ACADEMIC PRESS

Available online at www.sciencedirect.com

SCIENCE DIRECT®

Regulatory Toxicology and Pharmacology 38 (2003) 71–77

Regulatory Toxicology and Pharmacology

www.elsevier.com/locate/yrtph

Reduction of the biological potential of chrysotile asbestos arising from conditions of service on brake pads

Arthur M. Langer*

Ph.D. Program in Earth and Environmental Sciences, The Graduate School and University Center, 365 Fifth Avenue, New York, NY 10016, USA

Received 1 November 2002

Abstract

Data exist that show that chrysotile asbestos does not retain its mineral properties, or biological activity, at temperatures far below the olivine transformation point. Temperatures hundreds of degrees below this point cause the mineral to lose structural water with accompanying crystal structure degradation. The loss of structure is accompanied by modification of its surface and reduction or loss of biological activity. Using heating studies and milling as an approximation of thermal and mechanical shear stress that chrysotile is subjected to on a brake lining, biological blunting is shown to begin much earlier than the olivine transformation process. Minimal degradation of the chrysotile surface structure imparts a disproportionately great effect on its biological activity. Biological and epidemiological data for brake workers exposed to chrysotile asbestos should be viewed in context with the conditions of service to which the product was subjected over a lower range of temperatures than previously considered important.

Keywords: Chrysotile asbestos; Brake pad; Service conditions; Structural degradation; Surface modification

1. Asbestos dust in a brake mechanics environment

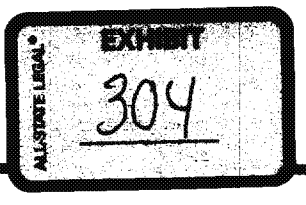
There have been many studies undertaken here in the United States, as well as overseas, concerning the release of asbestos into the air of garages where brake maintenance and repair is undertaken (read review in Nicholson et al., 1982). For example, a longitudinal study over 5 years (1972–1976) conducted by the National Institute for Occupational Safety and Health, cited in Nicholson et al. (1982), found a time-weighted mean of 0.18 f/ml of air for automobile brake repair work, with a range of 0.01–1.34 f/ml of air. The high value outlier was obtained in a garage in which dust control devices were absent.

Garages in which dust controls were present have consistently yielded values between 0.02 and 0.30 f/ml of air (Marsh, 1979; Raybestos Manhattan unpublished data, communicated to and reported in Nicholson et al., 1982). Municipal garages in New York City in the mid-1970s, with available control devices in place at that time, yielded a mean value of 0.22 f/ml of air for activities that included changing brake pads, brushing off wear debris, blowing off of wear debris, and sweeping up debris at the end on the work day (Nicholson et al., 1982).

Hickish and Knight (1970) found very low values for automobiles in Great Britain, but measured higher values for truck brake work. Outliers of up to 1.48 f/ml of air for an 8-hour time-weighted average were measured in some garages. Brake replacement, beveling, and cleaning truck brakes here in the United States also produced high excursion values, but much lower time-weighted averages than reported by Hickish and Knight. An average short-term excursion of about 37 f/ml of air, measured for a time period of several minutes, was obtained during the beveling of Department of Sanitation truck brakes yielding a time-weighted average of about 0.23–0.62 f/ml of air (Rohl et al., 1976). Rohl et al. used the NIOSH 7200 direct method for the analysis of personal air samples, but dust samples were prepared with the "rub-out" technique: This latter method of preparation tends to create diameter artifacts so that fiber number and width distribution measurements were not made.

* Corresponding author. Fax: 1-914-946-6524.
E-mail address: artlanger@aol.com.

0273-2300/03/$ - see front matter
doi:10.1016/S0273-2300(03)00070-9



The highest levels of asbestos measured in the air of garages followed work grinding and refurbishing truck brakes, compressed-air blowouts of wear debris from brake housings in general, and dry, uncontrolled sweeping of wear debris from the floors of these facilities (Nicholson et al., 1982).

