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Asbestos bodies in bronchoalveolar lavage in relation to asbestos bodies and asbestos fibres in lung parenchyma

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ABSTRACT: In Finland, unlike other countries, anthophyllite asbestos has been widely used due to its domestic production in 1918–1975. In this particular context, the aim of the present study was to analyse the relationship between asbestos bodies (ABs) in bronchoalveolar lavage (BAL) fluid and the concentration of ABs and the different amphibole asbestos fibres in lung tissue.

Sixty five BAL lung tissue sample pairs from patients with pulmonary disease were analysed. The concentration of ABs in BAL fluid and lung tissue was determined with optical microscopy, and the concentration, type and dimensions of asbestos fibres in lung tissue with scanning electron microscopy.

There was a significant correlation between the concentrations of ABs in BAL fluid and in lung tissue ($r=0.72$; $p<0.001$), between the concentrations of ABs and amphibole asbestos fibres in lung tissue ($r=0.73$; $p<0.001$), and between the concentration of ABs in BAL fluid and the concentration of amphibole asbestos fibres in lung tissue ($r=0.64$; $p<0.001$). In patients who had been exposed mainly to commercial anthophyllite, significantly higher concentrations of ABs were observed per total pulmonary amphibole fibre burden, as compared to patients whose main exposure was to crocidolite/amosite. The anthophyllite fibres in lung tissue were longer than the crocidolite/amosite fibres.

The relationship between asbestos body counts in lung tissue and in bronchoalveolar lavage fluid was similar to previous international observations. When using the asbestos body count to predict the underlying total pulmonary amphibole asbestos burden in Finnish patients, however, it should be borne in mind that the relationship between the two parameters seems to be different with anthophyllite as compared to crocidolite/amosite fibres.

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Occupational exposure to asbestos has been common in most industrialized countries during the past decades. In Finland, the use of asbestos peaked in the early 1970s [1]. Due to the long latent period of asbestos-related diseases, their incidence will continue to increase in most countries during the next 10–20 years. According to a recent analysis, the maximum incidence may be higher than previously predicted [2]. The lung cancer and mesothelioma of asbestos-exposed workers, thus, constitute a growing medicolegal problem. Accurate exposure assessment is crucial in these cases. The risk estimates of lung cancer and mesothelioma associated with increased pulmonary concentrations of asbestos fibres as well as reference fibre concentrations observed in the general population, based on electron microscopy, have recently been published in Finland [3–5].

In clinical routine, the intensity of asbestos exposure is often characterized by counting asbestos bodies (ABs) with optical microscopy in bronchoalveolar lavage (BAL) fluid samples. ABs are asbestos fibres that have been coated with ferroprotein by macrophages in the lung tissue [6, 7]. Their occurrence in the lungs is closely associated with the concentration of amphibole asbestos fibres [8], and their concentration in BAL fluid reflects their concentration in lung parenchyma [9, 10]. In Finland, the use of amphibole asbestos has been common due to the domestic production of anthophyllite asbestos. Approximately 40% of all asbestos used in Finland consists of anthophyllite [1]. ABs are formed preferably on long fibres [6]. As the distribution of fibre lengths differs between anthophyllite and other amphibole fibres [11, 12], it is probable that there is also a difference in the coating rate between these fibre types.

The aim of this study was to analyse the relationship between the concentration of ABs in BAL and lung parenchyma, the relationship between the concentrations of ABs and different amphibole asbestos fibres in lung parenchyma, and the value of BAL AB counting in the estimation of the pulmonary asbestos burden.

