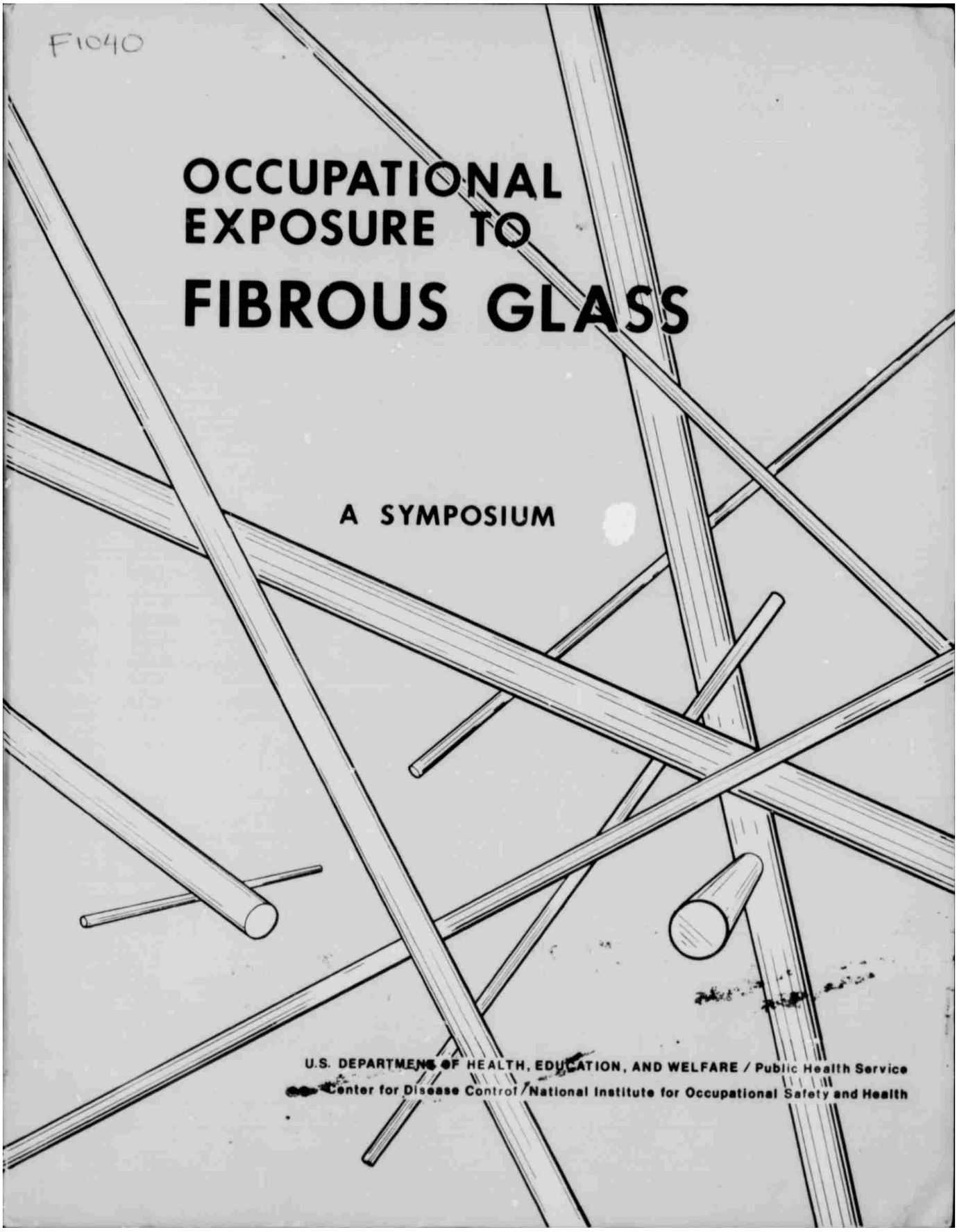


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OCCUPATIONAL EXPOSURE TO FIBROUS GLASS

Proceedings of a Symposium

Presented by the

Center of Adult Education
University of Maryland
College Park, Maryland
June 26 - 27, 1974

Sponsored by the

National Institute for Occupational Safety and Health

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health

Division of Criteria Documentation and Standards Development

April 1976

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FOREWORD

The National Institute for Occupational Safety and Health sponsored a symposium on "Occupational Exposure to Fibrous Glass," June 26 and 27, 1974. This was one of the first NIOSH-sponsored symposia. It demonstrated the utility of the symposium mechanism as a means of gaining input on scientific questions. The meeting provided an opportunity for presentation and discussion of current research related to fibrous glass exposure.

Fibrous glass is of concern in the field of occupational safety and health because it is used in a variety of processes in which many workers are potentially exposed. The effects of exposure to fibrous glass are of current interest to those involved with environmental as well as occupational health in this country and elsewhere due to its widespread use and potential as a substitute for materials such as asbestos.

This volume, containing the edited proceedings of the symposium, presents in one source an overview of the current state of research on fibrous glass exposure.

John F. Finklea, M.D.

John F. Finklea, M.D.
Director, National Institute for
Occupational Safety and Health

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PREFACE

Fibrous glass is a verstile material with a multitude of uses. The rising demand for this material has increased the need for understanding its potential occupational hazards. Despite its wide variety of uses, fibrous glass is a relatively new material with production essentially beginning in the 1930's. Yet today many questions still remain unanswered with regard to the respirability and long-term health effects of exposure to fibrous glass.

Although fibrous glass is the term for a single material it may be considered as sets of materials differentiated by dimension. The health effects may be different depending on the length and the diameter of the individual fibers. A glass fiber is the term used to describe a glass particle with a length to diameter ratio greater than 3 to 1. A more specific definition may include reference to the alignment of ions within a fiber to differentiate it from a crystal. There is special concern for glass fibers with diameters less than 3.5μ and especially less than 1μ. The regular production of these relatively small-diameter fibers is a more recent development in the fibrous glass industry.

The determination of the occupational health hazards of fibrous glass is all the more important because it is often used as a replacement for asbestos, another fibrous material. Asbestos has been associated with increased incidence of asbestosis and certain types of cancer usually after long latency or duration of exposure. The possibility of etiologic similarities between asbestos and fibrous glass due to physical factors rather than chemical composition underscores the importance of this symposium.

The primary purpose of the symposium was to fill, if the data existed, the previously identified gaps in our knowledge of the health effects of fibrous glass exposure, including long-term effects of exposure to small-diameter fibers and their mechanism of action. Filling certain of these gaps was considered essential at that time to the development of a recommended standard for occupational fibrous glass exposure. Subsumed by this purpose was the need to develop an understanding of the extent and type of fibrous glass in the work environment and the means of control.

The fibrous glass symposium provided a view of the then-current state of knowledge about fibrous glass exposure. It provided insight to many of the questions being asked by government, industry, researchers, and workers. It also generated new questions for consideration. As a result, the process leading to the development of a recommended standard for occupational fibrous glass exposure was strengthened.

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This symposium was sponsored by the National Institute for Occupational Safety and Health. Jon May, Ph.D., was chairman of the symposium steering committee. The committee included Philip Bierbaum, Paul Caplan, Alan Stevens, and William Wagner.

The symposium was presented by the University of Maryland under contract CDC-99-74-77.

These proceedings have been prepared from a verbatim, stenotyped recording of the symposium and from papers submitted by the speakers. Occasional editorial clarifications have been added.

Final editing of the document was performed under contract by William N. LeVee.

Acknowledgment must also be made for the efforts of Robin Burkhart, Joyce Huff, Evelyn Kellner, Martha Nelson and Cheryl Parsons in the production of this volume. Richard Carlson provided support for the graphic material.

Paul A. Schulte had NIOSH responsibility for the scientific editing and completion of the document.

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SCOPE OF THE SYMPOSIUM

Marcus M. Key
Vernon E. Rose

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SCOPE OF THE SYMPOSIUM

MR. ROSE: Good morning, ladies and gentlemen. I am Vern Rose, Director of the Office of Research and Standards Development of the National Institute for Occupational Safety and Health, a program of the Center for Disease Control in the Department of Health, Education and Welfare.

To officially open this Symposium on Occupational Exposure to Fibrous Glass, I am pleased to introduce the Director of NIOSH, Dr. Marcus M. Key, Assistant Surgeon General of the United States Public Health Service.

DR. KEY: Thank you. It gives me great pleasure to welcome all of you to this NIOSH symposium.

One of the questions that has been asked relative to this symposium regards the material itself, fibrous glass. In other words, why was fibrous glass chosen as the subject of the first NIOSH-sponsored symposium? The answer is quite simple. We had given considerable thought in the past to the symposium mechanism as a means of providing a forum for scientific discussion of various aspects of occupational safety and health. At the same point in time that our deliberations had jelled, resulting in our decision to seek scientific input and direction through the holding of symposia, we recognized the need for the development of a criteria document for fibrous glass. We had come to realize that significant gaps existed in the literature on the subject. Thus, it was a logical step to plan and hold a symposium involving all aspects of occupational fibrous glass exposure.

The symposium was developed with the idea that formal presentations followed by an opportunity for discussion between all participants would provide expert recommendations regarding future NIOSH actions relating to this material.

Although the National Institute for Occupational Safety and Health has a staff of trained scientists and technicians, we don't profess to know all the answers to the problems confronting us. We recognize that occupational safety and health expertise is available through universities and colleges, industry, various labor groups, state and local health departments, private consultants, and other sources in the United States and elsewhere. It has been, and will continue to be, our policy to solicit the assistance of experts wherever they may be located. The Symposium on Occupational Exposure to Fibrous Glass is one approach to seeking solutions to problems confronting us.

The National Institute for Occupational Safety and Health plans to conduct many more symposia in the future. Preliminary discussions are already are under way within the Institute to identify those compounds or exposure situations which would be appropriate subjects for future symposia. In this regard, we solicit your comments and recommendations.

Tentatively, we are planning to hold two symposia in addition to this one during the coming year.

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The speakers assembled for this symposium represent an outstanding assemblage of scientists recognized by their peers for their expertise in various areas related to fibrous glass exposure. The National Institute for Occupational Safety and Health greatly appreciates the willingness of these distinguished individuals to assist us in this important effort. I wish you success in your discussions and hope for a very profitable outcome.