Release of fiber from brake pads used in other devices has been investigated as well. Simulation studies on large presses, reported by Eimermacher (1980), revealed an average fiber release of 0.022 f/ml of air as an 8-h time-weighted average.

2. Brake mechanics and the asbestos diseases

Nicholson et al. (1982) examined 421 workers, members of several unions in New York City, who engaged primarily in brake service and replacement activities. The experience of this group was compared to other categories of workers who were also employed in the garage setting, e.g., auto body workers, under coaters, and welders. Among the brake repair workers, parenchymal markings (profusion ranking according to the ILO/UC classification equal to or greater than 1/0) were found in 101 of the 421 men (~24%), with 34 cases showing pleural thickening as well. However, pulmonary function within this group was better than found among persons in the general population of the state of Michigan examined just prior to this time. Pulmonary decrements were observed only among older workers who were cigarette smokers, men with occupational histories reflecting exposure to asbestos dust in other settings, e.g., shipyard employment, and among the auto body workers in general. Auto body repair involved exposure to mineral dust during grinding of fillers and to welding fumes. It was this group of garage workers that also showed the greatest profusion of markings and pulmonary function loss among all the categories of workers studied. Study of brake workers with uncomplicated work histories failed to show the presence of "serious" abnormalities.

Proportional mortality studies on groups of workers engaged in automotive or brake repair have shown that their cancer deaths (and for mesothelioma specifically) were equal to or less than values calculated for their respective control groups. Of significance, mesothelioma among these workers was either not found at all or equal to the number of cases anticipated for the population as a whole (e.g., McDonald and McDonald, 1980; Teschke et al., 1997; Teta et al., 1983; Woitowitz and Rodelsperger, 1994). In other mortality studies, lung cancer deaths were also low, and in instances where the anticipated number of cases was slightly greater than expected, the excesses were explained equally well by common confounders, e.g., cigarette smoking and diesel exhaust, rather than asbestos (e.g., Gustavsson et al., 1990; Jarvhol and Brisman, 1985).

3. Biologically important properties of the asbestos minerals

Most experimentalists in the asbestos field today would acknowledge that length of fiber is an important determinant of biological activity. Fibers with lengths well below 5 μm are considered "inert" while those exhibiting lengths just below this value exhibit markedly less activity than their counterparts of greater length when tested in the same system. Simply put, the physical status of the fiber and the state of aggregation of its dust are important variables in judging the degree of hazard associated with an asbestos exposure. Fiber length has been used as a handy "index" of activity. An overview of the fiber length issue is provided in Langer et al. (1978).

In that paper, Langer and colleagues focused on the importance of surface properties by providing data confirming that length reduction methods employed by experimentalists in the past inadvertently imposed both surface and structural modifications on the fiber. They noted that both length reduction and surface and structure modification co-varied so that early interpretations of biological outcome based on one parameter change (length) could be as well explained by other changes that were not obvious to, nor measured by, the investigators. A discussion of surface properties and activity, with a review of major papers in the literature, was provided in Langer and Nolan (1994a,b). The hypothesis advanced in those papers was that the surface chemistry and properties of mineral fiber (and minerals in general) were the *primary* determinants of biological activity. Further, any modification of these properties, by any means, would result in corresponding changes in activity.

4. Surface properties drive the activity of mineral fiber

Macnab and Harington (1967) demonstrated that asbestos fibers behave as other fibrogenic minerals do, that is, they are membrane active. They later demonstrated that the membrane activity of chrysotile asbestos might be blunted if the chemical functionalities that existed on the surface of the mineral were "blocked" with serum proteins, phosphate groups from buffered saline, and other negatively charged compounds (Harington et al., 1971). To support the magnesium functionality theory, they removal magnesium from the *surface* of the fiber with the chelator EDTA (ethylenediamine tetraacetic acid), retested it in the same in vitro system, and rendered chrysotile inactive. Later experi-

mental use of acid-treated chrysotile, leached of magnesium, showed that mesothelioma could not be induced in laboratory animals (Morgan et al., 1977). In all experimental studies the ionized magnesium functionality at the surface of the fiber was considered to be the site of biological interaction.

