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**RECENT DEVELOPMENTS IN THE
HEALTH EFFECTS OF TOBACCO USE:**

ETS: THE NEXT SMOKING GUN IN ASBESTOS LITIGATION

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I. INTRODUCTION

According to a 1989 Surgeon General report, tobacco smoking was responsible for more than one of every six deaths in the United States. Further, it was estimated that 90% of the lung cancer deaths in males and 80% of the lung cancer deaths in females in 1985 are causally related to tobacco smoking. U.S. Surgeon General, *The Health Consequences of Involuntary Smoking*, 1986. It is estimated that 434,000 deaths per year are attributable to tobacco smoking. Centers for Disease Control (1991a), *Smoking-Attributable Mortality and Years of Potential Life Lost - United States, 1988*.

The average American spends 90 percent of his or her life indoors. Consequently, the air quality of the indoor environment has critical health, economic and legal implications. Kelly S., *Indoor air pollution: An impetus for environmental regulation indoors?*, 6 B.Y.U. J. Pub. L. 295 (1992). In the past five years, numerous studies on the health effects of secondhand or environmental tobacco smoke have appeared in leading medical journals. As recently as October 18, 1997, the British Medical Journal published a compendium of the leading studies on the causal relationship between passive smoking and lung cancer and heart disease.

This article addresses the most recent developments in tobacco litigation and the health effects of secondhand smoking and offers some practical applications for asbestos litigation.

I. RECENT DEVELOPMENTS

A. Flight Attendants Class Action

Broin, Young, et al. v. Phillip Morris, et al - (Fla)

Broin was one of 60,000 flight attendants who sought damages from tobacco companies for ailments allegedly suffered as a result of breathing cigarette smoke in the aircraft passenger cabins during flights.

On October 10, 1997, following the presentation of the plaintiffs' case in chief, the tobacco industry agreed to pay \$300 million to start a secondhand smoke research foundation and \$49 million in court costs and legal fees. The agreement awards no money to the flight attendants but allows them to file individual suits against the cigarette industry later.

Importantly, the defendants agreed to waive the statutes of limitation that could have prevented thousands of flight attendants

whose illnesses dated back to the 1930's from filing individual actions. Compare Judge Newcomer's decision in *Barnes*, below.

H. Nevada Casino Dealers Class Action

Badillo et al v. R.J. Reynolds, et al. (Nev.)

Nine casino dealers have filed a lawsuit against the nation's tobacco companies in the Federal District Court in Reno, Nevada claiming years of exposure to secondhand smoke has impaired their health. The complaint suggests that upwards of 45,000 casino dealers working in Nevada could potentially become part of the suit.

C. Attorneys General State Suits

1. Scheduled to start October 27, 1997, Texas is one of 41 states scheduled for trial to recover costs for the treatment of sick smokers under the state Medicaid program for the poor.
2. During the summer of 1997, Mississippi and Florida negotiated settlements for a combined \$14.7 billion.

D. Barnes v. American Tobacco

Barnes v. American Tobacco, (ED Pa., 96-5903)

Memorandum Opinion by Judge Clarence C. Newcomer, 10/17/97

Judge Newcomer decertified a class of approximately one million or more Pennsylvania residents who were smokers and began smoking before age 19, who were seeking the establishment of a medical monitoring program. Judge Newcomer found that the class action implicated far too many individual issues to proceed on a class wide basis.

In a related decision, Judge Newcomer granted summary judgment based on the statute of limitations as to all but one of the representative plaintiffs of this class. Judge Newcomer determined that a medical monitoring claim accrues when the plaintiff is placed at a "significantly increased risk of contracting a serious latent disease." *Id.* at 29. Since many of these plaintiffs had admittedly been smoking for some time despite their knowledge of the dangers associated with smoking, the court granted the tobacco companies' motions.

E. Koop-Kessler Report on Tobacco Policy

Koop-Kessler Report on Tobacco Policy and Public Health (1997)

Dr. C. Everett Koop and Dr. David A. Kessler were asked by Congress to convene a committee on national tobacco policy in May 1997. They authored a report that addressed significant areas of public concern relating to smoking. Major recommendations included:

- FDA should continue to phase out nicotine and to remove ingredients that create dependence on cigarettes and other tobacco products, including tobacco, pipesmoking, cigars, etc.
- Smoking should be banned in all work sites and places of public assembly (e.g. sports stadiums, schools, campuses, public transportation)

The Koop-Kessler Report specifically noted that "scientific evidence now indicates that nonsmokers become seriously ill or die because of exposure" to environmental tobacco smoke.