Before closing, I should like to express my appreciation to the NIOSH Fibrous Glass Symposium Steering Committee for developing what promises to be an outstanding program. The committee was chaired by Dr. Jon May and included Mr. Philip Bierbaum, Mr. Paul Caplan, Dr. Alan Stevens, and Mr. William Wagner.

MR. ROSE: Before proceeding to a more specific discussion of the purpose and scope of this NIOSH symposium, I thought it might be helpful to review NIOSH's previous actions in this area, with particular emphasis on the Institute's activities in developing criteria for recommended standards ultimately to be promulgated by the Occupational Safety and Health Administration (OSHA) of the United States Department of Labor, as federal standards under the Occupational Safety and Health Act of 1970.

NIOSH and its predecessor organizations had initiated, in late 1968, various studies of workers exposed to fibrous glass, with primary emphasis on evaluating the mortality experiences of such exposed workers. A more complete discussion of aspects of NIOSH's activities in this area, with some preliminary results, will be presented in the formal part of the meeting.

I might point out that NIOSH's selection of substances on which to develop criteria documents is essentially based on a NIOSH priority list of 400-500 toxic substances and physical agents. I had the chance yesterday to review the list and noted that fibrous glass, as a substance for criteria development, ranked approximately 40th.

It might be pointed out that NIOSH essentially considered two parameters in developing such a priority list, into which many of you may have had some input.

The first parameter involves an estimate of the number of workers exposed in the work place in the United States. The second parameter is a subjective rating of the potential hazard of the material based on what is known in the available literature and information. These two factors are then combined to give a relative priority rating for a given chemical or physical agent.

According to the priority list, NIOSH had estimated some 200,000 workers in the United States had potential exposure to fibrous glass in their job situations. The relative rating on a scale of zero or some low level was up to about 6,000. This rating was achieved primarily by input from distinguished toxicologists working essentially with the Society of Toxicology in a rather formalized Delphi technique, to develop the subjective rating of toxicity. It was indicated that although the relative rating based on known information was rather low--a figure of about 400, as

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opposed to levels of 4,700 for phosgene and 4,300 for ethyl parathion--the combination of this level of toxicity with a large number of exposed workers moved fibrous glass to a rather high level in the priority list for criteria development.

Also, as data such as environmental levels and long-term follow up studies of exposed workers became available, NIOSH felt it appropriate to consider developing a criteria document on this substance with the goal of delineating exposure concentrations that are safe for various periods of employment and at which no employee would be expected to suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience. You are probably aware that this is a direct responsibility delegated in the Occupational Safety and Health Act to NIOSH.

It might be appropriate to summarize quickly the procedure that NIOSH developed for the gathering of information for developing a criteria document. The process usually is initiated under a contract arrangement with either a university consultant or a research organization to initially collect all the known world literature on the substance for which the criteria document is being developed. After a critical review of the available information, a draft document is prepared by the contractor and submitted to NIOSH for its first evaluation. After review and correction of this document, a second draft is prepared for internal review by a group of NIOSH professionals to find the weaknesses and/or the strengths of the proposal as it is being initially developed. A third draft of the document is then prepared and NIOSH selects a committee composed of external consultants generally represented by industry, organized labor, and government, both state and federal, to provide many differing points of view for this very critical review of the document.

After review by the external consultants, a fourth draft is prepared and then is made available to professional societies and governmental agencies for their evaluation and recommendations. I am sure that many of you in the audience have participated in reviewing these NIOSH criteria documents through a professional society such as the Society of Toxicology, the Society of Occupational Environmental Health, the American Industrial Hygiene Association, the American Occupational Medical Association or the American Medical Association.

Subsequent to these reviews and considering the suggestions and recommendations that are made at that time, a fifth draft is prepared, which essentially in the past has marked the end of the input by our contractors. This draft then has gone to my office for a final review prior to being presented to the Director of the Institute for his critique and approval.

However, it should be noted that, at the present time, we at NIOSH are considering a modification of this sequence in the review process, namely, that a one-day symposium be held to discuss some of the issues that have developed during the course of preparation of the criteria document. Discussion may involve the apparent lack of data or the evaluation of conflicting data or, perhaps, the epidemiological sampling methods or work practices. It is our objective to achieve, via an open forum, a critical

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evaluation of the collected data. The deliberations and recommendations of this symposium will be considered in the preparation of the final draft of the criteria document that will be submitted to the Director of NIOSH for his review.

After the document has been approved by the Director, it is then transmitted to the Assistant Secretary of Labor for consideration of the recommended criterion to be promulgated as a federal occupational health standard.

There is one additional point that I should like to make concerning future plans in the development of our criteria documents, which will have a direct impact, I think, upon the type of symposium that we are sponsoring today. In the past, we have not gone to great lengths to include in the criteria documents any comments as to lack of information or as to where gaps exist in the literature. From the very careful review of the literature conducted by NIOSH and the conclusions we try to draw from such a review, we are in a position to identify such gaps or lack of information.

Our plans call for functioning with the best information we have. Without discussing what type of research would be necessary to provide the total information needed to develop a meaningful standard, we do intend to identify information gaps in the criteria documents.

It is this needed information that will serve as the subject for discussion in a symposium such as this: a gathering where scientists can discuss what approaches and what research might be most profitable in the development of a standard.

Going back to the specific topic of our meeting, on June 30, 1972, NIOSH awarded a contract to the Tabershaw-Cooper Associates to conduct a literature search and to develop a criteria document for fibrous glass. Again, I want to point out that under this contract the contractor's involvement included half of the review stages established for the development of the document.

Over the next 15 months, essentially through September 1973, the criteria document went through several revisions that resulted from the comments and recommendations of the review groups. Subsequent to the final review by my office, the Office of Research and Standards Development, and based on recommendations stemming from the next to last review within NIOSH, a decision was made by the Director of NIOSH to delay the recommendation of a standard until certain actions could be taken. This decision was primarily based on the gaps in published knowledge of the health consequences of fibrous glass exposure and, consequently, our inability to determine a safe occupational level.

As a result of this decision NIOSH initiated two actions. The first involved plans to develop a work practice type recommended standard which would not include an environmental limit. This decision led to the development and the award of a contract, which over the next nine months, will involve observations and evaluations of different uses and

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applications of fibrous glass in industry, identifying good work practices and recommending adequate engineering controls.

We, of course, recognized the difficulties in developing safe work procedures where major questions exist regarding the ultimate health consequences of exposure to a substance. However, once a decision is made that an environmental limit cannot be established with the present knowledge, the only alternative to no regulation is the development of safe work practices and recommended engineering controls essentially designed to minimize the potential hazard of exposure to the substance in question.

The development at NIOSH of such a work practice and engineering controls document will follow the standard procedures I previously outlined which involve several internal and external reviews, including those by professional societies and external consultants.

I think that by making the decision to change the type of standard and by continuing with the various types of thorough reviews that we normally use for criteria documents,it will be at least another year before our recommendations as to work practices and engineering controls can be finalized.

The second course of action decided on by the Institute has led to the holding of this symposium. It involves a more thorough examination of what we know and what we don't know concerning the health effects of occupational exposure to fibrous glass. Indeed, our goal here is not to come up with answers to the problems or questions that we face, but rather, by bringing together the leading researchers in this field, to enable us to better document what is known, to identify ongoing or planned research on health effects, and to discuss, in a scientific forum, the type and content of research that will be most beneficial in further expanding our knowledge of health effects.

Toward this goal, this symposium has been structured to group the presentations and discussions into well defined research areas involving effects on experimental animals and on humans. Prior to getting into these two basic research areas on health effects, we shall first explore the uses and applications of fibrous glass in modern industry, followed by several papers on some basic aspects of the properties of fibers, with emphasis on respirability.

Also, prior to discussing health effects, this afternoon's session will be devoted to the important consideration of environmental sampling methods with some indications of the results that have been observed in the work place.

In considering the goals of the symposium, with emphasis on defining what we don't but should know and how we might best gain such knowledge, I should like to draw your attention to the discussion periods which have been set aside after each series of technical presentations. It is during these periods that scientific evaluations, concepts, and, where appropriate, recommendations, can best be presented and discussed. Because of the limited time available, however, it is important that such discussions be confined to the area of concern, whether it be sampling

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technology or health effects. It will be the responsibility of the individual session chairperson to insure that this time is used wisely. Consequently, the recognition of individuals who wish to comment and the expansion or termination of discussion at any point will be at the discretion of the chairperson. Should any individual feel that time was not adequate to get across his or her thoughts, NIOSH would be most pleased to receive his or her written comments subsequent to the close of the meeting.

Along these lines, there is another point I should like to make prior to getting into the first session. It involves your evaluation of the symposium, in the broadest sense, in contributing to the goal of worker health protection. As Dr. Key has indicated earlier, this is the initial attempt of NIOSH at sponsoring symposia related to ascertaining research needs. We should be most pleased to receive your comments and thoughts concerning this symposium and how future symposia might be structured or modified to better achieve the goals that we have set.

The final point I should like to make - and one which you may have questions about - concerns the availability of the papers, the transcripts, and the information that is presented here. As you will note, we are taking a verbatim transcript of this meeting. In addition, the individuals presenting technical papers will make them available to NIOSH. The University of Maryland will distribute the verbatim transcripts that are taken in this two-day meeting, including all the discussions and question and answer sessions. NIOSH also intends to publish edited proceedings of the symposium as a reference document.

I should like to introduce at this time Dr. Bobby Craft, the Deputy Associate Director for Programs from the Cincinnati operations of NIOSH.

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SESSION I

THE TECHNOLOGY OF FIBROUS GLASS, PHYSICAL AND AERODYNAMIC PROPERTIES

Chairman: Bobby Craft

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FIBROUS GLASS-MANUFACTURE, USE, AND PHYSICAL PROPERTIES

F.L. Pundsack

Introduction

DR. CRAFT: Session I addresses the subject of fibrous glass technology and covers the range of areas from the manufacture, use, and historical applications of fibrous glass to the aerodynamic considerations and deposition of fibrous glass in the human lung. We have six very interesting papers. I should like to ask that you hold all questions until after all the speakers have completed their presentations.

