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Letter to the Editor

Response to Letter from Paustenbach et al.

To the Editor:

We appreciate the interest expressed by Paustebach and colleagues in our study entitled, "Evaluation of the size and type of free particulates collected from unused asbestos-containing brake components as related to potential for respirability" which was published last year in this Journal (*AJIM* 46:545–533). We were somewhat surprised to note that Paustebach and colleagues "expected to find measurable TEM asbestos fibers on an unused brake component." We assume that they mean asbestos fibers that can be detected by analytical transmission electron microscopy (TEM). However, we do disagree with a number of their comments and conclusions.

They reference their study [Paustenbach et al., 2003] in which a "survey of the world's literature" on the subject of exposure to asbestos in automotive service facilities was carried out and comment that the airborne levels of asbestos are very low and in some cases, non-detectable. We conducted our study because it was unclear to us from the published literature, whether respirable asbestos fibers that were smaller than those which would be detected and included in a standard NIOSH count were readily released from new brake components or if they were tightly bound in "inert" matrix material and consequently not readily released.

Their comment goes to the salient point of our study, that is, that the asbestos fibers that are not included in such a fiber count or which are excluded by the count scheme still have the potential to cause disease. The use of phase contrast microscopy (PCM), which is used to establish a permissible exposure limit (PEL), is incapable of detecting small asbestos fibers that are nevertheless potentially capable of causing disease. Our "theoretical" classification [(Table I), Atkinson et al., 2004] of which asbestos structures in each group indicate which would be included in a PCM count as well as what would be 'missing' fibers. From the beginning what we did realize was that the literature contains abundant data, including data from our own studies, which indicate that the detection of chrysotile fibrils, most thinner bundles, and individual fibers of many amphiboles require the use of an analytical transmission electron microscope (ATEM) applied at a sufficiently high magnification to permit their recognition. The inclusion of fibers shorter than 5 μm in a count scheme is also important if one is to obtain an accurate representation of the populations of fibers in environmental Asbestos Hazard Emergency Response Act (AHERA) [Rohl et al., 1976; Rood and Streeter, 1984; Upton et al., 1991] as well as tissue samples [Langer et al., 1971; Churg and Warnock, 1980; Pooley and Ranson, 1986; Dodson et al., 2003].

It was not our original intent to discuss at great length phase contrast microscopy and NIOSH/OSHA counting schemes. However, since Paustenbach and colleagues have raised the issue of why we did not employ the PCM in counting fibers and have placed an appreciable emphasis on PCM-based data, it seems reasonable for us to elaborate further on what one can and cannot "see" with the PCM as well as the reasons that dictated the choice of instrument (ATEM) used in our study.

As Paustenbach and colleagues point out historical data on air quality obtained by PCM analysis (NIOSH/OSHA) in the work place of mechanics show that the PEL was often not exceeded. The use of the PCM and determination of PEL obviously tie together. The reader is recommended to review the article by Langer et al. [1991] that included the statement "the phase contrast microscope was both resolution limited and incapable of acquiring data useful in fiber identification" a conclusion with which we are in complete agreement. The authors furthermore discuss the basis for establishment of a definition of a fiber as per the OSHA-NIOSH count scheme

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by PCM [Langer et al., 1991]. Langer et al. [1971] in an earlier study pointed out "the optical microscope delivers a select, biased population. First, we can only study what the microscope sees and it only sees large fibers, those thicker than 0.5 μm in diameter." Rood and Streeter [1984] pointed out that "the detection limit of a good, correctly adjusted PCM may be 0.15 μm [Rooker et al., 1982]; however, under normal operating conditions, the detection limit is not so low [Ashcroft and Heppleston, 1973; Hwang and Graham, 1981; Hwang and Wang, 1983] and we shall assume a limit of 0.3 μm." Thus the resolution limit used in our study for the PCM seems rather liberal and indeed lower than published detection limits.

If one reviews the diameters of fibrils and most bundles found in our study, it is evident that they would be invisible by PCM and consequently not be included in any count carried out by PCM. The exclusion of asbestos structures derived from brake dust would be even further reduced if one used a count scheme excluding fibers below 5 μm be it done by PCM, scanning electron microscopy, or TEM. The point of our study and the reason for conducting this study was to determine, if respirable asbestos structures were released from new brake components in numbers that posed a potential health hazard irrespective of regulatory counting schemes, which in turn are not linked to the potential of a given fiber to cause disease. If an asbestos fiber is respirable it has the potential to contribute to the development of disease irrespective of whether limitations of the instrument used permit "seeing it or not" in a given air sample.

