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AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 45:229–237 (2004)

Asbestos in Brakes: Exposure and Risk of Disease

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Asbestos has been incorporated into friction products since the early 1900s. Epidemiological studies have been equivocal in their analysis of the incidence of disease among mechanics servicing brakes. Decomposition of asbestos occurs during the normal usage of the brake due to thermal decomposition into forsterite, although not all asbestos is so converted. Short fibers, below 5 μm in length, are also found in brake products. Several facts are discussed including the toxicity of the remaining asbestos fibers, short asbestos fibers, and the health implications of exposure to forsterite. Control methodologies, when used appropriately, have reduced exposure to asbestos during brake servicing, but have not been able to entirely eliminate exposure to asbestos, thus bring into question the controlled use of asbestos for friction product such as brakes. Even the so called "controlled" use of asbestos containing brakes poses a health risk to workers, users, and their families. Am. J. Ind. Med. 45:229–237, 2004. © *2004 Wiley-Liss, Inc.*

KEY WORDS: friction products; brakes; chrysotile; forsterite; epidemiology; short asbestos fibers; chrysotile

Historical Aspects of Asbestos Use in Brakes

Woven asbestos friction materials were first used in 1903 in the United States. Molded brake linings were developed in the early 1920s with increasing use until the introduction of the internal brake shoe around 1927. By 1940 virtually every automobile was equipped with molded brake linings [Sheehy et al., 1989]. Disc brakes came into being in 1965 and by 1975 virtually all US cars had such front brakes, however, rear wheel brakes remained mainly of the drum variety. Chrysotile asbestos was used almost exclusively, as the amphibole asbestos type tended to be too harsh and tended to score the brake drums, making them wear much faster. The chrysotile made up from 40 to 50% of the brake lining [Sheehy et al., 1989].

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Accepted 31 October 2003
DOI 10.1002/ajim.10334. Published online in Wiley InterScience (www.interscience.wiley.com)

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Decomposition of Asbestos Fibers in Brakes

The US Public Health Service reported a study by Lynch [1968] of decomposition products from brake linings. It found that a small fraction of asbestos fibers survived intact after normal brake use. While this study indicated a small fraction of actual asbestos fiber is released during normal wear into the urban atmosphere, it did not address the actual release of asbestos fiber during servicing and repair of the actual brakes [Lynch, 1968]. One of the first evaluations of worker exposure to asbestos, from brake servicing, found that the British Standard for asbestos of 2 fibers per cc was not exceeded in the general atmosphere but that personal exposures in the vicinity of the operation did exceed the standard on occasion [Hickish and Knight, 1970]. The chemical composition of asbestos materials is changed by the high heat generated during the braking process. Asbestos can begin to change at temperatures around 600°C into its decomposition product, due to a loss of water, into a form of iron magnesium silicate material, the end form which is known as forsterite, a non-fibrous material. During this decomposition of asbestos into forsterite not all fibers are changed and some remain as true asbestos fibers capable of causing disease. Studies conducted by General Motors'

researchers of brake wear debris demonstrate that 90,000 asbestos fibers per ng remain in that dust [Williams and Muhlbaier, 1980]. Fibers less than 5 μm in length outnumber fibers greater than 5 μm in length by a ratio of 300:1. This translates to approximately 300 billion asbestos fibers greater than 5 μm per g of wear debris and 90 trillion asbestos fibers less than 5 μm.

The role of forsterite in relation to health effects has not been well documented, but what little is known come from limited animal studies, that show the production of small granulomas with little fibrosis, from injection studies [Davis and Coniam, 1973]. Animal studies were not able to assess the possibility of long latent effects or associations with the production of mesothelioma, as the tests were terminated after 1 year. Although little fibrosis has been shown, the studies tend to support a more cytotoxic role for both heated chrysotile and brake lining dust, which is far more toxic than normal chrysotile. Further study is needed to assess the acute and chronic toxicity of forsterite [Davis and Coniam, 1973]. It has also been reported that during this decomposition process the majority of fibers that remain are of small diameter as well as below 5 μm in length [Rohl et al., 1976; Sheehy et al., 1989; Yeung et al., 1999] and thus are less harmful [Hatch, 1970].

