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Although a group of materials referred to generically by the term "asbestos" have been known and used, to a small extent, far back in antiquity, widespread and abundant use of these fibers did not begin until the last quarter of the nineteenth century, with further augmentation of production during the first half of the twentieth century. There are six distinct varieties of asbestiform fibrous minerals. Among some of these, there are striking differences in chemical and physical composition (1). Most commonly produced is chrysotile, a member of the serpentine family. The amphiboles are subdivided into crocidolite (blue asbestos), amosite, anthophyllite, tremolite, and actinolite. All are silicates, crystalline in nature, and characteristically are divisible into very small fibrils. Chrysotile and anthophyllite contain much lesser amounts of magnesium than either crocidolite or amosite, and the last two contain much more iron than the others. The crystalline structure and surface properties, which may be of importance in the biologic reaction, are also strikingly different among the varieties of asbestos (1). In view of the great variation of chemical and physical properties, it is most unsafe to predict that the biologic reactions of one variety of asbestos will be mimicked by another in terms of either actual consequences or mechanisms. To interpret the biologic actions of asbestos, it is imperative that the character of the exposure in terms of concentration, size, and types of fibers be known. This sort of data is scant or often nonexistent at present with respect to exposure of humans.

Pulmonary fibrosis: In 1907, Murray (2) reported the first cases of diffuse pulmonary fibrosis discovered among a group of persons who had experienced substantial and prolonged exposure to respirable-sized asbestiform fibers. Subsequently, the term "asbestosis" was applied to this reaction. Traditionally, asbestosis has connoted this kind of reaction and its sequelae with regard to altered pulmonary and cardiovascular function. Recently, the term has been corrupted to include such manifestations as pleural thickening, pleural plaques, and even the simple presence of ferruginous bodies. Resolution of the use of the word asbestosis must be obtained to bring clarity to scientific reporting.

Murray's report revealed the paradox of a life-saving agent possessing the capacity for human harm. At first, the conditions under which this biologic reaction came about seemed to be restricted to those created during fabrication, conditions limited to textile manufacturing at that time. In 1930, Merewether and Price (3) demonstrated that asbestosis posed a major threat to the health of workers in the asbestos textile trade. It is now known that in any of the several trades in which persons experience prolonged exposure to a high concentration of respirable-sized asbestiform fiber, it is probable that some of them ultimately will develop pulmonary fibrosis.

In response to the report of Merewether and

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[2] Presented before the Medical Session, as part of *Section 5A*, at the annual meeting of the American Thoracic Society, Miami Beach, Florida, May 25, 1960.

Price, the fabricators of asbestos-containing products took substantial measures to reduce the concentration of airborne fibers in the factories. The report of Sayers and Dreesen (4) in 1939 further stimulated the introduction of such corrective measures, especially in factories of the U.S. and in mines and mills of Canada where 95 per cent of the free world's asbestos was produced. Regrettably, substantial numbers of persons exposed to asbestos in their occupation have never come under a protective occupational hygiene program. Insulation applicators and some individual or small business fabricators fall into this category.

The preventive efforts that were applied led to considerable improvement in the factories, mills, and mine environments and a subsequent lessening of the occurrence and severity of pulmonary fibrosis. Unfortunately, the relaxation of precautions governing occupational health, common under the stresses of World War II, seriously interfered with many health protection programs, including that for the safe use of asbestiform fibers. As a result, the protection of workers using asbestos in mining and milling and especially in factory settings has not been as good as it might otherwise have been.

Until quite recently, the threshold limit value (TLV) for environments containing asbestiform fibers suggested tentatively by Sayers and Dreesen (4) in 1939 has served as a guide. Data entirely adequate for the purpose were not available in 1939, and it has taken years to recognize this inadequacy and to obtain the information upon which to base a more meaningful TLV. Fortunately, one of the major users of asbestos in the production of textiles and textile products has pursued for many years a program of environmental and population monitoring that now provides a more up-to-date experience (5, 6). These studies demonstrate that pulmonary fibrosis can be minimized when adequate protective measures are instituted, and that the concentration of airborne fibers must be the focal point of attention even though the technology of such monitoring may be new, difficult, and expensive. The U. S. Public Health Service, which began extensive studies into the problems of asbestos and health in 1961,[3] together with asbestos producers and manufacturers in the U.S. and Canada are at this time examining their experience with population exposure and pulmonary fibrosis during the past 30 years. It will be of interest to learn how their findings compare to those of the United Kingdom. A provisional TLV expressed in concentration of respirable-sized asbestos fibers has been set recently in the United Kingdom, and a slightly different value has been established in the U.S. (6, 7). Whether these are proper for all varieties of asbestos and all settings in which human exposure might occur is debatable. There is considerable evidence to suggest that chrysotile penetrates to the deep lung spaces less readily and is cleared from the lung more readily than either amosite or crocidolite (8). Also, chrysotile causes less pulmonary fibrosis in experimental animals than amosite or crocidolite (9).

