This file contains a number of changes from the original version. See the last page of the pdf for details.

Fiber Types, Asbestos Potency, and Environmental Causation

A Peer Review of Published Work and Legal and Regulatory Scientific Testimony

DAVID EGILMAN, MD, MPH

Scientific evidence and analysis offered in litigation and public policy testimony have an important role in occupational and environmental health, but are not subject to peer review. Critique and commentary, attempts at reproduction of results, and review of data offered in such testimony is essential. Peer review of such testimony should become part of the domain of medical and scientific journals. This paper is an effort to peer review the use of certain scientific methods in tort litigation and in testimony before regulatory agencies. In this issue of *IJOEH*, Azuma et al. show that background asbestos exposures can be considered to have caused mesothelioma. In contrast, epidemiologic studies and testimony by Teta et al. and Price and Ware, and pathologic studies and testimony by Roggli and others, claim that background exposures are benign. These are fatally flawed because of methodological and analytic errors. *Key words*: asbestos; litigation; peer review; chrysotile; public policy; mesothelioma

INT J OCCUP ENVIRON HEALTH 2009;15:202–228

Several recent episodes of the publication of works based on partial or fabricated data have again revealed the weakness of the peer review process. Dr. Scott Rubin fabricated data that appeared in at least 21 published peer-reviewed papers.[1,2] Jonathan Leo exposed the fact that in an article published in JAMA, authors misrepresented their consulting arrangements with Forest laboratories and concluded that Forest's drug Lexapro was better than placebo, but omitted data from the same study that showed that Lexapro is no better than counseling.[1,3] In response, the Editor of JAMA called Leo a "nobody and a nothing," tried to intimidate the Dean of his medical school, and banned him for life from publishing anything in JAMA.[1] JAMA then let the perpetrators of the misrepresentation explain away their misconduct in a letter to the editor and denied they had maligned Leo.[4,5]

Dr. Egilman is Editor-in-Chief of the *International Journal of Occupational and Environmental Health* and Associate Clinical Professor in the Department of Community Medicine at Brown University. Address correspondence to the author at: 8 N. Main St., Suite 404, Attleboro, MA 02703; email: <degilman@egilman.com>.

Disclosures: The authors has testified in asbestos litigation at the request of asbestos product manufacturers and injured workers.

These incidents remind us that the peer review process does not end with publication. This is true not only for published papers, but also for scientific argument and evidence presented as testimony offered for purposes of public policy-making and litigation. Azuma et al.'s paper in this issue, as well as letters from Hessell and Welch and colleagues, have motivated this commentary, which reviews the presentation of epidemiology- and pathology- based testimony in asbestos litigation and regulation.[6–8] The comments are designed to address general issues, but of necessity are comments on statements and/or publications of particular individuals. This commentary was reviewed by four experts, two of whom do not participate in U.S. asbestos litigation.

In this issue, Azuma et al. use real, although limited, exposure data to correlate environmental "background" asbestos exposures with mesothelioma incidence in Japan. "Background" has no universal definition.* Azuma et al. correlated mesothelioma cases with environmental exposure data and the weighted average number of asbestos ferruginous bodies detected in the lungs of the people with no identifiable point source of exposure either occupational, para-occupational or known environmental. Their data roughly confirm the U.S. Environmental Protection Agency's (EPA's) dose-response equation, which is consistent with a no threshold-effect level for asbestos-induced mesothelioma. Azuma et al. show that many, if not most, "background" mesothelioma cases are caused by ambient levels of asbestos which are attributable to asbestos released during building construction and from automobile and truck brakes, among other sources. Sprayed chrysotile and amphibole asbestos was used in the United States as well as Japan and other countries.

In addition to the Azuma paper, there is significant evidence that asbestos causes most mesotheliomas. Mark

*It is important to distinguish between occupational exposures (direct and bystander), non-occupational but clearly above-background exposures (e.g., neighborhood and residential exposures as well as "handyman" and "shade tree" mechanic type of exposures, both direct and indirect) and "environmental" exposures. "Background" exposures, as I use the term, refers to exposures with no identifiable point source that would elevate airborne respirable asbestos fiber concentrations in excess of those recorded for the environment at large. Azuma et al. refer to these exposures as "environmental."

and Yokoi reviewed all autopsies at Massachusetts General Hospital from 1896 onward, and failed to find any mesothelioma case before 1940.[9] They concluded that "the background level of diffuse malignant mesothelioma in Europe and in the United States prior to 1930 was extremely low," and that, "current cases in Boston are not attributable to any significant background level [non-asbestos cause] of the disease." In addition, Camus et al. reported seven "environmental" mesothelioma cases in women who lived near Canadian asbestos mines.[10] Camus et al. concluded that the EPA risk formula overestimated the risk of asbestos lung cancer 10-fold. They reported, but did not analyze, the mesothelioma risk. Unfortunately, Camus et al. relied on particle counting techniques that were inversely related to actual asbestos fiber counts.[11] (The higher the particle count, the lower the exposure.) In contrast, Swedish researchers who relied on fiber counts and controlled for smoking found that "low exposure" (10 fiber-years) relative risks ranged from 1.5 to 4.5, and argued the EPA model underestimated the risk at 1.10.[12] Gustavsson et al. found a non-linear dose–response relationship indicating that per-fiber risks were higher at low exposures than at high exposures. Pan et al. found a relationship between distance from natural outcroppings of chrysotile (occasionally containing tremolite) in California and concluded that the findings supported "the hypothesis that residential proximity to naturally occurring asbestos [NOA] is significantly associated with increased risk of mesothelioma mortality in California."[13]

Despite this rather consistent evidence of real risk of mesothelioma from "background exposures," some industry consultants have assumed in testimony and publication that background exposures are benign. In this commentary, I review these and related assertions on chrysotile potency and lung fiber counting, examining how they have been put to use in litigation and public policy hearings.

SEER DATA CANNOT BE USED TO ESTABLISH A THRESHOLD FOR ASBESTOS INDUCTION OF MEOTHELIOMA

Recent papers by Teta et al. and Price and Ware claim to establish a "safe threshold" below which asbestos does not cause mesothelioma.[14–16] These authors have attempted to use the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) data to estimate the "background" rate of mesothelioma in human populations.[14,15] They define "background" cases as mesotheliomas that occur in individuals who have no history of exposure to asbestos. From a scientific perspective, this approach is problematic since it is based on the unreferenced assumption and assertion that certain cohorts were never exposed to sufficient amounts of asbestos to develop asbestos-caused mesotheliomas, based on the false premise that there were constant rates of mesothelioma over time. They base this assertion on mesothelioma rates—not exposure data, interviews, medical record reviews or a search of medical literature.

In fact, scientists have published contrary information for more than a century and as recently as 2008.[17–22] The Swedish Family-Cancer Database is the largest cancer data base in the world that links job and other factors and cancer incidence. Using this data, Hemminki and Li reported that a comparatively "low [mesothelioma] risk among farmers [who have likely occupational exposures] suggests that the population at large is at a risk of mesothelioma from undefined sources in urban areas." They concluded that "Background exposures do cause mesothelioma and epidemiologic data on excess risk should use the lowest rates for the least exposed as controls. Occupational and para-occupation exposures are added to 'background' rates which have their own real risk."

The UK Health and Safety Executive (HSE) has also agreed that "background" exposures cause mesothelioma in adopting the position that:

> A PMR of 100 does not represent the 'background' risk of mesothelioma (the level that would be expected in the absence of asbestos exposure), A hypothetical group of men with zero exposure to asbestos would record" PMR of approximately 6. . . . An occupational group with a PMR greater than 100 indicates that the level of mesothelioma mortality is higher than average for all occupations.[23]

Disregarding this evidence, Teta et al. review SEER data and make the circular argument that mesotheliomas that occur in this cohort are, by definition, not caused by asbestos because the subjects were by definition not exposed, and therefore all cases are unrelated to asbestos.[15] But if the mesothelioma cases were not exposed to asbestos why look at any death data? Everyone agrees that absent exposure, asbestos is not a cause of mesothelioma. Teta et al. attempt to use mesothelioma rates to "prove" there were no exposures. SEER data cannot answer this question; exposure information can only come from patient histories and/or pathologic studies.

These papers are an example of using the wrong tool (epidemiology) and the wrong data (SEER) set to obtain a desired answer to a question.[24] Since all citizens in developed countries have lung asbestos burdens, there is no unexposed control group. There are many case reports of patients who developed mesothelioma after short, low-dose exposure. Most experts believe asbestos caused these cases.[18,25–38] Epidemiology based on the SEER data cannot answer the question about the effects of low-dose exposure to asbestos because it includes no exposure data, and because the pathologic diagnosis of mesothelioma can be confused with other cancers (such as lung or ovarian), has changed over time, and can be

TABLE 1 Mesothelioma Cases in Women Related to Domestic/Residential Exposure to Asbestos from Virginia Shipyards

	Name	DOB	DOD	Age at Death	Occupationally Exposed Family Member	Exposure Site	Occupation of Exposed Family Member
1.	A., Laura M.	06/19/1921	08/13/1998	77	Spouse	Newport News Shipyard	Pipefitter
2.	B., Bernice	12/28/1935	12/17/1989	54	Father	Newport News Shipyard	Joiner
3.	B., Dorothy	10/23/1919	09/05/1993	73	Spouse	Newport News Shipyard	Welder
4.	B., Dorothy W.	09/14/1924	02/19/2005	79	Spouse	Newport News Shipyard	Fitter/Machinist
5.	B., Juanita J.	05/02/1921	10/02/2006	85	Spouse	Newport News Shipyard	Machinist
6.	B., Marjorie S.	09/05/1918	07/16/1996	78	Spouse	Norfolk Naval Shipyard, various contractors in NC	Pipefitter, Carpenter
7.	B., Mary Louis	03/17/1922	02/07/2001	89	Spouse	Norfolk & Portsmouth Beltline, Portsmouth, VA	Hostler, Fireman, Engineer
8.	B., Sarah R.	08/21/1926	10/27/1992	66	Spouse	CSX Transportation, Inc., Clarksburg, WV	Brakeman
9.	B., Stachi B.	08/24/1915	12/24/1999	84	Spouse	Philadelphia Naval Shipyard, Norfolk Naval Shipyard	Pipecoverer, Insulator
10.	C., Jenell Estes	09/01/1926	09/18/1996	70	Step-grandfather	Norfolk Naval Shipyard	Plummer, Shipfitter, Supervisor
11.	C., Rosalee S.	12/13/1929	02/18/2002	71	Spouse	Newport News Shipyard	Machinist
12.	D., Betty L.	11/07/1932	05/09/2007	73	Spouse	US Navy at Newport News Shipyard	N/A
13.	D., Frances C.	01/29/1942	Living		Spouse; Spouse; Father	Newport News Shipyard	Machinist; Machinery Installation; Chipper
14.	D., Hope L.	01/21/1932	03/20/2005	73	Father; Spouse	Local #83, Norfolk, VA; Carpenter & Sons	Pipecoverer; Boiler Repairman
15.	E., Alma	06/25/1919	02/20/2009	88	Spouse	Newport News Shipyard	Laborer
16.	E., Dorothy M.	10/17/1920	07/02/2006	85	Spouse	Newport News Shipyard	Joiner
17.	E., Mary A.	05/11/1919	07/21/2005	86	Spouse; Spouse	Newport News Shipyard	Pipefitter; heating and boiling work
18.	F., Irene	10/28/1923	08/17/1986	63	Spouse	US Navy	Worked in engine rooms
19.	G., Dorothy Railey	10/25/1943	Living		Father; Spouse	Norfolk Naval Shipyard, Contractor, petroleum refinery; Virginia Power, Contel Telephone Co.	Storekeeper, Contractor, Laborer; Laborer, Lineman, Installer
20.	G., Dorothy Savage	09/17/1915	05/24/1990	75	Spouse	CE Thurston, Norfolk Naval Shipyard, F.H. Gaskins & Sons Co.	Pipecoverer at all three
21.	G., Frances H.	03/09/1922	10/14/2002	80	Spouse; Spouse	Local 540 Plumbers and Steamfitters; Newport News Shipyard	Pipefitter; pipefitter
22.	G., Lillian L.	11/04/1912	11/21/2002	90	Spouse	Newport News Shipyard	Sheetmetal
23.	H., Ronald L.	08/30/1940	03/16/1995	53	Father	Union Carbide, Charleston, WV	Insulator
24.	H., Sharon	02/03/1952	11/26/1995	44	Father	US Navy, Local #10, Richmond, VA	Metalsmith, Welder, Boiler maker

(*continued on next page*)

TABLE 1 (*continued*)

	Name	DOB	DOD	Age at Death	Occupationally Exposed Family Member	Exposure Site	Occupation of Exposed Family Member
25.	J., Iris Lee	01/08/1926	08/29/2003	77	Spouse	Norfolk Naval Shipyard	Pipefitter
26.	M., Daisy M.	01/06/1905	04/06/1989	84	Spouse	Norfolk Naval Shipyard	Pipecoverer, Insulator
27.	M., Diane T. Bunting	03/26/1952	02/19/2004	52	Father	Local #83, Norfolk, VA; Norfolk Naval Shipyard	Pipecoverer at both
28.	M., Dollie F.	03/01/1932	03/20/1993	61	Spouse	North Carolina Shipbuilding & Drydock Co., Fort Worth & Denver City Railway, Norfolk Naval Shipyard	Pipecoverer at all three
29.	M., Elizabeth Frances	06/17/1920	05/23/1983	63	Spouse	Norfolk Naval Shipyard, Armstrong World Industries, CE Thurston	Pipecoverer at all three
30.	M., Rebecca Louise T.	12/17/1931	09/29/2000	69	Spouse	US Navy, SUPSHIP	Machinist Mate, Mechanic, Machinist, Planner/Estimate
31.	O., Ruby Lee	11/11/1920	10/27/1990	70	Spouse	Norfolk Naval Shipyard, CSX Transportation, Norfolk Naval Shipyard	Sheetmetal Mechanic; Sheetmetal mechanic, Pipefitter, Supervisor; Pipefitter
32.	S., Callie Sue	03/31/1943	09/08/2007	64	Father	Norfolk Naval Shipyard	Pipefitter
33.	S., Leola Maxine	02/11/1929	03/10/1985	56	Spouse	Newport News Shipbuilding & Dry Dock	Handyman, Electrician
34.	S., Opal D.	11/02/1921	06/05/1987	66	Spouse	Newport News Shipbuilding & Dry Dock	Pipefitter
35.	S., Sharon Jane Mill	08/11/1950	06/10/1999	49	Father	US Navy, Norfolk Naval Shipyard	Machinist's Mate, Machinist
36.	W., Carolyn J.	10/22/1935	11/28/1999	64	Spouse	Consolidated Rail Corp.	Switchman, Brakeman, Conductor
37.	W., Emma Moore	10/16/1921	09/12/1995	74	Spouse	Newport News Shipbuilding & Dry Dock, US Navy, Norfolk Naval Shipyard	Pipecoverer at all three

influenced by the occupational history or absence thereof. These changes either may have reduced or increased the apparent rates of mesothelioma.

Asbestos Exposure and Mesothelioma in Women and Young Workers

Price and Ware come to the conclusion, which is contradicted by a cursory knowledge of the use of asbestos, that no mesothelioma case that occurred in a female was ever caused by asbestos because no woman had ever had experienced sufficient exposure to asbestos.[14] They based this on the claim that female mesothelioma rates remain "unchanged" from 1973–2000. Price and Ware's misuse of SEER data allowed them to conclude that all female cases were unrelated to asbestos since female mesothelioma rates had remained "constant." In fact, Price and Ware contradict themselves on the article's most important point, "The age-adjusted mesothelioma rate for females was constant at an average of approximately 0.30 per 100,000 between 1973 and 1982, when it showed a one-time increase to 0.40 per 100,000 [emphasis added]." They go on to state, "One might be tempted to interpret this change as a response to increasing environmental exposure." I agree. However, Price and Ware argue that since the rates remain constant after 1992, this post-1972 increase is not causally related to asbestos exposure, but is instead explained by changes in diagnostic techniques. This assertion is unreferenced and un-described changes in techniques could just as easily decrease as increase the number of mesothelioma diagnoses. In addition, para-occupa-

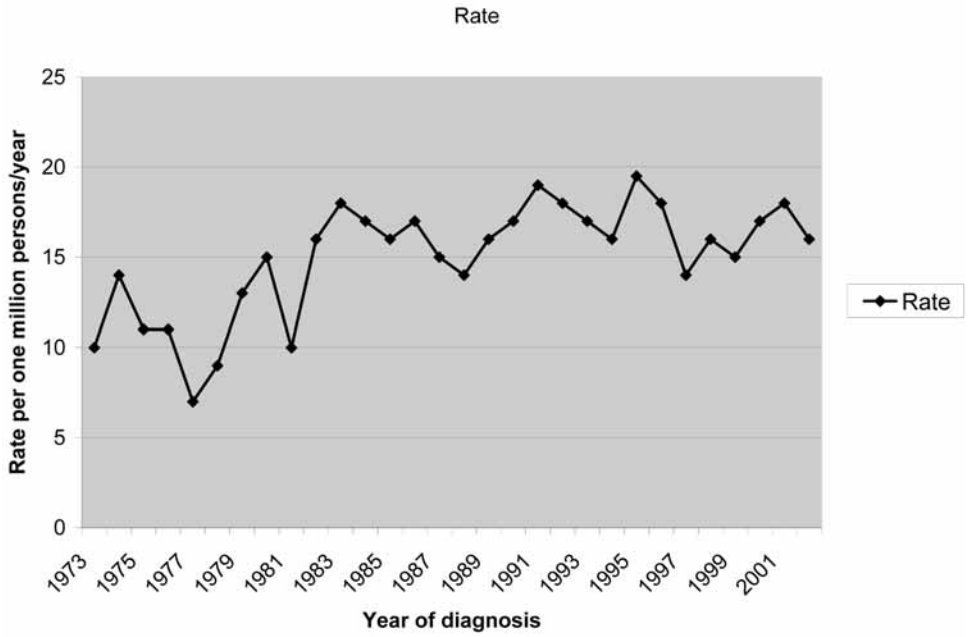

Figure 1—Women's Mesothelioma Rates age > 60, based on data reported in: Teta MJ, Mink PJ, Lau E, Sceurman BK, Foster ED. US mesothelioma patterns 1973-2002: indicators of change and insights into background rates. Eur J Cancer Prev 2008;17:525-34.

tional exposures from shipyard exposures reveal real risks to women.[39] Table 1 is a list of some cases in women with para-occupational exposure from the Newport News shipyard area. In this cohort, year of birth ranged from 1905 to 1952, and age of death ranged from 43 to 90, with 92% of the cohort older than 50. Of the 38 people in the cohort, 82% were exposed via their spouses and 21% were exposed by their father or both their father and spouse. Household exposures may be relatively high. Two exposed cases fit Teta et al.'s criteria for non-exposure (born in 1952).