Toxicity of Short Asbestos Fibers

Any assumption that short fibers, less than 5 μm in length, are not hazardous cannot be justified based on the available science. Because the analytical method of choice, for regulatory purposes, has been the phase contrast method (PCM) which counts only fibers greater than 5 μm in length, epidemiology studies that have been forced to compare doses in their cohorts to fibers greater than 5 μm in length. It must be noted that the PCM analytical method was chosen based on its ability to count fibers only and not on a health effect basis. While PCM has been the international regulatory method for analysis it is not able to detect thin diameter fibers (<0.2 μm in diameter), and because of this, it is suggested that transmission electron microscopy (TEM) should be an adjunct to PCM, since the evidence suggests that PCM may underestimate exposures and the health risks as found in the analysis of brake residue [Yeung et al., 1999].

Stanton and Wrench [1972] and Stanton et al. [1981] found that the longer, thinner fibers were more carcinogenic, but could not identify a precise fiber length that did not demonstrate biological activity. It must be kept in mind that Dr. Stanton has never said long fibers are bad and short fibers are good. In fact he appreciated that a large number of short fibers, individually of low tumorogenic probability might be more hazardous than fewer long fibers, individually of high probability [Greenberg, 1984]. It has been shown that it is not just the size and shape of the various asbestos fibers that are important in its ability to produce disease but other factors may play a role in the carcinogenicity of the mineral fiber [Wagner, 1980; Wylie et al., 1987]. Studies have also found that the majority of asbestos fibers in lung and mesothelial tissues were shorter than 5 μm in length, thus indicating the ability of the shorter fibers to reach the tumor site, remain there, and, therefore, their role in the etiology of disease is implicated [Dodson et al., 2001; Suzuki and Yuen, 2002]. National Institute for Occupational Safety and Health (NIOSH) research has found that in typical occupational environments fibers shorter than 5 μm in length outnumber the longer fibers by a factor of 10 or more [Dement and Wallingford, 1990]. Shorter fibers must be studied in more depth and they should not be disregarded especially when clearance is retarded [Oberdorster, 2001]. That chrysotile fibers tend to spit longitudinally as well as partially dissolve, resulting in shorter fibers within the lung, was reported in a review of several articles [Dement and Brown, 1993]. Additionally, Fubini [2001] argues that, because all asbestos appears to be nearly equally potent, length and fiber form does not appear to be the only influential aspect on the outcome of disease. He makes this conclusion based on the work of Boffetta [1998] which concludes that the specific type of asbestos is not correlated with lung cancer risk but that industry specific exposure appears to fit the linear slope best, a finding also supported by Dement and Brown [1993]. For mesothelioma, induction was related to the time since first exposure and potency with both industry type and asbestos type [Boffetta, 1998].

The Agency for Toxic Substances and Disease Registry (ATSDR), in response to concerns of short asbestos fibers resulting from the collapse of the World Trade Towers, asked a contractor to convene a panel of seven experts to evaluate the role of short fibers with human disease potential [ATSDR, 2003]. As to non-carcinogenic lung diseases associated with short asbestos fibers the report concludes that "...short fibers may be pathogenic for pulmonary fibrosis, and further research is needed to clarify this issue." The panel concluded that for carcinogenic effects of short fibers the current weight of the evidence is that short fibers less than 5 μm "...are unlikely to cause cancer in humans." While these conclusions were found in the executive summary of the report, a more in-depth review of the body of the report points to a less conclusive assessment for the role of short fibers in the etiology of cancer. In fact, in panel discussions it was noted that no epidemiologic studies have examined populations exposed only to short asbestos fibers. One epidemiology study that may have the ability to address this issue suffered from short latency to evaluate the development of cancer [Higgins et al., 1983]. Another study of workers having exposure for at least 5 years, in a gold mine with 94% of the asbestos fibers being less than 5 μm in length, found an increased mortality from respiratory cancers [10 obs. vs. 2.7 exp.: SMR 3.7; 95% CI = 1.78–6.81] and non-malignant respiratory diseases [8 obs. vs. 3.2 exp.: SMR 2.5; 95%