II. THE HAZARDS OF ENVIRONMENTAL TOBACCO SMOKE

A. What is Environmental Tobacco Smoke (ETS)?

1. Environmental tobacco smoke (ETS), otherwise known as secondhand smoke, "consists of exhaled mainstream smoke (MS), sidestream smoke (SS) emitted from the smoldering tobacco between puffs, contaminants emitted into the air during the puff, and contaminants that diffuse through the cigarette paper and mouth end between puffs." *Respiratory Health Effects of Passive Smoking and Other Disorders - The Report of the U.S. Environmental Protection Agency (1993)*, at p. 3-1.
2. ETS contains a vapor phase and a particulate phase. Sidestream smoke accounts for 80% of ETS. Trichopoulos D, *Risk of lung cancer and passive smoking*, Important Adv Oncol 1995: 77-84. Sidestream smoke contains essentially all of the same carcinogens and toxic agents that have been identified repeatedly in the past with mainstream smoke, but at greater levels. *Environmental tobacco smoke*, Cancer Facts, National Cancer Institute, National Institute of Health, 2/8/95, at 1.

3. Identified in this sidestream smoke are more than 4,000 compounds of which more than 60 have been identified as carcinogens, tumor initiators and tumor promoters including the following major groups:
- *Polyaromatic hydrocarbons* - 11 various compounds with known animal carcinogenicity, including benzo(a)pyrene, a "probable" human carcinogen;
 - *Aza-arenes* - four known animal carcinogens;
 - *N-Nitrosamines* - nine known animal carcinogens;
 - *Aromatic amines* - three known carcinogens, including 2-Naphthylamine and 4-Aminobiphenyl, both known human carcinogens;
 - *Aldehydes* - three known carcinogens including formaldehyde, a suspected human carcinogen;
 - *Miscellaneous organic compounds* - six carcinogens, including benzene and vinyl chloride, both known human carcinogens;
 - *Inorganic compounds* - seven carcinogens, including arsenic, chromium and polonium-210, all known or suspected human carcinogens;
 - *Miscellaneous compounds* - nicotine, HCN, zinc, cadmium and carbon monoxide.

From U.S. Surgeon General, *Reducing the Health Consequences of Smoking: 25 Years of Progress*, 1989; Wilner N, Feingold A, M.D. *Asbestos Medicine on Trial - A Medical/Legal Outline* 1996; 1: 299.

B. Hazard Identification

1. In 1986, the United States Surgeon General issued a report on the hazards of exposure to ETS. U.S. Surgeon General, *The Health Consequences of Involuntary Smoking*, 1986. The Surgeon General concluded that "involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers." *Id.*

2. In 1993, the US Environmental Protection Agency released a report that concluded:

- ETS is causally associated with lung cancer in nonsmoking adults and should be classified as a group A, or known human carcinogen, with approximately 3000 excess deaths yearly; approximately thirty percent of all lung cancers caused by factors other than smoking are attributable to ETS
- ETS produces an increased risk of development of acute lower respiratory tract irritation, asthma, and acute lower respiratory tract infections in children exposed in the home
- ETS is associated with an increased risk of SIDS or sudden infant death syndrome

Respiratory Health Effects of Passive Smoking and Other Disorders – The Report of the U.S. Environmental Protection Agency (1993); Wilner, Feingold, supra. at p. 304-05.

C. Diseases Associated With ETS

In 1972, when the Surgeon General first raised the issue of passive smoking, only a handful of studies were available. Since that time, numerous studies have illustrated conclusively that involuntary inhalation of cigarette smoke by nonsmokers causes lung cancer. Further, recent studies have suggested that ETS plays a role in a broad range of respiratory tract illnesses, including asthma, decreased lung function, coronary heart disease, COPD, vascular problems and other malignant neoplasms.

1. Lung Cancer

- a) Hackshaw K, Law MR, Wald NJ *The accumulated evidence on lung cancer and environmental tobacco smoke*, BMJ 1997; 315(7114).

