Lung Cancer

The Future of Asbestos Litigation?

By David N. Lutz
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Although there may be overlap between mesothelioma and lung cancer cases, differing medical issues may allow defendants to pare many cases through motion practice and to better defend others.

Asbestos litigation has become a multi-billion dollar cottage industry, largely because of mesothelioma cases. The rate of mesothelioma has peaked, or soon will peak, at roughly 3,000-3,500 diagnoses per year. The number of mesothelioma lawsuit filings has increased only slightly as most mesothelioma diagnoses now result in a lawsuit. While it is predicted that the rate of mesothelioma will not return to background for at least another 20 years, the incidence of mesothelioma will decline over those years and mesothelioma case filings will presumably also decline. As mesothelioma case filings decline in the future, defense lawyers have asked "What will be the next asbestos?"

The "Next Asbestos" Is Asbestos

In national seminars, plaintiffs' lawyers have said that they intend to return to cases alleging lung cancer as a result of asbestos exposure as mesothelioma cases decline. Although there are roughly 3,000–3,500 mesothelioma diagnoses per year, there are roughly 200,000 lung cancer diagnoses per year. Thus, if lung cancer cases could be linked to asbestos, there is a much greater pool of potential plaintiffs and cases. Some plaintiffs' lawyers have candidly admitted they have not been able to pursue lung cancer cases with full vigor because they have been so busy with the higher value mesothelioma cases. It appears that the "next asbestos" will be asbestos, specifically lung cancer cases. It is attractive to the plaintiffs' bar to return to lung cancer cases because much of the state-of-the-art and company-knowledge evidence appears on its face to be similar. In a cookie-cutter type of litigation, plaintiffs' lawyers may think that they can use the same playbook by merely substituting the mesothelioma section with a lung cancer section.

The effect on the defense community of this shift will likely be a trade of verdict

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potential for case volume. Settlement values in mesothelioma cases are in the seven figures in some jurisdictions and verdict potential can be eight figures. Verdicts in lung cancer cases tend to be lower than in mesothelioma cases and settlement values therefore tend to be lower, as well. Plaintiffs can claim that mesothelioma is a "signal" tumor that is usually caused by asbestos exposure, and it can be difficult for defendants to blame the plaintiff. The etiology of lung cancer, however, is very different. Although plaintiffs' experts attribute most mesotheliomas to asbestos, few lung cancers are caused by asbestos. Instead, most lung cancers are caused by smoking. Indeed, smoking is a stronger cause of lung cancer than asbestos is a cause of mesothelioma. *See* United States Cancer Statistics: 1999 - 2013 Incidence, WONDER Online Database, United States Department of Health and Human Services, Centers for Disease Control and Prevention and National Cancer Institute (2016) accessed at http://wonder.cdc.gov/ on Mar 7, 2017. The defense community can expect that the shift back to lung cancer cases will result in a higher volume of cases, each of which has a smaller average verdict potential and settlement value than mesothelioma cases.

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Smoking and Lung Cancer

Smoking causes 443,000 deaths per year and an economic burden of $193 billion annually. *See generally* Surgeon General 2010 Report. Half of long-term smokers die from smoking related disease. Each inhalation of tobacco smoke leads to the transfer of many chemical toxicants from the lungs to the blood stream, which carries them to almost every part of the body. The compounds cause damage to the genetic makeup of cells. There is a rapid and sharp increase in the biologic response from even low levels of exposure to tobacco. Genetic changes alter cellular pathways that foster uncontrolled cell growth and defeat normal mechanisms to restrain their growth. Smoking overwhelms lung defense mechanisms.