With regard to the prevention of pulmonary fibrosis, one would conclude in 1969 that in many settings the measures taken have not been adequate, and there are populations that have had little or no protection at all. The knowledge required to protect all workers is not available at present, and its development is one of the urgent tasks of the immediate future. Nevertheless, an important step has been taken toward further defining the conditions necessary for the prevention and ultimate abolition of significant pulmonary fibrosis in persons exposed to asbestos fiber.

Bronchogenic cancer: Although in 1935 Lynch and Smith (10) reported bronchogenic cancer in 2 patients with asbestosis, it was not until 1947 that the possibility of this second biologic influence of asbestos fiber was seriously raised, when Merewether (11) demonstrated bronchogenic cancer in 13 per cent of autopsies of persons who had asbestosis arising from employment in the textile trade. Eight years later, Doll (12) reported a frequency of lung cancer of ten times the expected rate in textile workers exposed for at least 20 years, during the period between 1922 and 1955. This experience has been confirmed in the textile trade by Newhouse (13), Enterline and Kendrick (14), and Mancuso and El-Attar (15). More recently, Selikoff and co-workers (16) reported a frequency of bronchogenic cancer eight times that anticipated in a group of insulation workers employed for more than 20 years who utilized a substantial portion of their time removing old or applying new insulation that contained asbestos. This

[3] Cralley, L. J.: Personal communication.

series of reports suggests that asbestiform fibers play an undetermined role in the augmented frequency of bronchogenic cancer in some categories of workers. In none of these categories, however, has there been a full exposition of other substances which coexist with asbestos in the environment and to which the worker has been exposed (5, 6, 17, 18). It is possible, therefore, that coexisting materials possessing carcinogenic potential might be implicated. It would be prudent to explore this possibility thoroughly before accepting asbestos per se as the carcinogenic factor.

In asbestos textile mills, the exposure involves primarily two varieties of asbestos, chrysotile and crocidolite. In some factories, oil has been applied to the fibers before final processing. Moreover, in the process of "opening" the fibers, a strong possibility exists that traces of metals known to have carcinogenic influences may have been added to the fibers (19–21).

Insulation workers have been exposed not only to chrysotile and amosite but also, especially in shipyards, to crocidolite. The nature of their work, as they remove old insulation and apply new in various places, exposes these workers to many nonasbestos substances, some of which may be hydrocarbons adsorbed onto the asbestos fiber. The exposure history of insulation workers is seriously lacking as to specific details of all of the materials that they may have inhaled in addition to asbestos.

Of the numerous questions which could be posed concerning the relationship of asbestos and bronchogenic cancer, the following three are of immediate practical importance: (1) Is the influence dose-related? (2) Is it related to variety of asbestos? (3) Does the relationship involve cocarcinogenesis? Some evidence bearing upon these questions is now available, but in no instance is it complete.

Data concerning the question of dose relationships do exist. The type of work related most strongly to the development of bronchogenic cancer has been in the textile and the insulation applications trades: the worker, in most instances, has been employed in either trade for 20 or more years. Merewether and Price (3), and Selikoff and associates (21) report an incidence of asbestosis (pulmonary fibrosis) in these groups of 80 per cent among persons exposed for 20 or more years. This indicates a heavy and prolonged exposure to asbestos for these two types of worker. Knox and associates (5), in a continuation of the study of textile workers previously reported by Doll (12), show that the incidence of lung cancer decreases with progressively smaller total exposures to asbestos fibers. Newhouse (13), also in an asbestos textile population, has shown that the occurrence of pulmonary cancer differs markedly with total exposure. She reports no increase of the incidence of bronchogenic cancer in a low to moderately exposed group, whereas in a heavily exposed group the incidence was augmented eightfold. Both groups were from the same textile mill and were similar with respect to duration of exposure and the length of time of observation. Enterline and Kendrick (14) demonstrated a stronger relationship between lung cancer and employment in the asbestos textile trade than exists in the less dusty manufacturing of asbestos building products or building materials. In a series of studies, Mancuso and El-Attar (15) suggested that cancer risk is lower with lesser exposures to asbestos. From these observations, the inference strongly favors a dose relationship between bronchogenic cancer and whatever effect asbestos fibers may ultimately be shown to have in its causation.

The question of whether the carcinogenic role of asbestos is related to the specific kind of fiber is more difficult to answer. For this purpose, populations having long-term exposure to only one variety of fiber are needed. Virtually all fabricating processes use more than one variety within the same premises at any one time or have used more than one variety at various times. Populations restricted to mining and milling in the production of asbestos fiber lend themselves best to a study of the effects of a single kind of fiber, and it is to these populations that one must look for an answer to the influence of a single variety of fiber.

