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Original Article

# Prevalence and Distribution of Asbestos Lung Residue in a Spanish Urban Population

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ABSTRACT

*Introduction:* The purpose of the present study is to analyse the prevalence and distribution of asbestos lung residue in the Barcelona urban population.
*Material and methods:* Lung autopsy samples were obtained from 35 individuals who had lived in Barcelona. The close family was interviewed in order to rule out asbestos exposure. Samples were obtained from three areas of the right lung during the autopsy: upper lobe apex, lower lobe apex, and lower lobe base. The samples were treated to remove organic material. The inorganic residue was analysed using a light microscope. The results were expressed as asbestos bodies (AB) per gram of dry tissue. Levels greater than 1000AB/g of dry tissue were considered as potentially causing disease.
*Results:* AB were detected in 29(83%) of the subjects, of which 86% had levels less than 300AB/g. Only one individual (3%) had values greater than 1000AB/g dry tissue. The asbestos residue was higher in the lower lung lobe in 17 individuals (48%) than in the rest, although no significant differences were seen as regards AB residue in the three lung areas studied.
*Conclusions:* The results of this study show that the urban population of Barcelona has asbestos levels in the lung that vary between 0 and 300AB/g dry tissue. No differences in the asbestos residues were detected in the lung areas studied in this population.


## Prevalencia y distribución del depósito pulmonar de amianto en población urbana española

*Palabras clave:*
Amianto
Microscopio óptico
Pulmón
Población urbana

RESUMEN

*Introducción:* El objetivo del presente estudio es analizar la prevalencia y distribución de amianto en pulmón de población urbana de Barcelona.
*Material y métodos:* Se obtuvieron muestras pulmonares necrópsicas de 35 individuos que habían residido en Barcelona. Se llevó a cabo una entrevista con el familiar más cercano para descartar exposición al amianto. En el acto necrópsico, se obtuvieron muestras de 3 zonas del pulmón derecho: apical del lóbulo superior, apical del lóbulo inferior y base del lóbulo inferior. Las muestras fueron tratadas para la eliminación de la materia orgánica. El residuo inorgánico fue analizado mediante microscopía óptica. Los resultados se expresaron como cuerpos de amianto (CA) por gramo de tejido seco. Se consideraron como niveles potencialmente causantes de patología aquellos que superaron los 1.000 CA/g de tejido seco.

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*Resultados:* Se detectaron CA en 29 sujetos (83%). El 86% de las muestras analizadas tenían niveles inferiores a 300 CA/g. Únicamente un individuo (3%) presentó valores superiores a los 1.000 CA/g de tejido seco. En 17 individuos (48%), el depósito de amianto era mayor en el lóbulo pulmonar inferior que en el resto, aunque no se observaron diferencias significativas en cuanto al depósito de CA en las 3 zonas pulmonares estudiadas.
*Conclusiones:* Los resultados del presente estudio demuestran que la mayoría de la población urbana en nuestro medio tiene niveles de amianto en pulmón que oscilan entre 0-300 CA/g de tejido seco. En esta población no se han detectado diferencias en el depósito de amianto, según las zonas estudiadas.


## Introduction

Asbestos is a natural silicate with a fine fibre structure. Sufficiently intense inhalation of asbestos fibres increases the risk of contracting various respiratory diseases, including benign lesions such as pleural plaques, pleural effusion, pleural fibrosis, and more severe pathologies such as malignant mesothelioma, lung cancer and asbestosis.[1] Asbestos was a frequently used material due to its wide industrial utility.[2] The boom in use of asbestos in Spain took place between 1970-1990; in 1992 Spain was the second largest European importer with 25,428t[2] and the total ban on its use did not come until 2002. Therefore, a large number of workers have been exposed to this mineral and will continue to be so into the future given the incorporation of asbestos into numerous structures and buildings. According to the voluntary registration of occupational respiratory diseases in Catalonia, diseases resulting from exposure to asbestos fibres are the second most common.[3]

The diagnosis of diseases caused by inhalation of asbestos is based on 3 factors: knowledge of exposure, patient manifestation of a compatible clinical profile and the exclusion of another disease that would justify the profile. In certain cases, there is a mismatch between the exposure and the clinical profile, which makes diagnosis difficult and often raises medical-legal issues. In these cases, it is necessary to determine the amount of asbestos in the lung tissue,[4,5] which requires a lung examination and the establishment of reference levels in each population through an analysis of individuals with no known occupational exposure.[6,7]