In any case, the constantly elevated rates are compatible with occupational and environmental exposures. A combination of changes in either exposure levels or population exposed (or both) could explain these findings. The SEER data provide no information on these questions. But even those data actually do show a broader change in women's mesothelioma rates over time. Price and Ware report age-adjusted rates which mask the increased rates of mesothelioma in women above 50.[40] Teta et al. disaggregated the same data by age groups without deleting "deviants" and concluded that females above age 60, "had increasing rates from about 1977 through the late 1980s." Teta et al. claimed these rates were "followed by an apparent decline around 1992." However, her data, presented in a graph that is reproduced here (Figure 1), do not show a decline after 1992. In fact the rates peaked in 1995, dip in 1997 and increase until 2002. Given the small numbers and the quality of the data, it is inappropriate to make any conclusions for this data set; it is especially wrong to base conclusions on "eyeballing the data." On the other hand, case reports and workplace- and environment-specific epidemiologic studies like that presented by Azuma et al. clearly show that women had environmental and occupational exposures that caused mesotheliomas.

It is instructive to note that Price and Ware's conclusions conflict with data from countries other than the United States. In England, mesothelioma rates in females increased by about 20% from 1989–1991 to 1995–1997, and more than doubled by 2002–2004.[41] Similarly, female mesothelioma rates in Australia rose about 3-fold between 1980 and 2000.[42] The same pattern has been reported from Italy.[43] These rates are likely to be more accurate than US reports because the national health insurance coverage in these countries likely encourages more complete discovery of cases and more sophisticated diagnostic methods.

Pathologic evidence of female exposures completely disproves Price and Ware's hypothesis. Roggli et al. reported that as many as 75% of female mesothelioma cases had a history of asbestos exposure, but 80% of these were para-occupational.[44] Lung tissue asbestos burdens were "elevated" in 70% of a series of female mesothelioma cases.[44,45]

Most far-fetched among their claims is Price and Ware's un-cited assertion that "In contrast [to men], female exposures to asbestos have been primarily environmental. In the 1930s through the 1960s, women generally did not work in industries in which men experienced high levels of exposure to asbestos." Given the sharp rise of female factory workers during World War II, as evidenced by the fame and success of the "Rosie the Riveter" campaign, it remains unclear how

Figure 2—Female Asbestos Textile Workers, c. 1922. Reproduced from: "Garlock 2009" Slide Presentation. Olwin Moeller v. Garlock Sealing Technologies, LLC. Case Number: 3:07-CV-65-H. United States District Court, Western District of Kentucky at Louisville.

anyone could make such an ungrounded assertion. In the 1940s, women comprised 20–30% of shipyard workers.[46] Approximately 12 million women worked in the defense industries and support services across the nation, including in shipyards, steel mills, and foundries.[46] During World War II, the Kaiser Company built shipyard child care centers for working mothers, which were funded through the United States Maritime Commission. Kaiser's two shipyard childcare centers in Portland served nearly 4000 children.[47] Given the volume of information confirming women's work in high-asbestos exposure occupations and commonplace domestic exposure from asbestos contaminated clothing, it is hard to imagine how Price and Ware reached the conclusion that women's asbestos exposures, "have been primarily environmental."

Even Wikipedia notes that Cooke first reported asbestosis in a female asbestos worker in 1924. In the 1930s, many textile workers were female.[48] Spinning, weaving, and sewing were traditionally "women's work" and exposures were far from innocuous (Figure 2).[49] Brown et al. reported on a cohort of asbestos textile workers employed between 1909 and 1977 that included 1265 women out of a total cohort of 3072 workers.[50] Removing the flashings of molded articles can result in high exposures, and women performed this work in the manufacture of small asbestos cement products and brakes (Figure 3).[51]

Teta et al. claim male and female rates for the post-1972 "unexposed cohorts" (male and female) are similar. But their respective point estimates are 1.15 and 0.94, a 20% difference.[15] There are few cases (72 male and 58 female), probably because the oldest member of the cohort was only 49 years old at the time of publication. Few asbestos researchers would venture any conclusions on such a small cohort with so short a potential latent period. Low doses are probably associated with longer latent periods.[52]

In addition, they assume that no one began any job with asbestos exposure until they were 19. However, many blue collar workers often begin formal employment at age 16 and children at still younger ages may work with or around their parents who change their own asbestos brakes. These exposures can be quite high, as shown in Table 2.

Teta et al. are more conservative than Price and Ware, claiming only that there was "little or no potential for occupational asbestos exposure [to men or women in the U.S.] after 1972," when the US Occupational Safety and Health Administration (OSHA) issued its first asbestos regulations. They state that asbestos use declined over the past 30–40 years. They go on to state unequivocally and without citation that:

> Since the mid-1970s, the potential for occupational and therefore domestic asbestos exposure would be minimal in the general US population, particularly for exposure to amphiboles. The mesothelioma rate in the population who entered the workforce after this time period of reduction of asbestos exposure would provide a reasonable estimate of the background rates of mesothelioma.[15]

Figure 3—Finishing Asbestos Gaskets. Reproduced from: "Garlock 2009" Slide Presentation. Olwin Moeller v. Garlock Sealing Technologies, LLC. Case Number: 3:07-CV-65-H. United States District Court, Western District of Kentucky at Louisville.

TABLE 2 Studies Showing High Asbestos Exposures During Brake Work

Author	Year	Exposure Type	Exposures Reported
Lee[68]	1970	Blow out	3–5 f/cc
Boillat & Lob[69]	1973	Brake work undefined	0.3–29.2 f/cc
Castleman & Ziem[70]	1985	Damp rag	High: 2.6 f/cc; TWA: 0.28 f/cc
		Squirt bottle	High: 0.54 f/cc; TWA: 0.21 f/cc
		Stoddard Solvent	High: 0.68 f/cc; TWA: <0.1 f/cc
		Dry rag	High: 0.81 f/cc; TWA: 0.2 f/cc
		Brake washer	High: 1.1 f/cc
Hatch[71]	1970	Compressed Air	Fibers >5 µm: 2.1-8.2; 10 minute avg: 0.8
Rodelsperger[72]	1986	Passenger car (various operations)	Mean: 3.8–4.7 f/cc
		Truck (various operations)	Mean: 4.4–9.9 f/cc
Kauppien & Korhonen[73]	1987	Truck (various operations)	<0.1–125 f/cc; TWA: 0.1–0.2 f/cc
		Grinding	7 f/cc
Hickish[74]	1968	Auto blow out	Peak exposure: 7.09 f/cc
Hickish[75]	1968	Auto brake work, various	TWA: 1.57–2.55 f/cc
Clark[76]	1976	Auto disc brake change	0.2–1.9 f/cc
Hatfield & Longo[77]	1998	Bendix Chrysler (filing and cleaning)	8.53–14.57 f/cc
Hatfield & Longo[78]	n.d.	Bendix Ford (filing and cleaning)	5.47–12.67 f/cc
Hatfield & Longo[79]	2000	Sweeping and cleaning brake shop	Personal Samples: 7.5–8.8 f/cc Area Samples: 2.0–2.4 f/cc
Hatfield, Longo & Newton[80]	2000	Grinding	4.83–12.51 f/cc
Hatfield, Longo & Newton[81]	2000	Hand grinding	12.57–21.43 f/cc
Hatfield, Newton & Longo[82]	2001	Hand sanding	0.5–0.96 f/cc
Rohl et al.[83]	1977	Blowing dust	6.6–29.4 f/cc
		Beveling	23.7–72.0 f/cc
Osborn[84]	1934	Grinding	17 mppcf
Roberts & Zumwalde[85]	1982	Compressed air	0.14–15.0 f/cc
Lloyd[86]	1975	Servicing brakes	3.75–37.3 f/cc
Longo, Mount & Hatfield[87]	2004	Hand sanding and grinding and other operations	19.7–35.7 f/cc

This is not true. This wishful thinking and derivative argument appear in the "Results" section of the paper, although the authors never provide evidence that they studied or reviewed literature on the question of exposure to asbestos at home, at work, or anywhere else.

Annual asbestos consumption in the US peaked in 1973 at 803,000 metric tons, but remained relatively stable above 550,000 metric tons (except for 1949) between 1947 and 1979.[53] For comparison, during WWII, use ranged between 232,000 and 398,000 metric tons.

OSHA has never banned asbestos use (the agency does not have the legal authority to ban the use of any substance), and exposures up to 5 fibers per cc (f/cc) were permitted until 1976, when permissible exposure limits (PELs) dropped to 2 f/cc. Even defense witnesses retained by asbestos companies testify that two years of exposure to Canadian chrysotile at the 5 f/cc level doubles the risk of developing mesothelioma.[54] Imports of asbestos for use in brakes increased three-fold between 1990 and 2002.[55] The EPA banned spray asbestos in 1973, and in 1977 the Consumer Products Safety Commission banned the use of asbestos in joint compound and spackling sold to the public. Currently, OSHA has enough inspectors to investigate every workplace in America about once every 113 years.[56] Halley's Comet passes by every 75–76 years (Figure 4).

In the absence of effective surveillance, asbestos regulations have often gone unheeded. For example, despite the 1972 OSHA regulations, workers at the Newport News Shipyard received no training in asbestos safety procedures until 1978.[57] Workers have testified that unprotected exposures from a variety of asbestos-containing products continued for several years after the training began.[57]

In 1983, the problem was so bad that Congress held hearings on the issue after complaints that the Navy was not monitoring shipyard workers who were exposed to up to 5 times the OSHA limit.[58] It is worth noting that by the mid 1970s, there were few women in the trades (about 1 in 12), but 30% of the clean-up workers were women.[59] Clean-up workers have the highest asbestos exposures in shipyards.[60]

Teta et al. repeatedly refer to amphibole asbestos as if it were the only exposure of concern and claim this exposure was eliminated on Navy ships in 1975. This is the year the Navy stopped adding new amphibole-con-

Figure 4 – The OSHA inspector meets Halley's Comet.

taining pipe covering to ships; the tons of previously applied insulation did not disappear that year.† It was removed during the next decade and the highest exposure occur during sweep up and removal.[58,60]

Exposures have continued into the 21st century: even an under-funded, short-handed OSHA has issued citations for overexposures to asbestos through 2008.[61] Some have turned circumvention of the OSHA standard into a profit making business.[62] A quick Google search reveals that the *Boston Globe* reported that:

> Albania Deleon, owner of Environmental Compliance Training of Methuen, sold training certificates to hundreds of undocumented workers who had not taken a mandatory training course from 2001 to 2006. Deleon then sent them out to remove asbestos at job sites in New England, and paid them under the table."[62]

OSHA has failed to enforce the asbestos standard in auto body shops.[63]

As asbestos has been used in joint compound, house paints, ceiling and floor tiles, vermiculite insulation and brakes, asbestos exposures among household members (50% of whom are women) also remain all too common in 2009. Expanded vermiculite (sold as WR Grace's Zonolite) was an easily poured insulation ideal for walls and attics. In 1985, the EPA estimated that 940,000 homes contained, or had once contained, vermiculite attic fill.[53] Asbestos (being relatively indestructible) does not degrade on its own. It must be removed and is often unknowingly released during renovations. OSHA does not regulate home renovation exposures unless they are performed by outside contractors.

A study of fetal asbestos content provides further evidence of potentially important and continuing current exposures.[64,65] Haque et al. studied asbestos content of lung, liver, skeletal muscle, and placenta digests of 82 stillborn infants. They found asbestos fibers in 50% of the fetal digests: 88% were chrysotile, 10% were tremolite, and 2% were actinolite and anthophyllite. Mean fiber counts were highest in the liver (58,736 f/g), followed by placenta (52,894 f/g), lungs (39,341 f/g), and skeletal muscle. The autopsies were conducted between 1990 and 1992 and the maternal ages ranged from 17–42, indicating that some maternal exposures occurred after 1972. Ampleford and Ohar reported a pleural mesothelioma in a 22-year-old woman born in 1980, whose father removed asbestos insulation from furnaces and pipes.[66] The fact that humans are exposed to asbestos in utero further complicates any epidemiologic efforts to establish a threshold for asbestos carcinogenicity. As noted above, there are no unexposed controls, as in utero exposure provides an ample latent period and exposures to a developing fetus are likely to be more toxic than adult exposures.[67] Because it appears that asbestos exposure is ubiquitous and begins in utero, epidemiologic studies cannot distinguish the effects of non-asbestos exposures that may appear to elevate mesothelioma rates (like radiation) from induction or promotion of the effect of asbestos.

AVAILABLE COHORT EPIDEMIOLOGIC STUDIES CANNOT ESTABLISH A "SAFE" THRESHOLD FOR ASBESTOS EXPOSURE AND CANNOT BE USED TO ESTABLISH RELIABLE RELATIVE FIBER POTENCY ASSESSMENTS

Some experts have used meta-analyses of asbestos cohorts to claim that exposure to chrysotile asbestos must exceed some "background" threshold to cause mesothelioma.[88–90] Recently, an EPA-appointed Science Advisory Board (SAB) focusing on asbestos concluded that the available historical exposure data was too scant to reliably differentiate any potential potency differences by fiber type as attempted by Berman and Crump.[89,91] Finkelstein commented, "In essence all of the input data would consist of guesses and the output of the model would not be credible."[91] As the EPA's SAB concluded, impinger data (which measured total particles and did not distinguish dust from fibers) "cannot" be "used to generate PCM comparisons."[91] There is some evidence that the asbestos-mesothelioma relationship may follow more than one dose-response curve. There are many case reports of mesothelioma in individuals with brief or "low dose" environmental or home exposure (see Table 1).[18,31] On the other hand, "only" 10% of even the most heavily exposed cohorts develop mesothelioma.[92] Clearly, genetic factors and other exposures interact to produce mesothelioma in some, but not all, people with similar exposures.

