Archives of Environmental Health: An International Journal
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/vzeh20

Chrysotile Asbestos in the Lungs of Persons in New York City

Arthur M. Longer PhD [a], Irving J. Selikoff MD [a] & Antonio Sastre [a]
[a] Environmental Sciences Laboratory, Mount Sinai School of Medicine of the City University, New York, USA
Published online: 29 Apr 2013.

To cite this article: Arthur M. Longer PhD, Irving J. Selikoff MD & Antonio Sastre (1971) Chrysotile Asbestos in the Lungs of Persons in New York City, Archives of Environmental Health: An International Journal, 22:3, 348-361, DOI: 10.1080/00039896.1971.10665854

To link to this article: http://dx.doi.org/10.1080/00039896.1971.10665854

Chrysotile Asbestos in the Lungs of Persons in New York City

Arthur M. Langer, PhD; Irving J. Selikoff, MD; and Antonio Sastre, New York

When inhaled, chrysotile tends to split into unit fibrils 200 to 400 Angstroms in diameter, invisible with optical microscopy. Further, it is altered chemically and physically in vivo. Therefore, attempts to identify unaltered chrysotile in the core of "asbestos bodies" have many pitfalls, especially when such attempts are limited by optical microscopy. High-magnification electron-microscopic examination of representative small samples of lung unequivocally showed chrysotile asbestos to be present in 24 of 28 consecutive New York city autopsy cases. Our data demonstrate that chrysotile fibers and fibrils are present in the lungs of New York city residents. Similar observations have been made in London. We anticipate that what is now known for New York and London will be found in other cities as well.

Asbestos Bodies: Their Nature and Occurrence.—Soon after the recognition of asbestosis, examination of lung tissue obtained from individuals with this disease demonstrated the presence of coated fibers which were first termed "curious bodies"[1,2] and, subsequently, "asbestos bodies."[3] Since these first individuals had been heavily exposed to asbestos at their work, it was assumed, undoubtedly with good reason, that the central fibrous core of these unusual structures was asbestos.

Submitted for publication May 22, 1970; accepted June 26.

From the Environmental Sciences Laboratory, Mount Sinai School of Medicine of the City University of New York.

Reprint requests to Environmental Sciences Laboratory, Mount Sinai School of Medicine, Fifth Ave and 100th St, New York 10029 (Dr. Selikoff).

When what appeared to be identical structures were next found in lungs of people not known to be occupationally exposed, the "asbestos-core" assumption was applied to these as well. It is of interest that the first description of such nonoccupational bodies in 1929 was made after examination of the lung tissue of a person who had lived across the road from an asbestos plant.[4]

In recent years a number of investigations, starting with the Capetown study of Thomson et al,[5] have demonstrated the presence of what appear to be "asbestos bodies" in the lungs (both in tissue sections and in expressed tissue fluid) of a large proportion of urban dwellers who had no recorded occupational exposure to asbestos. The percentage of instances in which they are found varies with the technique used in the investigation,[6] the amount of lung tissue examined, and the diligence and persistence with which they are sought. The percentage of positive findings has been reported to be as high as 97%[7] or 100%.[8]

The question whether the cores of the "asbestos bodies" observed in these nonoccupationally exposed individuals are necessarily asbestos has been revived. Although "asbestos bodies" occurring in the general population appear morphologically identical to asbestos bodies observed in asbestos workers, this does not guarantee that they necessarily have the same core. For epidemiological studies it would be useful to have unequivocal identification of the fibrous core.[9] This has not been easily accomplished because of technical factors.[10]

The possibility that what appear to be "asbestos bodies" may have other than an asbestos core has been accepted almost as long as asbestos bodies have been known,[11] especially when exposure to asbestos is not documented. When other exposures are identified, appropriate designations are useful ("talc bodies," "graphite bodies," "fibrous glass bodies," etc). When exposure is not documented, one can consider implicit the bodies' nonspecificity[12,13] or even use a descriptive term which is noncommittal, as "ferruginous body."[14] In such circumstances, the uncertainty concerning the nature of the fibrous core of random "asbestos bodies" has hampered evaluation of the significance of their demonstration in the lungs of individuals in the general population. Do they or do they not reflect nonoccupational community asbestos exposure?

Investigations have been pursued to obtain further information on the problem. These investigations have taken two directions: first, the study of asbestos bodies by more refined analytical techniques, as electron microprobe analysis[15,16] or electron diffraction[17]; second, the appreciation that the unique morphology of chrysotile asbestos allows its specific identification by high-magnification electron microscopy. Both lines of study have centered on chrysotile, since more than 90% of asbestos used in the United States is of this variety, and both have sought to answer the question: is chrysotile asbestos to be found in the lungs of individuals not known to be occupationally exposed to asbestos, and, if it is, how frequently?

It was recently reported that analyses were made of 28 asbestos bodies micromanipulated from the lungs of 28 individuals in the general population of Pittsburgh.[17] It was stated that chrysotile was not found in any instance, and it was inferred that asbestos contamination of urban air was unlikely to be responsible for the formation of the asbestos bodies. Unfortunately, since few details were given, it is difficult to fully evaluate the report.

We have investigated the problem in another way and come to another conclusion; chrysotile was specifically identified in lung tissue in 24 of 28 consecutive cases examined at autopsy in New York city. This suggests that it is commonly present in the lungs of urban dwellers at this time.

Mineral Fibers: Their Occurrence in Lung, Recovery, and Species.—It has been frequently noted that there is lack of correspondence between the ease of recovery of chrysotile asbestos from the lungs of experimental animals and the difficulty experienced in its recovery from the lungs of humans occupationally exposed to this mineral. This observation may be explained by a number of interrelated factors.

The first factor is the amounts of asbestos involved. In a series of experiments by W. E. Smith, MD, and associates (unpublished data), 25 mg of chrysotile asbestos were injected intratracheally into golden Syrian hamsters. Six hundred and fifty-one days later, some of the animals were killed and lung tissue forwarded to our laboratory for mineralogical analysis. The tissue was ashed in a low-temperature ashing device, and the unashed residue was water dispersed and examined by means of polarized light microscopy and electron microscopy. Everywhere within the partially digested tissue debris, chrysotile fibers were observed. The optical properties remained remarkably similar to the natural material before injection; asbestos bodies tended to form about the thinner fiber bundles. By electron microscopy (Fig 1, *A*), a large number of thick fibers were found that were not broken open into individual fibrils. Several of the larger fibers demonstrated incipient coatings. Electron diffraction patterns obtained on the thicker fibers (Fig 1, *B*) indicated that the chrysotile had apparently not altered structurally. High-magnification examination of the fibers with the electron microscope demonstrated the ultimate structure in the form of the internal capillary (Fig 1, *C*).

The recovery of chrysotile from experimental animals is technically simple. The 25-mg dose injected into the lungs of a hamster of a young or a modest age (50 to 100-gm range of animal weight) is massive. Considering the weight of a hamster and its ratio of lung weight, the amount of chrysotile injected would equal several percent of its lung weight. (This would correspond to huge amounts of chrysotile asbestos in the lungs of humans, in the order of 4 to 5 gm per human lung.) This massive dose, cou-

pled with relatively short-term biological residence, insured recovery of characteristic fibers.

