Numbers of Asbestos Bodies in Urban Patients with Lung Cancer and Gastrointestinal Cancer and in Matched Controls*

*Andrew M. Churg, M.D.** and Martha L. Warnock, M.D.***

We compared the numbers of asbestos bodies extracted from the lungs of 103 patients with lung cancer and 50 patients with gastrointestinal malignant neoplasms to the numbers of bodies extracted from lungs of control patients matched for age, sex, smoking habits, and, in some cases, occupation. All patients were urban dwellers over the age of 40 years, and none was a primary asbestos worker. No differences in the counts of asbestos bodies were observed between the tested and control populations. The numbers of asbestos bodies did correlate well with occupation; the highest counts were found in male manual laborers. We conclude that in the urban population studied herein, the numbers of asbestos bodies alone do not correlate with the presence of pulmonary or gastrointestinal carcinoma; however, uncoated asbestos fibers are also known to be present in the lung, and the possibility that such tumors may be related to the numbers of these fibers in lungs remains to be explored.

Except for cigarette smoke, the causes of the rising incidence of cancer of the lung are largely undetermined. Recent epidemiologic studies indicate that carcinoma of the lung has increased in frequency from 25/100,000 male subjects in 1951 to 60/100,000 male subjects in 1973[1] and that lung cancer is especially prevalent in industrial workers of lower socioeconomic class and in certain geographic areas in the United States (Chicago, where this study was performed, is in one of the counties with the highest death rate from lung cancer in the country).[2,3] These data cannot be accounted for by patterns of smoking but may correlate with industrial exposure or industrial air pollution.[4]

It is well known that inhalation of large amounts of asbestos may cause carcinoma of the lung, especially in smokers. Smaller amounts may be associated with mesothelioma.[5] Epidemiologic studies have also suggested that gastrointestinal cancers are associated with exposure to asbestos.[6]

The use of asbestos is now widespread in many industries and products, and there is increasing evidence of contamination with low levels of asbestos in air and water supplies,[4,7] as well as the possible hazard to the general population from such unlikely sources as road-paving materials[8] and even drugs.[9]

One manifestation of such contamination is the presence of both asbestos bodies and uncoated asbestos fibers in the lungs of almost everyone in the general population. There has been considerable controversy in the past about whether such bodies actually contain asbestos;[10] we have recently shown that almost all such bodies do contain cores of amphibole asbestos.[11,12] Several qualitative studies have failed to demonstrate an association between the presence of asbestos bodies in urban individuals and bronchogenic carcinoma.[13-15] but in a preliminary quantitative study, we[16] found that 27 percent of patients with lung cancer had more than 50 asbestos bodies per gram of lung, whereas only 5 percent of the unmatched control population had that number. Since then, we[17] have discovered that in our urban postmortem and surgical population, sex and occupation are important factors in determining the numbers of asbestos bodies found in the lungs; hence, an appropriate control population must be carefully matched for these variables. In the present report, we have counted asbestos bodies in 103 urban patients with bronchogenic carcinoma and compared them with 103 urban patients matched for age, sex, smoking history, and, where possible, occupation. A similar comparison was performed for 50 patients with gastrointestinal carcinoma and matched controls.

*From the Department of Pathology, Pritzker School of Medicine, Chicago.
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**Formerly at the Department of Pathology, University of California, San Francisco.
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Reprint requests: Dr. Warnock, Department of Pathology, HSW595, University of California School of Medicine, San Francisco 94143

Materials and Methods

Samples

Samples of lung were obtained from 363 patients for whom light-microscopic counts of asbestos bodies; detailed smoking, occupational, and residential histories; and complete histologic material were available. Occupational, smoking, and residential histories were obtained by interviews with the patients or relatives. Interviews were conducted without knowledge of the count of asbestos bodies by a social worker using pages 4 and 5 of the questionnaire on respiratory symptoms of the Medical Research Council, supplemented with questions on occupational history, hobbies, and use of insulating and construction materials at home. Two hundred fifty-two of these patients have been analyzed previously for correlation of occupation and counts of asbestos bodies.[17] All patients were seen at the University of Chicago Medical Center between February 1974 and May 1977. Samples of lung were taken from 103 patients with lung cancer (66 from autopsies and 37 from patients undergoing resection for lung cancer), from 50 patients dying with cancer of the esophagus, stomach, or colon, and from 210 control patients dying during the same period of time. The control population was composed of patients with either nonneoplastic disease or neoplasms other than cancer of the lung or gastrointestinal tract. Only urban patients who resided for more than 20 years in a city with a population of more than 100,000 and who were over the age of 40 years were included in the study. In order to determine the relationship of our counts of asbestos bodies to counts in patients with heavy exposure to asbestos, we analyzed samples of lung from six patients with asbestosis.

