ASBESTOS AIR POLLUTION IN URBAN AREAS

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INTRODUCTION

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 was, however, 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 (anthpohyllite) 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[5] reported 47 cases of pleural mesothelioma in a part of South Africa important for asbestos mining. He unearthed 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 by chance contact in a family setting. While pleural mesotheliomas 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 was soon added. Newhouse[7] studied all mesotheliomas at the London Hospital. She confirmed the close association with asbestos, 31 of the 76 cases having had occupational exposure. She also confirmed the importance of non-occupational contact: of the 45 who had not worked with the material, 9 had lived in the households of asbestos workers and 11 had lived decades before, within one-half mile of an asbestos plant. 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[9] that asbestos bodies were commonly to be found in the lungs of people in the general population of Capetown. He expressed a drop of lung tissue fluid onto a slide, as if preparing a smear for malaria examination, 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 concluded that they had inhaled

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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 Wagner's observations as an example, he 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 right off that Thomson's prediction 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. Too, 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 much with us. 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 5 deaths is due to lung cancer, one in ten is due to mesothilioma, one in ten to gastro-intestinal 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 community generally, 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 neoplasia hazard? An hypothesis could be formulated that such a threshold includes very little asbestos (since small amounts may still reflect billions of fibers and/or fibrils), that asbestos workers pass it early in their careers.

Such an hypothesis, or variations on it, would fit clinical observations made in the last several years. Neoplasms - lung cancer as well as pleural and peritoneal mesothelioma - occur in excess even among asbestos workers with little or no radiological evidence of asbestosis. It is now apparent that exposures insufficient to cause asbestosis may still produce neoplasia. The spread of this separation is not established, but is recognized by recent appreciation 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, if such a threshold indeed exists.

ASBESTOS IN LUNGS

It thus became important to know whether asbestos truly was a common contaminant of urban dwellers' lung. The demonstration that "asbestos bodies" were to be regularly found at autopsy in many cities of the world[16] confirmed Thomson's finding, but did not settle the question. There remained the nagging doubt that the cores were necessarily asbestos, an insecurity perpetuated by the technical difficulties involved in analyzing such cores.[17]

In recent months, the impasse has been resolved by avoiding the detour of asbestos bodies, with the 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 175u x $1cm^2$ of lung tissue

in each of these cases showed asbestos bodies in 1,449 (48.3%) (Table I). It is likely that, had a greater volume of tissue been submitted for study, asbestos bodies would have been found in all the 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. Fibers thicker than 1.0u 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 1.0u 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 with the number of asbestos bodies (Table 2).

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 the appreciation that the unique morphology of chrysotile allows its specific identification by high magnification electron microscopy.

Chrysotile fibers and/or fibrils were found in every specimen. (Figures 1,2). In 4 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 3). The morphological appearance and other characteristics of these fibers and fibrils and 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 percent of cases, but it was noted to be the most common and most abundant of all fibers detected.

The question inherent in Thomson's observations in 1963 "Is 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 paucity 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 percent 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 Wagner's cases, or Newhouse's 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 asbestos stigmata. This is rather urgently needed, with age, sex, residence and occupational exposure taken into account.

Studies now in progress in our Laboratory and elsewhere[25] indicate that suitable quantitative techniques for estimating asbestos lung content will be feasible and that fairly accurate estimates - approximating 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' lung 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.

Previous Epidemiologic Observations

An example of where an "obvious" explanation might also be inaccurate may be found in the assumptions adopted to explain Kiviluoto's 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.[26]

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 confidently be expected to have had the effect noted (the lapsed period between initial exposure and evidence of pleural calcification is 20, 30, 40 or more years).[27] 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.[28] It may be that similar explanations will become available for the finding of endemic pleural calcification in some rural areas of Czechoslovakia.[29,30]

Technical Factors

At first glance, 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 industrial circumstances, there is usually no problem in 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", etc.

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; but 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. (See below). Our own experience suggests that reticence is usually warranted when identifying single small fibers in ambient air samples, unless extended techniques are used.

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.[31] 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 non-fibrous and that the proportion could vary widely according to the materials used, process studied, etc. 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 3 and 4 show particles of clay and a diatom collected near a construction site during sampling by our Laboratory. 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 the particles. The question of size and magnification are critical.

