A commentary on Roggli's "The So-Called Short-Fiber Controversy"

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Dr. Victor Roggli republished a "literature review and critical analysis" of the toxicity of short fiber asbestos. His paper was originally prepared and presented at a conference of asbestos defense lawyers. His review omitted published papers that indicate that short fiber asbestos is a carcinogen. We critically review his paper.

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Recently Dr. Victor Roggli republished a "literature review and critical analysis" of the toxicity of short fiber asbestos.¹ He originally developed this review to present at a program arranged by The Defense Research Institute (DRI),¹ an organization run by and for defense lawyers. Roggli's target audience was lawyers who defend companies that mine, manufacture, and sell asbestos or asbestos-containing products. DRI advertised its program as allowing defense lawyers to:

Gain strategic insight from top experts and learn the latest developments in defending lung cancer cases [and] Keep on top of the latest developments in asbestos litigation! Hear about the latest medical advances in diagnoses and treatment. Learn how science impacts strategies and defenses. Attendance at this seminar will provide defendants, insurers, and attorneys with information to put to use immediately. Obtain knowledge from a cross-section of experts in a variety of sciences.

None of the lectures at DRI's program offered any opposing scientific perspectives. During his presentation, Roggli misrepresented the literature by omitting relevant papers that conflicted with his conclusion and libeled a dead researcher. In doing so, Roggli provides assistance in the litigation defense of companies who have been sued by workers that have asbestos-related diseases.

Roggli begins by claiming that Vorwald *et al.* concluded that "fibrogenicity" was related to fibers 20 μm or greater in length. He failed to note that Leroy Gardner's (who conducted the studies that Vorwald summarized and published after Gardner's sudden death) experiments were designed to test tissue reactions of several animals to various asbestos dusts in order to determine whether it was the chemical or the fibrous property of asbestos that results in asbestos toxicity.² Gardner incorporated four types of asbestos dust as part of his experiments: King's floats, short fiber, 100% ball milled, and long fiber.

Only 32.5% of the dust used in the "long fiber" inhalation experiments was fibrous and only 6.7 was longer than 10 μm . Less than 1% of the "short fiber" contained fibers greater than 10 microns. While Vorwald concluded that in comparison to the short fiber experiments "long fibers are chiefly responsible for asbestosis," hardly any of the short fibers were fibrous because Gardner ball milled the chrysotile to produce the test material. In addition, the ball milled asbestos caused "fibrosis typical of asbestosis was present 12 mo. after exposure ceased" in guinea pigs (36 month latency) but not after 24 months of exposure. The ball milled dose was 100 mppcf for first 7 months and 150 mppcf for the next 21 months (about 1/2 to 1/3 was $>10\mu\text{m}$ in length) while the long fiber dose ranged from 32 to 48 mppcf. Thus, Gardner's findings are more likely related to dose and latency rather than fiber length.

It is vital to recognize that Vorwald, at the request of the sponsoring brake manufacturing companies and MetLife Insurance Company, deleted results from Gardner's experiments that indicated asbestos was a carcinogen.³⁻⁵ Thus, Roggli's first citation in a paper prepared for asbestos defense lawyers was manipulated at the request of asbestos manufacturing company lawyers over 60 years ago.

Additionally, Roggli did not include an animal inhalation experiment published by King *et al.* in 1946. King *et al.* designed their experiment to test 'whether short-fibre asbestos is less damaging to the lungs of the rabbit than is long-fibre asbestos' specifically in light of Gardner's experiments.⁶ King *et al.* found that both long and short fibers produced fibrosis, but of different types: "[a]nimals in the long-fibre experiments developed a nodular reticulosis comparable with the experimental silicotic nodule," and "[a]nimals in the short-fibre experiment developed a diffuse interstitial reticulosis." They suggest that, on account of method, "[i]t is possible that the amount of asbestos fibres actually reaching the lung was very much smaller in amount in the case of Gardner's animals than our own."²

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Roggli then follows with citations from some, but not all, of Dr. Davis and colleagues' research.⁷⁻¹⁵ For example, peritoneal injection experiments by Bolton, Davis, and Donaldson state findings that contradicted Roggli's conclusion:

[T]he results showed that the chrysotile samples tended to be more carcinogenic than the amosite preparations and some of the possible reasons for this are discussed. It was concluded from estimations of particle size using the scanning electron microscope that relative fibre length did not provide a useful measure of the carcinogenicity of the seven dusts, owing in part to problems arising from the preparation of realistic, representative, samples for microscopy.¹⁶

The latter measurement issue relates to two of seven experimental dusts.