Materials and methods

Subjects

All individuals with both a BAL sample and a lung tissue sample analysed in 1992–1994 in the Laboratory of Pathology of the Finnish Institute of Occupational Health were included in the study. Altogether, 65 sample pairs were identified when the BAL and lung tissue analysis files of the laboratory were compared. The mean age of the subjects was 65 (range 44–85) yrs. There were 59 patients with lung cancer, one with mesothelioma, one with lymphoma, one with pneumonia, and three with benign pleural disease. All the subjects had been interviewed personally for their complete work history and asbestos exposure during an ongoing BAL study. The occupational histories were classified into four exposure categories [13]: 1) Heavy exposure - at least 1 yr of exposure in insulation work or in the manufacture of asbestos products, or at least 10 yrs of exposure in shipyard or construction work with definite or probable intermittent exposure to asbestos; 2) Moderate exposure - less than one year of exposure in insulation work or in the manufacture of asbestos products, or 1–10 yrs of exposure in shipyard or construction work with definite or probable intermittent exposure to asbestos, or more than 10 yrs of exposure in garage work involving brake repair; 3) Possible exposure - persons employed in other industrial occupations; and 4) Unlikely exposure - persons with lifelong employment in occupations with no known exposure to asbestos, *e.g.* office work and farming.

Forty five (69%) of the subjects studied were classified as heavily or moderately exposed, 15 as possibly exposed, and five as unlikely exposed. The high percentage of exposed individuals follows from the fact that most of the lung tissue samples (39 samples) were collected at necropsies performed due to suspicion of an asbestos-related death. The rest of the samples were collected from a series of surgically treated lung cancer patients without any selection as to exposure history.

BAL sampling and AB counting in BAL samples

BAL fluid samples were collected from patients who underwent diagnostic bronchoscopy for a suspected pulmonary malignancy at the Department of Pulmonary Medicine of the Helsinki University Central Hospital or at Laakso Pulmonary Hospital, Helsinki. The study protocol was approved by the Local Ethics Committee, and informed consent was obtained from the patients. BAL was performed with a fibreoptic bronchofibreoscope under local anaesthesia. A segment of the right middle lobe of the lung was washed with 10× 20 mL aliquots of saline solution as described in detail previously [14]. In case of an intrabronchial tumour in the right middle lobe, the lingula was washed.

The fresh lavage fluid was placed on ice in a glass container. No filtration was performed to trap mucinous contamination. For the determination of ABs, 40 mL of each original well-mixed sample was fixed in 20 mL of 96% ethanol. A sample containing 10 mL of BAL fluid was filtered through a 5 µm Millipore filter, using slight negative pressure at the end of the filtration. The filter was stained with an iron-staining Prussian blue dye, mounted on a glass slide and covered with a coverslip. ABs were identified according to previously described criteria [6], using an optical microscope and 200 fold magnification. The number of ABs was expressed per mL of BAL fluid. The variation in AB concentration between consecutive 10 mL aliquots of each BAL fluid sample was insignificant.

Lung tissue sampling

Lung tissue samples were collected during surgery (26 samples) or necropsy (39 samples). The delay between BAL fluid and lung tissue sampling ranged from 7 days to 34 months (mean 4.5 months). The tissue pieces for AB counting and electron microscopic fibre analysis were taken from the peripheral part of the lung, not including pleural or tumour tissue. In case of surgical bilobectomy or pulmectomy, the sample was taken from the lobe which appeared to be closest to normal. The necropsy samples were taken, if possible, from the left upper lobe.

AB counting in lung tissue samples

About 1 g of formalin-fixed lung tissue was dried at 60°C overnight, weighed, and digested with 30 mL of 5% potassium hydroxide overnight at 70°C in 50 mL disposable polypropylene tubes. The sample was then centrifuged at 1,800×g for 20 min. The sediment was washed three times (distilled water, 0.5 N HCl, and distilled water). The sample was then resuspended in 10 mL of distilled water in a mild ultrasound bath (1 min). AB concentration was determined according to a modified cytocentrifuge method of EHRLICH and SUZUKI [15]. Altogether 600–900 µL of the suspension were placed in a cytocentrifuge (Cytospin 3) and centrifuged on three glass slides (200–300 µL each) at 1,040 rpm (202×g) for 10 min. The glass slides were air-dried and covered with a coverslip. ABs were counted by phase contrast microscopy at 400 fold magnification. All three slides per sample were thoroughly examined. The results are presented as number of ABs per gram of dry weight.

