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Irving J. Selikoff MD [a], William J. Nicholson PhD [a] & Arthur M. Langer PhD [a]
[a] Environmental Sciences Laboratory, Mount Sinai School of Medicine, 10029, Fifth Avenue and 100th Street, New York, USA
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Scientific Communications

Asbestos Air Pollution

Irving J. Selikoff, MD; William J. Nicholson, PhD; and Arthur M. Langer, PhD, New York

Findings of mesothelioma among individuals living in the vicinity of asbestos plants suggested that asbestos air pollution might occur. Measurement of asbestos (chrysotile) content of ambient air in New York City and other locations showed levels of 10 to 50 × 10^{-9} gm/cu m, and lungs of New York residents examined at autopsy regularly showed chrysotile fibrils. Occurrence of asbestos air pollution is now established. What have not yet been defined, however, are the dimensions of disease hazard which may be associated. Epidemiological considerations suggest that it is improper to equate the several kinds of asbestos exposure: direct occupational, indirect occupational, exposure in family circumstances, neighborhood contamination, and general community asbestos air pollution. Since most urban air pollution is derived from commercial and industrial sources, the asbestos industry has both important responsibility and opportunity for its control.

EARLY in the 1960s, the problem of asbestos disease was disseminated from the occupational area into the general environment. Three germinal observations were responsible for this.

First, Kiviluoto reported finding 499 cases of parietal pleural calcification among 6,312 residents of a rural county in Finland.[1] This came very much as a surprise, since the type of calcification observed had previously been described primarily in asbestos workers,[2] usually more than 20 or 30 years after starting work[3]; yet the cases were not among asbestos workers, but among farmers and farmers' wives. The asbestos link, however, was there; the county did have an asbestos mine. The natural supposition was that asbestos air pollution from the mine was responsible, a presumption that was strengthened when the specific type of asbestos produced by the mine (anthophyllite) was found in the air up to 50 km away,[4] and asbestos bodies were demonstrated in the lungs of cattle grazing in the nearby fields.[1]

The same year (1960) saw a second worrisome communication. Wagner et al[5] reported 47 cases of pleural mesothelioma in a part of South Africa which was important for asbestos mining. They noted potential asbestos contact for most of the patients two or more decades before, in many instances merely the result of living in the general area or of chance contact in a family setting. While pleural mesothelioma had previously been attributed to asbestos exposure,[6] the strength of this association had not been appreciated. More pertinent was the demonstration that it could result from other than occupational exposure.

Amplification soon followed. Newhouse and Thompson[7] studied all mesotheliomas at the London Hospital. They confirmed the close association with asbestos, 31 of the 76 subjects having had occupational exposure. They also confirmed the importance of nonoccupational contact; of the 45 who had not worked with the material, nine had lived in the households of asbestos workers and 11 had lived, decades before, within one-half mile of an asbestos plant.

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

Lieben and Pistawka reported similar findings in Pennsylvania.[8]

What appeared to be a strong link in the chain of evidence for environmental asbestos disease was the report in 1963 by Thomson et al[9] that asbestos bodies were commonly to be found in the lungs of the general population of Capetown, South Africa. They expressed a drop of lung tissue fluid onto a slide, examined it by optical microscopy, and in one quarter of 500 consecutive autopsies, found structures apparently identical with those seen in asbestos workers' lungs. But these were not asbestos workers; they had been ordinary citizens of this large city. Thomson and associates concluded that the subjects had inhaled asbestos in the course of their urban living, from the many asbestos products about them. With knowledge of what exposure to these fibers could do under industrial circumstances as a background, and with the observations of Wagner et al[5] as an example, they suggested that we were now faced with a "modern urban hazard" and predicted that asbestos-associated neoplasms would rival cigarette-induced lung cancer in the future.

It should be noted that the prediction of Thomson and associates[9] is an extrapolation. It is a wide step from occupational exposure, with large numbers of asbestos bodies, to community contamination, with, as a rule, far fewer bodies, particularly with little knowledge of a dose-disease response relationship. Also, the particles described had the appearance of those seen in asbestos workers, but some uncertainty existed that these necessarily had an asbestos core,[10] especially since it had been known for 30 years that such bodies could be found after exposure to other fibers as well.[11]

Despite these caveats, the three reports, taken together, posed a problem that is now very real. Occupational asbestos exposure may be associated with serious risk; for example, among asbestos insulation workers in the New York metropolitan area at this time, approximately one in five deaths is due to lung cancer, one in ten is due to mesothelioma, one in ten to gastrointestinal cancer, and one in ten to asbestosis and cor pulmonale.[12-14] These men have been exposed to amounts of asbestos surely greater than those in the surrounding community, and we would not expect their risk to be duplicated in the general population. But is part of their risk disseminated, with the dusts from their work? What sort of dose-response curve are we dealing with? Is there a threshold which, once crossed, leads to serious hazard of neoplasia? An hypothesis could be formulated that such a threshold calls for very little asbestos (since small amounts may still reflect billions of fibers or fibrils, or both), and that asbestos workers pass it early in their careers.

Such an hypothesis, or some variation of it, is not inconsistent with observations made in the last several years. Neoplasms, such as pleural and peritoneal mesothelioma, occur in excess even among asbestos workers with little or no radiological evidence of asbestosis, suggesting that exposures insufficient to cause asbestosis may still produce neoplasia. The spread of this separation is not established, but it is apparent recently that while lower dust levels in industry may prevent much asbestosis, such levels will not necessarily prevent cancer.[15] It is not now known how low a threshold must be to prevent asbestos-associated neoplasms.