Our first speaker is Dr. Fred Pundsack who is the Senior Vice-President of the Johns-Manville Research and Development Center. He is a chemist by training and received both his B.S. and Ph.D. degrees from the University of Illinois. He has published numerous papers in the field on organo-metallic compounds and on the surface chemistry of inorganic minerals. He holds more than a dozen patents for various industrial processes and products. For the past 10 years, Dr. Pundsack has been concerned with the direction and management of industrial research. He is an active member of the Industrial Research Institute and of a number of other professional and technical societies.

Dr. Pundsack's paper defines fibrous glass as including mineral wool, and reviews the general methods employed in the manufacture of these products. Some of the physical properties of fibrous glass, particularly as they relate to thermal insulation applications, will be reviewed. He also will discuss the potential value of an epidemiological study of workers in mineral wool plants.

Presentation

DR. F. L. PUNDSACK: In a broad sense, the term "fibrous glass" encompasses any fibrous material that exists in the glassy state. In other words, if a material exists as a fiber (i.e., if it has a length-to-diameter ratio of, say, greater than 3:1), and the ions of which it is composed do not have a well-ordered, regular structure with respect to each other, then, strictly speaking, the substance is fibrous glass. In terms of this structural definition, the substance would be a "fibrous glass" regardless of the chemical composition.

Since this symposium is directed toward bringing together and reviewing our present knowledge of occupational exposure to fibrous glass and the possible biological effects of exposure, I plan to include mineral wool and such related terms as "rock wool" and "slag wool" within the broad category of fibrous glass. I believe it is appropriate to do so because the longest period of time since the onset of worker exposure to fibrous glass has occurred in the mineral wool industry. This may be an important consideration in epidemiological studies now under way or being planned.

I believe that several other papers in this symposium will discuss and illustrate the uses of fibrous glass. Therefore, in this paper I plan to review the general methods employed to manufacture fibrous glass,

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including mineral wool, and some of the physical properties of the products produced with those processes.

Mineral Wool

Mineral wool is a generic term used commonly to denote a glassy fibrous substance made by melting and fiberizing slags obtained from the smelting of metal ores including iron ores (slag wool), or by melting and fiberizing naturally occurring rocks (rock wool). Some crude forms of slag wool may have been produced as early as 1840, but it was only in the latter part of the 19th century and the early part of the 20th century that mineral wool began to be manufactured on a modest scale. Probably the first successful commercial manufacture of slag wool was in about 1885 in Manchester, England, using iron blast furnace slag as the raw material. Rock wool was first manufactured about 1900 in Alexandria, Indiana, by C. C. Hall, who used naturally occurring argillaceous limestone as his raw material. At least one slag wool operation, however, was in service in the United States prior to Hall's development of his rock wool process.

Although a few mineral wool plants were in operation in the United States, England and Europe in the early 1900's, it was not until after World War I that the industry began to develop and grow. By 1928, there were at least 8 mineral wool plants in the United States, and by 1939 that number had grown to 25 plants. The number of mineral wool plants in the United States probably peaked at between 80 and 90 plants in the 1950's, and then declined as fibrous glass wool made with a mixture of more or less conventional glass-making raw materials (e.g., silica sand, limestone, soda ash, etc.) began to penetrate the thermal insulation markets which had been held by mineral wool.

In the early manufacture of mineral wool, the raw materials, slag or rock, were melted in a cupola furnace and the molten stream of material was fiberized by passing it in front of a high-pressure steam jet. As the process evolved, more sophisticated designs of the steam jet were made in an effort to obtain more effective fiberization of the molten stream of slag. (Figure 1-1)

The molten stream of slag tends to be broken up into many small droplets by the steam jets, and as these droplets are swept out at high velocity in front of the jet, fibers are formed by the streaming out of "tails" from the heads of the droplets. Not all the droplets are converted into fibers by the process, and the non-fiberized droplets that remain in the product are referred to as "shot." In raw mineral wool, more than half the mass of the product may be "shot." (Figure 1-2)

Until the early 1940's, almost all mineral wool in the United States was made with one version or another of the steam jet process. Around the 1940's, however, at least two other processes were developed and commercialized for mineral wool production. The Powell process used a group of rotors operating at relatively high centrifugal speeds to collect and distribute the molten stream of slag in a thin film on the surface of the rotors, and then to fiberize the slag by throwing it off the rotors with centrifugal force. (Figure 1-3)

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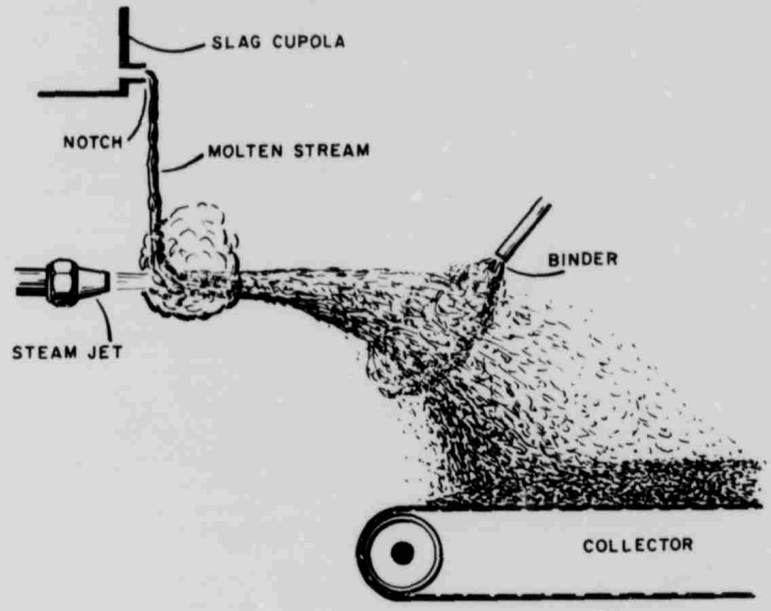


FIGURE 1-1. STEAM JET FIBERIZATION

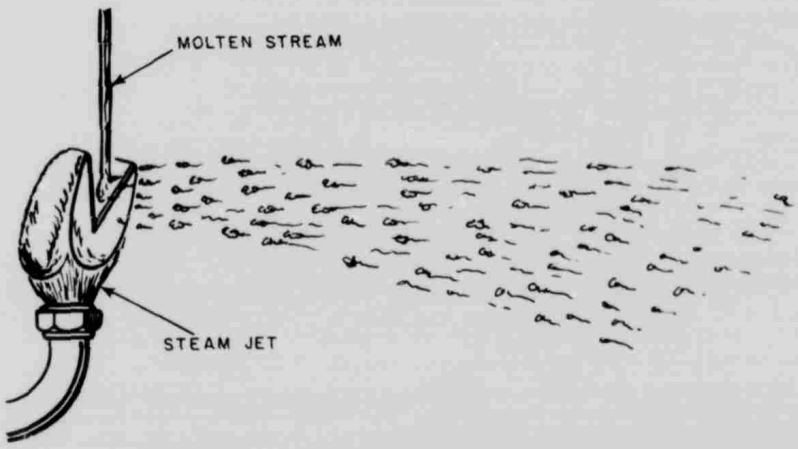


FIGURE 1-2. SHOT PROPELLED FIBERIZATION

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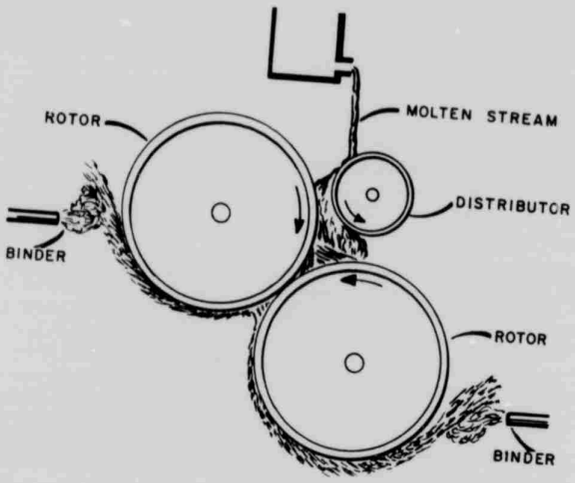


FIGURE 1-3. POWELL PROCESS

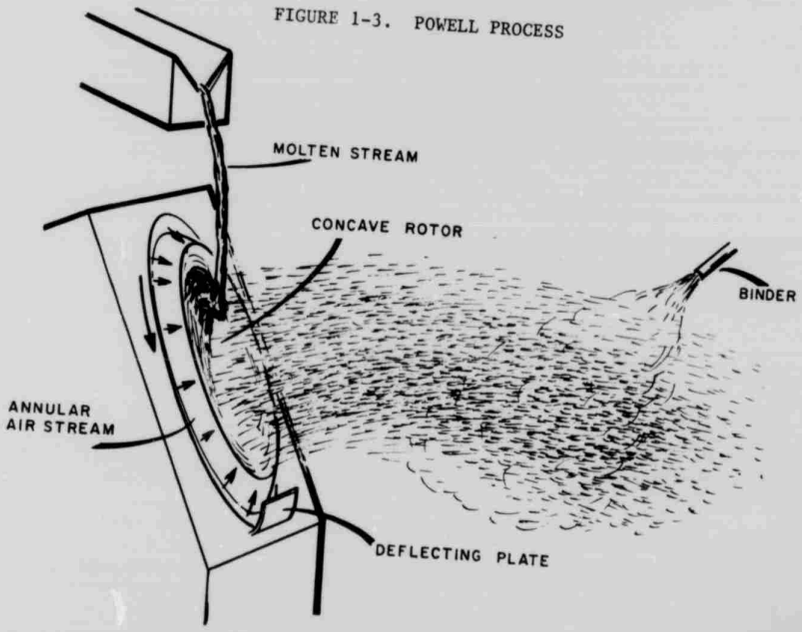


FIGURE 1-4. DOWNEY PROCESS

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A second fiberization method, which came into use at about this time was the Downey process, which combined a spinning concave rotor with steam attenuation. The molten stream was distributed in a thin pool over the surface of the dish-shaped rotor, and flowed up and over the edge of the dish where it was caught up in a high-velocity steam or air stream surrounding the dish and was fiberized. (Figure 1-4)

Although both the Powell and Downey processes had advantages over the steam jet method, they still produced a substance with a relatively high "shot" content. Even though they could produce a product with an average fiber diameter in the range, say, of 3.5 μ to 7.0 μ, the nature of the processes tended to make a product with a relatively broad distribution of fiber diameter.