On October 18, 1997, the British Medical Journal published a series of articles and editorials concerning passive smoking. One of the articles provided an analysis of 37 published epidemiological studies of the risk of lung cancer (4,626 cases) in non-smokers.

This study did not utilize workplace exposures due to the difficulty in measuring such exposure. The risk of lung cancer in non-smokers exposed to ETS was estimated by "extrapolating from the risk in smokers, using the urine or saliva concentrations of cotinine and nicotine (both sufficiently tobacco specific) in each." *Id.*

Twenty-nine of these studies were in peer reviewed journals, four in books, two in peer reviewed doctoral theses and three in published proceedings of scientific conferences. One study was an official report from a scientific organization. There were five cohort and 34 case control studies. *Id.* at p.2.

Result: The excess risk of lung cancer was 24% in non-smokers who lived with a smoker. *Id.*

See Table 1 at the conclusion of this article which illustrates each of the prior studies and relative risk. See Table 2 for a review by sex, geographical region, year of publication, and study design for relative risk.

This study also evaluated the dose-response relation between the number of cigarettes smoked by the spouse, the number of years living with the spouse and the risk of lung cancer. **This study showed a significant dose-response relationship whereby the risk of lung cancer increased with a concomitant increase in the number of cigarettes smoked per day by the spouse and in the number of years living with a spouse who smokes. *Id.*** The authors suggested that this study is consistent with the view that **exposure to environmental tobacco smoke is the equivalent of low dose smoking. *Id.***

Finally, the study also examined the relative risk according to the number of years the spouse lived with a smoker. This estimate showed a significant increase in risk with increasing duration of exposure. **This risk increased by 11% for every 10 years of exposure. *Id.***

- b) Morris P, *Lifetime excess risk of death from lung cancer for a U.S. female never-smoker exposed to environmental tobacco smoke*, Env. Res. 1995; 68 3-9.

Although the previous compendium evaluated exposure to ETS in the home, this study involved risk assessment to estimate the lifetime excess risk of death from lung cancer for a U.S. female never smoker exposed to ETS in different settings.

Since this study was conducted at least two years prior to the Hackshaw study, relative risks for social and occupational exposures were calculated by using a relative risk of 1.185 for home exposure. The Hackshaw study had calculated this risk to be 1.24.

Morris was evaluating the relative risks for ETS exposure in places other than the home to determine whether ETS should be regulated in public places and work sites. His study, which is presented in Table 3 at the conclusion of this article, summarizes the following risks:

- *Home Exposure*: 6.5 excess death per 10,000 never smokers;
- *Social Exposure*: 1.4 – 3.6 excess deaths per 10,000 never smokers;
- *Workplace Exposure*: 1.4 – 9.8 excess deaths per 10,000 never smokers;
- *Office Exposure*: 1.5 excess deaths per 10,000 never smokers;
- *Restaurant Exposure*: 3.0 excess deaths per 10,000 never smokers;
- *Bar Exposure*: 9.0 excess deaths per 10,000 never smokers.

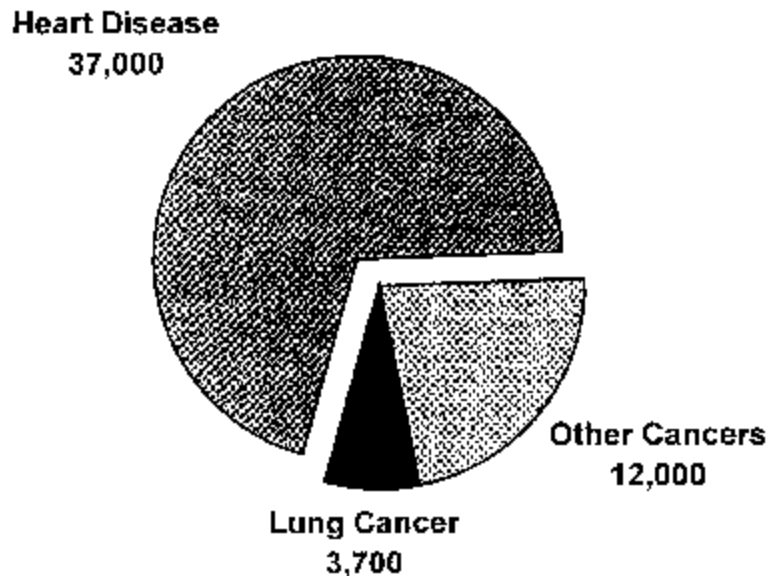
Id. at p.6.