Asbestos in Lungs

It thus became important to know whether asbestos truly was a common contaminant of urban dwellers' lungs. The demonstration that "asbestos bodies" were to be regularly found at autopsy in many cities of the world[16] confirmed the finding of Thomson et al,[9] but did not settle the question. It was not known whether the cores were necessarily asbestos, an uncertainty resulting from technical difficulties involved in analyzing such cores.[17]

In recent months, the impasse has been resolved by direct search for asbestos fibers and fibrils.[18] Investigation of 3,000 consecutive autopsies in New York had shown that "asbestos bodies" were common; optical microscopic examination of 175μ × 1 sq cm of lung tissue in each of these cases, showed asbestos bodies in 1,449 (48.3%) (Table 1). It is likely that, had a greater volume of tissue been submitted for study, asbestos bodies would have been found in all the

Table 1.—Asbestos Bodies in 3,000 Consecutive Autopsies in New York City, 1966 to 1968*

	Male		Female		Total	
Age	No.	%	No.	%	No.	%
<1	2/73	2.8	2/53	3.8	4/126	3.2
1-19	0/7	0.0	4/25	16.0	4/32	12.5
20-39	34/102	33.3	19/58	32.8	53/160	33.1
40-59	316/606	52.1	108/247	43.7	424/853	49.7
60-79	555/997	55.7	220/491	44.8	775/1,488	52.1
80+	106/186	57.7	83/155	53.5	189/341	55.4
Total	1,013/1,971	51.4	436/1,029	42.4	1,449/3,000	48.3

* From Langer et al.[18]

Table 2.—Numbers of Asbestos Bodies*

	Asbestos Bodies				
	0	1-4	5-14	15+	Total
Age					
<1	122	2	2	0	126
1-19	28	4	0	0	32
20-39	107	53	0	0	160
40-59	429	359	45	20	853
60-79	713	630	105	40	1,488
80+	152	156	40	3	341
Total	1,551	1,204	192	63	3,000
Sex					
Male	958 (48.6%)	802 (40.7%)	152 (7.7%)	59 (3.0%)	1,971 (100.0%)
Female	593 (57.6%)	392 (38.1%)	40 (3.9%)	4 (0.4%)	1,029 (100.0%)
Total	1,551	1,194	192	63	3,000

* From Langer et al.[18]

cases, except perhaps the infants and very young children.

The same examination showed that, in addition to coated particles ("asbestos bodies"), uncoated inorganic fibers were also readily seen (Table 2). Fibers thicker than 1.0μ were almost universally to be found; most of these are as yet still unidentified, although some were diatom fragments, glass fibers, or phytoliths. We were more concerned, however, with thinner fibers, less than 1.0μ in diameter, since these would be more consistent with chrysotile, the asbestos variety making up 95% of the asbestos used in the United States. Such thin fibers were also commonly present, being found in 1,038 of the 3,000 cases, and tending to vary proportionately with the number of asbestos bodies (Table 3).

The critical information was obtained by examining, in 28 of the 3,000 cases, a very small portion of lung (conservatively estimated at 10^{-6}) by a technique which allowed qualitative analysis,[17] with appreciation that the unique structure of chrysotile allows its specific identification by high magnification electron microscopy.

Chrysotile fibers or fibrils, or both, were found in every specimen (Fig 1). In four of the 28, background contamination could conceivably have been responsible for the findings. In 24 of the 28, the number found was greater than background counts could explain (Table 4). The morphological appearance and other characteristics of these fibers and fibrils are recorded elsewhere.[19]

It is evident that chrysotile asbestos is a common contaminant of the lungs of New York City residents at this time. Similar electron microscopic observations have been recorded in London,[20] where not only was chrysotile asbestos found in almost 80% of cases, but it was noted to be the most common and most abundant of all fibers detected.

The question inherent in the observations of Thomson and associates[9] in 1963, "is

Table 3.—Correlation of Inorganic Fibers with Asbestos Bodies in Lungs of 3,000 Consecutive Autopsies in New York City, 1966 to 1968*

Percentage of cases showing asbestos bodies in relation to occurrence of thin inorganic fibers

Asbestos Bodies	Thin Inorganic Fibers: 0 No.	0 %	1-4 No.	1-4 %	5-14 No.	5-14 %	15+ No.	15+ %
0	1,168	59.5	332	38.3	35	28.0	16	33.3
1-4	705	35.9	424	49.0	52	41.6	13	27.1
5-14	76	3.8	89	10.3	21	16.8	6	12.5
15+	13	0.66	20	2.3	17	13.6	13	27.1
Total	1,962	65.4	865	28.8	125	4.1	48	1.6

Percentage of cases showing thin inorganic fibers in relation to occurrence of asbestos bodies

Thin Inorganic Fibers	Asbestos Bodies: 0 No.	0 %	1-4 No.	1-4 %	5-14 No.	5-14 %	15+ No.	15+ %
0	1,168	75.3	705	59.0	76	39.6	13	20.6
1-4	332	21.4	424	35.5	89	46.3	20	31.7
5-14	35	2.3	52	4.3	21	10.9	17	27.0
15+	16	1.0	13	1.1	6	3.1	13	20.6
Total	1,551	51.7	1,194	39.8	192	6.4	63	2.1

* Correlation was done by optical microscopic study, from Langer et al.[18]

Table 4.—Chrysotile in 28 Cases Studied by Electron Microscopy

Group	No. of Chrysotile Fibers + Fibrils	Cases	Male	Female
1	≦9	4/28	3	1
2	10-50	11/28	5	6
3	51-99	6/28	1	5
4	100-200	4/28	4	0
5	≧201	3/28	3	0
"Blank grids"	≦9	...	...	...

chrysotile asbestos commonly found in the lungs of urban dwellers at this time?" has now been answered, "Yes, unequivocally."