Kiviluoto and Meurman (22) showed that exposure during the production of anthophyllite in Finland carries with it a threefold to fivefold increased risk of bronchogenic cancer for those in the age group older than 45 years with more than 10 years of exposure. At this time, there are no published data with respect to the frequency of bronchogenic cancer in the

crocidolite or amosite production areas of South Africa, the only place in the world where appreciable amounts of these types of fiber are produced. A verbal report[1] from South Africa suggests that the frequency is somewhat higher than anticipated in the general population of that country. Data from these regions will be awaited with great interest. In 1958, Braun and Truan (23) reported a study of the frequency of lung cancer in workers of the mines and mills producing only chrysotile in the eastern part of Quebec, Canada. From the data available to them at that time, they found no evidence of an augmented frequency of bronchogenic cancer in this group of employees. Although the period of observation was only five years and the group was diluted by short-term exposures, it contained more than 1,700 persons employed longer than 20 years, all of whom had had at least five years of exposure and 930 of whom were older than 55 years of age. The data would have been more meaningful if arranged in terms of total exposure and time from beginning of exposure. Nevertheless, if an excess of lung cancer equivalent to that seen in the settings referred to previously in this paper did actually exist in this population, it seems likely it would have been found. This same population is now being restudied after a much longer period of observation and in a more adequate way. Vigliani and associates (24) have found evidence of an augmented frequency of cancer of the lung and pleura in a group of 285 persons, previously compensated for asbestosis arising from all occupations, who died between 1943 and 1967. In a subgroup of 32 miners exposed to chrysotile only, there was no augmentation of the rate of cancer of lung and pleura. Although these studies suggest that chrysotile has little or no carcinogenic influence, more extensive and refined epidemiologic data are needed before reaching that conclusion.

The possibility that the carcinogenic role of asbestos is solely that of a cocarcinogen is very strong. The data of Selikoff and associates (25) are especially pertinent in this regard, in that not a single bronchogenic cancer has been found in an insulation worker who was a noncigarette smoker, even though the exposure to asbestos experienced by the nonsmokers was the same as that of those who smoked and did develop lung cancer.

[1] Webster, I.: Personal communication.

The considerable variation in the lung cancer incidence between populations of differing types of employment may be solely related to dosage, but it may also be the result of differing cocarcinogens. Gross and associates (26) produced a higher than anticipated frequency of pulmonary cancer in rats exposed to chrysotile. They attributed this variance, from the experience of others, to be related to contamination of the chrysotile fibers by nickel, cobalt, and chromium caused by the wear of the metal components of the mill used in preparing the fiber for administration to their animals. Cralley and associates (19, 20) showed substantial amounts of these trace metals in the asbestos fibers used commercially. Miller and associates (27) have shown that benzo (a) pyrene plus asbestos augments the yield of pulmonary tumors produced by benzo (a) pyrene alone. Harington and associates (28) raised the possibility that adsorbed hydrocarbons occurring natively or picked up in storage and handling may play a role in cocarcinogenesis.

As reviewed in 1969, the role of asbestos in augmenting the incidence of bronchogenic cancer appears to be dose-related, may be dependent upon the specific category of fiber, especially in relation to the degree of effect, and appears to be that of a cocarcinogen.

Mesothelioma: Subsequent to the earlier reports linking occupational exposure to asbestos and bronchogenic cancer, there were several suggestions that tumors of the peritoneal and pleural mesothelium might be similarly linked. Interest in this phenomenon remained dormant, however, until Wagner (29), in 1939, reported 20 cases of pleural mesothelioma associated with the Northwest Cape region of South Africa. He suggested that these were related to exposure to crocidolite asbestos because 14 of the persons were born or had spent their early youth near Kuruman, where crocidolite production is one of the major industries. This observation prompted an intensive search for all mesotheliomas in South Africa. Gilson (30) plotted the number and distribution of the South African cases up to 1962 and demonstrated a striking concentration of cases in the Northwest Cape. Although almost none was found to occur elsewhere in South Africa, the inference that the concentration of mesotheliomas coincided with the sole source of crocidolite in South Africa would not be warranted. A substantial quantity

of crocidolite has been produced for a period of many years, hundreds of miles away in the eastern Transvaal, the same area in which amosite and chrysotile asbestos are produced (31).

Investigators in other parts of the world have examined the distribution of mesotheliomas and the frequency of mesothelioma in populations occupationally exposed to asbestos, with the result that a strong relationship between exposure to asbestos fiber and the presence of mesothelioma has been indicated. Most of the cases outside South Africa, where mining and milling are implicated, have been reported in the United Kingdom and, to a lesser degree, in the U.S. In spite of a thorough search, no mesotheliomas have been found associated with anthophyllite asbestos production in Finland (22), and few are reported in France (32), where the subject has probably not been thoroughly investigated. In the United Kingdom, the association has been with populations of insulation workers and asbestos textile manufacturing factories (33). In the U.S., the relationship has been shown predominantly with insulation workers (16) and, to a lesser degree, with textile manufacturing employees (15). Recent study of the distribution of mesotheliomas in Canada will be commented upon later.

