Persistence of Natural Mineral Fibers in Human Lungs: An Overview

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Virtually all available data on persistence of naturally occurring mineral fibers in human lungs have been derived from studies of asbestos fiber loads. These studies indicate that, although both amphibole and chrysotile asbestos fibers are found in the lungs of the general population and exposed workers, amphibole fibers are universally present in disproportionately large and chrysotile fibers in disproportionately small amounts compared to their known abundance in the original inhaled dusts. Why this should be remains unclear. Most reports have shown that fiber accumulation is proportional to measured exposure for amphiboles, but this is not generally true for chrysotile. Very little information is available on actual fiber clearance rates from human lungs. For amosite and crocidolite, estimated clearance half-times are measured in years to decades, whereas for chrysotile the available, rather indirect, data suggest that the vast majority of fibers are cleared within months, although some fibers may be sequestered and very slowly cleared. Overall these studies suggest that the differences between amphibole and chrysotile fiber burdens in man reflect much faster clearance of chrysotile fibers, rather than a failure of chrysotile deposition. A variety of other naturally occurring fibers are commonly found in human lungs, but there are no data on their rates of accumulation or disappearance. — Environ Health Perspect 102(Suppl 5):229–233 (1994)

Key words: asbestos, lung, clearance, deposition

Introduction

Over the last two decades numerous studies have reported the human pulmonary burden of naturally occurring mineral fibers, particularly asbestos. While it is clear that everyone in the population carries a numerically substantial burden of fibers, and that persons with occupational exposure to asbestos carry considerably more, relatively few data exist on fiber persistence; there is a notable lack of information on rates of fiber accumulation and clearance. This review summarizes the data available from studies of human lungs; most of the review is directed to asbestos fibers since, apart from documentation of their presence, no information is available concerning accumulation or clearance of other fiber types.

Asbestos Fiber Burden in Relation to Fiber Type

Perhaps the most striking finding from the published studies on human lungs has been the consistent observation that, compared to chrysotile, amphiboles of all types preferentially accumulate in pulmonary tissue. This finding was foreshadowed by some of the original work of Wagner et al. (*1*), who demonstrated that, in rats exposed to asbestos, continuous exposure to amphiboles produced a continuous linear increase in recoverable amphibole fibers, whereas exposure to chrysotile showed only a small initial increase followed by a plateau. The issue of persistence in animal models is discussed at length elsewhere in these proceedings.

Table 1 lists published studies that supply information on the relative proportion of chrysotile and amphiboles found in lung samples (*2–16*). The preparative methods, types of electron microscope, and counting rules used in studies differ markedly, and these differences undoubtedly affect the results. Nonetheless, Table 1 shows that the proportion of amphibole present in the lung is markedly greater and the proportion of chrysotile markedly less than would have been present in the original inhaled dust. This is true of populations exposed only to ambient air, populations exposed largely to chrysotile, and populations with mixed chrysotile and amphibole exposure.

Because there is little exact information on the relative proportion of amphiboles and chrysotile in most exposure situations, it is difficult to estimate, on a fiber for fiber basis, the differences in accumulation of amphibole compared to chrysotile. However, some idea of the magnitude of this effect can be gleaned from the fact that, in the Thetford Mines region of Quebec, tremolite fibers make up at most a few percent of the chrysotile ore (*17*), but all of the studies of the lungs of chrysotile miners and millers from this area show an overwhelming preponderance (of the order of 75%, see Table 1) of tremolite. This is also true of miners and millers from the region of Asbestos, Quebec, a location in which the proportion of tremolite in the ore is even less than that of Thetford (*8,17*) (Table 1).

It is clear from these studies that the lung is an extraordinarily sensitive detection and accumulation system for amphibole fibers. Given the known much greater mesothelial carcinogenicity in man of amphiboles compared to chrysotile (*18*), preferential retention of amphiboles is extremely important in explaining the incidence of mesothelioma in exposed working populations, particularly when amphibole exposure has been fairly small.

Fiber Burden in Relation to Measured Exposures

The question of how preferential accumulation of amphiboles occurs is still unresolved. Two general possibilities exist: either there is much greater relative deposition of amphiboles compared to chrysotile, or chrysotile and amphiboles are deposited to the same extent but chrysotile is more rapidly cleared.