Glass Fibers

Glass fibers were developed on a successful commercial scale in the United States in the 1930's by Owens-Illinois, the Corning Glass Company, and the Owens-Corning Fiberglas Company. A merger subsequently was formed by the first two companies. Mr. H. V. Smith, who follows me on the program, will, I assume, discuss the history and evolution of the processes, so I will be brief in my remarks on glass fibers.

In evaluating occupational exposure to glass fibers, it is important to recognize that there are two distinct types of commercial fiber glass products: continuous filament "textile" glass, and discontinuous glass "wool." The former has wide application in textile fabrics and reinforced plastics, and the latter has broad usage as thermal insulation, and to a lesser extent as a filtration medium.

Continuous filament textile glass usually is made by combining and melting the glass raw materials in a furnace and flowing the molten glass through a forehearth where it is taken off into a series of small platinum tanks, i.e., bushings, fitted in the bottom with hundreds of very small diameter orifices. The glass flows through these orifices and the individual filaments are collected together in a strand, a binder is applied, and the strand is wound up on a rapidly rotating drum. This process produces a very accurately sized fiber with very little variation in diameter of the product. (Figure 1-5 and Figure 1-6)

Glass "wool" is produced by several processes. This sketch is a rather generalized view of a widely used method of manufacturing glass "wool." The glass batch materials are combined and melted in a furnace, and then led out through a forehearth to the fiberization devices. Binder is applied to the fiber as it flows through the collection chamber and the fiber is collected on a moving belt. A binder common in the industry then is phenolic resin. The collected fiber with the resin on it is passed through an oven in order to "cure" or "set" the resin, and the finished product is taken off the end of the line and packaged. (Figure 1-7)

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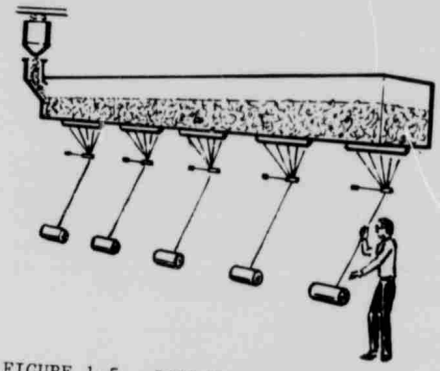


FIGURE 1-5. DIRECT MELT CONTINUOUS FILAMENT TEXTILE PROCESS

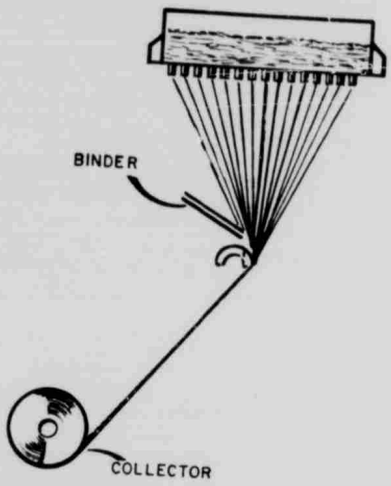


FIGURE 1-6. CONTINUOUS FILAMENT PROCESS

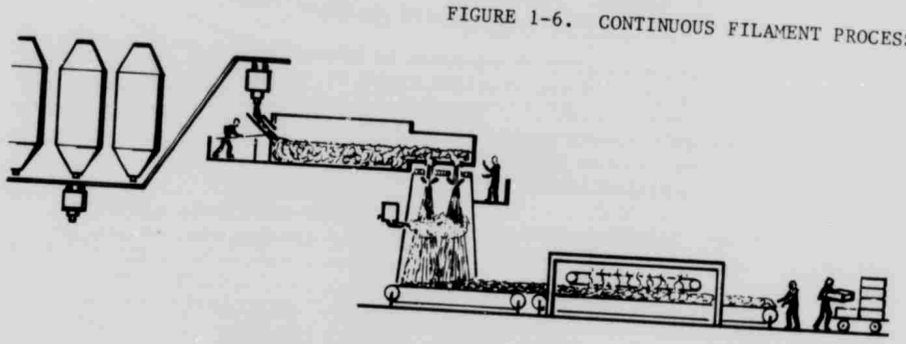


FIGURE 1-7. WOOL MANUFACTURING PROCESS

One type of fiberizing device used with this process is a rapidly spinning rotor. The molten glass stream is flowed into the bowl of the rotor, where it is distributed to the sidewall of the spinning rotor. The sidewall contains many small holes, and as the glass flows through these holes, it is attenuated by high-velocity jets around the outside periphery of the rotor. (Figure 1-8)

Another process for making glass "wool" does not utilize the rotor concept, but uses a high-pressure, high-velocity gas jet to fiberize rather coarse primary filaments of glass. In this flame attenuation process the primary filaments are fed in front of the high-velocity burners, a binder is applied to the attenuated fiber, and it is collected and cured by passing it through an oven. (Figure 1-9)

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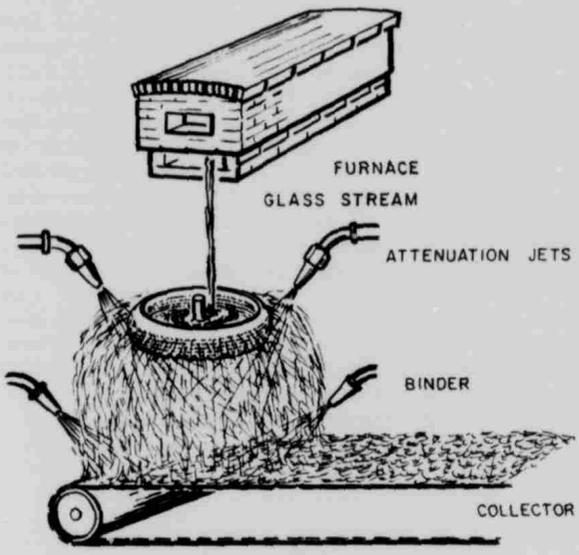


FIGURE 1-8. ROTARY PROCESS

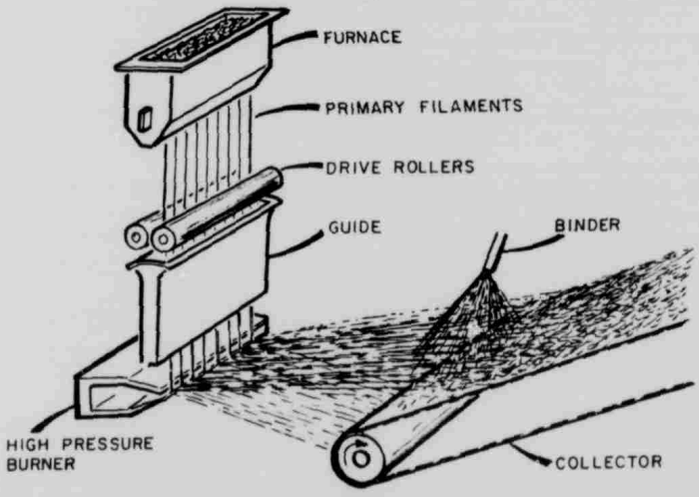


FIGURE 1-9. FLAME-ATTENUATION PROCESS

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Fibrous Glass--Thermal Conductivity and Filtration

The major use of fibrous glass "wool" is as thermal insulation. The fibers are an effective insulation because when they are formed into a mass of interlocking fibers they entrap air into many small cells and greatly restrict the flow of heat. The effectiveness of the insulation is dependent in large part on the size of the air cells and the structure of the interlocked glass fiber blanket or batt. This in turn is largely a function of the density of the product and the diameter of the fiber in the product. As the fiber diameter decreases, the thermal conductivity also decreases; that is, the material becomes a better insulator. However, in the smaller diameter regions, the relationship between fiber diameter and thermal conductivity is not linear - in fact, the curve is somewhat flat. Thus, as a result of balancing the trade-offs between fiber diameter, density, and cost of production, most commercial fibrous glass insulation is made with an average fiber diameter in the range of 4.0 μ-6.0 μ. There are, however, some highly specialized high-temperature insulations, such as the material that will go on the United States space shuttle, that are made with fibers having an average diameter of less than 1.0 μ.

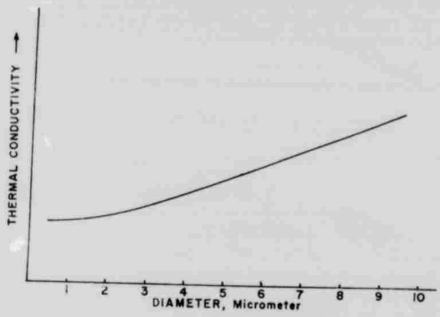


FIGURE 1-10. THERMAL CONDUCTIVITY AS A FUNCTION OF FIBER DIAMETER

In closing, I should like to reemphasize a final point. I believe that a carefully structured epidemiological study of the worker population from some of the older mineral wool plants in this country would be very productive in answering the question with which all of us are concerned: "What are the health effects of occupational exposure to fibrous glass?" It is reasonable to believe that workers in these early plants were exposed to very dusty conditions - much dustier than those existing today in fiber glass plants - and that the nature of the mineral wool process produced some fine diameter fibers (probably < 1.0 μ diameter) to which they were exposed. In addition, it may be possible to obtain a cohort of workers in which the elapsed time since onset of exposure is at least 35-40 years. I believe Dr. Konzen will discuss this type of study in more detail when he reviews the health research program of the Thermal Insulation Manufacturers Association.

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HISTORY, PROCESSES, AND OPERATIONS IN THE MANUFACTURING AND USES OF FIBROUS GLASS--ONE COMPANY'S EXPERIENCE

Harry V. Smith

Introduction

DR. CRAFT: This paper covers the experience of Owens-Corning Fiberglas and its predecessor organizations in the discovery and development of processes and products made of fibrous glass. The period covered is from 1931 to the present. Currently, fibrous glass products are used principally in thermal and acoustical insulation, as reinforcements for plastics in industrial and decorative fabrics, and other construction processing uses.