Relation of Asbestos Lung Burden to Environmental Asbestos Disease

There are few data at this time that would allow judgement of the significance of the asbestos we have found in lung tissue of urban residents. Whether or not the amounts present as the result of other than occupational exposure are associated with frequent risk of disease is not known. In part, this is a reflection of the scarcity of data concerning the asbestos content of lungs in general, including those of asbestos workers. Such data as are available suggest that the amount in lungs of the latter is quite small, ranging from 0.6% to 0.001% of lung weight.[21-22] It would be expected to be even lower in those not occupationally exposed.

No information is at hand concerning the asbestos content of the lungs among the cases of Wagner et al,[5] or Newhouse and Thompson,[7] or those of other cases with environmental disease.[23,24] Nor is there quantitative information concerning the asbestos content of the lung in individuals in the general population without stigmata of asbestos. This is rather urgently needed, with age, sex, residence, and occupational exposure taken into account.

Studies now in progress in our laboratory and elsewhere (F. D. Pooley, PhD, oral communication [July 1970]) indicate that suitable quantitative techniques for estimating asbestos lung content will be feasible and that fairly accurate estimates, approxi-

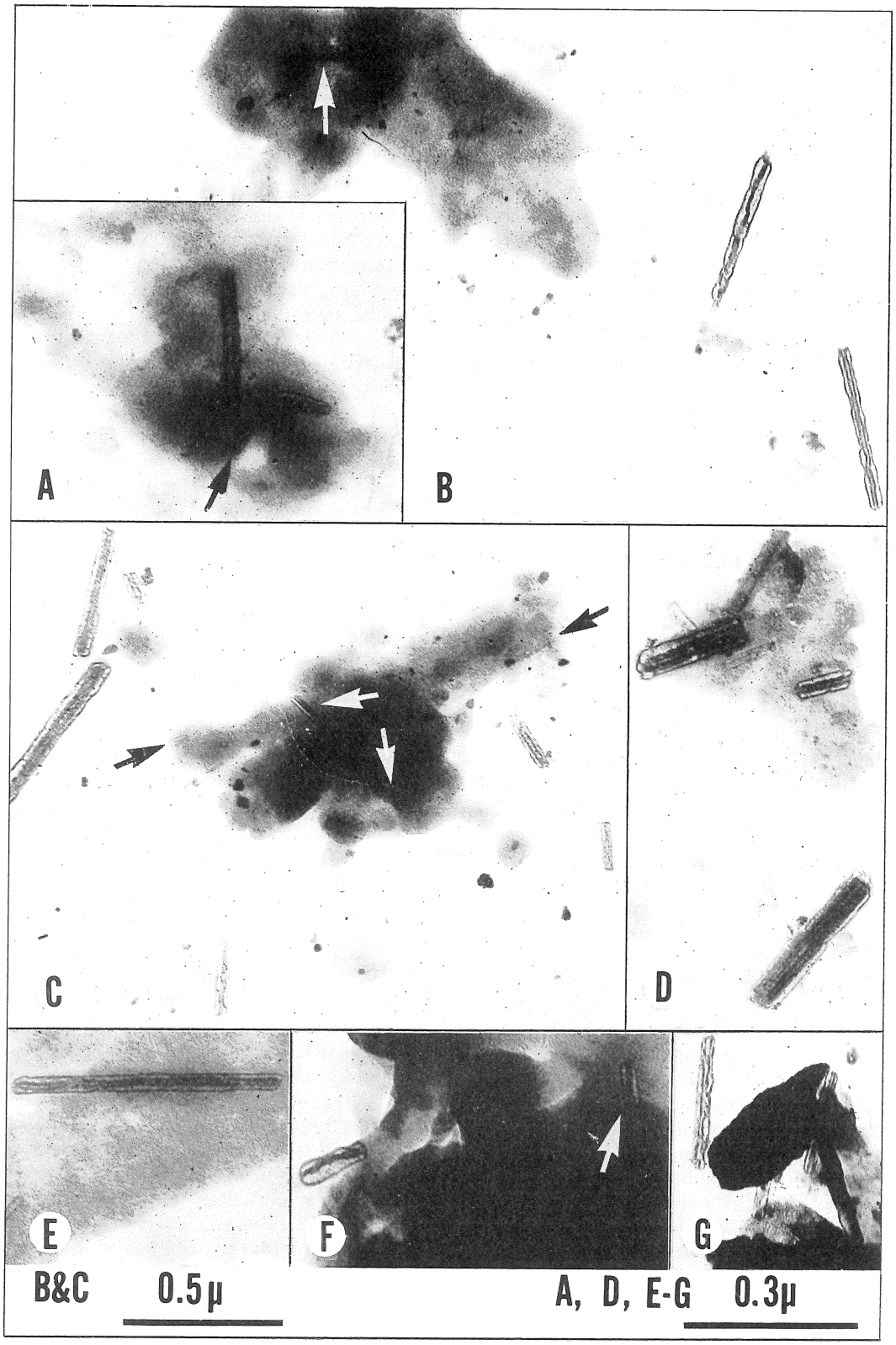

Fig 1.—Association of chrysotile fibrils and incompletely digested lung tissue. **Arrows** indicate "hidden" fibrils in partially digested tissue.

mating an order of magnitude, are to be anticipated in the future.