The logic of using ATEM to obtain a real representation of asbestos in brake dust has been cited in the literature. The Health Effects Institute-Asbestos Research document concluded "the TEM provides the most complete analysis currently available for airborne asbestos. The very high resolving power of the TEM, and its ability to provide the mineralogical identity of asbestos (when combined with selected area diffraction [SAED] and EDXA, make the TEM the method of choice for analysis of airborne asbestos samples." During evaluation of asbestos exposure during brake lining maintenance and repair, Rohl et al. [1976] found that "most fibers are too small to be seen by optical microscopy; almost all of them shorter than 0.4 μm in length; virtually all of respirable size (−5 μm)." Jacko et al. [1973] made size distribution measurements of asbestos fibers in brake dusts generated during dynamometer tests, using both optical and electron microscopy. They found, at magnifications of 22,000× that 30% of the fibers were from 0.25–0.5 μm in length and that 60% were longer than 0.5 μm [Rohl et al., 1976]. The data found in our study confirms that using an instrument not capable of resolving most asbestos structures based on diameter or excluding structures based on a count scheme that included only those greater than 5 μm in length would provide data on only a limited population of the dust and ignore a large amount of potentially injurious dust.

Of course, we could have analyzed our samples with PCM and then compared the results with ATEM data, as suggested although this would seem pointless for the reason stated by Pooley and Ranson [1986] that "it is possible using the electron microscope, to predict the asbestos fiber count that would be obtained by light microscopy, the reverse prediction cannot be made: it is impossible to determine the proportion of the various asbestos mineral types using the light microscope." Paustenbach et al. also take issue with our methodology, specifically citing the lack of control "rinsates." In the text we state that the water used was filtered through a 0.2 μm Nucleopore filter to remove any particulates as is standard in our lab. We periodically do run quality control on our reagents (including prefiltered water) and these are uniformly negative.

The authors also criticize the lack of a review of the world's literature on exposure to brake dust as it relates to industrial hygiene and epidemiology, and that our citations are biased. We maintain that this is unfair. Our study was a research report not a review article and, perhaps more to the point, was a study designed to answer the question—"What can be rinsed from the face of new brake components?" not an industrial hygiene or epidemiology-based study. An extensive literature review of published industrial hygiene or epidemiology-based studies would, in all probability, have been viewed as irrelevant by the referees of our manuscript. We did include the article by Paustenbach et al. [2003], which we thought to be an extensive review of these subjects. It is our position that we did quote the section from the Butnor study exactly as written [Butnor et al., 2003] as do Paustenbach and colleagues of the statement they have used. Since that study has been referenced it may be of interest to note the technique used for analyzing the tissue burden was scanning electron microscopy with a defined fiber being 5 μm or longer to be included into the count scheme. For reference the reader may choose to read our tables and determine how many of the total chrysotile asbestos structures (including fibrils and bundles) would have even been included in such a count. In essence a count of fibers (or structures) of 5 μm and longer will exclude most asbestos dust from brakes (and in tissue samples preferably favor the thicker and longer amphibole population) no matter what the instrument of choice. We believe from a health standpoint that this exclusion is arbitrary.

Finally, and perhaps of greatest importance, while we respect the position of Paustenbach et al. concerning the issue of exposures to brake dust and the potential for induction of diseases in man, we respectfully believe that the potential for cumulative exposure to asbestos, even short chrysotile, "raises further concerns," as to the potential for inducing disease. We are confused by their comment that on one hand one should expect to find free fibers on the surface of new brakes but that our findings of easily removed asbestos structures from the surface of new brakes carried no

implication as to the potential for asbestos release into the air. We would suggest that the reader considers the numbers of asbestos structures constituting the respirable components of each group of structures that were rinsed free from the binding material of the brake components and then logically consider if additional ones could be produced by sanding, grinding or other traumatic events during replacement (much less asbestos dust introduced into the air following physical wiping of brake drums or blowing the area with compressed air). It seems to us that we have actually identified only a small portion of the potential asbestos that could be made friable and thus become aerosolized during brake replacement or repair. If you find free asbestos structures of respirable size the logic eludes us as to how they may not, at least potentially, be inhaled if aerosolized and once deposited in the terminal airways have the potential to induce pathological responses at those sites or to be relocated to extrapulmonary sites. It should not be lost to our readers that data concerning tissue burden in extrapulmonary sites where asbestos induced diseases occur indicate that the populations of asbestos are often over whelmingly represented by short fibers (<5 μm). [Sebastien et al., 1980; Dodson et al., 1990; Suzuki and Yuen, 2001, 2002].

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