Fiber-fibril

Each asbestos fiber variety is quite different chemically, physically, structurally and morphologically.[32] 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 300Å-400Å (Figure 5). These unit fibrils cannot be seen with the optical microscope. The intact chrysotile fiber can be seen. Under industrial circumstances, this dichotomy is understood. It is recognized that when a population of fibers is counted, "invisible" fibrils are also present,[33] but that the optically visible fibers reflect, in varying proportions under different circumstances, the total chrysotile population, even though there be but 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, 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. Once again, we return to the problem of ultramicroscopic

size, since the proportion of fibers to fibrils cannot be determined without the electron microscope.

Size

It may be seen from the foregoing that 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 20,000X (direct) and probably over 30,000X. 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 1000Å 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"[34] is surely with us!

Quantitation

Under industrial circumstances, again, 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 appreciated. 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. First, to obtain a measure of the mass of asbestos per unit air volume at various locations in urban centers and later, for comparison 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. In such circumstances, 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, as the biological effect of a sample dominated by large fibers may be overestimated.

Second, 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 0.8 or 1.2 microns. (Millipore AA or RA filters were used). 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 cubic feet per minute through an 8" x 10" filter and small battery operated personnel monitoring samplers with a capactiy of 2 liters per minute through 8 cm^2, 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.[35] 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 2 to 5 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 so prepared of the dispersed chrysotile is scanned at 42,000X in an electron microscope. Typically, six grids are prepared of each sample and three 100 by 100 micron squares of each grid scanned. The mass of asbestos is obtained by measuring the volume of asbestos per grid square and multiplying by the appropriate density. Figure 6 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 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. Such a group is shown in Figure 7. 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

Table 4 gives the ranges of values for ambient air levels 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. 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 x 10^{-9} grams per cubic meter 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 300Å to 400Å in diameter and often 2000Å or smaller in length. Thus, 10^{-9} grams of chrysotile asbestos could represent a million fibrils.

That Manhattan has higher levels of asbestos that other boroughs is to be expected, as greater use of asbestos in building construction takes place in that borough. During the past 10 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 wide spread areas of Manhattan. This practice was of such obvious concern that the New York City Department of Air Resources instituted stringent regulations controlling the procedure in May, 1970.[36]

Table 5 records findings on several days at various sites in lower Manhattan near buildings under construction. Data were obtained prior to the implementation of New York City's regulations. During the two days on which data were obtained sites 1 and 4 were downwind from a spray source, 3 and 5 were upwind from any source, and 2 was 45° from one source and upwind from others.

The data in New York are being supplemented by measurements in other urban and more rural areas. Initial data are shown in Table 6. The sampling locations in Philadelphia were near sites where spraying was taking place but after the issuance of control regulations.

These data are preliminary. They serve to establish that, at least in the areas sampled, there is a background of chrysotile contamination of the ambient air and that this is considerably 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. For example, the pollution which might exist about a shipyard or an asbestos mill can hardly be equated with that in the ambient air of an urban community. It would be well, at least until much more is known of the dose-disease response relationship, to separate each type of asbestos air pollution, in the study of associated disease.

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[37] co-factors 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. Other examples could be given. The construction industry, for instance, employs over 4,000,000 men in the United States. They live in all areas, in all types of communities. Asbestos disease among them may not be entirely the result of asbestos air pollution in those communities. Special circumstances have also occurred. During World War II, shipyard employment rose rapidly in the United States, with close to 1,750,000 people engaged in ship building and repair at peak periods. These people, or at least those still alive, are now reaching 30 years from onset of shipyard employment and such asbestos disease as they might have as a consequence of asbestos exposure during their shipyard working days, will now be seen. Yet few of them are still employed as shipyard workers, so that unless this previous employment history is extracted, current disease could be improperly attributed to asbestos air pollution of various other sorts.

Stigmata of Asbestos-Associated Disease

The matter is further complicated by consideration of what is meant by disease caused by asbestos air pollution. Most of our current concepts are derived from the experiences of occupationally exposed workmen. While the same kind of disease can be seen in people not occupationally exposed, its attribution is much more difficult. It is complicated, moreover, by the necessity for separating those effects of asbestos which may be present but which do not actually constitute ill health, from those effects which should be categorized as disease.

Pleural plaques and pleural calcification, for example, are often associated with no disability, even when impressively extensive on roentgenograms.[1,3] The same could be said for the mere demonstration of asbestos fibers and asbestos bodies in lung and for scattered areas of minimal fibrosis associated with the asbestos fibers in the lung.