Asbestos Air Pollution

It is probably an entirely justified concept that the asbestos found in urban dwellers' lungs is derived from the inhalation of air contaminated with these fibers. Very little is known, however, of the conditions of such contamination, and facile assumptions should be avoided at this time.

An example of where an "obvious" explanation might also be inaccurate may be found in the assumptions adopted to explain Kiviluoto's[1] observations. It seemed natural to expect that the demonstrable anthophyllite asbestos air pollution from the mine and mill was etiologically related to the equally demonstrable asbestotic pleural calcification in the population living about that point source. It turns out, however, that this may not be the entire explanation, and that intimate contact with local asbestos-bearing rocks, including those used in building houses, saunas, barns, and the like, might also play a role.[25]

Indeed, the latter association better explains almost identical epidemiological findings in Bulgaria, where, again, pleural calcification was found in a rural population. In this district, too, an asbestos mine was being worked, but it was an underground mine and had opened only in 1943, too recently for its discharges to be expected confidently to have had the effect noted[26] (the lapsed period between initial exposure and evidence of pleural calcification is 20, 30, 40, or more years). As in Finland, the local field rocks have a high asbestos content and are used for various structures by the farming population, which then may have intimate contact with what is shed from them. More important, perhaps, the soils tilled by these farmers can be demonstrated to have asbestos fiber (anthophyllite) content. The importance of this observation was emphasized by the discovery that those farmers working plots without asbestos soil contamination had little pleural calcification, whereas farmers working soil with anthophyllite readily showed the radiological changes.[27] It may be that similar explanations will become available for the finding of endemic pleural calcification in some rural areas of Czechoslovakia.[28,29]

At first, it seems somewhat surprising that so few data are available concerning the asbestos content of ambient air, especially since so much is known regarding the asbestos content of air within the work place. Perhaps the best explanation is that once the factory gates are passed, a whole new set of technical problems is encountered, and sampling procedures, analytical approaches, and measuring methods useful for industrial controls are no longer applicable.

Fiber Identification.—Under the industrial circumstances, there is usually no problem to knowing exactly what is being measured, since the materials being used are either well characterized or can be readily analyzed. Thus, whatever fibers are seen can be confidently labelled as "chrysotile," "crocidolite," "amosite," "fibrous glass," and other types.

If these same fibers were to be seen in a random sample, especially if they are small, our confidence disappears. All that can be said is that inorganic fibers are present. Even then, if these fibers were found in very large numbers, identification could be readily accomplished by such mass techniques as x-ray diffraction. When they occur singly or randomly scattered in small numbers, these techniques are no longer applicable; and readily available alternate approaches, such as polarized light microscopy, hardly have the same definitive assurance. While it is true that analytical attack on single fibers is still possible, these approaches (including electron microprobe analyses, electron diffraction, and electron microscopy) are time-consuming and often restricted by the size of the particle available for analyses.

Particles and Fibers.—There has long been an anomaly in particle counting for asbestos threshold levels. In the United States, a threshold level was proposed in 1938 for occupational exposure to asbestos, based upon experiences necessarily limited to that point.[30] The recommendation reflected the instrumental restrictions of the times and were based upon counting of "particles" by optical microscopy. It was recognized that such particles could either be fibrous or nonfibrous and that the proportion could vary widely according to the materials used,

