A Quantitative Study of Environmental Asbestos Exposure in Honolulu

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The increased use of asbestos in various industries in past decades has led to increases in environmental asbestos pollution. Incidental exposure to asbestos is inevitable, and has generated public concern. We performed the following study aimed at determining the level of environmental asbestos exposure in Honolulu, and our results indicate that the levels of environmental asbestos in Honolulu are the lowest in the nation.

Introduction

Asbestos consists of a group of widely used fibrous silicates that are well known for causing adverse health effects to exposed occupational workers.[1-2] Asbestos bodies are asbestos fibers coated with iron and protein, and are unique histologic markers for asbestos exposure. These "curious" bodies were first described in patients with asbestosis about 50 years ago.[3] Since then, it has been well recognized that the asbestos body content in the lung is correlated with the degree of asbestos exposure and is considered a necessary finding in establishing the diagnosis of asbestos-related diseases.

The increased use of asbestos in consumer products and in construction materials in past decades has increased the chances of incidental, non-occupational exposure. Studies have confirmed that incidental exposure to asbestos dusts in the general population is also on the rise.[4-5] The health effects of incidental asbestos exposure, particularly in regard to the risk for mesothelioma, is still unknown[6] and has generated much public concern.[7-8]

In Honolulu, the naval shipyard at Pearl Harbor was the main source of local occupational asbestos exposure during the second world war[9] when exposure control was not strictly regulated. In recent years, autopsies have been frequently requested to document previous asbestos exposure of diseased workers. In order to determine the level of incidental, environmental asbestos exposure in our community and to establish a control background level of non-occupational asbestos exposure we sampled lung tissues of random autopsies from St. Francis Medical Center in Honolulu and quantified the asbestos bodies in these lung tissues. In this study, asbestos body counts from patients with known histories of occupational exposure to asbestos from Pearl Harbor naval shipyard are included for comparison.

Materials and Methods

Lung tissues from random autopsies from St. Francis Medical Center at Liliha, Honolulu were collected during a 10-year period from 1979 to 1988. The data obtained from each autopsy report included age, race, sex, occupational history, history of smoking, and presence or absence of asbestos related diseases. A total of 167 autopsies of patients without histories of occupational exposure to asbestos dusts and 18 cases of patients with known histories of occupational exposure to asbestos were analyzed. Of the 167 cases without occupational exposure to asbestos, 107 were male and 60 were female. Ages ranged from 15 to 93 with a mean age of 64. The ethnic backgrounds of these 167 cases were recorded as follows: 48 Caucasian, 40 Japanese, 31 Filipino, 18 Hawaiian, 15 Chinese, and 13 other or mixed race. Data from 18 patients with known histories of occupational exposure to asbestos were tabulated separately.

Extraction of Asbestos Bodies

Lung tissues were sampled from all 5 lobes. Approximately 10 gm. from each lobe was fixed in a 10% buffered formaldehyde solution. The lung tissue was minced, mixed, and pooled. Asbestos bodies were then extracted from 5 grams of the pooled lung tissue by Smith and Naylor's digestion method.[10] Briefly, the sampled lung tissue was dissolved in a domestic laundry bleach (5.25% sodium hypochlorite). The digested tissue sediment was then washed with chloroform and ethanol to remove organic substances. Following centrifugation, the final sediment that contained asbestos bodies was filtered onto a 5 μm pore size Millipore filter. Asbestos bodies were counted directly under a light microscope.