Hodgson and Darnton attempted to evaluate the relative potency of asbestos types using some of the same studies used by Berman and Crump.[88] Rogers and Major, referring to Australian exposure data used by Hodgson and Darton, noted that, "the[se] exposure

†Except for Unibestos, a 70% amosite insulation which was primarily used on nuclear vessels, chrysotile was the predominant and often exclusive fiber in most pipe covering.

values . . . should be recognized as 'guesstimates', made by people who have not been trained in occupational hygiene and who have no experience in asbestos dust monitoring."[93] In addition to using the 'guesstimates' of the Australian exposures, there was no exposure data for other crocidolite cohorts in their study, and the authors simply assumed an exposure level. Hodgson and Darnton then compared the crocidolite exposure guesstimates to the inaccurate exposure data from Canadian miner and miller cohorts. These McGill University studies funded by the Quebec Asbestos Mining Association found a slight inverse relationship between the particle counts they used and fiber counts.[94] Their dose estimates were slightly better than random guesses.[94] McGill researchers were aware of this problem and ignored it. In 1969, during a discussion on asbestos counting methods at an international conference on pneumoconiosis in Johannesburg, South Africa, McGill's Corbett McDonald asked, "Can an inaccurate instrument like the midget impinger (MI), give an accurate result?"[95] He was informed that it could not. Just as a stopped watch, which is correct twice a day, should not be used to tell time, unreliable exposure estimates should not be used to devise inevitably unreliable estimates of relative fiber potency. Hodgson and Darnton's comparison of dose-response relationships between these two large cohorts is as reliable as the square of the "guesstimate." Hodgson and Darnton were aware of these problems as well, and wrote, "Certainly these estimates are much less soundly based than one would wish." Unfortunately, they pressed on stating, "Some view does however need to be taken. . . ."[96] A wrong view based on inadequate data can be worse than no view at all; it can and has encouraged the continued use of chrysotile and been used to persuade juries that chrysotile products are harmless. Another weakness of the Hodgson-Darnton review is that it dealt with 17 cohorts representing special industries. It did not include any case-referent studies for end-use exposures, which represent the most common pattern for asbestos-associated mesotheliomas.[88] Despite these failings and contrary to the positions taken by Price and Ware and Teta et al., Hodgson and Darnton (whose model inherently adopts a no-threshold assumption) rely on these "guestimates" to calculate relative potency for crocidolite, amosite, and chrysotile for mesothelioma induction of 500:100:1.[88] Leigh and Robinson demonstrated the arbitrariness of these estimates.[97] They recalculated them and accounted for clearance of amphibole and found potency ratios to be 26:14:1 which represents a twenty fold difference for crocidolite.[97] An often-cited set of potency ratios in the literature is 30:15:1.[98]

Most other cohorts are too small to evaluate the effects of even moderate levels of exposure. Even fiber PCM counts may be misleading.[99] Hein et al. found that "Current PCM-based methods may underestimate asbestos exposures to the thinnest fibers, which were the strongest predictor of lung cancer or asbestosis mortality."[100] It is possible that amphiboles are more potent than amphibole-contaminated chrysotile, but existing epidemiology cannot support or rebut this theory no matter how often it is repeated. Peto et al. titled their recent discussion of the issue of chrysotile causation "Speculations on the Contribution of Chrysotile," and with respect to ecological epidemiology, speculation it is.[101] At a recent deposition, Teta's employer, Dr. Dennis Paustenbach, agreed that epidemiologic studies could not establish a "threshold" for the asbestos-mesothelioma dose response relationship, saying, ". . . why these epidemiologists are making these toxicology statements [that there is a threshold] is beyond me but that's their choice."[102] Ironically, these views on the limitations of epidemiology did not prevent him from elsewhere using epidemiologic studies to claim a threshold for the chrysotile-mesothelioma relationship.[103]

Pathologic Evidence o f the Importance of Short Fiber Chrysotile as a Cause of Mesothelioma

Substantial pathologic evidence contradicts the company-sponsored[‡] theory that chrysotile asbestos cannot cause mesothelioma.[11] At least four studies that look at pleural fiber levels by fiber type find that "short" chrysotile is often only the only fiber type found and is almost always the predominate fiber in patients with mesothelioma.[104–107] Lebouffant was the first to compare lung and pleural fiber types and sizes, and found that pleural and fiber types were different in the same patients. He stated that:

> As a matter of fact, in several cases of mixed dusts (chrysotile-amphiboles), there is significant chrysotile enrichment in the pleural tumor, contrary to the observations in the lung parenchyma in which . . . a relative amphibole enrichment was found. It thus appears that the chrysotile impoverishment of the parenchyma cannot be accounted for only by the dissolution of this mineral, but that there seems to be a preferential drainage of chrysotile towards the pleura.[107]

More importantly, he found that most fibers were short (<5μm)[§] (Figure 5).

Sebastien et al. compared the retention of fibers in parenchymal and pleural tissues in 29 patients with a variety of asbestos diseases and jobs.[105] All but one

[‡]Brake manufacturing companies GM, Ford and Chrysler funded Teta el al. WR Grace, the seller of Zonolite brand of vermiculite, funded Price's initial 1997 SEER paper.[16] Price and Ware used some data from the 1997 paper in the 2004 paper.[14]

[§]While "short" is a relative term, federal agencies adopted a regulatory convention of counting only fibers of 5 μm or longer. Ironically, the 5 μm cut-off was arbitrarily established because the predominance of short fibers in airborne samples made it difficult to count all fibers. The 5 μm "convention" has been carried over to lung fiber counting.

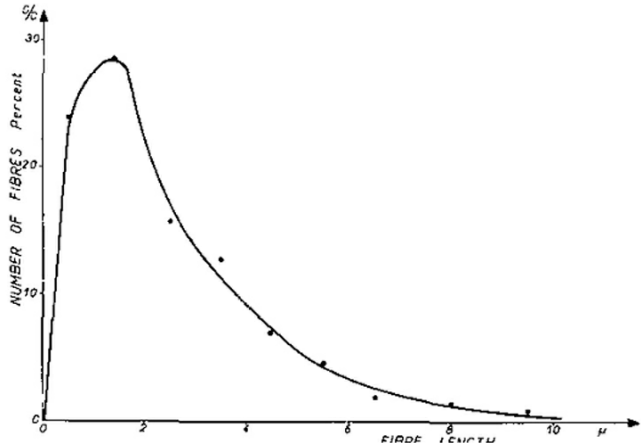

Figure 5—Relative Proportion of Short and Long Fibers in Lung Tissue. Reproduced from: LeBouffant L. Investigation and analysis of asbestos fibers and accompanying minerals in biological materials. Environ Health Perspect 1974;9:149-53.

worked with asbestos products and eleven had mesothelioma. In three of these cases, chrysotile was the only fiber they found in the pleura. Two of these three had significant (78% and 25%) amphibole lung parenchymal fiber counts. The third (with only chrysotile in the pleura and lung) was a 78-year-old female "unskilled worker" with no history of asbestos exposure.[105] Sebastien et al. concluded:

1. This study has obviously demonstrated that lung parenchymal retention is not a good indicator of pleural retention, the most striking feature being the absence of relationship between parenchymal and pleural concentrations, many pleural samples being free of asbestos fibers.

   The finding of many negative samples may be due to an heterogeneous topographic distribution of intratissular fibers in pleural area. If fibers are concentrated in pouctual areas, they can be ignored by the transmission electron microscope (TEM) which observes a very small size sample.

2. This study has demonstrated that the retention of asbestos fibers in parietal pleura was type and size related, and that inside the parietal pleura most of the fibers were short chrysotile fibers.
   The presence of fibers in pleural tissues involves the translocation of fibers to pleura, and then the penetration of fibers inside tissues. Thus, two possible explanations can be given for these findings:

   a) Only chrysotile fibers can be transported and reach the pleura.

   b) Fibers of all types can be transported to the pleural area, but only chrysotile fibers are retained in the pleural tissue.[105]

These finding have been reproduced by Suzuki and Yuen,[104] Dodson et al.,[106] LeBouffant,[107] and Kohyama and Suzuki.[108] Kohyama and Suzuki compared lung and pleural fibers in 13 insulation workers: three with asbestosis, three with asbestosis and lung cancer, and seven who had died from mesothelioma. Three had amosite and chrysotile in the lung but only chrysotile in the pleura.[108] Six cases had discordant crocidolite counts with elevated concentrations in the lung but no fibers in the pleura. Overall, counting all fiber sizes, chrysotile counts were similar in the lung and pleura; in three cases chrysotile concentrations were higher in the pleura than the lung. Suzuki, Yuen, and Ashley examined 168 mesothelioma cases and found that the majority of fibers were short (< 5u) (89%) and thin (<0.25 μm) (93%) chrysotile fibers.[109] Only 2.3% were consistent with the Stanton hypothesis that predicted that long fibers were more pathogenic than short fibers.[109] In a small series of 14 cases with and without history of asbestos exposure, Boutin et al. found that amphiboles outnumbered chrysotile fibers in pleural tissue from all cases.[110] Müller et al. could not replicate these findings, and stated, "In our collective of former miners of the Ruhr area we do not find asbestos fibers especially amphibole fibers directly located in black spots."[111] In Boutin et al.s' cases, the lungs contained 99% amphiboles, however they noted that chrysotile might have been hidden by debris.[110] In addition, Boutin et al. failed to find chrysotile in cases where there was documented chrysotile exposure, and suggested that "short and thin chrysotile fibers could be less easily detected among a 'background' of particles in anthracotic samples."[110] Despite this fact, and consistent with Suzuki and others, only 22% of these fibers in black spots were longer than 5μm. Therefore, the majority (77%) of pleural fibers were short (< 5μm). Black spots do not correlate with asbestos pathology; in fact, Michev et al. found that "pleural plaques were mostly seen in the areas with a lower prevalence of black spots."[112] Müller et al. found that, "The morphological finding of black spots is not an indicator for an existing mesothelioma or the possibility for the further development of a mesothelioma."[111]

Dodson et al. compared fiber types in the lung and pleura in 8 shipyard workers. All had amphibole and chrysotile fibers in the lung. One had only chrysotile in the lung.[106]

Animal studies support these human pathology findings. Short, thin chrysotile fibers induce pleural and peritoneal mesothelioma in rats.[113–116] Wagner's rat studies provide reliable evidence of relative potency. As in observations in humans, chrysotile lung retention was relatively short. After 24 months, the animals had fifty times more amphibole than chrysotile in the lung (Figure 6). Retained lung asbestos did not predict either lung tumor or mesothelioma risk. Canadian chrysotile was much more potent, on a weight basis, than the amphiboles (Figures 7 and 8).

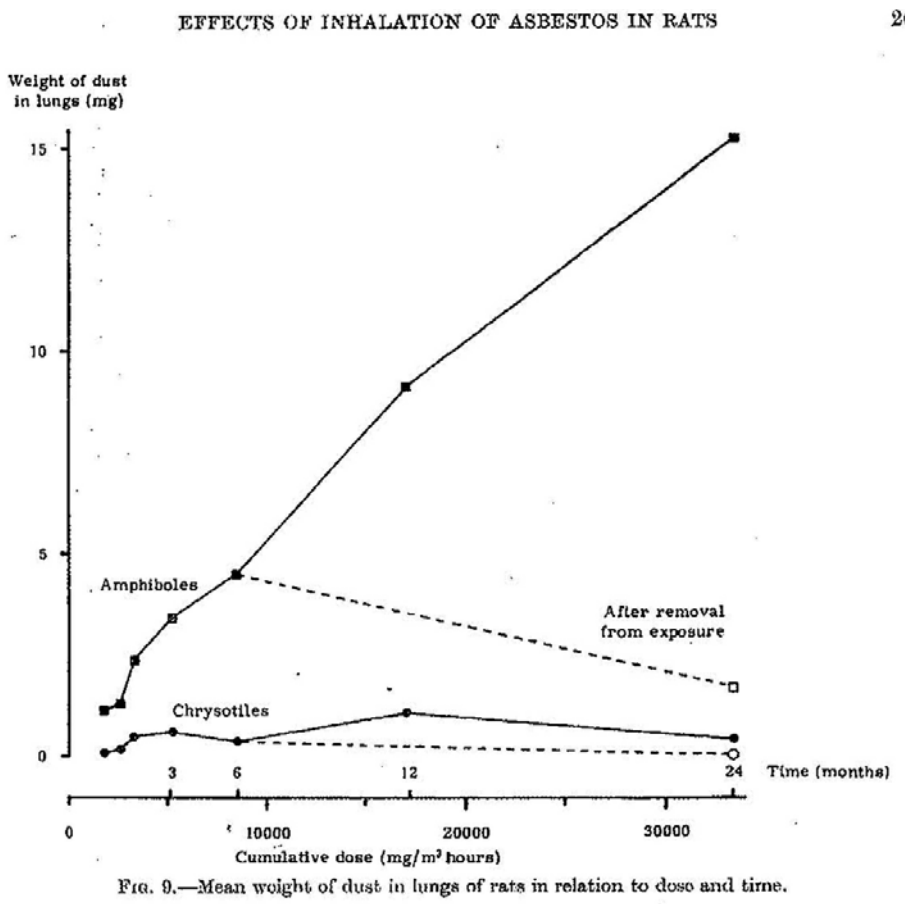

Figure 6—Mean weight of dust in lungs of rats in relation to dose and time. Reproduced with permission from: Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. Br J Cancer 1974;29:252-69.

It is universally accepted that asbestos must reach the pleura to initiate cancer formation.** Short fiber chrysotile is the predominate fiber at the site of the mesothelioma.[104,109] A minority of pathologists rely on SEM lung counts—which they admit are biased against finding chrysotile and biased toward finding amosite—to argue that a certain minimum lung concentration of chrysotile must be present to establish that chrysotile has contributed to cause any particular mesothelioma.[124] Roggli and colleagues are quite capable of comparing lung and pleural fiber burdens to disprove Sebastien's finding that his data ". . . obviously demonstrated that lung parenchymal retention is not a good indicator of pleural retention, the most striking feature being the absence of relationship between parenchymal and pleural concentrations," but they have chosen not to repeat his studies.[125] Asbestos fibers in the lung do not initiate mesothelioma formation. The fibers in the pleura cause the mesothelioma in the pleura and researchers from different countries studying workers in different jobs have repeatedly found that pleural fibers are overwhelmingly short thin chrysotile fibers.[104–107,109]

Selikoff found mesothelioma in 4.6% of amosite insulation and blanket manufacturing workers and 8% of insulation workers who used these products in addition to chrysotile products.[25] Thus chrysotile appears to double the risk of mesothelioma compared to amosite-only exposure. Acheson and Gardner reanalyzed lung fiber burdens in patients with mesothelioma and found that mixtures of amphiboles and chrysotile are associated with a relative risk of mesothelioma of 61, compared to 12 associated with amphiboles alone and 6 associated with chrysotile alone.[126] They reported that this pattern was closer to a multiplicative than an additive interaction between chrysotile and amphiboles.[126] The synergistic effect was strongest when the total fiber counts were low, which is the most common occurrence when Roggli dismisses chrysotile as a cause of a patient's mesothelioma.

Ecological epidemiology based on SEER data that include no information on history does not and cannot provide any useful information on individual risk or disease causation in general. Risk analyses, like those of Hodgson and Darnton and Berman and Crump, that rely on unreliable exposure estimates cannot establish

**Asbestos stimulates intrapulmonary production of cytokines sufficient to cause a mesothelial proliferation or pleural fibrosis and this may promote cancer cell growth. However, direct cellular contact appears to induce mutations.[117–123]

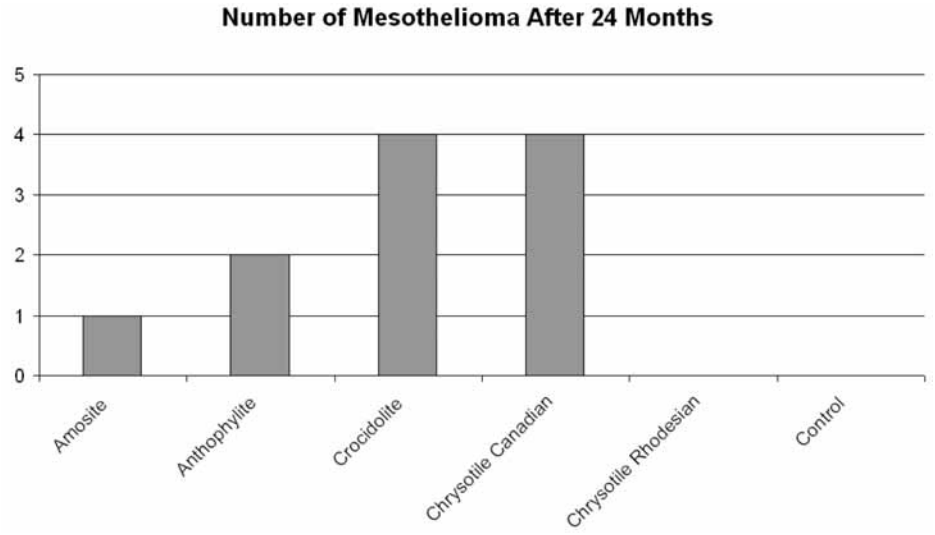

Figure 7—Relative Mesothelioma Potency by Fiber Type in Rats, based on data from: Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. Br J Cancer 1974;29:252-69.

fiber potency estimates. This is especially true when the results of both analyses conflict with animal experiments and human clinical data including clinical-pathologic evaluation of pleural tissue fiber levels. It is unscientific to use conversions that have been shown to be "guesstimates" to exclude known asbestos exposures as contributing causes of in specific individuals.[93,94,127] Rather, specific relevant clinical evidence and history of exposure can establish cause-effect relationships in an individual; pathologic studies of lung fiber counts that fail to reflect fiber types and systematically grossly undercount fiber types that are found at the site of the crime cannot only spread confusion or systematically mislead.[124,128] Risk assessments based on unreliable exposure data may make for interesting theoretical exercises, but "guesstimates" should not be mistaken for scientific argument.

THE USE OF FIBER ANALYSIS: A CASE STUDY OFHOW BAD SCIENCE CAN CONTRIBUTE TO BAD PUBLIC POLICY AND ERRONEOUS COURTROOM AND REGULATORY TESTIMONY

Some researchers have used lung fiber counts to claim that brake exposures do not contribute to mesotheliomas in brake workers.[129] Butnor, Sporn, and Roggli compared lung fiber counts in 10 brake mechanics to a group of historical controls, who they claimed had no occupational asbestos exposure.[129,130] They further claimed that brake asbestos exposures did not contribute to the development of mesothelioma in these particular workers because their exposures were not higher than their laboratory's fiber counts for all of their allegedly unexposed cases.[129]

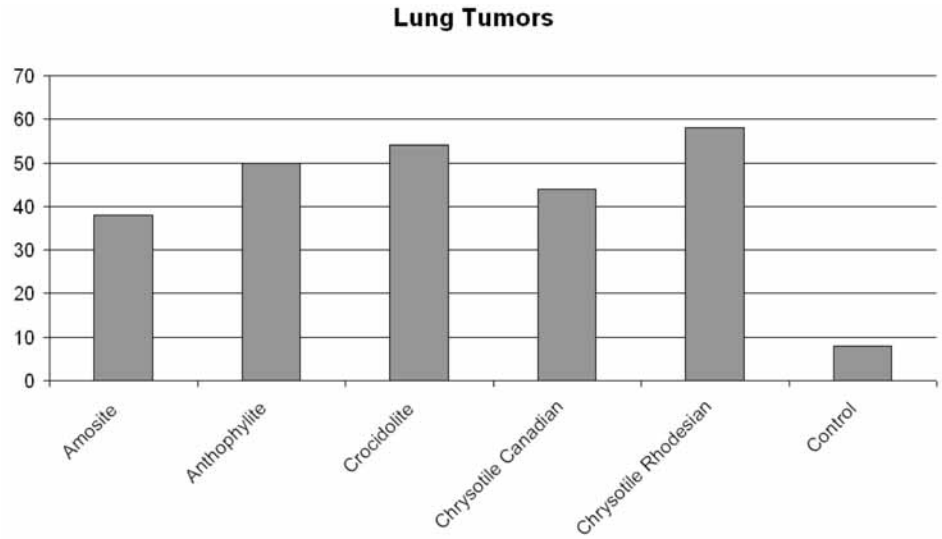

Figure 8—Relative Lung Tumor Potency by Fiber Type in Rats, based on data from: Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. Br J Cancer 1974;29:252-69.