Schnitzer blunted chrysotile activity by means of modification of the fiber's surface chemistry (e.g., Schnitzer, 1974). Schnitzer found that negatively charged polyanions, covalently bound to the mineral, inactivated the fiber surface. Magnesium again was the binding site. Polyanionic compounds worked best as antagonists, either markedly inhibiting or completely blocking membrane activity.

5. Heating and milling of chrysotile

Depending on the nature and magnitude of the manipulation, heating and milling of chrysotile induces profound change in the mineral. Heating of the fiber in the temperature range well below its complete breakdown temperature induces subtle thermal decomposition that creates a chemically, structurally and surface altered mineral (Hodgson, 1979).

Heat dehydroxylates chrysotile in a diffusion-controlled manner that begins at temperatures as low as 150 °C (see review of studies in Hodgson, 1979). Some 2.5% of the structural water is lost in the temperature range 150–500 °C. Complete water-loss occurs at 650 ± 20 °C, noted by a characteristic strong endothermic peak on differential thermal analysis (Martinez, 1966). This marks the complete breakdown of the mineral structure and creation of an amorphous mixture of silica and magnesia, which mineralogists have dubbed "serpentine anhydride."

The mechanism by which water is driven from chrysotile is thought to be donor–acceptor in character. The donor regions of the mineral supply cations and oxygen to the acceptor regions where they react with protons to form water. The donor regions thereby lose material to form "pores" in the surface structure. Monkman (1967) initially described the breakdown of the chrysotile "lattice" at about 525 °C, a temperature almost 300° below the major olivine transformation point. Incipient olivine formation has been recognized forming at temperatures hundreds of degrees below 810 °C (Naumann and Dresher, 1966).

Milling of chrysotile reduces fiber length and causes structural and surface changes as well. However, milling involves superimposed thermal events so that some effects cannot be process separated. Milling superimposes physical changes as well as chemical ones and similar alteration of the mineral's properties can be followed (Langer et al., 1978).

6. Conditions generated during brake pad service

Brake pads used in automobiles and trucks are made of a complex of inorganic property modifiers held in a phenolic resin thermo set matrix that is chrysotile fiber reinforced (Rohl et al., 1976). Vehicle motion is slowed ("decelerated") by conversion of forward motion (kinetic energy) into friction and heat (Anderson, 1969; Carroll, 1962). During the process the components of the pads are subjected to extreme thermal and shear stress (Anderson, 1987). The "rubbing interface" on the pad during "normal" service attains temperatures of up to 650 °C, but "hot spots" generate temperatures well above this, some reaching as high as 1000 °C (Anderson, 1987). The former temperature is sufficient to completely dehydroxylate chrysotile and the latter is well above the olivine transformation temperature, which occurs at about 810–820 °C.

The heating of chrysotile asbestos at the brake surface at temperatures sufficient to drive off structural water (differential thermal analysis indicates a gradual loss of structural water in the 150–500 °C range) is accompanied by about 30% loss of the fiber's structure, indexed as loss of X-ray counts for reflections of principal structural planes (Table 1). Virtually 70% of the chrysotile structure is lost by 575 °C. Total loss of structure occurs at about 650 °C the temperature at which water has been completely removed and chrysotile the mineral no longer exists. The end product is an

Table 1
Thermal stress of chrysotile and loss of mineral structure

Temperature of heating		X-ray intensity in CPS		Observations
Fahrenheit (F)	Centigrade (°C)	(002)	(004)	
750	~400	1100	700	No structural change
930	~500	900	500	30% of structure lost
1070	~575	300	300	70% of structure lost
1200	~650	~0	~0	100% of structure lost
1500	~810	0	0	100% of structure lost

These data are from Butler (1980).

The degradation of the chrysotile structure was followed by loss of X-ray counts under two major reflection peaks, the 7.3 Å *d*-spacing (002), and the 3.65 Å *d*-spacing (004). CPS, counts per second. At 650 °C chrysotile loses all its structural water and forms serpentine anhydride.

amorphous mixture of magnesia and silica (read in Hodgson, 1979).