Only morphologically typical asbestos bodies, i.e., those bodies with a characteristic central transparent fiber core and a golden-brown beaded or segmented iron-protein coat, were counted. Non-asbestos ferruginous bodies or "pseudoasbestos bodies" were carefully excluded from the counting. These non-asbestos ferruginous bodies appeared as aggregates of iron-protein particles without a transparent fiber core or with an irregular non-transparent core.[11]

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Table 1.—The Asbestos Body Counts of Non-occupational Group (167 cases)

Asbestos Bodies per Gram of Lung Tissue	Number of Cases Percentage of Total
0	58 (34.7%)
1 - 5	79 (47.3%)
6 - 10	18 (10.7%)
11 - 20	4 (2.4%)
over 20	4 (2.4%)

Table 2.—Non-Occupational Cases with More Than 10 Asbestos Bodies per Gram of Lung Tissue

Age	Sex	Occupation	Asbestos Body Count
72	M	Fireman	18
49	M	Mortgage Co. Clerk	12
76	F	Housewife	60
72	M	Electrician	70
74	M	Mining Engineer	20
61	M	Security Guard	21
78	F	School Teacher	25
56	M	Police Officer	11

Diluted aliquots were used when the counts were unusually high and difficult to count.

Results

Environmental Exposure of Asbestos

1. Asbestos Body Counts in the Lungs

Asbestos bodies were found in 108 (64.6%) of the 167 cases without occupational history of asbestos exposure. In Table 1, the distribution of asbestos body counts in the group without occupational exposure to asbestos is shown. Fifty eight cases (34.7%) showed no detectable asbestos bodies in their lungs, 79 cases (47.3%) had 1 to 5 asbestos bodies, 18 cases (10.7%) had 6 to 10 asbestos bodies, 4 cases (2.4%) had 11 to 20 asbestos bodies, and 4 cases (2.4%) had more than 20 asbestos bodies per gram of lung tissue.

We were particularly interested in further characterizing the 8 cases with greater than 10 asbestos bodies per gram of lung tissue. In Table 2, their occupations, age, sex, history of smoking, cause of death, and asbestos body counts are listed. Six were males and 2 were females. Their occupations consisted of a fireman, a mortgage company clerk, an electrician, a mining engineer, a school teacher, a police officer, a security guard and a housewife. One person (the fireman) died of lung cancer, and another (the mining engineer) died of chronic obstructive pulmonary disease. None were diagnosed with asbestosis or mesothelioma.

2. Age and Sex Distribution

In Table 3, the age and sex distribution of the cases without occupational exposure to asbestos are listed. Asbestos bodies were found in 76 (71%) of the 107 males and in 32 (53.3%) of the 60 females. Using the chi-square test, the difference between the males and the females is statistically significant ($p < 0.05$). In addition, there was an increased number of positive cases (defined as at least one asbestos body per gram of pooled lung tissue) in the older age groups. Below the age of 40, asbestos bodies were found in 5 out of 13 cases, a positive rate of 38%. The positive rate increased to 46% in the age range of 41 to 50, to 57% in the age range of 51 to 60, to 61.5% in the age range of 61 to 70, to 77.5% in the age range of 71 to 80, and to 80% in the group over the age of 80. The analysis failed to show significant differences based on ethnicity.

Table 3.—Age and Sex Distribution

Age	Number of Cases			Cases with Asbestos Bodies		
	Male	Female	Total	Male	Female	Total
under 40	11	2	13	4	1	5
41 - 50	9	4	13	4	2	6
51 - 60	19	14	33	13	6	19
61 - 70	23	16	39	16	8	24
71 - 80	32	17	49	28	10	38
over 80	13	7	20	11	5	16
Total	107	60	167	76	32	108

Table 4.—Smokers and Non-smokers (non-occupational)

Smoking History	Sex:	# Cases	# Cases with A B*	% +
Smokers:	Male	53	45	84.9
	Female	18	13	72.2
	Total:	71	58	81.7
Non-smokers:	Male	35	19	54.3
	Female	30	13	43.3
	Total:	65	32	49.2
Information not available:	Male	19	12	63.1
	Female	12	6	50.0
	Total:	31	18	58.1

*AB = asbestos bodies
Average number of asbestos bodies per gram of lung tissue
Smokers: = 4.5 asbestos bodies/gm.
Non-smokers: = 3.1 asbestos bodies/gm.

