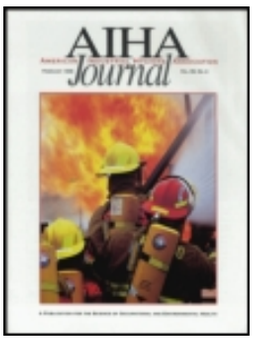

American Industrial Hygiene Association Journal

ISSN: 0002-8894 (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/aiha20

Mineral Fiber Content of Human Lungs

PAUL GROSS , RUSSELL A. HARLEY , JOHN M. G. DAVIS & LEWIS J. CRALLEY

To cite this article: PAUL GROSS , RUSSELL A. HARLEY , JOHN M. G. DAVIS & LEWIS J. CRALLEY (1974) Mineral Fiber Content of Human Lungs, American Industrial Hygiene Association Journal, 35:3, 148-151, DOI: 10.1080/0002889748507016

To link to this article: https://doi.org/10.1080/0002889748507016

Published online: 04 Jun 2010.

Mineral Fiber Content of Human Lungs

PAUL GROSS, M.D.,* RUSSELL A. HARLEY, JR., M.D.,*
JOHN M. G. DAVIS, PH.D.,† and LEWIS J. CRALLEY, PH.D.‡

Industrial Health Foundation, Inc., 5231 Centre Avenue, Pittsburgh, Pennsylvania 15232

Mineral fibers in the lungs of 13 Pittsburghers and 10 Charlestonians as well as of 7 workers exposed to asbestos dust have been quantitated, and the methodology has been described in some detail. Although wide variations in fiber concentration were found in people of the same sex and of the same community, the average concentration of optically visible pulmonary mineral fibers of Pittsburghers was about seven times as high as that of Charlestonians. The average pulmonary concentration of optically visible fibers in people not occupationally exposed to asbestos was about 0.7% of the average concentration of optically visible fibers in asbestotic lungs. From 54 to 71% of the EM-size fibers were less than 5 μm long. Of the EM-size mineral fibers in the lungs of people not occupationally exposed to asbestos, an average of 6.8% were identifiable as chrysotile.

Introduction

IT IS NOW RECOGNIZED that ambient air contains asbestos fibers and that the sources of these fibers are related not only to industry and industrial products but also to natural phenomena.[1] It is therefore not surprising that asbestos fibers have been identified in the lungs of people presumably not occupationally exposed to asbestos[2,3] (Gross, P. unpublished data). The presence of asbestos fibers in the lungs of these people has not, however, been associated with pulmonary disease referable to this mineral dust.

Also noteworthy is the fact that, in addition to identifiable chrysotile fibers, the lungs or urban dwellers contain much larger numbers of mineral fibers, the composition of which remains unknown unless expensive and time-consuming determinations are undertaken. Man-made fibers composed of glass or rock wool or even ceramic aluminum silicate could thereby be identified. There are also fibers derived from the skeletons of plants and diatom fragments to be found in the lungs. Fibers derived from the skeletons of plants become airborne in the smoke from burning coal, wood, paper, or leaves.[4]

There appears to be little question but that, of all the different kinds of mineral fibers inhaled during a lifetime, asbestos fibers are probably the most important, since they have a high pathological potential. However, there is likewise little question, at least with respect to the development of pulmonary fibrosis and lung cancer from inhaled asbestos dust, that a dose-effect relationship exists.[5] Unfortunately, at this time no data are available which would indicate at what dose level—that is, in terms of the number of asbestos fibers per gram of dry lung—a demonstrable asbestosis develops and how much more asbestos dust per gram of dry lung is required for the development of lung cancer. The question of primary concern is, of course, whether the asbestos dust

This work supported in part by the U.S. Public Health Service Contract CPE-70-107, National Institute for Occupational Safety and Health, and by the Institute of Occupational and Environmental Health, Montreal, Canada.

*Department of Pathology, Medical University of South Carolina, Charleston, South Carolina 29401.

†Institute of Occupational Medicine, Edinburgh, Scotland.

‡(retired) National Institute for Occupational Safety and Health, Post Office and Federal Court Building, Cincinnati, Ohio 45202.

content in the lungs of non-occupationally exposed people constitutes a health hazard.

The purpose of this inquiry is to provide data on the lungs of non-occupationally exposed people with regard to the number of mineral fibers in the optical range and in the submicronic range, the number of fibers less than 5 μm in length, and the number of fibers identifiable as probably chrysotile—all on a per-gram-of-dry-lung basis. In order to place these data in their proper perspective, they are compared with the data on the lungs of a few people occupationally exposed to asbestos dust. In addition, the methodology employed for the procurement of these data is described.