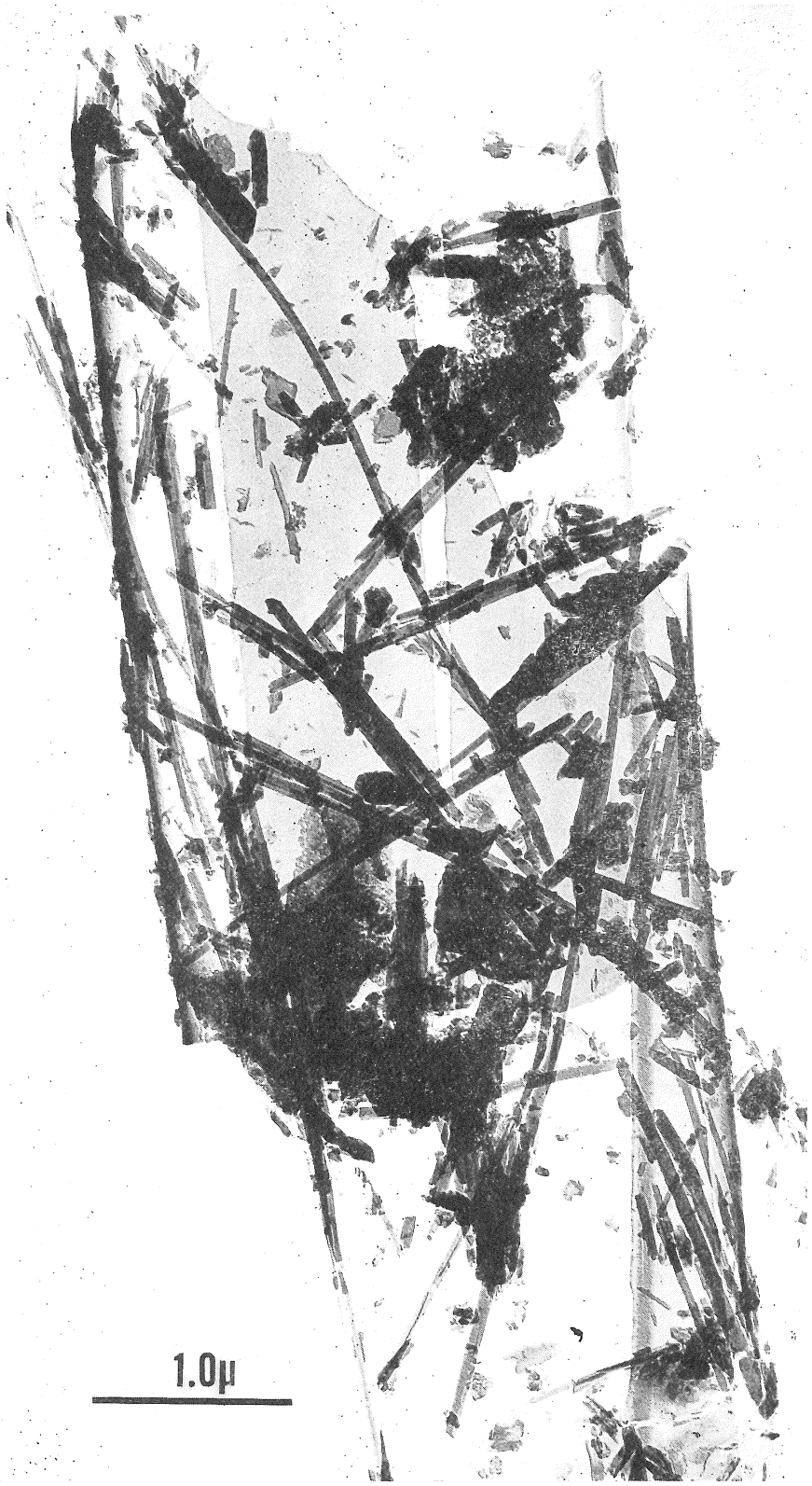

Fig 2.—Clay particle with adsorbed chrysotile fibrils, in air sample about construction site (×28,000).

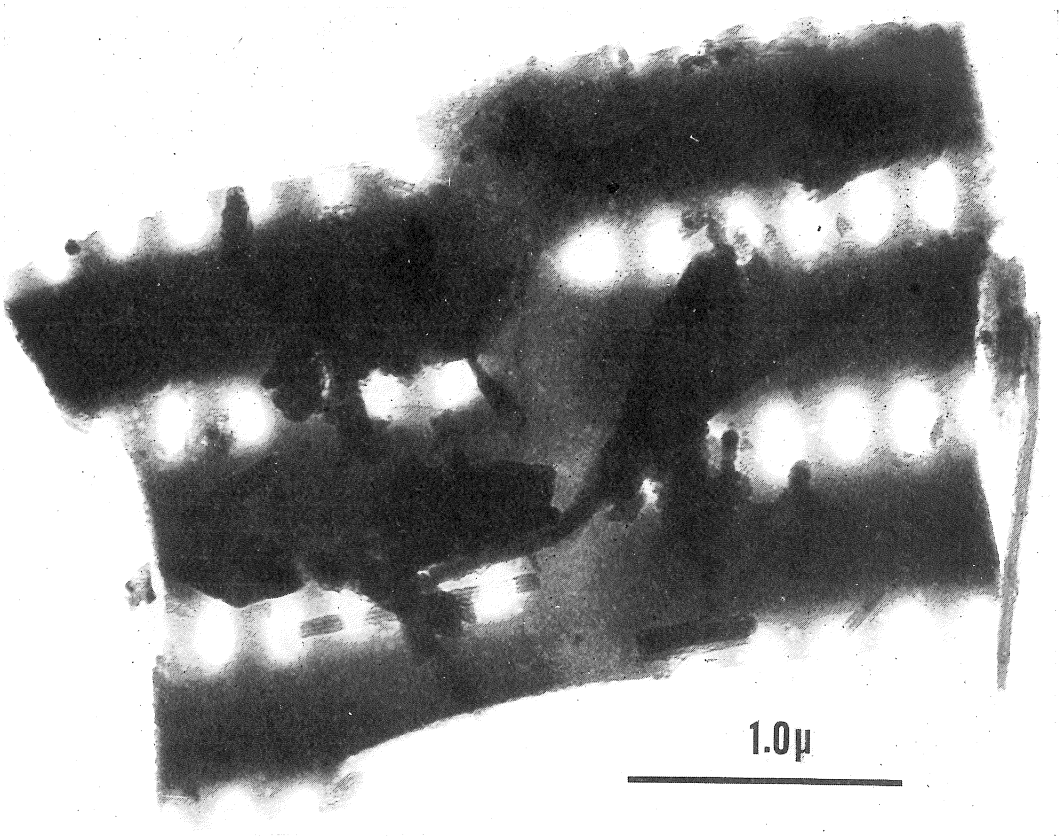

Fig 3.—Adsorption of chrysotile fibrils onto surface of diatom. Fibrils would not be seen by optical microscopy.