In Spain, the data relating to the pulmonary asbestos deposit in the population is limited to a study published by Monsó et al[8] in which necropsic samples were studied from 33 patients, 16 of which were residents of rural areas and 17 were from urban areas, along with samples from 8 patients with lung cancer without occupational exposure to asbestos. In this study, 50% of the individuals living in urban areas had asbestos bodies (AB) in the lung, compared to only 2 of the 16 (12.5%) living in rural areas.

To date there has been no study that provides more information on the Spanish population. This lack of evidence contrasts with the need for reference values, from which to differentiate the concentrations with pathological potential from those attributable purely to environmental exposures and that do not confer disease risk.

On the other hand, a key element in the interpretation of pulmonary AB values is the importance of the sample. Thus, to obtain representative samples for pulmonary asbestos deposits, it is crucial to know whether this mineral is deposited uniformly in the lung. The studies carried out so far, mostly on subjects exposed to asbestos, have yielded inconsistent results. The accumulation of asbestos in the various areas of the lung follows different patterns depending on the type of asbestos. Sebastien et al[9] showed a greater accumulation of chrysotile asbestos in peripheral areas. Morgan and Holmes[10] observed a greater concentration of anthophyllite in the lower pulmonary lobes, whereas Churg et al[11] found higher levels of asbestos in the upper regions of the lung. More recent studies continue to provide conflicting results, since Kishimoto et al[12] found chrysotile and amosite forming ABs, both in the upper and middle lobes, while Teschler et al[13] concluded that the lower lobes contained the most asbestos. It should be noted that the population examined in these studies[9-13] had known exposure to asbestos, the majority of which was occupational. So far, in our area, there has not been a single study done on the distribution of asbestos in the lung.

The objective of this study was to analyse the possible existence of AB deposits in Barcelona's urban population and to determine the distribution of this mineral in 3 areas of the lung.

## Material and Methods

### *Study Population*

The study was conducted on 35 necropsic lung samples from individuals residing in the city of Barcelona, collected prospectively from June 2004 to June 2005 at the Instituto Anatómico-forense of Barcelona (Forensic Lab of Barcelona), (Table 1). Information on possible exposure of the patient was obtained through interviews with next of kin: spouse, children, parents or siblings. The sampling criteria were set as follows: lack of lung disease and residency in Barcelona for at least 10 years.

### *Exposure Assessment*

After verifying that the individuals met the inclusion criteria, participation in the study was proposed. All interviews were carried out by one of the researchers. In all cases, informed consent was requested from the next of kin to carry out a study of asbestos in the lung tissue. A history of exposure to asbestos was investigated through a structured and comprehensive questionnaire, which had been used in previous studies.[14]

### *Protocol for Obtaining Samples*

In each necropsic procedure, $2cm^3$ lung samples were obtained from three areas of the right lung: upper lobe, apical segment (zone 1), lower lobe, apical segment (zone 2) and base of lower lobe (zone 3). The samples were fixed in formol and sent to our hospital's laboratory for analysis.

All samples were studied by a pathologist from our centre. The presence of chronic pulmonary disease was ruled out in all subjects.

### *Preparation of Lung Samples*

For each sample, two 0.5g fragments of lung tissue that did not contain pleura or vessels were weighed. Subsequently, one of the samples was frozen, lyophilised and weighed to determine the weight of dry tissue. Since there is international agreement regarding expressing the results of AB in relation to the grams of dry lung tissue, a study was conducted on the correlation between weight of