Cigarette smoke contains at least 69 chemicals known to cause cancer. Damage from tobacco smoke is immediate and chemicals in tobacco smoke reach the lungs quickly with every breath. There is no safe level of exposure of tobacco smoke; any exposure to tobacco smoke is harmful. Surgeon General 2010 Cover Sheet, Foreword. Smoking causes 85-95 percent of all lung cancers. Surgeon General 2010 Report. Smoking 20+ cigarettes (one pack) per day raises the risk of lung cancer 10-25 times. The duration of smoking adds more risk than the extent of exposure; *i.e.*, a longer duration of fewer packs per day adds more risk than a shorter duration of more packs per day. Selikoff's study of 17,800 insulators revealed that 14.5 percent of insulators smoked died of lung cancer, while barely 1 percent of non-smoker insulators died of lung cancer. Selikoff and Lee, *Asbestos and Disease* (1978) p. 327.

The plaintiff's deposition will likely secure the admission that the plaintiff knew that smoking caused lung cancer. After all, the first Surgeon General warnings date back to 1964 and typical plaintiff smokers have picked up a pack of cigarettes containing a warning tens of thousands of times in their lifetime. Plaintiffs' primary defense to the smoking defense will be that it is not their fault that they smoked because they were addicted. The deposition should therefore explore efforts to quit. Plaintiffs often say that they quit smoking for a period of time and then resumed later. The ability to quit and the voluntary choice to resume, however, is inconsistent with a claim of addiction.

Asbestos, Asbestosis, and Lung Cancer

It is generally accepted that asbestos-induced lung cancer is a function of fiber dose (and fiber burden), with a threshold for increased lung cancer risk. It has been widely believed that asbestosis is a prerequisite to asbestos-caused lung cancer because there is a close correlation between fiber burden levels associated with increased lung cancer risk and the presence of asbestosis. People with asbestosis have a higher rate of lung cancer than those without, and those with progressive asbestosis have higher risk than those with static asbestosis. *See generally* Churg and Green, Pathology of Occupational Lung Disease (1998); Roggli, *et al.*, Pathology of Asbestos Related Diseases, 3rd Ed (2014); and Asbestos Risk Assessment, Epidemiology and Health Effects (Dodson and Hammar eds., 2011).

Some experts believe that inflammation and interstitial fibrosis are responsible for lung cancer, hence asbestosis is a prerequisite. McDonald's study of chrysotile workers showed a statistically significant increased risk of lung cancer in workers with asbestosis, but no increased risk in workers without it. Studies of lung cancer in insulators, asbestos cement workers and amosite asbestos miners by Kipen, Newhouse, and Weil have found lung cancer almost exclusively in people with asbestosis. Studies have also found that the risk of lung cancer in people with severe asbestosis is higher than the risk of lung cancer in those with slight asbestosis. A study by Henderson found that in 35 lung cancer cases, most had asbestosis; those who did not have asbestosis were smokers. Karjalainen found that the risk of lung cancer increased as the asbestos fiber burden increased, but the association disappeared when they removed patients with asbestosis.

A study by Camus found no statistically significant increased lung cancer risk from environmental exposure to chrysotile in Quebec with an average exposure of 25 Fiber-Years (F-Y), defined as the extent of average exposure in fibers per cubic centimeter (f/cc) times the duration of exposure in years. Camus, et al, "Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer," *N Engl J Med* 1998; 338:1565-71. For example, exposure at a level of 1 f/

cc for 25 years would be 25 F-Y. Even under the Helsinki Criteria, regularly cited by plaintiffs' experts in mesothelioma cases, attribution to asbestos as a substantial contributing factor can be stated with probability at the 25 F-Y exposure level. The 2006 edition of *Asbestos Risk Assessment, Epidemiology and Health Effects* cites studies from Germany, Poland, and elsewhere that confirm that the risk of asbestos-caused lung cancer roughly doubles (which is arguably "more probable than not") at a cumulative exposure of 25 F-Y. Dodson and Hammar at 290. Even if asbestosis is not a prerequisite to asbestos-caused lung cancer, other experts believe that asbestosis is the only consistent and reliable marker that someone had exposure that was great enough to cause lung cancer. For example, prominent asbestos pathologist Victor Roggli cites a study that found that the fiber burden necessary to cause lung cancer was found in 82 percent of cases with asbestosis, but only 6 percent of cases without asbestosis. Thus, even if asbestosis is not a prerequisite to asbestos-caused lung cancer, the asbestos exposure necessary to cause lung cancer is comparable to the exposure necessary to cause asbestosis.

