Fiberglas Corp.*, 810 P.2d 549, 550 (Cal. 1991)(defendant may present evidence that the particular risk was neither known nor knowable by the application of the scientific knowledge available at the time of manufacture and/or distribution; otherwise, defendant would be a "virtual insurer" of the product's safe use).

[4] *Hawaii Fed. Asbestos Cases*, 665 F. Supp. 1454, 1458 (D. Haw. 1986); *Johnson v. Raybestos-Manhattan, Inc.*, 740 P.2d 548, 549 (Haw. 1987).

[5] *Murphy v. Chestnut Mountain Lodge, Inc.*, 124 Ill. App. 3d 508, 515, 464 N.E.2d 818 (1984)(admissible in a negligence claim but ruling is dicta as only a strict liability claim was presented); *Sappington v. Skyjack, Inc.*, 512 F.3d 440, 445, n. 3 (8th Cir. 2008)(under Missouri law, state of the art is defense by a manufacturer in a negligence case based on products liability).

[6] Compare *Normann v. Johns-Manville Corp.*, 593 A.2d 890, 894-895 (Pa. Super. Ct. 1991)(admissible applying New York law) with *Elmore v. Owens-Illinois, Inc.*, 673 S.W.2d 434, 438 (Mo. 1984)(applying Missouri law, "state of the art evidence has no bearing on the outcome of a [design defect] strict liability claim"); and, similarly, *Hawaii Fed. Asbestos Cases*, 665 F. Supp. 1454, 1458 (D. Haw. 1986); *Johnson v. Raybestos-Manhattan, Inc.*, 740 P.2d 548, 549 (Haw. 1987)(not admissible).

[7] *Gelsumino v. E. W. Bliss Co.,* 10 Ill. App. 3d 604, 608, 10 N.E.2d 110, 113 (1973)(not admissible); *Cunningham v. MacNeal Memorial Hospital*, 47 Ill. 2d 443, 453, 266 N.E.2d 897, 902 (1970)(not admissible); *Moehle v. Chrysler Motors Corp.*, 93 Ill. 2d 299, 305, 443 N.E.2d 575, 578 (1982)(safety standard); *Rucker v. Norfolk & W. Ry. Co.,* 77 Ill. 2d 434, 439396 N.E.2d 534, 537 (1979) (federal regulations). Although instructive, due to the statute of repose, claims for asbestos exposure are not brought in strict product liability under Illinois law.

[8] *Lane v. Amsted Indus., Inc.*, 779 S.W.2d 754, 759 (Mo. App. W.D. 1989)(admissible under Missouri law on the issue of punitive damages).

[9] *Murphy v. Chestnut Mountain Lodge, Inc.*, 124 Ill. App. 3d 508, 515, 464 N.E.2d 818 (1984); *Walker v. Trico Manufacturing Co., Inc.*, 487 F.2d 595, 600 (7th Cir. 1973).

[10] Selikoff, I. et al. *Mortality Experience of Insulation Workers in the United States and Canada*. Annals of the New York Academy of Sciences: Health Hazards of Asbestos Exposure. Vol. 330 (1979) at 92.

[11] Selikoff, I. et al. Asbestos and Disease. (Academic Press, New York: 1978) at 31.

[12] Dreessen, W. et al. A Study of Asbestos in the Asbestos Textile Industry. (U.S. Public Health Service, U.S. Government Printing Office: 1938) at 35, 117.

[13] Doll, R. *Mortality from Lung Cancer in Asbestos Workers*. British Journal of Industrial Medicine. Vol. 12 (1955) at 81, 86.

[14] Wagner, C. et al. *Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province*. British Journal of Industrial Medicine. Vol. 17 (1960) at 260.

[15] Wagner, C. *The Discovery of the Association between Blue Asbestos and Mesothelioma and the*

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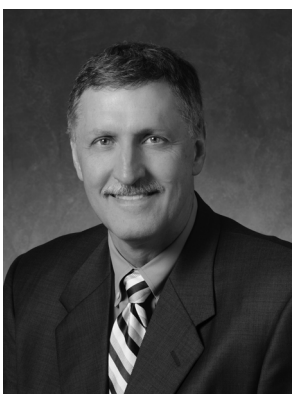

Zellmer speaks and writes extensively on products liability law and asbestos litigation. Such writing and presentation on asbestos litigation include subjects such as premises liability, application of statutes of limitations, medical causation, the litigation in historical context, and exclusivity of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

Aftermath. British Journal of Industrial Medicine. Vol. 48 (1960) at 399, 401.

[16] Selikoff, I. et al. *Asbestos Exposure and Neoplasia*. Journal of the American Medical Association. Vol. 88, No. 1. (1963) at 22.

[17] Selikoff, I. et al. *The Occurrence of Asbestosis Among Insulation Workers in the United States*. Annals of New York Academy of Sciences. Vol. 132 (1966) at 139.