**Table 1**
Characteristics of the study population

| Patient number | Age | Sex | Smoking habit | Job | Cause of death | Area 1 (ABs/g) | Area 2 (ABs/g) | Area 3 (ABs/g) |
|---|---|---|---|---|---|---|---|---|
| 1 | 87 | Male | Non smoker | Cable factory | Ischaemic heart disease | 62 | 19 | 437 |
| 2 | 79 | Female | Non smoker | Housewife | Heart failure | 0 | 20 | 22 |
| 3 | 43 | Male | Smoker | Messenger | Ischaemic heart disease | 0 | 0 | 0 |
| 4 | 35 | Male | Smoker | Messenger | Traffic accident | 0 | 0 | 0 |
| 5 | 67 | Male | Former smoker | Printing press | Aneurysm | 0 | 89 | 20 |
| 6 | 77 | Female | Non smoker | Housewife | Heart failure | 321 | 245 | 667 |
| 7 | 43 | Male | Non smoker | Teacher | Ischaemic heart disease | 22 | 0 | 0 |
| 8 | 44 | Male | Former smoker | Metallurgy | Ischaemic heart disease | 0 | 40 | 146 |
| 9 | 44 | Male | Smoker | Panel beater | Suicide | 44 | 0 | 21 |
| 10 | 76 | Female | Former smoker | Administrative work | Ischemic heart disease | 62 | 0 | 120 |
| 11 | 78 | Female | Unknown | Ironer | Head trauma | 77 | 195 | 266 |
| 12 | 37 | Male | Smoker | Waiter | Ischaemic heart disease | 20 | 0 | 0 |
| 13 | 54 | Male | Smoker | Electrician | Cerebral haemorrhage | 22 | 0 | 0 |
| 14 | 78 | Female | Non smoker | Metallurgy | Unknown | 0 | 20 | 0 |
| 15 | 64 | Female | Non smoker | Podiatrist | Heart failure | 82 | 90 | 21 |
| 16 | 43 | Male | Smoker | Journalist | Traffic accident | 0 | 0 | 0 |
| 17 | 72 | Female | Non smoker | Housewife | Suicide | 0 | 38 | 66 |
| 18 | 44 | Female | Smoker | Housewife | Pulmonary thromboembolism | 0 | 22 | 22 |
| 19 | 49 | Male | Smoker | Waiter | Heart failure | 0 | 0 | 0 |
| 20 | 54 | Male | Smoker | Graphic artist | Traffic accident | 0 | 19 | 40 |
| 21 | 85 | Female | Non smoker | Textile worker | Unknown | 170 | 260 | 283 |
| 22 | 74 | Male | Former smoker | Lathe operator | Sepsis | 21 | 0 | 0 |
| 23 | 74 | Male | Former smoker | Administrative work | Ischaemic heart disease | 0 | 19 | 19 |
| 24 | 85 | Female | Non smoker | Housewife | Choking | 123 | 127 | 255 |
| 25 | 67 | Male | Non smoker | Construction | Heart failure | 283 | 777 | 160 |
| 26 | 52 | Male | Former smoker | Driver | Digestive haemorrhage | 0 | 0 | 19 |
| 27 | 86 | Male | Smoker | Welder | Heart failure | 472 | 333 | 1.307 |
| 28 | 82 | Male | Former smoker | Sales agent | Ischaemic heart disease | 0 | 18 | 81 |
| 29 | 70 | Male | Smoker | Computer technician | Traffic accident | 87 | 42 | 154 |
| 30 | 76 | Male | Unknown | Watchman | Ischaemic heart disease | 78 | 77 | 64 |
| 31 | 42 | Male | Smoker | Mechanic | Suicide | 0 | 0 | 0 |
| 32 | 48 | Male | Former smoker | Electrician | Sudden death | 490 | 265 | 604 |
| 33 | 82 | Female | Non smoker | Housewife | Pneumonia | 21 | 0 | 22 |
| 34 | 65 | Female | Non smoker | Administrative work | Sudden death | 77 | 80 | 132 |
| 35 | 85 | Female | Non smoker | Domestic worker | Heart failure | 0 | 0 | 0 |