process studied, or other factors. Since the presumption is that only the fibers are responsible for the biological effect, such analytical dilution is hardly acceptable at this time, and current approaches to industrial threshold limit values are based entirely on fiber count. But even here, the matter is not so easily disposed of, since the admixture of particles and fibers often includes surface interactions among them. Figures 2 and 3 show particles of clay and a diatom collected near a construction site by one of us (A.M.L.). By light microscopy these would be categorized as "particles." Yet, by electron microscopy, it was found that numerous fibers (fibrils) were attached to the surface of the particles (opposite surface charges). In such circumstances, any biological effect of the fibers could be incorrectly attributed to particles. The question of size and magnification is critical.

Fiber-Fibril.—Each asbestos fiber variety is quite different chemically, physically, structurally, and morphologically.[31] Chrysotile seems unique in its tendency to physical instability under a number of circumstances. The chrysotile "fiber" is not a unit whole but is, rather, composed of a large number of individual fibrils, each from 300 to 400 Angstroms. Under industrial circumstances, it is recognized that when a population of fibers is counted, "invisible" fibrils are also present,[32] but that the optically visible fibers reflect, in varying proportions under different circumstances, the total chrysotile population, even though there is only one fiber for a very large number of fibrils. Also, with proximity to the industrial source, many fibers are still present, not having been subjected to influences which could result in their separation into fibrils.

In the ambient air, however, very little is known about the proportion of fibers to fibrils. The matter is of some importance,

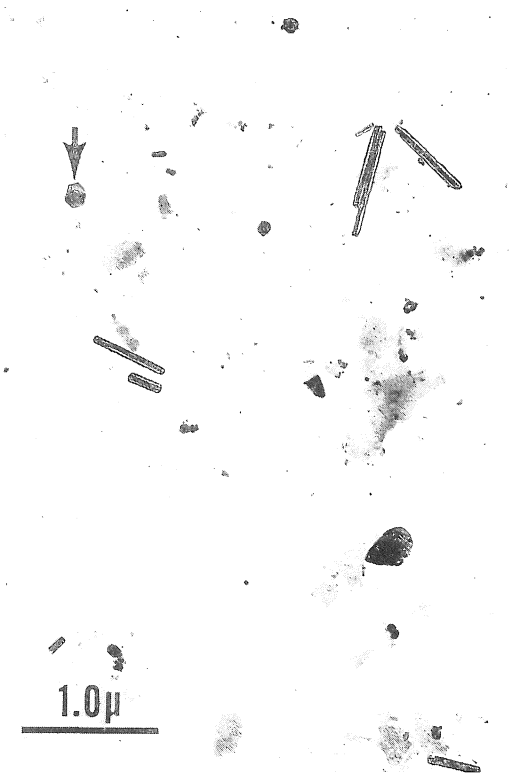

Fig 4.—Single, short fibrils collected 3/16 of mile downwind of chrysotile source. Arrow indicates position of clay particle.

since not only must number be categorized as fiber and fibril, but the surface area and potential biological effect might be quite different with the same total chrysotile mass, with different percentage fibrillation. The proportion of fibers to fibrils cannot be determined without the electron microscope.

Size.—Thus, a critical factor in studying asbestos air pollution is the utilization of techniques which will measure very small particles. It is unlikely that approaches which do not include the electron microscope will be effective. Indeed, one might add that high magnification electron microscopy will be needed, including magnifications of at least 20,000× (direct), and probably over 40,000×. While optical microscopy may be suitable as a guide for occupational asbestos exposure, it has insurmountable inadequacies in studying asbestos pollution of the ambient air.

In addition to the fine diameter of the fibrils, it has been our experience that many of them are quite short, as well. We have observed chrysotile fibrils with lengths less than 1,000 A in many instances; when these are enmeshed in sampling debris, not only is high magnification electron microscopy necessary, but visual scanning may be inadequate, and inferior to photographic recording.[17]

It may be worth noting that such small particles are at present subject to identification by their morphological characteristics only; structural and microchemical analysis by electron microprobe or electron diffraction study has many difficulties. It may be hoped that instrumental advances will remedy this situation (greatly improved microprobe definition can be anticipated, for example). Turkevich's "World of Fine Particles"[33] is surely with us.

Quantitation.—Under industrial circumstances, there is little difficulty in estimating the quantity of asbestos in a given sample of air. From this, the surface area of the fibers can be estimated or directly measured by nitrogen absorption or other techniques. It is even possible to conceive of gravimetric methods, rather than the more laborious counting of fibers. Quantitation is much more difficult in ambient air samples and, in our experience, only approximations can presently be obtained.

Current Approaches to Asbestos Air Sampling.—With the foregoing factors in mind, the absence of published information on asbestos levels in urban ambient air may be understood. Few air monitoring agencies have had available the technical equipment for the examination of ultramicroscopic asbestos fibrils. Moreover, even those fibers seen by optical microscopy in air samples required elaborate techniques (such as electron diffraction or microprobe analysis) for positive identification.