Roggli himself contradicted this interpretation of the importance of above-background exposures when he testified at the request of an injured worker:

> Once a patient is diagnosed with mesothelioma, one of the first questions to resolve is where and when he or she was exposed to asbestos. Because asbestos dust is so strongly associated with mesothelioma, proof of significant exposure to asbestos dust is proof of specific causation in a given case. The scientific and medical community have yet to determine a level of exposure to asbestos below which mesothelioma does not occur. While there is no threshold, there is insufficient evidence to implicate levels of exposure to asbestos that occur as a result of background or ambient air exposure. Very low levels of exposure above background, however, have been demonstrated to cause mesothelioma.[131]

Certainly brake workers have at least "Very low levels of exposure above background."

More importantly, Butnor, Sporn, and Roggli do not explain how their 10 cases were selected, except that they all came from a pool of cases that had been referred by plaintiff and defense lawyers, and that brake dust was the sole recognized source of asbestos exposure in all 10.[129] They report no effort to determine if the chosen cases were in any way (fiber counts, work history, referral source) representative of the entire pool of cases. The authors should have specified a specific selection methodology to avoid bias, especially given the fact that Roggli had already concluded and testified on numerous occasions that brake exposures cannot cause mesothelioma. Roggli's *a priori* hypothesis was that brake exposures do not cause mesothelioma. A more appropriate scientific test would have been an effort to find a worker with brake exposures only whose fiber counts exceeded those of all "controls." A failed effort to disprove this hypothesis would have increased the likelihood that it was correct. On the other hand, finding a single case with elevated fiber counts would have disproved the hypothesis.

Roggli has testified in court and regulatory hearings using the unsubstantiated assumption that "background" asbestos exposures do not contribute to mesothelioma risk. He has claimed[132] that:

1. Exposures to asbestos from some asbestos products do not increase the risk of contracting mesothelioma.
2. Mesotheliomas that occur in some individuals with occupational asbestos exposure and lung asbestos burdens are "idiopathic" if their asbestos fiber counts are not higher than "95% of the control levels."
3. Chrysotile asbestos from certain mines in California does not cause mesothelioma.

Recently, Chrysler used this argument to justify a court order to stop the burial of Harold St. John, a brake mechanic who had died of mesothelioma, to get access to his lungs to perform a fiber analysis.[133] A process server attended the funeral and, after the mourners had left, instructed the funeral director not to bury the body but to return it to the funeral home. Dr. Roggli testified at the request of Chrysler to establish the medical importance of lung tissue burden to justify the subpoena. Chrysler had and has Mr. St. John's pleural and tumor tissue but refused to examine it for fibers. Roggli does not consider pleural tissue fiber level to be relevant to the issue of asbestos causation.

I volunteered to testify for Mr. St John's family. On March 18, 2009 the New Jersey Appellate Court ruled that Chrysler had no need to remove Mr. St John's lung tissue and his family was allowed to bury him.[134]

There are many problems with the purported scientific basis of Chrysler's rather ghoulish request. The main problem is demonstrated in Figure 9, which shows a gentleman looking for his key. After helping him for awhile, you ask where he lost the key. The answer is, "On the next block." You then ask, "If you dropped it somewhere else, why are you looking for it here?" He answers, "Because the light is better."

As Roggli and everyone else acknowledge, the asbestos in the pleura—not the lung—is the cause of mesothelioma. As noted above, there is no relationship between the asbestos in the lung and that in the pleura. Chrysotile is biopersistent in the pleura—not the lung—and amphiboles predominate in the lung and not the pleura (see Figures 5 and 6). While several researchers have been able to analyze pleural tissue, Roggli and coauthors reject the use of pleural fiber counts because of the perceived difficulties in obtaining samples.[135]

There are many problems with the use of fiber counting to determine causation in individual cases. I review some of them here. Roggli summarized his use of fiber counting in his testimony in the St. John case:

> Well, I think that there are three scenarios that I could envision that you would see as a result of doing the fiber analysis in this case. One would be to find a fiber burden which is no different from our background or control population, which would indicate, in my opinion, that it's an idiopathic mesothelioma.
>
> The second would be that you would find elevated levels of commercial amphibole fibers, indicating that there was some exposure that has not been identified, other than to friction products, and that likely was the cause of the mesothelioma.
>
> And the third possibility is that you would find only elevated tremolite and/or chrysotile present in the tissues, and that would actually be a finding that would be favorable towards the Plaintiffs.[136]

In the third scenario, Roggli implicitly acknowledges, but avoids affirming, the fact that elevated

Figure 9—Looking Where the Light is Better. Art courtesy of Artt by Cartooncity.net.

tremolite and/or chrysotile would be evidence of mesothelioma caused by brake exposure. In the first two scenarios, Roggli uses fiber count data to assign causation to commercial amphibole exposures and exculpate brake amphibole (and/or chrysotile) exposures. In essence, Roggli compares lung fiber counts between mesothelioma cases and a group of "controls" whose fiber levels he claims represent "background" exposures. This can be misleading and underestimate asbestos contribution to causation if any of the following three scenarios occur.

1. If the fiber counts in "controls" are high because of unrecognized occupational, para-occupational (household or similar), or environmental exposure, all these comparisons will be biased against finding that the case's asbestos exposure contributed to cause the mesothelioma.
2. If Roggli's technique systematically undercounts chrysotile, it will underestimate the contribution of this fiber type (and total asbestos exposure) in exposed cases. Assuming there is a difference in chrysotile counts between the groups, undercounting chrysotile creates a bias against finding a difference between cases and "controls." If chrysotile is present in cases and not "controls," undercounting will result in low or no fiber detection in both cases and "controls."
3. If a higher percentage of chrysotile (compared to amphibole fibers) translocates to the pleura, lung fiber counts will underestimate the contribution of chrysotile to disease causation.

I now address the underlying studies and arguments that form the basis of Roggli's testimony.

1. Did controls have occupational or environmental exposures?

In court testimony, Dr. Roggli has been quite critical of the controls and techniques used by other scientists.[137] For example, he has criticized Dr. Abraham for relying on controls performed in another laboratory, saying "I think that—that that [*sic*] is not good science; and in my opinion the [Abraham's] numbers are not interpretable."[††] In a presentation to asbestos company defense lawyers, Roggli claimed that Suzuki's laboratory was contaminated with chrysotile.[104] However, he failed to note that 3.2% of Suzuki's cases were chrysotile-free, a fact which rebuts this criticism.[136,138] In addition, Suzuki ran controls in his 2005 paper to rule out contamination from water, fixative or formalin.[109]

Srebro, Roggli, and Samsa[109,138] selected twenty patients who they claimed had "no documented history of asbestos exposure and no evidence of asbestos-related disease" as controls to determine the lung burden of asbestos in people who they claimed had no occupational history of asbestos exposure (background exposures).[130] After looking at controls' fiber counts, however, the authors found high amosite levels in one patient. In response, they conducted "an extensive search through this patient's medical records and [made] two phone calls to surviving relatives [which] revealed that his employment history included installing furnaces, an occupation associated with asbestos exposure." This case was important evidence that their original screening had failed to exclude individuals with important occupational exposures. Srebro, Roggli, and Samsa excluded the "control" post-hoc based on the actual results of the only outcome of interest—lung fiber counts. Additionally, they failed to repeat this "extensive search" with the remaining "controls," despite the fact that at least 8 had occupations that are more usually associated with occupational asbestos exposure than "furnace installer" and three lacked any occupational history.

There is no justification for excluding only the control with the highest counts, other than the fact that the inclusion of this individual would have obviously signaled the inadequacy of their selection criteria for "unexposed" controls. Srebro, Roggli, and Samsa do not explain why they did not obtain more information on

[††]In this criticism Roggli emphasizes the lack or reproducibility of results between laboratories which complicates and undermines the value of non-research use of fiber counting.

the control with the next highest levels and so on down the line.[130] Had they used this same standard ("exclude controls with "high" counts) for all controls, they could and should have excluded every "control" but the one with the lowest fiber counts. I have previously described the use of arbitrary and non-standardized criteria for the selection of controls as "differential peeky bias."[139]

It is unclear why Srebro, Roggli, and Samsa failed to exclude control case 19 from the paper (never mind as a "control"). The paper was based on the premise that it was a study of patients who all had asbestos body counts within their laboratory's "normal range":

> This report presents a comparison of data for 18 mesothelioma cases with AB counts (by light microscopy [LM]) within our "normal" range *versus* data for 19 "control" cases with normal lungs at autopsy. Our normal range is 0 to 20 AB/g. . . . [italics in original].[130]

"Control" case 19 had 22 asbestos bodies per gram, which is higher than Roggli had repeatedly reported (both before, after, and in the 1995 publication of this paper) as the high end of his normal range.[45,129–130,140–143]

Srebro, the first author and a medical student with no training in occupational medicine at the time she collected the data, conducted the investigation to determine if the controls had a previous history of work with asbestos.[144] None of the controls were interviewed because they were all dead at the time the study was conducted.[144] The listed occupation for three of the 19 controls was NA (not available) and the researchers had no information on smoking for 10 of the "controls" (Table 4).[130] This indicates that Srebro failed to access or record from information sources that almost always contain this information, such as complete medical records or interviews with family members, to determine what jobs or environmental exposures the controls had.

Several of the study controls had likely occupational exposure to asbestos.[130] Control 24, one of the patients with unknown occupation, had the highest total "control" fiber count—more than three times the next highest "control" and the fifth highest level for all the cases reported (18 mesothelioma patients plus 19 controls).[130] Other "control" cases with possible occupational asbestos exposure included three manual laborers, two listed as "Air Force," two hospital workers, an electrical engineer, a spinning mill worker, a truck driver with esophageal cancer, and a garage owner. According to the U.S. National Institute of Occupational Safety and Health's (NIOSH's) Work-Related Lung Disease survey, hospital workers, truck drivers, electricians and farmers are in the top ten recorded industries in workers with mesothelioma.[145] For example, a garage owner likely will have entered the service area of a garage where asbestos exposures are all too common.[145] Similarly, manual laborers, Air Force veterans, electrical engineers, truck drivers and spinning mill workers all may have had occupational exposures to asbestos.[‡‡] Ironically, three years before the 1995 study was published, Roggli reported that manual laborers had occupational exposures and had median asbestos body counts of 830, nearly three times higher than levels in shipyard workers (295).[146]

Srebro, Roggli, and Samsa did not exclude potentially confounding "environmental" exposures when they labeled their "controls" as having had "background exposure."[147] They distinguish household and "environmental" from "background" exposures.[148] Roggli believes household and "environmental" exposures can cause mesothelioma, and has provided examples of environmental exposures that can cause mesothelioma, including "living near an asbestos manufacturing plant or a mine or a mill . . . in Louisiana many of the driveways and playgrounds down there used tailings that Johns Manville had from a manufacturing plant, deposits of tremolite, for example in the El Dorado area of California . . . and . . . Libby, Montana due to the mining operations."[148,149] In addition, Roggli believes that household exposures in patients who live with asbestos-exposed workers can cause mesothelioma.[127]

Srebro, Roggli, and Samsa's "controls" are indistinguishable from their mesothelioma cases. Srebro, Roggli, and Samsa reported that the mean amosite and tremolite, anthophyllite, and actinolite (TAA) levels were statistically significantly different between cases and controls (but failed to note that this was only true after they deleted the "control" patient with the highest fiber counts). Srebro, Roggli, and Samsa reported the mean amosite level for their mesothelioma cases as 270 uncoated fibers per gram of wet lung tissue (uf/gwt) but the correct value appears to be 240 uf/gwt. In the text they report that the one-tailed Wilcoxon test (performed after excluding the control case with elevated amosite levels) showed a statistically significant difference between cases and controls in amosite ($p<.006$) and tremolite ($p<.004$) lung burdens. However, this is incorrect and the authors cannot explain how they achieved this result.[§§] In the footnote below their Table 2, the authors write that the same result is a comparison of means, but Wilcoxon is not a comparison of means. Wilcoxon is a non-parametric test for assessing whether

[‡‡]I have reviewed a case of mesothelioma in a truck driver who received occupational exposure to asbestos by adjusting the brakes on his trucks.

[§§]Dr. Samsa responded to my request for an explanation of the statistical analysis and answered, "I'm afraid that I can't be of much assistance as, if my recollection from over a decade ago is correct, my role in the analysis was limited to the exploration of uni-modal versus bi-modal distributions. In re-reading the paper, one thing that would have been helpful to report was how the laboratory values that were below the threshold of detection were treated—for example, were they set to 0, to 1/2 the limit of detection, etc. In the absence of this information, it is difficult to comment on your questions. Perhaps the first author can be of more help."

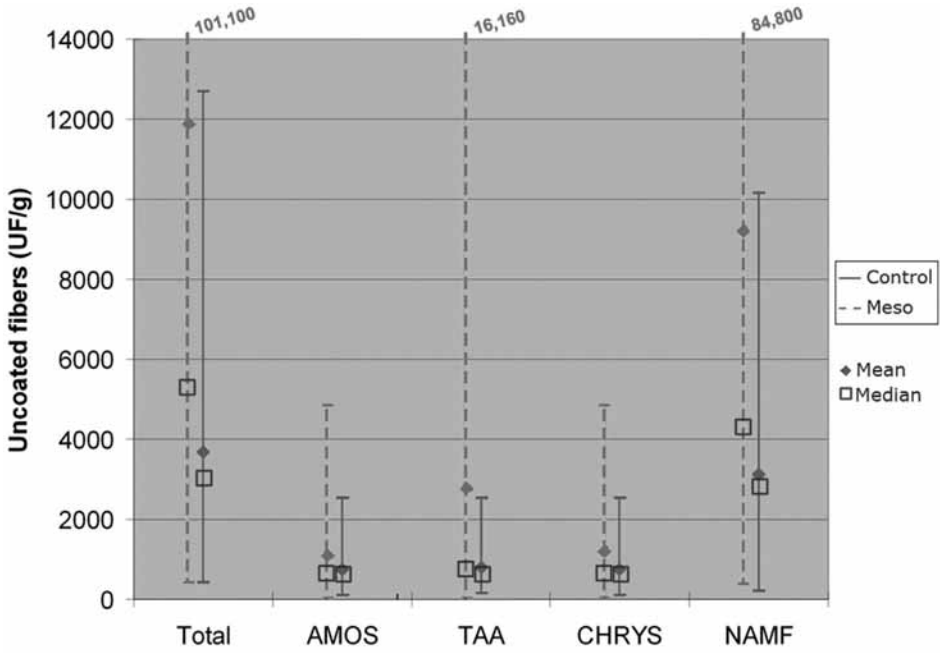

Figure 10—Medians, means, and ranges of uncoated fibers per gram of wet lung tissue in "controls" and mesothelioma cases, based on data from: Srebro SH, Roggli VL, Samsa GP. Malignant mesothelioma associated with low pulmonary tissue asbestos burdens: a light and scanning electron microscopic analysis of 18 cases. Mod Pathol 1995;8:614-21.

two independent samples of observations come from the same distribution. In any case, a two tailed test should have been performed since it was possible that some of the controls had higher fiber counts than the mesothelioma cases. Indeed this turned out to be the case in control 24 and the deleted control. For tremolite, the significance level for the one-tailed Wilcoxon result was p<.063 and for the two-tailed test was <.025. The significance level would be p=.05 for the correct, two-tailed test. However, I have been unable to repeat their statistical results. Even using Roggli's statistical method and after deleting the excluded "control," there was no significant difference for asbestos bodies, total fibers, and chrysotile between cases and controls.

Using the correct (two-tailed) test that accounts for left censored data, the controls and mesothelioma cases fiber counts are not different.[150] Fiber counts for both controls and cases overlap (see Figure 10). These results mean that either asbestos did not contribute to any of the cases or the controls do not represent exposures that are without risk. Based on the occupational histories and fiber counts, the latter is clearly the case.

There is no scientific basis to state that a "control" had no occupational exposure to asbestos if there is no information on their work history. It seems that this missing information invalidates Roggli's subsequent papers and individual case causation determinations based upon the data (or rather lack of data) in this study.