Neoplasia is a different matter. Here, there is considerable concern. Those neoplasms which have been found associated with occupational asbestos exposure have included bronchogenic carcinoma, pleural mesothelioma, peritoneal mesothelioma. Gastrointestinal cancer, oro-pharyngeal neoplasms[14] and other neoplasms are also under investigation, but more data are needed before their association can be considered firmly established.

There has been the problem of identifying neoplasms associated with environmental asbestos exposure against the background of random neoplasms of the same kind, not due to asbestos exposure. Lung cancer, for example, is highly associated with cigarette smoking and the added influence of environmental asbestos exposure is not easy to define. There is an advantage in studying mesotheliomas, since evidence suggests that, apart from asbestos exposure, it has been a fairly uncommon disease. Yet even here more data are needed. It would be good to know, for example, exactly what the incidence of mesothelioma has been in the past 70 years, during decades in which the neoplasm was often not considered by pathologists. Too, it would be useful to have information on the percentage of mesotheliomas which occurs other than with asbestos exposure. With such refinements, we will be able to use mesothelioma as an accurate index of asbestos disease under various environmental conditions.

Sources and Control

Sources for asbestos air pollution can be looked at in two ways. First, 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.

Both approaches are hampered by inadequate information at this time of the relative significance of peak exposures compared to constant background contamination. There are clinical experiences which indicate that heavy exposure 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.[38] 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. 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. It may be of interest to recollect that the original sources for chrysotile asbestos used by the company which later became the Johns-Manville Corporation, were in small deposits on Staten Island, in New York City.

Such surface deposits have long been known to geologists and are sometimes used as a guide in their explorations. The tale is told of fortuitous forest fires initiated by lightning in the 1860's in Quebec, resulting in clearing of brush overgrowth. This uncovered surface chrysotile-bearing serpentine formations, leading to the discovery of the valuable Quebec chrysotile fields.

Abrasion and weathering of such surface formations may be accompanied by release of chrysotile fibers into the surrounding air. It is expected that this has occurred. It is not known, however, whether this adds a measurable burden to the ambient air, but it should be considered as one source in the evaluation of ambient air levels. It is apparent that quantitative studies in this regard would be helpful; we are investigating the chrysotile content of ice core samples, accurately dated for many years past, in one study. Other approaches would be useful, and can be designed.

Soil contamination has already been mentioned (see above) and epidemiological data have been collected indicating that human exposure from this source can produce pleural changes.[27,28] Wider dissemination from this source is also possible, and should be investigated. Parenthetically, ground waters coursing through asbestos-bearing rock formations may be expected to become contaminated by random fibers, and water from such sources could contain occasional fibers, without invoking the necessity for asbestos air pollution.

Industrial and Commercial Sources

Although natural sources for asbestos air pollution should be considered, it is likely that they add but 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, and their weathering and ultimate disposal as waste. The relative contribution would vary with the type of product. In general, the potential for pollution varies with the degree of fixation of the fiber in the product. Under circumstances in which asbestos is loose or the material friable (asbestos cements, insulation materials, etc.) the likelihood of fibers becoming airborne is greater; conversely, where the fibers are more or less bound in the product's matrix, the likelihood is considerably decreased (asbestos cement materials, asbestos containing plastics, etc.). The spectrum of this variation requires detailed study, since control efforts will be guided by the findings.

Such analyses might well be focused, at the outset, in the construction industry. 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. Our experiences suggest that much education on appropriate methods for containing asbestos in commercial use will be necessary; here, the asbestos industry has both important responsibility and opportunity.

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Illustrations

Table I

Asbestos Bodies in 3,000 consecutive autopsies, N.Y.C. 1966-1968

Age	Male	Female	Total
<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%)	775/1488(52.1%)
80+	106/186 (57.0%)	83/155 (53.5%)	189/341 (55.4%)
	1013/1971(51.4%)	436/1029(42.4%)	1449/3000(48.3%)

A. Analysis by age.

	Asbestos Bodies				
	0	1-4	5-14	15+	TOTAL
<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	1488
80+	152	156	40	3	341

B. Analysis by sex.

	Asbestos Bodies				
	0	1-4	5-14	15+	TOTAL
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)
	1,551	1,194	192	63	3,000

Table II

Inorganic fibers (optical microscopy) in lungs of 3000 consecutive autopsies, NYC, 1966-1968. Correlation with asbestos bodies.

A.