In contrast to experimental animal studies, the amount of asbestos recovered from lungs of persons occupationally exposed to the fiber has ranged from reported values of 0.6% to 0.001% of lung weight.[18,19] Nagelschmidt noted[20] that the recovery of chrysotile asbestos from lung tissue is always low and recovery of amphibole asbestos generally high for human cases.

The second reason for the difficulty in extracting unaltered chrysotile fibers from human lungs lies in the fact that the nature of chrysotile asbestos is such that it tends to break down chemically and physically after prolonged biological residence. This is especially true of chrysotile fibers that have separated into their fine unit fibrils.

Chrysotile asbestos is chemically unstable even in distilled water.[21,22] An aqueous solution with a pH below 10.8 will cause the fiber to be leached of some of its magnesium. Chemical instability in a biological environment has been demonstrated several times. Morgan and Holmes,[23] following the "daughter" decay products of radioactive chrysotile asbestos, showed that magnesium was rapidly leached from the chrysotile fibers while in biological residence. This was also indicated by the work of Langer et al.[10] Electron microprobe studies of chrysotile asbestos fibers removed from the lungs of hamsters indicated a wide range of magnesium values in contrast to the narrow range in the fibers before injection. Similar results have been obtained in analyses of fibers and bodies removed from lung tissue of a Canadian chrysotile miner.

These findings are consistent with optical studies previously reported,[20,24] in which chrysotile asbestos removed from the lungs of workmen exposed to this fiber was found not to possess the same optical characteristics as did the materials to which they were exposed. Rather, the findings reflected chemically degraded fibers.

Marked physical changes in chrysotile may also take place in vivo. The chrysotile fiber composed of bundles of fibrils—breaks open, and the individual unit fibrils are separated. This has been demonstrated by Suzuki and Churg.[25] No such observations have been made with amphibole asbestos types, eg, amosite.[10]

This chemical and physical instability of chrysotile has made the quantitative extraction of asbestos fibers from the lungs of exposed individuals a problem that has not yet been completely solved. (See Nagelschmidt[20] for discussion.) As an alternative, it has been considered useful to at least obtain those fibers which had become coated (the "asbestos bodies"). While there are no data to quantitatively relate the presence of such asbestos bodies to the remainder of the asbestos present, it has been thought that it may at least reflect, to an undetermined degree, the presence of asbestos in general. Moreover, individual asbestos bodies can be removed for analysis of their coating and, more germane to the current problems, of their central, fibrous cores.

A useful method to obtain individual asbestos bodies for study is to isolate them by micromanipulation.[6] It should be noted, however, that micromanipulation must be done under the optical microscope, generally using low magnification. This inevitably produces a biased selection of large asbestos bodies from lung tissue,[10,16,26] since by definition the only bodies that can be manipulated are those that can be seen at such low magnifications! Micromanipulation of asbestos bodies precludes small asbestos bodies from analysis. Micromanipulation of asbestos bodies from human lung tissue, therefore, leads to particle selection. On the basis of the physical and chemical differences observed experimentally and determined theoretically for chrysotile and amphibole types, it may be concluded that the large recoverable fibers and bodies, selected by using the optical microscope, are more likely to be amphibole asbestos than unaltered chrysotile.

Timbrell et al[27] observed that what is seen by light microscopic methods often differs from what may be observed by electron microscope methods and that respirable-size fractions need not have the same size distribution or properties as those observed in the gross overall sample. Similarly, Lynch and Ayer[28] in describing the inadequacies of the impinger method of particle counting, concluded that only about one in 100 fibers present in the air could be seen by light

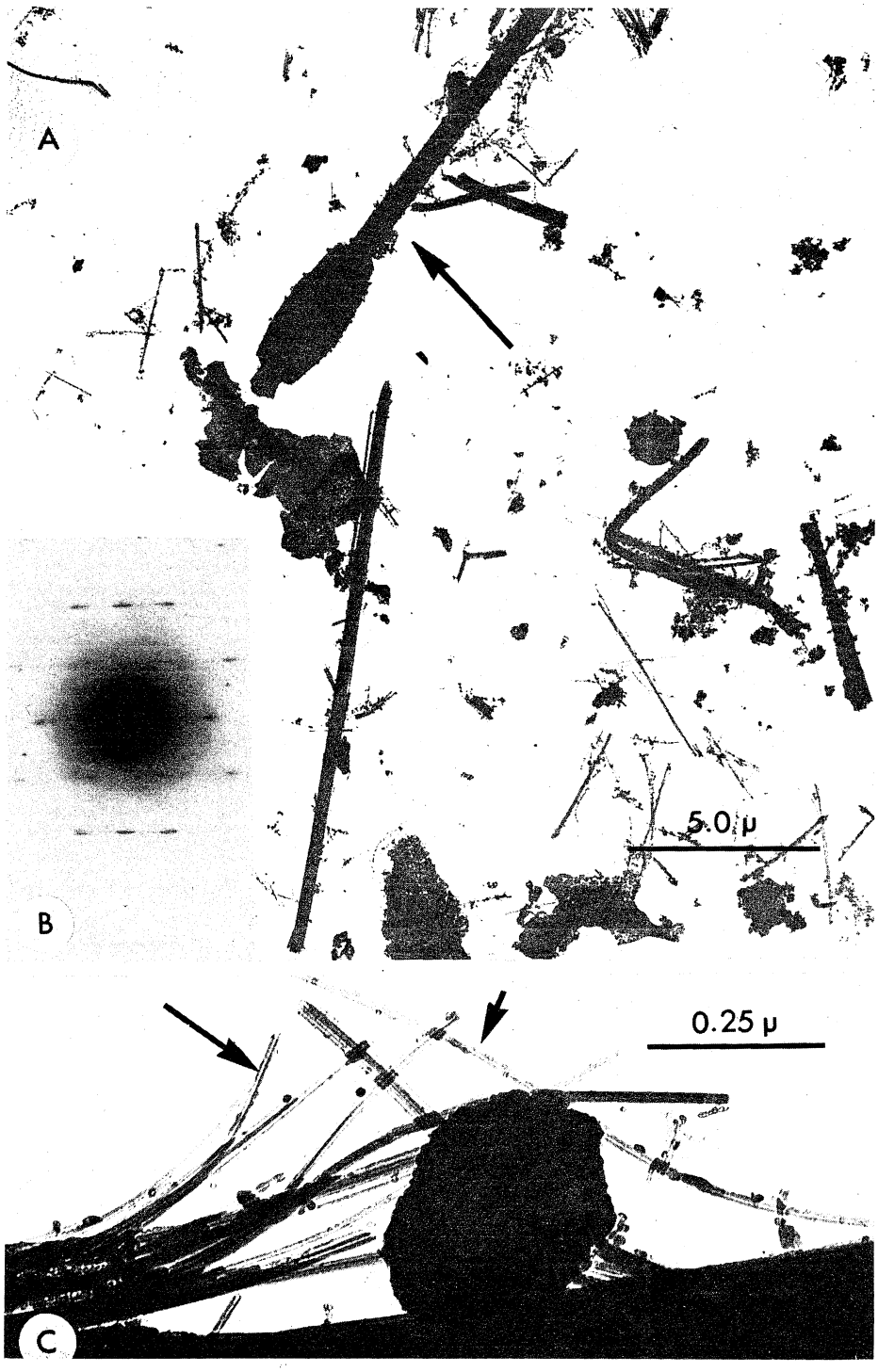


Fig 1.—Chrysotile extracted from hamster lung. Size range is smaller than light microscopy shows; fiber bundles are broken open (**A, C**). Chrysotile electron diffraction patterns (**B**) are obtainable. **Arrow (A)** indicates where on asbestos body area was selected for diffraction; **arrows (C)** show characteristic capillaries.