2. Cardiovascular Disease

It is well established that active smoking is a cause of heart disease. The chemicals in mainstream smoke that are instrumental in causing heart disease are carbon monoxide, nicotine and polyaromatic hydrocarbons. Wells A, *Passive smoking as a cause of heart disease*, JACC, 1994, Aug; 24(2): 546-54. Logic suggests that environmental tobacco smoke also results in heart disease, but perhaps at a lesser level. With lung cancer, the potentially fatal health effects usually require long-term exposure. With heart disease, the "potentially fatal effects are not only long-term and chronic but short-term and acute as well." *Id.* at 546.

As evidenced by the following diagram, heart disease is the most prevalent outcome from exposure to environmental tobacco smoke.

Deaths From Passive Smoking Total Deaths: 53,000



Pie chart of US deaths from environmental tobacco smoke. The majority of annual deaths are attributable to heart disease. Modified from Wells A *An estimate of adult mortality in the United States from passive smoking: A response to criticism*. *Environ Int* 1990; 16:187-193.

The most recent report from the California Environmental Protection Agency suggests that the above figures have grown dramatically:

- a) Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, *Health Effects of Exposure to Environmental Tobacco Smoke (Final Draft)*, Sacramento, CA (1997).

This report provides the most exhaustive survey of passive smoking and heart disease. The report lists both "heart disease mortality" and "acute and chronic coronary heart disease morbidity" as "effects causally associated with ETS exposure".
Id.

The report provides estimates of the total annual morbidity and mortality resulting from ETS exposure with the following estimates:

- *Sudden Infant Death Syndrome (SIDS):* 1900 to 2700 deaths per year;
- *Low-Birth Weight Babies:* 9700 to 18,600 cases per year;
- *Induced Asthma in Children:* 8000 to 26,000 new cases per year;
- *Bronchitis or Pneumonia:* 150,000 to 300,000 cases in children up to age 18 months per year. Of these, 7500 to 15,000 are hospitalized and 136 to 212 die;
- *Lung Cancer:* 3000 death per year;
- *Ischemic Heart Disease:* 35,000 to 62,000 deaths per year;

Id.

- b) Law MR, Morris JK, Wald NJ, *Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence*, BMJ, 10/18/97; 315(7114).

Three researchers from St. Bartholomew's and the Royal London School of Medicine evaluated prior epidemiological studies of exposure to environmental tobacco smoke and ischaemic heart disease. The studies utilized spouse's smoking as an objective measure of exposure to ETS. The writers also analyzed the dose-response relationship between smoking and heart disease.

Results: The relative risk at age 65 of ischaemic heart disease associated with ETS was 1.30. At the same age, the estimated relative risk for smoking one cigarette per day was similar at 1.39, while for smoking 20 cigarettes per day, the estimated relative risk was 1.78.

The writers were puzzled by this data since exposure to environmental tobacco smoke is only about 1% that of smoking, yet comparatively, the risk of heart disease from ETS is nearly half that of smoking. The researchers were not able to provide an alternative explanation for this large increased risk of heart disease from ETS except to note **that it is of similar magnitude to the effects of exposure to environmental tobacco smoke on lung cancer.**
Id.

- c) Kawachi I. et al, *A prospective study of passive smoking and coronary heart disease*, *Circulation* 5/20/97; 95(10): 2374-79.

Several Harvard researchers reported the findings of a 10-year study, which tracked more than 32,000 healthy women who never smoked ranging in ages from 36-61 and were free of diagnosed coronary heart disease, stroke and cancer. This study began in 1976 when 121,700 female nurses filled out detailed surveys every two years about their health and habits.

To measure the effects of passive smoking, the researchers asked the women in 1982 about their exposure, and then monitored new cases of heart disease for the next 10 years. The 32,000 plus women who became study participants were identified as non-smokers who reported being exposed to smoke regularly at home or at work.

Results: Compared with women not exposed to passive smoking, the relative risks of total coronary heart disease, when adjusted for a broad range of cardiovascular risk factors, were 1.51 among those reporting occasional exposure and 1.91 among women reporting regular exposure to passive smoking at home or work.