CI = 1.08–4.93; Gillam et al., 1976]. A subsequent study [McDonald et al., 1978], looking at the same mine reported above, of miners with 21 or more years underground experience, did not find such an increase for respiratory cancers but did for non-malignant respiratory disease. However, when analyzing the data in the previous study, for only those with 20 years or greater years of employment, both respiratory cancers [7 obs. vs. 2.18 exp.: SMR 3.2; 95% CI = 1.29–6.62] and non-malignant respiratory diseases [8 obs. vs 2.56 exp.: SMR 3.1; 95% CI = 1.35–6.16] were still significantly increased. Two other studies of miners, where 38% of the asbestos fibers were shorter than 5 μm in length also found excess mortality from lung cancer, mesothelioma as well as non-malignant respiratory disease and that the mortality patterns for mesothelioma were significant because they were much greater than that of crocidolite miners in South Africa and Australia [McDonald et al., 1986, 2002].

Animal studies can be misleading when looking at short fibers, especially as rodents clear short fibers from their lungs at a rate approximately 10-times faster than do humans [ATSDR, 2003]. Experimental models are limited also, due to the fact that only fibers of very limited length distributions have been tested [Dodson et al., 2003]. Further, when appropriate analytical techniques have been used the overwhelming majority of the asbestos fibers in the tissues have been found to be less than 5 μm in length [Dodson et al., 2003]. Only two of the seven ATSDR panelists felt there was a reasonable certainty of no harm from short fibers while the other five remained concerned about the ability of short fibers to cause harm [ATSDR, 2003]. In fact, tremolite asbestos fibers were found to produce the highest average fibrosis grades when exposures were to average tremolite fibers less than 5 μm in length [Nayebzadeh et al., 2001].

NIOSH Recommended Worker Protections for Brake Servicing Procedures

In 1975, the NIOSH convened a meeting with other government agencies, university scientists, industry representatives, and labor union officials. It was reported that average peak asbestos air concentrations for specific brake servicing operations, including blow-out, grinding, and beveling of new truck brake linings resulted in average peak asbestos air concentrations, within 10 feet of the operator of 10.5, 3.75, and 37.3 fibers/cc (>5 μm in length). Even when an analysis of the asbestos fibers was done it was found that overall almost all were shorter than 0.4 μm in length. These findings led NIOSH to conclude that enough asbestos is preserved to produce significant exposures during certain brake servicing procedures [Lloyd, 1975]. At that time NIOSH specifically recommended posting warning signs at all areas where brake repair work was to be done.

The sign should read:

Asbestos
Dust Hazard
Avoid Breathing Dust
Wear Assigned Protective Equipment
Do Not Remain in Area Unless Your Work Requires It
Breathing Asbestos Dust May Cause Asbestosis and Cancer

In addition to the warning signs, the Current Intelligence Bulletin (CIB) no. 5 [Lloyd, 1975] also gave very specific recommendations for protecting the worker.

Exposure Assessment of Asbestos in Brakes

A study of fiber release from brake pads of overhead industrial cranes found very small releases of asbestos fibers all well below the current OSHA standard [Spencer et al., 1999]. Such a finding is not surprising, as the normal use of brakes have not generally been shown to produce high fiber release to the general environment [Williams and Muhlbaier, 1982]. The greatest concern, however, lies with exposure to the person removing brakes or installing new brakes or through the manipulation of the pads with compressed air blow-out, wire brushing, or other such methods which can release airborne asbestos. Exposures during brake wear are also higher from vehicles using disc brakes than drum brakes (due to the smaller friction surface area of disc, allowing faster wear) and during high deceleration [Williams and Muhlbaier, 1982]. When evaluating what this means to persons near areas where such decelerations may occur the authors evaluated the amount of asbestos released into the environment in such an area. They chose to look at tollbooths in Connecticut where they concluded that a significant fraction of the ambient asbestos concentrations of 25 ng/m^3 could arise from braking. While this exposure appears low on a one time basis, continued exposures over a working lifetime to tollbooths operators would add to their total body burden of accumulated asbestos fibers and place them at higher risk of developing asbestos-related cancers. Bruckman et al. [1977] did report a case of mesothelioma in a tollbooth operator. These findings could also be important to other persons residing in areas where high decelerations take place, such as along high volume traffic areas, where residential housing is often located.

