Miller, Diane M. (CDC/NIOSH/EID)

From: David Egilman [david@neveragainconsulting.com]
Sent: Wednesday, February 24, 2010 3:01 PM
To: NIOSH Docket Office (CDC) 099-C ASBESTOS ROADMAP AM
Subject: ~~194 - Ten-Year Review of the NIOSH Radiation Dose Reconstruction Program comments~~
Attachments: Nicholson_1981 (2).pdf; nicholson Health Hazards of Asbestos Exposure (Effect of Aspect Ratio Fiber Counts - Winer Cossette).pdf; critique of hodgson.pdf

The relative potency data is inaccurate for several reasons:

1. You cannot convert particle counts to fiber counts (See my paper attached)
2. Fiber counts do not address the variability in fiber lengths that are not counted or the relative concentration of fiber lengths greater than 5 microns.

In other words for each fiber greater than 5 microns there are 10,000 to 100,000,000 million fibers shorter than 5 microns and there is similar variability for the number of fibers greater than 5 microns. (See attached graphs from Nicholson for fiber distribution from various product exposures.) The same phenomenon applies to mine samples from different samples. (See Winer attached)

THE CHARACTERIZATION OF ASBESTOS DUST LEVELS IN INSULATION WORK

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Submissions by Drs. Irving J. Selikoff and Kingsley Kay have documented the disease experience of the insulation workers in the New York and New Jersey Locals of the International Association of Heat and Frost Insulators and Asbestos Workers. To attempt to relate that health experience with asbestos dust levels to which the workers were exposed, industrial hygiene surveys have been made to asbestos use on construction sites in the New York-New Jersey area.

Asbestos Dust Exposures of Insulation Workers

The jobs studied included the blocking of boilers and breechings, covering pipes, insulating turbines (blocking, cementing, and spraying), cutting with hand saws and power saws, and mixing cement. The average concentrations found for several work practices are listed in Table 1. Individual counts ranged from less than 0.1 f/cm^3 to more than 100 f/cm^3. Some individual values measured are listed in appendix A. However, when averaged over the time workers spent on a given job using asbestos, the average exposure level was 6 f/cm^3. This average took into account the dustiest jobs in areas with poor ventilation as well as the clean ones in well-ventilated areas. If one considers that most asbestos workers spend more than half their working time with materials other than asbestos, the long-term average asbestos exposure of insulation workers today would be under 3 f/cm^3.

In comparing exposure levels with disease experience, it is

well to remember that today's disease may be the result of exposures 20, 30, or even 40 years ago. Some changes have taken place in insulation practices and insulation products during these years. Fibrous glass is now used extensively. Cork and felt are in little use today. But, even with the changes, it appears that dust concentrations, averaged over a day, during past years have not often exceeded 5 f/cm^3. In other words, significant disease is manifest in insulation workers exposed on average to levels of asbestos not significantly different from 5 fibers per cubic centimeter.

Variability of Estimates of Asbestos Exposure

The above correlation between disease and levels of past exposure, estimated from current measurements, is necessarily approximate. Such estimates of past exposures are, of course, fraught with obvious difficulties. However, as limited as our knowledge is of past exposure levels measured by optical microscopy, the correlation between disease and exposure is further complicated by the fact that light microscopy measures only a small fraction of the total number of fibers present. This would be acceptable if the fraction measured were a constant one. However, such is not the case. Figure 1 shows a typical fiber distribution found when an asbestos air sample is studied by electron microscopy. The longest fiber present in that photomicrograph is 2 microns long.

Figures 2 to 6 show the size distribution, measured using

electron microscopy, of several operations applying or removing asbestos materials. The fraction of fibers longer than 5 microns ranges from 0.004 to 0.29. Even when consideration is restricted to those operations involving only chrysotile, the fraction of fibers longer than 5 microns can differ by ten times. Thus, while it has been long recognized that counting asbestos-containing air samples with an optical microscope involves observation only of the "tip of the iceberg", it must also be recognized that we are dealing with qualitatively different icebergs having different densities.

At this time, it is well to recognize the scope of the problem associated with evaluating occupational asbestos exposures:

1. Maintenance of control by dust sampling is difficult and time-consuming,
2. The fiber count obtained cannot be related to a disease experience at all levels of exposure,
3. The physical assessment of an asbestos dust exposure is incomplete.

With these uncertainties, it is prudent, at this time, to reduce occupational asbestos exposures at least to levels possible with existing technology. This should be done by mandating procedural standards that must be met during the performance of any work involving asbestos. Such standards should include:

1. Exhaust ventilation of all power equipment

2. Mixing of asbestos materials in closed containers

3. Dust reducing coatings on asbestos materials

4. Downdraft cutting tables for hand sawing

5. Bagging of waste in enclosed containers

6. Removal of dust by vacuum cleaners

(Items 1, 2, 5, and 6 are included in the proposed standard.)

Tables 2 and 3 give levels that can be achieved in band saw ventilation and mixing asbestos cement in plastic bags. Table 4 give levels of exposure in insulation work that can be obtained using the above procedures.

Table I

AVERAGE DUST CONCENTRATIONS IN INSULATION WORK

Type of Work	Fiber Concentration (Fibers/cm^3)
Mixing and Applying Cement and Cloth Covering to Insulation Material	
Good Ventilation	2.5
Poor Ventilation	4.6
Cutting and Applying Insulation Block or Sections of Pipe Covering	
Good Ventilation	5.2
Poor Ventilation	11.5
At least 75% of an asbestos workers time, when using asbestos materials, is spent on the above jobs.	
Cutting Materials for later use	7.6
Spraying Turbines with Asbestos Fiber	
Spray man	67 (7)*
Hopper man	10.5 (1)*

* During the occasional jobs of spraying asbestos fiber, asbestos workers typically have worn half-mask respirators. The fiber concentration in parenthesis represents the dust concentration breathed by the worker with such a respirator

Table 2

Mixing Asbestos Cement

(Sampled during time of mixing, 5-9 min.)

Samples/operation		
6	Tub and Bucket Mixing	52
2	Plastic bag	6.3

Table 3

Power Saw Dust Collection

Samples/operation		
6	Uncontrolled	35
3	Collector A	0.5
1	B	2.0
4	C	0.7

Fig. 2

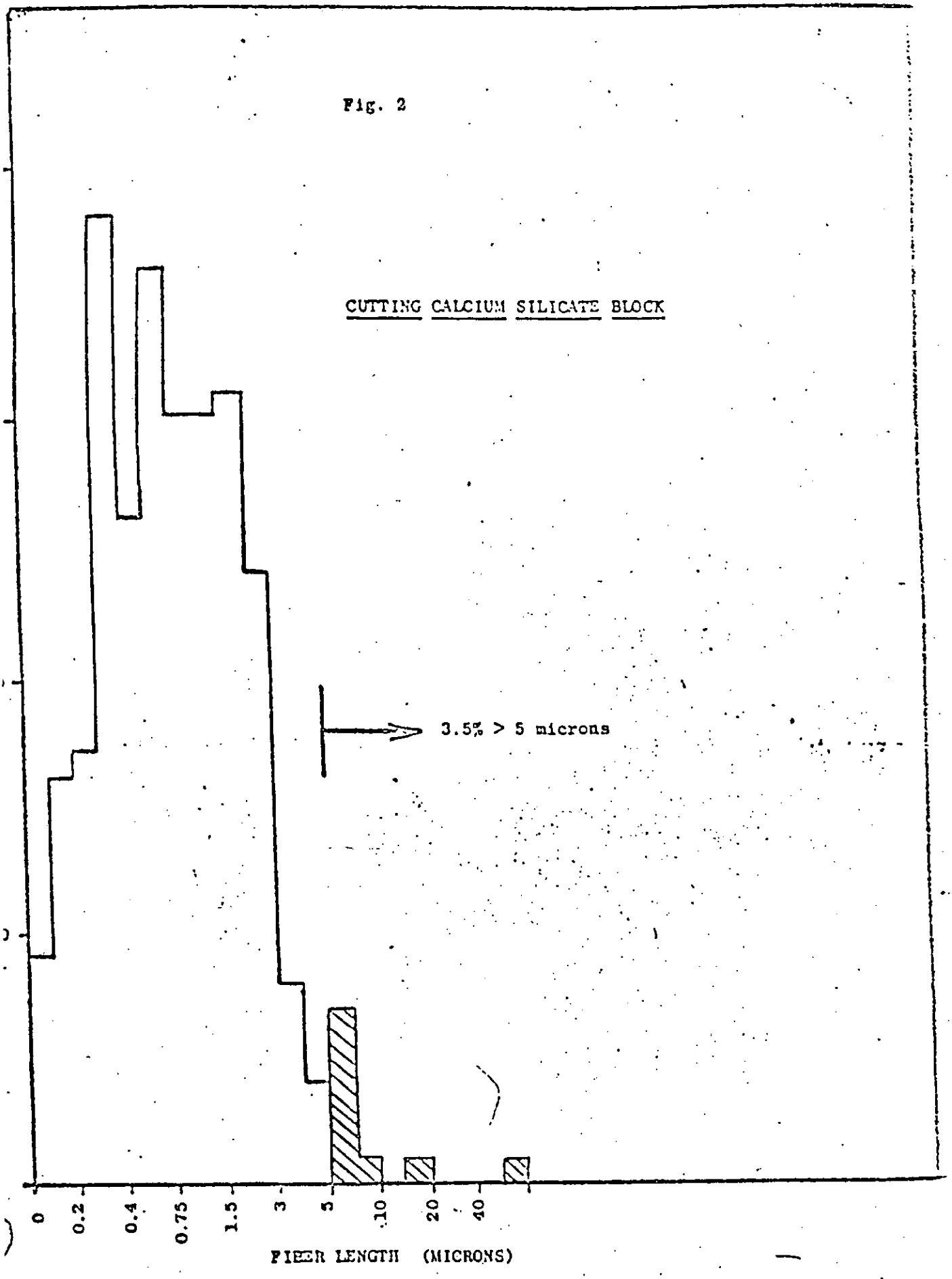
CUTTING CALCIUM SILICATE BLOCK
3.5% > 5 microns
0
0.2
0.4
0.75
1.5
3
5
10
20
40
FIBER LENGTH (MICRONS)