The cumulative exposure necessary to cause asbestosis is at least 20-25 F-Y. The Royal Ontario Commission concluded that asbestosis is unlikely at cumulative exposures under 25 F-Y. See Dodson and Hammar, *Asbestos Risk Assessment, Epidemiology, and Health Effects* (2nd Ed.), CRC Press 2011, p. 283. A Chinese x-ray study of manufacturing workers found 22 F-Y as a threshold for 1 percent prevalence of early stage asbestosis. *Id.* A study of asbestosis in South Carolina textile workers found that they usually had 20+ F-Y cumulative exposure. *Id.* Well-respected pathologist Churg concluded that asbestos-induced lung cancers do not appear until a threshold cumulative exposure is reached and that threshold is in the range of cumulative exposure necessary to cause asbestosis, probably 25-100 F-Y. Churg and Green, *Pathology of Occupational Lung Disease* (2nd Ed.) Williams & Wilkins (1998), p. 313, 341. At the current OSHA permissible exposure of .1 f/cc, it would take 200-250 years of asbestos exposure to cause cumulative exposure of 20-25 F-Y and potentially cause lung cancer.

There is no medical or scientific support for the proposition that low exposures to asbestos cause lung cancer. Although regulatory agencies apply a linear model to extrapolate downward from known and quantifiable risks at higher exposure levels, many experts believe that this downward extrapolation lacks scientific validity. Studies of cohorts with relatively low-level exposure (1-5 f/cc) do not show increased risk of lung cancer.

Plaintiffs' experts believe that it is the concentration of asbestos that is most important in causing lung cancer and not the presence of asbestosis. Whether asbestosis is a prerequisite or exposures sufficient to cause asbestosis is the threshold, asbestos exposure can cause lung cancer only after extensive exposure of long duration, either exposure sufficient to cause asbestosis or in conjunction with actual asbestosis.

Synergy

Synergy is the concept that the risk introduced by two toxins in combination is more than the cumulative risks added by each one individually. Synergy between smoking and asbestos exposure was originally shown in the Selikoff cohort of insulators in the 1960s and Selikoff urged the union members to quit smoking. In rough terms, Selikoff found that significant asbestos exposure increased lung cancer risk 5-fold, smoking increased it 10-fold, and the combination of both smoking and significant asbestos-exposure increased it 50-fold. The ratios applicable to the Selikoff cohort do not apply to most cohorts or plaintiffs, however, because the Selikoff cohort, by virtue of the fact that they were insulators, had exposures significantly higher than most cohorts or plaintiffs. Thus, the synergy data will overstate the contribution of asbestos exposure to lung cancer in most cases.

A recent study by Markowitz and colleagues confirms that there is synergism between smoking and asbestos exposure in insulators (one of the highest exposure groups ever studied), but only amongst those who have asbestosis. Markowitz, et. al., *Asbestos, Asbestosis, Smoking and Lung Cancer. New Findings from the North American Insulator Cohort*, 188 Am J Resp Crit Care Med 90, 92 (2013). Markowitz compared the relative risks of lung cancer amongst insulators with a control group population with neither asbestos exposure nor smoking. Exposure to asbestos as an insulator increased the risk to 3.6 that of the control group, whereas smoking increased the risk 10.3-fold. *Id.* at 91-92. The combined risk from heavy asbestos exposure and smoking increased the risk to 14.4, which is roughly additive (3.6 + 10.3). Amongst those with asbestosis, the risk of lung cancer in non-smokers was 7.4, but amongst smokers it was 36.8, which was *supra*-additive (but less than multiplicative). McDonald also found synergy amongst miners of chrysotile in Quebec.

The mechanism by which synergy happens in high exposure groups appears to arise from the effects of smoking on the body's defense mechanisms that otherwise protect against contaminants such as asbestos. For example, smoking damages the mucociliary escalator, which helps trap and clear inhaled contaminants. Smoking increases fiber retention in the lung and fiber penetration into the tissues. It is also believed that smoke carcinogens adsorb onto asbestos fibers and that smoking interferes with the clearance of asbestos, especially short fibers.