Available animal data (*19–23*) and human data appear to support the latter proposition, although the human data are at best fragmentary. Timbrell (*24*) suggested that amphibole fibers, because of

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their straight shape, are carried deep into the lung parenchyma, but the curled fibers of chrysotile tend to impact in the airways. Studies of the lungs of chrysotile miners and millers tend to refute this suggestion, since they demonstrate accumulation of both long and short chrysotile fibers in the distal parenchyma under the pleura (*25,26*). However, no information is available on what relative proportion of the inhaled dust reached the parenchyma.

Investigations of measured exposure and fiber burdens (Table 2) generally show a significant correlation between exposure and lung levels of amphiboles (*27,28*); but this is not always true, particularly when exposures have been relatively short and remote [(*29*); Churg, unpublished]. Most such investigations do not show a correlation between exposure and chrysotile concentration, and this is true even when detailed cumulative exposure data are available. An example of this phenomenon is derived from a large group of chrysotile miners and millers analyzed in our laboratory (Figure 1). Essentially identical results have been published by Sébastien et al. (*26*).

Table 1. Relative concentration of amphibole and chrysotile asbestos in various studies. Values as % of total asbestos fibers[a].

Report[b]	Total amphibole, %	Chrysotile, %	Amosite/crocidolite, %	Tremolite, %	Chrysotile, %	Comment %
General population: no special asbestos exposure						
San Francisco; *n* = 21 (*2*)	17	83	0.5	17	83	Mean values; all fibers >0.5 µm
Vancouver; *n* = 20 (*3*)	50	50	0	50	50	Median values; all fibers >0.5 µm
Charleston, SC; *n* = 58 (*4*)	2	98		2	98	
Sweden-site not detailed; *n* = 92 (*5*)	10	90	10		90	
Accident Victims–Canada age 61+; *n* = 14 (*6*)	28	72	12	16	72	>0.5 µm
East London; *n* = 56 (*7*)	17	83	17		83	
Rural population Eastern Quebec; *n* = 19 (*8*)	69	31	46	23	31	Fibers >0.5 µm geometric mean values
Mesothelioma cases without asbestos exposure in Great Britain; *n* = 21 (*9*)	24	76	24		76	
General population: chrysotile mining townships						
Town of Asbestos, Quebec; *n* = 22 (*8*)	51	49	37	14	49	Fibers >0.5 µm geometric mean values
Thetford Mines, Quebec; *n* = 7 (*10*)	50	50	0	50	50	Median values, fibers >0.5 µm
Thetford Mines, Quebec; *n* = 19 (*8*)	72	28	38	34	28	Fibers >0.5 µm geometric mean values
Workers in industries using predominantly chrysotile						
Textiles, Charleston, SC; *n* = 55 (*4*)	20	80		20	80	
Textiles, Charleston, SC; *n* = 72 (*11*)	37	63		37	63	Fibers >0.5 µm
Textiles, Rochdale; *n* = 24 (*12*)	61	39	51	10	39	
Asbestos cement workers; *n* = 74 (*5*)	25	75		25	75	
Mining and Milling, Town of Asbestos; *n* = 26 (*8*)	74	26	7	67	26	Fibers >0.5 µm
Mining and Milling,Thetford; *n* = 89 (*11*)	78	22		78	22	Fibers >0.5 µm
Mining and Milling, Thetford; *n* = 94 (this report)	81	19	0.5	81	19	Fibers >0.5 µm
Mining and Milling, Cyprus; *n* = 2 (*13*)	47	53	0.3	47	53	
Workers with mixed chrysotile–amphibole exposure						
Miscellaneous exposure; *n* = 110 (*14*)	94	6	89	5	6	Scanning EM
Factory workers, East London; *n* = 36 (*7*)	86	14	86		14	
Shipyard workers; *n* = 8 (*15*)	28	72	28	72		
Miscellaneous exposures; *n* = 53 (*16*)	11	89	9	2	89	Fibers >1 µm, mean values
Workers with mesothelioma; *n* = 16 (*9*)	24	75	24		76	
Shipyard workers/insulators; *n* = 161 (this article)	82	18	77	5	18	

[a] Left two columns show breakdown for all amphiboles versus chrysotile; right three columns show breakdown for different types of amphibole versus chrysotile. [b] Numbers in parentheses indicate references.