Preparation of Pulmonary Tissue

Lung fixed in formaldehyde solution (Formalin) was digested with bleach (sodium hypochlorite, 5.25 percent), and the digest was collected on membrane filters with a pore size of 0.45μ (Millipore), according to the method of Smith and Naylor[18] as modified by Churg et al.[17,19] Three samples with a wet weight of approximately 4 gm each were taken from each lung from autopsy (one sample from the upper and two from the lower lobe). Two samples were taken from resected lobes. Samples were taken from parenchyma away from large bronchi, tumor, consolidation, scar, or pleura. The values for the samples counted were averaged, and the result was expressed as the number of asbestos bodies per gm of wet weight of lung. For the samples from lungs with asbestosis, the size of the sample was decreased to 1 to 2 gm, and bodies in ten random areas measuring 0.04 sq mm were counted; the result was multiplied by an appropriate factor. The results for wet lung may be roughly converted to bodies per gram of dry weight by multiplying by a factor of 10.[17] Asbestos bodies were counted without knowledge of either the patient's disease or occupation.

Data on Patients

Slides and protocols from each of the 103 cases of lung cancer were reviewed to confirm the diagnosis and exact location of the tumor. Cases in which the possibility of a nonpulmonary primary carcinoma was raised were eliminated from the study. All surgical cases in which the preoperative work-up failed to disclose another primary cancer were included. Tumors were classified using the classification of the World Health Organization. Tumors were examined without knowledge of the counts of asbestos bodies or knowledge of occupational and environmental histories. Protocols and slides from the 50 patients with gastrointestinal cance were reviewed to confirm the diagnosis. Protocols for all the control patients were reviewed to confirm the absence lung and gastrointestinal cancer. Because we[17] have four that a level of approximately 100 asbestos bodies per gra appears to be a cutoff between persons with clear-cut se ondary occupational exposure to asbestos and persons with evident occupational exposure, we examined the type ar distribution of lung cancer in patients with greater or le than this number of bodies.

Case Matching and Statistical Methods

All data for the 363 patients were stored on comput disks. Patients with lung cancer were matched against co trols by sex, age within ten years, and pack-years of cig rettes within ten. For this study, patients with less than pack-years of smoking or patients who smoked cigars or pip were designated nonsmokers. Matching trials were performe with and without the patients with gastrointestinal cancers the control group. Since occupation has been shown influence the number of asbestos bodies in the lung in ma subjects,[17] an additional matching of patients with lu cancer and control subjects using age, sex, smoking, a occupation was performed. For this comparison, patients wi white-collar occupations were matched with patients wi white-collar occupations, but manual laborers were not su classified as to type of work. Matching was done largely the computer to decrease bias; however, five men had to matched by hand because they had smoked very heavily (1 to 220 pack-years). In these instances, persons with 100 more pack-years of smoking were considered suitab matches. Three women were similarly matched by hand. Th patients with gastrointestinal cancer were matched by t same criteria for sex, age, and smoking against the contr group that excluded patients with lung cancer. Since a given matching trial could not make use of all of the contr cases, the computer program was designed to permit differe sets of control patients to be matched against the patien with cancer. Multiple matching trials using this capabili were performed for each tested group.

Because the counts of asbestos bodies were marked skewed, with most counts being less than 300 (even for ma manual laborers), statistical tests assuming a normal distrib tion were not considered to be suitable; and nonparamet tests were chosen to analyze the data. The sign test a Wilcoxon's matched-pairs signed-ranks test were perform on the paired data.[20] These tests utilize information abo the direction and the magnitude of differences betwe paired values, respectively. In order to compare the grou more specifically on the basis of actual measured valu (rather than, for example, on the means or signs of pair differences) while avoiding assumptions about distributio we disregarded all matching and treated the groups wi cancer and the control group as independent samples for t Kolmogorov-Smirnov test.[20] Because our hypothesis was th the counts of asbestos bodies in the experimental group we "higher" than in the control group, we used a one-tailed te throughout.