The heating of chrysotile was also studied by the asbestos industry. They, however, followed application properties rather biological change. The industry reported degradation of fiber strength with increasing temperature. Even heating of chrysotile fiber for a time as short as 3 min at 650 °C resulted in 68% loss of the fiber's strength (Sinclair, 1952). Fiber strength and structural integrity co-vary.

7. Mechanical forces exerted on chrysotile during braking

The brake contact interface subjects the pad to mechanical shear stress. The stress may be severe, especially at times involving "emergency" stops. Chrysotile fiber is greatly reduced in size by this action as evidenced by the measurement of fiber lengths in brake drum dust (Rohl et al., 1976). These investigators studied recovered dusts by transmission electron microscopy and found that 56–99% of the fibers in these populations were less than 0.4 μm in length. Very few fibers in the wear debris examined were 5-μm or longer in length. Selected area diffraction characterization of surviving "long" fiber bundles indicated that structural degradation of the mineral had taken place.

8. Studies that mimic the conditions on brake pads during service

Langer et al., 1978) studied the physical milling of chrysotile asbestos and accompanying modification of its properties. The fiber source and character, techniques of study, preparation protocols, and analytical details were published in that report. These data are used here as an approximation of the effects on chrysotile caused by shear stress at the brake pad interface based on similar features that characterize both brake wear debris and milled fiber. First, there occurs a marked reduction of fiber length so that the resulting population is shorter than that which characterized the material originally used in the product. Secondly, the shortened fibers exhibit both structural degradation and surface property change.

The milling of chrysotile, and its effect on the size distribution of the mineral, was followed by electron microscopic examination. The particle population became so small that fiber measurement and characterization was beyond the resolving power of the light microscope. The sizing of the population necessitated that measurement be undertaken on photomicrographs obtained by transmission electron microscopy at 10,000× magnification. Some 5169 such measurements were made.

The milling of chrysotile asbestos for a length of time between 1 and 5 min (60–300 s) reduced the total measured fiber and fibril population to less than 5 μm in length (Table 2). The limit of detection of less than 5 μm objects, based on the number of objects below this size ($n = 3954$) is ~0.025%, less than three parts in 10,000. Fibrils (single unit strands of chrysotile about 0.3 μm in diameter) were reduced to less than 5 μm lengths in a time period shorter than 1 min (Table 2). Mechanical manipulation of chrysotile opens fiber bundles and reduces fiber length.

9. Accompanying reduction of crystallinity

The milling of chrysotile for a period of less than 1 min produced a loss in the mineral's crystallinity (Langer et al., 1978). This was first noted as the increased rapidity of fibers and fibrils to undergo electron beam damage (described in Langer et al., 1974). Table 3 illustrates two properties measured on the bulk powder, the reduction of X-ray counts under the strongest reflection of chrysotile (d-spacing ~7.3 Å), and a corresponding change in its infrared (IR) spectrum. Between five- and ten-minutes there was a marked loss of crystallinity followed as a loss of X-ray counts under the

Table 2
Change of fiber and fibril length with milling time

Sample	Fibers				Fibrils			
	Fiber length (μm)				Fibril length (μm)			
	N	<1	1.0–4.9	>5.0	N	<1	1.0–4.9	>5.0
As received	214	140 (64.4)	64 (29.9)	10 (4.7)	800	692 (86.6)	98 (12.3)	10 (1.2)
60-s	191	108 (56.5)	80 (41.7)	3 (1.8)	779	746 (95.7)	33 (4.3)	0 (0.0)
300-s	251	217 (86.5)	34 (13.5)	0 (0.0)	962	952 (99.0)	10 (1.0)	0 (0.0)
1200-s	162	160 (99.0)	2 (1.0)	0 (0.0)	829	827 (99.7)	2 (0.3)	0 (0.0)
3600-s	123	123 (100.0)	0 (0.0)	0 (0.0)	858	857 (99.9)	1 (0.1)	0 (0.0)

These data are from Langer et al. (1978).