In most of the studies to date, the question has been raised as to whether the population at risk with respect to mesothelioma is to be found outside as well as inside the factory or place of work. There has been a plethora of speculation by students of the problem that often has been accepted as fact or interpreted as such by lay writers. These dramatic speculations have further compounded an already difficult matter for those responsible for public protection as well as those responsible for the health of employees. Controversial or paradoxic issues about mesothelioma and asbestos deserve a more complete discussion than is possible in this paper. Among the controversial features about the relationship of asbestos exposure to mesothelioma of the pleura and peritoneum are the following.

There is a lively controversy among pathologists regarding the requirements for establishing the diagnosis of primary mesothelioma (34). Not only is there a question with respect to the histologic criteria, but even more important, the necessity for establishing by autopsy whether the tumor is primary or secondary. It is apparent that the overwhelming number of cases thus far being related to asbestos are diagnosed on the basis of biopsy alone. Further study will be needed to demonstrate whether mesothelioma is being over or under diagnosed.

If one accepts that some kind of direct or indirect relationship between asbestos and mesothelioma exists, does it involve a specific variety of fiber? The data from the U.S. and United Kingdom does not lend itself to resolving this question because chrysotile, amosite, and crocidolite are involved in the insulation workers' exposure, and chrysotile plus crocidolite in that of the textile manufacturing employees. If one examines the mining and milling exposures to the specific variety of fiber, interesting facts emerge. Kiviluoto and Meurman (22) did not find a single mesothelioma in a large population of miners and mill workers exposed only to anthophyllite. This group worked for three months or more between the period of 1935 and 1967. Although others have stressed the long period of time required for mesothelioma development, one would think that if anthophyllite by itself is implicated, at least one case would have been found by now in a setting known to cause asbestosis and a somewhat augmented frequency of bronchogenic cancer. In a study of the distribution of mesotheliomas in Canada, McDonald and co-workers (35) were unable to show excess frequency in the geographic area where chrysotile, but no other variety of asbestos, is produced.

The South African distribution of mesotheliomas is unusually interesting. Webster (38) reports that before the end of 1968, 179 cases of mesothelioma were agreed upon as meeting acceptable criteria. Of these, the history of exposure was known in 148 patients, and in 24 there was no asbestos exposure. Fifty-one patients had been exposed during the mining and milling of crocidolite in the Northwest Cape only. An additional 52 patients with mesothelioma, not exposed occupationally, were associated in some way with residence in the Northwest Cape. Sluis-Cremmer (31) has indicated that about 20 per cent of all crocidolite production in South Africa actually comes from mines located in the Transvaal, a few hundred miles from the Northwest Cape. He also indicated that although the exposure may have been somewhat less intense in these mines than in those of the Northwest Cape, it nevertheless was

high. The writer's inquiries confirm this and indicate further that the mines of the Transvaal have been in operation for as many years as those in the Northwest Cape. The mechanism for obtaining data regarding mesothelioma in the Transvaal crocidolite area is identical to that for the Northwest Cape. Because not a single case of mesothelioma has been discovered from the crocidolite mines or surrounding communities of the Transvaal, although 51 cases have come from the crocidolite mines and 52 additional cases from the neighboring community in the Northwest Cape producing exactly the same kind of fiber, the contention that crocidolite per se causes mesothelioma becomes difficult to accept. Sluis-Cremmer (31) pointed out this paradox in 1964. Webster (36), in his current report on asbestos exposures in South Africa, likewise calls attention to this disparity.

It is of equal interest that only 3 cases of mesothelioma have been reported as originating from the Transvaal, these being from the amosite asbestos mines (36). No cases of mesothelioma have been reported from the chrysotile mines of the Transvaal. On the basis of what is known about the number of persons employed, the intensity and duration of exposure, plus lapse time, one would expect far more than 3 cases of mesothelioma if amosite had the same potential as the factor causing mesotheliomas in the Northwest Cape. The dosage and particle size have not been sufficiently different in the Northwest Cape and Transvaal to explain the striking absence or minimal occurrence of mesotheliomas in the Transvaal.

The discovery of such a striking concentration of mesotheliomas in the Northwest Cape suggests the need for a thorough study of the differences between this region and other areas of South Africa in terms of possible etiologic factors. That something other than, or in addition to, asbestos plays a role in mesothelioma formation seems inescapable. The geology and climate of the Northwest Cape differs considerably from that of the Transvaal. Trace metals, chemicals, or oils adsorbed onto asbestos may be factors. If crocidolite is implicated, it is likely to be in the role of a cocarcinogen, some additional agent providing the difference between the experience of the Northwest Cape and the Transvaal. Much more study needs to be made of this aspect.