Electron microscopic fibre analysis of lung tissue samples

A tissue piece of about 100 mg wet weight was taken for the fibre analysis. Organic tissue was removed by low temperature ashing. Fibres were detected with a JEOL 100 CX-ASID4D electron microscope in scanning electron microscope (SEM) mode at a 5,000 fold magnification [4]. A length to width ratio greater than three and roughly parallel sides was used as a fibre criterion. Fibres longer than 1 µm could be detected. A minimum of 400 viewing fields were evaluated to find at least 4–30 fibres per sample, depending on the density. An analytical sensitivity (one fibre per sample) of about 0.1 million fibres per gram ($f \cdot g^{-1}$) of dry tissue could be achieved.

An energy dispersive X-ray microanalyser (Tracor TN 5500) was used to determine the fibre type by comparing peak ratios to standard spectra. Amosite and crocidolite have very similar X-ray spectra and are poorly

distinguishable. They are, therefore, not presented separately. In a previous Finnish study, crocidolite fibres accounted for the great majority of amosite/crocidolite fibres identified with transmission electron microscope (TEM) [16]. Chrysotile fibres are poorly detected with SEM and, consequently, the results represent the concentration of amphibole fibres. Chrysotile fibres were observed in four samples and tremolite fibres in four samples.

Fibre dimensions were measured directly on the screen in, altogether, 100 anthophyllite and 100 crocidolite/amosite fibres identified in 12 samples. Magnifications of up to ×100,000 were used.

Statistical methods

The distributions of concentrations of ABs in lung parenchyma and BAL and the distribution of concentrations of asbestos fibres in lung parenchyma were approximately log normal. Log transformed concentrations were, therefore, used in the linear regression analyses, and the correlation between the log transformed concentrations was calculated. In nine of the BAL samples and one of the lung tissue samples no ABs were found, and in one of the lung tissue samples no amphibole asbestos fibres were found. For data analysis, concentrations of 0.05 AB·mL^{-1}, 20 AB·g^{-1}, and 0.05 million f·g^{-1}, respectively, were used for these data points.

Results

ABs in BAL and lung tissue

ABs were detected in 86% of the BAL samples and 98% of the lung tissue samples. Their concentrations varied from <0.1 to 800 AB·mL^{-1} in BAL and from <40 to 890,000 AB·g^{-1} in lung tissue. The correlation between the concentrations of ABs in BAL and lung tissue is illustrated in figure 1. The simple regression equation log(AB·g^{-1} dry lung) = 3.39 + 0.590·log(AB·mL^{-1} in BAL) (r=0.72; p<0.001) was found between these two concentrations.

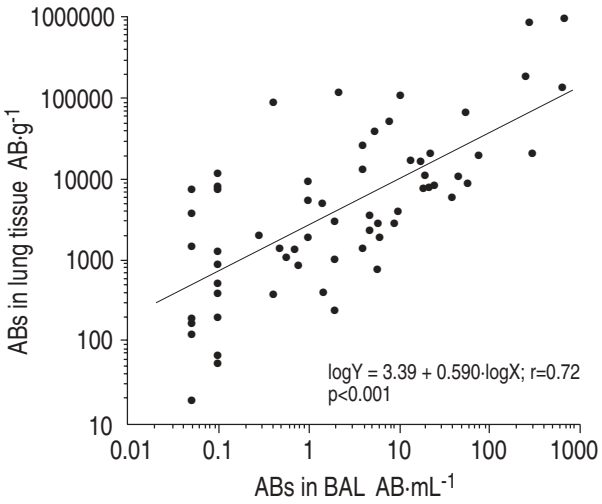

Fig. 1. – Relationship between asbestos body (AB) counts in bronchoalveolar lavage (BAL) fluid and lung tissue in 65 Finnish patients.