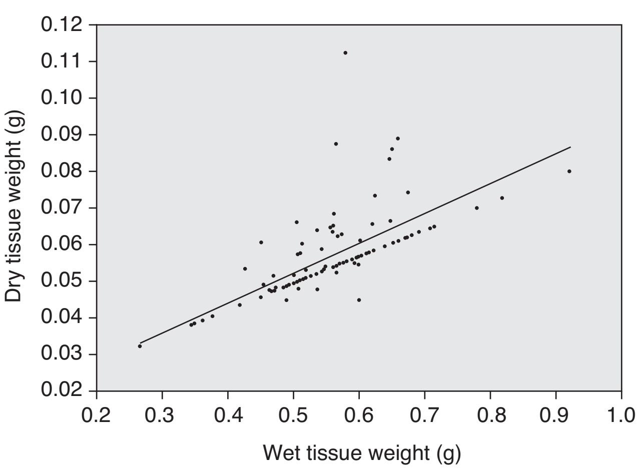


**Figure 1.** Correlation between weight of dry and wet tissue.

wet tissue and weight of dry tissue, yielding a Spearman correlation coefficient of 0.803 (fig. 1). The lyophilised sample was subsequently discarded, since lyophilisation can cause changes in the concentration and size of the fibres.[7]

Then 30cc of previously filtered sodium hypochlorite was added to the non-lyophilised portion of the tissue, and was left to shake for 24 hours to facilitate tissue digestion and removal of organic matter. Subsequently, the sample was centrifuged at 3700 rpm for 20 minutes. After removing the sodium hypochlorite, the sample was resuspended in filtered distilled water. To dissolve the AB in the liquid, the sample was placed in an ultrasonic bath (UCI-50, 300W, 50/60Hz, Raypa S.L.) for 10". After further washing, the sample was resuspended in 20cc of filtered distilled water. The resulting solution was filtered using a 0.45μm diameter filter (Millipore Membrane filters HAWP02500). The filter was dried in an oven overnight at 37° C and transferred to a microscope slide for transparency by means of an acetone vaporiser (JS Holdings 240v/110v) for subsequent reading.

*AB Analysis by Optical Microscopy*

The analysis of each of the filters was performed using an optical microscope (OM) (Olympus CX21FS2, Olympus Life Science Europa GmbH, Hamburg, Germany) at 400x magnification. Pulmonary asbestos content for each individual was obtained for each of the 3 lung areas analysed.

The OM examination helps identify ferruginous bodies. This study, which is in line with international standards,[7] assumed that these bodies correspond to ABs. In fact, although it is known that other minerals may have the same appearance as ABs, Churg and Warnock showed that in lung tissue samples from individuals exposed to asbestos, the majority of ferruginous bodies correspond to ABs.[15]

The highest value for the 3 zones analysed was considered to be the final value for each subject. Those levels that exceeded 1,000 ABs per gram of dry tissue were considered to be potential causes of pathology, in accordance with criteria established by the *European Respiratory Society* working group in 1998.[7]

*Statistical Analysis*

Data were expressed as mean and standard deviations (SD). The distribution of the AB values, analysed by means of the Kolmogorov-

Smirnov test, was normal, so the differences between the lung zones were analysed by means of the Student's t test. The Pearson correlation coefficient was calculated to establish the relationship between the various parameters analysed. The statistical analysis was performed using SPSS software, version 12.0, for Windows (SPSS Inc, Chicago, IL, USA).

## Results

### *Characteristics of the Study Population*

Table 1 shows the main characteristics of the study population and the concentration levels of asbestos in each of the 3 lung areas analysed in each individual.

The average age of the 35 patients was 64 years (range 35-87). Of the group, 22 were men (62.9%) and 13 were women (37.1%). In terms of occupation, the most significant were housewives, construction workers and those related to metallurgy. None of the families interviewed mentioned any history of workplace exposure to asbestos for the individuals studied. The principal causes of death were from heart disease and traffic accidents. The presence of chronic pulmonary disease was ruled out through interviews with the subject's next of kin. Furthermore, the histological examination performed in the study ruled out this condition.

### *Pulmonary Asbestos Content*

The mean (SD) of the values obtained was 167 (280) ABs/g of dry tissue. Only one individual (3%) had values higher than 1,000 ABs/g. Four individuals (11%) had a concentration between 300-1,000 ABs/g, while 30 (86%) had levels below 300 ABs/g (Table 1). In 6 of the individuals of this last group (17%) no ABs were found in the lung. There was no significant correlation observed between AB levels and age (correlation coefficient: 0.347).

The average concentration of asbestos in the group of men was 176 ABs/g (range: 0-1307) and in the group of women was 151 ABs/g (range: 0-667). There were no significant differences observed between the two groups. Information was available on the smoking habits of 33 of the patients, of which 20 were or had been smokers and the rest were non-smokers. There were no significant differences observed in the levels of ABs/g of dry tissue between the 3 groups.