Results

Pulmonary Cancers

Counts of asbestos bodies for the patients wit lung cancer varied from zero to 9,200/gm of lun and varied for the controls from zero to 1,980/gm o

Table 1—*Statistical Analysis of Cases*

Group	No. of Matched Pairs	Analysis of Matched Cases: Wilcoxon's P Value*	Results of Sign Test: Ca>Control**	Ca<Control†	P Value	Analysis of Cases Disregarding Matching (Kolmogorov-Smirnov Test): D Value	χ^2 ($df=2$)	P Value
Lung cancer								
Men vs all controls (matched for age and smoking)	69	0.17	34	35	0.5	0.12	2.0	>0.3
Men vs controls without gastrointestinal cancers (matched for age and smoking)	69	0.21	36	32	0.36	0.14	2.9	>0.2
Men vs controls without gastrointestinal cancer (matched for age, smoking, and occupation)	60	0.12	31	29	0.44	0.18	4.0	>0.1
Women vs all controls	34	0.19	18	15	0.36	0.06	0.2	>0.9
Women vs controls without gastrointestinal cancer	34	0.21	19	14	0.24	0.12	1.0	>0.5
Gastrointestinal cancer								
Men vs controls without lung cancer	31	0.37	14	16	0.57	0.16	1.6	>0.3
Women vs controls without lung cancer	19	0.12	12	7	0.18	0.21	1.7	>0.3

*Wilcoxon's matched-pairs signed-ranks test.
**Pairs with greater numbers of asbestos bodies in patient with cancer than in control.
†Pairs with greater numbers of asbestos bodies in control subject than in patient with cancer.

lung. Only two of the patients with lung cancer had counts over 2,000/gm; these were 9,000/gm and 9,200/gm, respectively. The average age of the women with lung cancer was 58 years and of the men was 61 years. The median count of asbestos bodies for women with lung cancer was 11/gm and for matched controls was 13/gm; the median count for men with lung cancer was 33/gm and for their matched controls was 44/gm.

Matching of patients with lung cancer and control subjects was initially based on age, sex, and smoking history. Two different control groups were selected. The first excluded patients with gastrointestinal cancers because it was thought that this group might have high counts of asbestos bodies and thus minimize differences between tested and control groups; for similar reasons, patients with lung cancer were not used as controls for patients with gastrointestinal cancers. The second control group included patients with gastrointestinal cancers.

In a separate matching trial, 60 of the 69 men with lung cancer were matched by occupation, as well as age and smoking history. Examination of the counts of asbestos bodies for each matched pair by the sign test indicated no significant differences between patients with lung cancer and control subjects. The number of pairs with higher counts in the patient with cancer than in the control subject approximately equaled the number where the reverse was true (Table 1). Similarly, the results for Wilcoxon's test showed no significant differences (Table 1). The χ^2 values which are derived from the D values for the Kolmogorov-Smirnov test for each of the comparisons for men and women were not significant (Table 1 and Fig 1). The multiple matching trials using different subgroups of the control population all gave similar results.

Results of classification of tumors by type and location in the lung for patients with counts above

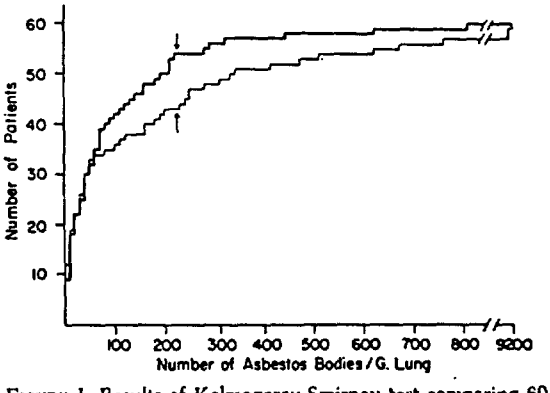

FIGURE 1. Results of Kolmogorov-Smirnov test comparing 60 men with lung cancer (*light line*) and their 60 matched control subjects (*heavy line*), treated as two independent random samples. Cumulative distributions of each sample are plotted at intervals of ten asbestos bodies. These distributions for men demonstrate also marked skewing of data. *Arrows* indicate largest deviation between curves. Because chance deviation of this size is not unlikely, this comparison provides no evidence that samples were from different populations.