Fig. 3

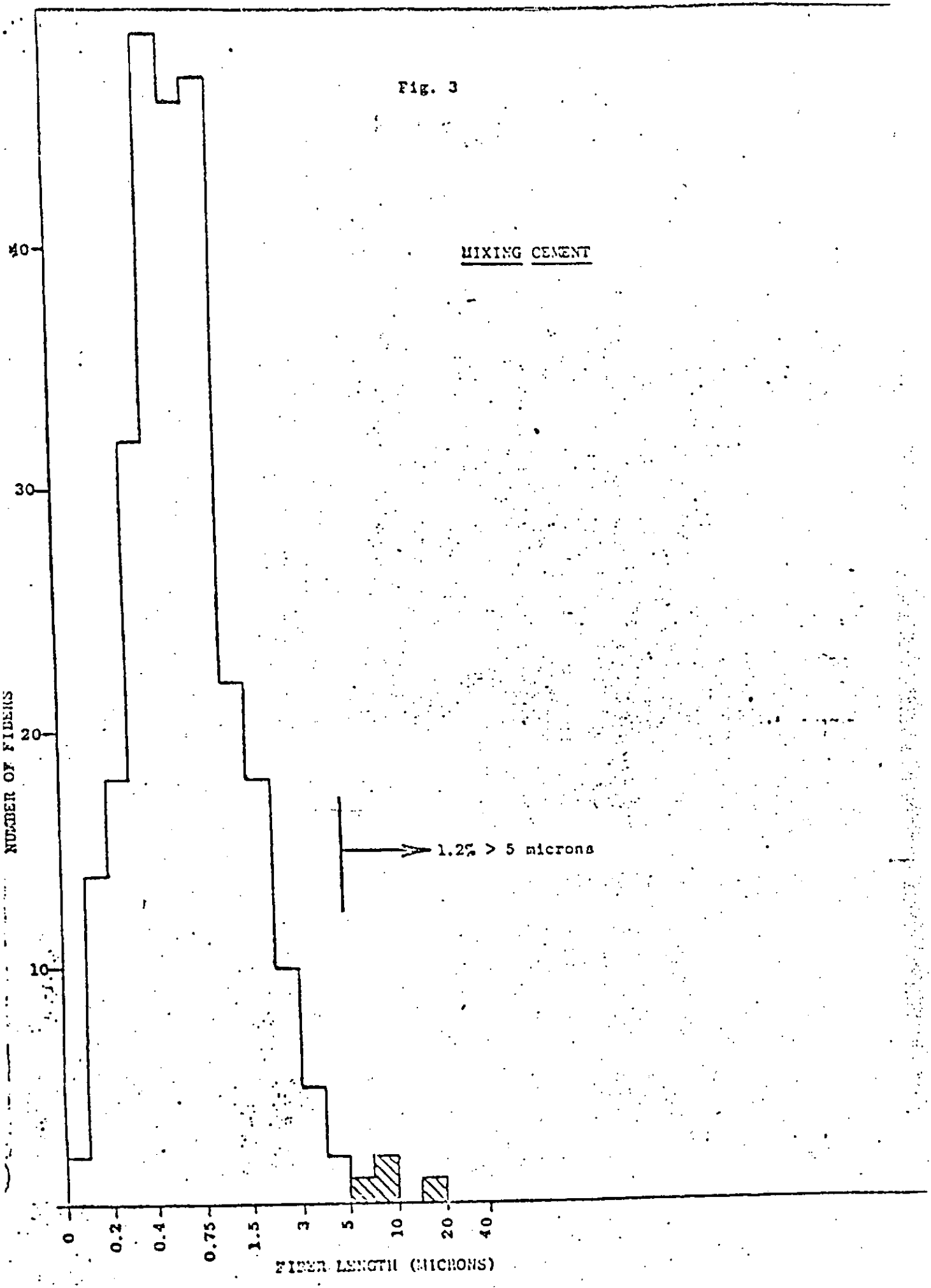
MIXING CEMENT
40
30
20
10
NUMBER OF FIBERS
1.2% > 5 microns
0
0.2
0.4
0.75
1.5
3
5
10
20
40
FIBER LENGTH (MICRONS)

Fig. 4

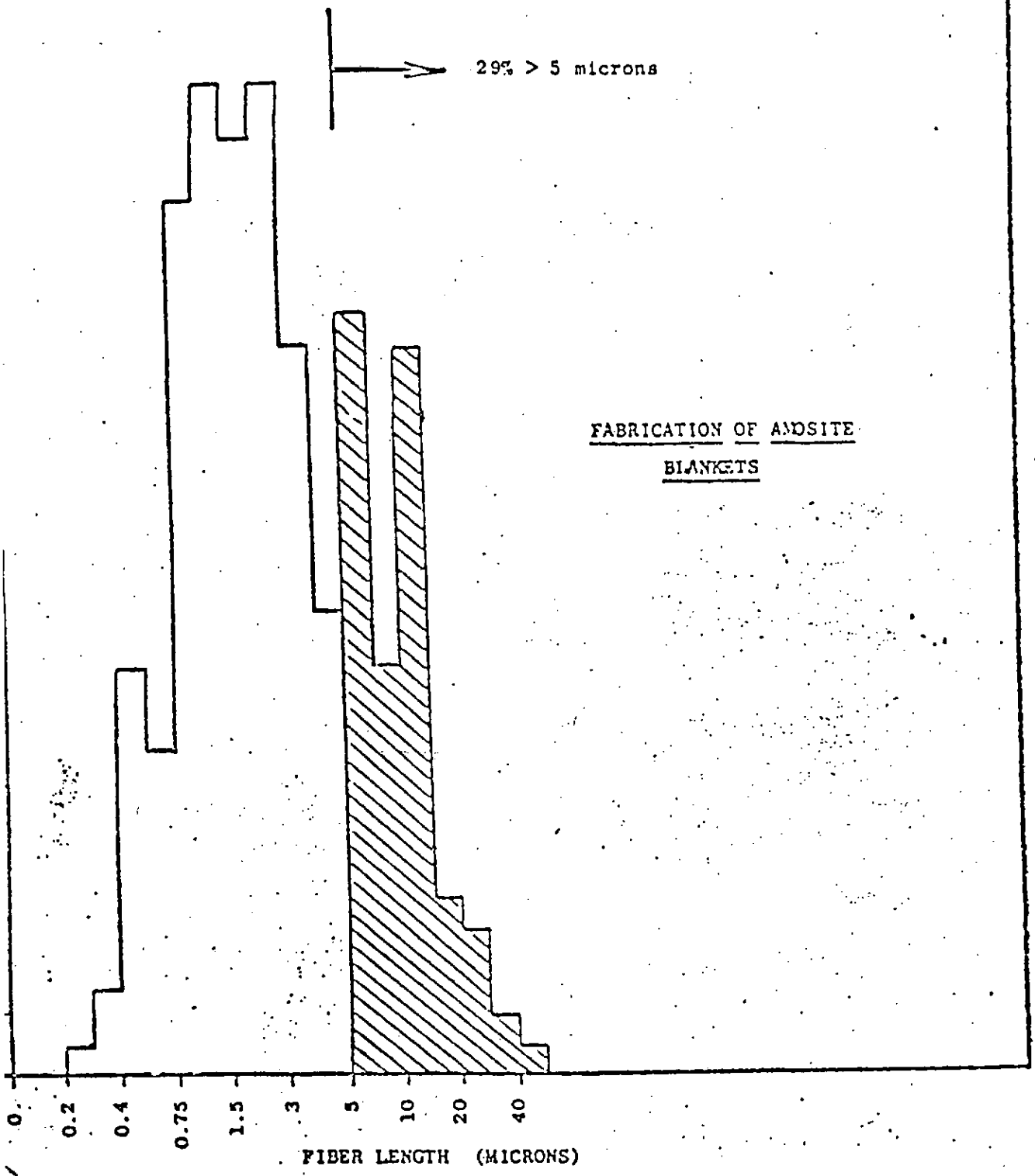
29% > 5 microns
FABRICATION OF ANDSITE
BLANKETS
0
0.2
0.4
0.75
1.5
3
5
10
20
40
FIBER LENGTH (MICRONS)

Fig. 5

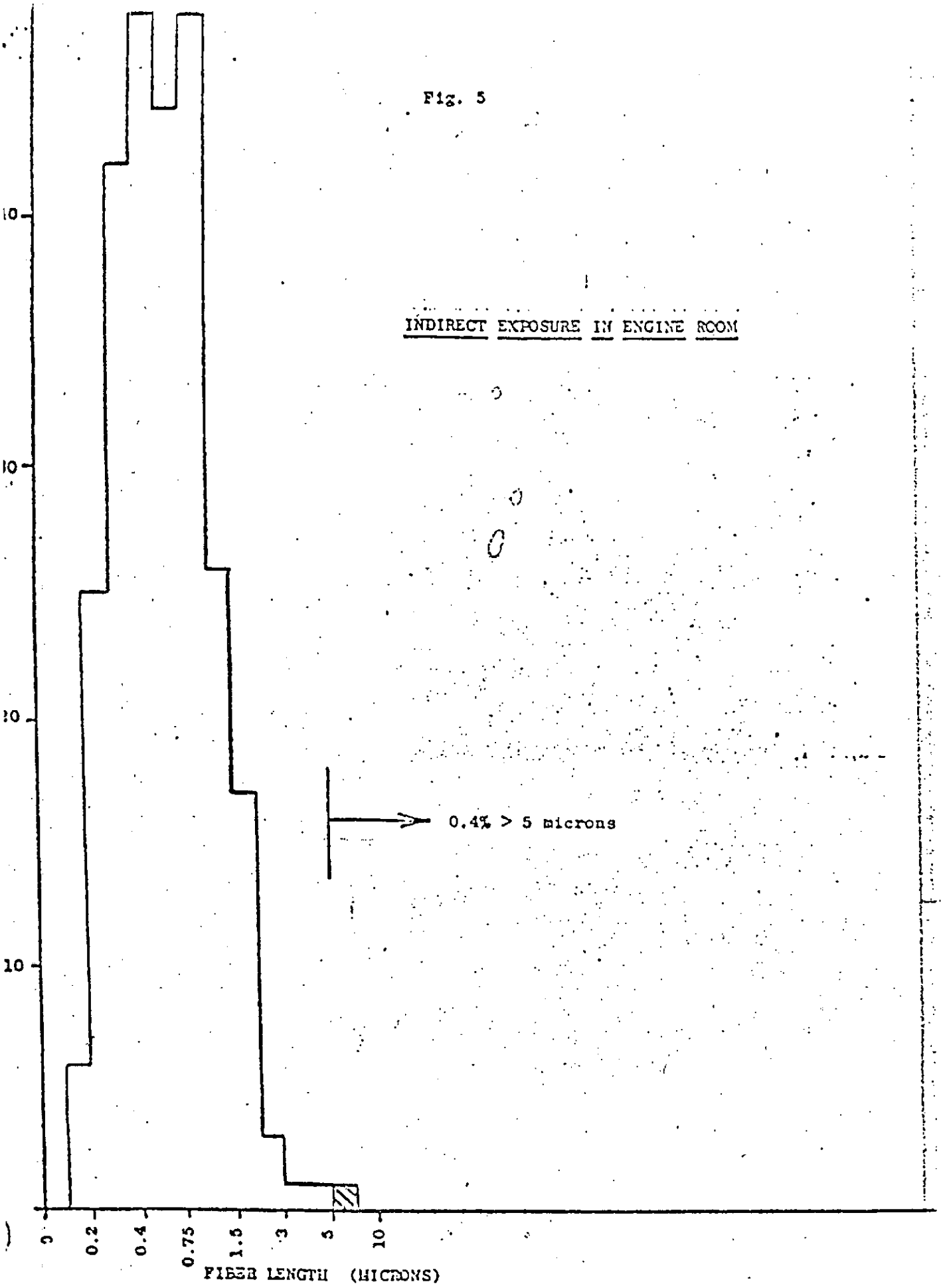
INDIRECT EXPOSURE IN ENGINE ROOM
10
10
10
10
0.4% > 5 microns
0
0.2
0.4
0.75
1.5
3
5
10
FIBER LENGTH (MICRONS)