- d) Wells AJ, *Passive smoking as a cause of heart disease* J Am Coll Cardiol, 1994, Aug; 24(2):546-54.

In a major review article, Wells concluded that the coronary death rate in the United States among nonsmokers was increased 20% to 70% by passive smoking.

The involuntary inhalation of cigarette smoke has the same adverse effects on the cardiovascular system including atherosclerosis, coronary vasoconstriction and intimal damage. Zhu B, Pannley W, *Hemodynamic and vascular effects of active and passive smoking*, Am Heart J, 1995; 130: 1270-75.

One study suggested that passive smoking may account for up to 20,000 deaths annually from atherosclerotic heart disease in non-smokers in the United States. Celermajer D, et al, *Passive smoking and impaired endothelium-dependent arterial dilatation in healthy young adults*, N Engl J Med, 1/18/96; 334(3):150-155.

3. Respiratory Problems

Tobacco smoke irritates the airways and has been established as a risk factor for obstructive respiratory conditions. Dayal H, et al, *Passive smoking in obstructive*

respiratory diseases in an industrialized urban population, *Envir Res* 1994; 65: 161-71. Some of these conditions are indicated below.

- a) Halken, et al, *Passive smoking as a risk factor for development of obstructive respiratory disease and allergic sensitization*, *Allergy*, 1995; 50: 97-105.

This review article analyzed several recent studies concerning the correlation between passive smoking and respiratory diseases.

Asthma: Most recent studies have shown a correlation between passive smoking and wheezy bronchitis and asthma in children. It was found that maternal smoking significantly increased the prevalence of asthma.

Respiratory Tract Infections: Previous studies have found a consistent effect of parental smoking on upper and lower respiratory tract infections in children, including pneumonia and bronchitis.

Allergies: Several studies of maternal smoking both during and after pregnancy found a positive correlation between the effect passive smoke has on the immune system in children.

Because there are numerous other genetic and environmental factors that cause these respiratory problems in adults, these studies were not conclusive as to the effects of passive smoking on adults since no studies have evaluated these factors properly.

- b) Dayal H, et al, *Passive smoking in obstructive respiratory diseases in an industrialized urban population* *Envir Research* 1994; 65: 161-71.

Researchers examined nine Philadelphia neighborhoods from 1985-86, evaluating data from 4200 individuals. The risk of obstructive respiratory disease associated with tobacco smoke in indoor air, independent of active smoking, ambient air pollution and some other sources of air pollution was examined.

Results: The findings of this study were consistent with prior studies which found a relative risk of 1.3 for chronic lower respiratory infections related to ETS, and a relative risk of 1.2 for asthma and wheezing for children exposed to ETS at home.

- c) *Respiratory Health Effects of Passive Smoking and Other Disorders - The Report of the U.S. Environmental Protection Agency* (1993), at p. 7-1, et seq.

It is well established that "active smoking causes significant reductions in lung functions and significant increases in prevalence of respiratory symptoms." *Id.* at 7-69. Recent studies have suggested that adult non-smokers exposed to ETS may have small reductions in lung function, and may increase the frequency of respiratory symptoms in adults by as much as 30% to 60% higher in the ETS-exposed non-smoker than an unexposed non-smoker. *Id.*

4. Other Cancers

Cancers of the kidney, bladder, esophagus, digestive tract and oral cavity have been strongly linked to cigarette smoking. Although studies evaluating the risks of other cancers from exposure to passive smoking have not been conclusive, given the inability to control for environmental and other confounding factors, an association between non-respiratory tract cancers and spouse's smoking was studied by Hirayama in *Cancer Mortality in Non-Smoking Women With Smoking Husbands Based on a Large-Scale Cohort Study in Japan*, *Prev Med* 1984;13: 680-690.

As the studies increasingly implicate environmental tobacco smoke in pulmonary and cardiovascular deaths, it logically follows that passive smoking will be causally linked to various neoplasms associated with active smoking. Below is a recent table from Peto in *Lancet* which outlines the diseases and percent mortality.