Table 1.—*Epidemiological Data on 28 Studied Cases*

Case	Age	Sex	Occupation	Cause of Death	Asbestos Exposure	Comments
1	66	F	Domestic	Polycystic kidneys	0	Used asbestos ironing pads
2	70	M	Truck driver	Lung cancer	0	...
3	45	M	Home repairs	Glomerulonephritis	Some used in repairs	Lived near shipyard
4	82	M	Tailor	Arteriosclerotic heart disease	0	...
5	80	M	Linen supplier	Colon cancer	0	Asbestos ironing board covers
6	53	M	Bartender	Hypernephroma	0	Construction laborer, 1940's
7	75	M	Porter, office	Hypertension	0	...
8	27	M	Computer operator	Melanoma, eye	0	...
9	46	M	Appliance factory	Sepsis	0	...
10	48	M	Plumber	Chronic glomerulo-nephritis	Material in trade	Oil burner repairs
11	61	M	Attorney	Reticulum cell sarcoma	0	...
12	77	M	Lens polisher	Pancreatic cancer	0	Home repairs, asbestos cement
13	81	M	Tailor	Pancreatic cancer	0	Asbestos ironing board covers; lived near shipyard
14	50	F	Broker	Myocardial infarction	NA*	...
15	48	F	Housewife	Myocardial infarction	0	...
16	61	F	Housewife	Lung cancer	0	...
17	68	M	Chauffeur	Heart block	0	...
18	80	F	Office secretary	Myocardial infarction	0	...
19	42	F	Grocery clerk	Cirrhosis of the liver	0	Asbestos ironing board covers
20	59	F	Bookkeeper	Myeloid metaplasia	0	Riveter, WW II; asbestos gloves
21	79	F	Housewife	Bronchopneumonia	NA*	...
22	58	F	Stenographer	Stroke	0	Asbestos ironing board covers
23	76	F	Housewife	Myocardial infarction	0	...
24	75	F	Seamstress	Glomerulonephritis	0	Once lived near yard with building materials
25	45	M	Musician	Aortic insufficiency	0	...
26	72	M	Salesman	Metastatic carcinoma, primary site not determined	0	...
27	64	F	Housewife	Cryptococcal meningo-encephalitis	0	Asbestos ironing board covers
28	76	M	Dress manufacturer	Myocardial infarction	0	...

* NA, not available.

microscopic methods. This was determined by comparison of light microscopic samples with the same material studied by electron microscopy.

Asbestos and Asbestos Bodies.—Thus, several considerations explain the difficulty of demonstrating chrysotile asbestos in human lung tissue if one is limited to those particles visible by optical microscopy: (1) Chrysotile tends to break into fine fibrils, often smaller in diameter than 0.5μ, the limit of resolution of the light microscope, and so not visible by this instrument. (2) It may be inhaled as fibrils or fibers; the latter, once inhaled, may separate into the unit fibrils and thus become "invisible." (3) Chrysotile's chemical and physical nature makes it subject to attack in tissue fluids, in contrast to amphibole asbestos. These factors make for "survivor populations" of the latter when fibers are extracted from human lung. All in all, without the electron microscope, one must feel insecure when searching for chrysotile asbestos in lung tissue, and techniques based upon light optical microscopy are studded with pitfalls.

In summary, chrysotile asbestos is unlikely to remain as large fiber bundles in man. "Asbestos bodies" in man, that are easily visible in the light microscopic size range, are not likely to be chrysotile nucleated or, if so nucleated, not to be unaltered chrysotile. If

Table 2.—*Comparison of Particle Counts Obtained by Light and Electron Microscopy*

	Optical Microscopy		Electron Microscopy					
	Asbestos Bodies			Chrysotile		Other Fibers		
Case	O_2 Ashing	KOH Digestion	Asbestos Bodies	Fibers	Fibrils	Thick*	Thin*	Platy Particles (Clays, Talc)
1	3	0	1	14	40	7	1	+
2	1	0	0	0	9	15	0	0
3	0	0	0	0	4	4	1	0
4	3	0	0	1	2	31	0	0
5	3	4	0	1	15	1	2	0
6	0	0	0	9	203	0	4	+
7	0	0	0	1	132	0	1	+
8	3	2	0	36	308	13	10	+
9	0	0	0	0	37	63	0	0
10	1	32	0	1	24	11	0	0
11	9	3	1	8	99	43	7	+
12	1	0	0	5	114	14	11	0
13	0	2	0	3	17	0	0	+
14	0	0	0	5	74	4	1	+
15	1	2	0	2	55	9	4	+
16	0	1	0	6	64	6	2	0
17	0	2	0	2	55	2	0	+
18	0	0	0	1	15	4	5	+
19	1	0	0	0	7	2	2	0
20	1	5	1	5	23	0	3	0
21	0	0	0	9	14	3	1	+
22	2	1	0	3	18	8	2	+
23	0	0	0	6	10	25	3	+
24	0	4	0	5	45	2	0	+
25	1	4	0	16	254	7	6	+
26	0	1	0	3	23	9	0	+
27	1	0	0	4	77	1	0	+
28	0	0	0	18	168	13	3	+

* Thick fibers, those with length:diameter ratio < 10:1; thin fibers, length:diameter ratio > 10:1.

Table 3.—*Distribution of Chrysotile in 28 Cases Studied*

Group	No. of Chrysotile Fibers and Fibrils	Cases	Men	Women
1	≦ 9	4	3	1
2	10-50	11	5	6
3	51-99	6	1	5
4	100-200	4	4	0
5	≧ 201	3	3	0
"Blank grids"	≦ 9	...	...	...

we are to seek chrysotile, it would appear necessary to include a search at a submicroscopic level.

Present Study

Our laboratory has been concerned with an investigation of 3,000 consecutive deaths in New York city.[9] Of this number, approximately 45% have been found to have what appear to be asbestos bodies in their lungs. A still larger proportion had "uncoated" fibers present (I. J. Selikoff, MD, unpublished data). This is in keeping with our observation that asbestos bodies represent a small proportion of fibrous particles present in the lung tissue of asbestos workers.