The question of whether there is a tolerable exposure or threshold limit value for asbestos without contracting an enhanced risk of developing mesothelioma has been raised by the finding of what, on first inquiry, seemed to be minor exposures to the fiber in nonoccupational settings as a background for some of the cases of mesothelioma reported in South Africa (36) and the United Kingdom (33). Although originally it had been thought that there were many such examples of mesothelioma developing outside an occupation or occupation-associated environment, better and more complete exposure histories reduced this proportion greatly. At present, 80 per cent or more of the mesotheliomas thus studied in the United Kingdom and South Africa have been shown to have an occupational or paraoccupational association with asbestos fiber. The level of this exposure is substantial. Most cases occurring outside actual "on the job occupational exposure" occur through an exposed worker living on the same premises, who brings fiber into the home on his clothing, or through contamination of the environment surrounding an industrial operation which has cast airborne fiber into the atmosphere for years and thus exposed persons living or working in such a neighborhood. This was especially exemplified in the Northwest Cape region of South Africa where roads were made of asbestos-bearing waste rock, where women were employed in hand cobbing or in gleaning asbestos fiber from the waste heaps, and where the mill disseminated blue fiber quite widely around its location. There is reason to believe that these various types of exposure have been substantial in the population thus involved during the past 40 years.

With respect to neighborhood or paraoccupational exposure to asbestos in the U.S., it is of interest that Borrow and co-workers (37) called attention to the fact that none of their series of mesothelioma, gleaned from a community medical practice, fell into this category of exposure.

Is it reasonable to believe that the cases of mesothelioma in which no occupational or paraoccupational asbestos exposure can be disclosed, amounting to less than 20 per cent of those reported from the United Kingdom and South Africa, in fact represent a reaction to the casual exposures which might be experienced by the public at large? In the United

Kingdom, where an intensive search for mesothelioma has been pursued actively during the past four years, fewer than 100 cases thus far have been discovered in persons who have not had a known substantial exposure to asbestos (33). If one adds to this a portion of the 132 cases about which there was no information, the number of persons who have not had a discoverable substantial exposure, but in whom a mesothelioma has developed, is less than 200. This number is such a minuscule proportion of the adult population from which the cases were gleaned that it becomes difficult to entertain the view that the asbestos fibers that may be in the air and respired in a casual fashion by the public at large pose a genuine carcinogenic threat.

If one postulates that there are respirable-sized fibers of asbestos in the ambient air breathed by the general public, the strikingly rare occurrence of mesothelioma not related to occupation strongly suggests that there is a tolerable level of airborne asbestos fiber which does not cause an undue risk of development of mesothelioma. The findings of Newhouse (13) and McDonald and associates (35), which indicate that a genuine occupational exposure to asbestos has been tolerated without evidence of an increased risk of developing a mesothelioma, suggest that the tolerable level is substantial. The evidence strongly favors the concept that the casual exposure of the general public has not exceeded such a level in the past.

Ferruginous (asbestos) bodies. In spite of the foregoing considerations, additional developments have caused some investigators to suggest that an epidemic of mesotheliomas will occur among the general public in the near future (38). This apprehension is based on the finding of Thomson and associates (39), confirmed by several others, that structures identified as being similar to asbestos bodies (commonly seen in persons known to have an intensive exposure to asbestos fibers) are found in 25 per cent or more of the adult general public by direct examination of lung tissue or of the juices pressed from such tissue. The frequency of occurrence of these bodies in the lungs of the general public apparently depends upon the volume of lung tissue examined and the intensity with which the search is made, because Uridiian and co-workers (40) report that in their series almost 100 per cent of the population exhibited these bodies. These structures have been assumed by many to represent coated asbestos fibers and, therefore, to indicate an exposure to the inhalation of asbestos fiber, hence a higher risk of developing mesothelioma. Several facts militate against this conclusion.

The coated fibers found in the lungs of the public have not been proved to be asbestos fibers. When they are found in persons who have experienced a heavy exposure to the inhalation of asbestos fiber, it is reasonable to assume that the bulk of these bodies do represent asbestos fiber. It has been shown, however, that other materials which are fibrous in nature will produce bodies of an identical appearance (41). Cralley and associates (42) have listed 20 mineral materials that are fibrous in nature and that may be widely disseminated in the environment of the general public. Because of the numerous other types of fibers that may, and almost certainly do, gain access to human lungs where they become coated with an iron-protein complex, Gross and co-workers (43) suggested the term ferruginous bodies for these structures and suggested that they be called asbestos bodies only when it is known with certainty, or can be presumed on the basis of known exposure, that the central fiber of the body is in fact asbestos. This would appear to be a far more accurate designation and scientific attitude than to insist on calling all of these bodies asbestos bodies whether or not they are known to be such.

The literature concerning ferruginous bodies has become quite large, and sweeping inferences from their presence in the general population have been made (38, 44). Without attention to the necessary quantitative and qualitative factors, it has been predicted that the experience of the general public with respect to mesothelioma will parallel that which has thus far been disclosed in certain very specific occupational and paraoccupational settings. A number of facts indicate a great need for caution before arriving at such a serious conclusion. To transfer the experience of a specific occupational or paraoccupational population to the general public requires evidence that the exposure of the public does, in fact, corre-

spond to those populations in which an increased risk of mesothelioma is said to exist. This is especially true now that it has been demonstrated that there are some occupational populations in which the exposure to asbestos fiber exceeds many fold that of the general public and in which no evidence of an unusual mesothelioma risk was found (13, 22, 35). What is the present situation in this regard?