Materials and Methods

The lungs or portions of lungs fixed in formaldehyde were obtained from the Departments of Pathology of the University of Pittsburgh and of the Medical University of South Carolina. They were not selected except for a deliberate preponderance of women over men (15 women versus 8 men) in order to minimize bias caused by occupational exposure to dust in general and fibrous dust in particular. In addition, formalin-fixed lung tissue derived from workers known to have been occupationally exposed to asbestos dust was obtained for examination.

The lung tissue was dried to constant weight and defatted in acetone. Weighed portions were placed in 5% sodium hypochlorite (commercial bleach) for digestion. The amount was usually 10 gm of dry lung tissue from people not occupationally exposed to asbestos, and 2 gm from asbestos workers. After complete digestion, the sediment was washed several times and subjected to treatment with perchloric acid to remove most of the carbonaceous material. The remaining sediment was washed, dried, and weighed. It was then suspended in water to which phenol had been added to prevent growth of microorganisms. The strength of the suspension was generally such that the amount of sediment in it was equivalent to 1 gm of dry lung per milliliter of suspension. Prior to counting, it was usually necessary to subject the suspension to ultrasonic treatment in order to break up aggregates.

Preparatory to counting, a clean cover slip (20 × 20 mm), one surface coated with glycerinized egg albumin, was loosely cemented in its mid-portion, coated side up, to a small flat-headed nail protruding from a board. The cement was a small drop of balsam or similar mounting medium. A pipet delivering 100 μl was used to deposit the measured aliquot of the suspension upon the cover slip. This was allowed to dry, and the cover slip was heated to about 70°C to coagulate the albumin and thereby fix the sediment to the cover slip. The latter was next placed upon a glass slide, sediment down, and water was introduced by capillary attraction between the two surfaces. Optical counting of all fibers on the cover slip was performed at a magnification of 125× with the aid of a reticule in one ocular and a mechanical stage.

For the counting of the submicronic fibers, a known number of latex spheres (1.0 μm in diameter) was added to an aliquot of the sediment suspension to yield a definitive number of spheres per unit volume of suspension. A tiny drop of this mixture was placed upon an electron microscope (EM) grid and allowed to dry. A determination was then made of the number of fibers per sphere, and this number was converted into the number of EM-size fibers per gram of dry lung by means of a simple calculation. Similarly, counts were made of fibers less than 5 μm long and of the fibers that morphologically resemble chrysotile.

Results

The data obtained from the lungs of 30 people are listed in Table I.

The following statements apply to the data: There is wide variation in the concentration of optically visible mineral fibers as well as in the concentration of mineral dust in the lungs of people, even of the same sex

TABLE I
Mineral Fiber Content of Human Lungs

	Number of Cases	Number of Women	Age	Mineral Dust (mg/gm dry lung)	x 10^3/gm Dry Lung: Optical-Size Fibers	EM-Size Fibers	Fibers Less Than 5 μm
Pittsburgh	13	7	67	9.5 (2.6-33.0)	45 (5-170)	1800 (470-6750)	1200 (370-4810)
Charleston	10	8	60	2.8 (0.2-9.3)	7 (0.5-48.8)	1300 (150-6600)	900 (47-4650)
Asbestos workers	7	0	61	19 (5-35)	4000 (22,000-24,000)	150,000 (19,300-340,000)	80,000 (15,700-146,000)

TABLE II
Mineral Fiber Content of Human Lungs

	EM-Size Fibers/ Optically Visible Fibers	Percent Fibers Less Than 5 μm Long	Chrysotile ($\times 10^3$)	Percent Chrysotile of All EM-Size Fibers	Percent Chrysotile of Fibers Less Than 5 μm Long
Asbestos workers	181 (10-1125)	71	160	7.4	10.5
Pittsburgh	815 (114-3200)	71	130	6.1	9.2
Charleston	325 (14-880)	53			

and in the same community. Wide variations are also found in the concentrations of EM-size fibers, of fibers shorter than 5 μm, and of fibers identifiable as chrysotile in the lungs of these people. However, in spite of the wide range of values, some categorization of the data is evident.

Although the range of concentrations of optically visible fibers in the lungs of people not occupationally exposed to asbestos is from 400 to 170,000, with an average of about 25,000, the range of concentrations of such fibers in known asbestotic lungs is of an entirely different magnitude, with an average of about 4 million. More important, whereas the percentage of mineral fibers identifiable as chrysotile averages only about 7% (Table II) in people not occupationally exposed to asbestos, the percentage of asbestos fibers in *asbestotic* lungs must be well over 95%.

It is of interest that the concentration of EM-size mineral fibers in lung tissue ranges from 100 to over 1000 times that of optical-size mineral fibers. Of potential significance, for reasons to be discussed later, is that from 54 to 84% of the EM-size mineral fibers are less than 5 μm long.