For the present investigation, 28 consecutive cases were chosen (Table 1). All had been longtime residents of New York city; none had been asbestos workers. In each, the lungs had been frozen at autopsy. From each, inorganic materials were extracted for study.

This investigation was undertaken to determine the presence or absence of submicroscopic uncoated asbestos fibers in the lungs of urban dwellers who died in New York city. Comparison of light microscopic

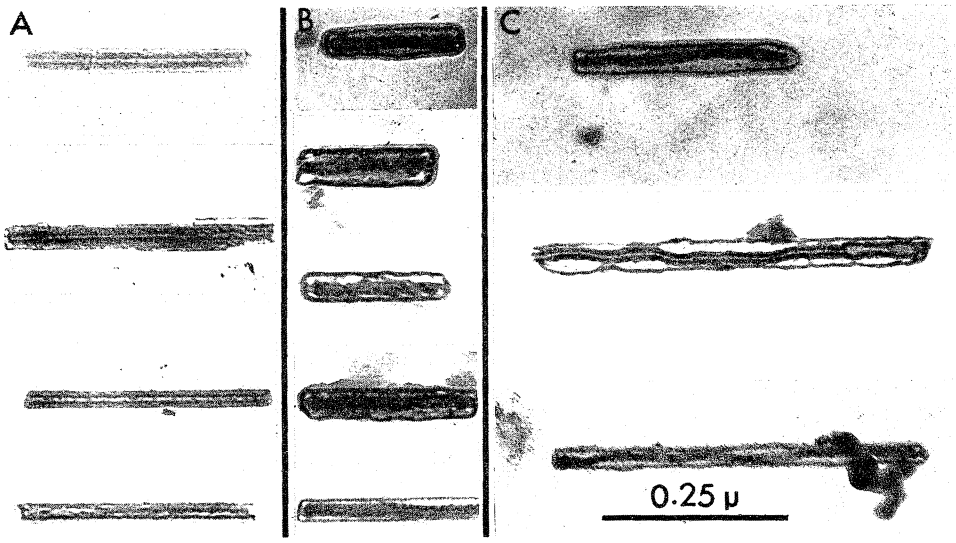


Fig 2.—Chrysotile morphology range in recovered mineral dusts. Rare well-formed fibrils with undeformed capillaries and thick, electron-dense walls without amorphous coatings, (**A**); more often, electron-dense wall encapsulated in amorphous coating (**B**); very often, fibrils with deformed internal capillaries and thin crystalline walls encapsulated in thick amorphous covering (**C**).

findings with those by electron microscopy was made. Study was further undertaken to determine whether the fibers found were or were not chrysotile asbestos.

Separation Techniques and Preparation.—From each of the 28 frozen lungs, 1 cc of tissue was cut and placed in a thick-walled centrifuge tube. A 40% KOH solution was added to the tube until the solution entirely covered the lung specimen (3 ml required). The centrifuge tubes were then placed in a hot water bath, and the water heated to boiling. Digestion was carried out for one hour starting from the time the water began to boil. After one hour very little lung residue remained in most instances. However, in some it was necessary to continue the digestion for an additional hour. It was found that this was sufficient to complete the digestion. The tubes were centrifuged at 15,000 to 17,000 rpm for approximately 30 minutes (head design indicates $F > 30{,}000$ g). The supernatant was next decanted, and the residue washed with distilled water. Washing, centrifugation, and decantation were repeated 3 times until the residue was free of KOH. Examination of the supernatant with a polarized light microscope indicated that no optically visible asbestos bodies or fibers were present in the supernatant after any of the spinning periods.

The residues were examined by polarized light microscopy. The mounting medium used in all cases was a highly viscous liquid which minimized particle migration[29]; scanning was commonly done under 250X, 400X, and 500X magnification. High-magnification examination was occasionally undertaken at 1,000X. Each of the 28 cases examined microscopically showed some residual undigested organic materials present, despite the apparently "complete" digestion.

Tissue extracts were also prepared for study by electron microscopy. Small splits (approximately 1 mg) of the washed residues from the 28 specimens were pipetted into 28 smaller test tubes. Each of the latter 28 test tubes was filled with 2 ml of distilled water. The residue and medium were agitated (for dispersal) for 30 seconds. Small proportions of the dispersant residues were removed, and one drop pipetted onto a polyvinyl methylal (Formvar) coated 200-mesh copper electron microscope (EM) locator grid. The drop of water was allowed to remain quietly on top of the grid for 15 minutes to allow the settling of solid materials. At the end of that time, the liquid drop was drawn off with wetted filter paper.

The amount of material that actually settled out in this time onto the polyvinyl methylal grid was invisible to the unaided