Roggli also stated that the Agency for Toxic Substances and Disease Registry (ATSDR)'s 2002 report regarding the World Trade Center "concluded that there was no convincing evidence that short fibers caused disease."¹⁷ This is not true; the report does not contain any such statement. It does include the following:¹⁷

On penetration:

For fibers less than 5 µm in length, Dr. Lippmann indicated, the information available on particle deposition and longer fibers suggests that fiber diameter likely has the greatest influence on deposition patterns. He noted that fibers less than 5 µm in length will have diameters less than 1.66 µm, assuming the aspect ratios are at least 3:1. This panelist estimated that 10–20% of short fibers with diameters between 0.1 and 1.6 µm will deposit in the lungs of healthy people. (Section 2–4)

This panelist then reviewed two preliminary studies of fiber translocation, one in humans (Boutin *et al.*²¹) and the other in goats, which were based on more robust methods using controls. He noted that one study found that 22.5% of fibers detected in the pleura were longer than 5 µm ... (Section 2–8)

Second, another panelist summarized the findings from a study of rats exposed via inhalation to kaolin-based refractory ceramic fibers with geometric mean length of 4.5 µm. The study reported that the fate of the fibers depended on fiber length: fibers in the pleural tissue 32 days after exposure had a geometric mean length of 1.5 µm and geometric mean diameter of 0.09 µm, while fibers in the parenchymal tissue were much larger with geometric mean length of 5 µm and geometric mean diameter of 0.3 µm. Thus, the study indicates that very thin fibers smaller than 5 µm – fibers that would not be counted by conventional phase contrast microscopy (PCM) asbestos sampling methods – are capable of translocating to the pleural tissue. (Section 2–9)

To illustrate this issue further, the panelist presented data on the distribution of fiber lengths measured in surgical lung tissue samples from six miners and four cement plant workers who were exposed to asbestos fibers (primarily chrysotile) and non-asbestos fibers. The men were hospitalized with various lung diseases,

which were mostly not related to their asbestos exposures. In these individuals, the majority (71%, by fiber count) of lung-retained chrysotile asbestos fibers were shorter than 5 µm, with lesser amounts (25%) of chrysotile asbestos fibers between 5 and 20 µm, and even lesser amounts (4%) of chrysotile asbestos fibers longer than 20 µm. A similar pattern was observed for the lung-retained non-asbestos fibers, with an even greater number of fibers shorter than 5 µm (85%) and none longer than 20 µm. (Section 2–11)

On the inferences that can be drawn from animal studies relative to human effects:

Role of laboratory animal studies in evaluating depositional patterns in humans. The panelists acknowledged that laboratory animal studies have provided additional insights on how fibers deposit in the lung, but the panelists noted that inter-species differences in lung airway structure limit the utility of the animal data. One indicated, for instance, that laboratory animal studies have the advantage of being able to characterize lung fiber burdens at different time frames following highly controlled dosage conditions. On the other hand, he added, airway branching patterns in humans are nearly symmetrical, while rats (and most other mammals) have asymmetrical branching patterns. Such differences in branching patterns influence the cross-sectional air flow profiles, which in turn affect fiber deposition behavior. Consequently, lung deposition patterns in laboratory animals are expected to differ from those in humans. (Section 2–4)

Another panelist showed how modeling results of lung deposition patterns support this expectation. Based on predictions of a mathematical lung dosimetry model developed by the International Commission on Radiological Protection, this panelist illustrated differences between rats and humans in estimated deposition fractions of fibers in alveolar regions. His figure indicated that the predicted deposition fraction in humans was greater than that in rats for all fiber lengths considered, and this difference was most striking for longer fibers. Specifically, the model predicted that virtually no fibers with aerodynamic diameters of 3 µm and aspect ratios of 10:1 deposit in the alveolar region of rats, while more than 25% of these same fibers are predicted to deposit in the alveolar region of humans. Such predictions, this panelist noted, raise questions about whether rats are good models for humans in terms of fiber deposition in the lung. (Section 2–1)

On human health effects:

Second, this panelist reviewed studies of gold mine workers in South Dakota who also were exposed to cumingtonite-grunerite asbestiform material. He indicated that an initial study of this cohort found increased mortality from malignant respiratory disease among workers with at least 5 years of exposure. A follow-up study of this same cohort which considered workers who had worked for 21 years or longer, found no such increase, but did report increased risks of silicosis and tuberculosis. Average exposure concentrations for this site were 4.82 (±0.68) fibers per cubic

centimeter, with 94% of airborne fibers being less than 5 µm in length. (Section 3–2)