Cessation of Smoking

Since most lung cancer cases involve plaintiffs who are former smokers, a battleground issue will be the effect of quitting on their lung cancer risk. Although a smoker can reduce the lung cancer risk by quitting, former smokers are still at increased risk of lung cancer. The Surgeon General 2004 report confirmed that even after many years of not smoking, the risk of lung cancer in former smokers remains higher than in persons who never smoked. The Surgeon General also reports that the risk for dying of lung cancer drops only by half 10 years after a smoker quits.

The Markowitz study found that current smokers had a 19.2-fold increased lung cancer risk over never smokers. Even those who had quit 10-19 years earlier had a 5.2-fold greater lung cancer risk and those who quit 20-29 years earlier were still at 2.6-fold increased risk of lung cancer. Even amongst those who had quit over 30 years earlier, there was still a 1.5-fold increased risk of lung cancer. Roggli has confirmed that the risk to former smokers is more than the

risk to never-smokers, but less than for current smokers. Although risk declines over years of cessation, even people who quit 20 or more years ago remain at increased risk.

There are no pathologic features of a lung cancer that permits distinction between asbestos-caused and smoking-caused. Significant asbestos exposure is proven by asbestosis, but some level of exposure may be inferred by tissue markers, such as pleural plaques. Although plaintiffs' experts cite pleural plaques as evidence of attribution of a mesothelioma, they are not markers of exposure necessary to cause lung cancer because the threshold exposure necessary to cause pleural plaques is much smaller than the exposure necessary to cause asbestosis and lung cancer. There are, however, diagnostic markers of extensive smoking history, including emphysema, chronic bronchitis, and chronic obstructive pulmonary disease (COPD).

Whether asbestosis is a prerequisite or exposures sufficient to cause asbestosis is the threshold, asbestos exposure can cause lung cancer only after extensive exposure of long duration, either exposure sufficient to cause asbestosis or in conjunction with actual asbestosis.

Attribution Between Smoking and Asbestos Exposure

The causation standard in many jurisdictions is a variant on the "substantial factor" test. Since most lung cancer cases involve smoking and (alleged) asbestos exposure, a pivotal issue will be how to attribute the lung cancer amongst multiple potential causes. Various medical experts have developed criteria.

Churg, a well-respected asbestos pathologist, opines that 1) if there is asbestosis in a never-smoker, then asbestos caused it; 2) if the plaintiff has asbestosis and is or was a smoker, then smoking also contributed; and 3) if there is smoking history but no asbestosis, then the lung cancer should be attributed to smoking and not asbestos. Churg and Green, Pathology of Occupational Lung Disease 348 (1998). Dr. Roggli opines that lung cancer should be attributed to asbestos exposure only if asbestosis is present or tissue reveals asbestos burden within range observed in patients with asbestosis Roggli, *et al.*, Pathology of Asbestos Related Diseases,197 (3rd ed. 2014).

Friedman's chapter in the book edited and authored in part by prominent plaintiffs' experts Hammar and Dodson noted that the probability that asbestos was a contributing factor relies on the dose-response relationship between cumulative exposure and lung cancer and requires a careful account of the duration and intensity of asbestos exposure, the presence of objective evidence of asbestos exposure as non-malignant disease, and the smoking history. Friedman, *Dodson/Hammar 2006 Book* p.354. Friedman applies the following attribution criteria: 1) if there is asbestosis, then it was asbestos-caused; 2) if there are bilateral pleural plaques or bilateral pleural thickening, then asbestos contributed; 3) if there is documentable cumulative exposure history of 25 F-Y, then asbestos contributed; 4) if asbestos bodies are present in sufficient quantity to cause asbestos disease, then asbestos contributed, 5) if there is no asbestosis or pleural plaques and available pathology shows no interstitial fibrosis or asbestos bodies/fibers, then it is not attributable to asbestos regardless of exposure history and 6) if there is no asbestosis or pleural disease, pathology is not available, a 25 F-Y exposure history cannot be reliably documented, and there is significant smoking history, then asbestos did not contribute. These criteria do not address the fact that even if asbestos contributed, smoking also contributed if the plaintiff smoked. The reliance on pleural plaques is subject to challenge because pleural plaques can occur at levels of exposure below 25 F-Y, and they therefore do not demonstrate 25 F-Y of exposure.