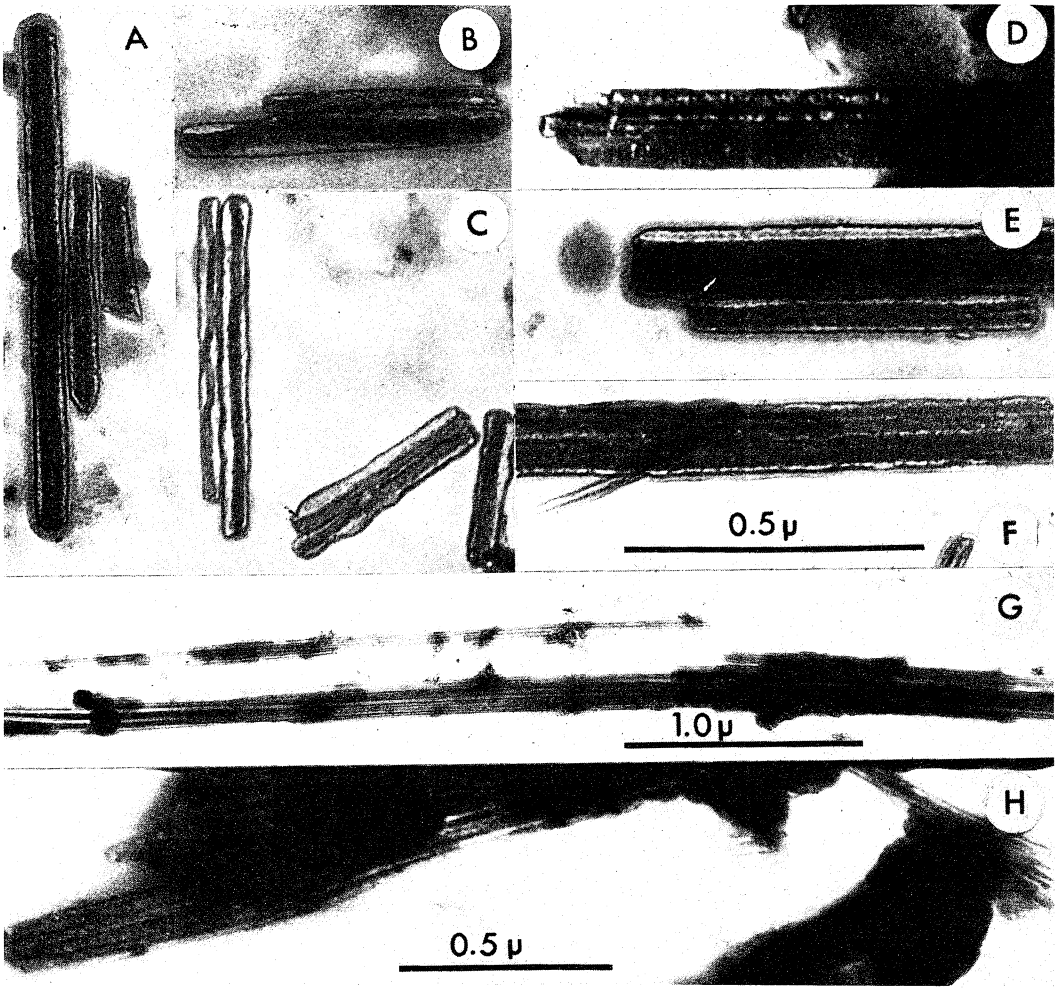


Fig 3.—Chrysotile fiber size range (thickness, length); fibers several fibrils thick (A to F) to fibril bundles (G, H). Fibrils range from well-formed (A) to highly deformed (C). Fiber length ranges from several thousand Angstroms to several microns.

eye. Only 9 of 248 squares of each grid were examined under the EM beam. The technique used in this investigation provides an answer to the qualitative question: is chrysotile a ubiquitous contaminant of the tissue examined? It is evident that only an infinitesimal fraction of lung tissue was scanned (conservatively estimated at 10^{-6} of the total lung burden). Quantitative studies are in progress; these will be useful for epidemiological investigations concerned with the biological significance of the current qualitative findings.

Instrumentation.—Identification and location of asbestos fibers and fibrils required the use of the highest magnification available on the electron microscope (RCA EMU 3G). A fibril is defined as the smallest characteristic fiber unit of chrysotile asbestos, with a thickness of 200 to 400 Angstroms. Scanning was done at magnifications of 31,000X with an acceleration voltage of 100 kv. Identification of chrysotile was confirmed using the 7X binocular attachment, or at 217,000X.

Identification Criteria.—In order to justify identification of chrysotile asbestos as it appears after extraction from lungs, it was necessary to review and extend information concerning the morphological nature of chrysotile. The range of morphological characteristics of chrysotile was studied in considerable detail, and the findings are described

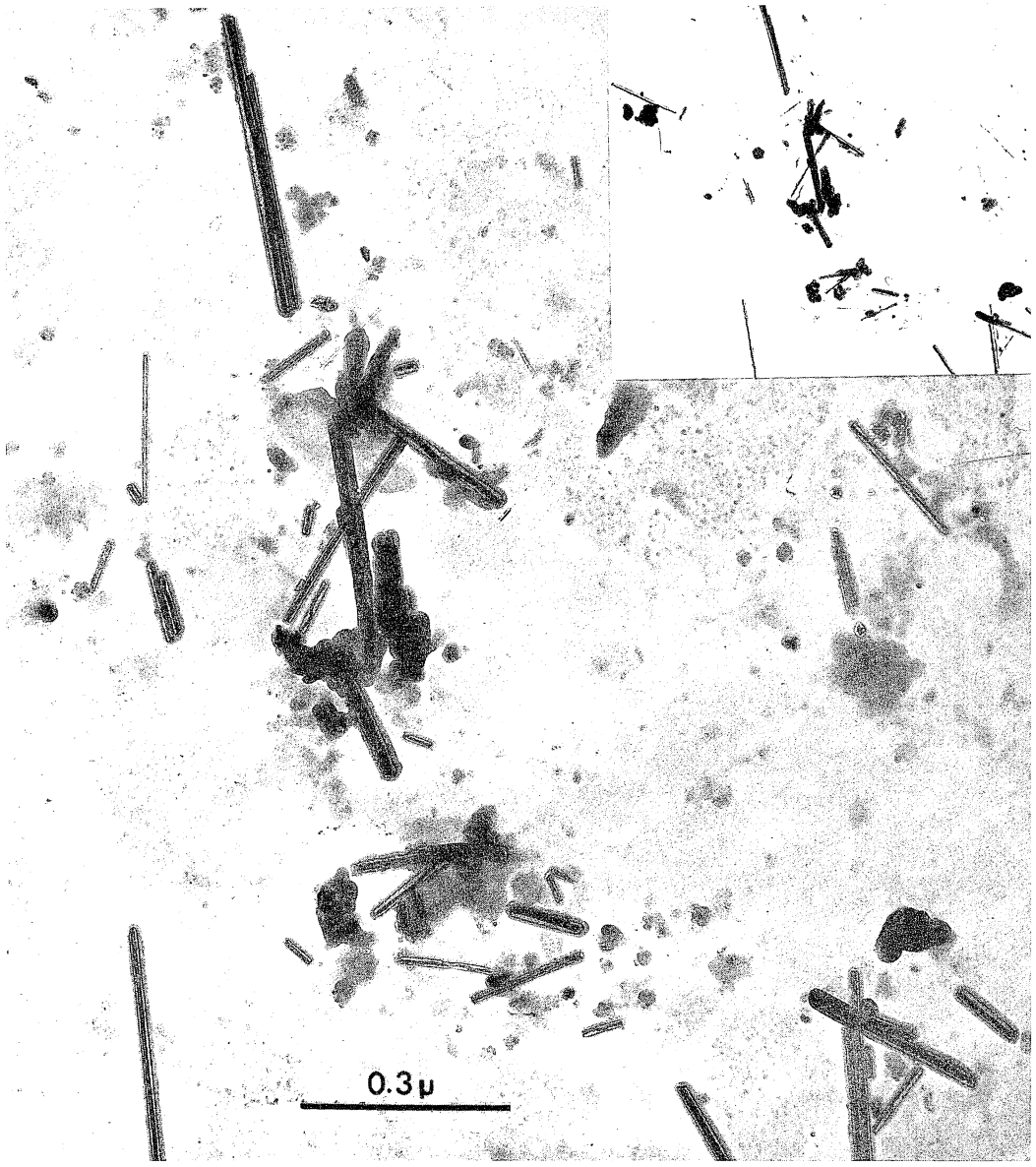


Fig 4.—Chrysotile field at magnification as on EM viewing screen (insert); and photographic enlargement. Even with photographic plate's high contrast, lower-magnification count, only 45 fibers and fibrils were counted; enlargement shows 59 actually present.

elsewhere (A. M. Langer, PhD, unpublished data).[30]

Background Problems.—We have observed that polyvinyl methylal grids which were scanned at the low magnification of 20,000X were void of "background" fibrils. However, other "blank" grids, scanned at higher magnifications of 31,000X were found to sometimes contain a background of single, isolated chrysotile fibrils. The range of values observed in these control grids was zero to two fibrils per field, and no more than nine background fibrils for nine fields per grid scanned.

