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# Asbestos

## Controversies Regarding The Role Of Asbestos Exposure In The Causation Of Lung Cancer: The Need For An Evidence Based Approach

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# Commentary

## Controversies Regarding The Role Of Asbestos Exposure In The Causation Of Lung Cancer: The Need For An Evidence Based Approach

**By**
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The incidence of asbestos-related diseases has steadily declined since the early 1990s.[1] This trend is not surprising. In light of the federal Occupational Safety and Health Administration's ("OSHA") dictates and workplace controls which took hold in the 1970's, asbestos exposure does not in any way resemble the levels of exposure witnessed in the 1940's, 1950's and 1960's.[2] And in recent years the aggregate volume[3] of asbestos cases has significantly decreased due to steps taken to address non-malignancy cases through tort reform laws and judicial rulings.[4]

As a result, in the past decade the primary focus of the asbestos plaintiffs' bar has been on high-value cases involving malignant pleural mesothelioma.[5] But there is a finite number of these high value cases. According to the American Cancer Society, as well as the recent National Cancer Institute data, the number of malignant pleural mesothelioma cases diagnosed annually in the United States has steadied — in the range of 3,000 cases per year.[6] Because of the lack of available mesothelioma cases, there has been a recent increase in the number of lung cancer lawsuits filed as asbestos cases.

Unlike an asbestos-related disease such as mesothelioma, which has a historically strong association with certain asbestos exposures, lung cancer can be, and is most often, caused by tobacco abuse without any history of asbestos exposure. In fact, at least eighty percent of all lung cancers are caused by cigarette smoking. And most workers who develop lung cancer are either current smokers or have a significant smoking history that undeniably increased their risk for developing lung cancer. A 20-pack year habit increases a smoker's risk of lung cancer ten-fold or more.[7] In contrast, in order to demonstrate a doubling of the risk of lung cancer in non-smokers with asbestos exposure, a very significant exposure to asbestos, i.e., at the very least a total asbestos exposure dose of 25 f/cc-years is required.[8]

To overcome this challenge, a growing number of the asbestos plaintiffs' bar seek to disregard science-based attribution criteria and advocate that asbestos is the cause of many lung cancers. This disregard for causality based on science takes two approaches: one which finds any level of asbestos exposure causative of lung cancer (while discounting or ignoring substantial smoking histories that are legally and medically sufficient explanations as the cause for most lung cancers), and the other, which finds the presence of asbestosis (while discounting or ignoring accepted diagnostic criteria). Both approaches disregard years of well-established causation criteria that distinguish smoking-induced lung cancers from asbestos-related lung cancers.

This paper discusses the asbestos plaintiffs' bar's efforts to redefine tobacco-induced lung cancers and turn them into claims for asbestos-related cancers. Because of the recent surge in lung cancer case filings, and given that the American Cancer Society estimates approximately 225,000 lung cancers are diagnosed annually in the United States,[9] there is a need to return to an evidence-based approach founded upon epidemiology, and to develop medicolegal guidelines for asbestos-related lung cancer lawsuits.

## Rarity Of Exposure Sufficient To Cause Asbestosis In Current Lung Cancer Claims

Exposure to asbestos or the mere presence of asbestos in lung tissue is not sufficient to cause lung cancer. It is well known that all persons are exposed to background levels of asbestos in the ambient air. Therefore, every person's lungs contain some level of asbestos fiber. And yet, the consensus of published scientific studies is that the population at large is not at an increased risk to develop lung cancer — or any other cancer, as a result of exposures to asbestos at background levels.[10]

Even individuals with exposures above background levels are not at an increased risk to develop lung cancer until they encounter a substantial (and quantifiable) dose of exposure — a dose that requires many years of direct work with asbestos in a trade such as a career insulator with a work history pre-dating OSHA and concomitant safety and health regulations.[11] Studies demonstrate that asbestos workers who smoke but do not have asbestosis have the same lung cancer risk as non-asbestos workers who smoke at a similar level.[12] Thus, it is generally accepted that the diagnosis of asbestosis or the equivalent dose of exposure is the requisite foundation to any evidence based approach for the attribution of asbestos exposure to lung cancer.