As indicated in foregoing discussions, conclusions with regard to the relationship between mesothelioma and asbestos fiber per se, even in an occupational setting, are rather uncertain at this time. The roles of types of fiber, cocarcinogenesis, and dosage are so far from being clear in these special occupational settings as to give little assurance that they can be transferred in any meaningful way to the nonoccupational related or general public populations.

Although a scant number of ferruginous bodies has been demonstrated in lungs from the general public, there has been no demonstration that these are in fact the result of exposure to asbestos fiber rather than to one or more of the many other materials known to be capable of producing ferruginous bodies. At this time, the determination of whether the fibrous core of a ferruginous body is in fact asbestos is extraordinarily difficult and quite beyond the means of other than the most sophisticated laboratories. In spite of this, the necessary investigation must be done before the contention that all ferruginous bodies in the general public are in fact asbestos fibers can be accepted.

Quantitative estimates indicate that the number of ferruginous bodies found in the lungs of the general public is a small fraction of the number found in the lungs of those who had been occupationally exposed. If one accepts the contention that ferruginous bodies reflect asbestos exposure, one must conclude that the general public has had far less exposure than those persons in occupations using asbestos fiber. That some persons with known occupational exposure to asbestos fiber appear to have but few ferruginous bodies complicates an interpretation of quantitative measurement of ferruginous bodies. It is clear that much more comprehensive and reliable data with regard to the dynamics and the quantitative and qualitative aspects of ferruginous bodies is necessary before any meaningful interpretation of their importance for the general public can be made.

The finding of ferruginous bodies in a large proportion of the general population has led to the contention that the augmented use of asbestos in the manufacture of products has greatly increased the degree to which the general public has been exposed to this fiber. This assumption has been based upon the simple statistic of asbestos consumption, with no attention being paid to how the fiber is used nor the degree to which its ultimate fabricated form would lend itself to dissemination of fiber among the public (30). Although it is possible that augmented consumption of asbestos fiber led to a greater output of these fibers from factories into the air surrounding the factory, this does not constitute a generalized public environmental exposure. Tabershaw (45) has pointed out that asbestos is used for the most part in fabrication of materials that are unlikely to become airborne. It is not widely appreciated that the bulk of the products using asbestos actually bind the fiber firmly in the product, and there is subsequently very little opportunity for release of this fiber except by slow wear of the products.

The estimated consumption of asbestos fiber in the U.S. has increased from 125,000 short tons in 1918 to 720,000 in 1965; most of the increase occurred after 1940.[6] Once the fiber, in the form of a product, is put into place, it is not released except by gradual wear, which in most examples is minimal. During factory processing, the public at large is not exposed.

During the use of certain products, such as insulation removal and application, fibers are released and those in the immediate vicinity, workmen and public, may be exposed. This category of product can be thought of as having fibers that are "loosely bound," in contrast to such products as asbestos cement or floor tile which actually incorporate the fibers and bind them very tightly. The quantity of asbestos fiber used for "loosely bound" products, including insulation (figure 1), has increased from 39,000 only to 55,000 short tons between 1920 and 1965.[6]

The commercial grade of fiber indicates the kind of products in which it will be used.

[6] Pundsack, F.: Personal communication.