Rohl et al. [1976] reported that chrysotile asbestos was found in all dust samples taken from car brake drums, with 2–15% in each sample in both fiber and fibril forms, with average concentrations from blowing the dust of 16 fibers/ml of air and that measurable concentrations were also found up to 75 feet from the actual worksite some 15 min after blowing out ceased. Also, Lorimer et al. [1976] found mean fiber concentrations of 3.8 fibers/ml among New York brake repair workers, which is similar to other studies that

show intact chrysotile fibers may be released from brake materials.

Grinding of the brake linings produced the most asbestos fiber release, some as high as 125 fibers/cm^3, during brake maintenance of truck and buses in a study by Kauppinen and Korhonen [1987]. Up to 8.2 f/cm^3 was found during cleaning of the drum brakes of passenger cars using a compressed air jet to remove the brake dust. Overall the average concentrations were between 0.1 and 0.2 f/cm^3 for an 8 hr TWA for truck and bus repair, and under 0.05 f/cm^3 for passenger car repair. This study clearly demonstrated that while average concentrations can average much lower, excursion levels can reach much higher concentrations during certain operations of the brake maintenance and repair process exceeding the OSHA excursion limits and the 8 hr TWA for asbestos. NIOSH researchers found that when looking at the fibers from the brake repair area 30% were chrysotile, 20% forsterite, and 50% were unknown, leading the researchers to conclude that excessive exposure to asbestos fibers occurs during brake servicing [Roberts and Zumwalde, 1982]. EPA reported that millions of asbestos fibers can be released during brake and clutch servicing and that such asbestos can linger around the garage long after brake jobs are done and can be breathed in by everyone inside the garage which can present a hazard for months or years. Grinding of used brake block linings has been shown to release up to 7 f/cm^3 and beveling new linings up to 72 f/cm^3 and even light grinding of the new linings up to 4.8 f/cm^3 [USEPA, 1986].

Control methods have been shown effective in reducing the concentrations of asbestos fibers in the air in areas where brake repair takes place, however, when such reductions have occurred, the risk of asbestosis is probably eliminated but the risks of cancer are not [Sheehy et al., 1987]. Both Kelly and Cheng [1986] and Rodelsperger et al. [1986], found asbestos concentrations during brake repair to have average exposures that reach 0.28 and 0.1 f/cc, respectively, one above and the other at the current OSHA PEL for asbestos. A study of asbestos exposure during brake inspection and replacement of light-duty vehicles found fiber counts, when not using compressed air blow down, that range from 0.05 to 0.2 f/cc and when using compressed air the average when up to 0.9 f/cc which remained elevated for 15 min after blow out. While this is below the OSHA 30 min excursion limit of 1 f/cc (OSHA, 29 CFR Asbestos, 1910–1001) it does indicate the release of asbestos fibers during brake pad manipulation, and in inspection and replacement [Weir and Meraz, 2001]. In a study that looked at both brake and clutch repair workers in Knoxville and Knox County Tennessee it was found, that when using the worst asbestos control techniques, that those working up to 7.25 hr per day would reach and exceed the current OSHA standard of 0.1 f/cc during brake repair. Citing Anderson et al. [1973] concentrations of asbestos reached as high as 29 f/cc at distances of 5 feet from the brake drums and 4.8 f/cc at distances as far away as 20 feet. Peak concentrations of asbestos fibers in the breathing zone of brake mechanics were reported to be as high as 15 f/cc when using dry brushing, wet brushing, or compressed air during brake repair and 0.2 f/cm^3 as an 8 hr TWA.