The idea of using asbestosis as a marker to demonstrate a causal link between asbestos exposure and lung cancer is not new. The use of asbestosis as a marker of exposure dates back to studies in the 1950s including the work of Doll and through the 1970s with the work of Selikoff.[13] Exposure dose for attribution has also been examined and quantified in fiber per cc years.[14] And though some scientific studies place the threshold dose for attribution in the range of 100 f/cc years of total asbestos exposure, it has also been accepted that in a non-asbestosis case, an equivalent fiber burden of at least 25 f/cc years is necessary to attribute lung cancer to asbestos exposure.[15] Either way, asbestos exposures in these concentrations have been and continue to be the only reliable marker for the potential attribution of a lung cancer to asbestos. And even so, it must be remembered that even at these concentrations, the issue is an increased risk of developing lung cancer, not necessarily proof of attribution.

Similarly, for many years, it has been accepted that asbestosis does not — and will not, occur absent a substantial and sustained history of exposure.[16] The prevailing opinion has been — and is — that a minimum of 25 f/cc years exposure history is required to cause radiographic findings consistent with the diagnosis of asbestosis under the ILO guidelines (a universally accepted guideline in the diagnosis of asbestosis).[17] And the incidence of asbestosis in exposure above the low threshold of 25 f/cc is varied in the published studies — suggesting that a dose far greater than 25 f/cc may be required.[18]

In 1976, the federal Occupational Safety and Health Administration ("OSHA") lowered the permissible exposure limit for asbestos in the workplace to 2 f/cc at a time-weighted exposure average.[19] Under this standard, a worker must have fifty years of sustained asbestos exposure to reach the lifetime dose of 100 f/cc years — the dose that has been clearly associated with asbestosis. In subsequent years, the permissible exposure limit was lowered such that even marginally compliant workplaces posed no threat to the workers of a lifetime dose that would reach the exposure level required for asbestosis.[20] Accordingly, the expected decrease in the incidence of the disease asbestosis occurred, and is widely accepted.

True asbestosis occurred in worker populations with extensive and extended pre-OSHA exposures, such as shipyard workers who spent years tearing off insulation in ships, or asbestos miners and millers who worked extensively with raw asbestos.[21] These are the populations whose lifetime exposures could exceed the 25-100 f/cc year dose needed to produce asbestosis.[22] On the other hand, the jobs in which an infinitesimally small exposure to asbestos may have occurred are legion because of the widespread use of asbestos-containing products — but those products produce very little (if any) asbestos exposure and have never been considered a cause of lung cancer — until now, by many plaintiffs' experts.

## Any Increase In Claims Of Asbestos-Related Lung Cancer Is Scientifically Unsupported

Despite the fact that few living persons had exposure to asbestos at a level that even remotely approaches the long-recognized dose required to cause asbestosis, many members of the asbestos plaintiffs' bar have recently been filing an increasing number of lung cancer lawsuits alleging they are asbestos induced. To address obvious causation hurdles, plaintiffs' experts redefine the criteria for the diagnosis of asbestosis (by employing far weaker criteria), or change the criteria for attribution such that any reported asbestos exposure or any exposure "above background" might be, or is, a legal cause of the lung cancer.

Working off loose medical criteria, plaintiff experts in today's litigation often claim to find "asbestosis" in workers who have been in the vicinity of an asbestos-containing product and at best, have only minimal or incidental exposure to asbestos from activities such as a limited number of brake repair jobs, removing a few gaskets, or handling bonded products. Similarly, these same experts opine that every workplace exposure, no matter how trivial, combines with smoking to induce lung cancer. This initiative substitutes proof of the requisite exposure dose through the diagnosis of asbestosis, lung fiber burden analysis or legitimate dose reconstruction, with subjective testimonial work history that references the presence of asbestos or asbestos products and "dust." The asbestos plaintiffs' bar's causation theory for these cases is a departure from traditional causation analysis for attribution of asbestos exposure to a lung cancer.