### *Distribution of Asbestos Content per Lung Area*

The mean values of ABs/g for each of the pulmonary lobes in the autopsied population studied were 72 (range: 0-490) for zone 1, 80 (0-777) in zone 2, and 141 (0-1307) in zone 3. In 17 individuals (48%), the asbestos deposit was greater in the lower pulmonary lobe (area 3) (fig. 2). However there were no significant differences observed in terms of AB deposit, taking into account the 3 areas studied nor in the possible comparisons between two zones. The Pearson correlation coefficient between the different lung zones was 0.6 between zones 1-2, 0.7 between zones 1-3, and 0.8 between zones 2-3.

## Discussion

This study provides the first data on the distribution of AB in the urban Spanish population. The results of this study show that there are no differences in the asbestos deposit in the analysed lung areas.

The average concentration of asbestos in the lower pulmonary lobes was slightly higher than in the upper lobes, although this difference was not statistically significant. The homogeneity of the asbestos deposit was also shown by the good correlation of values in the different areas. As detailed in the introduction, several studies previously published in other countries have shown conflicting results in exposed individuals. Our population, however, had no occupational exposure to asbestos. The results seem to indicate that lower asbestos exposures do not result in differences in the deposit of ABs between different lung areas. This fact is relevant when evaluating results obtained from clinical samples, which normally correspond to one area of the lung. Therefore, according to the results of this study, it can be confirmed that in these individuals the analysis of one single area of the lung is representative of the whole lung. However, despite the absence of statistical differences, we have observed a case in which the above must be questioned. The case in question is that of an 86-year-old man, a smoker, who had worked as a welder. The asbestos analysis showed values of 472.333 and 1,307 ABs/g of dry tissue in the upper lobe and the middle and lower lobes, respectively. Therefore, in this case, according to the studied area, results were obtained above and below the threshold of 1,000 ABs/g of dry tissue, which has been accepted internationally as an initiator of pulmonary and pleural pathology.[7] In our opinion, this case could involve occupational exposure that was not known to the family. In fact, working as a welder can be considered a risk for inhalation of asbestos and it is well known that the exposure questionnaires, obtained from family members, may suffer from a low diagnostic sensitivity.[7] With this exception, we believe there is high reliability in the diagnosis of pulmonary asbestos, even when analysing only one area of the lung.

To date only one study has been published in Spain that provides values for pulmonary asbestos in the population that is not exposed to it in the workplace.[8] Ours is now the second study and has a greater number of cases relating to the urban population. The values measured in the study were lower than 300 ABs/g of dry tissue in 86% of the study population. Compared to the Monsó et al study, the current study observed a lower percentage of cases with absence of asbestos (6/35, [17%]) compared to 8/18 (44%). Comparing the average of cases that showed presence of asbestos, Monsó et al found a mean value of 95.14 ABs/g of dry tissue, while in the current study the value was 201 and an isolated case with a value greater than 1,000. These differences may be due to the fact that our study analysed 3 lung areas and determined the maximum global value to be 3. We cannot rule out either the fact that there are differences in the degree of exposure among the population, even when that exposure is not due to the workplace. The presence of asbestos in the lungs of subjects that were believed not to be exposed can be explained by the ubiquitous presence of the silicate in the cities' air. In addition to environmental inhalation, which can be increased if

