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LETTER TO THE EDITOR

Report of a recent "brake" through in the fiber burden-mesothelioma dialogue

Marsh et al. concluded that "elevated lung levels of tremolite in the lungs of brake workers with elevated levels of amphiboles arose from concurrent exposures to commercial amphibole and chrysotile asbestos in occupational settings other than brake repair work." Then, based on this "finding", they conclude that "the weight of the scientific evidence does not support a role for occupational exposure to brake dust and other friction products in the development of mesothelioma" (Marsh, 2011).

These statements are incorrect. The authors assume that occupational settings, where brake work is conducted, is free of amphibole exposure. This is not true. Commercial amphiboles were used to manufacture many friction-related components. Victor/Dana used crocidolite in gaskets until at least 1963, Borg Warner used crocidolite in transmission friction plates, Ford hygienists were aware of the use of amosite in brakes, Maremont used crocidolite during the Canadian asbestos strike, and Borg Warner admitted that it used crocidolite in brakes in one set of sworn interrogatories, and denied this in another set of interrogatory answers (Hickish, 1968; Borg-Warner, 1992 and 1996; Borg-Warner, 2006; Sherr, 2011; Egilman, 2011). Automobile mechanics who worked on brakes would typically work with these products. Thus, it is incorrect to assume that brake workers acquired elevated lung amphibole levels from anywhere but automobiles absent evidence that a patient had exposure to amphiboles at some other occupation. Working with automobiles can be the source of amphibole lung levels, and explains the results in this paper.

The authors argue that chrysotile brakes can not be linked to mesothelioma because brake work never results in chrysotile lung burdens sufficient to cause mesothelioma. However, this is not true.

Dr. Roggli found elevated levels of chrysotile in the lungs of two workers with pulmonary fibrosis whose only asbestos exposure was as a consequence of the work with asbestos brakes (Case 1, 1983 and Case 2, 1994) (Roggli, 2011) Dr. Roggli explained that he has failed to publish these cases because the patients did not suffer from mesothelioma (Roggli, 2011). However, this is irrelevant as the authors' theory is based on the general proposition that working with brakes never results in elevated lung chrysotile levels. Any brake worker with elevated levels is a Black Swan case that disproves the authors' theory. For example, even absent lung fiber data, Dr. Roggli has opined that a particular brake worker's mesothelioma was idiopathic because he has not seen any brake workers who had above-background levels of asbestos in their lungs without a corresponding elevated level of commercial amphibole fibers (Roggli, 2011). Dr Roggli's methodology has been thoroughly critiqued elsewhere (Egilman, 2009).

In recent testimony, Dr. Roggli agreed with our interpretation of the importance of these results. He confirmed that mechanics' exposures can lead to levels of chrysotile and non-commercial amphiboles that cause increased fiber burden in their lung tissue, and he would have attributed causation to their exposures had they had mesothelioma (Roggli, 2011).

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Patient	Asbestos bodies	Total fibers	Commercial amphiboles	Chrysotile	Tremolite	Silicate	Antho-phylite	Actino-lite	Total uncoated fibers	Non-commercial asbestos fibers
Case 1	0	10	0	6	1	3	0	0	42,800	8500
Case 2	0	7	0	0	4	0	1	2	21,800	7000

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Declaration of interest

Dr. Egilman serves as a consultant in asbestos litigation at the request of asbestos manufacturing companies and injured people.

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