Fig. 6

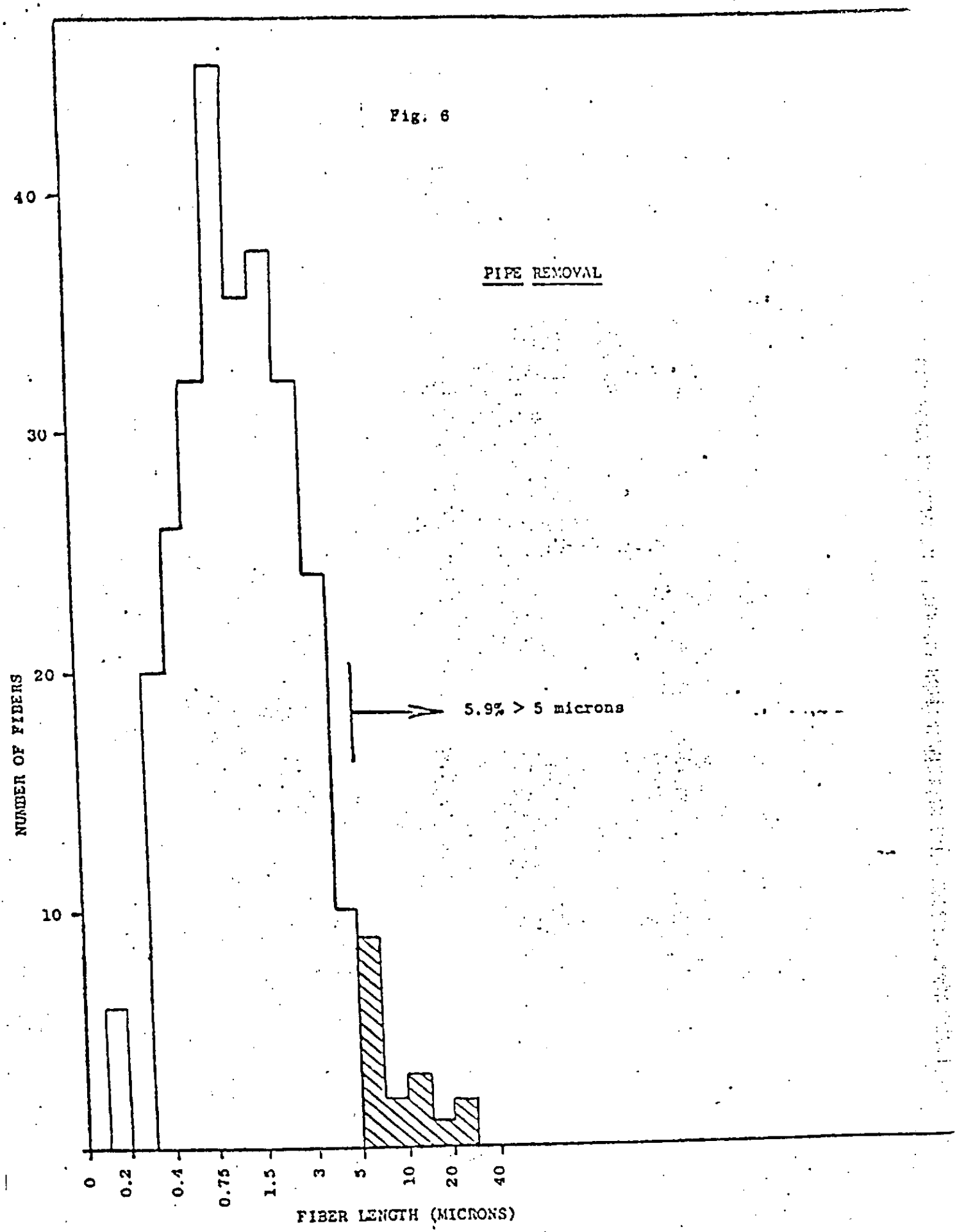
PIPE REMOVAL
40
30
20
10
NUMBER OF FIBERS
5.9% > 5 microns
0
0.2
0.4
0.75
1.5
3
5
10
20
40
FIBER LENGTH (MICRONS)

Table 4

VERAGE DUST CONCENTRATIONS IN INSULATION WORK WITH IMPROVED INDUSTRIAL HYGIENE PRACTICES

ype of Work	Fiber Concentration Possible with Improved Industrial Hygiene Practices (fibers/cc)	Fiber Concentration Possib With Improved Practices an of Approved Disposable Res (fibers/cc)
ixing and Applying Cement and loth Covering to Insulation Material		
Good Ventilation	0.5	0.05
Poor Ventilation	1.0	0.1
itting and Applying Insulation Block r Sections of Pipe Covering		
Good Ventilation	2.5**	0.3
Poor Ventilation	5.0**	0.5
itting Materials for later use	1.5	0.2
praying Turbines with Asbestos Fiber		
Spray man	0.7*	
Hopper man	0.1*	

The reduced fiber levels shown here are possible with the use of a portable air supplied respirator Similar dust reductions can be made during the removal of old insulation material, another extremel dusty job.

The use of an approved disposable respirator is highly recommended for this work.

Appendix A

Mixing cement and finishing pipes including covering with cloth -- no blocking

Samples/job site	Time Weighted Average
1	1.6
6	3.8
8	3.2
3	0.7

Range of counts 0.5 to 9.2

Mixing and applying cement to tanks

Samples/job site	Time Weighted Average
11	2.5

Range of counts 0.4 to 5.2

Pipe covering -- cement mixing and application

Samples/job site	Time Weighted Average
8	6.1

Range of counts 1.3 to 12.3

Blocking pipes outside including cutting and fitting -- no cement mixing -- good ventilation

Samples/job site	Time weighted Average
8	1.2

Range of counts 0.6 to 2.4

Boiler room blocking with very poor ventilation (including boilers, breechings, and room ceiling) -- no cement mixing

Samples/job site	Time Weighted Average
2	21.1
3	8.6
6	10.2

Range of counts 6.4 to 37

Hand sawing of calcium silicate pipe covering and block for future use

Samples/job site	Time Weighted Average
6	6.8

Range 4.3 to 22

Turbine spraying

Samples/job site	Time Weighted Average
6	66

Range of Counts 31 to 100

Spray hopper tending

Samples/job site	Time Weighted Average
3	10.3

Range of counts 4.6 to 22

Peak Exposures

Mixing of hydraulic, fast setting, cement containing 15% asbestos. (sampled only for duration of mixing)

Samples/job site	average count	time
1	46	9 min
1	76	4 min
2	16	5 min

Mixing of non-hydraulic cement containing 50% asbestos

Samples/job site	average count	time
1	10	8 min.

Band saw cutting in good ventilation with no collector

Samples/job site	average count	time
2	36	1 min
4	16	2 min

ANNALS OF
THE NEW YORK ACADEMY
OF SCIENCES

VOLUME 330

HEALTH HAZARDS OF ASBESTOS EXPOSURE

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PUBLISHED BY THE NEW YORK ACADEMY OF SCIENCES
ANYAA9 330 1-814 (1979)

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES

Volume 330

The New York Academy of Sciences
2 East 63rd Street
New York, New York 10021

THE EFFECT OF ASPECT RATIO ON FIBER COUNTS: A PRELIMINARY STUDY

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The "Naturally Occurring Inorganic Fiber Task Group" in ASTM Committee D-22 (Occupational Health and Safety) has been developing an ASTM standard for fibers that include asbestos. The method of counting fibers, the NIOSH technique,[1] was determined to have a high degree of variance. This was confirmed at a recent workshop of the National Bureau of Standards. The technique, however, is the best available for the purpose at this time, and has two advantages; it is relatively simple and can be easily learned. If the precision of the method were to be increased, and simultaneously if the "fibers" counted were truly asbestos, then the disadvantages of this relatively simple method would be overcome to a large degree.

The NIOSH technique has adopted the criteria of a 3:1 aspect ratio and >5 μm in length for identifying and counting of fibers. Specialists in grinding techniques have known that particulates of 3:1 aspect ratio could be created with some minerals. As well, nonasbestos fibrous materials were known to occur in association with asbestos materials, e.g., nemalite and brucite with chrysotile. Specificity of asbestos fibers is therefore very important.

Electron microscopic studies by the U.S. Bureau of Mines[2] showed that of the nonasbestiform varieties, such as anthophyllite, tremolite and hornblende, 95% produced cleavage fragments with an aspect ratio smaller than 10:1 and 70% smaller than 3:1. On the other hand, the majority of commercially milled chrysotile asbestos had an aspect ratio greater than 10:1. This would suggest that increasing the aspect ratio to >3:1 could significantly improve the discrimination between true fibers and nonfibrous particulates for counting purposes.

A study to observe the effect of increasing the aspect ratio was suggested. It was hoped that this increase would allow greater discrimination between true fibers and nonfibrous particulates.

A round robin series was begun to test this hypothesis. Objectives of the initial series were to indicate problem areas and also to point the way to a more refined round robin series that would be statistically designed to allow for an analysis of variance. A parallel study of fiber count and identification on the same samples was included in the initial series. Two government, three research, and six industrial laboratories participated in the study.

Counting Criteria and Experimental Method

The technique for counting generally followed the NIOSH method using a membrane filter. Two samples from each of six plants were obtained on membrane

0077-8923/79/0330-0661 $1.75/0

TABLE 1
ORIGIN OF FILTER SAMPLES BY INDUSTRY

Sample No.	Industry
1–2	Asbestos–Cement
3	Brake linings (carding)
4	Brake linings (press)
5–8	Mining (mill)
9–10	Textile (twister)
11–12	Textile (blender)

filters taken from different machines. The types of industry from which the samples were taken are shown in TABLE 1.

Each filter was whole or had had one or two wedge-shaped samples removed from it. A sample from each of the twelve membrane filters was sent to five participating laboratories and the sixth wedge was counted by all six mining laboratories. Each laboratory mounted and counted the sample, using its standard technique and criteria, except that it was asked to count "respirable" fibers (less than 3 μm diameter) and to allocate them to the seven groups defined by length and aspect ratios in TABLE 2. Each laboratory was asked to examine every sample with two counters. The laboratory microscopes are described in TABLE 3.