**Table: Mortality From Selected Diseases
Smoking Attributed and Total (From Peto 1992)**

Cause of Death	Smoking Attributed Deaths	Total Deaths Per 100,000	Percent (%) Smoking Attributed
Lung Cancer	203	217	93.5
Upper Aero-Digestive Cancer	47	69	68.1
Other Cancer	64	362	17.7
COPD	71	87	81.6
Other Respiratory	14	45	31.1
Cardiovascular Disease	297	876	33.9
Other Medical	78	228	34.3
Total All Fatal Conditions*	774*	2,216*	34.9*

From Peto, R, Lopez, AD, Boreham, J., Thun, M, Heath, C., *Mortality from Tobacco in Developed Countries: Indirect Estimation From National Vital Statistics*, Lancet, 1992: 339: 1268-78. *(Edited)

D. Practical Applications in Asbestos Litigation

The most recent studies conclusively prove the importance of obtaining as much information about a particular asbestos plaintiff as possible prior to trial. This information contributes to ascertaining alternative, establishing mitigation, and apportionment.

1. Written Discovery

Written discovery in many jurisdictions has been standardized by the courts and parties, and does not explore ETS exposure. Additional questions concerning the plaintiff's social and employment history is essential to establish the association with prolonged environmental tobacco smoke. For example, new areas of inquiry should include:

- whether prior employers provided a smoke free environment for the plaintiff;
- whether plaintiff worked in any environment where smoking was prevalent (e.g. bartenders, waitresses, casino dealers, flight attendants, police officers);
- whether plaintiff's family history included parents, spouse, children, or extended family who were smokers;
- whether plaintiff's neighbors, friends or acquaintances with whom plaintiff spent some time were smokers.

2. Oral Discovery

Obviously, many of the areas of inquiry requested from the plaintiff in written discovery should be the foundation for questioning in the discovery deposition of the plaintiff, the plaintiff's spouse, coworkers of the plaintiff and the plaintiff's treating physician. Again, this information will be useful for providing alternative causation in the non-smoking plaintiff in the traditional lung cancer case.

3. Medical Experts

Your medical expert should be completely aware of the current literature and epidemiological studies concerning passive smoking. Attached to this article is a bibliography of some of the most recent articles for your expert to review. You must become familiar with these articles to effectively cross-examine the plaintiff's expert, as well as to guide your expert's testimony.

4. Trial

One drawback in emphasizing ETS at a trial is the difficulty of establishing plaintiff's fault. For mainstream or direct smoking, the issue of fault is simple; for ETS, the plaintiff is the victim. Consider these issues in the preparation of your case. Not to be discussed in this article is the joinder of cigarette companies in asbestos litigation. See prior DRI articles for exploration of this issue.

IV. LUNG CANCER AND THE CIGARETTE SMOKER

Cigarettes cause lung cancer of all cell types, in all lung zones. Almost all lung cancer patients are either smokers or former smokers. The Surgeon General estimated that 85% "of all lung cancers are directly attributable to cigarette smoking." U.S. Surgeon General, *The Health Consequences of Smoking*, 1982.

Hammond's prescient epidemiological study in 1958 of 187,783 men over a 44 month period compares favorably with present research concerning the relative risk of lung cancer and cigarette smoking. This relative risk is set forth in the following table:

**Table: Risk of Lung Cancer From Cigarette Smoking
(From Hammond)**

Smoking Category	Lung Cancer Per 100,000 Man Years	Relative Risks
.5 to 1 packs/day	107.8	8.4
1 to 2 packs/day	229.2	17.9
> 2 packs/day	264.2	20.6

From Hammond EC, Horn D, *Smoking and Death Rates Report on Forty-Four Months of Follow-Up of 187,788 Men*, JAMA 1958; 166(10):1159-1172. (Edited)

The debate in medical as well as legal arenas will continue to rage concerning the necessity for asbestosis as an obligatory precursor to asbestos-related lung cancer. Illerdal, G, Henderson, D, *Asbestos, asbestosis, pleural plaques and lung cancer*, Scand J Work Environ Health 1997; 23:93-103. However, given the hypothesis that almost all lung cancer patients are either present or former smokers or exposed to environmental tobacco smoke of some quantity, experts cannot ignore the probability that the majority of lung cancers are primarily caused by cigarette smoke. "Occupational risks cause a small percentage of lung cancers." Wilner, Feingold, *supra*, at 289.

V. CONCLUSION

Plaintiff's class action lawyers have recently drawn the nation's attention forcibly to the secondhand smoking evidence. Attorneys specializing in asbestos defense can take advantage of this raised national awareness by emphasizing this somewhat controversial evidence.