3. Smokers versus Non-Smokers

In Table 4, the differences of asbestos body counts between the smokers and non-smokers are listed. Of the 167 cases without occupational exposure to asbestos, 71 (53 males and 18 females) were cigarette smokers, 65 (35 males and 30 females) were non-smokers, and in 31 cases (19 males and 12 females) information on smoking was not available. Asbestos bodies were found in 58 (81.7%) of 71 smokers and 32 (49.2%) of 65 non-smokers demonstrating a statistically significant difference between smokers and

Table 5.—Patients with Known Occupational Asbestos Exposure

Case No.	Age	Asbestos Body Count*	Malignancy (cause of death)
1.	65	1200	Ca of the lung
2.	68	320	Mesothelioma
3.	45	125	Ca of the lung
4.	69	1200	Mesothelioma
5.	65	600	Ca of the larynx
6.	56	8060	Mesothelioma
7.	84	1000	Ca of the lung
8.	74	45	
9.	65	1000	Ca of the lung
10.	58	37	
11.	63	1050	Ca of stomach
12.	65	900	Ca of the lung
13.	63	120	
14.	80	100	Mesothelioma
15.	62	930	Ca of the lung
16.	66	116	Mesothelioma
17.	65	1575	
18.	54	390	Ca of the lung

*Per Gram of Lung Tissue.
All 18 patients were male

non-smokers (Chi-square test, $p<0.05$). In addition, a greater number of asbestos bodies were detected in the lungs of smokers than non-smokers, as smokers had an average of 4.5 and non-smokers had an average 3.1 asbestos bodies per gram of lung tissue.

Occupational Group

Asbestos body counts and clinical data from the 18 patients with known histories of occupational exposure to asbestos are summarized in Table 5. The counts varied from 37 to 8060 per gram of lung tissue. In Figure 1, a graph of the total asbestos body counts from cases with and without histories of occupational exposure to asbestos is shown. An overlap is noted where two cases with histories of occupational exposure to asbestos had low counts of 37 and 41 asbestos bodies, and where two cases without histories of occupational exposure to asbestos (a housewife and an electrician) had high counts of 60 and 70 asbestos bodies per gram of lung tissue. The average overall count for the cases with occupational exposure was 1043 and for the cases without occupational exposure was 3.4 per gram of lung tissue.

Clinically, all 18 patients with histories of occupational exposure to asbestos had fibrotic pleural plaques, and 14 of the 18 had peribronchiolar fibrosis and a diagnosis of asbestosis. Five of the 18 patients died of mesothelioma and 7 of the 18 died of lung cancer. In contrast, of the 167 non-occupational cases, none died of mesothelioma, 4 died of lung cancer.

Discussion

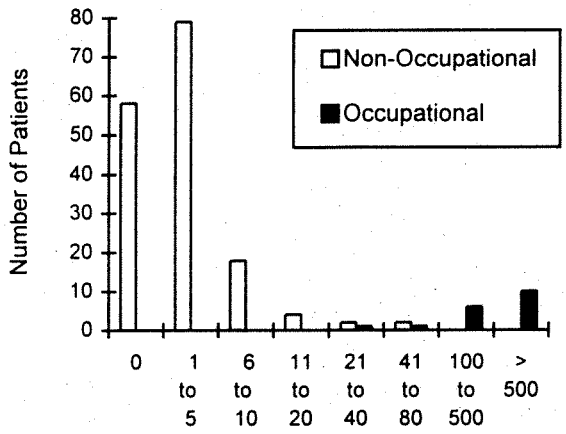
In this study the quantification and distribution of asbestos bodies in patients with histories of occupational exposure to asbestos are comparable to data reported by others.[12-13] Two of 18 cases had less than 100 asbestos bodies per gram of lung tissue, and the remaining cases had asbestos body count up to 8060, with an overall average count of 1,043 asbestos bodies per gram of lung tissue. Fourteen of the 18 patients had malignancies, including 7 cases of lung cancer, 5 cases of mesothelioma, one case of adenocarcinoma of the stomach and one case of squamous cell carcinoma of the larynx. The high incidence of malignancy may be due to high alert in connection with asbestos exposure as these cases were requested for autopsy to establish evidence of asbestos exposure for litigation and compensation purposes.