The consensus is that smoking is the recognized culprit in at least eighty percent of all lung cancer cases.[23] The lung cancer risk attributable to smoking has grown steadily over time — 11.35 times that of non-smokers in the study period 1959 to 1965 (Cancer Prevention Study CPS-I), 22.36 times that of non-smokers in the study period 1982-1988 (CPS-II).[24] Any other lung cancer cause or contribution diminishes to insignificance when compared with smoking alone. The estimates as to the portion of cases unrelated to tobacco ranges from 4% to 10%, though the exact incidence is uncertain. The primary factors closely tied to lung cancer in never smokers are radon, secondhand tobacco and indoor air pollution.[25] Other causalities include environmental tobacco smoke (ETS), cooking fumes, inherited genetic susceptibility, occupational and environmental exposures to carcinogens, hormonal factors, preexisting lung disease, and oncogenic viruses.[26] Even absent a smoking history, the risk attributable to asbestos exposure is roughly equal to that presented by exposure to heavy metals or polycyclic aromatic hydrocarbons, and is lower than the risk attributed to work-related environmental tobacco smoke. At the end of the day, the litigation trend to seek out and attribute smoking lung cancers to the combined effects of smoking and minimal asbestos exposure is a distortion of the science — a science that requires the presence of asbestosis or evidence of a lifetime dose of asbestos exposure sufficient to cause asbestosis, for attribution.

## The Abandonment Of Traditional Standards Of Causation: Theories Of Helsinki, Synergy And Diagnostic Criteria

Traditionally, the medical community was in agreement that asbestosis, or at least a dose consistent with the dose required to contract asbestosis, was necessary to link asbestos exposure to lung cancer. The consensus required a showing of exposure to asbestos at very high concentrations — not "any exposure above background" as espoused by the new generation of asbestos plaintiffs' experts. Under standard toxicology principles and tort law causation standards (e.g., proof of a sufficient dose to cause the disease), plaintiffs should have an increasingly difficult time pursuing smoking lung cancer cases in the asbestos arena. But so far, they have not. Instead, experts utilized by the asbestos plaintiffs' bar have launched a three prong attack under the guise of Helsinki, the concept of "synergy," and reframed diagnostic criteria. Each of these is addressed in turn.

*The Misuse of Helsinki.* Recently, asbestos plaintiffs' experts have distorted observations published following a symposium in Europe that has become known in asbestos litigation parlance as "the Helsinki Criteria." The symposium in Helsinki was a European-focused gathering to accommodate a claiming process not unlike the no fault and limited proof administrative process used in the workers' compensation system in the United States. The trade-off in this system of limited proof reduces the burden, but also limits compensation — a system separate and distinct from proof and compensation in the tort law system practiced in U.S. courts.

Cleverly, but disingenuously, the asbestos plaintiffs' bar grasp Helsinki Criteria and regularly try to apply its

European no-fault standards (but not the resultant limits on compensation) to both mesothelioma and lung cancer cases. Specifically, the plaintiffs try to substitute Helsinki for the causality element required in the U.S. tort system where compensation is virtually unlimited. The Helsinki Criteria was generated by only nineteen symposium participants and has never been adopted by any scientific medical body. Moreover, deficiencies in the Helsinki criteria are identified and analyzed by eminent scientists.[27]

This misuse of the Helsinki Criteria is wrong for two fundamental reasons. First, the Helsinki approach is a regulatory surrogate for actual causation and cannot be the substitute in the United States legal system for the traditional requirement founded on epidemiology, of a quantified dose proving to be causative. Second, even the generous Helsinki Criteria require a very heavy exposure to asbestos before declaring diffuse fibrosis to be asbestosis:

1. One year of heavy exposure or five to ten years of moderate exposure;

2. Estimated cumulative exposures to mixed (amphibole and Chrysotile) fibers of 25 fiber years;

3. A lung fiber burden within the range recorded for asbestosis in the same laboratory;

4. Retained fiber levels of 2 million amphibole fibers per gram of dry lung tissue as determined by electron microscopic analysis;

5. Asbestos body concentrations determined by light microscope analysis greater than 10,000 per gram of dry lung tissue.

Yet, the asbestos plaintiffs' experts ignore these intense exposure requirements and substitute "exposure," or "dust" or even a person's mere presence in a workplace in which asbestos is present as the basis for finding asbestosis. This distortion of Helsinki based on a subjective narrative work history, takes the place of years of scientific analysis on the importance of dose to the determination of causation and attribution. And that subjective narrative becomes the gravamen of causation. This juxtaposition of the "any exposure" theory into the Helsinki context converts a lung cancer diagnosis into a compensable tort suit. In so doing, principles of proof in law, science and medicine are abandoned.