To present this paper, we have Mr. Harry V. Smith, who became a part of the glass industry in 1932 when he joined Owens-Illinois, which was one of the Owens-Corning parent companies, as a laboratory assistant in the New Uses and Research Division in Columbus, Ohio. He has been involved in several jobs and operations within the company since that time, including serving as Chief Engineer. He helped in pioneering fibrous glass manufacturing techniques and established the company's first quality control department. His present position is the Director of Quality Assurance for the Owens-Corning Fiberglas Corporation.

Presentation

Mr. SMITH: I was asked to give you my view on the history, the products, the processes and the end uses of fibrous glass. I shall not deal with the health aspects. I was fortunate to be connected with the birth of this industry and shall summarize the history of fibrous glass and one company's experience, that of Owens-Corning Fiberglas.

As I am sure you know, glass itself is old. It is made up of metallic oxides combined through melting at a high temperature. Its accidental manufacture probably approaches the age of fire. Glass fiber has been produced over an extended period of time. For example, a dress fabricated from glass fibers for the Empress Eugenie was exhibited at the St. Louis World's Fair in 1893. This fabric was manufactured by hand, not by commercial process.

European activity in the manufacture of glass fiber preceded that in this country. The Gossler process, wherein glass filaments were wound on a large drum and then removed as a hank and spread, and the Hager process, in which glass filaments were spun from a horizontal rotating disc, were used for commercial production prior to 1930. The collection devices were limited and considerable hand fabrication was required before a final useful product was formed.

In the 1930-31 period, Owens-Illinois Glass Company decided to undertake work toward uncovering new uses for glass and established the New Uses and Research Division under the direction of Dr. James Slayter. Dr. Slayter had developed the pneumatic application of mineral wool to residences and was familiar with the rock wool processes and products. He

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recognized the opportunity to refine these processes and to expand the uses of mineral fibers by using glass as a basic material. This, to me, was the start of the modern fibrous glass industry.

In 1935, Corning Glass Works joined in the activity. By 1938, Owens-Illinois and Corning had joined forces to provide a single, well-supported company to insure the progress of the industry. The result was the formation of Owens-Corning Fiberglas.

Products and Processes

The current processes for the manufacture of fibrous glass materials and products have evolved through a number of steps, each aimed at improved control over filament diameter, average and range, and at higher productivity and lower costs.

The products made from fibrous glass are defined on a performance basis, i.e., hardness, tensile strength, air flow resistance, noise reduction coefficient, thermal properties, etc. Combinations of density, filament diameter, and binder content are selected to provide the user with the thermal, acoustical, or structural properties required for the intended use. Most of the products, that is, insulations, thermal and acoustical, and plastic reinforcements, employ average filament diameters greater than 6 μ.

To deal with the history of process and product, I shall present them in two general classifications: construction materials products and textile and plastic reinforcement products.

Construction Materials Products

The first commercial fibrous glass product made by Owens-Illinois was the Dust Stop Air Filter, used in heating and ventilating systems. Glass was taken from a bottle furnace, fed through clay orifices, the stream of hot glass processed through a spiral annular steam blower to produce coarse curled filaments. The fiber was collected loosely on a conveyor, coated with hot petrolatum as a dust-catching adhesive, and packaged in fiber board frames. This was the first of the throwaway filters and it gained acceptance in industrial, commercial and residential applications. Prior to the introduction of this product, the only filter available was a permanent type which had to be cleaned on occasion; a dirty, messy job.

The usefulness of the product is illustrated by the fact that throw-away filters utilizing the fibrous glass media are still produced and sold today by the millions per year.

The coarse fibers produced for air filters were not suitable for thermal insulation. Developmental work was undertaken using smaller multiple streams and different kinds of steam and air blowers for attenuation, to secure finer fibers. The rapid wear of the clay orifices led to the employment of precious metals as orifice liners. Electrically heated precious metal orifice plates allowed an increased number of

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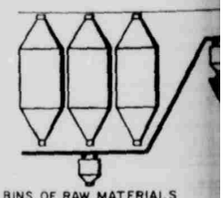
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orifices per unit and the use of slot blowers for attenuating a row of streams of glass. An average fiber diameter in the range of 9-14 μ could be made. This provided thermal values which were desirable in the marketplace.

To facilitate the handling of the product, a mineral oil lubricant was applied to control the brashiness and to avoid dusting of fiber.

The first installation of house insulation was made in a Lima, Ohio, residence in early 1933. The product was at a 1.5 pound density as compared to the 6 to 8 pound density required for mineral wools.

Further refinement established process control for a more consistent product at higher throughput. This process became known as the "Owens" process, which was used for all construction material products, including air filters, from 1934 through 1954.

The construction uses of fibrous glass insulation were hindered by its soft, blanket-like form. A rigid or semi-rigid material was needed to expand use and application. Developmental work resulted in the use of a binder applied to the filament prior to collection and cured to provide a controlled shape and form. The fibers and binder, in the uncured form, were collected on a conveyor from a stream of air. When the product was cured, it was bonded in a mass which confined the fiber, and controlled the density and dimensions. This process refinement was put into commercial use in 1939. (Figure 2-1 and Figure 2-2)

In 1954, as a feedback from a licensee, St. Gobain, the rotary forming process, wherein a rotating perforated cup was used to feed multiple streams of glass through a flame, was added to the processes already available. The provision of a higher number of glass streams allowed higher throughput, gave greater fiber length and essentially avoided non-fibrous material. (Figure 2-3)

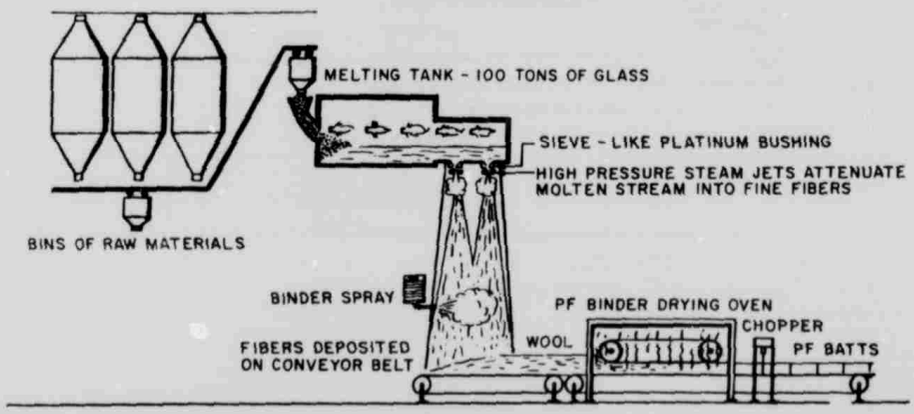


FIGURE 2-1. GLASS FIBER WOOL FORMING LINE

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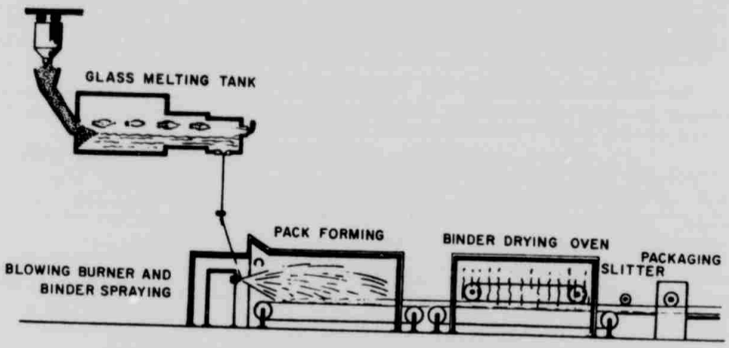


FIGURE 2-2. SUPERFINE WOOL FORMING LINE

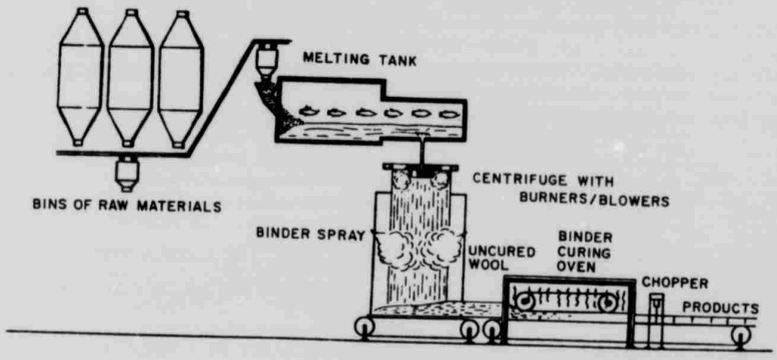


FIGURE 2-3. GLASS FIBER WOOL CENTRIFUGAL FORMING LINE

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By 1958, work at Owens-Corning Fiberglas, combining the features of the rotary forming process secured from St. Gobain with the experience of the Owens process using steam and air attenuation, resulted in a modified centrifugal process, combining steam, air, and flame drawing. This allowed tighter control over filament diameter and range, higher throughput and lower costs.

Today, this process is used for essentially all construction material products. These include: residential, appliance, pipe and roof insulation, formboard, and flexible insulation blankets. The Owens steam/air attenuation process is used for filtration media for throw away air filters.

The benefits of these products are well known. For example, the use of house insulation of fibrous glass can save heating energy in a residence in one year amounting to approximately 16 times the energy required to produce the material.

Flame Blown Process

Investigation into various means of producing glass filaments led to the discovery of the flame blown process, wherein a glass rod is fed into a flame for attenuation.

Initially, the glass source was marbles; currently, the rods are manufactured directly from a small furnace. The first large-scale manufacture of materials by this process took place in 1940-41.

This became very important in World War II when kapok was in short supply; and the process was used in Newark, Ohio to supply an alternate flotation material for life preservers.

To get the fiber into usable form, mineral oil lubricant and a binder are applied to the filaments, and the binder is cured to give a flexible blanket-like material. The quantity of material made with this process is relatively small compared to the total quantity of construction material and textile products. Most of the products manufactured by this process are used in such applications as flexible insulations in prefabricated buildings and as external insulation on air conditioning ducts. These have an average filament diameter of 4-6 μ.

There are sophisticated small volume product applications which need filament diameters averaging 4 μ or below. Such applications include high performance thermal and acoustical insulation for aircraft and space vehicles, flotation fiber as a replacement for kapok in life preservers, and high efficiency filtration media.

In all cases, the fiber has a lubricant applied and the binder is cured so as to retain the filaments in a cohesive blanket and to minimize dusting of fiber release.