One group of samples, similar to those used for the optical microscope count, was also sent to the Ontario Research Foundation.[4] The particulates were counted and identified by transmission electron microscopy and compared to the count obtained by phase contrast optical measurement.

RESULTS AND DISCUSSION

Phase Contrast Microscopy

Fiber Count

The results were received with fibers classified into the first seven groups (TABLE 4) as well as a field count. Also shown is a summary of the results on all twelve filters as means and standard deviations expressed as a percentage of the total count. The percentage of the variations (as standard deviations) and fiber/unit area is about 10% greater than on fibers as parts per total; this is presumably due to variations in filter density and/or visual acuity of counters.

TABLE 2
GROUP CLASSIFICATIONS[3]

Length \ Aspect ratio	3:1 to 5:1	5:1 to 10:1	>10:1
5 to 10 μm	1	2	3
10 to 50 μm	4	5	6
> 50 μm			7

MICROSC

Counter Code*	Mic
I A1	Bausch
B2, 3	Bausch
C4, 5	Bausch
D6, 7	Bausch
E8	Bausch
F9	Bausch
G G10, 11, 12	Petro Ze
H13, 14	Olympus
I15, 16, 17	Dynasco
R J18	
J19	Zeiss
K20	Leitz

*I Industry, G Governme

Effect of Length

The analysis of fibers

Effect on Aspect Ratio

The analysis of fibers
It can be seen that the
the length groups (TABLE

MEAN AS PERCENTAG

Length \ Aspect ratio	3:1
5 to 10 μm	
10 to 50 μm	
> 50 μm	
Totals	16

STANDARD DEVIA

Length \ Aspect ratio
5 to 10 μm
10 to 50 μm
> 50 μm

TABLE 3
MICROSCOPE TYPE AND RELATED DATA USED IN COUNTING[3]

Counter Code*	Microscope	Magnification	Graticule Type	Graticule Size μm^2	Diameters Defined
I A1	Bausch & Lomb	400X	Porton	3000	
B2, 3	Bausch & Lomb	400X	Porton	3000	
C4, 5	Bausch & Lomb	400X	Porton	3000	
D6, 7	Bausch & Lomb	430X	Porton	2500	
E8	Bausch & Lomb	400X	Porton	3000	
F9	Bausch & Lomb	430X	Porton	2500	
G G10, 11, 12	Petro Zeiss	500X	Circular	45000	
H13, 14	Olympus		Porton	3310	
I15, 16, 17	Dynascope 70	400X	Porton	4030	
R J18		400X		5500	as mean
I* J19	Zeiss	500X	Patterson	3440	at midpoint
K20	Leitz	500X	Porton	3310	

*I Industry, G Government, R Research; Labs A to K; Counters 1-20.

Effect of Length

The analysis of fibers into groups by length is shown in TABLE 5.

Effect on Aspect Ratio

The analysis of fibers by aspect ratio is shown in TABLE 5.
It can be seen that the variability of the aspect ratio groups is much greater than the length groups (TABLE 6).

TABLE 4
MEAN AS PERCENTAGE OF TOTAL COUNT FROM ANALYSIS OF FIBER/SQ MM[3]

Length \ Aspect ratio	3:1 to 5:1	5:1 to 10:1	>10:1	Total
5 to 10 μm	13.8%	18.7%	24.1%	56.6%
10 to 50 μm	2.9	7.9	26.6	37.4
> 50 μm	—	—	5.9	5.9
Totals	16.7%	26.6%	56.6%	99.9%

STANDARD DEVIATION AS A PERCENTAGE OF TOTAL COUNT FROM ANALYSIS OF FIBERS/SQ MM[3]

Length \ Aspect ratio	3:1 to 5:1	5:1 to 10:1	>10:1
5 to 10 μm	23.6%	13.4%	19.1%
10 to 50 μm	8.6	8.7	13.5
> 50 μm	—	—	6.0

TABLE 5
ANALYSIS OF ASPECT RATIOS[3]

	Percentage of Fibers by Aspect Ratio		
	3:1 to 5:1	5:1 to 10:1	>10:1
Analysis by Proportion of Total			
Mean Overall	18.2	27.4	54.4
Standard Deviation	16.5	11.2	22.1
S.D./Mean	0.91	0.41	0.41
Analysis by Fibres/Unit Area			
Mean Overall	16.7	26.6	56.7
Standard Deviation	20.8	16.5	34.0
S.D./Mean	1.25	0.62	0.60
Range of Slide Means	9 to 37	21 to 34	38 to 68

The interpretation of the results are discussed for each length region below:

(a) *Long fibers: >50 μm*

All fibers less than 3 μm diameter and longer than 50 μm had aspect ratios greater than 10:1. There was only one group in this study (No. 7). Generally, 1 to 2% of the fibers were longer than 50 μm on the textile mill slides. Nearly all the individual fibers seen on the slides had a diameter of less than 3 μm, although some bundles apparently had greater diameters.

(b) *Medium length fibers: 10–50 μm*

The fibers in this category formed 20 to 30% of the total number of the mining and asbestos cement slides and 40 to 50% of the brake linings and textile slides. The overall average was 34%.

The counters disagreed considerably on the allocation of fibers across the three aspect ratio regions.

TABLE 6
ANALYSIS OF LENGTH[3]

	Percentage of Fibers by Length		
	5 to 10 μm	10 to 50 μm	>50 μm
Analysis by Proportion of Total			
Mean Overall	61.6	34.0	4.4
Standard Deviation	10.8	10.7	3.1
S.D./Mean	0.17	0.31	0.75
Analysis by Fibers/Unit Area			
Mean Overall	56.6	37.4	5.9
Standard Deviation	24.6	18.3	6.0
S.D./Mean	0.47	0.49	1.0
Range of Slide Means	28 to 77	21 to 51	1 to 12

(c) *Short fibers: 5–10 μm*

The fibers in this category formed
sbestos cement slides and 28 to 58
overall mean was 62% with a standar

Difficulties E

The difficulties noted by the co
found-robin so that we may avoid so
in the preliminary study. Their comm

1. Assigning aspect ratios for fib
fiber diameter was small.
2. Some of the slides were too d
light causing counting difficult

SUMMARY OF O

Sample	Fibers Counted	Fi
Asbest 1	208	
Asbest 2	45	
Lake 3CP	225	
Lake 2FF	194	
2K258 (T1)	204	
2K215 (T2)	202	
2K326 (T2)	214	
CCM 16.0.2	200	
CCM 16.0.3	203	
Atlas 1	207	
Atlas 2	119	

3. Many of the particulates in 3:1
based on their prismatic appear
4. It was found to be much more
than its length.
5. The major problem in judging l
the marks on the graticule to t
that is dependent on both count

IDENTIFICATION AND COUNTING F

Problems were encountered with t
this problem was overcome.
Up to 10 grid squares were count

(c) *Short fibers: 5–10 μm*

The fibers in this category formed 70 to 76% of the total number of the mining and asbestos cement slides and 28 to 58% of the brake linings and textiles slides. The overall mean was 62% with a standard deviation of 11%.

Difficulties Encountered in Counting

The difficulties noted by the counters are important in planning a new major round-robin so that we may avoid some of the ambiguities and problems encountered in the preliminary study. Their comments are noted below:

1. Assigning aspect ratios for fibers 5 to 10 μm in length was difficult when the fiber diameter was small.
2. Some of the slides were too dense (too many fibers/unit area) and some too light causing counting difficulty.

TABLE 7

SUMMARY OF OPTICAL FIBER COUNT DATA[3]

Sample	Fibers Counted	Fields Scanned	Area Scanned, mm^2	Fiber Density, Fibers/mm^2
Asbest 1	208	155	0.513	405
Asbest 2	45	350	1.159	39
Lake 3CP	225	350	1.159	194
Lake 2FF	194	350	1.159	167
JK258 (T1)	204	26	0.086	2372
JK215 (T2)	202	52	0.172	1174
JK326 (T2)	214	40	0.132	1621
CCM 16.0.2	200	155	0.513	390
CCM 16.0.3	203	69	0.228	890
Atlas 1	207	193	0.639	324
Atlas 2	119	350	1.159	103

3. Many of the particulates in 3:1 and 5:1 aspect ratio groups were not chrysotile based on their prismatic appearance, yet all fibers were counted as asbestos.
4. It was found to be much more difficult to determine the aspect ratio of a fiber than its length.
5. The major problem in judging length was the failure to adjust the dimensions of the marks on the graticule to the exact length required. This can lead to a bias that is dependent on both counter and microscope.

IDENTIFICATION AND COUNTING BY TRANSMISSION ELECTRON MICROSCOPY (TEM)[4]

Problems were encountered with the slides from which solvent had evaporated but a problem was overcome.

Up to 10 grid squares were counted, if required, to obtain a total of 100 fibers.

TABLE 8

SUMMARY OF ROUTINE FIBER COUNTS FOR ALL FIBERS[4]

Sample	Fiber Concentration (Fibers/ml)** Mean	95% Confidence Interval	Estimated Mass Concentration (Nanograms/mm³)	Concentration Equivalent to 1 Fiber Detected (Fibers/ml)	Fiber Type*
Asbest 1	230	160-300	3800	1.37	C
	<1.4			1.37	OF
Asbest 2	150	110-190	4700	1.40	C
	1.4			1.40	OF
Lake 3CP	320	240-390	45000	1.57	C
	9.4			1.57	OF
Lake 2FF	200	130-270	2700	1.29	C
	1.3			1.29	OF
2K258 (T1)	937	780-1100	22000	7.38	C
	<7.4			7.38	OF
2K215 (T2)	860	510-1200	36000	7.72	C
	7.7			7.72	OF
2K326 (T2)	870	500-1200	20000	8.28	C
	17			8.28	OF
CCM 16.0.2	750	660-840	26000	7.25	C
	<7.3			7.25	OF
CCM 16.0.3	840	380-1300	18000	7.63	C
	<7.7			7.63	OF
Atlas 1	150	100-190	16000	1.28	C
	<1.3			1.28	OF
Atlas 2	96	57-130	1000	0.98	C
	<0.98			0.98	OF

**Concentration assuming 1 m³ sampling volume and 1/6th filter supplied.
*C = Chrysotile, OF = Other Fibers (Mineral particulate >3:1 aspect ratio).

Chrysotile was identified by morphology (tubular structure) and occasional checks were made by electron diffraction and energy dispersive x-ray analysis of selected areas of the fibers. Other mineral fibers with greater than 3:1 aspect ratio were counted also. Fibers longer than 5 μm were counted and tabulated separately.

RESULTS AND DISCUSSION

A summary of the optical fiber count data is shown in TABLE 7 and a summary of the relevant fiber counts and identification is shown in TABLE 8.

Practically all the fibers were chrysotile and therefore the other fibers will not be considered further in this discussion.

It is interesting to note that, in the higher size range, large variations occurred between the different samples.