Laboratory simulated studies while not always showing similar results may not tell the entire story of actual brake work, as in the case of drilling and grinding simulated by Weir and Meraz [2001]. They report "... that the majority of fibers collected on filters from the air stream have resin deposits attached." They state correctly that the aerodynamic behavior of such fibers is different from the "clean" fibers in their inhalation into the respiratory system. They also say that "... to a great extent, (fibers) remain bound to the matrix of the underlying brake material." The real question is what happens to the remainder of those fibers not included in the "great extent, remain bound..." which the authors fail to address. And what happens to the nominally bound fibers once trapped in the lung tissues, where fibers may be released. The EPA evaluated the release of fibers from three commercial products (asbestos cement sheet. millboard, and brakes). They found that grinding brakes produced a release of asbestos fibers and other asbestos containing structure types. Asbestos fibers accounted for 46.0% of the asbestos structure types released and when evaluated by fibers/cc/g of asbestos milled were as high as 486.1. This study shows that fibers are indeed released when asbestos containing brakes are ground; that many of the released asbestos structures are indeed fibers and not just bound into bundles, clusters, or matrices; and that sufficient amounts exceed the then 1985 OSHA 15-min ceiling of 10 f/cc and the current excursion limit of 1 f/cc over a 30 min time period [Faigout, 1985]. A more recent study by Blake et al. [2003] has also found higher concentrations of asbestos fibers released during arc grinding but were not above the current OSHA 8 hr TWA PEL. Chrysotile fibers were the only fiber type found in this study of six brake shoe changes and one cleaning test. While this study does not indicate, under the conditions of the study, a high concentration of asbestos fiber concentration over the 8 hr TWA, it does re-iterate that encapsulated fibers of asbestos are released as the brakes are manipulated. Other, so-called encapsulated products, similar to brakes, such as gaskets have also been shown to release respirable asbestos fibers exceeding both the earlier 15 min and the current 30 min excursion limits of the OSHA PEL and in some cases the OSHA 8 hr TWAs [Longo et al., 2002].

Fiber release studies of actual brake repair and replacement and the laboratory simulation studies both demonstrate the ability of encapsulated asbestos containing brake products, to release respirable asbestos fibers at concentrations capable of causing asbestos related disease. But another aspect, generally overlooked when evaluating the ability of asbestos to cause disease in auto mechanics,

is the adequacy of floor cleaning methods to remove the residual asbestos containing dusts. One study [Phillips and Hamilton, 1994] evaluated this process and found 23% of the businesses allowed employees to dump wheel dram dust directly on the shop floor and that 29% of the firms used dry sweeping instead of wet methods for clean-up, thus reintroducing the aerosolized asbestos fibers into the air for workers to breathe. For these workers, overall exposures would have been greater because of the additive nature of these additional exposures, from clean-up, to those already encountered while servicing the brakes during the repair or replacement process.

Evidence of Disease in Persons Exposed to Asbestos From Brakes

The National Safety Council reported that asbestos used in brakes was potentially harmful [Castrop, 1948]. Mesothelioma has been reported among brake mechanics [McDonald et al., 1970], their wives [Ziem, 1984], and children [Environmental Protection Agency, 1986]. Mesothelioma has been described among a variety of asbestos exposed persons. Huncharek et al. [1989] describes a 47-year-old lifetime non-smoking man whose only known exposure to asbestos occurred while he was a brake mechanic from age 30 to 41, giving him a latency of 17 years. Langer and McCaughey [1982] reported only chrysotile fibrils in the lung parenchyma tissue of a 55-year-old brake repair worker of which 10% were longer than 10 μm. They further describe that "... besides this submicroscopic chrysotile fibre in brake drum housing there is a more significant source of free, unaltered fibre in the bevelling, refurbishing, and refitting of brake pads. There is thus ample opportunity, during brake maintenance and repair, for contact with chrysotile fibre both in drum debris (where it will usually be in a transformed state) and as long and predominantly unaltered fibers liberated by machining." Langer and McCaughey [1982] also reported that pathological diagnosis of asbestos-related diseases in people exposed to chrysotile is complicated because asbestos-bodies do not form readily. Vianna and Polan [1978] reported two mesotheliomas in women whose husbands had exposure to brake linings, and one woman who was a textile worker and whose husband was a brake lining worker. Godwin and Jagatic [1968] reported two cases of mesothelioma, one in a 43-year-old woman, with peritoneal mesothelioma, who had spent 3 years weaving brake linings made of chrysotile asbestos and the second in a 50-year-old man who worked 5 years in a Canadian asbestos mine who gave X-ray diffraction evidence of only chrysotile present in his body.