2. Did controls have typical/representative "background" exposures?

"Background" asbestos lung levels are a function of background ambient air concentrations, which are related to geographic location. Areas adjacent to asbestos manufacturing plants and mines and cities in general have high levels compared to other areas. There is no standard "background" exposure, as ambient air levels and lung fiber counts vary.[151] Srebro, Roggli, and Samsa did not report any information on the geographic distribution of their "controls" and never evaluated environmental or household exposure differences.[147] Further, Srebro, Roggli, and Samsa do not distinguish "environmental" or household exposure levels from "background" exposures. Since Roggli himself believes that environmental and household exposures cause mesothelioma,[130] his "controls" do not represent a threshold for the induction of mesothelioma.

3. Did the counting method reflect actual fiber levels?

Roggli's scanning electron microscope (SEM) method cannot "see" the thin chrysotile fibers that are most common in the lung and the pleura, which leads to undercounting of chrysotile and the misleading conclusion that chrysotile is not an important cause of mesothelioma. Roggli uses a scanning electron microscope (SEM) set at a magnification of only 1000× (the method is capable of 10,000–20,000×), which misses chrysotile fibers that are <0.15μm in diameter.[152] As a result, he fails to count most chrysotile fibers which are, on average, .03μm–.07μm in diameter.[129,136]

The EPA evaluated various measurement methods and concluded:

> SEM, for purposes of this rulemaking, was determined to be inadequate for building clearance for the following reasons: (1) Currently available methodologies are not validated for the analysis of asbestos fibers; (2) SEM is limited in its ability to identify the crystalline structure of a particular fiber. (SEM analysis is therefore confined to the identification of structures by elemental composition and morphology); (3) recent studies conducted by NBS have evaluated several types of scanning electron microscopes and the variability between these instruments. (NBS has found the image contrast of the microscopes is difficult to standardize between individual scanning electron microscopes); and (4) currently no laboratory accreditation program exists for accrediting SEM laboratories.[153]

All other US agency protocols that relate to fiber counting use only TEM analysis. These include EPA's Asbestos Hazard Emergency Response Act Protocol,[154] NIOSH 7402,[155] ASTM Air sample analysis,[156] ASTM Dust sample analysis,[157] International Organization for Standardization (ISO) air sample analysis,[158] EPA bulk sample analysis,[153] and EPA Super Fund site air analysis.[159]

The EPA scientific advisory board on asbestos used strong language to support the use of TEM:

> Multiple binning should be evaluated, *but only using TEM-analyzed environmental exposure data that is directly associated with health outcomes*. Studies continue to reveal the importance of fiber width in potency. Fiber width is the most critical dimension in determining deposition site in the respiratory system, plays a significant role in determining surface area exposed to tissue, and may be a factor in mobilizing fibers from alveoli to pleural space. Future attempts to model fiber potency should have at least two bins for width. One possible width division could be an aerodynamic diameter of 2.5 μm, which is the cut point for EPA fine (~respirable) particles. This would be ~0.5 for amphibole asbestos and ~0.65 μm width for chrysotile. . . . *only TEM-analyzed environmental exposure data that is directly associated with health outcomes should be used for risk assessment*. [emphasis in original].[91]

Follow-up on chrysotile-exposed textile workers has shown that thin fibers significantly contribute to the risk of contracting asbestos-related lung cancer and mesothelioma.[160,161] Roggli admits that his method undercounts chrysotile fibers, but claims that TEM undercounts amosite.[124] While it is undisputed that amphibole is a cause of mesothelioma, Roggli's flawed method—which systematically undercounts chrysotile—supports his conviction that chrysotile has not contributed to mesothelioma causation in certain individuals or more generally in those exposed to certain chrysotile-tremolite products. His technique is biased in a direction that supports his argument.

Since chrysotile fibers are biopersistent in the pleura and not the lung, while amphiboles are biopersistent in the lung and often fail to reach the pleura, over time chrysotile levels will decrease in the lung. Using a one year half-life for chrysotile and a 20-year half-life for amphiboles, the amount of chrysotile remaining in the lung 30 years after exposure would be 1 billionth of what was inhaled, while almost 30% of the amosite would still be present in the lung.

Elsewhere, Roggli has undermined the validity of Srebro, Roggli, and Samsa's conclusions on the question of chrysotile causation in general and the contribution of chrysotile-containing products (like asbestos brakes) to the induction of a mesothelioma in any particular individual. In a 2000 paper, he states that, "Fiber burden studies do not accurately reflect past exposures to chrysotile."[162] At that point, he maintained that these studies "afford limited information regarding the role of chrysotile asbestos-related lung cancer since chrysotile is broken down in and removed from the lung. And long, thin, greater than 5 micron chrysotile fibers are not readily detectable by our technique."[162]

4. Did the counting method count short fibers?

Roggli's method fails to count fibers that are shorter than 5 μm in length, leading to further undercounting of chrysotile and over-emphasis on the role of amphiboles in causation.

All scientists who have published on pleural fiber counts find short fibers to be the most common and often the only pleural fiber.[105,163,164] The combined effect of using an insensitive instrument and the deciding to not count short fibers is dramatic. In one blinded cross-laboratory comparison on the same patient, Dodson et al. found 84 chrysotile fibers while Roggli reported only one.[164]

5. Was a standard procedure used for all cases and controls?

Srebro, Roggli, and Samsa used two different methods to prepare tissue specimens. They described these methods as follows:

> AB counts for all 19 control cases and for 6 mesothelioma cases (Cases 2 and 13 to 17) were quantified using the technique of Smith and Naylor for approximately 5-g samples of lung tissue. In six mesothelioma (Cases 3, 5, 6, 7, 10, and 18) limited lung tissue (<1.0 g) was available. For these cases, our laboratory developed a hypochlorite digestion procedure [modified from Williams et al].[130]

Srebro, Roggli, and Samsa then cited a 1986 paper (by Roggli, Pratt, and Brody) that compared the valid-

ity of the two techniques, stating "on average, [the modified techniques values were] within 10% of values determined by the Smith and Naylor procedure."[143] However, the earlier paper's comparison related only to asbestos bodies. Srebro, Roggli, and Samsa failed to report the range of counts for their subjects, which showed that values differed by 10 fold (0.31–3.53) between techniques. This ten-fold range indicates that the different techniques are not comparable.

6. Can fiber counting determine how long a fiber has been in the lung?

Pathologic evaluations cannot determine when a fiber entered the body and recent exposures do not contribute to cancer formation.[136]

7. Was crocidolite found in cases or controls?

Srebro, Roggli, and Samsa did not find any crocidolite in any patients, but Roggli misreports this fact in subsequent publications. Srebro, Roggli, and Samsa never found crocidolite, but in every subsequent publication of his data, Roggli lists the amosite counts as "AC" (amosite and crocidolite). This is misleading, because Roggli's SEM method will miss the vast majority of crocidolite fibers, which are too thin to be seen at Roggli's preferred microscope setting.[165]

8. Was a standard method used to compare cases to his "controls"?

Srebro, Roggli, and Samsa's paper states that their "study demonstrated that approximately one-third (6 of 18) of the mesothelioma cases have asbestos fiber burdens greater than 95% of the control levels" They concluded that these cases were caused by asbestos.[130] The authors fail to explain what this is 95% *of*. However, in recent testimony, Roggli explained: "There was one control case which we eventually threw out because we discovered through work that Dr. Srebro did that that person had an occupational exposure. That's the 95 percent."[144]

A close reading of the paper shows that their comparison was ad hoc:

- They discounted values for cases if there they only found one fiber.

Srebro, Roggli, and Samsa wrote that in mesothelioma cases 6, 7, and 14, amosite fibers were detected but were not clearly above background level because the calculated values were based on a single fiber detected (*versus* none detected in controls)[italics in original]." Srebro, Roggli, and Samsa classified all these cases as of "uncertain etiology."[130] Roggli recently testified on this issue, saying, "[W]e typically require 2 [fibers] to be an unambiguous result. Two fibers. One fiber even though it's more than we found in our control still might be just a matter of chance and [an] ambiguous result."[144]

- In contrast, they did not discount values for "controls" if the fiber estimate was based on only one fiber.

Tremolite fiber counts in controls 20, 22, 24, 25, 31, and 35 are all based on finding a single fiber. Two of these "single fiber" controls (20 and 24) had the highest tremolite values for all "controls" (2540 and 1770, respectively). In all subsequent evaluations, Roggli only attributes causation to tremolite exposure if levels are above these two "one fiber" controls. Except for a single chrysotile fiber found in controls 29 and 34, Srebro, Roggli, and Samsa found no chrysotile in any "controls," but nevertheless use these as a basis for comparison with case chrysotile levels.[130]

Roggli's inconsistent exclusion of fiber counts based on the finding of a single fiber has an important impact on his conclusions. Roggli discounts brake exposure as a cause for mesothelioma in case 5 in his 2003 brake study, based on two of the single fiber tremolite "control" cases (20 and 24).[129] Otherwise, case 5 has higher tremolite levels than all but one of his "controls" (and no amphiboles). This would meet his original criteria of determining that asbestos caused a mesothelioma if he finds a fiber count that exceeds those reported in 95% of his controls.

Finkelstein reviewed Butnor, Sporn, and Roggli's comparison of brake worker and control fiber counts and elegantly showed that the authors performed an incorrect statistical analysis by comparing medians.[166] Finkelstein's correct analysis revealed that cases had significantly more tremolite than the 1995 "controls" (Figure 11). Roggli responded to Finkelstein's critique by stating, "What Dr. Finkelstein seems to ignore is that in 'every case' with an elevated level of chrysotile or non-commercial amphibole fibers, there was also an elevated level of commercial amphibole fibers (amosite or crocidolite)."[167] However, Roggli found no amosite or crocidolite in either case 4 or 5. Case 4 had no asbestos bodies, and according to Roggli chrysotile can form asbestos bodies.[141]

- Srebro, Roggli and Samsa discounted fibers found when the result was positive but below the highest detection limit in any of the "controls."

Srebro, Roggli and Samsa discounted the results in case 2 because they claimed that they used a larger tissue sample size (5 g) than that used for some of the other cases, and as a result had a lower detection limit than all of the controls. However, case 2's tissue sample was the same size as that of all the "controls." The paper reports that analysis was conducted with "approxi-

mately 5-g samples" for all controls and "six mesothelioma cases (Cases 2 and 13 to 17)." Although case 2 worked at shipyards during WWII and had amosite fibers in his lung, Srebro, Roggli and Samsa classified it as of "uncertain etiology."

- Srebro, Roggli and Samsa discounted chrysotile as a cause even if the levels were greater than 95% of all "controls."

Srebro, Roggli, and Samsa wrote,

> One additional case (Case 17) demonstrated a chrysotile fiber count greater than 95% of the levels for the controls but not greater than all of the control values. However, SEM is not as sensitive at detecting chrysotile fibers, which are frequently less than 5 μm in length and thinner than 0.1 μm.

The authors here paradoxically conclude that although this case met their criteria for assigning asbestos causation—and indeed would have exceeded those criteria if their own methodology had been more accurate—that the role of asbestos causation in this case was nevertheless "uncertain." This appears to be an admission that their methodology is fatally flawed, at least with respect to the evacuation of chrysotile as a cause of mesothelioma.

Roggli has changed his criteria for comparing new cases to his historical controls on several occasions. In 2002, he compared new cases to the highest detection level for chrysotile in any "background case" (control), which was 2540 uf/gwt (see Table 2).[128] In 2003, he compared new cases to the highest actual recorded value for chrysotile (1000 uf/gwt) but continued to compare tremolite to the highest detection limit in any "control."[129] He also dropped the 95% comparison after the original paper, and since 2002 he appears to require that the case have fiber levels that "exceed all controls" to qualify as a potential asbestos-caused case.[136]

As noted at the beginning of this section and consistent with his interpretation of cases 4 and 5, Roggli has testified that he would not attribute causation to brake exposure even if the patient had elevated lung chrysotile and/or tremolite levels, essentially admitting that he does not follow his own methodology if the results conflict with his prior opinion that brake exposures cannot cause mesothelioma.[136]

9. Are there problems with the approach to attribution of causation in individual cases?

Roggli has asserted that lung fiber counts must exceed "background or control" to establish causation.[124] This presumes that there is a threshold for asbestos induction of mesothelioma and that the threshold is at or below "background." However, Roggli himself has stated that "no threshold has been identified for asbestos exposure below which mesothelioma will not occur."[124] In the lung, there is no qualitative difference between asbestos fibers from "background" exposure and those from asbestos products. If asbestos can cause mesothelioma, then fibers from "background" or ambient air can cause mesothelioma.[10,12,26–28,160,168–174] Therefore, there is no reason to exclude them as causes of mesothelioma. In this issue, Azuma et al. provide further evidence that there is no threshold for asbestos induction of mesothelioma, as Roggli has done in an affidavit:

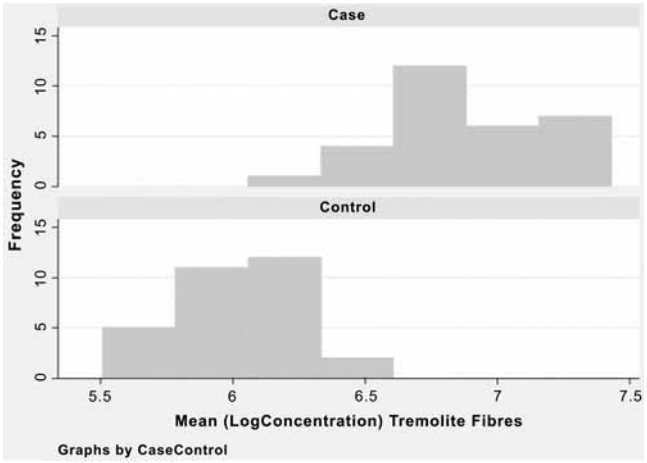

Figure 11—Tremolite levels of brake workers vs. controls, reproduced with permission from: Finkelstein MM. Asbestos fibre concentrations in the lungs of brake workers: another look. Ann Occup Hyg 2008;52:455-61.

> It is also my opinion that it is the total dose of asbestos, regardless of fiber type, that the patient experiences that causes the disease, . . . It is further my opinion that each and every exposure to asbestos that an individual with mesothelioma experienced in excess of a background level is a substantial contributing factor in the development of the disease.[131]

If a threshold for asbestos induction of mesothelioma exists, and lung fiber burden drives pleural levels, then "background" exposures that reach the pleura will be added to "occupational" exposures and contribute to induction of the cancer and/or promote its growth. According to Roggli, it is the combined total dose of asbestos which causes mesothelioma.[136] Roggli believes that seven fiber-years of chrysotile exposure are required to cause mesothelioma, except for household or environmental exposure.[136] However, if two products each contribute one-half of the dose necessary (in Roggli's view) to cause the mesothelioma, he will not attribute any role in causation to either one.[127] This reasoning has no scientific basis. When it comes to legal causation, which calls for a contribution to be "significant" at some comparative exposure level, it may be reasonable to conclude that an exposure was trivial (for example 1 fiber out of one billion), but Roggli's position that an exposure that constitutes 50% of the sufficient dose is trivial is erroneous. Interestingly, in some

TABLE 3 Various Presentations of the Same Data on Control Fiber Counts Originally Reported by Srebro, Roggli, and Samsa (1995)[130]

Paper	AB/gm	Total Fibers/gm	Crocidolite	Amosite	Amosite/ Crocidolite (AC)	Tremolite, Actinolyte, Anthophylite (TAA)	Chrysotile	Number of Controls
Original 1995 actual data[130]	3 (<0.2–22)	Median: 2990 Range: 420–12,700 Mean: 4330	Reported as "not identified" and 0	Median: ND* Mean: ND*	No data presented as AC combination	Median: ND* Mean: 470 Range: <170–2540	Mean: 75 Median: ND*	19
2000[161]	2.9 (0.2–22)	Median: <600 Range: <170–<2540		Amosite levels reported as amosite and crocidolite (AC)	Median: <600 Range: <100–2540	Median: <600 Range:<170–<2540	Median: <600 Range: <100–<2540	19
2002[128]	3 (0.2–22)	Not reported	Not reported	Amosite levels reported as AC	Median: <600 Range: <100–<2540	Median: <600 Range: <170–<2540	Median: <600 Range: <100–<2540	19
2003[129]	3 (<0.2–22)	Not reported		Amosite levels reported as AC	Median: <600 Range: <100–<2540	Median: <600 Range: <170–2540	Median: <600 Range: <100–1000	20

Shading shows data that is incorrect or different from other reports of the same data.
*ND= below detection Limits

circumstances Roggli does not adhere to his own seven fiber-years of exposure rule. For instance, Roggli will attribute asbestos causation in household exposure mesothelioma cases despite the fact that he doesn't "know any way in a household-contact case to apply the [his] seven to ten fiber cc year rule [for chrysotile] or the .01 fiber cc rule for amphibole fibers."[148]

Roggli's position is different from the legal standard as described by Keeton and Prosser, that:

> In products liability involving asbestos, where the plaintiff has sufficiently demonstrated both lung disease resulting from exposure to asbestos and that the exposure was to the asbestos products of many different, but identified, suppliers, no supplier enjoys a causation defense solely on the ground that the plaintiff would probably have suffered the same disease from inhaling fibers originating from the products of other suppliers.[175]

and:

> When the conduct of two or more actors is so related to an event that their combined conduct, viewed as a whole, is a but-for cause of the event, and application of the but-for rule to them individually would absolve all of them, the conduct of each is a cause in fact of the event.[175]

10. Is the assumption that automobile mechanics do not work with brake and/or clutch products that contain amosite or crocidolite correct?[136]

In an introduction to a 1968 paper that reported asbestos exposures in brake mechanics, Ford Motor Company's industrial hygienists wrote, "The brake linings in current use may contain 40 to 60% asbestos when manufactured—the asbestos being normally in the chrysotile from, and occasionally in the amosite form."[176] Borg Warner used crocidolite in some automobile clutches and brake bands.[177] Maremont used crocidolite in its automobile parts operation which produced brakes, clutches, and mufflers.[178] Several brake and clutch manufacturing companies purchased amphibole fibers from the North American Asbestos Corporation between 1954 and 1974.[179,180] These include Bendix, Victor, Raybestos-Manhattan and Delco Moraine, a General Motors subsidiary. In a government review, Blau reported that manufacturing companies had used amosite and crocidolite in brakes.[181] Some brake patents called for the use of either crocidolite or amosite (see Table 5).