The progressive milling of chrysotile produces a less than 5-μm length population of fibers and fibrils in less than 5 min. Fibrils are length reduced more rapidly than fibers. Total particle population measured was 5169 fibers and fibrils.

Table 3
Degradation of chrysotile structure with milling time

Sample	XRD (7.36 Å) X-ray counts	IR spectrum of Si–O–Mg at 1020 cm^{-1}
As received	8450	Standard chrysotile spectrum
60-s	8360	Standard chrysotile spectrum
300-s	7461	Slight broadening at 1020 cm^{-1}
1200-s	4977	Marked broadening at 1020 cm^{-1}
3600-s	3541	Marked broadening at 1020 cm^{-1}

These data are from Langer et al. (1978).

Chrysotile's 7.36 Å *d*-spacing corresponds to its (002) plane, its strongest reflection.

The broadening of the 1020 cm^{-1} reflection reflects an increase in the Si–O–Mg bond length and a corresponding decrease in bond strength.

The milling process imparted a 58% loss of structure in 3600 s, a structural degradation corresponding to loss measured when chrysotile is heated to 575 °C for 24 h.

7.3 Å reflection and an ever-increasing bond length and change in co-ordination between the silica and brucite layers of the mineral (indicated by both shift and broadening of specific vibration bands in the IR spectrum).

Distortion of chrysotile's crystal lattice was also studied by means of electron spin resonance (Langer et al., 1978). Loss of hyperfine resonance was observed with progressive milling with accompanying resonance spectral broadening and reduced intensity. The authors reported the same effect when the mineral was heated to temperatures slightly below 450 °C. Heating to temperatures in the 600–800 °C range produced a marked shift in the position of the resonance peak with an accompanying loss of peak intensity. These changes have been interpreted as dehydroxylation of chrysotile's brucite layer in the former instance and the recoordination of the anhydrous magnesium silicate to olivine (forsterite).

10. Alteration of chrysotile surface properties

Both milling and thermal events altered chrysotile's ability to physisorb or interact with organic compounds (Langer et al., 1978). The authors noted that the ability of chrysotile to reduce the stable free radical diphenylpicrylhydrazyl to the hydrazine state was markedly reduced when the mineral was milled. Further, its ability to bind polar molecules was also greatly reduced.

11. Change in membranolytic behavior

Langer and Nolan (1994a,b) also studied the biological behavior of manipulated chrysotile. Table 4 shows the fiber's hemolytic behavior and its antagonist binding properties. In the time period between 1 and 5 min the ability of the chrysotile to rupture red cell membranes was reduced by one-third. It was found that this activity loss was associated with ~12% loss of the mineral structure (Table 3). The structural disruption appeared to begin on the mineral surface. Milling beyond this time reduced chrysotile membrane activity to even greater degree.

Table 4
Alteration of membrane activity and surface binding character with milling time

Sample	Hemolysis percent (1 mg/ml)	Amount of chrysotile to produce 50%H (mg)	Antagonist CMC 4 μg/ml %H
As received	100.0	0.4	21
60-s	93.8	0.4	38
300-s	65.0	1.0	29
600-s	28.8	2.6	19
1200-s	15.0	4.4	11
3600-s	—	7.5	6

These data are from Langer et al. (1978).

The antagonist compound used was carboxymethylcellulose, CMC.

Alteration of the surface character was demonstrated by the change in the ability of the mineral to bind compounds that antagonize hemolysis. Although milling increased the surface area of chrysotile dust, it altered the brucite layer's proton distribution thereby disrupting its electron flow properties and reducing its membrane activity (Langer et al., 1978).

12. Discussion

For the most part, studied groups of workers that have been chrysotile-exposed in brake repair environments show no statistically significant excess asbestos-associated cancer mortality. The small excesses of lung cancers that have been observed are explained equally well by the presence of confounding agents. Brake installers and maintenance workers appear to exhibit no increased risk of mesothelioma.