Asbestos fibres in lung tissue

Amphibole asbestos fibres were detected in all but one of the 65 lung tissue samples. Their concentrations ranged from <0.1 to 740 million f·g^{-1}. Most of the lung tissue samples contained both anthophyllite and crocidolite/amosite fibres. The concentrations of anthophyllite fibres ranged from <0.1 to 230 million f·g^{-1}, and the concentrations of crocidolite/amosite fibres from <0.1 to 740 million f·g^{-1}. The concentration of amphibole asbestos fibres correlated with the concentration of ABs in lung tissue (fig. 2) and BAL (fig. 3). When the concentrations of anthophyllite and crocidolite/amosite fibres were included in the same statistical model (multiple linear regression), both were found to correlate closely with the parenchymal concentration of ABs (p<0.001 for both fibre types). The coefficient of anthophyllite fibres in the regression equation was, however, higher than the coefficient of crocidolite/amosite fibres: log(AB) = 3.91 + 0.79·log(antho)

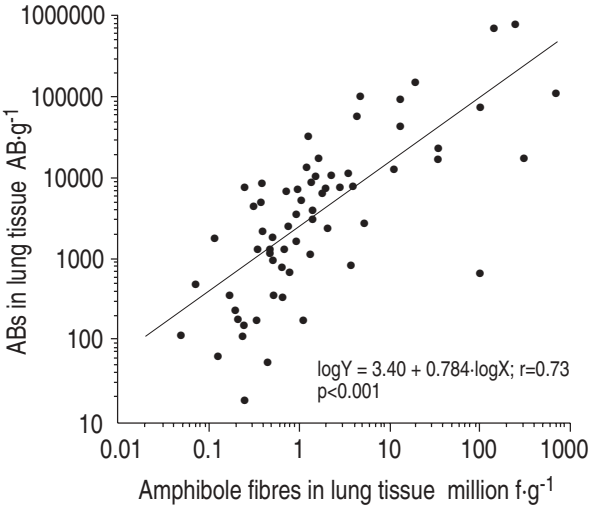

Fig. 2. – Relationship between concentration of asbestos bodies (ABs) and amphibole asbestos fibres in lung tissue samples of 65 Finnish patients.

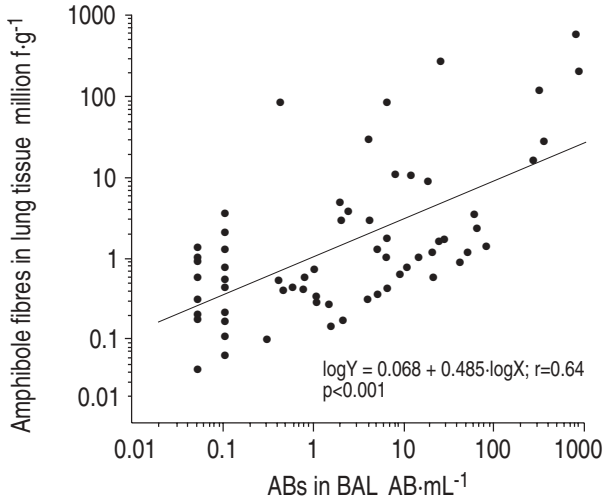

Fig. 3. – Relationship between concentration of asbestos bodies (ABs) in bronchoalveolar lavage (BAL) fluid and concentration of amphibole asbestos fibres in lung tissue in 65 Finnish patients.

Table 1. – Concentration of asbestos bodies (ABs) and amphibole asbestos fibres in lung tissue samples with predominant exposure (more than 80% of all amphibole fibres) to anthophyllite or crocidolite/amosite fibres