11. Did researchers consider evidence of synergy between chrysotile and amosite?

Roggli has attributed causation solely to amphibole fiber, irrespective of the chrysotile or tremolite count, unless he can estimate a 7–10 year chrysotile exposure.[148]

TABLE 4 "Background" vs. Mesothelioma Patients, Adapted from Srebro, Roggli, and Samsa[130]

Patient	Age/Sex	Diagnosis[a]	Occupation[b]	Smoke[c]	AB/g[d]	Uncoated Fibers (UF/g)[e] AMOS[f]	TAA[g]	CHRYS[h]
Mesothelioma Cases								
1	56/M	BPL-R	Painter/spackler	70 PY	<15.0	<4060	16,160	<4060
2	60/M	EPE	Shipyard worker (WWII)	S	14.4	60	60	<60
3	/M[i]	EPL-R	Brake repair		14.0	1440	2170	720
4	60/M	BPE	Truck driver (vermiculite)	132 PY	<12.4	1360	<680	<680
5	68/F	DPL-L	No history of exposure		9.8	<660	1980	<660
6	52/M	SPL-R	Ship engine room/ brake repair	40 PY	7.1	280	550	<280
7	65/M	EPL-R	Navy	52 PY	6.4	1070	7490	4280
8	77/M	SPL-R	Sales		<5.2	<390	1570	<390
9	68/M	EPL-L	Railroad machinist	35 PY	<5.0	<510	<510	<510
10	67/M	BPL-R	Merchant marine	NS	<3.0	<1080	1080	<1080
11	61/M	EPL-R	Carpenter	20 PY	<3.0	<310	<310	<310
12	65/M	BPC	Weigh station employee		2.8	<690	690	<690
13	58/F	DPL-L	Teacher aide (building exposure)		2.8	<870	4330	<870
14	66/M	BPL-L	Brake repair	XS (16 yr)	2.6	120	240	<120
15	57/F	PL	Wife of shipyard worker	40 PY	2.0	<4860	9720	<4860
16	45/M	PE	Attorney (building exposure as student)	24 PY	1.0	<1220	1220	<1220
17	53/M	EPL-L	Accountant (building exposure)		<0.2	<640	1270	640
18	45/M	EPL-R	No history of exposure		<3.0	<440	<440	<440
Control Cases								
19	64/M	MI	Hospital, farmer	NS	22.0	<990	<990	<990
20	76/M	ALL	Manual labor		19.6	<1770	1770	<1770
21	40/M	GBM	Manual labor		9.7	<100	210	<100
22	61/M	Esophageal cancer	Truck driver		8.9	<400	400	<400
23	64/M	Melanoma	Air force	NS	7.4	<570	<570	<570
24	64/M	Alzheimer's	NA		5.4	<2540	2540	<2540
25	59/M	Gastric cancer	Guard	NS	3.5	<470	470	<470
26	53/M	ABE	Air force	NS	3.0	<760	<760	<760
27	71/M	CLL	Music	50 PY	3.0	<300	890	<300
28	61/M	CAD	Garage owner		2.8	<170	<170	<170
29	51/M	Cirrhosis	Manual labor		2.2	<1000	<1000	1000
30	53/M	Hepatoma	Spinning mill		2.2	<650	1310	<650
31	28/M	ALL	Air Force	NS	1.0	<960	960	<960
32	36/M	Pancreatitis	NA		1.0	<790	<790	<790
33	67/M	GBM	NA		0.8	<430	<430	<430
34	71/M	ESRD	Business supply store	XS (pipe)	0.4	<510	<510	510
35	64/M	MI	Electrical engineer	NS	0.4	<370	370	<370
36	85/M	CVD	Manual labor	XS	0.2	<600	<600	<600
37	63/M	AAA	Hospital aide		<0.2	<600	<600	<600

Key (Shading not in original)

= Case with single amosite fiber dismissed as "uncertain etiology" (Cases 2, 6, 14)

= Case with counts below or equal to controls dismissed as "uncertain etiology" (Cases 5, 8 – 12)

= Case with chrysotile levels above 95% of controls dismissed as "uncertain relationship" to asbestos (Case 17)

= Causal case

[a]AAA, abdominal aortic aneurysm; ABE, acute bacterial endocarditis; ALL, acute lymphoblastic leukemia; B, biphasic; CAD, coronary artery disease; CLL, chronic lymphocyctic leukemia; CVD, cardiovascular disease; D, desmoplastic; E, epithelial; ESR, end-stage renal disease; GBM, glioblastoma multiforme; L, left; MI, myocardial infarction; PC, pericardial; PL, pleural; PE, peritoneal; R, right; S, sarcomatous.
[b]NA, not available.
[c]NS, nonsmoker; PY, pack-years; S, smoker (unknown duration/quantity); XS, ex-smoker.
[d]AB/g, asbestos bodies per gram of wet lung by light microscopy.
[e]UF/g, total uncoated fibers ≥5 μm (in length) per gram of wet lung by scanning electron microscopy.
[f]AMOS, amosite.
[g]AA, tremolite, anthophylite, actinolite.
[h]CHRYS, chrysotile.
[i]No age reported by Srebro, Roggli, Samsa

TABLE 5 United States Brake Patents which Include Amphibole Fibers

Patent Number	Comment	Year
2227424	Johns Manville patent for a brake lining includes the following description: "In the friction materials of the present invention, heat resistant fibers of chrysotile, amosite, or other variety of asbestos fibers adapted for use in friction materials are used as the fibrous component associated with the friction composition."	1941
2943010	Raybestos patent for laminated composite fabric break lining in which "examples of the types of asbestos fibers which are suitable for use in this process are chrysotile, crocidolite or amosite."	1960
3624234	Raybestos-Manhattan's patent for a friction material for use in "automotive and industrial brakes or clutches" called for typical materials including 20% anthophylite and 20% chrysotile asbestos.	1969

Roggli's fiber year estimates are based on the QAMA mine studies, whose exposure monitoring never measured fibers and whose results were no better than guesses.[94] He has not examined the validity of this data.*** McDonald, the designer of the Quebec Asbestos Mining Association studies, in an unpublished paper made public in the Tobacco Company archives, wrote:

> . . . converting from particles to fibers a difficult and dubious operation. Even in chrysotile mining and milling, the range of conversion ratios is at least 40-fold. A problem of similar magnitude concerns the equivalence in fiber terms of measurements made in the general environment, nearly all of which are gravimetric and usually expressed in nanograms per cubic meter $(ng/m)^3$. The conversion factor relating mass to optical fiber concentration had a range of 5–150 and probably varied with fibre type.[182]

Roggli claims the 7–10 fiber-year dose is the dose at which the mesothelioma rate doubles in miners.[133] However, "doubling doses" are not required to establish a cause-effect relationship.[183,184] This is particularly true when there is pathologic or historical evidence of exposure to asbestos.

At any rate, as described above there is substantial evidence that chrysotile and amphiboles act super additively or synergistically even if chrysotile itself is not a complete carcinogen.[126] Therefore, it is more likely that chrysotile is a contributing cause of mesothelioma when amphiboles are present as well. Roggli agrees and has testified that, "An amosite and chrysotile insulation worker would have about twice the risk of an amosite factory worker or crocidolite Australian mines [*sic*] or chrysotile amphibole textile factory workers."[127] This opinion seems to have had no impact on his assessment of the potential contribution of chrysotile in American workers, almost all of whom have been exposed to both.

Roggli has stated that the chrysotile-mesothelioma relationship has no threshold.[127] This is inconsistent with his position that "background" exposures do not contribute to cause mesothelioma.[127] There are rare mesotheliomas for which no point source of exposure in excess of that in the general environment can be identified. Such cases can be attributed to general environmental "background" exposure, leaving aside the unsolvable issue of whether there exist any spontaneous mesotheliomas entirely unrelated to asbestos (this impossible to establish even in childhood cases since there is neonatal exposure to asbestos).

12. Do lung fiber types and levels predict or "drive" pleural levels?

In 1992, Roggli asserted that, "[T]here is growing consensus that the fiber burdens that accumulate in the lung are the primary determinant of later disease."[185] Wagner and Pooley offer the hypothesis that

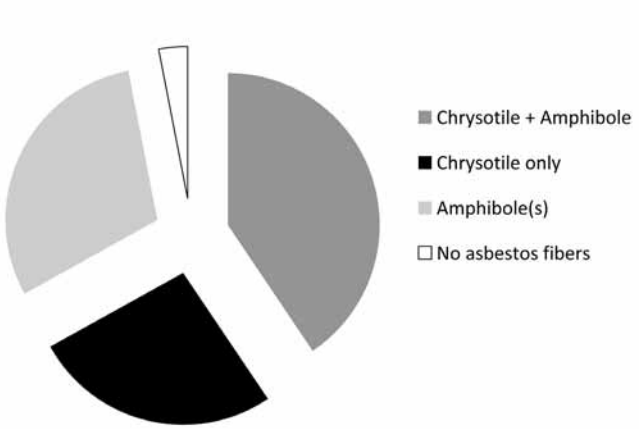

Figure 12—The predominant fibers in the lung, based on data from: Suzuki Y, Yuen SR. Asbestos tissue burden study on human malignant mesothelioma. Ind Health 2001;39:150-60. Comparison to Figure 13, which shows pleural fibers in the same patients, establishes that chrysotile goes to the pleura, while amphiboles stay in the lung.

***Roggli's claim that at least 7 f/cc/years of exposure are required to attribute a mesothelioma to asbestos is based a "pocket risk assessment" relying on exposure and disease data from the QAMA studies. When faced with evidence that QAMA data was potentially unreliable, Roggli agreed that there was the potential for a "garbage-in, garbage out" phenomenon.[135]

"those diseases associated with exposure to mineral fiber are due to the fiber retained in the lungs," but do not address the relationship between lung and pleural fiber burdens.[186]

Churg's finding contradicts Roggli's assertion, as he notes:

> A different approach is to examine fiber burden in lung tissue of patients with mesothelioma. This procedure ensures that mesotheliomas induced by occult amosite or crocidolite exposure will be detected as such, but it suffers from unknown patterns of fiber clearance over time and also from the fact that, while amphibole accumulates readily in lung, chrysotile does not.[187]

13. Is fiber counting reliable?

Fiber counting is unreliable due to wide intra- and inter-laboratory variability. It is a non-standard technique that cannot be used to determine causation in individual cases.

Roggli has written:

> The wide variety of preparative techniques and analytical methodologies that have been employed by various investigators make it difficult to extrapolate results from one laboratory to another. The actual analytical result obtained on any one sample can be profoundly influenced by the steps employed in the analytical procedure. Interlaboratory comparison trials demonstrate that striking differences can occur among laboratories even when the same sample is analyzed.[152]

He further states,

> In addition to inter-laboratory variation, intralaboratory variation can occur, which may be due either to changes in a laboratory's procedures over time, or to variation in fiber content from one site to another within the lung. Morgan and Holmes have reported a five to tenfold site-to-site variation based on analyses of multiple samples from a single lung using phase contrast light microscopy.[152]

There is also sampling variability. In testimony, Roggli has agreed that the numbers of fibers can vary from site to site within the lung by a factor of anywhere from two to five.[127] In addition, he has agreed that there are as many as ten short chrysotile fibers for every one he can count >5µm.[127] Few scientists have the temerity to overlook test variability of this magnitude to conclude anything about a scientific theory or individual causation—except in the case of a finding that in and of itself disproves a theory (identification of a black swan disproves the theory that all swans are white). This 15,000% lack of precision would appear to violate the Daubert standard for "reliability."[188]

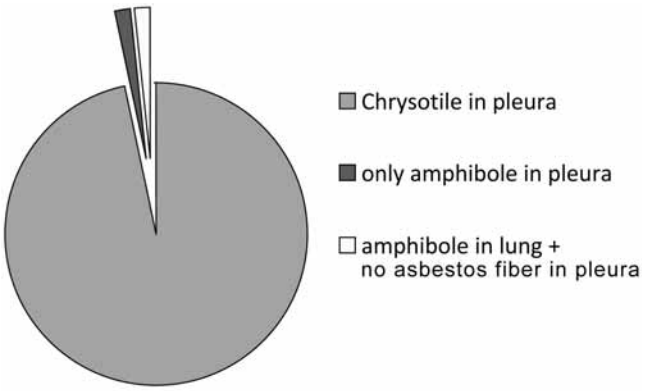

Figure 13—The predominant fibers in the pleura, based on data from: Suzuki Y, Yuen SR. Asbestos tissue burden study on human malignant mesothelioma. Ind Health 2001;39:150-60. Comparison to Figure 12, which shows lung fibers in the same patients, establishes that chrysotile goes to the pleura, while amphiboles stay in the lung. Amphiboles were found in the pleura in 23.5% of cases.

14. Do fibers differentially locate to the pleura in a way that lung counts systematically underestimate chrysotile pathogenicity?

Short thin chrysotile fibers are the most commonly found fiber in tumors and in the pleura of patients with mesothelioma, asbestosis, and lung cancer (see Figures 12 and 13).[104] Roggli's method systematically underestimates or misses chrysotile exposure while overemphasizing amphibole exposures. Except for workers who fabricated Unibestos products and some individuals who had only environmental tremolite exposures (e.g. Libby, Montana residents), all exposures to US residents included at least some chrysotile exposure. Because Roggli systematically underestimates chrysotile exposures and because chrysotile is always overrepresented in the pleura compared to the lung, Roggli's results often attribute causation to the wrong fiber and they are almost always misleading. In this case, more information (lung fiber count versus no count) is worse than no information—it is misleading. Roggli has also claimed that pleural tumor fiber counts are unreliable without stating why this is so.[136] This is only true if fibers appear to absent, as the tumor can dilute the fiber concentration (absence of evidence is not evidence of absence). If fibers are found in a tumor or plaque, however, this is always important.[109]

In contrast to Teta et al. and Price and Ware, Azuma et al. consider fiber burdens, exposure data, and mesothelioma rates in their study design and thus provide evidence that low exposures to asbestos cause "background" cases. Their results are comparable to those of Iwatsubo et al.,[174] Rödelsperger et al,[189†††] Mag-

†††Rödelsperger used lung cancer controls, thus conflating his analysis of relative potency of different fibers, but his data on the exposed population is consistent with a no-threshold dose-response model.

nani et al.,[34,35] and Maule et al.[36] Human pathologic studies of pleural tissue that do not exclude "inconvenient" data buttress this conclusion.

CONCLUSION

This is the first peer-reviewed publication of which we are aware that "peer reviews" testimony. In our view, review of the presentation of scientific ideas that are presented in court and at hearings is at least as important as peer review of published research and academic reviews. We look forward to publishing other similar reviews in the future and encourage our readers to submit them.

I want to thank David Madigan, PhD for providing the statistical analysis of data from Srebro et al., William Longo, PhD for use of his data, and Susanna Bohme, PhD for her editorial advice.