Asbestos Bodies	Thin inorganic fibers			
	0	1-4	5-14	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%)
	1,962 (65.4)	865 (28.8)	125 (4.1)	48 (1.6)

B.

Asbestos Bodies	Thin inorganic fibers				
	0	1-4	5-14	15+	TOTAL
0	1,168 (75.3%)	332 (21.4%)	35 (2.3%)	16 (1.0%)	1,551 (51.7)
1-4	705 (59.0%)	424 (35.5%)	52 (4.3%)	13 (1.1%)	1,194 (39.8)
5-14	76 (39.6%)	89 (46.3%)	21(10.9%)	6 (3.1%)	192 (6.4)
15+	13 (20.6%)	20 (31.7%)	17(27.0%)	13(20.6%)	63 (2.1)

Table III

Chrysotile in 28 Cases Studied

Group	Numbers of Chrysotile Fibers + Fibrils	Cases	Male	Female
1	= or < 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	= or > 201	3/28	3	0
"Blank Grids"	= or < 9	---	---	---

Table IV

Chrysotile Content of Ambient Air in N.Y.C.
Preliminary Results

Sampling Locations	Asbestos air level in 10^{-9} grams/m^3
Manhattan	25-60
Bronx	25-28
Brooklyn	19-22
Queens	18-29
Staten Island	11-21

Table V

Chrysotile content of N.Y.C. air in vicinity of spray fireproofing with asbestos-containing materials

Site	Asbestos level in 10^{-9} grams/m^3
1- Downwind from source	45-180
2- 45° from source	15-30
3- Upwind from source	20
4- Downwind from source	45
5- Upwind from source	20

Table VI

Chrysotile content of air in three selected locations

Location	Asbestos level in 10^{-9} grams/m^3
Philadelphia, Pa.	45-100
Ridgewood, N.J. (suburban)	20
Port Allegany, Pa.*	10-30

*We have also found amosite fibers in the air of this community; a factory using this material is present.