[18] McDonald, J.C. et al. *The Epidemiology of Mesothelioma in Historical Context*. European Respiratory Journal. Vol. 9 (1996) at 1932.

[19] Selikoff, I. "Joint Health Research Effort Starts for Insulation Men." Insulation Hygiene Progress Reports. Vol. 1, No. 1 (Spring 1969) at 6.

[20] Federal Register, vol. 37, no. 110: 11318 (June 7, 1972).

[21] Id. The finding refers to "no harm," not just the prevention of harm from asbestosis.

[22] NIOSH, Public Health Service, Center for Disease Control, U.S. Department of Health, Education and Welfare. Revised Recommended Asbestos Standard (December, 1976) at 92.

[23] Section of Occupational Medicine. Attitudes to Occupational Health Hazards within the EEC. Journal of the Royal Society of Medicine. Vol 72 (April, 1979) at 287, 290.

[24] Hain, E. et al. Retrospective Study of 150 Cases of Mesothelioma in the Hamburg Area. International Archives Arbeitsmed. Vol. 33, no. 1 (1974) at 15.

[25] Id.

[26] Zielhuis, R.L. et al. *Pleural Mesothelioma and Exposure to Asbestos: A Retrospective Case-Control Study in the Netherlands.* International Archives of Occupational and Environmental Health. Vol. 36, Issue 1 (March, 1975) at 1.

[27] Greenberg, M. et al. *Mesothelioma Registry: 1967-1968*. British Journal of Industrial Medicine. Vol. 31 (1974) at 91, 95, 96. The authors stressed their pains-taking efforts to determine any asbestos exposure whatsoever of any subject in the study. Occurrence of mesothelioma without asbestos exposure was approximately 16 percent of all cases.

[28] Huncharek, M. et al. *Domestic Asbestos Exposure, Lung Fibre Burden and Pleural Mesothelioma in a Housewife*. British Journal of Industrial Medicine. Vol. 46 (1989) at 354, 355. Environmental exposure is not always low level exposure. Case, B. et al. Applying Definitions of "Asbestos" to Environmental and "Low-Dose" Exposure Levels and Health Effects, Particularly Pleural Mesothelioma. Journal of Toxicology and Environmental Health, Part B, Critical Reviews. Vol. 14 (1-4) (2011) at 3. For example, Jefferson Parish, Louisiana is a geographic area with a high incidence of malignant mesothelioma due to the concentration of asbestos product manufacturing in the area and the spread of asbestos by-products on the roads, driveways and elsewhere.

[29] NIOSH at 32. Clearly NIOSH was careful to use the terminology of "association" rather than advocating this data as proof of "causation."

[30] Gillam, J.D. et al. *Mortality Patterns among Hard Rock Gold Miners Exposed to an Asbestiform Mineral.* Annals of New York Academy of Sciences. (1976) at 336.

[31] Id. at 341. The authors reported 3 lung cancers with less than 20 years employment and 7 lung cancers with more than 20 years employment. There was no report whether any of these men had concurrent asbestosis. However, exposures for fibers longer than 5 microns ranged as high as 1.29 f/cc and total fibers ranged as high as 11.79 f/cc. At these exposure levels with this length of employment, these subjects with lung cancer had exposures in a range that could and likely would cause asbestosis. Id. 339, 341.

[32] Id. 92.

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

[34] Id. at 2-9.

[35] Id. at 10.

[36] 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).

[37] Higginson, J. *Proportion of Cancer Due to Occupation*. *Preventive Medicine* 9: 180-188 (1980).

[38] Hogan, M.D. and Hoel, D.G. Estimated Cancer Risk Associated with Occupational Asbestos. Risk Analysis. Official Publication of the Society for Risk Analysis 1: 67-76 (1981).

[39] 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.

[40] 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.

[41] 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.

[42] 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).

[43] Iwatsubo, Y. et al, *Pleural Mesothelioma: Dose-Response Relation at Low Levels of Asbestos Exposure in a French Population-based Case-Control Study*, American Journal of Epidemiology Vol. 148:2 (1998) at 133. 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*, American Journal of Industrial Medicine. Vol. 39 (2001) at 262. 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 Vol. 56 (2014) at 6, 11.

[44] Boffetta, P. et al. *A Systematic Review of Occupational Exposure to Synthetic Vitreous Fibers and Mesothelioma*. Critical Reviews in Toxicology. Vol. 44(5) (2014) at 436, 440; Siemiatycki, J. et al. *Invited Commentary: Is It Possible to Investigate the Quantitative Relation between Asbestos and Mesothelioma in a Community-based Study?* American Journal of Epidemiology. Vol. 148, No. 2 (1998) at 143; Case, B.W. et al. *Applying Definitions of 'Asbestos' to Environmental and 'Low-Dose' Exposure Levels and Health Effects, Particularly Malignant Mesothelioma*. Journal of Toxicology and Environmental Health, Part B. Vol. 14:3 (2011) at 3.