References

1. JAMA Editor Calls Critic a 'Nobody and a Nothing.' 2009. [cited 2009 March 3] Available from: http://blogs.wsj.com/health/2009/03/13/jama-editor-calls-critic-a-nobody-and-a-nothing/.)
2. Winstein K, Armstrong D. "Top Pain Scientist Fabricated Data in Studies", Hospital Says. Wall Street Journal. 2009 Mar11,2009.
3. Robinson RG, Jorge RE, Moser DJ, et al. Escitalopram and problem-solving therapy for prevention of poststroke depression: a randomized controlled trial. JAMA. 2008;299:2391-400.
4. DeAngelis CD, Fontanarosa PB. Conflicts Over Conflicts of Interest. JAMA 2009;301:2.
5. Robinson RG, Arndt S. Incomplete financial disclosure in a study of escitalopram and problem-solving therapy for prevention of poststroke depression. JAMA. 2009;301:1023-4.
6. Azuma K, Uchiyama I, Chiba Y, Okumura J. Mesothelioma Risk and Environmental Exposure to Asbestos; Past and Future Trends in Japan. Int J Occup Environ Health. 2009;15:6.
7. Hessell P. Inaccuracies in Article on Mesothelioma in Brake Workers [Letter]. Int J Occup Environ Health. 2009;15.
8. Welch L, Anderson HA, Balmes J, et al. Research on Mesothelioma from Brake Exposure: Corporate Influence Remains Relevant Concern [Letter]. Int J Occup Environ Health. 2009;15.
9. Mark EJ, Yokoi T. Absence of evidence for a significant background incidence of diffuse malignant mesothelioma apart from asbestos exposure. Ann N Y Acad Sci. 1991;643:196-204.
10. Camus M, Siemiatycki J, Meek B. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. N Engl J Med. 1998;338:1565-71.
11. Egilman D, Fehnel C, Bohme SR. Exposing the "myth" of ABC, "anything but chrysotile": a critique of the Canadian asbestos mining industry and McGill University chrysotile studies. Am J Ind Med. 2003;44:540-57.
12. Gustavsson P, Nyberg F, Pershagen G, et al. Low-dose exposure to asbestos and lung cancer: dose-response relations and interaction with smoking in a population-based case-referent study in Stockholm, Sweden. Am J Epidemiol. 2002;155:1016-22.
13. Pan XL, Day HW, Wang W, et al. Residential proximity to naturally occurring asbestos and mesothelioma risk in California. Am J Respir Crit Care Med 2005;172:1019-25.
14. Price B, Ware A. Mesothelioma trends in the United States: an update based on Surveillance, Epidemiology, and End Results Program data for 1973 through 2003. Am J Epidemiol. 2004; 159:107-12.
15. Teta MJ, Mink PJ, Lau E, et al. US mesothelioma patterns 1973-2002: indicators of change and insights into background rates. Eur J Cancer Prev. 2008;17:525-34.
16. Price B. Analysis of current trends in United States mesothelioma incidence. Am J Epidemiol. 1997;145:211-8.
17. Patel AV, Bogner PN, Klippenstein D, Ramnath N. Malignant pleural mesothelioma after household exposure to asbestos. J Clin Oncol. 2008;26:5480-3.
18. Miller A. Mesothelioma in household members of asbestos-exposed workers: 32 United States cases since 1990. Am J Ind Med. 2005;47:458-62.
19. Leigh J, Davidson P, Hendrie L, Berry D. Malignant mesothelioma in Australia, 1945-2000. Am J Ind Med. 2002;41:188-201.
20. Castleman BI, Berger SL. Asbestos : medical and legal aspects. 5th ed. New York, NY: Aspen Publishers; 2005.
21. Schneider J, Rodelsperger K, Bruckel B, et al. Pleural mesothelioma associated with indoor pollution of asbestos. J Cancer Res Clin Oncol. 2001;127:123-7.
22. Schneider J, Straif K, Woitowitz HJ. Pleural mesothelioma and household asbestos exposure. Rev Environ Health. 1996;11:65-70.
23. Health and Safety Executive. Mesothelioma Occupation Statistics: Male and Female Deaths Aged 16-74 in Great Britain. Suffolk: HSE Books; 2006.
24. Egilman DS, Billings MA. Abuse of epidemiology: automobile manufacturers manufacture a defense to asbestos liability. Int J Occup Environ Health. 2005;11:360-71.
25. Seidman H, Selikoff IJ, Hammond EC. Short-term asbestos work exposure and long-term observation. Ann N Y Acad Sci. 1979;330:61-89.
26. Lin RT, Takahashi K, Karjalainen A, et al. Ecological association between asbestos-related diseases and historical asbestos consumption: an international analysis. Lancet. 2007;369:844-9.
27. Welch LS. Asbestos exposure causes mesothelioma, but not this asbestos exposure: an amicus brief to the Michigan Supreme Court. Int J Occup Environ Health. 2007;13:318-27.
28. Welch LS, Acherman YI, Haile E, Sokas RK, Sugarbaker PH. Asbestos and peritoneal mesothelioma among college-educated men. Int J Occup Environ Health. 2005;11:254-8.
29. Musti M, Pollice A, Cavone D, Dragonieri S, Bilancia M. The relationship between malignant mesothelioma and an asbestos cement plant environmental risk: A spatial case-control study in the city of Bari (Italy). Int Arch Occup Environ Health. 2009; 82:489-97.
30. Berry M. Mesothelioma incidence and community asbestos exposure. Environ Res. 1997;75:34-40.
31. Greenberg M, Davies TA. Mesothelioma register 1967-68. Br J Ind Med. 1974;31:91-104.
32. Mollo F, Magnani C. European multicentric case control study on risk for mesothelioma after non-occupational (domestic and environmental) exposure to asbestos. Med Lav. 1995;86:496-500.
33. Magnani C, Ivaldi C, Botta M, Terracini B. Pleural malignant mesothelioma and environmental asbestos exposure in Casale Monferrato, Piedmont: Preliminary analysis of a case-control study. Med Lav. 1997;88:302-9.
34. Magnani C, Agudo A, Gonzalez CA, et al. Multicentric study on malignant pleural mesothelioma and non-occupational exposure to asbestos. Br J Cancer. 2000;83:104-11.
35. Magnani C, Dalmasso P, Biggeri A, et al. Increased risk of malignant mesothelioma of the pleura after residential or domestic exposure to asbestos: a case-control study in Casale Monferrato, Italy. Environ Health Perspect. 2001;109:915-9.
36. Maule MM, Magnani C, Dalmasso P, et al. Modeling mesothelioma risk associated with environmental asbestos exposure. Environ Health Perspect. 2007;115:1066-71.
37. Vianna NJ, Polan AK. Non-occupational exposure to asbestos and malignant mesothelioma in females. Lancet. 1978;1:1061-3.
38. Chahinian AP, Pajak TF, Holland JF, Norton et al. Diffuse malignant mesothelioma. Prospective evaluation of 69 patients. Ann Intern Med. 1982;96:746-55.
39. Kilburn KH, Lilis R, Anderson HA, et al. Asbestos disease in family contacts of shipyard workers. Am J Public Health. 1985;75:615-7.
40. Strickler HD, Goedert JJ, Devesa SS, Lahey J, Fraumeni JF, Jr., Rosenberg PS. Trends in U.S. pleural mesothelioma incidence rates following simian virus 40 contamination of early poliovirus vaccines. J Natl Cancer Inst. 2003;95:38-45.
41. Health and Safety Commission. Health and Safety Statistics 1998/99. Sudbury: HSE Books; 1999.
42. Australian Safety and Compensation Council. Preparing an Estimate of the National Pattern of Exposure to Asbestos in Cases of Malignant Mesothelioma. Australian Government Department of Education, Employment and Workplace Relations; 2008 June.

43. Mirabelli D, Stura A, Gangemi M, Bertolotti M, Maule MM, Magnani C. [Incidence of malignant mesothelioma in Piedmont, 1990-2001]. Epidemiologia e prevenzione. 2007;31:132-8.
44. Roggli VL, Oury TD, Moffatt EJ. Malignant mesothelioma in women. Anat Pathol. 1997;2:147-63.
45. Roggli VL, Sharma A, Butnor KJ, Sporn T, Vollmer RT. Malignant mesothelioma and occupational exposure to asbestos: A clinicopathological correlation of 1445 cases. Ultrastruct Pathol. 2002;26:55-65.
46. World War II in the San Francisco Bay Area [Internet]. National Park Service, US Department of the Interior; n.d. Tending the Home Front: The Many Roles of Bay Area Women During WWII [cited 2009 Feb 27]. Available from: http://www.nps.gov/history/Nr/travel/wwIIbayarea/womenatwar.htm.
47. Hassan A. Shipyard Day Care Centers of World War II: The Kaiser Experiment 2009 [Internet].[cited 2009 April 2]. Available from: http://wwiishipyarddaycare.tripod.com/intro.htm.
48. Wikipedia [Internet]. Wikimedia Foundation. [2009]. Asbestos [cited 2009 February 20]. Available from: http://en.wikipedia.org/wiki/Asbestos/.
49. Greenberg M. Asbestos Exposures to Women. Personal Communication to: David Egilman. 2009.
50. Brown DP, Dement JM, Okun A. Mortality patterns among female and male chrysotile asbestos textile workers. J Occup Med. 1994;36:882-8.
51. Merewether ERA, Price CW. Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry. HM Stationary Office; 1930.
52. Finkelstein MM. A study of dose-response relationships for asbestos associated disease. Br J Ind Med. 1985;42:319-25.
53. Saldivar A, Soto V. Asbestos in the United States: Occurrences, Use and Control 2008 [Internet]. Proquest; 2008. [cited 2009 February 26]. Available from: http://www.csa.com/discoveryguides/asbestos/review.pdf.
54. Roggli V. Deposition. Ralph Stanley vs. Anderson et al., Civil Action No. 03-C-9600, Kanawa County, WV; 2005:80.
55. Schneider A. U.S. imports of asbestos brake material are on rise. St Louis Post Dispatch. 2003 Oct 26.
56. Seminario P. Testimony Before the Senate Employment and Worker Safety Subcommittee of Health, Education, Labor and Pensions Committee Hearing on "Is OSHA Working for Working People." 2007 April 26. Available from: http://www.aflcio.org/issues/safety/upload/SeminarioOSHA20070426.pdf.
57. Morton S. Deposition Testimony. Morton vs Owens Illinois et al, Newport News Virginia. 2006.
58. United States Congress. Hearing Transcript. Oversight Of The Navy Department's Enforcement Of OSHA; 1983.
59. Glass A. (President, Steelworkers Union, Newport News Shipyard) Personal Communication to: Robert Hatten R; 2009.
60. Harries PG. Asbestos dust concentrations in ship repairing: A practical approach to improving asbestos hygiene in naval dockyards. Ann Occup Hyg. 1971;14:241-54.
61. Garcia A. OSHA Violations Indicate Enforcement of Asbestos Standard [Internet] Future Environment Designs; 2008 September 05 [cited 2009 Feb 20]. Available from: http://futureenv.blogspot.com/2008/09/osha-violations-indicate-enforcement-of.html
62. Saltzman J. Trainer in asbestos removal convicted. Boston Globe. 2008 November 21.
63. Oversight Report: OSHA's Failure To Monitor And Enforce Asbestos Regulations In Auto Repair Shops. Washington, DC: U.S. House of Representatives, Congressman Dennis Kucinich; 2004 Mar. [cited 2008 Feb 2]. Available from: http://kucinich.house.gov/UploadedFiles/OSHA.pdf.)
64. Haque AK, Hernandez JC, Dillard EA, 3rd. Asbestos bodies found in infant lungs. Arch Pathol Lab Med. 1985;109:212.
65. Haque AK, Vrazel DM, Uchida T. Assessment of asbestos burden in the placenta and tissue digests of stillborn infants in South Texas. Arch Environ Contam Toxicol. 1998;35:532-8.
66. Ampleford EJ, Ohar J. Mesothelioma: You do not have to work for it. Diagn Cytopathol. 2007;35:774-7.
67. Barton H, Cogliano J, Firestone M, et al. Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. EPA/630/R-03/003F. 2005 March. Available from: http://www.epa.gov/ttn/atw/childrens_supplement_final.pdf.
68. Lee GL. Removing Dust From Brakes Assemblies During Vehicle Servicing- Alternative Cleaning Methods. Annals Occup Hyg. 1970;13:33-6.
69. Boillat MA, Lob M. Risque d'asbestose chez les travailleurs occupes a remplacer les garniture de freins [Risk of asbestosis in workers employed in replacing automobile brake linings]]. Schweiz Med. 1973;103:1354-9.
70. Castleman B, Ziem G. Control Technology for Brake and Clutch Work. In: Castleman B, ed. Asbestos: medical and legal aspects. 5th ed. New York: Aspen Publishers; 1985.
71. Hatch D. Possible Alternatives to Asbestos as a Fraction Material. Ann Occup Hyg. 1970;13:25-9.
72. Rodelsperger K, Jahn H, Bruckel B, et al. Asbestos Dust Exposure During Brake Repair. Am J of Indust Med.1986;10:63-725.
73. Kauppien T, Korhonen K. Exposure to Asbestos During Brake Maintenance of Automotive Vehicles by Different Methods. Am Ind Hyg Assoc J. 1987;48:499-504.
74. Hickish DE. Exposure to Asbestos Dust during Brake Maintenance Operations on Commercial Vehicles, Fleet Repair Garage, Dagenham. Report 41/68. 1968 October. Produced by Ford in Coates. Plaintiff Exhibit FD5036, http://www.egilman.com/Documents//Asbestos/Brakes/Ford/amosite%20and%20brakes/hickish%20amosite%20and%20brakes.pdf
75. Hickish DE. Exposure to Asbestos during the Servicing of Brake of Passenger Cars. Report 52/68. Produced by Ford in Coates. Plaintiff Exhibit FO-55. Available from: http://www.egilman.com/Documents//Asbestos/Brakes/Ford/Knowledge%20of%20Excess%20Exposures/hickish%20secret%20october%20amosite%201968.pdf.
76. Clark AR. Asbestos Exposure Associated with Automotive Brakes Systems. Union Carbide Internal Correspondence. 1976 July 8.
77. Hatfield RL, Longo WE. Bendix Brakes for Chrysler Vehicles: Work Practice Simulation Demonstration. Suwanee, GA: Materials Analytical Services, Inc; n.d.
78. Hatfield RL, Longo WE. Bendix Brakes for Ford Vehicles: Work Practice Simulation Demonstration. Suwanee, GA: Materials Analytical Services, Inc; n.d.
79. Hatfield R, Longo W. Arc Grinding II Cleanup. Raleigh, NC: Materials Analytic Services, Inc.; 2000.
80. Hatfield N, Longo W, Newton LR. Arc Grinding of Brake Shoes: Work Practice Study. Raleigh, NC: Materials Analytical Services, Inc.; 2000.
81. Hatfield N, Longo W, Newton LR. Hand Grinding of Brake Shoes: Work Practice Study. Raleigh, NC: Materials Analytical Services, Inc.; 2000.
82. Hatfield N, Newton LR, Longo W. Hand Sanding of Brake Shoes: Work Practice Study. Raleigh, NC: Materials Analytical Services, Inc.; 2001.
83. Rohl AN, Langer AM, Klimentidis R, et al. Asbestos content of dust encountered in brake maintenance and repair. Proc R Soc Med. 1977;70:32-7.
84. Osborn SH. Forty-Ninth Report of the State Department of Health for the Year Ended June 30, 1934. State of Connecticut Public Document No. 25. Hartford, CT: Bureau of Occupational Diseases; 1934.
85. Roberts DR, Zumwalde RD. Industrial Hygiene Summary Report of Asbestos exposure assessment for brake mechanics. Cincinnati, OH: Industrial Hygiene Section, Industrywide Studies Branch, Division of Surveillance, Hazard Evaluations and Field Studies, National Institute for Occupational Safety and Health; 1982 November 22. Report No.: 32.4.
86. Lloyd JW. Current Intelligence Bulletin 5: Asbestos: Asbestos exposure during servicing of motor vehicle brake and clutch assemblies. Cincinnati, OH: National Institute for Occupational Safety and Health; 1975 Aug 8. Available from: http://www.cdc.gov/NIOSH/78127_5.html
87. Longo WE, Mount MD, Hatfield RL. Occupational Exposure to Asbestos-Containing Brake Shoes During Hand Grinding and Sanding. Raleigh, NC: Materials Analytical Services, Inc.; 2004.
88. Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. Ann Occup Hyg. 2000;44:565-601.
89. Berman DW, Crump KS. A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. Crit Rev Toxicol. 2008;38 Suppl 1:49-73.