Other interesting aspects of the results obtained include differences in the average concentrations of mineral dust and optical-size mineral fibers in the lungs of Pittsburghers versus those in the lungs of Charlestonians. For mineral dust, these were 9.5 mg/gm versus 2.8 mg/gm of dry lung, respectively; and for the fibers, 45.2 $\times$ 10^3 versus 6.5 $\times$ 10^3 fibers per gram of dry lung in Pittsburghers and Charlestonians, respectively. Finally, it should be noted that there was no relationship between the concentration of mineral dust and the concentration of mineral fibers in the lungs.

Comments

At this time, it may be appropriate to point to the fact that neither the amount nor the character of the dust found in the lungs of the subjects examined necessarily reflects the present status of the ambient air in the communities from which they came, inas-

much as this dust was accumulated over the lifetime of these people. Furthermore, much of the individual's life was spent indoors, often in poorly ventilated rooms. Fifty years ago and later, depending upon geographic location and economic circumstances, roads were unimproved and many homes were heated by individual stoves and fireplaces, some of which smoked. These could have been the sources of much of the particulate content of the lungs rather than the ambient community air. The wide range of values shown in Table I merely reflects the nonhomogeneity of the people from which the data are derived. The different economic circumstances, the frequency of travel and relocation, as well as occupational exposures to smoke and dust, often determine the quality of the air breathed.

In spite of the nonhomogeneity of the subjects, as a group, the Pittsburghers differ appreciably from the Charlestonians with respect to the concentration of mineral dust and mineral fibers in their lungs. The average concentration of mineral dust in the lungs of Pittsburghers is more than three times that in the lungs of Charlestonians, whereas the average pulmonary concentration of mineral fibers is seven times as high in Pittsburghers as it is in the Charlestonians. This difference probably reflects the dissimilarity over a span of more than one-half of a century in the ambient air of a city that was known for its high industrial smoke emissions and that of a relatively clean, seacoast community.

It can be said with justification that the pulmonary content of mineral fibers in the optical range is similar to the visible portion of an iceberg. The pulmonary content of optically invisible, submicronic fibers, like the submerged portion of the iceberg, is far larger, ranging to more than 1000 times as great. However, there the similarity to the iceberg stops. Unlike the danger posed by the submerged portion of the iceberg, there is considerable doubt that the optically invisible, submicronic fibers in nonoccupationally exposed persons constitute a health hazard. This doubt is based on recent experimental evidence that asbestos fibers shorter than 5 μm are not fibrogenic[6-8] (P. Gross, unpublished data; G. W. Wright, private communication) and the finding that the vast majority of the identifiable chrysotile fibers present in the lung sediment of non-occupationally exposed people are shorter than 5 μm. The failure of autopsy protocols in these cases to note the presence of pulmonary disease attributable to asbestos serves to strengthen this doubt. Also of significance is the great disparity between the mineral fiber concentrations in the lungs of people not occupationally exposed to asbestos and those in asbestotic lungs. Nevertheless, it must be added that the concentration of asbestos fibers associated with *early* asbestotic inflammation has not yet been determined.

References

1. *Asbestos: The Need for and Feasibility of Air Pollution Controls.* National Academy of Science, Washington, D.C. (1971).
2. Pooley, F. D., *et al.*: The Detection of Asbestos in Tissues. In *Pneumoconiosis. Proceedings of the International Conference, Johannesburg, 1969* (H. A. Shapiro, ed.), pp. 108-116, Oxford University Press, Capetown (1970).
3. Langer, A. M., *et al.*: Electron Microprobe Analysis of Asbestos Bodies. In *Pneumoconiosis. Proceedings of the International Conference, Johannesburg, 1969* (H. A. Shapiro, ed.), pp. 57-69, Oxford University Press, Capetown (1970).
4. Gross, P., *et al.*: A Quantitative Study of Fibrous Dust in the Lungs of City Dwellers. In *Inhaled Particles III* (W. H. Walton, ed.), Vol. II, pp. 671-681, Unwin Brothers, The Gresham Press, Old Woking, Surrey, England (1971).
5. Wright, G. W.: Asbestos and Health in 1969. *Amer. Rev. Resp. Dis. 100:*467-479 (1969).
6. Webster, I.: The Pathogenesis of Asbestosis. In *Pneumoconiosis. Proceedings of the International Conference, Johannesburg, 1969* (H. A. Shapiro, ed.), pp. 117-119, Oxford University Press, Capetown (1970).
7. Hilscher, W., *et al.*: Zusammenhänge zwischen Asbestose und Faserlänge. *Naturwissenschaften 57:*356-357 (1970).
8. Smith, W. E., *et al.*: Biologic Differences in Response to Long and Short Asbestos Fibers. *Amer. Ind. Hyg. Ass. J. 33:*A162 (1972).