The larger fibers, >5 μm, which had been counted separately, produced the results shown in TABLE 9. A comparison of the fiber count by optical microscope vs. TEM is graphically shown in FIGURE 1. A relationship definitely appears to exist between the two methods. The correlation coefficient of 0.978 is highly significant.[4]

No such relationship exists betwee
length, FIGURE 2. This is partially attri
being more dispersed than others becau

The majority of fibers counted by
results of fibre count analysis for two sa
and CCM16-02 respectively. Each tabl
fibers. In TABLE 11, about 55% of the
41% between 5:1 and 10:1 and 4% with

In TABLE 10, fibers with a greater t
the total fibers, the percentage of fibers
about 24%, and less than 1% have fibers

Co

Results of the preliminary round rob
optical microscope is increased when a
relative error decreased from 93 to 4
decreased by approximately 45%.

It was previously recognized that the
be counted, particularly in the very sm
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The vast majority of fibers counted
greater than 10:1. This was true for all s
of the total fibers counted in each sampl
ratio to greater than 5:1 should increase
contrast microscope technique, particula

T

COMPARATIVE SUM

Sample	Fiber Count in Fil: Optical >5 μm	TEM >
Asbest 1	405	2.4 ×
Asbest 2	39	9.7 ×
Lake 3CP	194	6.9 ×
Lake 2FF	167	9.5 ×
2K258 (T1)	2372	2.0 ×
2K215 (T2)	1174	7.9 ×
2K326 (T2)	1621	9.4 ×
CCM 16.0.2	390	1.8 ×
CCM 16.0.3	890	3.5 ×
Atlas 1	324	3.8 ×
Atlas 2	103	2.3 ×

*Large area of grid separately scanned for fi

No such relationship exists between the two methods for fibers less than 5 μm in length, FIGURE 2. This is partially attributed to the origin of the samples, some fibers being more dispersed than others because of their industrial origin.

The majority of fibers counted by TEM had an aspect ratio exceeding 10:1. The results of fibre count analysis for two samples are shown in TABLES 10 and 11, 2K326 and CCM16-02 respectively. Each table contained a total count of approximately 100 fibers. In TABLE 11, about 55% of the fibers have an aspect ratio greater than 10:1, 41% between 5:1 and 10:1 and 4% with less than 5:1.

In TABLE 10, fibers with a greater than 10:1 aspect ratio amount to about 76% of the total fibers, the percentage of fibers between 10:1 and 5:1 aspect ratio amount to about 24%, and less than 1% have fibers with an aspect ratio of less than 5:1.

CONCLUSION

Results of the preliminary round robin indicate that fiber count precision using the optical microscope is increased when a 10:1 aspect ratio is used instead of 3:1. The relative error decreased from 93 to 42% although the number of fibers counted decreased by approximately 45%.

It was previously recognized that the optical microscope did not allow all fibers to be counted, particularly in the very small lengths and diameters. Using a TEM, a relationship was found to exist between the fiber count for optical microscope phase contrast technique and that by TEM, for fibers >5 μm in length. The ratio of fibers counted to fibers present is about 1:50 respectively. No such relationship exists for fibers in the range of less than 5μm in length; however, in these shorter lengths an average fiber count ratio of 1:1000 was found.[4]

The vast majority of fibers counted by TEM, >5 μm long, has aspected ratios greater than 10:1. This was true for all samples, ranging from about 60% to over 80% of the total fibers counted in each sample.[4] It would appear that increasing the aspect ratio to greater than 5:1 should increase the precision of the fiber count by the phase contrast microscope technique, particularly because of the relationship found between

TABLE 9

COMPARATIVE SUMMARY OF FIBER COUNTS[4]

Sample	Fiber Count in Fibers/mm² on Original Membrane Filter			
	Optical >5 μm	TEM >5 μm*	TEM all Fibers	TEM all Fibers, 95% Confidence
Asbest 1	405	2.4×10^4	3.8×10^5	$(2.70–5.00) \times 10^5$
Asbest 2	39	9.7×10^3	2.5×10^5	$(1.80–3.20) \times 10^5$
Lake 3CP	194	6.9×10^3	5.3×10^5	$(4.00–6.50) \times 10^5$
Lake 2FF	167	9.5×10^3	3.3×10^5	$(2.20–4.50) \times 10^5$
2K258 (T1)	2372	2.0×10^5	1.6×10^6	$(1.33–1.88) \times 10^5$
2K215 (T2)	1174	7.9×10^4	1.4×10^6	$(0.85–2.00) \times 10^6$
2K326 (T2)	1621	9.4×10^4	1.5×10^6	$(0.86–2.06) \times 10^5$
CCM 16.0.2	390	1.8×10^4	1.3×10^6	$(1.14–1.45) \times 10^6$
CCM 16.0.3	890	3.5×10^4	1.4×10^6	$(0.63–2.20) \times 10^6$
Atlas 1	324	3.8×10^3	2.5×10^5	$(1.70–3.20) \times 10^5$
Atlas 2	103	2.3×10^3	1.6×10^5	$(0.95–2.20) \times 10^5$

*Large area of grid separately scanned for fibers, >5 μm only, to increase statistical validity.

Mines Ltd., East Broughton, Qu
Lake Asbestos of Quebec Ltd.,
Association, Thetford Mines, Qu

The results of a study of the
counting method are reported. Tl

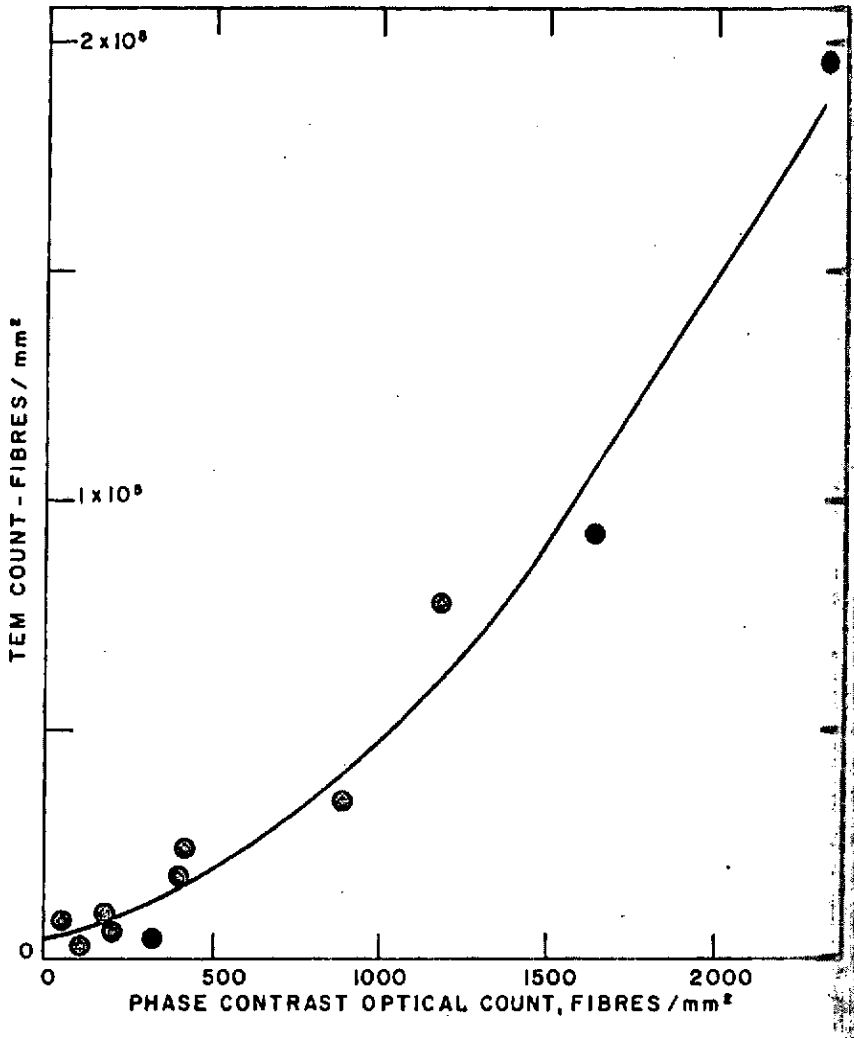

FIGURE 1. Relationship between TEM count and phase contrast optical count for fibers longer than 5 μm[4]

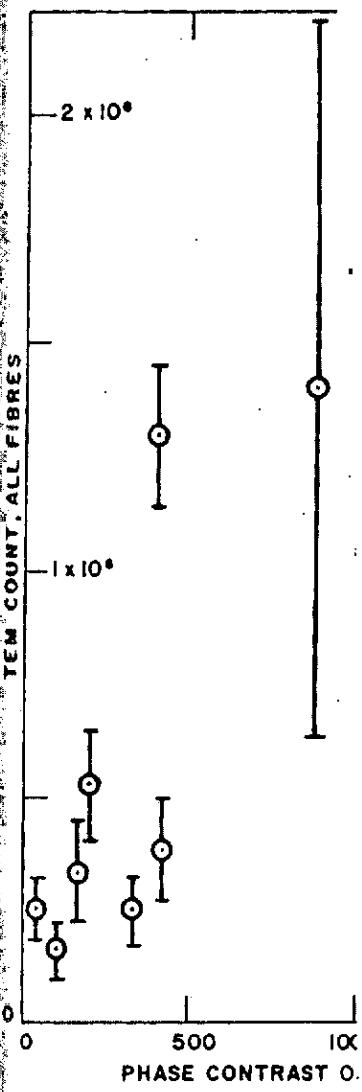

FIGURE 2. Relationship between TEI

the TEM and optical microscope methods. This can be verified in the next round robin series. It is hoped that the second series, which is now being statistically designed, will avoid some of the problems encountered in the present study.

The research and government organizations participating in the preliminary round robin series were: Environment Canada, Ottawa; Environmental Protection Service, Montreal, Quebec; McGill University, Montreal, Quebec; Ontario Research Foundation, Mississauga, Ontario; and U.S. Bureau of Mines, Particulate Center, College Park, Maryland.

The industrial laboratories participating were: Asbestos Corporation Ltd., Thetford Mines, Quebec; Bell Asbestos Ltd., Thetford Mines Quebec; Carrey Canadian

Mines Ltd., East Broughton, Quebec; Canadian Johns-Manville, Asbestos, Quebec; Lake Asbestos of Quebec Ltd., Black Lake, Quebec; and Quebec Asbestos Mining Association, Thetford Mines, Quebec.