On animal studies:

In the second study (Davis and Jones⁸), seven malignant cancers were observed among rats exposed via intraperitoneal injection to a "short" chrysotile fiber mixture, while 22 malignant cancers were observed among those exposed to the "long" fiber mixture. Cancers in the former group, however, have since been attributed to contamination of the "short" fiber samples with longer chrysotile fibers. (Section 3–6)

Roggli summarizes the findings of this Davis and Jones⁸ animal study, stating that there were "[c]onsiderably more fibrosis and tumors from long fibers" and claims that "[t]he authors concluded that their results were due to some contamination of short-fiber preparations with long fibers." While Jones and Davis discussed the quality of the short fiber preparation, they concluded that, "[t]his short-fibre chrysotile sample certainly represented the shortest chrysotile that has been available for long-term inhalation studies and has permitted a meaningful comparison to be made with long-fibre dust using both inhalation and injection techniques," and "[t]hese figures could indicate that whereas only the relatively long fibres stimulate fibrosis, the shorter fibres (< 5 µm in length) which were present in much larger numbers in the *short-fibre preparation*, *do have some carcinogenic potential*" [emphasis added].⁸

Additionally, Roggli cites one of Pott's papers to support the argument that long fibers cause tumors, but he failed to discuss a second study by Pott *et al.* which concluded that "[m]illed chrysotile with 99.8% fibers [less] than 5 µm in length are carcinogenic in our experience" [emphasis added].^{18–20}

Roggli emphasizes that there are some longer fibers of amphiboles in the black spots analyzed by Boutin *et al.*, but omits that the authors noted that their preparations had debris that may have caused them to fail to find chrysotile which is typically shorter and/or thinner than amphiboles.²¹ Roggli omits the fact that Boutin *et al.* noted that 77.7% of the amphiboles were less than 5 µm in length.

Roggli states that McDonald *et al.* found that short fibers did not contribute to mesothelioma risk; however, he failed to note that they only considered lung concentrations, and that in another paper, they found that the overwhelming majority of fibers found in the pleura were short-fiber chrysotile.^{22,23}

Roggli argues that fiber size > 8 µm (otherwise known as the "Stanton fiber") with no minimum width is the critical size for asbestos carcinogenicity. However, Stanton's actual conclusion was: "[t]he probability of pleural sarcoma correlated best with the numbers of fibers that measured 0.25 µm or less in diameter and more than 8 µm in length, but *relatively high correlations were also noted with fibers in other size categories having diameters up to 1.5 µm and lengths greater than 4 µm*" [emphasis added].²⁴ It should be noted that Stanton only evaluated amphibole asbestos

and had no minimum width requirement. Stanton made only one reference to chrysotile: "[c]hrysotile, although as carcinogenic as the amphiboles at comparable dimensions, could not be included since it has proved difficult to be measured with any degree of precision."²⁴

Finally, Roggli posthumously criticizes Suzuki's research, claiming that his samples were contaminated: "[t]he vast majority of chrysotile fibers identified were less than 1 µm in length, which raises the issue of contamination or fragmentation during processing." Roggli also testified concerning contamination in Suzuki's laboratory:¹⁹

Q: Okay. The – the tests that he did would have indicated, No. 1, whether the actual materials holding the specimens, both the controls and the ones that were of interest, it would have indicated whether or not there was asbestos contamination in the equipment itself; correct?

A: Sure.

Q: The slides?

A: Sure.

Q: Okay. Would it have also showed contamination if there was any in the water that was being used; correct?

A: I think he tested for that, yes.

Q: Right. And there was also bleach used in the process?

A: Yes.

Q: Okay. His blanks on that would have also indicated that – whether or not the bleach was contaminated; correct?

A: Yes.

Q: And his blanks would have also indicated whether the paraffin that was used was contaminated; right?

A: Yes.

Roggli thought that Suzuki had not tested blank paraffin blocks. He was "pretty sure":

[...] I don't think he tested for paraffin. That was one of the – that was one of the criticisms that I had because I maintained that even after his 2005 article.

Q: Okay. So other than it could have happened to this deceased doctor's lab results, what proof do you have that it did?

A: I don't think there is any definite proof. What there is a lot of circumstantial evidence that occurs that it – that it could have occurred and that would be one of the reasons to explain his findings.