Table 2. Studies examining the relationship between measured exposure and fiber burden.

Report	Amosite/crocidolite	Tremolite	Chrysotile	Exposure measured as
Chrysotile textile workers, Rochdale (*12*)[a]	Yes	Yes	No	Years of exposure
Chrysotile textile workers, Charleston, SC (*4*)		Yes	Yes	Cumulative exposure
Chrysotile textile, workers, Charleston, SC (*11*)[a]		Yes	No	Years of exposure
Chrysotile textile workers, Charleston, SC (*11*)[a]		Yes	Yes	Intensity index
Chrysotile miners and millers, Thetford (*26*)		Yes	No	Cumulative exposure
Chrysotile miners and millers, Thetford (*11*)[a]		No	No	Years of exposure
Chrysotile miners and millers, Thetford (*11*)[a]		No	No	Intensity index
Chrysotile miners and millers, Thetford (this article)		No	No	Years of exposure
Chrysotile cement workers (*27*)	Yes	Yes	No	Years of exposure
Amosite processing plant workers (*28*)	Yes			
Crocidolite gas mask factory workers (*29*)	No			
Mixed exposure factory workers (*7*)	Yes		No	Exposure index
Shipyard and insulators (this article)	No	No	No	Years of exposure

[a] Calculations by A. Churg from the published data. [b] Numbers in parentheses indicate references. [c] Yes indicates statistically significant correlation; no indicates no correlation.

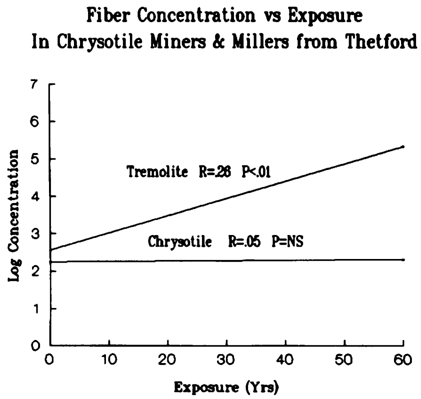

Figure 1. Fiber concentration versus exposure in 94 chrysotile miners and millers. Only tremolite shows a significant correlation.

However, some reports do demonstrate lung chrysotile burdens that are proportional to exposure (*4,11*), providing evidence against the idea of deposition failure.

Some studies do find a correlation between measured exposure and chrysotile burden, which raises the question of whether different fiber-related parameters are actually being measured. The most obvious of these is fiber size, since experimental data suggest that long fibers are more persistent than short fibers (*19,20,30*). However, we were unable to demonstrate correlations of exposure and fiber burden for any particular size of chrysotile fiber found in the lungs of chrysotile miners and millers, but burdens of both long and short fibers of tremolite produced equally good correlations with exposure (Churg, unpublished). Sébastien et al. (*26*), who counted only long (>5 µm) chrysotile fibers, were similarly unable to show such correlations in the lungs of Thetford miners and millers.

Another possibility is that fiber accumulation patterns are quite different in different industrial settings. In this regard it is striking that the two studies demonstrating an exposure correlation for chrysotile (Table 2) both come from a group of textile workers, suggesting that there may be something special about the fibers to which these workers are exposed. This suggestion has also been raised in regard to the high incidence of carcinoma in the same population (*11*).

Fiber Burden in Relation to Time Since Last Exposure

Only a few studies have examined the relationship between fiber burden and time since last exposure, and there is a major problem in using these reports to estimate clearance half-times, since absolute fiber concentrations for any specimen are known to vary markedly from laboratory to laboratory (*31*). Nonetheless, these studies are consistent in that, where rates can be estimated, the clearance half-times for amphiboles are generally fairly long, ranging from years to decades (Table 3; Figure 2).

Table 3. Studies examining the relationship between time since last exposure and fiber load.

Report	Amosite/crocidolite[c]	Tremolite[c]	Chrysotile[c]
Chrysotile textile workers, Rochdale (*12*)[a]	6	9	No
Chrysotile miners and millers, Thetford (Varying exposure levels, fibers >5 µm) (*26*)		7–110	6–48
Chrysotile miners and millers, Thetford (this article)		No	No
Crocidolite miners, South Africa (*33*)	6		
Shipyard and insulators (this article)	20 years	No	No

[a] Calculations by A. Churg from the published data. [b] Numbers in parentheses indicate references. [c] Number indicates calculated clearance half-time in years; No, no correlation.