SUMMARY

The results of a study of the inter-laboratory precision obtained by the NIOSH counting method are reported. Three different aspect ratios (a) 3:1, (b) 5:1, and (c)

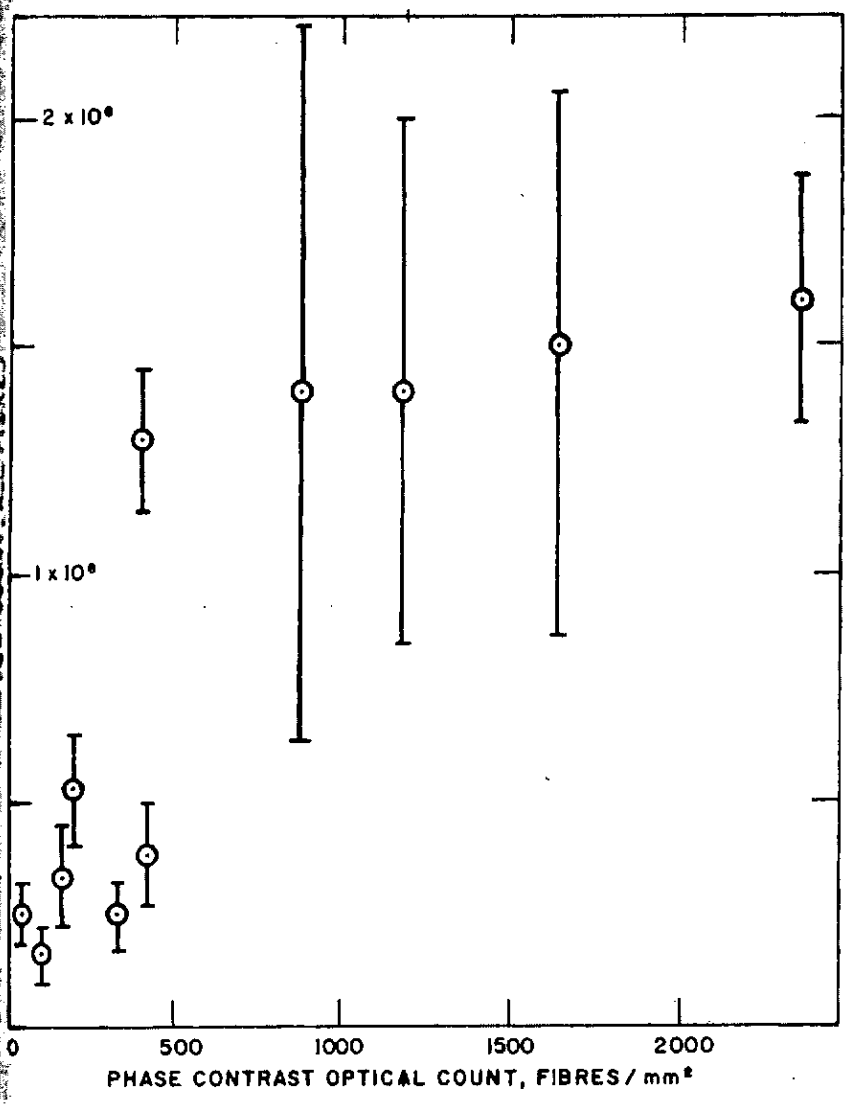

FIGURE 2. Relationship between TEM count of all fiber lengths and optical fiber count.[4]

TABLE 10

ASBESTOS FIBER COUNT ANALYSIS: CHRYSOTILE RAW SIZING DATE[a]

Sample: 2K326 (T2)								
Length μm	Width μm	Aspect Ratio	Length μm	Width μm	Aspect Ratio	Length μm	Width μm	Aspect Ratio
0.82	0.045	18.0	1.41	0.136	10.3	0.59	0.091	6.5
0.27	0.045	6.0	0.36	0.045	8.0	1.18	0.091	13.0
2.09	0.136	15.3	2.68	0.136	19.7	0.73	0.045	16.0
2.50	0.045	55.0	0.68	0.045	15.0	0.50	0.045	11.0
0.41	0.045	9.0	0.55	0.045	12.0	1.14	0.091	12.5
1.18	0.136	8.7	0.95	0.091	10.5	0.55	0.045	12.0
3.27	0.045	72.0	1.05	0.273	3.8	0.95	0.045	21.0
1.41	0.136	10.3	0.73	0.045	16.0	0.45	0.091	5.0
1.86	0.091	20.5	2.86	0.045	63.0	0.27	0.045	6.0
1.45	0.136	10.7	0.45	0.045	10.0	0.32	0.045	7.0
2.77	0.136	20.3	0.27	0.045	6.0	0.73	0.045	16.0
1.18	0.045	26.0	1.86	0.045	41.0	1.59	0.182	8.7
2.05	0.136	15.0	3.27	0.091	36.0	1.18	0.045	26.0
2.09	0.136	15.3	1.32	0.045	29.0	1.45	0.045	32.0
0.59	0.045	13.0	5.59	0.045	123.0	6.23	0.045	137.0
5.73	0.091	63.0	6.23	0.136	45.7	4.64	0.091	51.0
1.45	0.045	32.0	1.64	0.045	36.0	2.95	0.045	65.0
0.73	0.091	8.0	0.73	0.045	16.0	2.05	0.091	22.5
20.55	0.045	452.0	0.55	0.045	12.0	0.27	0.045	6.0
0.95	0.045	21.0	0.36	0.045	8.0	0.32	0.045	7.0
1.64	0.091	18.0	7.36	0.045	162.0	2.91	0.045	64.0
0.73	0.045	16.0	0.91	0.045	20.0	18.18	0.045	400.0
0.32	0.045	7.0	1.00	0.045	22.0	0.73	0.045	16.0
0.36	0.045	8.0	0.73	0.045	16.0	1.14	0.136	8.3
4.14	0.045	91.0	2.09	0.045	46.0	0.64	0.091	7.0
1.18	0.182	6.5	1.36	0.091	15.0	0.32	0.045	7.0
0.73	0.045	16.0	7.45	0.045	164.0	0.59	0.091	6.5
1.18	0.045	26.0	5.64	0.045	124.0	1.41	0.136	10.3
0.64	0.045	14.0	0.32	0.045	7.0	0.73	0.045	16.0
0.55	0.045	12.0	0.36	0.045	8.0	1.00	0.091	11.0
0.36	0.045	8.0	1.23	0.045	27.0	1.50	0.045	33.0
1.00	0.091	11.0	0.82	0.045	18.0	1.09	0.045	24.0
2.09	0.045	46.0	1.59	0.045	35.0	0.91	0.045	20.0
0.59	0.045	13.0	0.27	0.045	6.0	4.82	0.091	53.0
0.59	0.045	13.0	0.95	0.136	7.0	1.27	0.045	28.0

10:1, were applied as fiber counting criteria. Maximum and minimum length covering the following ranges were also applied as criteria for the choice of particles to be counted: (a) 5 to 10 μm (b) 10 to 50 μm (c) >50 μm. Twelve laboratories (6 industry, 2 government and 3 research) counted two samples from each of 6 plants (1 asbestoscement, 1 brake linings, 2 ore treating mills, 2 textiles).

Results obtained are summarized hereunder:

Counting Criteria		Mean Count	Coefficient of
Aspect Ratio	Min. Length	%	Variation
3:1	5 μm	100	0.43
5:1	5 μm	80	0.38
10:1	5 μm	53	0.54
3:1	10 μm	38	0.49

It appears that a 5:1 aspect ratio i
precision than 10 μm minimum length.
(ORF) of the same samples at high resol
between transmission electron microsc
using fibers >5 μm in length. The major
of >10:1, a much smaller amount was
number of fibers with an aspect ratio of

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ASBESTOS FIBER COUNT ANALY

			Sample
Length μm	Width μm	Aspect Ratio	Length μm
1.68	0.045	37.0	0.95
2.73	0.045	60.0	0.91
0.23	0.045	5.0	0.59
0.41	0.091	4.5	0.59
5.50	0.045	121.0	0.55
1.55	0.045	34.0	0.36
0.59	0.091	6.5	0.73
1.05	0.182	5.7	0.41
0.41	0.045	9.0	2.59
0.59	0.045	13.0	0.86
1.05	0.136	7.7	1.09
0.32	0.045	7.0	0.32
0.86	0.091	9.5	0.41
0.73	0.045	16.0	0.27
0.23	0.045	5.0	0.64
0.64	0.045	14.0	8.05
0.50	0.045	11.0	0.36
1.14	0.091	12.5	1.68
0.36	0.045	8.0	3.09
0.50	0.045	11.0	0.95
0.77	0.045	17.0	0.36
0.68	0.045	15.0	0.36
0.41	0.045	9.0	1.86
3.23	0.136	23.7	0.23
0.41	0.045	9.0	0.27
0.50	0.045	11.0	0.59
0.32	0.045	7.0	1.27
2.41	0.045	53.0	1.77
0.50	0.045	11.0	0.50
0.68	0.091	7.5	2.09
0.32	0.045	7.0	1.00
0.32	0.045	7.0	3.50
0.32	0.045	7.0	0.55
1.05	0.091	11.5	1.14
0.41	0.045	9.0	

ACKN

Appreciation and thanks are exten
particularly to G. Knight and M. Trud
ing samples and statistical data.

It appears that a 5:1 aspect ratio is better than 3:1 and 5 μm leads to better precision than 10 μm minimum length. However, a parallel study at one laboratory (ORF) of the same samples at high resolution indicated that correlation was excellent between transmission electron microscopy fibre count vs. visual microscope count using fibers >5 μm in length. The majority of the fibers were in the aspect-ratio class of >10:1, a much smaller amount was in the aspect ratio class of >5:1<10:1; the number of fibers with an aspect ratio of less than 5:1 was very small.

TABLE 11

ASBESTOS FIBER COUNT ANALYSIS: CHRYSOTILE RAW SIZING DATA[4]