These findings have previously been explained by low cumulative exposure to asbestos dust, short fiber length, fiber type, conversion of chrysotile to olivine in the high temperatures generated during brake service, and the presence of confounders. Generally omitted from consideration in these studies is the accompanying partial modification of the fiber's properties that blunts activity without ever requiring conversion to olivine. The presence of "short fiber" might actually be an index of altered fiber.

Many investigators consider only the olivine transformation temperature as the point at which chrysotile is converted to a biologically inactive substance. However, mechanical and thermal events cause effects hundreds of degrees below this temperature. Profound changes in the mineral's crystal structure, with

accompanying changes in its surface character, begin to occur at temperatures as low as 150 °C. Seventy percent of the mineral's structure is lost by 575 °C. At temperatures of about 650 °C the chrysotile completely dehydroxylates to form "serpentine anhydride," a material void of structure and absent of chrysotile properties.

13. Conclusions

Studies have shown that thermal treatment and mechanical manipulation of chrysotile alters both its surface and structure. Service conditions created on brake pads both heats and tears down the fiber. Chrysotile subjected to these severe conditions cannot, and does not, retain its natural properties. Chrysotile biological activity is thereby greatly reduced, and can become virtually nil hundreds of degrees below the olivine transformation temperature. Complete transformation of the mineral is not required to result in loss of activity. Exposure to brake wear debris, which has been created as the result of these forces, may be associated with little or no risk of asbestos disease. Blowouts and cleanups of wear debris, both visually dusty work practices, might constitute no asbestos hazard to workers.

Exposure to fiber in these work environments might occur as the result of product manipulation during brake installation. Beveling and arcing of pads to fit vehicles, cleaning and refurbishing of brake surfaces (especially large truck brakes, see Rohl et al., 1976) produce dust that has not been subjected to the same conditions as pad service. These practices require the attention of the dust control engineer and industrial hygienist. Modern dealerships supply brake pads that are manufactured as model-specific replacements. Design change has aided in the improvement of workplace safety.

There are several other issues to consider. The NIOSH-OSHA environmental assay instrument for mineral fiber (phase contrast optical microscopy) generally cannot distinguish between altered and unaltered fiber. Such assays in the past "overstated" exposure and attendant hazard. The assay tool could distinguish form only. Workers in such environments were afforded that much more protection per fiber count. Further, structurally damaged chrysotile is more sensitive to chemical degradation, and based on size and damage measurement, the fibers likely lack biopersistence and might breakdown more readily in the lung.

Acknowledgments

The following acknowledgment of support was given in the two major papers from which data were taken. Langer et al. acknowledged support under a Center Grant from the National Institute of Environmental Health Sciences, ES 00928. Rohl et al. were supported by a grant from the Health Research Council of New York City HRC U 2329. Rohl et al. also acknowledged financial assistance from the Ford Motor Company.

References

Anderson, A.E., 1969. Wear in brake materials. Proceedings of the American Society of Metals Wear Conference.

Anderson, A.E., 1987. Brake system performance. Effects of fiber types and concentrations. Proceedings of the Fibers in Friction Materials Symposium. The Asbestos Institute, Montreal, Canada, pp. 2–49.

Butler, M.A., 1980. The Physical and Chemical Characteristics of Serpentine Rocks and Minerals. Ph.D. Thesis submitted to the Department of Mineral Exploitation, University College Cardiff, Wales, 348 pp.

Carroll, W.G., 1962. The manufacture of brake linings. Br. Plastics 8, 414–417.

Eimermacher, J.P., 1980. Clutch and Brake Lining Investigation. Test Report Document No. 4344. Cincinnati Incorporated, September 19, 1980. 7p and attachments, unpublished study.

Gustavsson, P., Plato, N., Lidstrom, E.B., Hogstedt, C., 1990. Lung cancer and exposure to diesel exhaust among bus garage workers. Scand. J. Work Environ. Health 16, 348–354.

Harington, J.S., Miller, K., Macnab, G., 1971. Hemolysis by asbestos. Environ. Res. 4, 95–104.