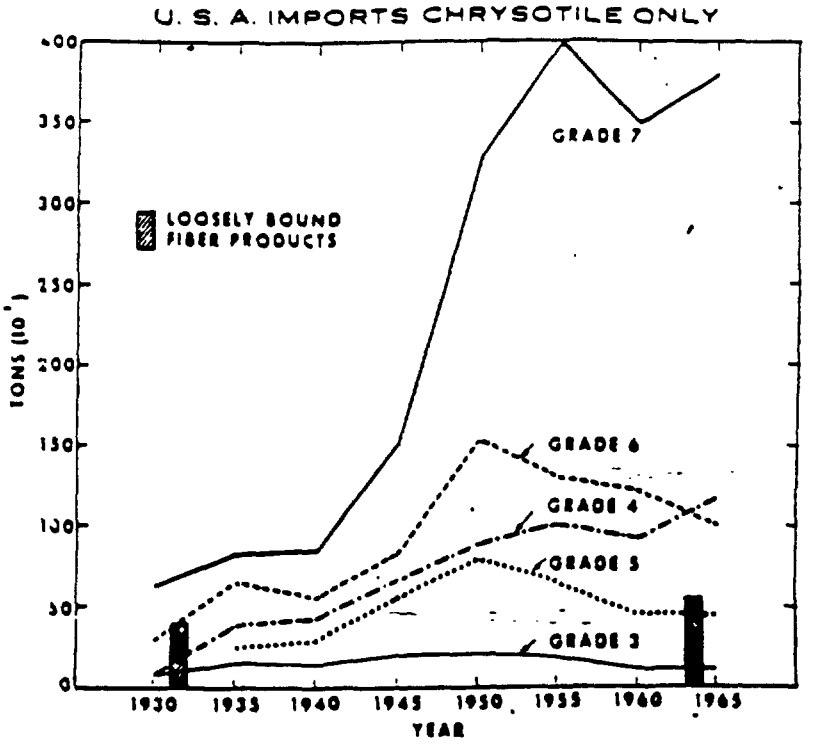

FIG. 1. Amount of chrysotile asbestos (short tons) used in the U. S. during 1930 to 1968, according to the grade of fiber. Longest fibers are grade 3 and shortest are grade 7. "Loosely bound" products include insulation and other materials that may release fibers during use, installation, or removal.

That most of the augmentation of consumption of chrysotile has been in grades 4, 6, and 7 (especially 7) is shown in figure 1. Grade 7 goes predominantly into asphalt and vinyl floor tile, which usually has a life of 20 to 25 years or more before it is changed, as a result of decoration alterations rather than wear. The bulk of grades 4 and 6 goes into asbestos cement products, especially asbestos cement pipe, in which the replacement time is indefinite—systems now in use are more than 30 years old. Air contamination does not occur from this source. Although there may be some weathering of asbestos cement shingles and siding, the rate of wear is very slow. It can readily be seen that although there has been an augmented utilization of asbestos fiber, the largest fraction of it has gone into strongly bound products designed to last many years. The wear from these products is also far less than the total product by the time the material is discarded.

Asbestos forms an essential part of friction materials. Concern has been expressed that the public is exposed to large quantities of asbestos fiber liberated by the wear of automobile brake linings. This is unwarranted. The amount of asbestos fiber used in friction material in the U.S. was estimated at 33,000 tons, less than 5 per cent of the total in 1965.[8] Studies of the material released during the use of brakes (46) demonstrated that the high heat of friction destroys the asbestos converting it to an amorphous material no longer having the characteristics of asbestos. Of the total materials removed from brake linings during their use, less than 5 per cent appears as actual asbestos. The amount of asbestos fiber released by normal friction material wear must be small indeed.

The fibers that might be released from all kinds of wear either become intimately bound with the soil or are captured in either rain water or in water used for cleansing. Ultimately the fibers are distributed predominantly where water is distributed. A study of river water has shown that asbestos fiber is almost universally present although in quite small amounts. How

TABLE 1

AMOUNT OF COMMERCIAL TALC USED FOR VARIOUS PRODUCTS IN THE U.S. (SHORT TONS IN 1967)

Total U.S. consumption	826,000
Ceramics	220,000
Paint	131,000
Roofing	72,000
Insecticides	69,000
Paper	45,000
Refractories	41,000
Rubber	31,000
Toilet preparations	28,000
Other	167,000

Total U.S. consumption of asbestos in 1965 was 720,000 short tons.

much of this comes from wear of products and how much from leaching of soil is of course not determined. The concept that asbestos is indestructible is fallacious, and the belief that it accumulates on the surface of streets and other places seems ill-founded.

The possibility that sources of fiber other than commercial asbestos may lead to ferruginous bodies must be considered. Talc is a form of magnesium silicate that exists in several forms, has large industrial use, and commonly is contaminated by fibrous substances such as tremolite and even chrysotile. It should be noted that talc production has been shown to cause pulmonary fibrosis and possibly influence the development of bronchogenic cancer (47). As shown in table 1, the consumption of commercial talc in the U.S. exceeds that for asbestos. Attention to some of the products made suggests that the opportunities for airborne talc dissemination may have been augmented to an even greater degree than has asbestos. Cralley and associates (48), in an assessment of 22 cosmetic talcum powders purchased in the U.S., found that an average of 20 per cent of the particles were fibers. These fibers are fully capable of producing ferruginous bodies in the experimental animal. It is of interest to note that the amount of commercial talc that ultimately finds its way into toilet preparations is far in excess of the amount of asbestos used in the manufacture of insulation materials. Insecticide preparations consume a larger tonnage than all of the unbound kinds of asbestos fiber put together. Rubber, which utilizes a large amount of commercial talc, may contribute to the discharge of fibers in the general environment as a result of wear. It is obvious from these data that commercial talc provides a potent source at least equivalent to that of asbestos for supplying respirable-sized fibers that could produce ferruginous bodies. It is difficult to conceive of a better way of having fibers inhaled than the use of cosmetic talcum powders. In this regard, it is of interest that Avril and Chamaux (49) found no ferruginous bodies in 135 autopsies in France. It is claimed that the cosmetic talcum in France has no fibers.* The extent to which other mineral or vegetable fibers known to be present in the environment of the general public may contribute to the production of ferruginous bodies is as yet unknown.