Fig. 1

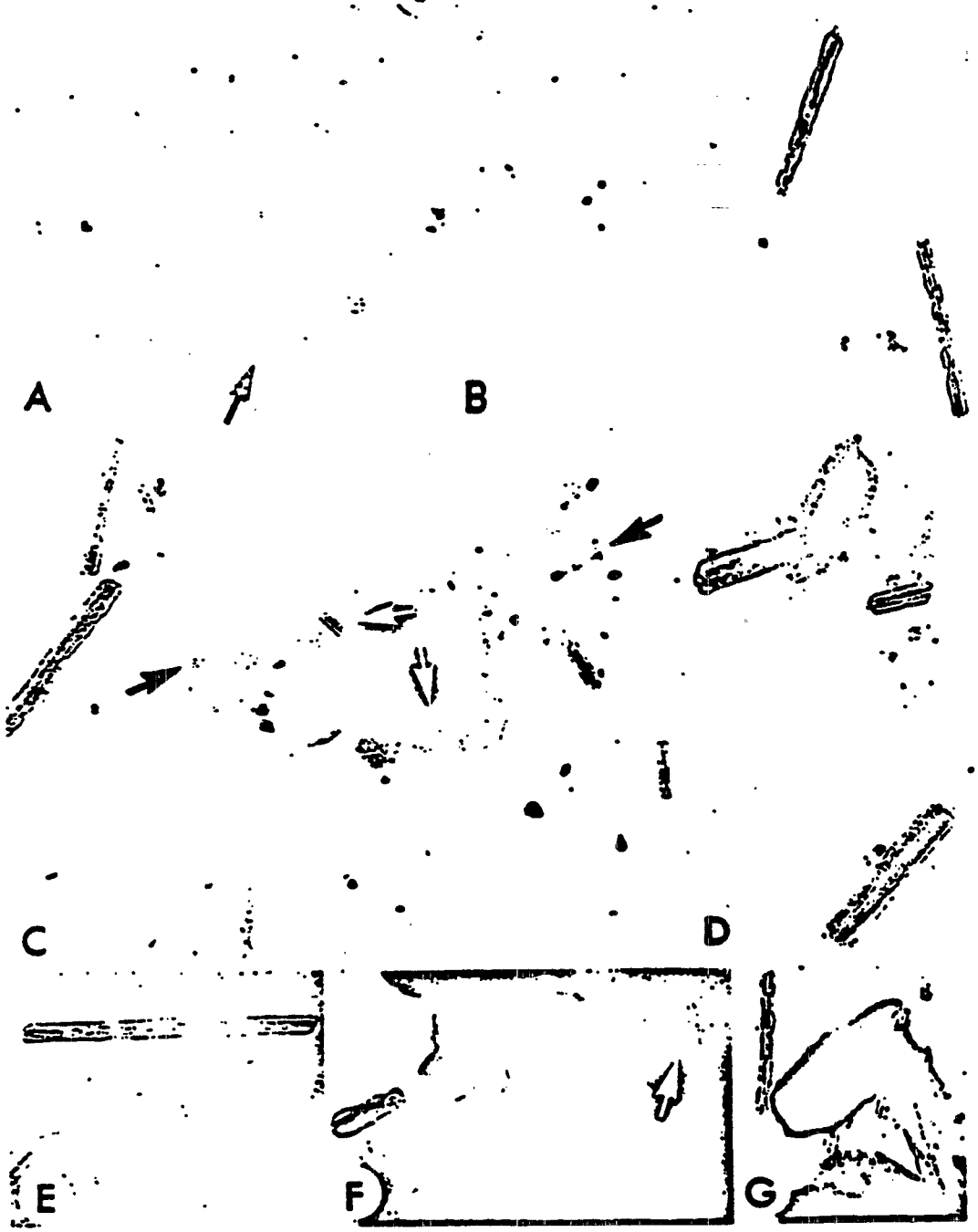

Fig. 2

Fig. 3

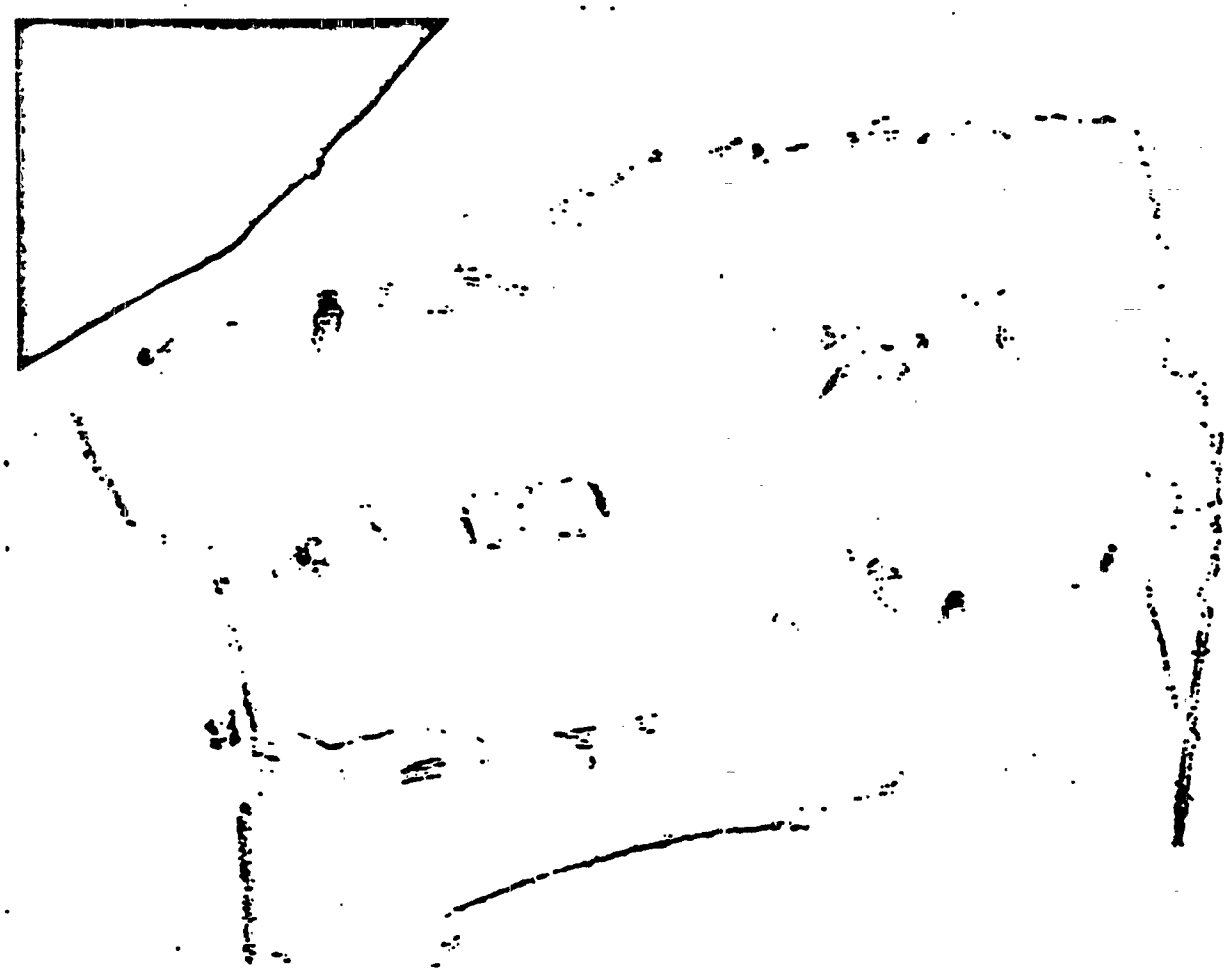

Fig. 4

CHEV BB

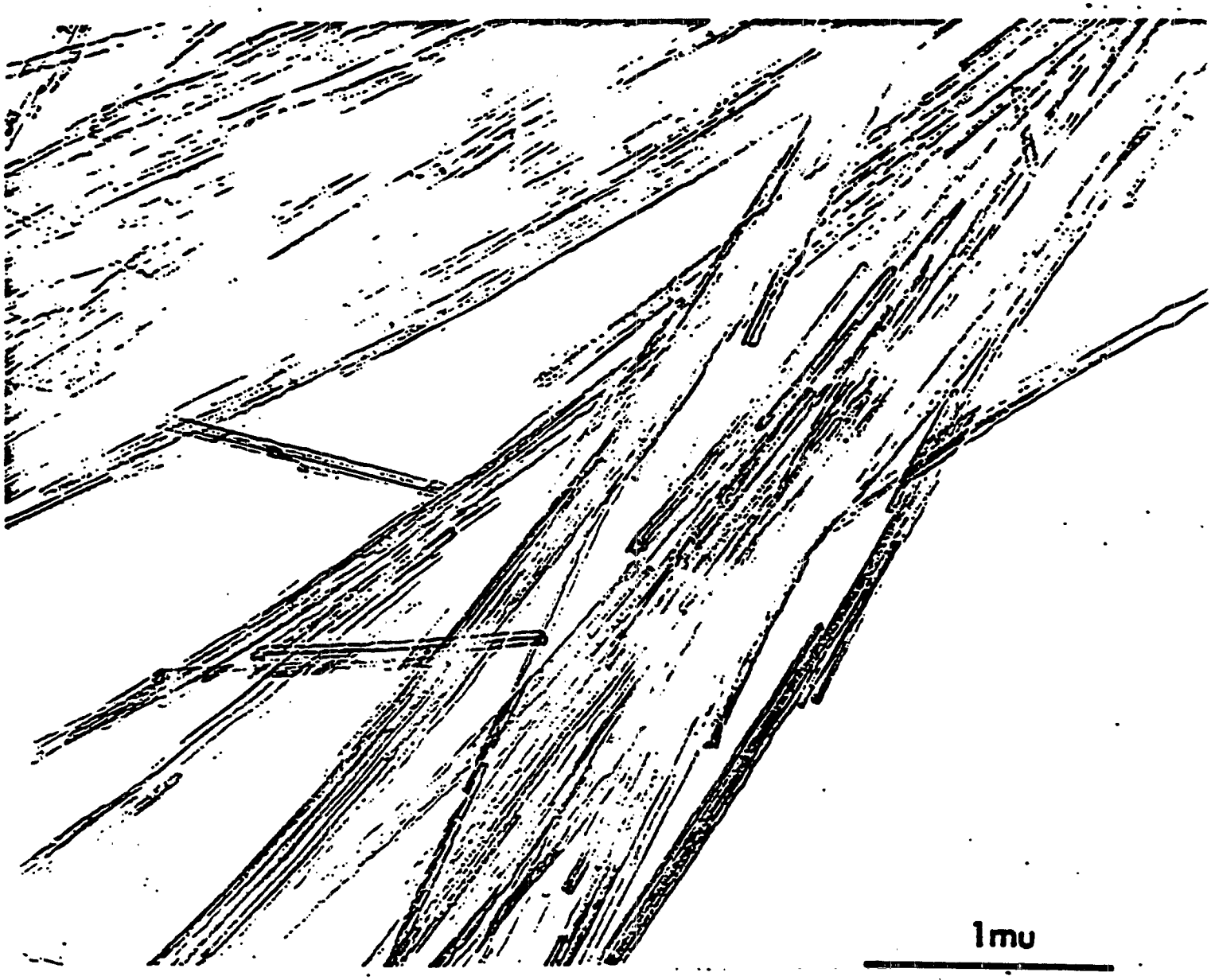

Fig. 5

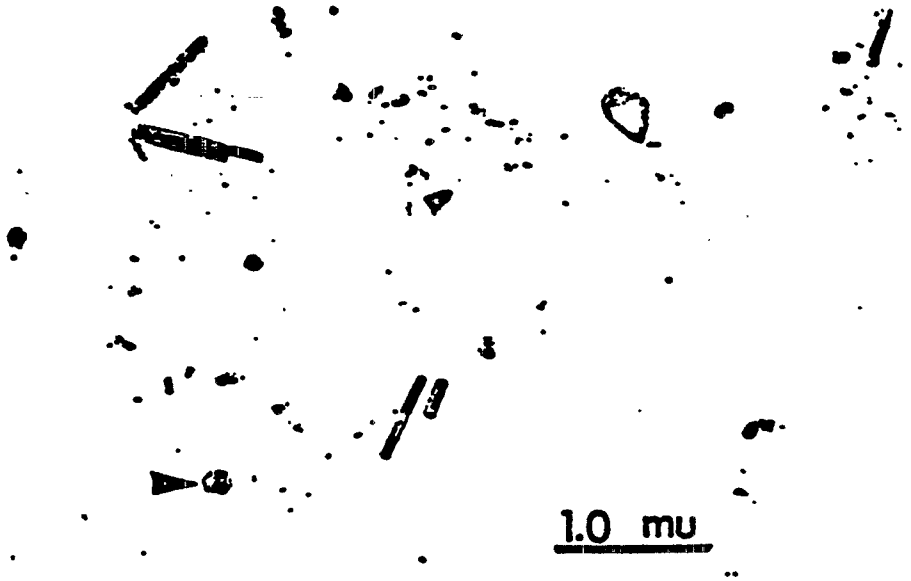

Fig. 6

0.4 mu

Fig. 7

The following items are suggested when purchasing a phase-contrast microscope for use in evaluating airborne asbestos dust for comparison with the A.C.G.I.H. Threshhold Limit Value (T.L.V.) 1970.

1. Microscope body with a binocular head and a fine focus accuracy of .005 mm.

2. 10X Huygenian eyepiece (s)

3. Porton reticle

4. Mechanical stage

5. Koehler illumination (preferably built in and having provisions for adjusting light intensity)

6. Abbe condenser with an adjustable iris

7. 40 - 45 X (.65 N.A. at least) Positive (bright field) phase-contrast objective