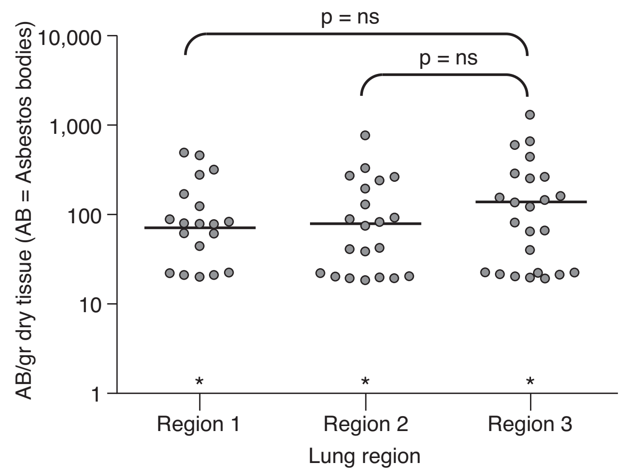


**Figure 2.** Pulmonary asbestos deposits in the different areas studied.

**Table 2**
Asbestos bodies in lung tissue samples of non-exposed populations of different countries

| Country or area | Population (n) | ABs/g of dry tissue | Reference |
|---|---|---|---|
| Barcelona (Spain) | 8 | 0/8 > 100 | 8 |
| Canada | 81 | 9/81 > 750 | 16 |
| Quebec (Canada) | 49 | 24–471 | 17 |
| Switzerland | 137 | 4/137 > 500 | 18 |
| Vancouver (Canada) | 20 | 280 (average) | 19 |
| Tokyo (Japan) | 390 | 0/90 > 40<br>5/300 > 400 | 20 |
| Giessen (Germany) | 41 | 3/41 > 3.000 | 21 |

the person has lived near industries that handle asbestos, there are other sources of exposure that often go unnoticed both at home and in the workplace.

With respect to series from other countries, it is noteworthy that the ranges of ABs/g of dry tissue vary depending on the population but are comparable to those obtained in our study. In most of those series, the average values were lower than 500 ABs/g of dry tissue,[8,16-20] although individuals with values above 1,000 were encountered. In one German series, 3/41 non-exposed subjects presented levels higher than 3,000 ABs/g of dry tissue[21] (Table 2). This variability may be due in part to technical differences between laboratories, which confirms the need for reference values obtained for each population with each sample treatment protocol used. As for the extreme values above 1,000 ABs/g of dry tissue, as was noted earlier, these reflect the lack of sensitivity that the case history can have in the detection of exposure to asbestos.

On the other hand, the possibility that an AB analysis underestimates the exposure should not be ruled out. Indeed, it should be noted that asbestos, after being inhaled, goes through a drainage process that alters its final deposit. Chrysotile, for instance, is known to undergo considerable drainage after inhalation, which means that the chrysotile deposit detected years afterwards may not reflect the intensity of the exposure.[22] This has special significance in our environment, since it has been reported that the majority of asbestos imported in recent years corresponds to chrysotile.[2] In short, one must contemplate the possibility that, as in all cases that analyse AB content, said deposit does not fully reflect the actual amount of asbestos a population has been exposed to.

The AB content of our population was analysed in relation to certain variables of interest. In regards to smoking, the Selikoff et al[23] study showed increased deposits of asbestos in smokers with lung cancer. In our study, an increased deposit of ABs was not detected among smokers as compared to non-smokers. Our results are similar to those obtained by Monsó et al,[7] which suggest that smoking does not change the deposit of asbestos in the lung for low levels of exposure. In terms of age, there was no significant correlation with AB values in the lungs in our population. Previously, a Canadian study observed a tendency for the average value of pulmonary asbestos to rise as the population aged, although a statistical difference was not demonstrated.[16] Although one might think *a priori* that the deposit of ABs should be proportional with the number of years lived in an urban setting, the clearing effect produced in the case of chrysotile,[22,24] one of the most common fibres employed in our environment, has already been discussed. However, we believe that this issue is open to further analysis in series with greater numbers of subjects.

A limitation of this study may be possible bias due to the indirect collection of work history. In this respect, De Vuyst el al[7] affirm that studies carried out in reference population groups (autopsic) allow for optimum sampling, while lung cancer groups make it is possible to perform a direct anamnesis, although with risk of bias towards a greater load of asbestos, and therefore an optimum correlation between antecedents and pulmonary asbestos. In our case, since the objective was to analyse the possible existence of AB deposits in the urban population of Barcelona that had no respiratory pathologies, it was necessary to opt for an autopsy population when sampling, assuming that the indirect medical history may not be accurate.

In conclusion, the results of this study show that the majority of the urban population in our area has levels of asbestos in the lung, with values falling below 300 ABs/g of dry tissue. As a result, we believe that the threshold of 1000 ABs/g is fully applicable for differentiating asbestos contents with pathological values. In the analysis of the lung areas studied, there were no differences detected in terms of asbestos deposits, with good correlations between values.

## Conflict of Interest

The authors affirm that they have no conflicts of interest.

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