The Helsinki Criteria for attribution of lung cancer to asbestos are:

1. Asbestosis *or*
2. Concentration of 5000+ Asbestos Bodies/gram dry lung tissue *or* 2+ million amphibole fibers >5 microns long/gram dry *or* 5+ million amphibole fibers >1 micron long, *or*
3. Estimated cumulative exposure of 25 F-Y, *or*
4. Occupational history of one year of heavy exposure (manufacture of asbestos products, asbestos spraying, insulation work or demolition of old buildings) or 5-10 years of moderate exposure (such as construction or shipbuilding).

Helsinski Workshop Consensus Report, "Asbestos, asbestosis, and cancer: The Helsinski Criteria for diagnosis and attribution, *Scand J Work Env Health* 1997; 23:311-16. Prominent plaintiffs' experts Hammar and Dodson endorse the following criteria for attribution:

1. Asbestosis as a marker of substantial cumulative exposure, *or*
2. The occurrence of asbestosis in the same workforce, *or*
3. Cumulative exposure of 25 F-Y if mixed fiber, 20 F-Y for amphibole exposures, 25 F-Y for textile workers, and 200 F-Y for pure chrysotile exposures such as Canadian chrysotile miners and friction product exposures, *or*
4. History of 5+ years of exposure before 1975 or 5-10 years exposure after 1975 for exposure to textile workers or insulation workers, or one year of frequent spraying of asbestos insulation; Canadian chrysotile miners and friction products workers are excluded from this factor, *or*
5. For never smokers or smokers who quit 30+ years earlier cumulative exposure of 5 F-Y or exposure at 1/3 the duration of the prior factor, *or*
6. Concentration of amphibole fibers at 5th percentile for cases of asbestosis (which is 25 F-Y for pure amphibole).

Douglas Henderson and James Leigh, *Asbestos and Carcinoma of the Lung*, in Asbestos Risk Assessment, Epidemiology and Health Effects 296-7 (Dodson and Hammar eds., 2011).

These criteria are similar to criteria used in European countries. Germany also uses a 25 F-Y approach. The British Industrial Injuries Advisory Council uses asbestosis or employment as an asbestos textile worker, asbestos sprayer, or asbestos insulation worker (including shipbuilding) for 5+ years before 1975 or 10+ years after 1975.

These criteria are particularly important for "friction" (vehicle brake and clutch) defendants. Roggli noted that the exceptions for friction exposures arise from the low rate of lung cancer among auto maintenance workers, which can be explained by relatively low dust levels, low proportion of asbestos in the dust, and preponderance of very short chrysotile fibers. Nicholson from the Selikoff group estimated brake mechanics exposure at .1-.3 f/cc, insulation work at 15 f/cc, manufacturing at 20-40 f/cc, and ship building/repair at 2. They did not attribute any increased risk of lung cancer (or mesothelioma) to automotive maintenance workers. The Koskinen study in Finland assigned exposure factors of 10 for pipe insulation, 2 for construction work in building repair, 20 for asbestos spraying, and 1 for brake/clutch repair. *See generally* Friedman, *Dodson/Hammar 2006 Book* p.357.

Defense counsel will want to learn plaintiffs' medical experts' methodology for attributing lung cancer to asbestos exposure and the basis for attribution in this particular case.

Diagnosis of Asbestosis

Application of these criteria suggests that plaintiffs will seek evidence that they have underlying asbestosis. A diagnosis of mild asbestosis, particularly if done by chest x-ray rather than CT scan, however, is not necessarily diagnostic of an asbestos-related disease because chest x-rays have a significant rate of false positives and because interstitial fibrosis diagnosed as asbestos-caused may in fact be smoking-caused.