Table 1 - Epidemiological studies of the risk of lung cancer in lifelong non-smokers whose spouses smoked relative to the risk in those whose spouses did not smoke							
Study	Year, country	Women			Men		
		Lung cancer cases	Controls	Relative risk (95% confidence interval)	Lung cancer cases	Controls	Relative risk (95% confidence interval)
Case-control studies							
Chan et al	1982, Hong Kong	84	139	0.75 (0.43 to 1.30)			
Correa et al	1983, USA	22	133	2.07 (0.81 to 5.25)	8	180	1.97 (0.38 to 0.32)
Trichopoulos et al	1983, Greece	62	190	2.13 (1.19 to 3.83)			
Buffler et al	1984, USA	41	196	0.8 (0.34 to 1.90)	11	90	0.51 (0.14 to 1.79)
Kabat et al	1984, USA	24	25	0.79 (0.25 to 2.45)	12	12	1 (0.10 to 5.07)
Lam	1985, Hong Kong	60	144	2.01 (1.09 to 3.72)			
Garfinkel et al	1985, USA	134	402	1.23 (0.81 to 1.87)			
Wu et al	1985, USA	29	62	1.2 (0.50 to 3.30)			
Akiha et al	1986, Japan	94	270	1.52 (0.87 to 2.63)	19	110	2.10 (0.51 to 8.61)
Lee et al	1986, UK	32	66	1.03 (0.41 to 2.55)	15	30	1.31 (0.38 to 4.52)
Koo et al	1987, Hong Kong	86	136	1.55 (0.90 to 2.67)			
Pershagen et al	1987, Sweden	70	294	1.03 (0.61 to 1.74)			
Humble et al	1987, USA	20	162	2.34 (0.81 to 6.75)			
Lam et al	1987, Hong Kong	199	335	1.65 (1.16 to 2.35)			

Table 1 - Epidemiological studies of the risk of lung cancer in lifelong non-smokers whose spouses smoked relative to the risk in those whose spouses did not smoke

Study	Year, country	Women			Men		
		Lung cancer cases	Controls	Relative risk (95% confidence interval)	Lung cancer cases	Controls	Relative risk (95% confidence interval)
Gao et al	1987, China	246	375	1.19 (0.82 to 1.73)			
Brownson et al	1987, US	19	47	1.52 (0.39 to 5.96)			
Ceng et al	1988, China	54	93	2.16 (1.08 to 4.29)			
Shimizu et al	1988, Japan	90	163	1.08 (0.64 to 1.82)			
Inoue et al	1988, Japan	22	47	2.55 (0.74 to 8.78)			
Kalandidi et al	1990, Greece	90	116	1.62 (0.90 to 2.91)			
Sobue	1990, Japan	144	731	1.06 (0.74 to 1.52)			
Wu-Williams et al	1990, China	417	602	0.79 (0.62 to 1.02)			
Liu et al	1991, China	54	202	0.74 (0.32 to 1.69)			
Jockel	1991, Germany	23	45	2.27 (0.75 to 6.82)	9	70	2.68 (0.58 to 12.36)
Brownson et al	1992, US	431	1166	0.97 (0.78 to 1.21)			
Stockwell et al	1992, US	210	301	1.6 (0.80 to 3.00)			

Table 1 - Epidemiological studies of the risk of lung cancer in lifelong non-smokers whose spouses smoked relative to the risk in those whose spouses did not smoke							
Study	Year, country	Women			Men		
		Lung cancer cases	Controls	Relative risk (95% confidence interval)	Lung cancer cases	Controls	Relative risk (95% confidence interval)
Du et al	1993, China	75	128	1.19 (0.66 to 2.13)			
Liu et al	1993, China	38	69	1.66 (0.73 to 3.78)			
Fonham et al	1994, USA	651	1253	1.26 (1.04 to 1.54)			
Kabat et al	1995, USA	67	173	1.1 (0.62 to 1.96)	39	98	1.63 (0.69 to 3.85)
Zaridze et al	1995, Russia	162	285	1.66 (1.12 to 2.45)			
Sun et al	1996, China	230	230	1.16 (0.80 to 1.69)			
Wang et al	1996, China	135	135	1.11 (0.67 to 1.84)			
Cohort studies							
Garfinkel	1981, USA	153	176,586	1.18 (0.90 to 1.54)			
Hirayama	1984, Japan	200	91,340	1.45 (1.02 to 2.08)	64	20,225	2.25 (1.06 to 4.76)
Butler	1988, USA	6	9,199	2.02 (0.48 to 8.56)			
Cardenas et al	1997, USA	150	192,084	1.2 (0.80 to 1.60)	97	96,445	1.00 (0.60 to 1.80)
All studies (37 studies of women, 9 studies of men)*							
	1981-97	4,626	477,924	1.24 (1.13 to 1.36) (P<0.001)	274	117,260	1.34 (0.97 to 1.84) (P<0.07)
*In addition, there were two studies which gave results only for men and women combined: Hole et al (7 lung cancer cases) relative risk 2.14 (95% confidence interval 0.45 to 12.83); Janerich et al (188 lung cancer cases), relative risk 0.75 (0.48 to 1.18).							