Hickish, D.E., Knight, K.L., 1970. Exposure to asbestos during brake maintenance. Ann. Occup. Hyg. 13, 17–21.

Hodgson, A.A., 1979. Chemistry and physics of asbestos. In: Michaels, L., Chissick, S.S. (Eds.), Asbestos. Wiley, New York, pp. 67–114.

Jarvhol, B., Brisman, J., 1985. Asbestos associated tumors in car mechanics. Br. J. Ind. Med. 45, 645–646.

Langer, A.M., Nolan, R.P., 1994a. Factors controlling the biological potential of inorganic dusts: surface chemistry and character. In: Mohr, U. et al. (Eds.), Toxic and Carcinogenic Effects of Solid Particles in the Respiratory Tract. ILSI Monographs. ILSI Press, Washington, DC, pp. 147–157.

Langer, A.M., Nolan, R.P., 1994b. Chrysotile: its occurrence and properties as variables controlling biological effects. Ann. Occup. Hyg. 38, 427–451.

Langer, A.M., Mackler, A.D., Pooley, F.D., 1974. Electron microscopical investigation of asbestos fibers. Environ. Health Perspect. 9, 63–80.

Langer, A.M., Wolff, M.S., Rohl, A.N., Selikoff, I.J., 1978. Variation of properties of chrysotile asbestos subjected to milling. J. Toxicol. Environ. Health 4, 173–188.

Macnab, G., Harington, J.S., 1967. Hemolytic activity of asbestos and other mineral dusts. Nature (London) 214, 522.

Marsh, J., 1979. Unpublished data from Raybestos Manhattan communicated to William Nicholson. Cited in Nicholson et al., 1982.

Martinez, E., 1966. Chrysotile asbestos: relationship of the surface and thermal properties to the crystal structure. Can. Mining Metallur. Bull. 69, 414–420.

McDonald, A.D., McDonald, J.C., 1980. Malignant mesothelioma in North America. Cancer 46, 1650–1656.

Monkman, L., 1967. Thermal decomposition of chrysotile asbestos. In: Proceedings of the First International Conference on the Asbestos Minerals. Published Abstract. Oxford University, May 1967.

Morgan, A., Davies, J.C., Wagner, J.C., Berry, G., Holmes, A., 1977. The biological effects of magnesium-leached chrysotile asbestos. Br. J. Exp. Pathol. 58, 465–473.

Naumann, A.W., Dresher, W.H., 1966. The influence of sample texture on chrysotile dehydration. Am. Mineral. 51, 1200–1211.

Nicholson, W.J., Daum, S.M., Lorimer, W.V., Velez, H., Lilis, R., et al., 1982. Investigation of the health hazards in brake lining repair and maintenance workers occupationally exposed to asbestos. Report to the National Institute for Occupational Safety and Health. NIOSH Contract 210-77-0119. Unpublished Report, 99 pp. and appendix.

Rohl, A.N., Langer, A.M., Wolff, M.S., Weisman, I., 1976. Asbestos exposure during brake lining maintenance and repair. Environ. Res. 12, 110–128.

Schnitzer, R.J., 1974. Modification of the biological surface activity of particles. Environ. Health Perspect. 9, 261–266.

Sinclair, W.E., 1952. Asbestos: Its Origin, Production and Utilization. Mining Pub., Salisbury House, London.

Teschke, K., Morgan, M.S., Checkoway, H., Franklin, G., Spinelli, J.J., van Belle, G., Weiss, N.S., 1997. Mesothelioma surveillance to locate sources of exposure to asbestos. Can. J. Public Health 88, 163–168.

Teta, M.J., Lewinsohn, H.C., Meigs, J., Vidone, R.A., Mowad, L.Z., Flannery, J.T., 1983. Mesothelioma in Connecticut, 1955–1977. Occupational and geographic associations. J. Occup. Med. 25, 749–756.

Woitowitz, H.J., Rodelsperger, K., 1994. Mesothelioma among car mechanics. Ann. Occup. Hyg. 38, 635–638.