In their 2015 article, *Smoking-associated fibrosis and pulmonary asbestosis*, Kradin, et al. (a plaintiffs' expert in mesothelioma cases) histopathologically studied 24 cases diagnosed as asbestosis of International Labor Organization (ILO) severity >/= 1/0 on chest x-ray. They found that only 6 of the 24 cases actually had asbestosis and that the other 18 cases (which had a mean smoking history of 53 pack-years) showed interstitial fibrosis that was caused by smoking and not asbestos exposure. They concluded that "the clinical diagnosis of mild asbestosis cannot be reliably distinguished from interstitial fibrosis in heavy smokers." All six whose asbestosis was confirmed pathologically had an ILO chest x-ray of 1/1. This study suggests that a chest x-ray read by a B-Reader as 1/0 is likely not a correct diagnosis of asbestosis and is, instead, likely interstitial fibrosis caused by smoking.

Although the Kradin study confirms that mild interstitial fibrosis diagnosed as asbestos on chest x-ray may actually be smoking-caused, another 2015 article suggests that a chest x-ray report of interstitial fibrosis may itself be inaccurate because of false positives. In *Screening of Miners and Millers at Decreasing Levels of Asbestos Exposure: Comparison of Chest Radiography and Thin-Section Computed Tomography*, Terra-Filho and colleagues reported on a study in Brazil that compared the results of chest x-rays and chest CT scans in current and former asbestos miners. In all groups other than the highest exposure group, the chest x-ray found more pulmonary abnormalities than the CT. The authors concluded that chest x-rays were associated with false-positive findings and diagnosed interstitial abnormalities that CT scans revealed were not actually there. This inaccuracy of chest x-rays was more pronounced at lower asbestos exposure levels. The study found the opposite with respect to pleural plaques—that chest x-rays tend to miss existing pleural plaques that a CT scan can detect. The study also found, consistent with the Kradin study, that there was a higher incidence of interstitial abnormalities in current and former smokers than in never smokers, both by chest x-ray and (even more pronounced) by CT. The study also found a similarly higher rate of pleural plaques amongst current and former smokers than never smokers. This is, of course, consistent with literature confirming a higher retention of asbestos fibers in smokers.

Other Causes of Lung Cancer

The most challenging lung cancer cases are those with no smoking history and those cases may rival mesothelioma cases in verdict potential and settlement value.

Defense counsel will want to learn plaintiffs' medical experts' methodology for attributing lung cancer to asbestos exposure and the basis for attribution in this particular case.

Such cases are rare, as almost all lung cancers occur in current or former smokers. *See* Roggli, *et al.*, Pathology of Asbestos Related Diseases, 3rd ed. (2014); Asbestos Risk Assessment, Epidemiology and Health Effects, (Dodson and Hammar eds., 2nd ed. 2011).

Radon is the second leading cause of lung cancer in the United States, behind smoking. *See generally* Environmental Protection Agency, *A Citizen's Guide to Radon: The Guide to Protecting Yourself and Your Family From Radon (*2002). Radon exposure may serve as an alternative explanation for (or at least a contributing cause to) the plaintiff's cancer. Nearly 1 in 15 homes in the United States is estimated to have elevated radon levels, whether due to cracks in the home's foundation, cracks in the walls, or contamination of a groundwater water supply. Multiple studies around the world have shown that increased exposure to radon in a residential setting increases a person's risk of lung cancer, even if that person has never smoked. *See* Daniel Krewski *et al.*, *Residential Radon and*

Risk of Lung Cancer: A Combined Analysis of 7 North American Case-Control Studies, 16 Epidemiology 137 (2005); Janakiraman Subramanian and Ramaswamy Govindan, *Lung Cancer in Never Smokers: A Review,* 25 J. of Clinical Oncology 561, 563–64 (2007). Incidence of lung cancer in those exposed to radon increases, however, if the exposed person was also a smoker. The EPA recommends repairing any home with more than 4 picocuries per liter (pCi/L) of radon in its indoor air, though it warns that even as few as 2 pCi/L can be dangerous. Environmental Protection Agency, *Home Buyer's and Seller's Guide to Radon 17* (2013). Defense counsel should investigate the plaintiff's living conditions, particularly if the plaintiff's home uses a private well or public groundwater supply.