Sample: CCM 16.0.2								
Length μm	Width μm	Aspect Ratio	Length μm	Width μm	Aspect Ratio	Length μm	Width μm	Aspect Ratio
1.68	0.045	37.0	0.95	0.091	10.5	1.41	0.091	15.5
2.73	0.045	60.0	0.91	0.045	20.0	0.27	0.045	6.0
0.23	0.045	5.0	0.59	0.045	13.0	1.36	0.091	15.0
0.41	0.091	4.5	0.59	0.091	6.5	0.41	0.091	4.5
5.50	0.045	121.0	0.55	0.045	12.0	0.23	0.045	5.0
1.55	0.045	34.0	0.36	0.045	8.0	0.50	0.045	11.0
0.59	0.091	6.5	0.73	0.091	8.0	1.41	0.091	15.5
1.05	0.182	5.7	0.41	0.045	9.0	3.00	0.273	11.0
0.41	0.045	9.0	2.59	0.045	57.0	3.95	0.045	87.0
0.59	0.045	13.0	0.86	0.091	9.5	0.36	0.045	8.0
1.05	0.136	7.7	1.09	0.227	4.8	0.73	0.091	8.0
0.32	0.045	7.0	0.32	0.045	7.0	0.64	0.182	3.5
0.86	0.091	9.5	0.41	0.091	4.5	0.50	0.045	11.0
0.73	0.045	16.0	0.27	0.045	6.0	1.23	0.045	27.0
0.23	0.045	5.0	0.64	0.045	14.0	0.86	0.045	19.0
0.64	0.045	14.0	8.05	0.045	177.0	0.41	0.045	9.0
0.50	0.045	11.0	0.36	0.045	8.0	0.73	0.091	8.0
1.14	0.091	12.5	1.68	0.045	37.0	0.73	0.091	8.0
0.36	0.045	8.0	3.09	0.273	11.3	0.86	0.091	9.5
0.50	0.045	11.0	0.95	0.091	10.5	0.64	0.045	14.0
0.77	0.045	17.0	0.36	0.045	8.0	0.68	0.091	7.5
0.68	0.045	15.0	0.36	0.045	8.0	2.82	0.045	62.0
0.41	0.045	9.0	1.86	0.091	20.5	1.32	0.045	29.0
3.23	0.136	23.7	0.23	0.045	5.0	1.32	0.045	40.0
0.41	0.045	9.0	0.27	0.045	6.0	0.64	0.045	14.0
0.50	0.045	11.0	0.59	0.091	6.5	0.68	0.091	7.5
0.32	0.045	7.0	1.27	0.045	28.0	0.64	0.045	14.0
2.41	0.045	53.0	1.77	0.136	13.0	1.18	0.091	13.0
0.50	0.045	11.0	0.50	0.045	11.0	1.45	0.182	8.0
0.68	0.091	7.5	2.09	0.136	15.3	1.77	0.045	39.0
0.32	0.045	7.0	1.00	0.091	11.0	0.64	0.045	14.0
0.32	0.045	7.0	3.50	0.409	8.6	0.45	0.045	10.0
0.32	0.045	7.0	0.55	0.045	12.0	2.77	0.182	15.2
1.05	0.091	11.5	1.14	0.045	25.0	1.45	0.136	10.7
0.41	0.045	9.0						

ACKNOWLEDGMENTS

Appreciation and thanks are extended to the many participants in this study and particularly to G. Knight and M. Trudeau of QAMA for coordinating and summarizing samples and statistical data.

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INFLUENCE OF CR
MINERALS ON *I*

Lalita D. Palekar,† Charl

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The health hazards caused by ex
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of these minerals to which people n
activities can also have adverse effec
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of all minerals and their varieties
consuming project. A more practical
properties of minerals and their v
simultaneously in selected biological
be made to relate specific mineralogi

Several attempts have been mad
chemical properties of minerals w
revealed that hemolysis of mamm
area,[1,2] others have shown that it d
indicated a relationship to magnesi
strated that still another mineral ch
habit—is responsible for sheep eryth
cytotoxicity.[3]

The study is designed in an atte
characteristics in the biological sys
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studies, these minerals are ground
samples are characterized for chem
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*This report presents the results of
Contract Number 68-02-2566) and GCA
the United States Environmental Protect

0077-8923/79/03

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]

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

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

*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-

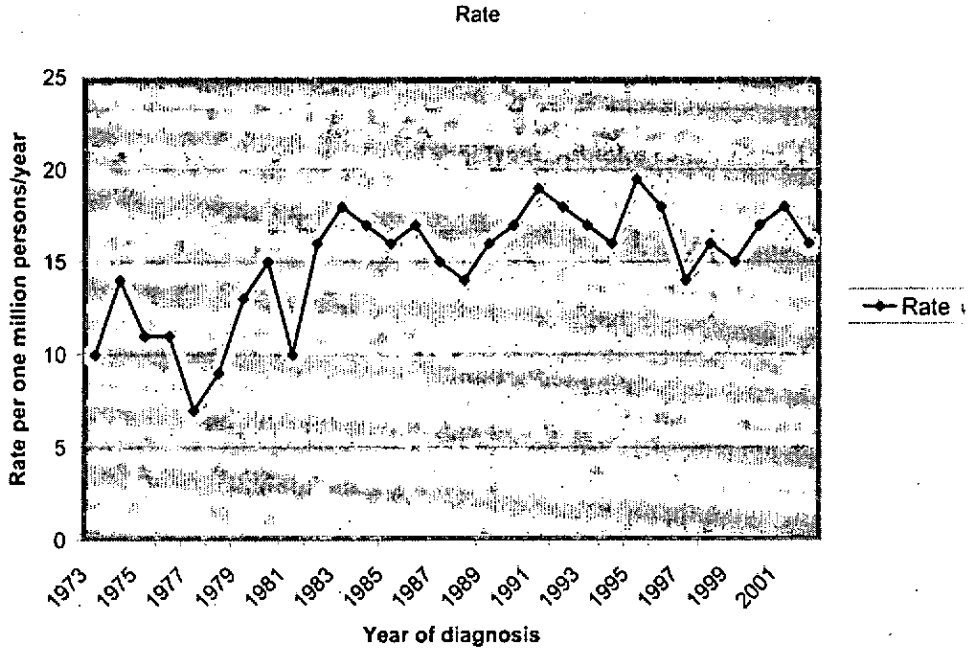

Figure 1—Women's Mesothelioma Rates age > 60, based on data reported in: Teta MJ, Mink PJ, Lau E, Sceurman BK, Foster ED. US mesothelioma patterns 1973-2002: indicators of change and insights into background rates. Eur J Cancer Prev 2008;17:525-34.

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

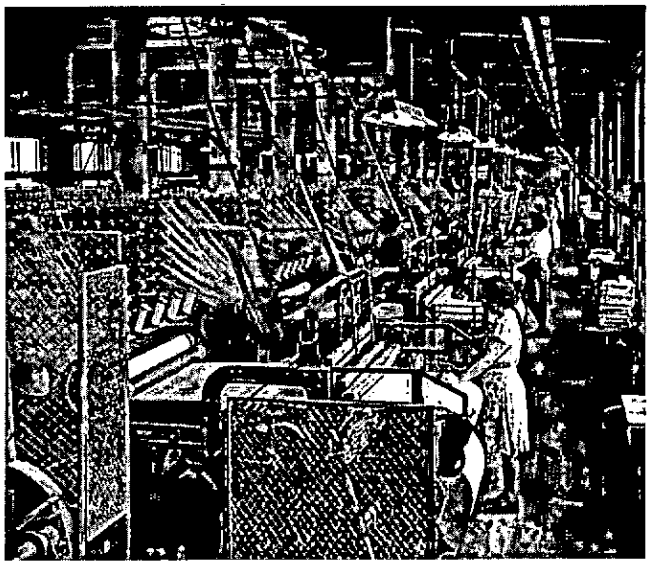

Figure 2—Female Asbestos Textile Workers, c. 1922. Reproduced from: "Garlock 2009" Slide Presentation. Olwin Moeller v. Garlock Sealing Technologies, LLC. Case Number: 3:07-CV-65-H. United States District Court, Western District of Kentucky at Louisville.

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.

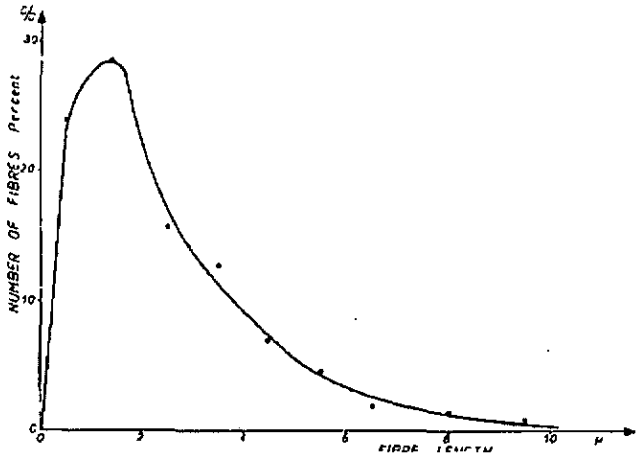

Figure 5—Relative Proportion of Short and Long Fibers in Lung Tissue. Reproduced from: LeBouffant L. Investigation and analysis of asbestos fibers and accompanying minerals in biological materials. Environ Health Perspect 1974;9:149-53.

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).

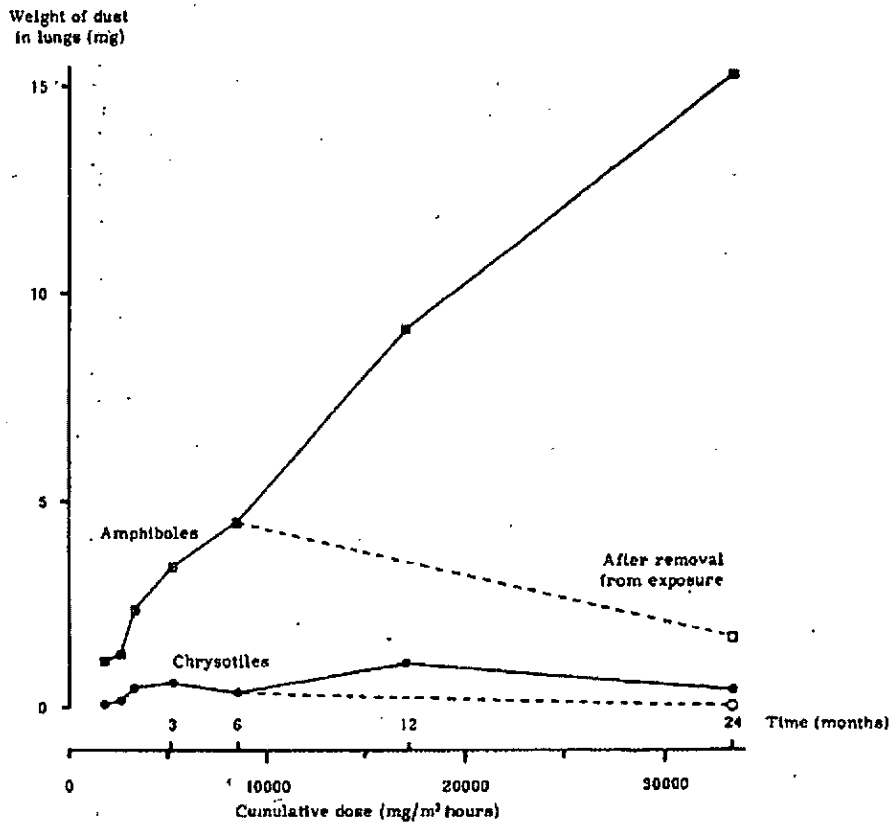

FIG. 9.—Mean weight of dust in lungs of rats in relation to dose and time.