*The Distortion of "Synergy."* Separate and apart from misapplication of the Helsinki Criteria, the asbestos plaintiffs' experts cling to a "synergy" theory between smoking and asbestos in the causation of lung cancer. The problem asbestos plaintiffs' experts face is smoking. Virtually every lung cancer case on the asbestos docket involves a heavy smoker, often up to 100 pack years or more. There is no question this kind of smoking history is more than sufficient to cause lung cancer. And to avoid this obvious hurdle, plaintiff experts resort to the "synergy" concept by claiming that asbestos fibers combined with smoking cause lung cancer. Rather than running from the dramatic smoking history, they embrace it (gingerly) and malign to convince the courts and juries that "synergy" comes into play whenever there is an asbestos exposure of any sort.

This approach greatly distorts the synergy concept. Years ago, some studies indicated a synergistic relationship between smoking and heavy asbestos exposure in the causation of lung cancer.[28] In addressing the union of asbestos insulation workers at their international convention, asbestos-related disease pioneer Dr. Irving J. Selikoff spoke directly and candidly with the insulators about the effects of smoking and the risk of lung cancer:

> "[F]rom 1963, to the present time, in 1967 . . . I have yet to see a lung cancer in an asbestos worker who didn't smoke cigarettes . . . cancer of the lung could be wiped out in your trade if you people wouldn't smoke cigarettes, period."[29]

Though Dr. Selikoff embraced the theory of synergy between asbestosis and smoking in the causation of lung cancer, he did not have the benefit of more recent and more complete studies (i.e. CPS-II). The more recent studies of smoking cohorts of the era involved in current cases demonstrate a far higher incidence of disease related to smoking alone and calls into question the synergistic effect.

Under the "synergy theory," early reports suggested that the lung cancer risks associated with smoking and asbestosis were greater than the combined risks associated with the independent presence of either smoking or asbestosis. Smoking patterns and cigarette composition underwent significant changes from the pre-WWII

era continuing today. Today's plaintiffs who are classified as smokers associate with the later studies that clearly demonstrate the combination of risks associated with smoking and asbestosis are additive and not multiplicative — that is, not synergistic.

But fundamentally, synergy does not even come into play unless both the smoking and asbestos exposures are separately sufficiently extensive to induce the lung cancer independent of each other. Thus, a heavily exposed asbestos worker who only smoked a few packs of cigarettes one year could not blame smoking for "synergistically" causing his lung cancer. And a heavy smoker cannot claim a small amount of asbestos exposure played any role in his lung cancer. Once again, the plaintiff experts' attempts to extend causation to the lowest levels of exposure runs counter to long-established and reliable scientific studies. Those studies make clear that asbestos exposure, absent the requisite dose level to cause asbestosis, does not result in an increased incidence of lung cancer whether or not the person was a smoker. Some experts in fact, believe that synergy only exists between heavy smoking and asbestosis itself, not just asbestos exposures.[30]

*The Plaintiffs' Reinvention of Diagnostic Criteria for Asbestosis.* A small group of writers, most of whom are experts for the asbestos plaintiffs' bar, advocate that any asbestos exposure "above background" — levels that do not (and cannot) cause asbestosis, combines with smoking to cause lung cancer. This unscientific approach ignores decades of epidemiology and well-established conclusions regarding the relationship between asbestosis and the occurrence of lung cancer. The asbestos plaintiffs' bar standard lacks certifiable scientific support and is unworkable.

Recognizing this, the plaintiff experts bolster "exposure" claims by diagnosing asbestosis in persons who clearly do not have asbestosis. Though there is a legitimate debate as to whether it is the physical presence of asbestosis that presents the lung cancer risk, or the dose of asbestos exposure that results in asbestosis that represents the lung cancer risk — that debate is premised upon a legitimate diagnosis of asbestosis. A diagnosis of asbestosis indicates a very heavy exposure, and no such diagnosis can occur in its absence.