It is not clear to what extent family history provides an alternative cause of lung cancer and to what extent it represents an "egg-shell" plaintiff, who is at increased individual susceptibility to lung cancer caused by exposure to a carcinogen.

The plaintiff's deposition should also explore exposure to heavy metals and other chemical agents. Heavy metal exposure is thought to cause lung cancer by a mechanism that is not well understood. In addition, arsenic, cadmium, nickel, metal dust, PAH, and vinyl chloride are also thought to be associated with lung cancer.

Recent research suggests that two-thirds of lung cancer cases do not have a specific reason. Several studies have suggested that susceptibility to lung cancer is heritable, and that certain genetic markers, mutations, or other idiosyncrasies may drastically increase a person's risk of lung cancer. Amongst American women who have never smoked, Wu et al found that a history of respiratory tract cancer in a parent or sibling was linked to a 30 percent increased risk of lung cancer. Anna H. Wu et al, *Family History of Cancer and Risk of Lung Cancer among Lifetime Nonsmoking Women in the United States,* 143 Am. J. of Epidemiology, 535 (1996). The only association for which the confidence interval exceeds 1.0, however, is a family history of lung cancer in a sister of an adenocarcinoma patient. It is likely that several different factors contribute to genetic susceptibility to lung cancer, and significant progress has been made with genome-wide association studies to begin isolating chromosomal regions associated with lung cancer. *See* Ian A. Yang et al, *Generic Susceptibility to Lung Cancer and Co-Morbidities,* 5 J. Thoracic Disease S454 (2013). It is not clear to what extent family history provides an alternative cause of lung cancer and to what extent it represents an "egg-shell" plaintiff, who is at increased individual susceptibility to lung cancer caused by exposure to a carcinogen. *See* Restatement (Third) of Torts: Phys. & Emot. Harm §31 (2010) ("When an actor's tortious conduct causes harm to a person that, because of a pre-existing physical or mental condition or other characteristics of the person, is of a greater magnitude or different type than might reasonably be expected, the actor is nevertheless subject to liability for all such harm to the person"). Merely demonstrating that the plaintiff was an "egg-shell" plaintiff because of genetic susceptibility to lung cancer may not help the defense if it merely proves that a lower threshold of asbestos exposure caused it.

Cost Effective Defense

Given that the case volume will likely increase, it is imperative that defendants defend lung cancer cases even more cost-effectively than they have defended mesothelioma cases, since there will be a higher case volume. As the docket includes increasing volume of cases with lower settlement value and verdict potential, there will also be additional pressure on defense costs and efficiency. The initial triage to identify the level of workup justified in a particular case may be even more important in a lung cancer case so defense resources can be allocated where most needed.

"Low dose" defendants will find that the dose defense is also helpful in lung cancer cases, perhaps even more so than it is in mesothelioma cases. In evaluating lung cancer risk, Henderson used an equation based in part on cumulative exposure and an industry-specific factor Douglas Henderson and James Leigh, *Asbestos and Carcinoma of the Lung,* in Asbestos Risk Assessment, Epidemiology and Health Effects 284 (Dodson and Hammar eds., 2011). He used an industry factor of .0001 - .0020 for chrysotile miners and friction product manufacturers and a factor of .003 - .0900 for asbestos cement, textile, and insulation workers. Thus, Henderson calculated that cement, textile, and insulation workers have 30-45 times more exposure than friction product manufacturers. Friction defendants can cite studies that exposures for friction product end users would be a small percentage of the exposures of friction product manufacturers, which in turn is a small percentage of the exposures of insulation workers, such as those in whom Selikoff found a 5-fold increase in lung cancer risk.