Observations

Lung Residues.—*Light Microscopy.*—The results of optical microscopic scanning for the 28 cases are given in Table 2. Two preparation techniques are compared, KOH

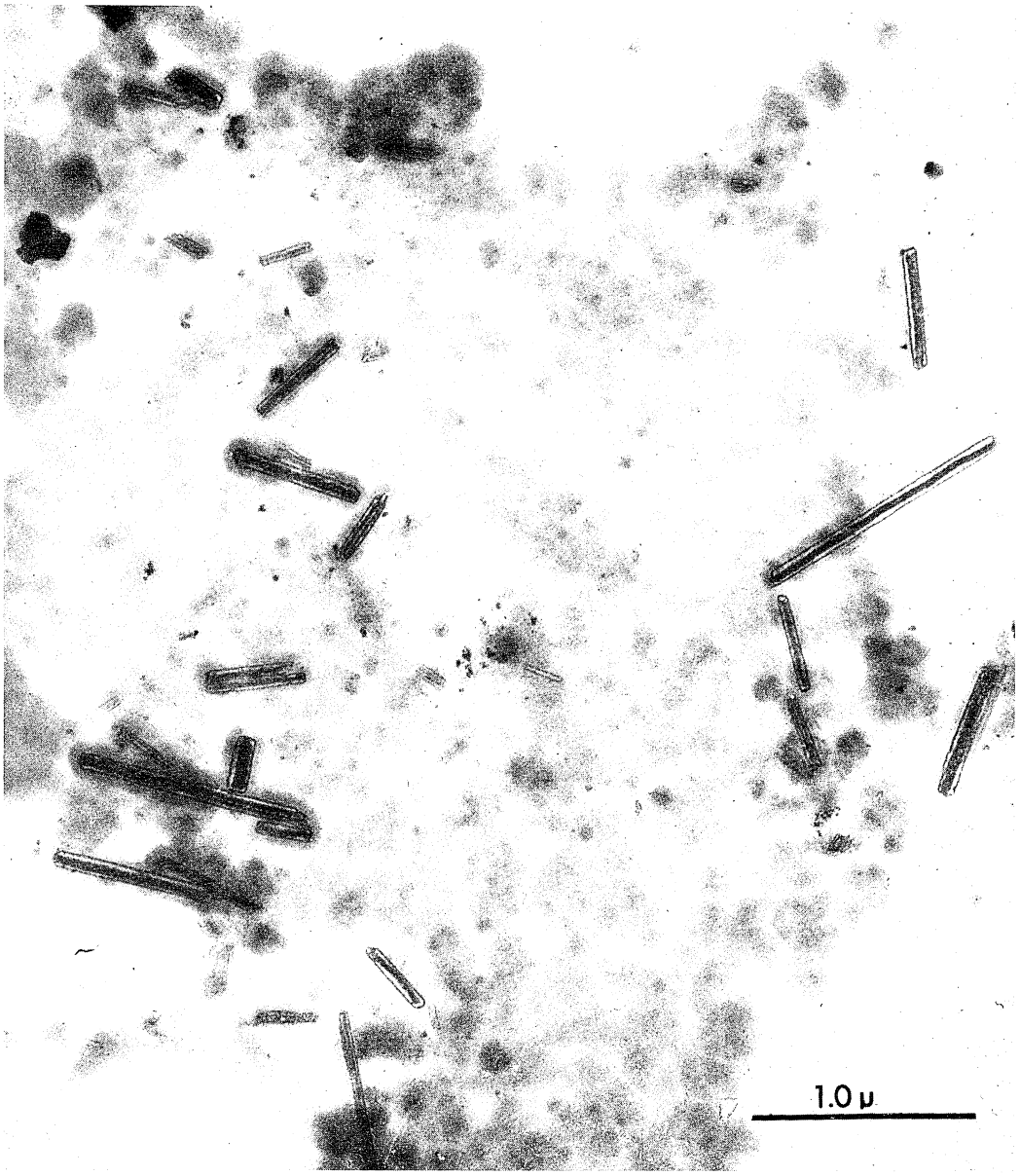


Fig 5.—Chrysotile field in partially digested tissue. Although tissue was electron transmissive, count obtained on screen was 20 compared with count of 31 on photograph. All short and thin fibrils were "missed" on viewing screen.

and ashing (Table 2). A comparison of asbestos body count obtained with the low temperature ashing technique and the KOH digestion method shows 14 of 28 positive by the ashing technique and 13 of 28 positive by the KOH technique. However, agreement was present in only eight cases of both group totals. Combining both techniques, 19 of 28 cases, or 68% of the cases, were positive for definite asbestos bodies. Of the eight in agreement, the number of bodies obtained with the KOH technique was higher in five of the eight cases. These observations were expected because the amount of tissue sampled in each instance differed (1,000 cu mm, KOH; 17.5 cu mm, ashing); different portions of the same lung were examined for each technique.

Lung Residues.—*Electron Microscopy.*—A number of substances were observed to be present on the prepared grids: chrysotile asbestos (fibrils and fibers); polygonal platy particles (clay and talc minerals); electron-dense fibrous particles (resembling amiphibole asbestos minerals); fibrous glass; asbestos bodies; and diatom fragments. The occurrence of chrysotile in our cases is shown in Table 3.

There are characteristic morphological appearances to the chrysotile particles found in human lung tissue. The fibrils range from those with relatively undeformed internal capillaries, thick crystalline electron-dense walls, and thin amorphous edges (Fig 2, *A*) to others with thinner crystalline walls surrounding slightly deformed capillaries which are in turn surrounded by thicker edges of amorphous material Fig 2, *B*. Some fibrils are highly deformed and are encapsulated by thick-walled amorphous material (Fig 2, *C*). Many of the fibrils observed appear deformed. The size of the fibrils found in human lung range from several hundred Angstroms to several microns in length. The average size of fibrils observed from human lung tissue appears to be 0.2μ to 0.3μ in length.

We have also found chrysotile fibers to be present. A fiber is defined as any chrysotile aggregate greater than one fibril in thickness. The range in morphological characteristics of the fibers may be seen in Fig 3. It is of interest to note that some of the morphological characteristics of the fibers are similar to those described by Suzuki and Churg in their study of the origin of asbestos bodies in hamsters.[25] Tables 2 and 3 demonstrate that 24 of the 28 cases had asbestos fibers in their lungs in numbers higher than background counts could explain. Those with high fiber counts had high fibril counts as well. Three of the four "negative" cases were observed by light microscopy to possess asbestos bodies (Table 2).

Chrysotile fibers and fibrils tend to occur in fields. Fibrils and small thin fibers, which apparently consist of only two or three unit fibrils still bonded, predominate the fields.

We found only three cases to contain more than 200 chrysotile fibers and fibrils in the grids studied by us (Tables 2 and 3). It may be of interest that we have observed these highly positive cases only among men.