Mesotheliomas have also been observed in pets. In one study of 18 dogs diagnosed with mesothelioma, the owners for 16 were identified and 12 were able to identify possible sources of asbestos exposure. The owners of two of the dogs were car and truck mechanics, respectively [Glickman et al., 1983].

Epidemiological studies have been equivocal. For example Rushton et al. [1983] concluded that their study, while negative, suffered from small numbers of men and that further follow up time would be required to determine any definite causal mortality patterns. Teta et al. [1983] reported a relative risk of 0.65 for automobile repair and related service when they observed a single case of mesothelioma among auto repair workers in their 220 cases reported in the Connecticut Tumor Registry from 1955 to 1977. They concluded that difficulties in ascertaining occupational histories, in their study population thus indicated a better need for record keeping as well as a lack of detailed information regarding the residual cases could obscure the true number of occupationally exposed cases. Teta et al. [1983] also found three case of mesothelioma, one definite peritoneal, one probable pleural, and one possible pleural, in worker at the Raybestos brake manufacturing facility in Connecticut. Robinson et al. [1979] also found among the deaths observed in a friction production plant 17 were the result of mesotheliomas, representing 4.3% of the deaths.

Rodelsperger et al. [1986], report that approximately 300,000 mechanics in the automotive service stations in Germany are exposed to asbestos. In their clinic they have observed four cases of mesothelioma which is clearly not representative of the overall incidence of mesothelioma among brake mechanics, but an indication of the disease occurring in such workers. Wong [1992] reports that the three cases (actually four) observed by Rodelsperger et al. [1986] are not over the background rate. Given that there might exist a background level of mesothelioma occurring in the absence of exposure to asbestos, even though there is no proof of this, this "natural level" is probably much lower than the 1–2/million/year which has often been cited [Hillerdal, 1999], therefore, three or four cases may well be more significant than attributed to by Wong. Jarvholm and Brisman [1988] reported no excess of mesothelioma but a slight increase in lung cancer among car mechanics. In their cancer linkage study of using Swedish census and death register data they found a case of mesothelioma, but concluded that because other exposures could not be ruled out that such a study methodology can not answer the question concerning cancers among car mechanics. Hansen [1989] discovered a case of mesothelioma which she attributed to asbestos exposure in her study of Danish auto mechanics. Spirtas et al. [1994] reports 33 cases of mesothelioma in persons having stated as part of their occupational history brake repair work. One of the confounding factors preventing Spirtas et al. [1994] from calculating a relative risk was that an overwhelming majority of those workers had also been exposed as insulators or shipbuilders.

In a study of mesothelioma among car mechanics in Germany, the authors found no evidence of an increased risk

of mesothelioma. They concluded, however, that if there was a mesothelioma risk it could not be detected by their methodology. Further they stated that the absence of chrysotile fibers in the lung tissue of one of the cases did not exclude the possibility that, decades before, chrysotile fibers were active at the target cells [Woitowitz and Rodelsperger, 1994].

An additional limitation of the Woitowitz and Rodelsperger [1994] study is that the authors looked only at lung tissue and not pleural tissues. The authors also excluded a number of cases because the workers had other asbestos exposures in addition to their brake work. This exclusion is significant because both asbestos exposures at brake work and during other employments add to an individual's overall body burden of asbestos intake, thus, the role of exposures during brake work can not be discounted in considering an individual's overall risk of developing asbestos-related diseases. Another limitation was the use of hospital controls that had contracted lung cancer which might implicate exposures to asbestos. A recent evaluation of the German mesothelioma registry records 48 cases of mesothelioma in the automobile sector [Neumann, 2001].

Teschke et al. [1997] who found 6 mesothelioma cases in vehicle mechanics among 51 cases of mesothelioma did not find an excess of this disease in this occupation. The authors concluded that most of their cases of mesothelioma were explainable by exposure to asbestos and acknowledge their findings were based on small numbers of cases and any judgments about any causal associations would be speculative.