Genetic Susceptibility and Markers

Although there is no pathologic "marker" that distinguishes a smoking caused lung cancer from one that is caused by something else, genetic studies have identified certain genetic changes that are common or unusual in smoking-caused lung cancers. For example, G:C to T:A transversions on the p53 tumor suppressor gene are common in smoking-caused lung cancer. In addition, K-ras and an increased methylation rate of p16 are also associated with smoking-caused lung cancer. The presence of these genetic markers may allow an expert to attribute the lung cancer to smoking. On the other hand, chromosomal aberrations involving 16p DNA gain and p53 transition mutations are rare

in smoking-caused lung cancers. EGFR-TK (epidermal growth factor receptor tyrosine kinase) is also rare in smoking-caused lung cancer. *See generally* Subramanian and Govindan, *Lung Cancer in Never Smokers: A Review*, 25 J Clin Oncol, No. 5, 561-70 (Feb 10, 2007). It is important to review medical records for such genetic testing because it may help a medical expert opine about whether a lung cancer is or is not more likely smoking-caused. If genetic testing has not been done, defendants might consider moving to compel it.

Defenses

An important issue in discovery will be whether the plaintiff and plaintiff's experts claim that the plaintiff has asbestosis and pleural changes and that should be a topic of interrogatories and deposition questions. In a mesothelioma case, it is often challenging to support a comparative fault defense because it is often difficult to find something that the plaintiff did and knew or should have known not to do that caused the disease. In a lung cancer case, however, contributory negligence and assumption of risk should be prominent defenses. The first Surgeon General warnings about the hazards of smoking appeared in 1964, so most lung cancer plaintiffs picked up a pack of cigarettes with a health warning tens of thousands of times. In doing so, they voluntarily assumed a known risk. To the extent that the smoking contributed to their disease, it is also causal comparative fault. The fact that they ignored cigarette warnings is also relevant to whether they would have followed different warnings with respect to asbestos-containing products. Plaintiffs typically argue that they were addicted to smoking. Yet, most plaintiffs also testify that they quit for some period of time, which may serve to undercut claims that they were addicted (*e.g.*, Did a relapse caused by addiction actually cause them to resume smoking six months later, or was it a voluntary choice to start again?).

Motion Practice

Although the case volume of lung cancer cases will likely be much higher in the future than mesothelioma cases, most lung cancer cases will not satisfy the criteria for reliable attribution to asbestos exposure. It is imperative that defendants expose the scientific unreliability in this majority of cases and file motions to exclude expert opinions of attribution. Of course, the factual support for these motions will arise in effective depositions of plaintiffs' experts. These depositions should take plaintiffs' experts through the various attribution criteria and show that the expert's opinion is based on criteria and methodology that is not generally accepted.

In addition to motions to exclude expert opinions of attribution, defendants should also continue the success they have had in mesothelioma cases in excluding expert opinions that every asbestos exposure (above background or that are "special" exposures) contribute. Defendants have obtained favorable outcomes with such motions, particularly in low dose chrysotile cases. These motions should be even more compelling in a lung cancer case, particularly cases with smoking history. There is no scientific support for the proposition that each and every low dose chrysotile exposure is a substantial factor in a lung cancer, particularly where there is smoking history.

Experts

Defendants will need an expert pathologist and most of the pathologists they use in mesothelioma cases will also be outstanding in lung cancer cases. Defendants will also need a medical expert in additional to a pathologist to testify about the causation and attribution of lung cancer, particularly in cases with smoking history. Given the importance of asbestosis and potentially pleural plaques, defendants will often also want an expert radiologist or B-reader.

Conclusion

As mesothelioma diagnoses and lawsuits eventually decline, asbestos defendants can expect an increase in lung cancer cases alleging asbestos exposure as a cause. Although there may be overlap in certain issues between mesothelioma and lung cancer cases, the medical issues are very different. Through application of principles discussed in this article, defendants may be able to pare down the number of cases through motion practice and be better able to defend the others. FD