Table 1 is from Hackshaw, Law, Wald, *The Accumulated Evidence on Lung Cancer and Environmental Tobacco Smoke*, *BMJ*, Vol. 315, No. 7114 (10/18/97)

Table 2 - Relative risk of lung cancer and exposure to environmental tobacco smoke in the 39 epidemiological studies according to sex, geographical region, year of publication, and study design

	No of studies	No of lung cancer cases	Pooled relative risk (95% confidence interval)
By sex:			
Women	37	4,626	1.24 (1.13 to 1.36)
Men	9	274	1.34 (0.97 to 1.84)
Both*	39	5,095	1.23 (1.13 to 1.34)
By geographical region†:			
USA	14	1,959	1.17 (1.05 to 1.31)
Europe‡	6	439	1.53 (1.21 to 1.94)
Japan	5	550	1.28 (1.04 to 1.57)
China and Hong Kong**	12	1,678	1.22 (0.99 to 1.50)
By year of publication†:			
1981-5	10	809	1.29 (1.06 to 1.58)
1986-90	15	1,591	1.28 (1.07 to 1.54)
1991-7	12	2,226	1.19 (1.06 to 1.33)
By study design†:			
Case-control	33	4,115	1.24 (1.12 to 1.38)
Cohort	4	511	1.27 (1.05 to 1.53)

*Including the two studies of men and women combined.

†Studies of women only.

‡United Kingdom, Sweden, Greece, Russia.

**Excluding Wu-Williams et al. the pooled estimate was 1.30 (95% confidence interval 1.09 to 1.56)

Table 2 is from Hackshaw, Law, Wald, *The Accumulated Evidence on Lung Cancer and Environmental Tobacco Smoke*. *BMJ*, Vol. 315, No. 7114 (10/18/97)

TABLE 3
Lifetime Excess Risk of Death from Lung Cancer for a U.S.
Female Never-Smoker Exposed to Different Sources of ETS

Exposure	Relative risk ^a	95% CI ^b	Excess risk ^c
	1.185	(1.014, 1.386)	6.5×10^{-4}
Social ^d			
0.20	1.037	(1.003, 1.077)	1.4×10^{-4}
0.55	1.102	(1.008, 1.212)	3.6×10^{-4}
Workplace ^e			
0.05	1.009	(1.001, 1.019)	9.8×10^{-6}
0.30	1.056	(1.004, 1.116)	6.1×10^{-5}
Workplace ^f			
0.45	1.083	(1.006, 1.174)	9.0×10^{-5}
0.70	1.130	(1.010, 1.270)	1.4×10^{-4}
Offices ^g			
0.73	1.135	(1.010, 1.282)	1.5×10^{-4}
Restaurants ^h			
1.50	1.278	(1.021, 1.579)	3.0×10^{-4}
Bars ^h			
4.46	1.825	(1.062, 2.722)	9.0×10^{-4}

^a Estimated relative risk for those exposed to ETS.

^b 95% confidence interval.

^c Excess risk for 35 years (ages 45-79) of home or social exposure and 21 years (ages 45-65) of workplace exposure.

^d Social exposure level divided by domestic exposure level. Based on cotinine studies.

^e Workplace exposure level minus social exposure level divided by domestic exposure level. Based on cotinine studies.

^f Workplace exposure level divided by domestic exposure level. Based on cotinine studies.

^g Workplace exposure level divided by domestic exposure level. Based on air measurement studies of nicotine and particulates.

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