8. Annular ring condenser diaphragm (corresponding to the objective)

9. Phase ring centering telescope

10. Green filter

Note:

Most manufacturers sell a basic body unit and built-in illumination system as a unit.

Phase-contrast accessories can usually be purchased as a kit usually consisting of a 10X, 40X and 90X objective, an Abbe condenser containing appropriate annular ring diaphragms, a phase ring centering telescope, and a green filter. It is to the microscopist's advantage to purchase the kit.

A list of manufacturers of phase-contrast microscopes is given here to aid in selecting a proper instrument.

Bausch and Lomb
Scientific Instrument Division
72624 Bausch Street
Rochester, New York 14602

3/72

TF 110.1 Asbestos

Hacker Instruments
Box 646
West Caldwell, New Jersey 07006

E. Leitz Inc.
Rockleigh, New Jersey 07647

Nikon Inc.
Instrument Division
Garden City, New York 11530

Olympus Microscopes
Micro Optics Company
28165 Greenfield
Southfield, Michigan 48075

American Optical Corporation
Reichert Products
Buffalo, New York 14215

Unitron Instrument Company
Microscope Sales Division
66 Needham Street
Newton Highlands, Massachusetts 02161

Carl Zeiss
444 Fifth Avenue
New York, New York 10018

Additional Information on airborne asbestos dust evaluation is available in the paper, Equipment and Procedures for Mounting Millipore Filters and Counting Asbestos Fibers by Phase Contrast Microscopy by S. G. Bayer and R. D. Zumwalde.

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