Table 2—*Type of Lung Cancer According to Count of Asbestos Bodies*

Group and No. of Asbestos Bodies*	No. of Cases (Percent)			
	Squamous Cell Carcinoma	Adeno-carcinoma	Large Cell Carcinoma	Small Cell Carcinoma
Women				
<100/gm	6 (21)	12 (41)	6 (21)	5 (17)
>100/gm	3 (75)	1 (25)	0	0
Men				
<100/gm**	15 (34)	18 (41)	5 (11)	6 (14)
>100/gm	10 (40)	12 (48)	0	3 (12)

*Value of 100 asbestos bodies per gram of lung was arbitrarily selected as cutoff between counts below that value (environmental exposure) and counts above that value (occupational exposure).[17]

**One adenosquamous tumor has been omitted.

and below 100 bodies per gram are given in Tables 2 and 3. No differences between the two groups are apparent for type of tumor or location, with the single exception of the high frequency of squamous carcinoma and low frequency of adenocarcinoma in the women with high counts.

Gastrointestinal Cancers

Counts of asbestos bodies for the patients with gastrointestinal cancers varied from zero to 559/gm. The median count of asbestos bodies for women with cancer was 27/gm and for matched controls was 15/gm; the median count for men with cancer was 20/gm and for matched controls was 40/gm. Twenty-three of 31 male patients and ten of 19 female patients were smokers. The average age of the women was 63 years and of the men was 65 years. The locations of the gastrointestinal cancers are given in Table 4.

Matching of patients with gastrointestinal cancer and control subjects was based on age, sex, and smoking. All patients with lung cancer were excluded from the control group. Examination of the

Table 3—*Location of Lung Cancer According to Count of Asbestos Bodies**

Group and No. of Asbestos Bodies	No. of Cases (Percent)	
	Upper and Middle Lobes	Lower Lobes
Women		
<100/gm of lung	16 (70)	7 (30)
>100/gm of lung	3 (75)	1 (25)
Men		
<100/gm of lung	23 (64)	13 (36)
>100/gm of lung	18 (82)	4 (18)

*Origin of six tumors in women and 11 in men could not be attributed to specific lobar bronchus.

Table 4—*Gastrointestinal Cancer by Site*

Group	No. of Cases (Percent)		
	Esophagus	Stomach	Colon
Men	7 (22)	12 (39)	12 (39)
Women	4 (21)	5 (26)	10 (53)
Total	11 (22)	17 (34)	22 (44)

paired counts of asbestos bodies showed no significant differences between patients with gastrointestinal cancer and controls for the sign test or Wilcoxon's test (Table 1). The χ^2 values for the Kolmogorov-Smirnov test for each of the comparisons for men and women were not significant (Table 1).

Smoking and Age vs Counts of Asbestos Bodies

Figures 2 and 3 indicate that asbestos bodies do not increase with age or pack-years of cigarettes smoked for male manual laborers. Comparison of counts of asbestos bodies and cigarettes smoked per day also failed to show a correlation.

In patients with asbestosis, the numbers of asbestos bodies varied from 8,000/gm to 520,000/gm in our samples (Table 5). In most of the cases, a limited amount of lung was available for analysis. Marked discrepancies in replicate counts were noted

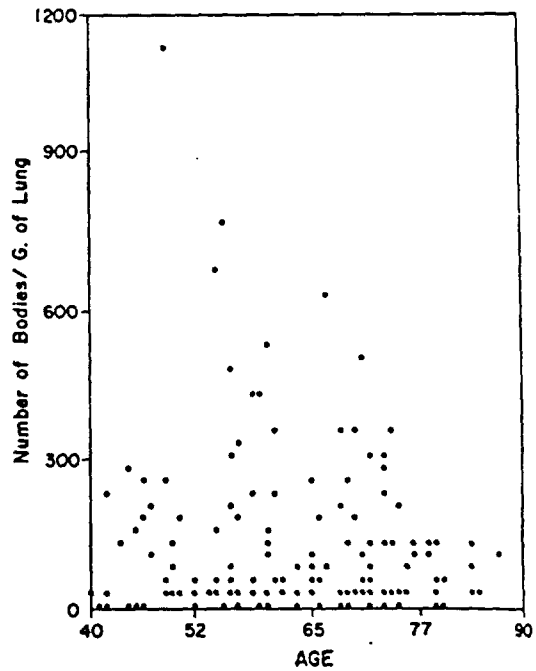

FIGURE 2. Relation of number of asbestos bodies per gram of lung to age of male manual laborers. There is no evidence for increase in numbers of asbestos bodies with age. Six patients with more than 1,200 asbestos bodies per gram of lung were excluded; all but two of these were less than 53 years old.