Plaintiff experts find support for weakened diagnostic criteria for asbestosis in certain publications of the last decade. For instance, the most recent American Thoracic Society (ATS) Criteria for Diagnosis of Nonmalignant Asbestos Related Diseases,[31] weakens the causation and attribution standards of the past by failing to require an extended and intense history of asbestos exposure, instead resorting to the vague phrase "occupational and environmental history of exposure."[32] The asbestos plaintiffs' bar and its experts manipulate these criteria to diagnose asbestosis in lung cancer claimants, where asbestosis does not exist and concomitant exposures do not support a diagnosis of asbestosis in the first instance. These "false positive" findings are inconsistent with the medical literature and easily convert a smoking lung cancer into an asbestos/synergy lawsuit.

And along with the ATS change in the definition of what constitutes asbestosis, so too has the asbestos plaintiffs' bar articulation of the dose necessary to achieve these less stringent criteria for diagnosis. In particular, the diagnostic criteria used in Selikoff's original insulators (for whom the diagnosis of asbestosis for all of insulators with lung cancer was made on pathology or under the "Original ATS Diagnostic Criteria") is substituted using a more lenient standard such as is found in today's litigation-influenced literature.[33] It is scientifically invalid to read the medical literature on asbestos and cigarette smoking — which has not changed in the last ten years or more — by substituting the definition of the foundational requirement of asbestosis with the far more liberal diagnostic definition advocated by the asbestos plaintiffs' bar today.

## The Need For Medicolegal Guidelines

Courts faced with one or more of these new lung cancer cases need to apply standard and well-accepted scientific principles to remove unwarranted cases from the legal system. The alternative is an explosion of asbestos lawsuits when the real incidence of asbestos-induced disease is in decline. Courts should start this process with a return to legitimate principles of toxicology and the primary requisite of proving causation through dose and epidemiology. The concept of receiving a sufficient dose to cause disease is at the heart of toxicology. "All substances are poisonous — there is none which is not; the dose differentiates a poison from a remedy."[34] Courts across the country in the last ten years have rejected the notion that every asbestos exposure is a cause of disease because that hypothesis is

unproven, speculative, and inconsistent with logic and good science.[35] Even if certain asbestos courts have grown accustomed (inappropriately, given the importance of a dose assessment) to allowing low exposure mesothelioma cases to go to the jury without proof of causative dose, those courts must take a stand against these speculative, low exposure lung cancer cases. Experts universally agree that lung cancer requires a much higher exposure level and thus mere speculation about seeing "dust" or being in the presence of asbestos should never suffice to defeat summary judgment in a smoking lung cancer case.

In addition to requiring proof of a causation dose, courts addressing lung cancer — clothed as asbestos cases, must insist that plaintiffs prove doses of exposure consistent with those in published epidemiological studies documenting excess of lung cancer (controlled for smoking) in asbestos-exposed worker populations. There is no shortage of such studies. Asbestos is one of the most heavily studied workplace substances in history. And generally, tobacco is the most heavily studied carcinogen of all time. Epidemiology is the "gold standard" for latent disease causation. If the asbestos plaintiffs' bar cannot point to epidemiological studies showing increased lung cancers at low exposure levels (and they cannot, because no such body of studies exist), courts should summarily dismiss affected cases and prevent them from proceeding at the outset.

The United States Federal Judicial Center's *Reference Manual on Scientific Evidence* Second Edition (2000) and Third Edition (2011) rely on Sir Bradford Hill's approach to the determination of cause and effect relationships. Indeed, Dr. Hill's criteria[36] underscore the foundation upon which science from the most basic level upward is based: correlation does not demonstrate causation. In the context of lung cancer, while requisite levels of asbestos exposure may be a risk factor in the development of lung cancer, it is not axiomatic that asbestos exposure is causative of lung cancer. It is well-established that asbestos exposure is nearly universal in urban populations in the United States, yet, even most asbestos plaintiffs' experts concede that ambient asbestos exposures in urban environments is not a scientifically recognized cause of asbestos induced disease, including lung cancer. Rather, the level of exposure recognized as a cause is thousands to ten thousands times higher than the innocuous level of background exposure.

## Conclusion

The recent surge in lung cancer filings ignores well-recognized studies on lung cancer causation. Instead, the asbestos plaintiffs' bar advances an ambiguous approach that advocates for an unquantifiable synergy between smoking and lung cancer. The standard is unworkable and replaces science with speculation and conjecture; neither of which has any place in the field of medicine or law. Moreover, the abandonment is not founded on any epidemiological evidence, but rather subjective reports of work history because exposures sufficient to cause asbestosis are now exceedingly rare given the promulgation of OSHA and other regulations. The bottom line is that the arguments advanced by the asbestos plaintiffs' bar and their experts abandon the epidemiological moorings that establish the threshold necessary before lung cancer can be attributed to asbestos exposure while at the same time ignoring the known relationship between smoking and lung cancer.