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]

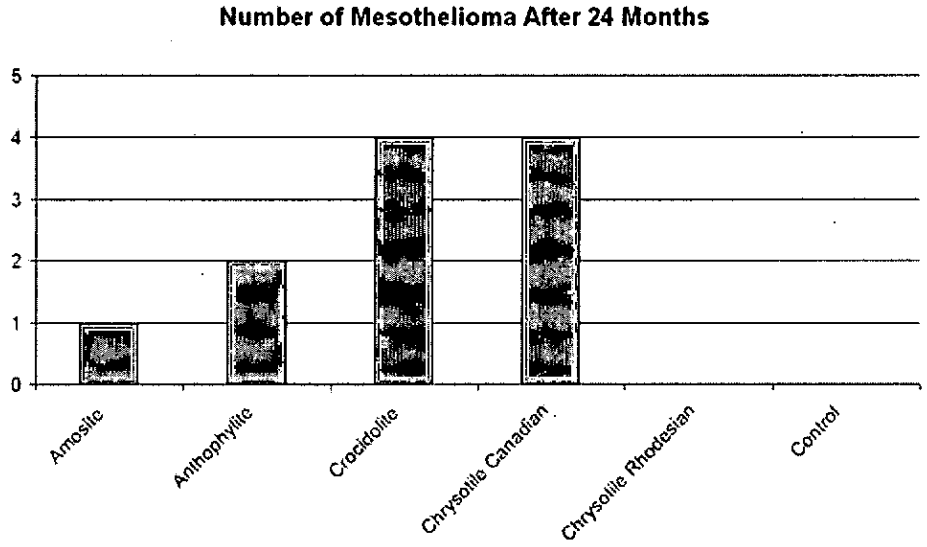

Figure 7—Relative Mesothelioma Potency by Fiber Type in Rats, based on data from: Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. Br J Cancer 1974;29:252-69.

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]

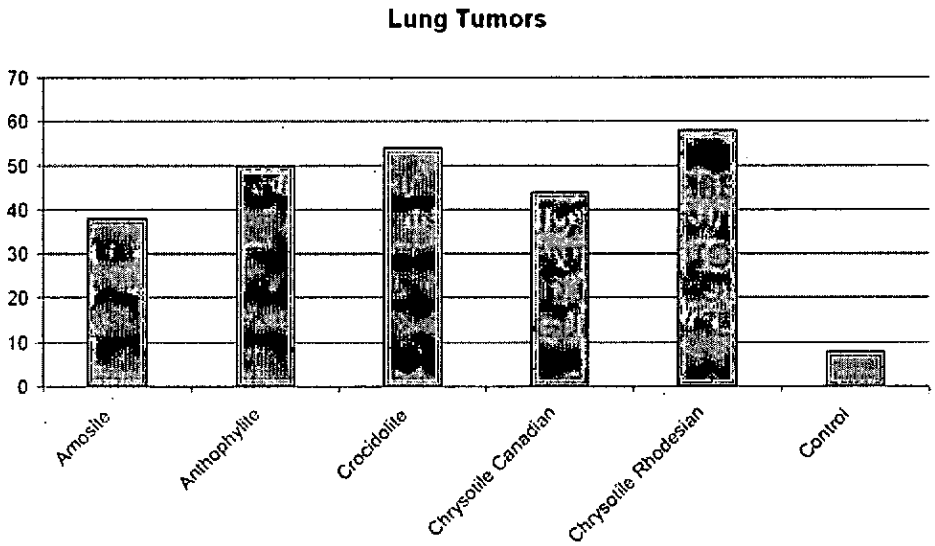

Figure 8—Relative Lung Tumor Potency by Fiber Type in Rats, based on data from: Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. Br J Cancer 1974;29:252-69.

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

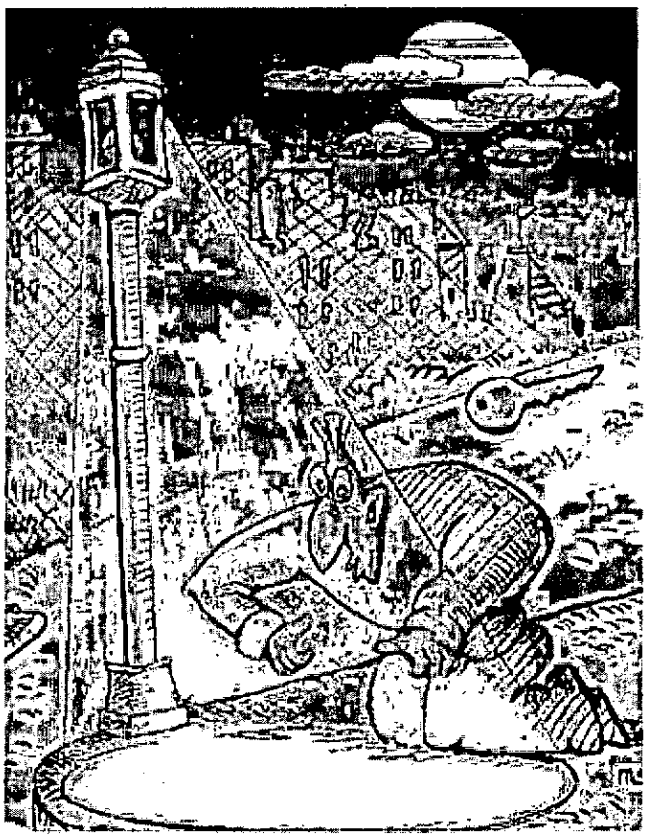

Figure 9—Looking Where the Light is Better. Art courtesy of Artt by Cartooncity.net.

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."

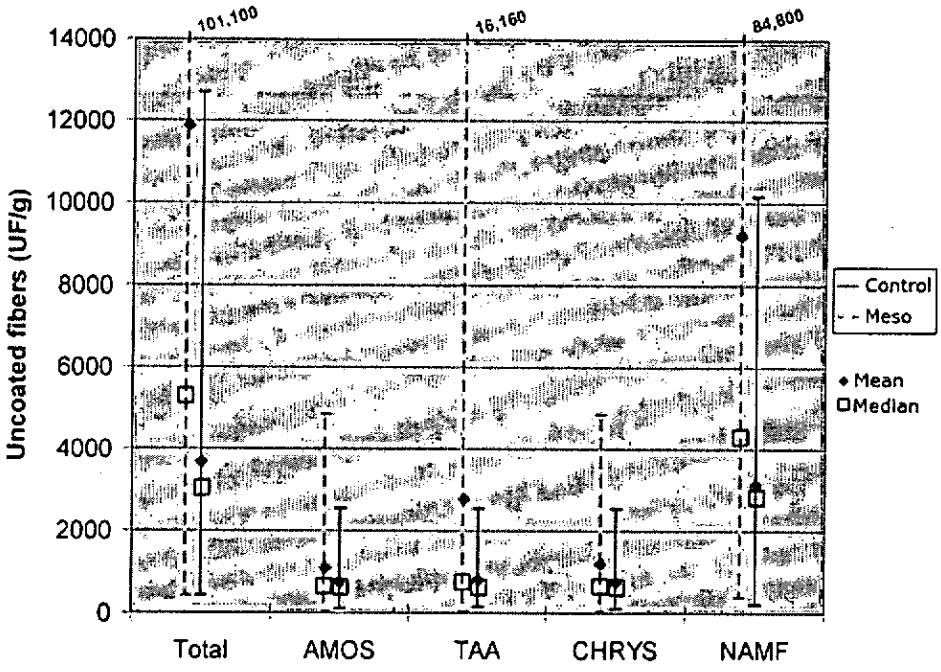

Figure 10—Medians, means, and ranges of uncoated fibers per gram of wet lung tissue in "controls" and mesothelioma cases, based on data from: Srebro SH, Roggli VL, Samsa GP. Malignant mesothelioma associated with low pulmonary tissue asbestos burdens: a light and scanning electron microscopic analysis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

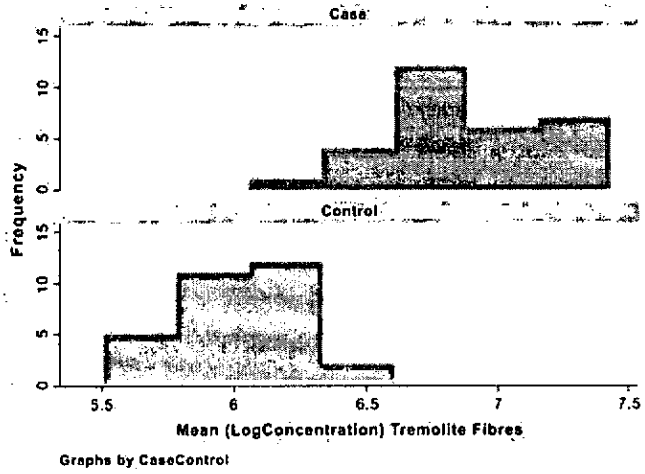

Figure 11—Tremolite levels of brake workers vs. controls, reproduced with permission from: Finkelstein MM. Asbestos fibre concentrations in the lungs of brake workers: another look. Ann Occup Hyg 2008;52:455-61.

> 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 lymphocytic 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

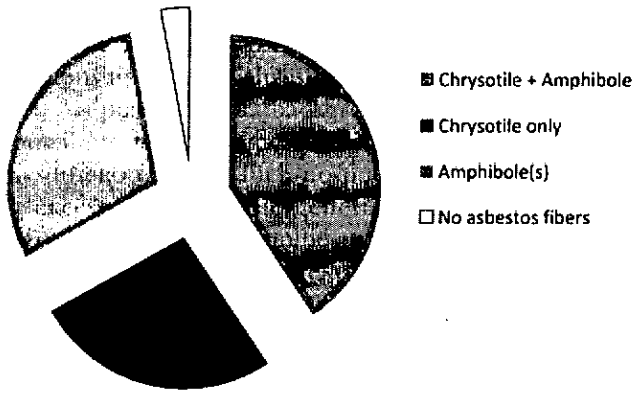

Figure 12—The predominant fibers in the lung, based on data from: Suzuki Y, Yuen SR. Asbestos tissue burden study on human malignant mesothelioma. Ind Health 2001;39:150-60. Comparison to Figure 13, which shows pleural fibers in the same patients, establishes that chrysotile goes to the pleura, while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

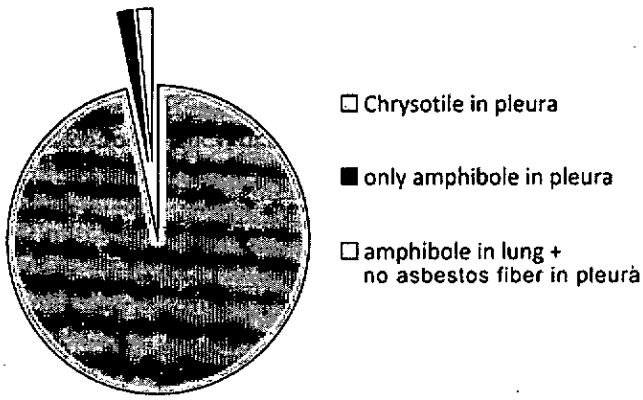

Figure 13—The predominant fibers in the pleura, based on data from: Suzuki Y, Yuen SR. Asbestos tissue burden study on human malignant mesothelioma. Ind Health 2001;39:150-60. Comparison to Figure 12, which shows lung fibers in the same patients, establishes that chrysotile goes to the pleura, while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

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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.