Case No.	ABs in lung tissue	Amphibole fibres in lung tissue	Proportion of ABs of amphibole fibres	Proportion of the predominant fibre type of all amphibole fibres	Main type of exposure
	AB·g^{-1}	million f·g^{-1}	%	%	
Predominant exposure to anthophyllite					
1	890000	253	0.35	91	Asbestos product plant
2	780000	146	0.53	>97	Asbestos product plant
3	170000	19.0	0.89	85	Asbestos product plant
4	105000	12.9	0.81	90	Construction
5	25000	34.8	0.072	86	Asbestos product plant
6	13000	3.5	0.37	93	Construction
7	8800	2.8	0.31	87	Construction
8	8400	4.1	0.20	85	Stevedore
Predominant exposure to crocidolite/amosite					
1	120000	739	0.016	>97	Asbestos sprayer
2	87000	100	0.087	>97	Shipyard
3	50000	13.1	0.38	84	Construction, shipyard
4	20000	335	0.006	>97	Shipyard
5	20000	35.0	0.057	89	Shipyard
6	15000	10.8	0.14	92	Construction
7	3100	5.6	0.055	86	Construction
8	2600	2.1	0.12	89	Power plant
9	730	102	0.0007	>97	Shipyard

+ 0.43·log(cro/amo), where AB refers to pulmonary concentration of asbestos bodies (AB·g^{-1}) and antho and cro/amo to pulmonary concentrations of anthophyllite and crocidolite/amosite fibres (million f·g^{-1}), respectively.

Table 1 presents the concentrations of ABs and amphibole asbestos fibres in the lung tissue samples of eight cases with anthophyllite as the main fibre type and 9 cases with crocidolite/amosite as the main fibre type. All the samples that contained at least 2 million f·g^{-1} and in which the predominant fibre type accounted for at least 80% of all fibres are included in table 1. In samples that contained mainly anthophyllite fibres, the number of ABs in lung tissue divided by the number of amphibole asbestos fibres in lung tissue was significantly higher (p=0.0043; t-test). In the samples that contained mainly anthophyllite fibres, the concentration of ABs in lung tissue (optical microscopy) was on the average 0.44% (range 0.07–0.89, SD=0.29) of the concentration of amphibole asbestos fibres in lung tissue (electron microscopy). In the samples that contained mainly crocidolite/amosite fibres the respective figure was 0.096% (range 0.007–0.38, SD=0.12), and in the four cases that contained only crocidolite/amosite fibres, even lower. The subjects with main exposure to anthophyllite, had been exposed either in an asbestos product plant processing the anthophyllite asbestos from Paakkila mine or in handling anthophyllite-containing pipe and boiler insulations in construction work or in stevedore work. The subjects exposed mainly to crocidolite/amosite fibres, had been exposed as an asbestos sprayer or as a bystander during asbestos spraying in shipyards, construction work, or power plants.

Anthophyllite fibres detected in the lung tissue samples were longer (median length 7.5 µm, range 1.6–91 µm) than crocidolite amosite fibres in the same samples (median length 3.2 µm, range 1.1–40 µm). The proportion of fibres longer than 10 µm was 45% in anthophyllite and 7.5% in crocidolite/amosite.

Distribution of parenchymal concentrations according to AB concentration in BAL fluid

Tables 2 and 3 present the distribution of AB and amphibole asbestos fibre concentrations in lung tissue by concentration of ABs in BAL fluid. Of the cases with less than 1.0 AB·mL^{-1} in BAL, 92% showed less than 10,000 AB·g^{-1} and 96% less than 10 million amphibole fibres per gram dry lung tissue. Of the cases with ≥10 AB·mL^{-1} in BAL, all had ≥1,000 AB·g^{-1} and 90% ≥1.0 million amphibole fibres·g^{-1} in lung tissue.

Table 2. – Comparison of AB in BAL fluid and lung tissue

AB·mL^{-1}	AB·g^{-1} dry lung tissue			
in BAL fluid	n	<1000	1000–9999	≥10000
<0.10	9	6 (67)	3 (33)	0 (0)
0.10–0.99	17	8 (47)	7 (41)	2 (12)
≥1.0	39	3 (8)	18 (46)	18 (46)
≥5.0	27	1 (4)	11 (41)	15 (56)
≥10.0	19	0 (0)	6 (32)	13 (68)

AB: asbestos body; BAL: bronchoalveolar lavage. Percentage values are presented in parentheses.