The purpose of this article is not to call for a change in the standard required to attribute lung cancer to asbestos exposure; it is simply a call for a return the standard based on epidemiological evidence — evidence which supports an increased risk of developing lung cancer based upon a diagnosis of asbestosis or an exposure dose equal (based on objective laboratory analysis, rather than subjective report and recollection) to that at which asbestosis is known to occur under long-recognized diagnostic standards. Absent these criteria, there is simply insufficient epidemiological evidence to conclude that asbestos exposure played any role in the development of a lung cancer. At the same time, the known relationship between smoking and the development of lung cancer is too significant to be disregarded.

## Endnotes

1. *See* ASBESTOS AND ITS DISEASES 140 (John E. Craighead et al. eds.) (2008) ("Now, asbestosis is a disappearing disease. Modern governmental regulations controlling the use of asbestos and public awareness have reduced substantially its incidence.")

2. *See* Eduardo C. Robreno, *The Federal Asbestos Product Liability Multidistrict Litigation (MDL-875): Black Hole or New Paradigm?*, 23 WIDENER L.J. 97, 102 (2013).

3. Since the asbestos litigation began, hundreds of thousands of lawsuits have been filed by plaintiffs who were not sick and had no asbestos-related impairment. Cardozo Law School Professor Lester Brickman, an expert on asbestos litigation, has said, "the 'asbestos litigation crisis' would never have arisen" if not for the claims filed by the non-sick. Lester Brickman, *Lawyers' Ethics and Fiduciary Obligation in the Brave New World of Aggregative Litigation*, 26 WM. & MARY ENVTL. L. & POL'Y REV. 243, 273 (2001); *see also* James A. Henderson, Jr. & Aaron D. Twerski, *Asbestos Litigation Gone Mad: Exposure-based Recovery for Increased Risk, Mental Distress, and Medical Monitoring*, 53 S.C. L. REV. 815, 823 (2002) ("By all accounts, the overwhelming majority of claims filed in recent years have been on behalf of plaintiffs who . . . are completely asymptomatic.").

4. *See* Mark A. Behrens, *What's New in Asbestos Litigation?*, 28 REV. LITIG. 501, 502 (2009) ("In the earlier years of asbestos litigation, most cases were filed by people with cancer and other serious conditions. From the late 1990s until recently, the vast majority of claimants were not sick. The mass recruitment of non-malignant claims has ceased, and the litigation is re-focused on people with mesothelioma (a type of cancer) and other serious conditions.").

5. *See id.*

6. *See* Am. Cancer Soc'y, *What Are the Key Statistics About Malignant Mesothelioma?*, http://www.cancer.org/cancer/malignantmesothelioma/detailedguide/malignant-mesothelioma-key-statistics; Mesothelioma Incidence by Gender, 1975-2010 (chart with data from Nat'l Cancer Inst., SEER Incidence Rates), http://www.asbestos.com/mesothelioma/incidence.php. A 2015 review of asbestos-related liabilities reported to the Securities and Exchange Commission by more than 150 publicly traded companies showed that "[f]ilings have shown no decline in the last seven years, a finding that is perhaps inconsistent with predictions of epidemiological models." Mary Elizabeth Stern & Lucy P. Allen, *Defense Costs Dropped in 2014, While Claim Filings, Dismissal Rates, and Indemnity Dollars Remained Steady*, at 1 (NERA Economic Consulting June 4, 2015), http://www.nera.com/content/dam/nera/publications/2015/ PUB_Asbestos_Litigation_Trends_0615.pdf; *see also* Jenni Biggs et al., *A Synthesis of Asbestos Disclosures from Form 10-Ks — Updated* 1 (Towers Watson June 2013) (mesothelioma claim filings have "remained near peak levels since 2000."), http://www.towerswatson.com/Download Media.aspx?media=%7B67C014AD-30A0-43A8-BF7D-987809C35E76%7D.