Data have been reported recently by Selikoff and Hammond (50) showing that the frequency of occurrence of ferruginous bodies is essentially the same in lungs obtained by autopsy in 1934 as in 1967, a finding difficult to reconcile with the contention that the augmented use of asbestos during the past 35 years has greatly enhanced the exposure of the general public to asbestos fiber. It also raises the question of whether the fibers in these ferruginous bodies are in fact asbestos. A similar study in Great Britain failed to demonstrate any ferruginous bodies in lung samples obtained at necropsy in 1936 or in lung samples from females autopsied in 1946 (51). Although they may have been fewer than now, there surely must have been potent sources for the dissemination of both talc and asbestos in Great Britain before 1936 and 1946.

The previous findings provide little or no evidence to suggest that an epidemic of mesothelioma among the public at large is to be expected. In a more positive way, the data from South Africa indicate that during the past five or six years the number of new cases of mesothelioma have been rather constant at approximately 20 per year, and in Great Britain (33) only a moderate increase has been shown.

Although the study of ferruginous bodies is of interest, the necessity for the central core to be identified and for quantitative data to be obtained in order that more meaningful conclusions be reached about their import

* Avril, J.: Personal communication.

suggests that this is an area of study with diminishing returns. A direct approach to the more easily identifiable naked fibers contained in the lung would appear to be a more fruitful avenue of search. Still better would be a direct approach to the types and amounts of fiber present in the ambient air of the various environmental locations that are of interest with respect to occupational, paraoccupational, and general public exposure. Although this would pose the disadvantage of not giving information with regard to long-term past exposure, there is equally no guarantee that ferruginous bodies truly represent the complete history of past exposure. The direct approach to the study of fiber populations in the ambient air would have the advantage of telling us what the conditions are for the public at present.

Pleural plaques: Attention has been directed toward another phenomenon said to be the result of past exposure to asbestos fiber. Since the first recognition that prolonged inhalation of asbestos fibers might in some persons lead to diffuse pulmonary fibrosis, clinicians have been aware that areas of thickening of the pleura occur in some such persons. At times, this thickening is localized leading to the appearance of plaques, some of which may be calcified. The pleura effects range all the way from minimal findings to extensive and bizarre, irregularly shaped, calcific masses. Pleural plaques with or without calcification are a matter of considerable interest at the present time. Unexpectedly, these manifestations are often absent in persons who have well-developed diffuse pulmonary fibrosis resulting from prolonged exposure to asbestos fiber, and they may be very extensive in persons with little or no diffuse fibrosis. In fact, rather extensive pleural plaques have been reported as being common in large populations not directly exposed occupationally to asbestos, but living in or near the region where asbestos fiber is being produced or used (52). There are, of course, other causes for pleural plaques and calcification, and, in some instances, it is unwise to ascribe these findings to the inhalation of asbestos fiber. In a geographic area where asbestos fiber is known to be present in considerable concentration in the ambient air, there is every reason to believe that from an epidemiologic point of view it is proper to consider these findings as evidence of asbestos exposure.

Long-term implications of pleural plaques are a matter of interest. In general, unless the thickening of the pleura is extensive, the effects of this patchy thickening on various aspects of pulmonary function are minimal or absent.[7] There is no evidence that the presence of pleural plaques connotes anything with respect to the ultimate development of mesothelioma. In fact, two of the geographic areas in which the largest concentrations of pleural plaques have been reported, Finland (52) and eastern Quebec Province,[8] do not have a demonstrated excessive frequency of mesothelioma. To use the presence of pleural plaques as a marker of previous asbestos exposure is of limited value, in view of the fact that other causes lead to this condition and especially in view of the fact that the absence of plaques in no way connotes the absence of asbestos exposure.

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The relationship between asbestos and health of humans, as reviewable in 1969, indicates that serious exposures to asbestos fiber can occur in certain occupational and immediately adjacent environments. Fortunately, the concentration of asbestos fibers in these settings can be controlled. The environment of the insulation workers is more difficult to control than the more fixed and permanent environment of the factory and associated neighborhood. An approximation of the concentration of respirable-sized asbestos fiber that can be tolerated without the risk of pulmonary fibrosis has been achieved. There is reason to believe that this concentration will also minimize the risk of pulmonary malignancy including mesothelioma. Because of the long time lapse associated with the development of malignancy, longer periods of observation are desirable for a final decision on this point. There is reason to believe that the occupational use of asbestos fiber can achieve the same degree of safety as the use of explosives, radiation, and beryllium, although extensive alterations may be required in the control of production and manufacturing.

[7] Bucklake, M.: Personal communication.
[8] Cartier, P.: Personal communication.

the containment of asbestos fiber so that none escapes to the outside, and the proper disposal of solid wastes containing the fiber.

The risk to the general public posed by the mostly intermittent exposure to the low concentrations of asbestos likely to be present in the ambient air has been exaggerated beyond the limits imposed by known facts. Research to develop the necessary additional information upon which appropriate and realistic measures for safe use of asbestos fiber can be taken should be the goal in the next few years.

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