Table 3. – Comparison of AB concentrations in BAL fluid and amphibole asbestos concentrations in lung tissue

AB·mL^{-1}	Amphibole asbestos fibres million fibres·g^{-1} dry lung tissue			
in BAL fluid	n	<1.0	1.0–9.9	≥10.0
<0.10	9	6 (67)	3 (33)	0 (0)
0.10–0.99	17	13 (76)	3 (18)	1 (6)
≥1.0	39	12 (31)	16 (41)	11 (28)
≥5.0	27	5 (19)	12 (44)	10 (37)
≥10.0	19	2 (11)	9 (47)	8 (42)

AB: asbestos body; BAL: bronchoalveolar lavage. Percentage values are presented in parenthesis.

Discussion

The concentrations of ABs in BAL fluid and lung parenchyma were found to correlate with each other and with the concentration of amphibole asbestos fibres in lung parenchyma. Most of our patients were exposed both to anthophyllite and crocidolite/amosite fibres, and the concentration of ABs in lung parenchyma correlated with both the concentration of anthophyllite and crocidolite/amosite fibres. According to multiple linear regression analysis, however, higher AB concentrations would be expected for a given pulmonary anthophyllite content as compared to an identical pulmonary concentration of crocidolite/amosite fibres. A similar statistically significant difference was found when only cases with predominant exposure to one of the fibre types were considered.

The general objective of asbestos fibre measurement in biological samples is to provide clinicians or epidemiologists with better information on past asbestos exposure. When AB counting in BAL samples is used as an indicator of past cumulative asbestos exposure, the validity of the following two general assumptions is crucial: 1) the number of ABs in BAL fluid correlates with the number of ABs in lung parenchyma; and 2) the number of ABs in lung parenchyma correlates with the past cumulative asbestos exposure.

In three previous studies, the correlation between parenchymal and BAL AB concentrations has been analysed with optical microscopy [9, 10, 17]. Table 4 summarizes the results of two of these studies and our results. The regression equations predicting the underlying parenchymal concentration from the concentration of ABs in BAL fluid are very similar. The equations predict nearly identical parenchymal concentrations for concentrations ranging 1–10 AB·mL^{-1} in BAL. Despite the numerous methodological differences in sample preparation and AB counting, the similarity of the results is striking, and strongly supports the view that there is a well-established structural relationship between the concentrations of ABs in BAL and lung parenchyma. However, all three series contained only few cases with more than 100 AB·mL^{-1} in BAL or more than 100,000 AB·g^{-1} in lung parenchyma, and the consistency of the results in such high concentrations is not equal to that in moderate concentrations. In each of these studies, there has also been a wide variation in the ratio of BAL and parenchymal concentration readings in individual cases, and the correlation between the two concentrations is lower if only restricted concentration areas are analysed [17]. Part of this variation may arise from the regional variation of AB concentrations in the different anatomical sites of the lungs [9, 18].

Chrysotile fibres and short amphibole fibres are rarely coated [6, 7], and the concentration of ABs in BAL or lung parenchyma therefore reflects mainly the concentration of long amphibole fibres in lung parenchyma. Our results indicate that this relationship is different in persons exposed to anthophyllite as compared to persons exposed to crocidolite/amosite. The average number of ABs divided by the total amphibole asbestos concentration was about five times higher among those exposed mainly to anthophyllite as compared to those exposed mainly to crocidolite/amosite. This is to be borne in mind when the number of ABs in lung parenchyma or BAL is used as an estimate of the cumulative asbestos exposure in Finland. The observed difference in the coating of anthophyllite and crocidolite/amosite fibres is most probably due to the difference in fibre length between these fibre types. In lung parenchyma, fibres longer than 10 µm were about six times more frequent among anthophyllite than among crocidolite/amosite fibres. In this respect, our results apply only to the anthophyllite fibres detected in the lungs of Finnish patients who have been exposed to commercially exploited and industrially processed anthophyllite fibres. Such fibres are probably on average longer than anthophyllite fibres from environmental exposure or contamination of other minerals that sometimes occur in lung tissue samples of patients from countries where anthophyllite has not been in commercial use.

