FILE NAME: Brakes (BRK)

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DOC#: BRKмрм

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AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 47:276–277 (2005)

Letter to the Editor

RE: Asbestos in Brakes: Exposure and Risk of Disease

To the Editor:

We read with interest the recent article by Richard A. Lemen entitled "Asbestos in brakes: Exposure and risk of disease" [Lemen, 2004]. However, we were disappointed that Dr. Lemen omitted a number of key references that bear on this important issue.

With respect to the decomposition of asbestos in brakes, Dr. Lemen refers to the studies by Lynch [1968], Williams and Muhlbaier [1982], and Rohl et al. [1976], but fails to reference the more recent study by Langer regarding the reduction of the biological potential of chrysotile asbestos arising from conditions of service on brake pads [Langer, 2003]. Instrumentation required to identify and measure degree of alteration of free fiber was not used by Rohl and colleagues [1976], or by the other investigators in their respective studies. Although Rohl et al. [1976] detected some forsterite in brake drum dust, no further analysis was attempted to determine if the individual fibers released from brake pads had suffered physical or thermal degradation. The X-ray diffraction analyses given characterized the fiber in binder particulates (unaltered) as well as free fiber (in a range of physical states).

Regarding the putative toxicity of short fibers, Dr. Lemen quotes JMG Davis' study on brake dust [Davis and Coniam, 1973], but fails to quote Dr. Davis's extensive work that failed to demonstrate the pathogenicity of fibers less than 5 μm in length [Davis et al., 1986; Davis and Jones, 1988]. Furthermore, Dr. Lemen omits the study by Berman et al. [1995], which reanalyzed Davis' data and concluded that fibers less than 10 μm in length are not pathogenic. In addition, he references three studies by McDonald et al. [1989] but omits the study by these authors which analyzed human lung tissue samples and concluded that fibers less than 8 μm in length did not contribute to the development of mesothelioma.

With respect to exposure assessment of asbestos in brakes, Dr. Lemen quotes a variety of studies, but fails to quote the extensive review of this subject by Paustenbach et al. [2003].

The evidence regarding excess mesothelioma among brake workers deserves special attention. Dr. Lemen quotes a letter by Otto Wong, but fails to quote the meta-analysis by Dr. Wong that found an overall relative risk of mesothelioma among auto mechanics of 0.90 with a 95% confidence interval of 0.66–1.23 [Wong, 2001]. Dr. Lemen also quotes the study by Roggli et al. [2002] but omits the authors' conclusions regarding mesothelioma and brake mechanics or their separate study of lung fiber burden analyses in brake mechanics [Butnor et al., 2003]. Dr. Lemen also refers to the Australia Mesothelioma Surveillance Program, which is not a formal epidemiologic study.

Unfortunately, Dr. Lemen's article does not provide the reader with a balanced view of the available evidence. We would urge those interested in this area not to draw conclusions regarding exposure to brake dust and mesothelioma risk before reading these additional sources.

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Accepted 1 June 2004
DOI 10.1002/ajim.20129 Published online in Wiley InterScience (www.interscience.wiley.com)

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AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 47:278–280 (2005)

Letter to the Editor

Reply to Victor L. Roggli and Arthur M. Langer

To the Editor:

I am happy to see that my article on Asbestos and Brakes has generated interest to this often under-estimated risk. Dr. Roggli and Dr. Langer suggest several articles were not recognized in my paper which, if they had been included, would allow a more complete analysis of the risks or lack thereof to workers exposed to asbestos-containing brake dust.

Dr. Langer's paper [Langer, 2003] dealing with the reduction of the biological potential of chrysotile asbestos offers a position in contrast to that discussed in my paper; however, his analysis does support the necessity for continued vigilance to protect brake repair workers, especially in new brake applications. Dr. Langer's analysis purports the elimination of the biological potential of chrysotile because of its transformation into the supposed innocuous degradation product of chrysotile, fosterite, a substance my paper points out lacks suitable analysis to determine its long-term effects and while some chrysotile does convert to fosterite, the evidence, other than Dr. Langer's paper, has not been consistent. Dr. Langer contends that "[C]hrysotile biological activity is thereby greatly reduced, and can become virtually nil hundreds of degrees below the olivine transformation temperature." This is not supported by the data presented in my paper nor by a follow-up paper by Rohl et al. [1977] which is in fact a follow-up of the Rohl et al. [1976] analysis which Dr. Langer contends was never done, that concludes, by using the transmission electron microscopy (TEM), that "[M]ost of the chrysotile retained is characteristic morphology without significant alteration, and selected area electron diffraction patterns obtained on representative fibers demonstrated the preservation of crystal structure as well."