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Textiles

Concurrent with the activity described previously, research was conducted into the properties of the filament proper. It was found that the glass filament had tensile strength in excess of 1MM pounds per square inch, and that its strength/weight ratio was superior to that of steel.

It was also determined that glass compositions which were used for bottles and windows were not suitable when drawn into fibers because of the amount of alkali exposed on the surface. The need for a glass which had little exposed surface alkali led to the development of special glasses, one of which is "E" glass, a low alkali calcia-alumina-silicate which has excellent electrical properties. Alternate glasses which had high chemical durability were also developed. These glasses gave a base for developing the filament textile fibers.

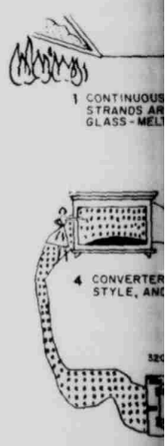
It was recognized that, to take advantage of the high tensile strength available with glass filaments, continuous lengths were needed. In 1934, work was undertaken to mechanically draw 100, 200, 400, or more filaments, collect these into a strand, apply a size made up of a lubricant, a film former, and a coupling agent, and wind this on a tube for further processing. (Figure 2-4)

Initially, the interest was in the electrical market because of the electrical properties of "E" glass. The use of fibrous glass electrical insulation permitted Class B electrical equipment to be operated at higher temperatures than had previously been possible. Generally speaking, this use of fibrous glass allowed the reduction in size of motors and transformers by one frame size and conserved copper and other materials. Cloth made from the "E" glass continuous filament was resistant to temperatures at about 1000F. During World War II, this led to the use of glass cloth as shades for parachute flares.

The reinforcement of plastic was explored wherein the glass filament or strand was coupled to the plastic so as to utilize the high tensile strength of the glass to provide an order of magnitude change in the physical properties of the composite as compared to the properties of the plastic alone. The reinforcement improved dimensional stability, impact resistance, flexural strength, and rigidity, and reduced the mass required for the end use. The reinforcement of plastic allowed lower weight than would be required with steel or aluminum for equivalent strength. This led to the use of fibrous glass in boats, automobiles and in many functional parts formerly made of steel or by die casting.

Initially, due to low throughput per unit, marbles were used to feed individual units. Later the fiber-forming units were connected to furnaces rather than going through the production and conversion of the marbles. The OCF Anderson, South Carolina plant, built in 1951, was the first textile fiber plant to produce fiber directly from the melting unit.

Work on the surface of the glass filament led to the development of processes for treatment of fabrics which provided them with flexibility so as to allow their use as decorative fabrics. This gave the drapery industry the opportunity for a flame resistant, no-iron window treatment.



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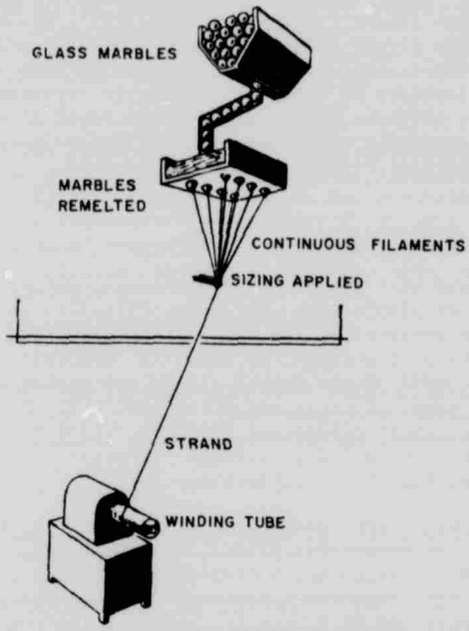


FIGURE 2-4. CONTINUOUS FILAMENT YARNS PROCESS

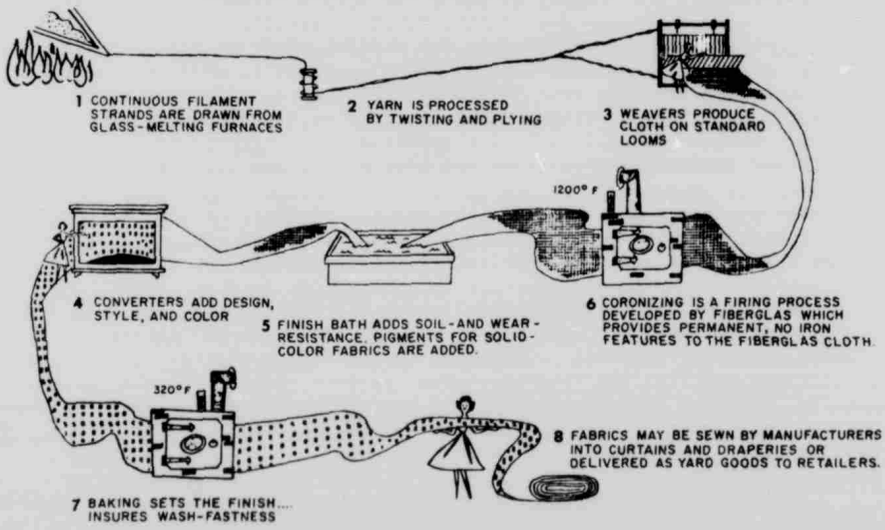


FIGURE 2-5. HOW FABRICS ARE MADE FROM GLASS FIBER YARN

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While average filament diameters of 2.5-15 μ may be produced by this textile process, the actual filament diameter used depends on the end use of the product. The drapery fabrics which need to have maximum flexibility employ filament diameters of 4 and 6 μ. Plastic reinforcements when the glass is added for strength use larger average fiber diameters from 9 to 11 μ. The process provides a minimum range in diameter because the filaments are pulled mechanically. (Figure 2-5)

Staple

It was found that there were applications which needed greater bulk in yarn than that available with continuous filaments yarn. To meet this need, fibers were produced by the Owens steam/air attenuation process, and collected as a veil on a conveyor or drum. A mineral oil lubricant was applied and the veil drawn into a sliver and packaged. This sliver was then twisted into yarn. Typical end uses of this form are filtration cloth, electrical cable filler and some decorative fabrics. The products are made in filaments of 6-9 μ in average diameter. The product became known as staple fiber because of its bulky form.

Fibers in mat form were also found to be desirable for use as reinforcements or for decorative effect. In this case, the filaments or strands were collected as a web, a binder applied, and then dried and cured to provide a thin mat. Such mats are used in electrical laminates, for underground pipe wrap and as a base for built-up roofing. Average filament diameters of 10-15 μ are used in these products.

Currently, the company is using the staple process for textiles, the continuous strand process for textile fabrics, textile yarns and plastic reinforcement, and the mat process for reinforcement of plastics, built-up roofing, electrical laminates, etc. The AF process is used for essentially all insulation products used in construction. A modification of the Owens process is used for the filtration media in Dust Stop Filters. The flame-blowing process is only used for specialty, fine fiber, high performance thermal and acoustical insulation and high efficiency filtration media.

The processes are basically the drawing of fiber, either mechanically or by gases, applying a lubricant, a film former, and a coupling agent, curing that binder and then fabricating the fiber into the end use product.

No attempt will be made to delineate all the end uses which fall into two general categories: the applied and the process materials. The applied products include building, appliance, roof, pipe, and house insulation, and flexible blankets. The process materials are generally textile products including yarn, tire cord, plastic reinforcement slivers, rovings, and mats. Plastic reinforcements go into boats, cars, air conditioners, lawn mowers, and other products.

There are more than 35,000 individual product applications. Each is aimed at a specific end use. Usually the material is combined by the customer into his product for final sale to the consumer.

HISTORY

Introduction

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HISTORY OF THE USES/APPLICATIONS OF FIBROUS GLASS

John M. Barnhart

Introduction

DR. CRAFT: Our next paper, will be presented by Mr. John M. Barnhart, Executive Director of the Thermal Insulation Manufacturers Association, Inc., Mt. Kisco, New York. Mr. Barnhart is a graduate of Rensselaer Polytechnic Institute and a member of a variety of technical societies and professional organizations. In his paper he will explain the purposes of the Association and will discuss some of the uses and applications of fibrous glass.

Presentation

MR. BARNHART: The Thermal Insulation Manufacturers Association was established in the early 1940's and has undergone several name changes since that time to encompass all types of thermal insulation.

The purposes for which the association was formed are:

(1) To develop and disseminate technical information and promote the sale and use of thermal insulation products.

(2) To assist in the development of performance and application standards, new uses, products and markets for our industry.

(3) To encourage energy conservation through the use of thermal insulation.

(4) To develop and sponsor environmental and occupational health and safety programs.

(5) To coordinate matters affecting thermal insulation with professional groups and other industries.

Our membership represents the major manufacturers in the United States of commercial industrial insulation, i.e., fibrous glass, mineral wool, and calcium silicate types of thermal insulation.

Our activities include fire testing, acoustical testing, the development of application standards, thermal testing, friction factor testing, dynamic thermal testing, and the development of comprehensive safety and health programs.

In cooperation with governmental agencies, industry, and code groups, we assist in developing performance codes and application standards.

We maintain membership and are active in the American Society for Testing Materials, the American Society of Heating, Refrigeration and Air Conditioning Engineers, the National Fire Protection Association, the Building Research Institute, the Metal Building Dealers Association, and the Industrial Health Foundation.

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Thermal insulations are those materials or combinations of materials which retard the flow of heat energy by conductive, convective, and radiative transfer modes. Thermal insulations are used for both hot and cold applications.

By retarding heat flow, thermal insulations may serve one or more of the following functions:

(1) Conserve energy by reducing heat loss or gain of piping, ducts, vessels, equipment and structures.

(2) Control surface temperatures of equipment and structures for personnel protection and comfort.

(3) Facilitate temperature control of a chemical process, a piece of equipment, or a structure.

(4) Prevent vapor condensation at surfaces having a temperature below the dew point of the surrounding atmosphere.

(5) Reduce temperature fluctuations within an enclosure when heating or cooling is not needed or available.

Fibrous glass thermal insulation is used to control heat flow in temperature ranges from below zero to 1000F. It is also used to reduce noise and vibration.

Fibrous glass is manufactured in two basic forms i.e., textile fibers and wool fibers. Textile fibers are used in reinforcements for plastics, rubber, and paper. They are also used in fabrics for industrial and commercial uses, and electrical insulation.