When the relationship between AB concentration and total fibre burden is interpreted, it must be emphasized that wide interlaboratory variation occurs in the determination of pulmonary fibre concentrations with electron microscopy [19].

Our results are consistent with previous reports in that a low or negative count of ABs in BAL does not rule out heavy exposure [9, 10, 17, 20]. This is best illustrated in two samples with a very high concentration of crocidolite/amosite fibres and only a relatively low concentration of ABs in lung tissue and BAL; 100 million f·g^{-1} of crocidolite/amosite, 730 AB·g^{-1}, and 6 AB·mL^{-1} were observed in the samples of a shipyard worker with mesothelioma, and concentrations of 330 million f·g^{-1} crocidolite/amosite, 20,000 AB·g^{-1}, and 23 AB·mL^{-1} in the samples of a shipyard worker with pulmonary adenocarcinoma and asbestosis. In these cases, the regression equations of figures 2 and 3 would have predicted a much lower amphibole fibre concentration in lung tissue. Figure 3 also shows a number of cases with an elevated parenchymal amphibole fibre concentration but with a BAL AB content at or below the detection limit. All but one of the six cases with ≥1 million amphibole f·g^{-1} in lung parenchyma and ≤0.1 AB·mL^{-1} in BAL, had more than 1,000 AB·g^{-1} in lung parenchyma. This probably

Table 4. – Correlation between parenchymal and BAL asbestos body (AB) concentrations (optical microscopy) in three international studies

Regression equation	Predicted parenchymal AB concentration (AB·g^{-1}) for 1, 10 and 100 AB·mL^{-1} in BAL			Cases n	[Ref]
	1 AB·mL^{-1}	10 AB·mL^{-1}	100 AB·mL^{-1}		
log(AB·g^{-1}) = 3.25 + 0.77 log(AB·mL^{-1}) (r=0.74)	1800	10500	62000	69	[10]
log(AB·g^{-1}) = 3.34 + 0.684 log(AB·mL^{-1}) (r=0.73)	2200	10600	51000	100	[9]
log(AB·g^{-1}) = 3.39 + 0.590 log(AB·mL^{-1}) (r=0.72)	2500	9500	37000	65	Present study

AB: asbestos body; BAL: bronchoalveolar lavage .

demonstrates that, in addition to cases with an unexpectedly low fraction of coated fibres, this cluster may contain cases with a poorly representative BAL sample.

Most of the ABs extracted from the human lungs are formed around amphibole fibres [6], and the pulmonary concentration of ABs correlates with the pulmonary amphibole concentration but not with the concentration of chrysotile fibres [8]. Elevated concentrations of ABs formed on chrysotile fibres can, however, be found in the lungs of workers with heavy exposure to chrysotile [21], but the concentration of chrysotile fibres in lung tissue or BAL is a poor indicator of cumulative chrysotile exposure [8, 20]. In Canadian chrysotile miners, tremolite fibres constitute only a small percentage of the airborne fibres in the mines, but have accounted for up to one half or more of the pulmonary asbestos fibres in the miners [22, 23]. We used scanning electron microscopy, which is known to underestimate the concentration of chrysotile fibres. Consequently, only one of the 65 lung tissue samples in our series contained chrysotile fibres as the main fibre type. This was from an Estonian immigrant who had worked as a pipe insulator in the former Soviet Union from 1970 to 1990. A concentration of 0.1 $AB \cdot mL^{-1}$ was detected in BAL in 1993 and 70 $AB \cdot g^{-1}$ and 2.6 million $f \cdot g^{-1}$ in lung tissue in 1994.

Due to the above-mentioned restrictions of bronchoalveolar lavage asbestos body counting, a complete chronological work history based on personal interview is often the best way to evaluate past exposure to asbestos. In cases with inconclusive or lacking work history, bronchoalveolar lavage asbestos body counting offers, however, a reasonably reliable estimate of the past exposure.

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