Table 6.—Comparison of Environmental Asbestos Exposure in US

Investigator	Location	# Cases	% (+)
Utidjian et al*	Pittsburgh	100	97
Rosen et al*	N.Y. City	86	93
Breedin and Buss*	N. Carolina	100	93
Churg and Warnock*	Chicago	252	96
Bhagavan and Koss*	Baltimore	145	91.1
Yang et al.	Honolulu	167	64.6

*References (4, 12, 14, 15, 16)

Fig 1.—Asbestos Bodies Per Gram Lung Tissue



In the non-occupational group, asbestos bodies were found in 108 (64.6%) of the 167 cases. The detection rate of 64.6% is significantly lower than that of several other reported similar studies as seen in Table 6.[4,12,14-16] In other studies using the same digestion method, asbestos bodies were detected in more than 90 % of the population from many different geographical areas in the United States and one study by Breedin and Buss included both urban and rural populations.

The results suggest that environmental (non-occupational) exposure to asbestos is extremely low in Honolulu. This may result from the relative isolation of Honolulu from industrial sources of asbestos, as well as from the effects of ocean air and trade winds in constant cleaning of the environment. Two persons from our non-occupational group, an electrician and a housewife, had relatively high asbestos body counts (70 and 60 asbestos bodies per gram of lung tissue respectively), which are nevertheless within the cut off point of 100 suggested for non-occupational exposure by Churg and Warnock.[12] The electrician actually belongs to the group of "secondary asbestos workers" who use asbestos-containing products in their

jobs,[12] and this may explain his relatively high asbestos body counts. The asbestos source of the housewife was not clear. Neither of these 2 persons was diagnosed with any asbestos-related diseases.

The results also showed a cumulative effect of age with a clear trend towards increased rate of detection of asbestos bodies with advancing age. The oldest age group had the highest positive rate at 80%. The difference of asbestos exposure between men and women in our series may be partly due to work-related secondary exposure and partly due to a higher incidence of the smoking habit in men than in women. In our study, more men than women were smokers, and the smoking habit was a significant factor in increased retention and the incidence of asbestos bodies in the lungs as shown in Table 4. Asbestos bodies were detected in 81.7% of the smokers and in 49.2% of the nonsmokers. Fifty three (74.6%) of the 71 smokers were male and 18 (25.3%) of the 71 were female. Smokers also had higher average counts of asbestos bodies in the lung (4.5 asbestos bodies/gm) than did nonsmokers (3.1/gm). In animal studies, cigarette smoking has been noted to impair the lung's ability to clear asbestos fibers,[17] and smoking also facilitated the penetration of asbestos into the bronchiolar wall.[18] This increased retention of asbestos among the smokers may contribute to the observed synergistic effects of smoking and asbestos in carcinogenesis.[19]

Although Honolulu is among the cleanest environments regarding incidental asbestos exposure, the public should still be reminded that asbestos products such as popcorn ceilings, roof tiles and many electrothermal insulating materials are still present in and around the living environment. Many houses, apartments, and school buildings in the community built before the enforcement of regulatory legislation are possible sources of environmental asbestos exposure, and the resulting low dose exposure is not completely harmless. Emphasis must be placed on strict control measures for building demolition and for continued public awareness of careful handling of these existing asbestos-containing building materials.[20]

Summary

Incidental environmental asbestos exposure is inevitable anywhere in the world and this problem has generated public concern. The present study demonstrates that environmental exposure to asbestos in Honolulu is among the lowest in the nation, as compared with many cities and even rural areas on the mainland United States. In addition, our results also demonstrate that there is a cumulative effect of asbestos in the lungs with advancing age, and that cigarette smoking increases the chance of retention of asbestos in the lungs.

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