Present approaches to quantitating asbestos levels in ambient air, using ultramicroscopic techniques, have sought two objectives. The first is to obtain a measure of the mass of asbestos per unit of air volume at various locations in urban centers and later, for comparative purposes, in more rural areas. This would provide a stable estimate, since fiber size distributions change as sampling is undertaken at different distances from asbestos emission sources. Equal numbers of fibers can represent significantly different

amounts, by weight, of asbestos. Moreover, it is not currently known how strongly biological effect is dependent on fiber size. This being so, a measure by mass or weight may be more conservative for the establishment of air quality criteria and standards, since the biological effect of a sample dominated by large fibers may be overestimated.

Secondly, fiber size distribution is important. At the moment, to obtain such a distribution at each sampled site is time-consuming, and the presence of other material and existing transfer and sampling methods may distort the observed fiber distribution. However, experiences in current studies suggest that these problems can be overcome, and that complete fiber size distributions will be obtained at selected sites in the near future.

Sample Preparation.—In our studies, air samples were collected on membrane filters having an effective pore size of either 0.8μ or 1.2μ. While this pore size is larger than the largest dimension of some asbestos fibrils, it has been found that the surface charge properties of the filter and the asbestos, as well as the circuitous path through the filter, allow virtually complete collection of all asbestos material.

Both high-volume samplers capable of drawing 40 cu ft/min through an 8 × 10-inch filter, and small battery-operated personnel monitoring samplers with a capacity of 2 liters/min through 8 sq cm, were found effective in sample collection. Portions of each collected sample were ashed in an activated oxygen asher which oxidized the membrane filter and all organic or carbonaceous material in the sample.[34] The residue, consisting mostly of fly ash and mineral matter, was dispersed on a microscope slide by grinding in a solution of 1% nitrocellulose in amyl acetate for two to five minutes. Upon evaporation of the amyl acetate, the dispersal was scanned for uniformity by optical microscopy and a representative area chosen for transfer to an electron microscope grid for scanning.

During this procedure, those chrysotile fiber bundles present are broken into their elementary fibril form. The grid is scanned at 42,000× in an electron microscope. Typically, six grids are prepared of each sample, and three 100μ × 100μ squares of each grid are scanned. The mass of asbestos is obtained by measuring the volume of asbestos per grid square and multiplying by the appropriate density. Figure 4 shows several isolated fibrils in an ambient air sample as seen by electron microscopy. Usually, only single fibrils, if any, are observed in the microscopic field.

However, as one takes samples near sources of asbestos, more large fiber bundles are present in the initial sample. As these are dispersed during sample preparation, occasionally some groups of fibrils remain intact. The presence of these bundles, or even localized fibril clumps, gives rise to a variation between samples greater than one would expect from statistical considerations alone. However, the scanning of many grid squares serves to average their effect.

Results of Initial Investigations.—Ambient air levels were measured at various sites in New York City. These samples were taken at selected locations of the sampling network of New York City's Department of Air Resources. The sites were all located on public buildings distant from any known significant source of asbestos. The chrysotile content of ambient air in the various sampling locations was as follows: Manhattan, 25 to 60, asbestos air level in 10^{-9} gm/cu m; Bronx, 25 to 28, asbestos air level in 10^{-9} gm/cu m; Brooklyn, 19 to 22, asbestos air level in 10^{-9} gm/cu m; Queens, 18 to 29, asbestos air level in 10^{-9} gm/cu m; and Staten Island, 11 to 21, asbestos air level in 10^{-9} gm/cu m. While preliminary in nature, the samples from Manhattan tended to be higher than those from other boroughs. Lowest values were usually from sites most distant from densely populated business areas (Staten Island).

While amounts ranging from approximately 10 to 100 × 10^{-9} gm/cu m of sampled air may appear to be exceedingly small quantities of asbestos, it is well to recall that chrysotile asbestos easily fragments into ultimate fibrils 300 to 400 A in diameter and often 2,000 A or smaller in length. Thus, 10^{-9} gm of chrysotile asbestos could represent a million fibrils.

That Manhattan has higher levels of asbestos than other boroughs is to be expected, as greater use of asbestos in building construction takes place in that borough. During the past ten years, it has been common

practice to spray fireproofing material containing from 10% to 30% asbestos, onto girders, spandrels, and decking of high-rise office buildings. Often, inadequate precautions were taken by contractors to contain the spray material and extensive "snowfalls" of asbestos-containing material took place over widespread areas of Manhattan. This practice was of such obvious concern that New York City has banned the spraying of asbestos fireproofing, effective as of Feb 26, 1972.[35]

Prior to the implementation of New York City's regulations, data were obtained in lower Manhattan at various sites in the vicinity of buildings under construction where spray fireproofing with asbestos-containing materials was used. During the two days on which data were obtained, the asbestos levels in 10^{-9} gm/cu m were as follows: site 1, downwind from a spray source, 45 to 80; site 2, 45° downwind from one source and upwind from others, 15 to 30; site 3, upwind from any source, 20; site 4, downwind from a spray source, 45; and site 5, upwind from any source, 20.