With one exception, none of the data in Table 3 show a correlation between chrysotile concentration and time since last exposure. The exception is the calculation provided by Sébastien et al. (*26*), who estimated clearance half-times of up to 48 years for chrysotile. These data are hard to interpret (as the authors note), since their clearance half-times decrease with increasing exposure. Moreover, only fibers longer than 5 µm were counted, as opposed to most reports which include much shorter, and presumably more rapidly cleared, fibers.

We have also approached this problem by examining the tremolite:chrysotile ratios in small groups of chrysotile miners and millers whose time since last exposure was recent, intermediate, or remote, and in a larger series of 94 chrysotile miners (*32*) (Figure 3; Table 4). No differences were found in the ratio over time. The data of Sébastien et al. (*26*) and those of Wagner et al. (*7*) also show no trend toward increasing tremolite:chrysotile ratios with increasing time since last exposure.

Most studies fail to find a correlation between chrysotile burden and time since last exposure, for which the measurements are generally in years, and the tremolite:chrysotile ratio does not change with time since last exposure. These facts suggest that preferential chrysotile clearance must occur and be completed very rapidly, probably within weeks to months of exposure, because were preferential clearance a slow process, the tremolite: chrysotile ratios should increase with time since last exposure, and clearance half-times should be readily apparent. The long

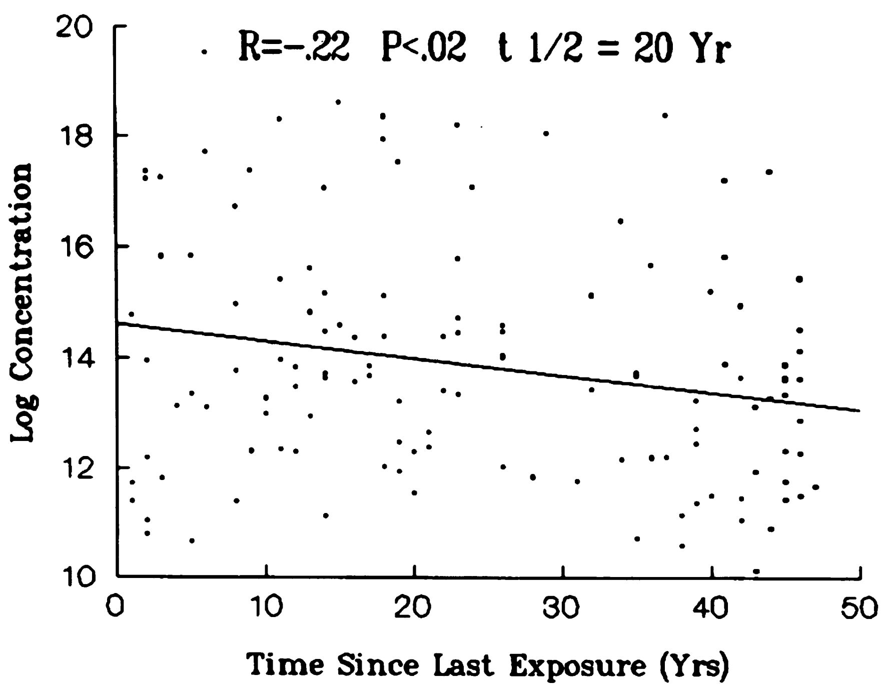

Figure 2. Fiber concentration versus time since last exposure in 161 shipyard and insulation workers. A significant negative correlation is found.