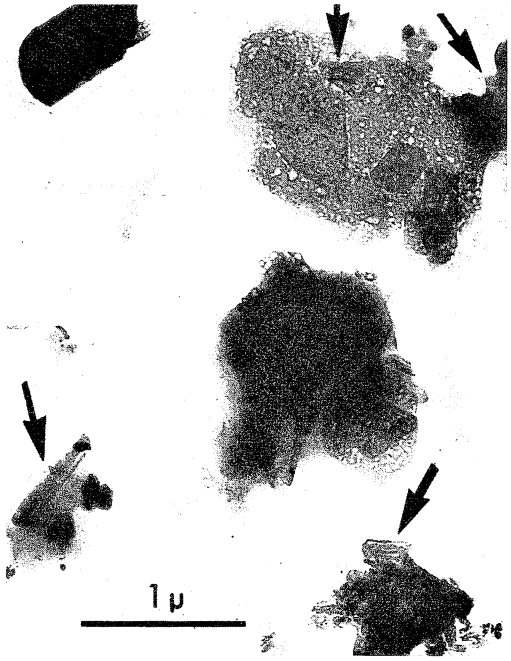
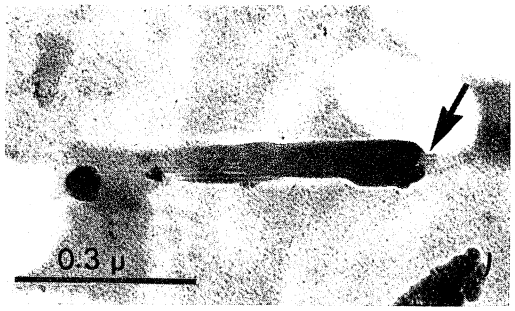


Fig 6.—Example of association of platy particles with chrysotile asbestos. **Arrows** indicate location of chrysotile fibrils. Note also presence of partially digested tissue.

Fig 7.—Asbestos body observed by means of EM. Photomicrograph shows parallel bundle of chrysotile fibrils coated with electron-dense material which resembles incipient bodies described in Suzuki and Churg.[25]



Scanning and Counting.—Photographic recording of EM fields consistently gave more accurate counts than simple visual scanning of the EM screen, a function of increased magnification, contrast, particle size, and search in partially digested debris.

Figure 4 demonstrates the difference in

counting obtained at different magnifications. At the low magnification shown in the *upper right* of Fig 4, the number of fibers and fibrils counted was only 45. However, when the magnification was increased photographically, the count increased to 59. Occasionally, even at the highest magnification, organic debris (partially digested tissue) obscures chrysotile from view.

Some fiber fields were counted by scanning and were then photographed; recounting of the fields photographed indicates that they were initially "undercounted" (Fig 5). The findings reported here are based upon scanning values, and are not corrected for photographic results; the fiber and fibril counts given, therefore, reflect minimum values.

There exists a close association of undigested tissue and chrysotile fibers and fibrils. Chrysotile fibrils occur not only next to, but in the midst of, partially digested tissue. Fibrils inside the undigested tissue tend to be thin-walled and relatively small. Clay minerals are present in the undigested tissue as well. Asbestos "counts" from photographs again demonstrated the presence of more chrysotile than could be counted directly on the screen.

Figure 6 demonstrates the close association of chrysotile with well-formed polygonal plates, morphologically similar to clay and talc minerals. This association is striking because chrysotile fibrils tend to be both "surface-" and edge-sorbed." Sheet silicates tend to have a negative surface charge and a positive edge charge. This suggests that one of the following mechanisms is at work: (1) The chrysotile fibrils possess a range of surface properties. (2) Different sheet silicates are involved in the association. (3) Both 1 and 2. (4) The association is artifact. The morphology of the sheet silicates indicates that they may be kaolinite (Compare with kaolinite plates shown in Beutelspacher and van der Marel,[31] Fig 19 to 32, pages 53 to 59; Fig 259 to 260, Page 271; Fig 238, A and B, page 252. Several of the sheet materials resemble bentonites, Fig 130, page 137, as well as labile chlorite, Fig 152 and 153, page 161). This association may be important in determining the origin of the chrysotile source. The nature of the pitted surface texture is likely artifact produced under the electron beam (compare with naturally occurring clay minerals).

In the extracts examined, only three of 28 cases showed what appeared to be asbestos bodies on the EM level. In each of these three cases, asbestos bodies were observed with the light microscope. Figure 7 shows one of these bodies. The asbestos body is morphologically like asbestos bodies observed by Suzuki and Churg[25] and appears to be nucleated on chrysotile. It is a paradox that, although asbestos bodies were readily observed with the light microscope, very few were observed with the EM. The reasons may lie in the preparation technique: the water drop is drawn off the EM substrate by means of a wet filter paper; the moisture is quickly and strongly "sorbed" into the blotting material; nearly all of the larger particles are "pulled" along with the water, as evidenced by brown discoloration of the wetted filter paper. It is likely that only the smallest and most highly surface-charged particles remain attached to the substrate.

Diatom fragments, other fibrous materials including fibrous glass and amphibole asbestos types and sheet silicates were observed to be present in the lung dust residues. The occurrence of fibrous particles (other than chrysotile and currently unidentified) is relatively frequent. Diatom fragments, although occasionally observed, were rare.

Comment

Unaltered chrysotile is uncommonly found as the core fiber in asbestos bodies removed from lungs of people in the general population. In an electron microprobe study of 16 cases from which such asbestos bodies were recovered and analyzed,[15] none were chemically equivalent to unchanged chrysotile. Fifteen of 16 analyses, however, were consistent with magnesium-leached chrysotile, and one was consistent with amphibole (amosite). We have similarly examined asbestos bodies from the lungs of a Canadian chrysotile miner and from hamsters injected with chrysotile, all of which gave results consistent with magnesium-leached fibers.

This could well have been predicted, in that biological environments would seem ideal for this. Indeed, with the rapid splitting of the fibers into their unit fibrils, the surface

area is greatly increased, augmenting the leaching process (air-milling of the fibers increases the surface area from 4 sq m/gm to almost 60 sq m/gm).[32]

There are other good reasons why the large fibers which we inevitably choose by optical microscopy will tend not to be unaltered (or even, in many instances, altered) chrysotile. The optical microscope delivers a select, biased population. First, we can only study what the microscope sees—and it only sees large fibers, those thicker than 0.5μ in diameter. Chrysotile, it has been noted, tends to split into finer fibrils in biological environments, and degradation to smaller particles occurs. Amphiboles (anthophyllite, tremolite, amosite, crocidolite) resist such physical and chemical attack. Therefore, selection of large asbestos bodies by means of light microscopy, resulting in a biased population of larger particles which were not altered in biological residence, stacks the analytical deck.

These considerations indicate that study of asbestos bodies need tell us little about the chrysotile content of human lungs. Rather, if we want to know whether chrysotile is present in lungs, we should look for chrysotile. In a sense, the wrong question has perhaps been asked; instead of "What is the nature of the core of the asbestos body?", it seems more profitable and more direct to ask the question, "Is chrysotile asbestos present in the lungs of urban dwellers?"