7. William J. McCarthy et al., *Lung Cancer in Never Smokers, Epidemiology and Risk Prediction Models*, 32 RISK ANALYSIS (Suppl. 1) S69 (2012), http://www.ncbi.nlm.nih.gov/pubmed/ 22882894.

8. *Id.*

9. *See* Am. Cancer Soc'y, *Learn About Cancer*, http://www.cancer.org/cancer/lungcancer-non-smallcell/detailedguide/non-small-cell-lung-cancer-key-statistics.

10. *See* PATHOLOGY OF OCCUPATIONAL LUNG DISEASE 341 (Andrew Churg et al. eds.) (1998) ("The fact that any exposure to asbestos, no matter how small, leads to an increased lung cancer risk is unsupported by any direct epidemiologic evidence . . .").

11. *See Id.*

12. William Weiss, *Asbestosis: A Marker for the Increased Risk of Lung Cancer Among Workers Exposed to Asbestos*, 115 CHEST 536 (1999), http://journal.publications.chestnet.org/ data/Journals/CHEST/21906/536.pdf.

13. *See* Doll, R., *Mortality from Lung Cancer in Asbestos Workers*, 12 BRIT J. INDUS. MED. 81-86 (1955).

14. Concentration of asbestos fibers is expressed as the number of fibers per cubic centimeter (cc) of air multiplied by the number of occupational years that an individual was exposed to at that concentration.

15. *See e.g.* ASBESTOS: RISK ASSESSMENT EPIDEMIOLOGY, AND HEALTH EFFECTS 572 (Ronald F. Dodson et al. eds.) 2011; Kevin Brown, *A Threshold for Asbestos Related Lung Cancer* 43 BRIT J. INDUS. MED. 556-558 (1986).

16. *See* MINERAL FIBERS AND HEALTH 143 (Douglas Liddel et al. eds) (1991) ("... lung cancer risk from general environmental asbestos exposure, such as buildings containing well-maintained asbestos products, are either non-existent or so low that they could never be detected.")

17. *See* PATHOLOGY OF ASBESTOS-ASSOCIATED DISEASES 54-55 (Tim D. Oury et al. eds.) (3d ed. 2014).

18. *See* Geoffrey Berry et al., *Asbestosis: A Study of Dose Response Relationships in an Asbestos Textile Factory*, 36 BRIT. J INDUS. MED. 98-112 at tbl 4 (1979), http://oem.bmj.com/content/36/2/98.short; Ludovic M. Lacquet et al., *Roentgenograpic Lung Changes, Asbestosis and Mortality in a Belgium Asbestos Cement Factory*, 30 IARC SCIENTIFIC PUBLICATIONS 783, 789 (1980), http://www.researchgate.net/publication/15971434_Roentgenographic_lung_changes_asbestosis_and_mortality_in_a_Belgian_asbestos-cement_factory (no cases of asbestosis below 100 f/cc and one case in the range of 100-200 f/cc).

19. Occupational Safety & Health Admin., *Occupational Exposure to Asbestos: Regulatory History*, https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=PREAMBLES &p_id=775.

20. *See id.*

21. PATHOLOGY OF ASBESTOS-ASSOCIATED DISEASES, *supra* note 17, at 54.

22. *Id.*

23. *See* McCarthy, *supra* note 7. Studies have shown that historically, blue-collar workers tend to have a much more significant smoking history than the general population. *See* D. Cal Ham et al., *Occupation and Workplace Policies Predict Smoking Behaviors: Analysis of National Data from the Current Population Survey*, 53 J. OCCUP. ENVTL. MED. 1337 (2011).

24. *See, e.g.*, Michael J. Thun et al., *Trends in Tobacco Smoking and Mortality From Cigarette Use in Cancer Prevention Studies I (1959 through 1965) and II (1982 through 1988)* in SMOKING AND TOBACCO CONTROL MONOGRAPH 8: CHANGES IN CIGARETTE-RELATED DISEASE RISKS AND THEIR IMPLICATIONS FOR PREVENTION AND CONTROL (Nat'l Cancer Inst. Feb. 1997), http://cancercontrol.cancer.gov/brp/tcrb/monographs/8/m8_complete.pdf.