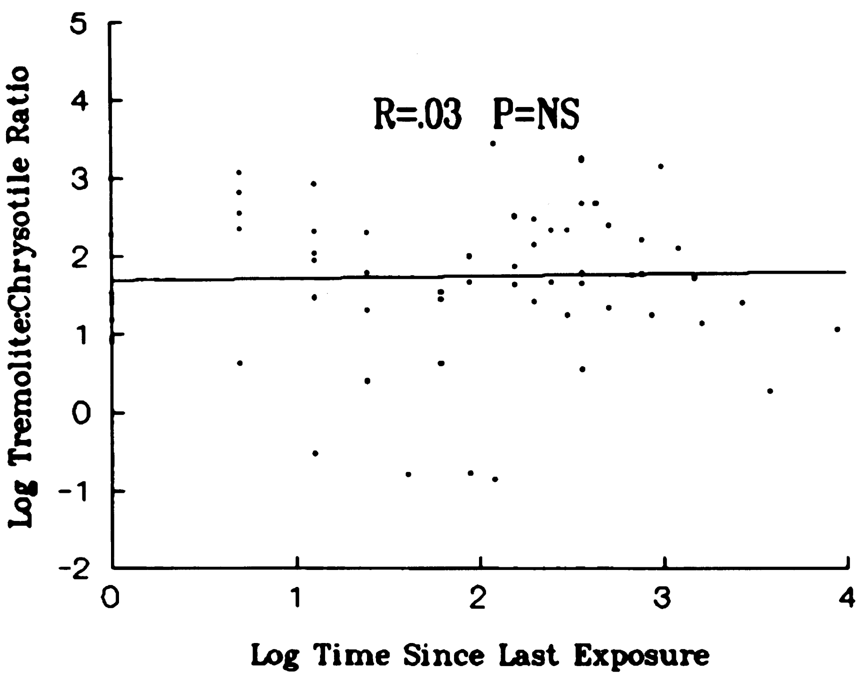

Figure 3. Tremolite:chrysotile ratio versus time since last exposure in 94 chrysotile miners and millers from Thetford. The ratio does not change over time.

Table 4. Chrysotile:tremolite concentration ratio and chrysotile composition in the lungs of Thetford miners and millers.[a]

Years from last exposure	Median C:T Ratio	Chrysotile composition Mg:Si Ratio (no. of fibers)
		1.2 (100–Native fiber)
<2	0.22	1.08 (880)
12–15	0.11	0.94 (537)
22–25	0.15	0.95 (397)

[a]Data taken from Churg and dePaoli (*32*).

Table 5. Types of naturally occurring nonasbestos fibers reported in human lungs.[a]

Erionite
Talc
Attapulgite
Silica
Rutile (Titanium)
Kaolinite
Mica
Feldspars
Vermiculite
Mullite
Calcium sulfate

[a]Data is taken from references (*6,7,34–38*).

Table 6. Nonasbestos fibers as a proportion of all fibers in human lungs.

Report	Mean percent of total
General population, San Francisco (*2*)[a]	50
Rural population, Texas (*37*)	26
Town dwellers, asbestos, Quebec (*8*)	56
Rural dwellers, Quebec (*8*)	40
Miner & Millers, asbestos, Quebec (*8*)	20
General population, London (*7*)	68

[a]Numbers in parentheses indicate references.

chrysotile clearance half-times calculated by Sébastien et al. (*26*) might represent values for a sequestered (interstitial?) fiber pool that is removed very slowly from the lung, and for which clearance rates are similar to those for tremolite.

Mechanisms of Chrysotile Fiber Removal

Much energy has been put into investigating the proposition that chrysotile is rapidly removed from lung because of magnesium leaching and eventual dissolution of the fiber. Although leaching of chrysotile fibers can be demonstrated easily in a test tube, there are few data on fibers recovered from human lungs, and these are contradictory. Langer et al. (*34*) suggested that leaching is indeed present, but only about 25% of their fibers showed leaching and the amount of magnesium loss appeared small. Sébastien et al. (*26*) found as much as 50% leaching in a few fibers, but most fibers did not show much magnesium loss. Jaurand et al. (*35*) reported marked leaching in fibers recovered from different lungs; indeed, some fibers were reported as having no magnesium at all. By contrast, we found an average of about 20% magnesium leaching after 20 years from last exposure (Table 4). To us the data on leaching are not definitive, and rapid fragmentation of the relatively fragile chrysotile fibers into short pieces readily removed by macrophages may also occur. Certainly we found this to be true in an intratracheal instillation model where there was rapid removal of chrysotile and essentially no leaching at all (*21*).