The answer to the latter question is unequivocally "yes." Data presented here demonstrate that this is so in New York city. Similar EM observations have been recorded in London, where Pooley et al[33] have not only found chrysotile asbestos in almost 80% of their cases, but noted it to be the most common and most abundant of all fibers detected. We anticipate that what is now known for New York and London will be found in other cities as well.

Conclusions

In 28 consecutive cases of urban dwellers who died in New York city, electron microscopy showed chrysotile asbestos to be present in all examined (28 of 28). Of these, possibly four of 28 may have been made "positive" by the occurrence of background fibril contamination. It is noteworthy that asbestos bodies were found in three of four of these "negative" cases.

Chrysotile asbestos often occurred in association with other substances, including platy particles (clay or talc), fibrous glass, and, occasionally, diatoms.

The question has been put forward: Is chrysotile asbestos present in the lungs of urban dwellers at this time? Our data demonstrate that the answer is unequivocally "yes" in New York city. Similar observations have been made in London. We anticipate that what is now known in these two cities will be found in other urban areas as well.

This study was supported in part by Public Health Service grant EC-00160.

One of us (A.M.L.) received support by career award ES448-12 from the National Institute of Environmental Sciences.

References

1. Stewart MJ, quoted by Cooke WE: Asbestos dust and curious bodies found in pulmonary asbestosis. *Brit Med J* **2**:578-580, 1929.

2. Stewart MJ, Haddow AC: Demonstration of the peculiar bodies of pulmonary asbestosis ("asbestosis bodies") in material obtained by lung puncture and in the sputum. *J Path Bact* **32**:172, 1929.

3. Gloyne SR: The presence of the asbestos fiber in the lesions of the asbestos workers. *Tubercle* **10**:404-407, 1929.

4. Haddow, cited in Report of annual meeting of British Medical Association, Manchester. *Lancet* **2**:230, 1929.

5. Thomson JG, Kaschula ROC, MacDonald RR: Asbestos as a modern urban hazard. *S Afr Med J* **27**:77, 1963.

6. Baden V, Schwartz J: Demonstration of asbestos bodies: Comparison of available techniques, in Anspach M (ed): *Proceedings of the Second International Conference on Biological Effects of Asbestos* (Dresden, East Germany, 1968), to be published.

7. Utidjian MD, Gross P, deTreville RTP: Ferruginous bodies in human lungs: Prevalence at random autopsies. *Arch Environ Health* **17**:327-333, 1968.

8. Bignon J: Incidence of pulmonary ferruginous bodies in France. *Environ Res*, to be published.

9. Selikoff IJ, Hammond EC: Environmental epidemiology: III. Community effects of nonoccupational environmental asbestos exposure. *Amer J Public Health* **58**:1658-1666, 1968.

10. Langer AM, Rubin I, Selikoff IJ: Electron microprobe analysis of asbestos bodies, in Webster I (ed): *International Conference on Pneumoconiosis*, (Johannesburg, South Africa, 1969), to be published.

11. Williams E: "Curious bodies" found in the lungs of coal-workers. *Lancet* **2**:541-542, 1933.

12. von Rüttner JR: Über Asbestose- und Pseudoasbestosekörperchen. *Schweiz Z Allg Path* **15**:628-631, 1952.

13. Schiller F: Adsorptionserscheinungen an Stauben in vivo: Pseudoasbestose- und Schaumankör-

perchen. *Arch Gewerbepath Gewerbehyg* **14**:664-672, 1956.

14. Gross P, Cralley LJ, deTreville RTP: "Asbestos" bodies: Their non-specificity. *J Amer Industr Hyg Assoc* **28**:541-542, 1967.

15. Langer AM: Electron microprobe analysis (study of asbestos fibers and bodies from lung tissue), in Sunderman FW, Sunderman FW Jr (eds): *Laboratory Diagnosis of Disease Caused by Toxic Agents.* St. Louis, Warren H Green Inc, 1970, pp 126-136.

16. Schwartz J, Langer AM: Extraction techniques and instrumental analysis of asbestos bodies removed from human lung tissue, in Anspach M (ed): *Proceedings of the Second International Conference on Biological Effects of Asbestos,* to be published.

17. Gross P, deTreville RTP, Haller M: Pulmonary ferruginous bodies in city dwellers: A study of their central fiber. *Arch Environ Health* **19**:186-188, 1969.

18. Sundius N, Bygden A: Der Staubinhalt einer Asbestosis-Lunge und die Beschaffenheit der sogennannter Asbestosis-Körperchen. *Arch Gewerbepath Gewerbehyg* **8**:26-80, 1938.

19. Beattie J, Knox FJ: Studies of mineral content and particle size distribution in the lungs of asbestos textile workers, in Davies CN (ed): *Inhaled Particles and Vapours.* New York, Pergamon Press Inc, 1961, pp 419-433.

20. Nagelschmidt G: Some observations of the dust content and composition in lungs with asbestosis, made during work on coal miners pneumoconiosis. *Ann NY Acad Sci* **132**:64-76, 1965.

21. Hargreaves A, Taylor WH: An x-ray examination of decomposition products of chrysotile (asbestos) and serpentine. *Mineral Mag* **27**:204-216, 1946.

22. Nagy B, Bates TD: Stability of chrysotile asbestos. *Amer Mineral* **27**:1055-1058, 1952.

23. Morgan A, Holmes A: Neutron activation techniques in investigations of the composition and biological effects of asbestos, in Webster I (ed): *International Conference on Pneumoconiosis,* to be published.

24. Beger PJ: Über die Asbestosiskörperchen. *Virchow Arch Path Anat* **290**:280-353, 1933.

25. Suzuki Y, Churg J: Structure and development of the asbestos body. *Amer J Path* **55**:79-107, 1969.

26. Stumphius J, Meyer PB: Asbestos bodies and mesothelioma. *Ann Occup Hyg* **11**:283-293, 1968.

27. Timbrell V, Pooley F, Wagner JC: Characteristics of respirable asbestos fibres, in Webster I (ed): *International Conference on Pneumoconiosis,* to be published.

28. Lynch JR, Ayer HE: Measurement of asbestos exposure. *J Occup Med* **10**:21-24, 1968.

29. Edward GH, Lynch JR: The method used by the U. S. Public Health Service for enumeration of asbestos dust on membrane filters. *Ann Occup Hyg* **11**:1-6, 1968.

30. Berkley C, Churg J, Selikoff IJ: Detection and localization of mineral fibers in tissue. *Ann NY Acad Sci* **132**:48-63, 1965.

31. Beutelspacher H, van der Marel HW: *Atlas of Electron Microscopy of Clay Minerals and Their Admixtures.* New York, American Elsevier Publishing Co Inc, 1968.

32. Speil S, Leineweber JP: Asbestos minerals in modern technology. *Environ Res* **2**:166-208, 1969.

33. Pooley FD, Oldham PD, Chang-Hyun UM, et al: The detection of asbestos in tissues, in Webster I (ed): *International Conference on Pneumoconiosis,* to be published.