90. Berman DW, Crump KS. Update of potency factors for asbestos-related lung cancer and mesothelioma. Crit Rev Toxicol. 2008;38 Suppl 1:1-47.
91. Kane A. (Chair, SAB Asbestos Committee). Letter to Johnson S. (EPA Adminstrator), SAB Consultation on EPA's Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos Cancer Potency Factors for Inhalation Exposure to Asbestos 2008. EPA-SAB-09-004 Available from: http://yosemite.epa.gov/sab/sabproduct.nsf/77CFF6439C00ABF3852575010077801F/$File/EPA-SAB-09-004-unsigned.pdf.
92. Selikoff IJ, Seidman H. Asbestos-associated deaths among insulation workers in the United States and Canada, 1967-1987. Ann N Y Acad Sci. 1991;643:1-14.
93. Rogers A, Major G. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure: The Wittenoom data. Ann Occup Hyg 2002;46:127-8; author reply 8-9.
94. Gibbs G, LaChance M. Dust-fiber relationships in the Quebec chrysotile industry. Arch Environ Occup Health. 1974;28:3.
95. Shapiro HA. Pneumoconiosis; Proceedings of the international conference, Johannesburg, 1969. Cape Town, New York,: Oxford University Press; 1970.
96. Hodgson J, Darnton A. Letter. Ann Occup Hyg. 2001;45:336-8.
97. Leigh J, Robinson B. The history of mesothelioma in Australia 1945-2001. In: Robinson B, Chahinian A, eds. Mesothelioma. London: Martin Dunitz; 2000:55-86.
98. Henderson DW. Supplementary Report, On Causation, for Mr. Frank Gregory Lansley. Adelaide, South Australia; 2006 February 27.
99. Costas E, Garrido A, Goyanes VJ. Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. N Engl J Med. 1998;339:1000; author reply 1-2.
100. Hein MJ, Stayner LT, Lehman E, Dement JM. Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. Occup Environ Med. 2007;64:616-25.
101. Peto J, Rake C, Gilham C, Hatch J. Occupational, domestic and environmental mesothelioma risks in Britain: A case-control study. Health and Safety Executive; 2009. Research Report No.: 696. Available at: http://www.hse.gov.uk/research/rrpdf/rr696.pdf.
102. Paustenbach D. Deposition Testimony. Gray vs.John Crane, Inc., et al. , Virginia Circuit Court for the City of Newport News, Case Number CL0800724PT; 2009 Apr 6.
103. Pierce JS, McKinley MA, Paustenbach DJ, Finley BL. An Evaluation of Reported No-Effect Chrysotile Asbestos Exposures for Lung Cancer and Mesothelioma, Crit Rev Tox. 2008; 38;191-214.
104. Suzuki Y, Yuen SR. Asbestos tissue burden study on human malignant mesothelioma. Ind Health. 2001;39:150-60.
105. Sebastien P, Janson X, Gaudichet A, Hirsch A, Bignon J. Asbestos retention in human respiratory tissues: comparative measurements in lung parenchyma and in parietal pleura. IARC Sci Publ. 1980:237-46.
106. Dodson RF, Williams MG, Jr., Corn CJ, et al. A comparison of asbestos burden in lung parenchyma, lymph nodes, and plaques. Ann N Y Acad Sci. 1991;643:53-60.
107. LeBouffant L. Investigation and analysis of asbestos fibers and accompanying minerals in biological materials. Environ Health Perspect. 1974;9:149-53.
108. Kohyama N, Suzuki Y. Analysis of asbestos fibers in lung parenchyma, pleural plaques, and mesothelioma tissues of North American insulation workers. Annals NY Acad Sci. 1991;643:25.
109. Suzuki Y, Yuen SR, Ashley R. Short, thin asbestos fibers contribute to the development of human malignant mesothelioma: Pathological evidence. Int J Hyg Environ Health. 2005;208:201-10.
110. Boutin C, Dumortier P, Rey F, et al. Black spots concentrate oncogenic asbestos fibers in the parietal pleura. Thoracoscopic and mineralogic study. Am J Respir Crit Care Med. 1996;153:444-9.
111. Müller KM, Schmitz I, Konstantinidis K. Black spots of the parietal pleura: morphology and formal pathogenesis. Respiration. 2002;69:261-7.
112. Mitchev K, Dumortier P, De Vuyst P. 'Black Spots' and hyaline pleural plaques on the parietal pleura of 150 urban necropsy cases. Am J Surg Pathol. 2002;26:1198-206.
113. Wagner JC, Berry G. Mesotheliomas in rats following inoculation with asbestos. Br J Cancer. 1969;23:567-81.
114. Wagner JC, Berry G, Timbrell V. Mesotheliomata in rats after inoculation with asbestos and other materials. Br J Cancer. 1973;28:173-85.
115. Pott F, Huth F, Friedrichs KH. Tumorigenic effect of fibrous dusts in experimental animals. Environ Health Perspect. 1974;9:313-5.
116. Wagner JC, Berry G, Skidmore JW, et al. The effects of the inhalation of asbestos in rats. Br J Cancer. 1974;29:252-69.
117. Appel JD, Fasy TM, Kohtz DS, et al. Asbestos fibers mediate transformation of monkey cells by exogenous plasmid DNA. Proc Natl Acad Sci USA. 1988;85:7670-4.
118. Adamson IY, Bakowska J, Bowden DH. Mesothelial cell proliferation: a nonspecific response to lung injury associated with fibrosis. Am J Respir Cell Mol Biol. 1994;10:253-8.
119. Adamson IY. Early mesothelial cell proliferation after asbestos exposure: in vivo and in vitro studies. Environ Health Perspect 1997;105 Suppl 5:1205-8.
120. Kumar-Singh S, Weyler J, Martin MJ, Vermeulen PB, Van Marck E. Angiogenic cytokines in mesothelioma: a study of VEGF, FGF-1 and -2, and TGF beta expression. J Pathol. 1999;189:72-8.
121. DeLong P, Carroll RG, Henry AC, et al. Regulatory T cells and cytokines in malignant pleural effusions secondary to mesothelioma and carcinoma. Cancer Biol Ther. 2005;4:342-6.
122. Yoshimoto A, Kasahara K, Saito K, Fujimura M, Nakao S. Granulocyte colony-stimulating factor-producing malignant pleural mesothelioma with the expression of other cytokines. Int J Clin Oncol. 2005;10:58-62.
123. Hegmans JP, Hemmes A, Hammad H, et al. Mesothelioma environment comprises cytokines and T-regulatory cells that suppress immune responses. Eur Respir J. 2006;27:1086-95.
124. Roggli VL, Vollmer RT. Twenty-five years of fiber analysis: what have we learned? Hum Pathol. 2008;39:307-15.
125. Sebastien P, Masse R, Bignon J. Recovery of ingested asbestos fibers from the gastrointestinal lymph in rats. Environ Res. 1980;22:201-16.
126. Acheson ED, Gardner MJ. Mesothelioma and exposure to mixtures of chrysotile and amphibole asbestos. Arch Environ Health. 1979;34:240-2.
127. Roggli VL. Deposition Testimony. Wanda T. Jones, v. Dana Corporation, et al., Circuit Court For The City Of Newport News Virginia, Law No. 39028T-01.; 2006 Jan 13.
128. Roggli VL, Sharma A, Butnor KJ, Sporn T, Vollmer RT. Malignant mesothelioma and occupational exposure to asbestos: A clinicopathological correlation of 1445 cases. Ultrastruct Pathol. 2002;26:55-65.
129. Butnor KJ, Sporn TA, Roggli VL. Exposure to brake dust and malignant mesothelioma: a study of 10 cases with mineral fiber analyses. Ann Occup Hyg. 2003;47:325-30.
130. Srebro SH, Roggli VL, Samsa GP. Malignant mesothelioma associated with low pulmonary tissue asbestos burdens: a light and scanning electron microscopic analysis of 18 cases. Mod Pathol. 1995;8:614-21.
131. Roggli VL. Affidavit, Exhibit 1. Bretzke vs. Ford Motor Company, District Court of Ramsey County, Minnesota Court File No. 62CV081189. 2008 Sept 13.
132. Roggli VL. Comments Regarding The U.S. EPA's Proposed Approach for the Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure for Asbestos; 2008. http://yosemite.epa.gov/sab/sabproduct.nsf/33EEF78BBA9E6D0085257491006E9276/$File/Asbestos+Public+Comments+Victor+Roggli+July+21-22+2008+Meeting.pdf.
133. Grave Robbers: Chrysler Subpoenas Body At Funeral [Internet]. WCBSTV. 2009 Mar 7. [cited 2009 Mar 14]. Available at: http://wcbstv.com/topstories/graveside.subpoena.harold.2.952746.html
134. Order on Emergent Application. Harold St. John and Florence St. John v. Affinia Group, Inc., et al. Superior Court of New Jersey Appelate Division. 2009 Mar 18. [cited 2009 Mar 29]. Available from: http://amlawdaily.typepad.com/stjohnappeal.pdf.
135. Roggli VL. Deposition testimony, Paz vs AW Chesterton et al., Circuit Court, Third Judicial Circuit ,Madison County, Illinois. 1/27/2006
136. Roggli VL. Testimony. Harold R. St. John And Florence Diane St. John vs. Affinia Group, Incorporated, et al., Superior Court Of New Jersey, Law Division: Middlesex County Docket No. Mid-L-5110-08; 2009 Mar 11.
137. Roggli VL. Testimony in Lilly v. John Crane et al, Circuit Court of Newport News, Virginia, Case No. 38861AF. 2001 Sept 19.

138. Roggli VL. The Friction Products Debate (PowerPoint Presentation). Defense Research Institute; 2008 Nov.
139. Egilman DS, Billings MA. Differential peeky bias. Int J Occup Environ Health. 2006;12:294.
140. Roggli VL, McGavran MH, Subach J, et al. Pulmonary asbestos body counts and electron probe analysis of asbestos body cores in patients with mesothelioma: a study of 25 cases. Cancer. 1982; 50:2423-32.
141. Ingram P, Shelburne JD, Roggli VL. Microprobe analysis in medicine. New York: Hemisphere; 1989.
142. Roggli VL, Longo WE. Mineral fiber content of lung tissue in patients with environmental exposures: Household contacts vs. building occupants. Ann N Y Acad Sci. 1991;643:511-8.
143. Roggli VL, Pratt PC, Brody AR. Asbestos content of lung tissue in asbestos associated diseases: a study of 110 cases. Br J Ind Med. 1986;43:18-28.
144. Roggli VL. Testimony. Robert Frank Smith and Mary Lou Smith v. Chrysler LLC, et al., Los Angeles Superior Court Docket No. 396072. 03/13/2009
145. Work-Related Lung Disease Surveillance Report, 2002. Cincinnati, OH: National Institute for Occupational Safety and Health; 2003. Report No.: 2003-111.
146. Henderson DW. Malignant mesothelioma. New York: Hemisphere Pub. Corp.; 1992.
147. Roggli VL. Questions on your paper response. Email Communication to: David Egilman; 2009 March 17.
148. Roggli VL. Deposition. Windnagle vs Georgia Pacific et al., Circuit Court Of The 11th Judicial Circuit In and For Miami-Dade County Florida CASE NO: 06-28424 CA 42; 2007 Nov 20.
149. Roggli VL. Environmental asbestos contamination: What are the risks? Chest. 2007;131:336-8.
150. Unknown. Regression by Maximum Likelihood Estimation for Left-censored Data [Internet] Available from: http://rss.acs.unt.edu/Rdoc/library/NADA/html/cenmle.html.
151. Toxicological Profile for Asbestos [Internet]. Agency for Toxic Substances and Disease Registry; 2001 Sept. Available from: http://www.atsdr.cdc.gov/toxprofiles/tp61.html .
152. Roggli VL, Oury TD, Sporn TA. Pathology of asbestos-associated diseases. 2nd ed. New York: Springer; 2004.
153. Environmental Protection Agency. Method for the Determination of Asbestos in Bulk Building Materials. US Environmentqal Protection Agency. 1993 July. Report No.: EPA/600/R-93/116.
154. Environmental Protection Agency. Asbestos Hazard Emergency Response Act. 40 CFR Part 763, Asbestos-containing materials in schools. 1987 October 30.
155. National Institute of Occupational Safety and Health. Asbestos by TEM. NIOSH Manual of Analytical Methods (NMAM), Fourth Edition. 1994 August 15.
156. Standard test method for airborne asbestos concentration in ambient and indoor atmospheres as determined by transmission electron microscopy direct transfer (TEM). ASTM; 1998.
157. Standard Test Method for Microvacuum Sampling and Indirect Analysis of Dust by Transmission Electron Microscopy for Asbestos Structure Number Surface Loading: Dust Sample analysis- ASTM D 5755-03. 2003 Oct 1.
158. Chatfield EJ. Ambient Air, Determination of Asbestos Fibers, Indirect-Transfer Transmission Electron Microscopy Method. International Standard Organization;1999.
159. Chatfield EJ, Berman DW. Interim Superfund Method for the Determination of Asbestos in Ambient Air, Part 1: Method. U.S. Environmental Protection Agency. Publication No: 540/2-90/005a; 1990 May.
160. Loomis D, Dement JM, Wolf SH, et al. Lung Cancer Mortality and Fiber Exposures among North Carolina Asbestos Textile Workers. Occup Environ Med. 2009 Mar 11 [epub ahead of print].
161. Stayner LT. Canada, chrysotile and cancer: Health Canada's asbestos international expert panel report. J Occup Environ Med. 2008;50:1327-8.
162. Roggli VL, Sanders LL. Asbestos content of lung tissue and carcinoma of the lung: a clinicopathologic correlation and mineral fiber analysis of 234 cases. Ann Occup Hyg. 2000;44:109-17.
163. Suzuki Y, Yuen SR. Asbestos tissue burden study on malignant mesothelioma. Ind Health. 2001;39:160.
164. Dodson RF, Hammar SP, Poye LW. A technical comparison of evaluating asbestos concentration by phase-contrast microscopy (PCM), scanning electron microscopy (SEM), and analytical transmission electron microscopy (ATEM) as illustrated from data generated from a case report. Inhal Toxicol. 2008;20:723-32.
165. Asbestos in Public and Commercial Buildings:A Literature Review and Synthesis of Current Knowledge. Cambridge, MA: Health Effects Institute, 1991.
166. Finkelstein MM. Asbestos fibre concentrations in the lungs of brake workers: another look. Ann Occup Hyg. 2008;52:455-61.
167. Roggli VL, Sporn TA, Case BW, et al. Comments on Asbestos fibre concentrations in the lungs of brake workers: another look. Ann Occup Hyg. 2009;53:191; author reply 2-3.
168. Gustavsson P, Ahlbom A, Andersson T, et al. Calculation of fractions of lung cancer incidence attributable to occupational exposure to asbestos and combustion products in Stockholm, Sweden. Eur J Epidemiol. 2003;18:937-40.
169. Gustavsson P, Albin M. Low-dose occupational exposure to asbestos and lung cancer risk. Med Lav. 2006;97:357.
170. Stayner L, Kuempel E, Gilbert S, et al. An epidemiological study of the role of chrysotile asbestos fiber dimensions in determining respiratory disease risk in exposed workers. Occup Environ Med. 2008;65:613-9.
171. Stayner L, Smith R, Bailer J, et al. Exposure-response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. Occup Environ Med. 1997;54:646-52.
172. Stayner LT, Dankovic DA, Lemen RA. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. Am J Public Health. 1996;86:179-86.
173. Chen WJ, Mottet NK. Malignant mesothelioma with minimal asbestos exposure. Hum Pathol. 1978;9:253-8.
174. Iwatsubo Y, Pairon JC, Boutin C, et al. Pleural mesothelioma: dose-response relation at low levels of asbestos exposure in a French population-based case-control study. Am J Epidemiol. 1998;148:133-42.
175. Keeton P, Prosser WL. Prosser and Keeton on the law of torts. 5th ed. St. Paul, Minn.: West Pub. Co.; 1984.
176. Hickish DE, Knight KL. Report 41/68: Exposure to Asbestos during the Servicing of Brakes of Passenger Cars. In; 1968.
177. Cline W. Borg Warner Corporation answers to interrogatories in Re: All Asbestos Litigation Borg. In: In The Circuit Court Of The Third Judicial Circuit Madison County, Illinois; 2006.
178. Jenkins W. Maremont answers to interrogatories in the case of London vs. Flintcote et al. Circuit Cort of Pleas case no. 6849 Philadelphia County, Pa. In; 1984.
179. North American Asbestos Corporation Annual Report. 1957. Available from: http://www.egilman.com/browse.php?display=list&dir=Asbestos/Brakes/amphibole_use/.
180. NorthAmerican Asbeestos Company Customer List. 1976. Available from: http://www.egilman.com/browse.php?display=list&dir=Asbestos/Brakes/amphibole_use/.
181. Blau PJ. Compositions, Functions, and Testing of Friction Brake Materials and Their Additives: Bechtel; 2001.
182. McDonald J. Linear Extrapolation for Risk Estimation as Low Level Exposure: The Asbestos Example In: London Univ; McGill Univ; Natl Heart + Lung Inst: Philip Morris; Unknown.
183. Greenland S. Relation of probability of causation to relative risk and doubling dose: a methodologic error that has become a social problem. Am J Public Health. 1999;89:1166-9.
184. Egilman D, Howe S. Against anti-health epidemiology: corporate obstruction of public health via manipulation of epidemiology. Int J Occup Environ Health. 2007;13:118-24.
185. Henderson DW. Malignant mesothelioma. New York: Hemisphere Pub. Corp.; 1992.
186. Wagner JC, Pooley FD. Mineral fibres and mesothelioma. Thorax. 1986;41:161-6.
187. Churg A. Chrysotile, tremolite, and malignant mesothelioma in man. Chest.1988;93:621-8.
188. Egilman DS, Kim J, Biklen M. The Use and Abuse of Medical and Scientific Evidence Inside the Courtroom-An Epidemiologist's Critique of the Judicial Interpretation of the Daubert Ruling. Food and Drug Law Journal. 2003;58.
189. Rodelsperger K, Woitowitz HJ, Bruckel B, et al. Dose-response relationship between amphibole fiber lung burden and mesothelioma. Cancer Detect Prev. 1999;23:183-93.

Errata

Errors appeared in the original version of the article "Fiber Types, Asbestos Potency, and Environmental Causation: A Peer Review of Published Work and Legal and Regulatory Scientific Testimony," by David Egilman, which appeared in the April/June 2009 issue of *IJOEH.* Dr. Egilman thanks Dr. Victor Roggli for calling attention to these errors.

The errors have been corrected in this version of the article to read as follows:

Page 214. "On March 18, 2009, the New Jersey Appellate Court ruled that Chrysler [*not* Ford] had no need to remove Mr. St. Johns lung tissue…"

Page 216. "Srebro…conducted the investigation to determine if the controls had a previous history of [*not* "history of a previous history of"] work with asbestos."

Page 218. "In one blinded cross-laboratory comparison on the same patient, Dodson et al. found 84 chrysotile fibers while Roggli reported only one.[164]" [*not* "Dodson et al. found 84 fibers per gram while Roggli reported only one.[163]"]

Page 218. "Roggli…claims that TEM [*not* SEM] undercounts amosite."

Page 221. "Borg Warner used crocidolite in some automobile clutches and brake bands." [*not* "…used crocidolite some automobile clutches…"]

Page 222. Table 4. Case 13 is a "Caused Case" and should appear with no shading.