However, Suzuki ran controls of both his environment as well as paraffin when he used blocks of embedded tissue.^{25,26} Suzuki describes his effort to control for contamination in his published paper:²⁶

Negative control sampling of the digested bulk tissue sample. To exclude the possibility of environmental contamination of asbestos fibers from air and water, negative control samples are prepared. To obtain negative control samples, the procedure described above is performed without the digested tissue sample. [...] Negative control sampling of the tissue section sample. To obtain the negative control, the same procedure described

Table 1 Animal studies cited by Boulanger *et al.* and omitted by Roggli

Study	Study design	Comment by Boulanger <i>et al.</i>
Schinwald <i>et al.</i> ³⁰	Injection of several types of fibers including amosite with different lengths in the pleural cavity of mice.	"They showed a threshold effect demonstrating that fibers beyond 4 μm in length are pathogenic to the pleura."
Davis <i>et al.</i> ¹⁵	Compared the effects of six samples of tremolite injected in the peritoneal cavity of rats.	"They noted that size alone did not explain the different carcinogenic potencies between the different samples."
Wylie <i>et al.</i> ³¹	A size analysis of crocidolite samples used by Stanton <i>et al.</i>	"... for samples containing a low number of fibers > 8 μm in length and $\leq$ 0.25 μm in diameter, the correlation coefficient was low enough to suggest that other parameters (other categories of size, shape, or other factors) may play a role in carcinogenicity."
Brown <i>et al.</i> 1986 ³⁹	Examined 15 samples of different particles: V79/4 (Chinese hamster lung fibroblasts) and A549 cells (human lung adenocarcinoma)	"The results showed a correlation for a fiber length greater than 3 μm (V79/4) or 4 μm (A549 cells). The correlation improved as the length increased. For diameter, the only significant association was related to the fiber diameter <0.2 μm (A549)."

Table 2 Role of fiber type and dimensions in human studies cited by Boulanger *et al.* and omitted by Roggli

Study	Study design	Findings per Boulanger <i>et al.</i>
Adib <i>et al.</i> ³²	Review of 135 cases with an asbestos-related disease for which a mineralogical analysis had been requested.	Workers with asbestosis, mesothelioma, or lung cancer had mostly short fibers in their lung.
Loomis <i>et al.</i> ³³	TEM data from North and South Carolina asbestos textile workers exposed to chrysotile.	Cumulative exposure to all fibers counted by TEM was significantly associated with lung cancer risk, and to fibers of every length.
Stayner <i>et al.</i> ³⁴	TEM data from North and South Carolina asbestos textile workers exposed to chrysotile.	Cumulative exposures to all fiber size classes, including fibers $\leq$ 5 μm in length, were statistically significant predictors of lung cancer mortality.
Loomis <i>et al.</i> ³⁵	A matrix of fiber size-specific exposure estimates was constructed using determinations of fiber numbers and dimensions through analysis of 160 historical dust samples.	"Loomis <i>et al.</i> ³⁶ conclude that exposure to fibers throughout the range of lengths and diameters was significantly associated with increased risk of lung cancer."

above is performed without the section sample. To rule out the possibility of contamination from paraffin, whenever possible, plain paraffin from the same block is examined using the same methods described above.

Ironically, in 2015, Roggli testified that he himself only began running reagent and blank controls regularly in 2010 "... when you [the plaintiff lawyer asking the questions] started asking questions about it."²⁷ Roggli uses scanning electron microscopy (SEM) to count asbestos fibers and this method cannot visualize or count fibers less than 0.15 μm in width.²⁸ Therefore, his method that cannot visualize many, if not most, chrysotile fibers that served as his "control."

The inadequacy and/or biased nature of Roggli's "so-called" review is also revealed by his omission of much of the relevant literature which Boulanger *et al.* included in a 2014 review of the same topic (Tables 1 and 2).²⁹ Boulanger *et al.*'s entire conclusion from a more compressive review of the topic contrasts with Roggli's:

In view of the experimental and epidemiological studies, the toxicity of SAF [short asbestos fiber] cannot be dismissed. The potential toxicity of SAF remains widely debated in the scientific community. The lower effect of SAF in comparison with LAF [long asbestos fiber] is mostly founded on experimental studies as few epidemiological studies took short fibers into consideration. Additional data are needed as recent epidemiological studies suggest a risk for short fibers.