by Site

	Colon
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	10 (53)
	22 (44)

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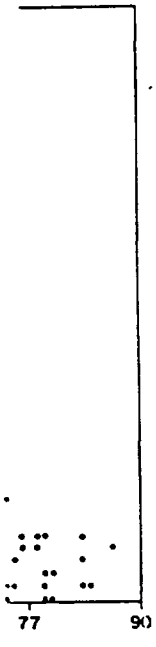

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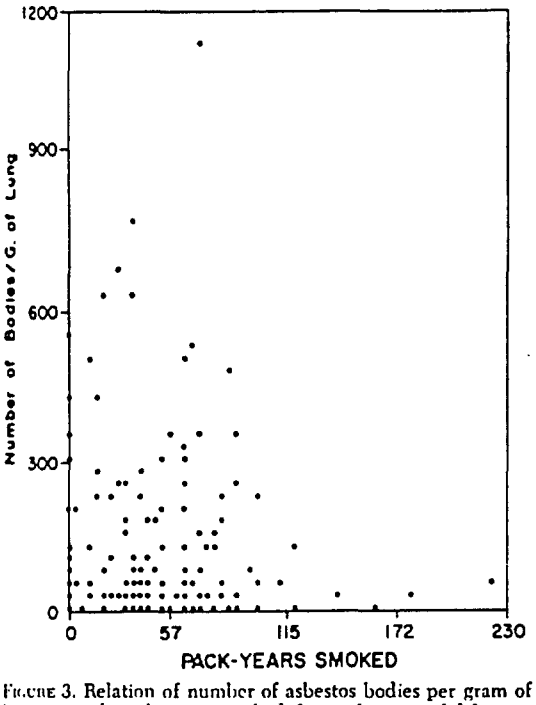

FIGURE 3. Relation of number of asbestos bodies per gram of lung to total pack-years smoked for male manual laborers. There is no evidence of increase in numbers of asbestos bodies with increasing pack-years smoked. Smokers with zero pack-years include pipe and cigar smokers. Six patients with more than 1,200 asbestos bodies per gram of lung were excluded; all smoked less than 60 pack-years.

in cases 5 and 6, suggesting that sampling is important in scarred lungs, where distribution and clearance may be abnormal. By contrast, in our sample population, only seven of our counts were over 800 asbestos bodies per gram; the vast majority of our patients had counts that were two or three orders of magnitude below counts in the patients with asbestosis.

DISCUSSION

In this study, we failed to find differences in the numbers of asbestos bodies between patients with lung or gastrointestinal cancers and matched controls. The lack of statistically significant differences between groups with cancer and control groups, the absence of large numbers of patients with cancer who had counts greater than the matched control subject, and the low median values for numbers of asbestos bodies in groups with cancer and control groups all indicate to us that, for practical purposes, asbestos bodies in this matched population are not associated with these cancers.

Table 5—*Numbers of Asbestos Bodies in Patients with Asbestosis*

	Asbestos Bodies per Gram of Lung	
	Sample 1	Sample 2
Patient 1	370,000	430,000
Patient 2	340,000	520,000
Patient 3	12,000	27,000
Patient 4	23,000	62,000
Patient 5	15,000	360,000
Patient 6	8,000	170,000

We did, however, find an association between occupation and counts of asbestos bodies; 60 percent of the construction workers, 41 percent of the steel workers, and 25 percent of the other male manual laborers had more than 100 asbestos bodies per gram of lung, whereas only 7 percent of men and women with white-collar occupations had more than 100 asbestos bodies per gram. Somewhat similar results have been obtained by Selikoff and Hammond[21] and by Doniach et al[22] using semiquantitative methods. In our study the number of asbestos bodies seems to be unrelated to smoking in male manual laborers (Fig. 3). Also, the numbers of asbestos bodies do not increase with age after 40 years (Fig 2), although others have found that such an increase does occur;[21] however, careful matching for age is still necessary because asbestos-induced neoplasms are related to the time since the onset of exposure, with a period of latency of 15 to 50 years between initial exposure and appearance of the cancer.