In a dose-response study to low levels of asbestos exposure, in a French-based case-control study 82% of motor vehicle mechanics had frequent exposure. The authors found a clear dose-response relation between cumulative exposures and pleural mesothelioma and that a significant excess of the mesothelioma was observed at levels that were probably below the limits adopted in most industrial countries [Iwatsubo et al., 1998]. Anderson et al. [1973] reports that 58 mesotheliomas were reported among Australian brake mechanics having no other exposures to asbestos and that only a small fraction of the total 82,827 mechanics in Australia worked with brake blocks or brake linings. He concludes that these 58 cases represent 1,062,946 person-years. If one rounds off the total mechanics to 100,000 mechanics this represents 45 mesotheliomas per million person-years and that if one doubles this number to 200,000 mechanics to include retirees and workers who moved to other occupations then the mesothelioma rate becomes 22.6 per million person-years, a rate substantially above the upper limit of the estimated background rate of 1–2 mesotheliomas per million person-years or around a 10-fold excess. The Australian mesothelioma tumor registry has reported that when analyzing their data for the years 1945 through 2002 that the overall lifetime risk of mesothelioma for vehicle mechanics was 0.7% which is much higher than the background lifetime risk for Australia of 0.007% (70 per million). When analyzing the mesothelioma cases between 1986 and 2001 there were 78 cases with a history of asbestos exposures during either brake lining repair or manufacturing work. Broken down it was reported that 73 were among brake lining repair workers, 5 from manufacturing workers, and that in 43 brake lining repair was their only source of asbestos exposure. When looking at 1980–1985, eight more cases were reported with only brake lining work as their source of exposure. The most recent data for the years 2002–2003 report another eight cases, three of which reported only brake lining work as their only source of asbestos exposure. One of the authors, Dr. James Leigh, reports that when using only the data from 1986 to 2001 (43 cases of vehicle mechanics with no other exposures) and statistics on the number of mechanics in the country that the resulting rate of 32.5 per million person-years represents over 30-times the commonly accepted "background" mesothelioma rate of 1 per million person-year. When equating this to the lifetime risk, calculated by the author of 0.38%, this amounts to approximately 55-times the background lifetime risk of 70 per million (0.007%) [Leigh, 2003; Leigh and Driscoll, 2003].

Conclusions Concerning Asbestos and Brakes

A review of the published peer reviewed literature reveals at least 165 cases of mesothelioma in end-product users of friction products. Additional government studies have reported other cases. These numbers can not be attributed ambient air exposure or to chance alone [Newhouse and Thompson, 1965: 1; Godwin and Jagatic, 1968: 1; McDonald et al., 1970: 2; Oels et al., 1971: 1; Greenberg and Davies, 1974: 1; Castleman et al., 1975: 1; Vianna and Polan, 1978: 2; McDonald and McDonald, 1980: 11; Kagan and Jacobson, 1983: 1; Guillon, 1984: 1; Huncharek, 1987: 3; Woitowitz and Rodelsperger, 1994: 16; Teschke et al., 1997: 6; Agudo et al., 2000: 3; Neumann, 2001: 48; Roggli et al., 2002: 24 = 165; and Leigh and Driscoll, 2003: 43]. Milham and Ossiander [2001] of the Washington State Department of Health report: 7; the Environmental Protection Agency, 1986: 6 cases].

The results of the exposure studies, experimental studies, case reports, and findings from the equivocal epidemiological studies by no means exonerate the brake mechanic from being susceptible to a causal relationship between asbestos exposure and mesothelioma. In conclusion several facts remain evident:

- encapsulated asbestos containing brakes do release asbestos fibers when the brakes are both used and manipulated and at concentrations capable of causing disease;

- short asbestos fibers (<5 μm in length), often found in brakes and brake residue, have been shown to pose a risk of disease;
- the application of control methodologies has resulted in the reduced exposures to asbestos, but have not been able to entirely eliminate exposures;
- additional asbestos exposures, beyond those encountered directly from work with brakes, occur from faulty work practices and clean-up methods which are not appropriate;
- OSHA has stated that their current standard for asbestos [0.1 f/cc] will not eliminate the risk of asbestos-induced cancers [OSHA, 1986], and;
- the International Program for Chemical Safety has concluded that no threshold has been identified for carcinogenic risks for chrysotile asbestos, the principle fiber type used in asbestos-containing brakes [IPCS, 1998].

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