Based on literature data determining the role of fiber size in biological effects of asbestos fibers and on our present knowledge on their mechanism of action, it appears that the measurement of airborne asbestos concentrations limited to fibers with a length > 5 μm leaves out other types of fibers that may also have health adverse effects. MET analyses of air samples reveals that SAF are systematically present, and in significant proportions. Consequently, regarding the size dependent biological effects of asbestos, it seems that the content of both LAF and SAF present in air samples should be taken into consideration. Measuring SAF would make it possible to identify pollution sources and the need for action to anticipate a possible health risk.

Roggli also asserts that coal miners in the Ruhr valley were not exposed to asbestos, which is incorrect. Asbestos disease in hard coal miners is rare; however, "among a workforce of 300,000 employed between 1950 and the 1970s, there were 140 reported mesothelioma cases. Exposures were mainly due to wearing asbestos filter masks, contact with asbestos insulation material, and employment as metalworkers."³⁶ See Fig. 1 for an image of an asbestos respirator filter.

Roggli is not the first defense expert to use the term "So-Called" in the title of a paper which helps companies defend against dust disease lawsuits. In response to the silicosis lawsuits brought by miners who dug the Union Carbide tunnel at Gauley Bridge, West Virginia, Gardner published a paper intended to discredit the idea that the workers could have developed silicosis within a few years of exposure entitled

Figure 1 Picture of German asbestos respirator filter (courtesy of Hans-Joachim Weitowitz and Xaver Baur. Reprinted for general interest).

"The Pathology of So-Called Acute Silicosis."³⁷ In 1938, L.G. Cole and his son, W.G. Cole, were hired by the John B. Pierce Foundation to prepare a text on silicosis. The Coles reviewed many X-rays and pathologic findings from Gauley Bridge workers and the Coles reported on their meetings and discussions with Drs. Pancoast and Gardner concerning their denial of the existence of acute silicosis. They did not review the Gauley Bridge X-rays seen by Sampson and Gardner as most of these roentgenograms were supposedly accidentally destroyed. Referring to one of the accidentally destroyed X-rays, they wrote, "At least they are not available for study, a situation while fortunate for some is most unfortunate for the advancement of medical knowledge."³⁸ Of their meeting with Gardner, the Coles reported that "the source material for [Gardner's] report and ours was the same, and the moment Gardner saw microscopic sections, he said, 'Oh, I know where that case came from.' It is therefore evident that his description and ours deal with the same morbid changes."³⁸ The Coles were not aware of the fact that, contrary to their published paper, Gardner's secret pathology reports to the companies indicated that these tunnel drillers had suffered from uncomplicated silicosis, and not tuberculosis.⁵

The Coles concluded that many of the Gauley Bridge workers had been denied compensation due to the views put forth by Gardner and Pancoast in their testimonies, which were erroneously accepted by the jury. Moreover, the Coles argued that the workers did indeed suffer from acute silicosis. Because the workers had so rapidly developed the disease, the Coles argued, they did not display the conventional symptoms of the disease, yet were entitled to compensation just the same.³⁸ According to their report:

Gauley Bridge workers died within a few years, but because they were unfortunate enough to have inhaled

dust which did not cause typical roentgenological nodulation, they received no proper compensation. One boy who has since died of silicosis in tuberculosis received for his pains \$18.50. Fate fed them dust which killed but which did not cause morbid changes conforming with a neat definition of the industrial hygiene committee of America.³⁸

The Coles suggested that the reoccurrence of "such a fiasco as the Gauley Bridge incident, or the inspired insertion of the words 'so-called' before the title of an article on acute silicosis, may be avoided by an understanding of the pathology and roentgenology of this vicious form of pneumoconiosis."³⁸

Gardner and the companies responsible for the Gauley Bridge silicosis disaster understood both the pathology and the roentgenology of acute silicosis. Unfortunately, they believed that it was acceptable to falsifying facts to protect the financial interests of their research sponsors at the expense of workers' health.

This is still not understood by some.

Notes

1 The primary author was a member of DRI. The document is available from the DRI website: http://www.dri.org/DRI/course-materials/2014-Asbestos/pdfs/23_Roggli.pdf.

2 Gardner kept his animals in a contained space with asbestos dust. King *et al.* administered "a known weight of asbestos (and aluminum) directly into the trachea."

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Disclosure statement

Dr. Egilman testifies in asbestos toxic tort litigation at the request of injured workers and family members of exposed workers and at the request of asbestos product

manufacturing companies. Triet Tran works for Never Again Consulting.

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