25. *See* Am. Cancer Soc'y, *Do We Know What Causes Non-Small Cell Lung Cancer?* (last revised Mar. 4, 2015) ("Lung cancer in non-smokers can be caused by exposure to radon, secondhand smoke, air pollution, or other factors. Workplace exposures to asbestos, diesel exhaust, or certain other chemicals can also cause lung cancers in some people who do not smoke."), http://www.cancer.org/cancer/lungcancer-non-smallcell/detailedguide/non-small-cell-lung-cancer-what-causes.

26. *See* McCarthy *supra* note 7.

27. *See* Allen Gibbs et al., *The 'Helsinki Criteria' for Attribution of Lung Cancer Cases to Asbestos Exposure; How Robust are the Criteria?*, 131 ARCHIVES OF PATHOLOGY & LAB. MED. 181 (2007), http://www.thefreelibrary.com/The+'Helsinki+Criteria'+for+attribution+of+lung+cancer+to+asbestos...-a0163098555.

28. *See e.g.* E.C. Hammond et al. "Asbestos Exposure, Cigarette Smoking and Death Rates" 330 ANN NY ACAD SCI 473-490 (1979).

29. J. Irving Selikoff's Address to the Delegates of the Twenty-first Convention of the International Association of Heat and Frost Insulators and Asbestos Workers, Chicago, Ill., Sept. 1967, at 71, http://www.worldcat.org/title/dr-irving-j-selikoffs-address-to-the-delegates-of-the-twenty-first-convention-of-the-international-association-of-heat-and-frost-insulators-and-asbestos-workers-chicago-illinois-september-1967/oclc/12288308.

30. *See* Robert N. Jones et al., *Asbestos Exposure, Asbestosis, and Asbestos-Attributable Lung Cancer*, 51 THORAX (Suppl. 2) S9 (1996), http://thorax.bmj.com/content/51/Suppl_2/S9.citation?51/Suppl_2/S9.

31. *See* Am. Thoracic Soc'y, *Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos* (adopted Dec. 12, 2003), 170 AM. J. RESPIRATORY & CRITICAL CARE MED. 691 (2004), http://www.thoracic.org/statements/resources/eoh/asbestos.pdf.

32. *See id.* at 692 Table 1.

33. *See, e.g.*, Steven B. Markowitz et al., *Asbestos, Asbestosis, Smoking and Lung Cancer: New Findings for the North American Insulator Cohort*, 188 AM. J. RESPIRATORY & CRITICAL CARE MED. 90 (2013), http://www.atsjournals.org/doi/pdf/10.1164/rccm. 201302-0257OC.

34. David L. Eaton, *Scientific Judgment and Toxic Torts—A Primer In Toxicology For Judges And Lawyers*, 12 J.L. & POL'Y 5, 11 (2003).

35. *See, e.g.*, Betz v. Pneumo Abex LLC, 44 A.3d 27 (Pa. 2012); Bostic v. Georgia-Pacific Corp., 439 S.W.3d 332 (Tex. 2014); Moeller v. Garlock Sealing Tech., LLC, 660 F.3d 950 (6th Cir. 2011); Martin v. Cincinnati Gas & Elec. Co., 561 F.3d 439 (6th Cir. 2009); Smith v. Ford Motor Co., No. 2:08-CV-630, 2013 WL 214378 (D. Utah Jan. 18, 2013); Anderson v. Ford Motor Co., 950 F.Supp.2d 1217 (D. Utah 2013); Sclafani v. Air & Liquid Sys. Corp., No. 2:12-CV-3013, 2013 WL 2477077 (C.D. Cal. May 9, 2013).

36. In his 1965 address, Sir Bradford Hill asked in the situation where "our observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance," then "what aspects of that association should we especially consider before deciding that the most likely interpretation of its causation?" Dr. Hill then provided a list of prime considerations, to be applied to a significant body of epidemiologist studies to determine whether the findings of those studies support a causation conclusion; among them: 1) Statistical strength; 2) Consistency of the Association; 3) Specificity of the Association; 4) Temporality of the Association (Exposures occurs before disease, also considerations of latency); and 5) Biological Gradient (Dose response curves). Austin Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 PROCEEDINGS OF THE ROYAL SOC'Y OF MED. 295 (1965), http://www.edwardtufte.com/tufte/hill. ■

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