Other Naturally Occurring Fibers in Human Lungs

Many reports have documented the occurrence of nonasbestos, naturally occurring mineral fibers in human autopsy lungs (*34–38*) (Tables 5,6). In some instances they account for a very large fraction of the fiber burden in the lung. For example, Wagner et al. (*7*) noted that mullite fibers constituted 68% of the total fiber number in their control lungs. While these data suggest that nonasbestos fibers probably persist in lung tissue, there is no actual information on this point, and, except for the well documented mesothelial carcinogenicity of erionite, the pathogenic effects of these fibers, if any, are also unknown.

REFERENCES

1. Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. Br J Cancer 29:252–269 (1974).
2. Churg A, Warnock ML. Asbestos fibres in the general population. Am Rev Respir Dis 122:669–678 (1980).
3. Churg A, Wiggs B. Fiber size and number in users of processed chrysotile ore, chrysotile miners, and members of the general population. Am J Ind Med 9:143–152 (1986).
4. Green FHY, Harley R, Vallyathan V, Dement J, Pooley F, Althouse R. Pulmonary fibrosis and asbestos exposure in chrysotile asbestos textile workers: preliminary results. In: Biological Effects of Chrysotile (Wagner JC, ed). Philadelphia:Lippincott, 1986; 59–68.
5. Pooley FD, Mitha R. Fiber types, concentrations, and characteristics found in lung tissues of chrysotile-exposed cases and controls. In: Biological Effects of Chrysotile (Wagner JC, ed). Philadelphia: Lippincott, 1986;1–11.
6. Case BW, Sébastien P, McDonald JC. Lung fiber analysis in accident victims: A biological assessment of general environmental exposure. Arch Environ Health 43:178–179 (1988).
7. Wagner JC, Newhouse ML, Corrin B, Rossiter CER, Griffiths DM. Correlation between fibre content of the lung and disease in East London asbestos factory workers. Br J Ind Med 45:305–308 (1988).
8. Case BW, Sébastien P. Fibre levels in lung and correlation with air samples. In: Non-occupational Exposure to Mineral Fibres (Bignon J, Peto J, Saracci R, eds), IARC Scientific Publications No. 90. Lyon:International Agency for Research on Cancer, 1989;207–219.
9. Gibbs AR, Jones JSP, Pooley FD, Griffiths DM, Wagner JC. Non-occupational malignant mesothelioma. In: Non-occupational Exposure to Mineral Fibres (Bignon J, Peto J, Saracci R, eds), IARC Scientific Publications No. 90. Lyon:International Agency for Research on Cancer, 1989;219–228.
10. Churg A. Lung asbestos content in long-term residents of a chrysotile mining town. Am Rev Respir Dis 134:125–127 (1986).
11. Sébastien P, McDonald JC, McDonald AD, Case B, Harley R. Respiratory cancer in chrysotile textile and mining industries: exposure inferences from lung analysis. Br J Ind Med 46:180–187 (1989).
12. Wagner JC, Berry G, Pooley FD. Mesotheliomas and asbestos type in asbestos textile workers: a study of lung contents. Br Med J, 285:603–606 (1982).
13. McConnochie K, Simonato L, Mavrides P, Christofides P, Mitha R, Griffiths DM, Wagner JC. Mesothelioma in Cyprus. In: Non-occupational Exposure to Mineral Fibres (Bignon J, Peto J, Saracci R, eds). IARC Scientific Publications No. 90, Lyon:International Agency for Research on Cancer, 1989;411–419.
14. Roggli VL, Pratt PC, Brody AR. Asbestos content of lung tissue in asbestos associated disease: A study of 110 cases. Br J Ind Med 43:18–29 (1986).
15. Dodson RF, Williams MG, Corn CJ, Brollo A, Bianchi C. Asbestos content of lung tissue, lymph nodes, and pleural plaques from former shipyard workers. Am Rev Respir Dis 142:843–847 (1990).
16. Langer AM, Nolan RP. Fibre type and burden in parenchymal tissues of workers occupationally exposed to asbestos in the United States. In: Non-occupational Exposure to Mineral Fibres (Bignon J, Peto J, Saracci R, eds). IARC Scientific Publications No. 90, Lyon:International Agency for Research on Cancer, 1989;330–335.