There are many epidemiologic studies on the incidence of lung cancer in workers in the asbestos industry. These have recently been reviewed by Becklake.[5] Several of these reports have attempted to grade the degree and length of exposure to asbestos; and, in general, an increased incidence of lung cancer was not found in persons in the groups with lowest exposure, even after long latent periods.[5] Since our patients almost certainly have less exposure to asbestos than these workers, our results are in keeping with the epidemiologic data.

The type and distribution of lung cancer in asbestos workers were examined by Kannerstein and Churg.[23] They studied lungs from 50 male patients with occupational exposure and lungs from 50 male control patients and found that there were no differences in histologic type, but the asbestos-associated tumors tended to occur more frequently in the lower lobes. In the present study, all tumors occurred more frequently in the upper lobes, quite the reverse of the distribution seen with asbestos-associated cancers.

Attempts to determine possible asbestos-related risk for workers in nonasbestos industries and for members of the general population have been rela-

tively few. In one of these studies, Whitwell et al[24] compared the numbers of asbestos fibers visible by phase-contrast microscopic examination in lungs of 100 patients with lung cancer, 100 control subjects, and 100 patients with mesothelioma and found that patients in the group with lung cancer and the control group had similar distributions of fiber counts. Whitwell et al[24] found little overlap between their control group and their patients with mesothelioma who had asbestosis, although there was considerable overlap of patients with mesothelioma who did not have asbestosis and controls. Most of the controls had lived in Liverpool, England, but 22 percent of the patients with lung cancer had lived in rural areas. No matching for occupation or place of residence was attempted, despite the fact that Whitwell et al[24] noted large discrepancies in counts according to occupation. They concluded that levels of exposure to asbestos that do not cause asbestosis are not etiologically related to lung cancer.

While the counts from many of our patients fall within the values cited by others, it is difficult to compare results from different laboratories, particularly since few details of sampling, preparation of tissue, and methods of counting are provided. In our laboratory, we have found that in lungs from the general population, two or three samples from the same lung yield a relatively narrow range of results (mean number of bodies ± 50 percent). In lungs from primary asbestos workers who have asbestosis, the range in a single lung is considerably wider and often approximates one order of magnitude (Table 5). Such discrepancies are probably related more to a nonuniform distribution in scarred lung, rather than to technical details; hence, one must be cautious about interpreting counts from only one or two areas in a scarred lung. Nevertheless, we have observed a very great separation between values found in the general population, only seven of which were over 800 asbestos bodies per gram, and those in asbestos workers.

We wish to emphasize that even a strong positive correlation between numbers of asbestos bodies and carcinomas would not, by itself, define an increased incidence of cancer in these patients; such a conclusion would require studies of incidence based on large populations with varying levels of asbestos bodies. Nevertheless, our negative findings should not yet be interpreted to mean that there is no danger to the general population from asbestos. In asbestos workers the number of uncoated fibers in the lung is far greater than the number of asbestos bodies; similarly, the number of fibers visible by light-microscopic examination in such workers is only a fraction of the number demonstrable by electron-microscopic examination.[5] Whether this is also true of the general population has not been determined.

When this study was begun, we had no information concerning the type of cores found in ferruginous bodies in the general population. We have now examined over 500 ferruginous bodies by electron optical techniques and have found that over 95 percent contain a core of amphibole asbestos;[11,12] we have also demonstrated that it is possible routinely to pick out those which do not contain asbestos by simple light-microscopic examination.[12] Thus, there is no doubt that the counting of asbestos bodies in lungs of the general population serves as one marker of exposure to asbestos; however, it is known that uncoated asbestos fibers (generally chrysotile asbestos) too small to form asbestos bodies may be found in most lungs[25] and that most of the asbestos used in this country is chrysotile, rather than amphibole. Therefore, in order to establish an association between the total pulmonary burden of asbestos and carcinoma of the lung in the general population, it will be necessary to measure total asbestos fibers in a controlled study similar to this one.