Wool type fibers are used primarily for thermal and acoustical insulation products. Most of the insulation material is used in homes, commercial and industrial buildings, equipment and appliances, transportation, and for industrial and commercial mechanical applications.

My principal interest here today is in the area of fibrous glass thermal insulation. The different types of thermal insulation and their uses are:

(1) Building insulation - this type of insulation is a flexible blanket. It is used in homes and is installed between the roof rafters and the studs in the side walls.

(2) Equipment insulation - this is a flexible type of a fibrous glass blanket used as lining of refrigerators, stoves, furnaces, hot water heaters, etc.

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(3) Transportation - fibrous glass molded type insulation is used on the hoods of automobiles, in the bodies of cars and also in reefer ships.

(4) Ceiling board - an acoustical ceiling board is used for acoustic, thermal and decorative purposes. This material is a rigid board type.

(5) Roof insulation - this rigid board type of product is used for roof insulation on a factory or warehouse deck.

(6) Pipe insulation - molded type fibrous glass pipe insulation is used to control heat on a steam line. It is also used for temperature control in cold applications, in chemical processing plants, utility plants, for commercial-mechanical applications, etc.

(7) Board insulation - this is a semi-rigid type of fibrous glass board used on chemical processing tanks.

(8) Cold storage insulation - the board type of insulation is used in cold storage structures.

(9) Duct systems - rigid fibrous glass board is used as a low velocity air transmission systems.

(10) Exterior duct covering - fibrous glass board type of thermal insulation may be applied to the exterior of air transmission systems.

(11) Metal building insulation - a semi-rigid type of fibrous glass is used for installation on the roofs of prefabricated metal buildings.

Fibrous glass thermal and acoustical insulation are products that are essential to our industrial, commercial, and economic well being.

Because of the unique properties of the material, there are some thermal and acoustical fibrous glass insulations for which suitable substitute materials are not available.

We currently are undergoing a major energy crisis. Many of the fibrous glass products discussed today are critical to energy conservation in a vast, diversified number of operations.

Dr. J. L. Konzen, Chairman of the TIMA Medical-Research Committee, will present our health research program in a later session.

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DR. CRAFT: We shall accept questions for the next 5 minutes.

MR. DEMENT: I have just one basic question and it is one that we have been trying to answer at NIOSH for about a year. We are aware of the fact that a small portion of the fibers produced are the so-called microfibers which are less than 1 μ in diameter. Would any one of you care to estimate what percentage of the fibers produced are these microfibers?

DR. PUNDSACK: I don't have an exact figure, but I would estimate that the production of so-called microfibers would not exceed 1% or so of the total poundage of fibers produced in the United States.

MR. SMITH: I would agree with that total assessment. Actually one would have to define more specifically the particular kind of fibers because the range of fibers used depends upon the product application. I am certain that in the case of the fibers where one needs less than the 4 μ average for the very specialized application, the amount would be considerably higher.

MR. BIERBAUM: I would like to pursue that a little further. What is the prognosis for the future production and use of the smaller diameter fibers? In your statement you mentioned their use in space shuttles and other applications. Are there any other potential uses for these small diameter fibers that would cause more concern in the future?

DR. PUNDSACK: I think that Mr. Smith brought up a good point in terms of defining what we are talking about, and I would like to define the so-called microfibers as those in the region of 1 μ or less in diameter. Most of these fibers tend to be used in the area of filtration, especially filtration applications such as clean rooms for electrical assemblies, filtration of some fluids, and also in the area of sophisticated insulations. I would estimate that probably the use of microfibers as a percentage of the total usage of glass fiber, that is, the total growth of the market, would be in the same general range that it is now. However, as the total glass fiber market grows, the actual poundage of those fibers produced would increase from the present levels.

MR. SMITH: I would like to add that generally speaking, as the filament diameter becomes finer, the cost of it per pound increases exponentially. So there's always a cost lid on how much volume of small fibers one is likely to produce.

MR. CAPLAN: I would like to inquire further about the types of binders that are used with fibrous glass. I think, Dr. Pundsack, you mentioned phenolic resins. Are there any other types of binders that are used, and roughly what percentage of the final product is the binder?

MR. SMITH: This type of question is similar to the question of what fiber diameter do you use. There are a number of binders employed. If plastic reinforcements are being used with a polyester, the binder has to be polyester-compatible. If they are being used with epoxy, the binder must be epoxy-compatible. In the insulation fields, by and large most of

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the binders are phenol formaldehyde resins, together with a mineral oil lubricant. For some applications where material is used at higher temperatures, additional nitrogen is added to give the flame-resistance or punking characteristics needed in the end use. To try to define all the variables that are needed for each end use would be impossible because of our very limited time here.

DR. PUNDSACK: He wanted to know what percentage is binder.

MR. SMITH: The percentage of binder in the final produce can be less than 0.5% in textiles. But in insulation products, it can run from a low of 4% to a high of 12%. Where structural properties are needed, as in roof insulation, it can be up to 12%.

MR. STEINFURTH: You mentioned a while ago that possibly 200,000 people were exposed to fibrous glass. Does this also include the public? I am considering now fiber glass-lined duct work, and so forth. In regard to that, I am talking about the submicron particles that would be inhalable.

DR. CRAFT: I believe that figure was given by Mr. Rose, and it does not include the general public. These are industrial employees.

MR. MAITRA: We ourselves have been concentrating on the study of fiber glass and mineral fiber. Has NIOSH considered studying any types of fiber other than these two broad categories?

DR. CRAFT: I think we shall have to defer that question until later on in the symposium when that subject will be covered.

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AERODYNAMIC CONSIDERATIONS AND OTHER ASPECTS OF GLASS FIBER

Vernon Timbrell

Introduction

DR. CRAFT: Our next speaker is Dr. Vernon Timbrell. He is a physicist at the Pneumoconiosis Unit of the British Medical Research Council at Penarth, Wales. This is a unit at the Llandough Hospital. Members of this unit started studies on asbestos diseases about 10 years ago. Dr. Timbrell is the principal researcher concerned with the physical aspects of these kinds of problems.

Recent animal experiments conducted by Dr. Vogner, Dr. Barry, and Dr. Timbrell have shown that many different types of fiber, including some samples of fibrous glass, can produce pleural mesotheliomas in rats. Dr. Timbrell recently has put together a joint team of physicists and biologists to study the mechanisms of formation of these fiber-induced tumors.

He will speak about what a respirable glass fiber is, using evidence from studies of both fibrous glass and asbestos. He also will present some very recently collected size data.

Presentation

DR. TIMBRELL: Pleural mesotheliomas have been produced in rats by intrapleural injection of asbestos fibers. [Wagner et al, 1973 and Stanton and Wrench, 1972] These tumors also have occurred following the injection of other types of fibers, including glass fibers. [Wagner et al, 1973 and Stanton and Wrench, 1972] Frequency of mesotheliomas in the asbestos injection studies was substantially greater than was observed in experiments in which rats were exposed to aerosols of the asbestos fibers. [Wagner et al, 1974] These results confirm that testing materials by intrapleural inoculation is unrealistic in that the procedure circumvents the barriers which inhaled particles must surmount to reach this specific membrane. In this paper, the aerodynamics of the penetration of fibers to the periphery of the lung and the physical factors in the development of pleural mesotheliomas are discussed.

Pulmonary Deposition

Even after particles have been formed, made airborne, and brought within the breathing zone, they are subjected to physical processes which determine whether or not they are deposited in the respiratory system. In the last four decades, a large amount of information has been collected on these processes by experiments using particles of spherical or near-spherical shape [Davies, 1954, 1961, 1967 and Walton, 1971] and through measurements of the size of particles of compact shape, [Cartwright and Nagelschmidt, 1961] retained in occupationally exposed lungs. This evidence is valid for particles of fibrous form since these processes operate on particles irrespective of the shape. The marked contrast, however, between the large length of up to 200 μ of fibers and the small

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diameters of up to 10 μ of compact-shaped particles observed in lungs indicates that the pulmonary deposition of fibers involves extra factors related to the elongated shape of these particles.

Retention of long fibers in lungs implies that the particles are capable of deep penetration into the respiratory tract. Fibers could achieve this deep penetration if they were deposited by physical forces in the upper airways and then translocated by other means to the air sacs. It would not be necessary to postulate such a double mechanism if the aerodynamic behavious of fibers alone enabled them to achieve deep penetration. In the absence of evidence on this question, or of an experimental method with which to investigate it, an analytical approach was adopted. [Timbrell, 1965] This involved physical studies on the aerodynamic characteristics of fibers to determine, on the basis of the known mechanisms of particle deposition, the size of the largest fiber capable of reaching the alveolar regions of the lung. Agreement between the result of these studies and the maximum size of fibers recovered from lungs indicated that deep penetration of long fibers during inhalation was feasible and that no novel translocation mechanism need be invoked. An outline of these studies is given below, together with an account of our subsequent animal studies to obtain further information on the influence of fiber length and shape on the behavior of fibers in the respiratory system.

Although asbestos fibers have been the center of interest in our studies, they are not ideal experimental fibers, and therefore we often have used glass fibers in view of their advantageous features: good cylindrical shape, availability of samples with convenient ranges of fiber diameters, and the possibility of controlling fiber shape and of producing spheres of the same glass.

The fact that the compact-shaped particles in rat lungs have size distributions closely resembling those of compact-shaped particles in human lungs, and a similarity in the length of the longest fibers in rat and human lungs, suggests that inhalation experiments in rats provide good indications of what happens in man. [Cartwright and Skidmore, 1964]

Deposition Mechanisms

The four mechanisms by which particles are deposited in the respiratory system are:

(1) Sedimentation, in which deposition is proportional to particle free-falling speed (i.e., proportional to aerodynamic diameter squared) and to the time available for settling.

(2) Impaction, due to inertia of the particles when a change in direction of air flow occurs; deposition by this mechanism is also proportional to particle free-falling speed.

(3) Brownian displacement, which is significant only for small particles in small air spaces.

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(4) Interception, which occurs when the effective radius of a particle is less than the distance by which the center is separated from an airway surface, in which case deposition is a function of linear dimensions.

Interception may thus provide dimension barriers to the penetration of particles, in addition to the aerodynamic barriers provided by the other mechanisms.