17. Sébastien P, Plourde M, Robb R, et al. Ambient air asbestos survey in Quebec mining towns. Part 2–Main study. Environment Canada Report 5/AP/RQ-2E, 1986.
18. McDonald JC, McDonald AD. Epidemiology of malignant mesothelioma. In: Asbestos-related Malignancy (Antman K, Aisner J, eds). New York:Grune and Stratton, 1986;31–35.
19. Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Smith T. The pathogenicity of long versus short fibre samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. Br J Exp Path 67:415–430 (1986).
20. Davis JMG, Jones AD. Comparisons of the pathogenicity of long and short fibre chrysotile asbestos in rats. Br J Exp Path 69:717–737 (1986).
21. Churg A, Wright JL, Gilks B, DePaoli L. Rapid short term clearance of chrysotile compared to amosite asbestos in the guinea pig. Am Rev Respir Dis 139:885–890 (1989).
22. Roggli VL, Brody AR. Changes in numbers and dimensions of chrysotile asbestos fibers in lungs of rats following short-term exposure. Exp Lung Res 7:133–147 (1984).
23. Roggli VL, George MH, Brody AR. Clearance and dimensional changes of crocidolite asbestos fiber isolated from lungs of rats following short-term exposure. Environ Res 42:94–105 (1987).
24. Timbrell V. Deposition and retention of fibres in the human lung. Ann Occup Hyg 26:347–369 (1982).
25. Churg A, DePaoli L, Kempe B, Stevens B. Lung asbestos content in chrysotile workers with mesothelioma. Am Rev Respir Dis 130:1042–1045 (1984).
26. Sébastien P, Begin R, Case BW, McDonald JC. Inhalation of chrysotile dust. In: Biological Effects of Chrysotile (Wagner JC, ed). Philadelphia:Lippincott, 1986;19–29.
27. Albin M, Johansson L, Pooley FD, Jakobsson K, Attrewell R, Mitha R. Mineral fibres, fibrosis, and asbestos bodies in lung tissue from deceased asbestos cement workers. Br J Ind Med 47: 767–774 (1990).
28. Dodson RF, Williams MG, O'Sullivan MF, Corn CJ, Greenberg SD, Hurst EA. A comparison of the ferruginous body and uncoated fiber content in the lungs of former asbestos workers. Am Rev Resp Dis 132: 143–147 (1985).
29. Jones JSP, Smith PG, Pooley FD, Berry G, Sawles EW, Maderley RJ, Wignall BK, Aggarwal A. The consequences of exposure to asbestos dust in a wartime gas-mask factory. In: Biological Effects of Mineral Fibres (Wagner JC, ed). IARC Scientific Publications No. 30, Lyon:International Agency for Research on Cancer, 1980; 637–653.
30. Morgan A. Effect of length on the clearance of fibre from the lung and on body formation. In: Biological Effects of Mineral Fibres (Wagner JC, ed), IARC Scientific Publications No. 30, Lyon: International Agency for Research on Cancer, 1980; 329–335.
31. Gylseth B, Churg A, Davis JMG, Johnson N, Morgan A, Mowe G, Rogers A, Roggli V. Analysis of asbestos fibers and asbestos bodies in human lung tissue samples. An international laboratory trial. Scand J Work Environ Health 11:107–110 (1985).
32. Churg A, DePaoli L. Clearance of chrysotile from human lung. Exp Lung Res 14:567–574 (1988).
33. Du Toit RSJ. An estimate of the rate at which crocidolite asbestos fibres are cleared from the lung. Ann Occup Hyg 35: 433–438 (1991).
34. Langer AM, Rubin IB, Selikoff IJ. Chemical characterization of asbestos body cores by electron microprobe analysis. J Histochem Cytochem 20:723–734 (1972).
35. Jaurand MC, Bignon J, Sébastien P, Goni J. Leaching of chrysotile asbestos in human lungs: Correlation with in vitro studies using rabbit alveolar macrophages. Environ Res 14:245–254 (1977).
36. Churg A. Nonasbestos pulmonary mineral fibers in the general population. Environ Res 31:189–200 (1983).
37. Dodson RF, Williams MG, Corn CJ, Rankin RL. A comparison of asbestos burden in non-urban patients with and without lung cancer. Cytobios 56:7–15 (1988).
38. Sébastien P, Gaudichet A, Bignon J, Baris YI. Zeolite bodies in human lungs from Turkey. Lab Invest 44:420–426 (1981).