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References

1 Vincent RG, Pickren JW, Lane WW, et al: The changing histopathology of lung cancer: A review of 1,682 cases. Cancer 39:1647-1655, 1977

2 Mason TJ, McKay FW, Hoover R, et al: Atlas of Cancer Mortality for U.S. Counties: 1950-1969 (publication NIH 75-780). US Department of Health, Education, and Welfare, 1976

3 Brown SM, Selvin S, Winkelstein W: The association of economic status with the occurrence of lung cancer. Cancer 36:1903-1911, 1975

4 Selikoff IJ, Nicholson WJ, Langer AM: Asbestos air pollution. Arch Environ Health 25:1-13, 1972

5 Becklake MR: Asbestos-related diseases of the lung and other organs: Their epidemiology and implication for clinical practice. Am Rev Respir Dis 114:187-227, 1976

6 Selikoff IJ, Hammond EC, Seidman H: Cancer risk of insulation workers in the United States. *In* Bogovski P, Gilson JG, Timbrell V, et al (eds): Biological Effects of Asbestos (publication No. 8). Lyon, France, IARC Scientific Publications, 1973, pp 209-216

7 Nicholson WJ: Analysis of amphibole asbestiform fibers in municipal water supplies. Environ Health Perspect 9:165-172, 1974

8 Rohl AN, Langer AM, Selikoff IJ: Environmental asbestos pollution related to use of quarried serpentine rock. Science 196:1319-1322, 1977

9 Nicholson WJ, Maggiore CJ, Selikoff IJ: Asbestos contamination of parenteral drugs. Science 177:171-173, 1972

10 Gross P, deTreville RTP, Cralley LJ, et al: Pulmonary ferruginous bodies. Arch Pathol 85:539-546, 1968
11 Churg A, Warnock ML: Analysis of the cores of ferruginous (asbestos) bodies from the general population: 1. Patients with and without lung cancer. Lab Invest 37:280-286, 1977
12 Churg A, Warnock ML, Green N: Analysis of the cores of ferruginous (asbestos) bodies from the general population: 2. True asbestos bodies and pseudo asbestos bodies. Lab Invest 40:31-38, 1979
13 Nizze H: Exposure to asbestos and the genesis of pleural plaques and neoplasia. Arch Pathol 95:213-214, 1973
14 Breedin PH, Buss DH: Ferruginous (asbestos) bodies in the lungs of rural dwellers, urban dwellers and patients with pulmonary neoplasms. South Med J 69:401-404, 1976
15 Anjilvel L, Thurlbeck WM: The incidence of asbestos bodies in the lungs at random necropsies in Montreal. Can Med Assoc J 95:1179-1182, 1966
16 Warnock ML, Churg AM: Association of asbestos and bronchogenic carcinoma in a population with low asbestos exposure. Cancer 35:1236-1242, 1975
17 Churg AM, Warnock ML: Correlation of quantitative asbestos body counts and occupation in urban patients. Arch Pathol Lab Med 101:629-634, 1977
18 Smith MJ, Naylor B: A method for extracting ferruginous bodies from sputum and pulmonary tissue. Am J Clin Pathol 58:250-254, 1972
19 Churg A, Sakoda N, Warnock ML: A simple method for preparing ferruginous bodies for electron microscopic examination. Am J Clin Pathol 68:513-517, 1977
20 Siegel S: Nonparametric Statistics for the Behavioral Sciences. New York, McGraw-Hill Book Co, 1956, pp 68-83, 127-136
21 Selikoff IJ, Hammond EC: Asbestos bodies in the New York City population in two periods of time. *In* Shapiro HA (ed): Pneumoconiosis: Proceedings of the International Conference, Johannesburg, 1969. London, Oxford University Press, 1970, pp 99-105
22 Doniach I, Swettenham KV, Hathorn MKS: Prevalence of asbestos bodies in a necropsy series in East London: Association with disease, occupation, and domiciliary address. Br J Ind Med 32:16-30, 1975
23 Kannerstein M, Churg J: Pathology of carcinoma of the lung associated with asbestos exposure. Cancer 30:14-21, 1972
24 Whitwell F, Scott J, Grimshaw M: Relationship between occupations and asbestos-fibre content of the lungs in patients with pleural mesothelioma, lung cancer, and other diseases. Thorax 32:377-386, 1977
25 Langer AM, Selikoff IJ, Sastre A: Chrysotile asbestos in the lungs of persons in New York City. Arch Environ Health 22:348-361, 1971