The data in New York have been supplemented by measurements in other urban and in more rural areas. The chrysotile content of air in three selected locations was as follows: Philadelphia, 45 to 100, asbestos level in 10^{-9} gm/cu m; Ridgewood, NJ, 20, asbestos level in 10^{-9} gm/cu m; and Port Allegany, Pa, 10 to 30, asbestos level in 10^{-9} gm/cu m (amosite fibers were also found in the air of this community; a factory using this material was present).

These data, though still limited, serve to establish that, at least in the areas sampled, there is a background of chrysotile contamination of the ambient air and that this may be higher about construction sites in urban areas. Much more information will be required, however, before reliable estimates can be made concerning quantitative levels of such contamination.

Epidemiological Perspectives

Environmental Disease.—The occurrence of asbestos pollution of urban air is now established. What has not been defined, however, are the dimensions of disease which may be associated with this pollution. Indeed, it is hardly proper to speak of "asbestos air pollution" in general terms. There are different sets of circumstances in which such pollution can occur, varying in intensity, intimacy, and duration of exposure.

Lapsed Period.—A problem common to all types of asbestos air pollution is the long lapsed period between onset of exposure and appearance of disease. In general, this is 20, 30, 40, or more years insofar as neoplasia is concerned. There are variations, of course. It may be that intensity of exposure is one such variable; others could include fiber variety, fiber size, competitive risk of asbestosis,[36] cofactors such as cigarette smoking,[13] trace elements, and perhaps other concomitant air pollutants.

Defined Populations.—It should be recognized that the different kinds of asbestos air pollution are not limited to well separated compartments. Exposure to general community asbestos air pollution may be overwhelmed by indirect occupational exposure, in the case of a construction workman. Similarly, the asbestos inhaled by virtue of family contact in the household of an insulation worker could hardly be attributed to the scant asbestos fibrils in the air of a rural community in which that employee happened to live. Such permutations are common and may be misleading unless identified. When considering neighborhood air pollution about an asbestos plant (30 years ago, since it would be these people with whose fate we would now be concerned) it is well to remember that, at least in the 1930s and 1940s, people who worked in a plant tended to live near it. Thus, the population about a plant being studied would have to be well characterized, to identify those with direct occupational exposure, before the effects of neighborhood contamination could be evaluated.

Sources and Control

Sources for asbestos air pollution can be looked at in two ways. They can be identified and measured without reference to exposed populations. Epidemiological attention may then be attracted to these "contamination sources in search of disease." Alternatively, sources for asbestos air pollution can be studied in relation to their potential for exposure of human populations.

At this time, both approaches are hampered by inadequate information on the relative significance of peak exposures compared to constant background contamination. There is clinical evidence which indicates that heavy exposures for brief periods (days, weeks, months), with retention of the inhaled fibers for the rest of the individual's lifetime, may carry serious disease potential.[37] Therefore, intermittent high peak exposures may carry an unusual risk, especially when added to the cumulative retention associated with background pollution over long periods of time.

Adequate information is needed concerning the natural history of asbestos air pollution, including persistence, variations with meteorological conditions, and ultimate fate.[38] Asbestos fibers, being mineral, may persist in the environment for long periods. It is not known, however, if this is so, or to what extent attrition occurs by a variety of physical processes. We have found amosite asbestos fibers in the settled dust and in the household air, within homes which had been occupied 15 years before by workmen of an amosite factory. Similarly, both settled dust and ambient air in a construction workman's home contained chrysotile fibers at levels beyond those usually observed as background. Neighborhood contamination from factory sources may also be associated with long persistence of the mineral fibers. In preliminary studies, we have found this to be true of both superficial soil contamination and settled dust on attic rafters in such neighborhoods. It is apparent that information concerning persistence and fate of asbestos air pollution would be important, if only as a background to the evaluation of air levels from current emission sources.

Natural Sources of Asbestos Air Pollution.—It is likely that some air contamination occurs from natural sources. Serpentine rock outcroppings occur in many parts of the United States and other countries. Studies in our laboratories suggest that, on an ultramicroscopic level, serpentine very frequently contains some fibrous mineral components, which are properly classified as chrysotile. In addition, some outcroppings contain frank chrysotile veins. Such chrysotile-containing rocks are widely distributed, although not necessarily in commercial concentrations. Nevertheless, abrasion and weathering of such surface formations might be accompanied by release of chrysotile fibers into the surrounding air.

Industrial and Commercial Sources.—Although natural sources for asbestos air pollution should be considered, it is likely that they add only an infinitesimal amount to the asbestos air burden in urban areas. Most is derived from commercial and industrial sources. Here, emission-source inventories can be prepared and would include transport and storage of raw fiber supplies, manufacture of the many useful asbestos-containing products, transport and end-use of these products, their weathering, and ultimate disposal as waste. In general, the potential for pollution varies with the degree of fixation of the fiber in the product.

Approximately two thirds of the asbestos used in the United States is used in construction products. Ship building and repair, and waste disposal of asbestos products are important areas for study. Spraying of asbestos-containing mineral fiber insulation has already been mentioned. Factory emissions are an obvious, and controllable, difficulty. Housekeeping in all asbestos-using facilities may turn out to be a knotty problem, and ultimately associated with much asbestos air pollution.

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ERRATUM

In a recent article by Sherwin and Yuen, "Silicone Fluid for the Metering and Monitoring of Nitrogen Dioxide" (24:331, 1972), the number "5.23 ppm" in the third line of column 1 on page 331 should read "3.93 NO_2."