Dr. Roggli and Dr. Langer suggests my paper lacks objectivity in that it does not review the Davis et al. [1986], Davis and Jones [1988], or Berman et al. [1986] reanalysis of the Davis data nor the McDonald et al. [1989] papers concerning the lack of toxicity for short fibers. First, the Davis papers do not conclude a lack of toxicity for fibers shorter than 5 μm in length. What the Davis papers do show is that: (1) long fibers produced six times more fibrosis and three times more tumors than the short-fiber preparations after inhalation; (2) injection studies, at the highest dose levels 25 mg, found little difference in the number of tumors produced by both long- and short-fiber chrysotile, while at lower levels there was a significant difference between the long- and short-fiber preparations with the longer fibers being more carcinogenic; (3) the mean tumor induction period was longer for the short-fiber preparation in producing mesotheliomas at both the 25 mg and 2.5 mg dose level and the authors conclude "...would probably have been seen with the 0.25 mg dose if the short-fiber chrysotile had produced any mesotheliomas at this level;" and (4) the authors state that the alteration of the short-fiber chrysotile produced by ball-milling is subject to a level of crystal damage, which is sufficient to make results difficult to interpret in relation to hazards resulting from short fibers produced during the manufacture of asbestos products or during the subsequent usage of these materials. Berman et al. [1986] using a risk analysis model of their choice simply choose to eliminate all fibers less than 5 μm in length as "Structures <5 μm in length do not appear to make any contribution to lung tumor risk." Their support for this assumption is unwarranted given the conclusions of the Davis et al. papers and the scientific evidence to date.

McDonald et al. [1989] examined 78 cases of mesothelioma from autopsy between 1980 through 1984 with matched referents to evaluate the lung burden of long versus short fibers, concluding that the role of short-fibers was nil. As I stated in my paper, looking only at lung burden analysis for chrysotile short-fibers may not be the most appropriate analysis to determine the role of either chrysotile or short-fibers as they are cleared from the lung rapidly

Dr Lemen is retired Surgeon General, USPHS and retired Deputy Director and Acting Director, NIOSH and has testified as a plaintiff's expert in brake exposure cases.
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Accepted 1 June 2004
DOI 10.1002/ajim.20130. Published online in Wiley InterScience (www.interscience.wiley.com)

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compared to longer non-chrysotile fibers. This same criticism is applicable to the Butnor et al. [2003] analysis of ten cases of mesothelioma among brake exposed workers where analysis was only of lung tissue. Butnor et al. [2003] dismiss the 'hit-and-run' hypothesis for chrysotile as 'flimsy' and having no solid scientific support and cite Hesterberg et al. [1994, 1995, 1996] studies, of man-made vitreous fibers, as their proof for this contention. While there is clear proof of the biopersistence for amphibole asbestos, the lack of such biopersistence of other fibers, as shown in the Hesterberg et al. papers, provide support to the contrary, that pathogenicity of a fiber is dependent upon more than simply the dose, dimension, and the durability of the fibers found in the lung [Hesterberg et al., 1995]. It is also of interest that chrysotile asbestos produced fibrosis, lung tumors, and mesothelioma in rats after inhalation studies as shown in the Research and Consulting Company (RCC) studies cited in the Hesterberg et al. [1995] paper.

Finally, to rely upon the Wong [2001] meta-analysis or the recompilation by Paustenbach et al. [2003] for support of the proposition that brake dust exposures pose no mesothelioma risk is weak at the very best. Wong [2001] uses a technique, meta-analysis, to pool data from available epidemiology studies and concludes a lack of causal association between exposure to brakes and mesothelioma. First, meta-analyses of observational studies present inherent biases such as selection bias and confounding biases as discussed in my paper. Secondly, meta-analysis is a technique, which was first envisioned for evaluating clinical studies where combining results, based on individual participation data, would be less likely to suffer from such biases and its further application to the analysis of observational studies becomes problematic. If the data relied upon for meta-analysis have flaws then the outcome of the meta-analysis will also suffer from the impact of such flaws as will their conclusions. As Dr. Wong states in his paper, which he attributes to IARC: "In addition, all studies that are judged to be methodologically sound should be consistent with a relative risk of unity for any observed level of exposure to the agent and, when considered together, should provide a pooled estimate of relative risk which is at or near unity and has a narrow confidence interval, due to population size." The observational studies selected for analysis by Wong [2001] do not meet this standard and thus are inappropriate for use in pooling their data because of their heterogeneity in several areas including their cohort selection criteria; their exposure categories; and their varying risks of exposure to asbestos. Until such time as methods of controlling heterogencity are developed, like those employed in clinical trials, meta-analysis of observational epidemiology studies will remain problematic and their conclusions remain unclear as are those purported by Dr. Wong. Therefore, systematic reviews, as done in my paper, remain a far superior method to the meaning of observational epidemiology studies where heterogencity issues remain.

Paustenbach et al. [2003] report exposures to asbestos from some 200 historical samples taken of brake dusts, which generally show low exposures but do report the occurrence of findings above the current PEL. Their findings are of very limited use as they represent only a small fraction of the brake dust exposures encountered. That is to say 200 samples among the estimated 5 million brake-repair workers exposed to asbestos in the United States is far less than a representative sample of the spectrum of potential exposures encountered by American workers to asbestos-containing brake dust. What the Paustenbach et al. [2003] data show is that asbestos can and is released from brakes and as a result brake-repair workers can be exposed to asbestos. Any comparison of this data set to the total universe of exposures to brake-repair workers is a phantom stretch of the imagination.

In conclusion, Dr. Roggli and Dr. Langer have presented no evidence that negated the findings or conclusions of my paper and I thank them for their comments, which have helped to strengthen and further affirm my conclusions. Finally, my paper draws the conclusion that as long as brakes contain asbestos there remains the real threat that workers will be exposed to releases of asbestos, which have the potential to cause worker diseases related to exposure from asbestos-containing brake dusts.

Richard A. Lemen, PhD, MSPH*

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