This list indicates the important part played by aerodynamic size in determining what happens to particles brought within the breathing zone. Those with falling speed greater than the velocity of air entering the nostrils are not taken into the respiratory system. Particles of smaller aerodynamic size may enter the nose, only to be deposited on the mucous lining or the nasal hairs. There is in general increased pulmonary deposition with decreasing aerodynamic size to a maximum deposition (c 60%) at about 2 μ. Below this aerodynamic diameter the deposition falls off to a minimum (ca 20%) at about 0.4 μ, only to increase again at smaller size due to increasing Brownian movement. Ordinarily 90% of particles of compact shape recovered from human and rat lungs have aerodynamic diameters less than 10 μ.

This last fact was used in the attempt to resolve the question whether a translocation or other novel mechanism must be postulated to explain the observation of 200 μ long fibers in lungs. This required a demonstration that a fiber 200 μ long could have an aerodynamic diameter of 10 μ or less. This in turn required a determination of the relationships between the aerodynamic diameters of fibers and the linear dimensions.

Aerodynamic Size of Fibers

Relationships between aerodynamic size and dimensional parameters of fibers were investigated with the aid of a gravitational aerosol spectrometer. [Timbrell, 1972b] In this instrument the aerosol is injected from a point source into an air flow along a wedge-shaped channel, resulting in the formation of a deposit which is a falling speed spectrum of the particles. Figure 4-1 shows a photomicrograph of a small area of a deposit obtained by sampling an aerosol comprising glass fibers and shellac spheres. Because of the limited range of spectrum covered by the microscope field, all the particles in the photomicrograph, the fibers and the spheres, have the same aerodynamic diameter, the magnitude of which may be calculated from the diameter and the density of the spheres. The photomicrograph depicts the relationships between the aerodynamic size and the linear dimensions of fibers. The fact that the fibers show a substantial variation in length indicates that the length of a fiber has only limited influence on its aerodynamic size. In contrast, virtual equality of the fiber diameters indicates a close relationship between the diameter of a fiber and its aerodynamic size.

Figures 4-2 and 4-3, which depict the relationships between fiber diameter (D), length (L), and aerodynamic diameter (Dae) were constructed from measurements made on glass fibers (density 2.25g/cc) examined in a number of microscope fields corresponding to different values of Dae.

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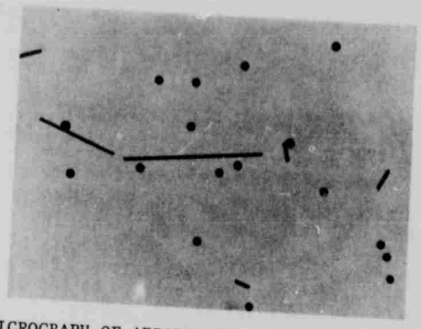


FIGURE 4-1. PHOTOMICROGRAPH OF AEROSOL SPECTROMETER SAMPLE OF SHELLAC SPHERES (1.1g/cc) AND GLASS FIBERS (2.25g/cc) OF THE SAME AERODYNAMIC DIAMETER

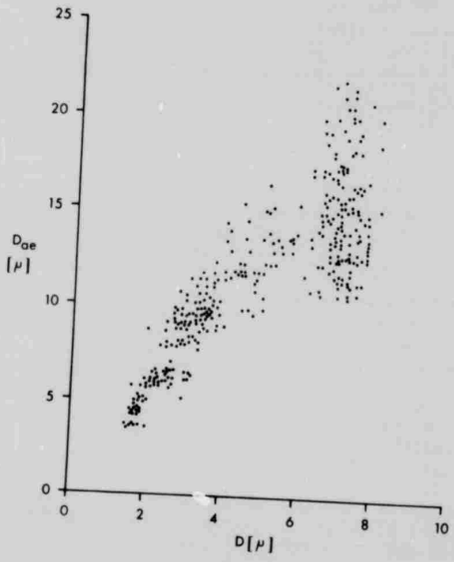


FIGURE 4-2. RELATION BETWEEN DIAMETER AND AERODYNAMIC DIAMETER OF GLASS FIBERS (2.25g/cc)

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Figure 4-2 substantiates the correlation between Dae and D. The scatter in the points is attributable to the limited but finite influence of fiber length on aerodynamic size and to the fact that the falling speed of a fiber depends on the orientation of the fiber axis during settling, being greater with axis vertical than with axis horizontal. The points near the upper boundary of the pattern relate to fibers with high values of aspect ratio (L/D), while points near the lower boundary in general relate to fibers with low aspect ratio near to the limit (3:1) beyond which they would cease to be regarded as fibers.

Figure 4-3 substantiates the limited influence of L on Dae. For the regression line, Dae/D increases only from 2.6 to 3.6 when L/D increases from 10 to 300. The scatter in the points is attributable to the variable orientation of fibers during settling, the points near the upper boundary of the pattern relating to fibers falling with fiber axis vertical while those close to the lower boundary relate to fibers falling with axis horizontal. In general there is a 40% variation of aerodynamic diameter for the two orientations.

Of particular interest is the relationship between Dae and D for fibers with such high values of L/D that they are of distinct elongated shape: for these the gradient of the upper boundary of the pattern in Figure 4-2 gives Dae/D as about 3, so that for a long glass fiber with a Dae of 10 μ the value of D is about 3.5 μ. This result, considered in conjunction with the evidence provided by studies on particles of compact shape in human lungs, indicates that when fibers (of density 2.25 g/cc) are less than about 3.5 μ in diameter, they may be very long and yet be capable of penetrating to the alveolar regions. There is therefore no need to postulate any new mechanism to explain the observation of 200 μ long fibers in excised lungs. This value of 3.5 μ obtained for the upper limit of fiber diameter is in satisfactory agreement with data reported by Gross et al, [1971a] on the size of fibers recovered from the lungs of city dwellers, when allowance is made for density variations. It is also in agreement with the data reported by Gross et al, [1971b] on fibers in the lungs of fiber glass workers where 90% of the fibers were less than 3.5 μ in diameter.

The fact that 3.5 μ approximates to the diameter of the thickest long fibers that we have observed in rats following inhalation exposure promotes confidence in the use of this animal as an indicator of the behavior of the human lung.

While fibers as thick as 3.5 μ can achieve deep pulmonary penetration, the tendency of aerosols to contain many more thin than thick fibers ensures that the number of fibers of 3.5 μ diameter recovered from lungs will be small compared with the number of thinner fibers. This feature is demonstrated by the data of Gross et al on human lungs and our own data on rat lungs.

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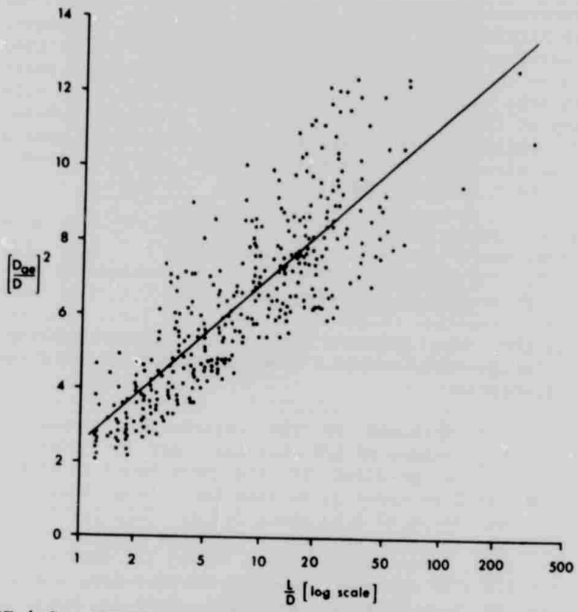


FIGURE 4-3. RELATION BETWEEN LENGTH AND AERODYNAMIC DIAMETER OF GLASS FIBERS (2.25g/cc) IN UNITS OF FIBER DIAMETER

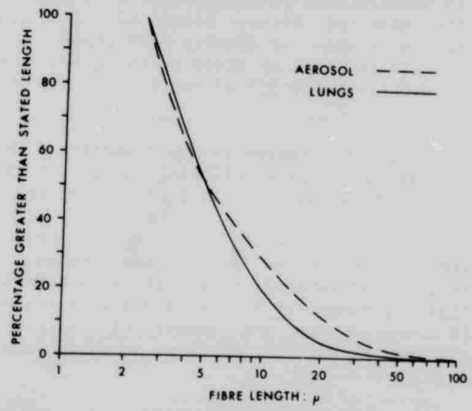


FIGURE 4-4. LENGTH DISTRIBUTIONS OF AMOSITE FIBERS IN AEROSOL AND IN RAT LUNGS

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Fiber Length

The nasal hairs present an aerodynamic-barrier to the penetration of particles of compact shape and also to fibrous particles. Whereas the nasal hairs are too widely spaced for them to present an efficient dimension-barrier to compact-shaped particles of the size that can enter the respiratory system, it was of interest to examine how efficient a dimension-barrier they would present to fibrous particles. Accordingly, a calculation was made for a model situation in which the nasal hairs provided a number of gratings in series: the inter-hair spacing was taken as 100μ and, in the absence of extra-nasal fiber-orientating forces, the fibers were assumed to have random orientation. The results gave increasing magnitude of deposition with increasing fiber length, with virtually no penetration above 200μ. The agreement between this figure and the maximum length of fibers recovered from lungs, and the fact that the airways in the upper respiratory tract are too wide to present dimension-barriers even to such long fibers, indicate that the nasal hairs provide a barrier which limits the length of respirable fibers. Another factor limiting length is that long fibers tend to have large diameters and, consequently, substantial falling speed. The falling speed of most long fibers allows little time in the breathing zone and is often too great for them to be taken into the respiratory system in either nose or mouth breathing.

The respiratory bronchioles are much narrower than the large airways. Since their diameters are comparable to the length of the longest respirable fibers, it was of interest to estimate the efficiency of deposition of fibers by interception in these fine airways. This sharply raised the question whether, in the presence of the parabolic velocity profile ordinarily existing in the laminar air flow in these fine tubes, fibers exhibit random or preferred orientation. In the absence of evidence and of methods for in vivo examination, the problem was investigated with the aid of the aerosol spectrometer, which has laminar air flow and a parabolic velocity profile. Because of the possibility that small deviations from perfect cylindrical form might influence the result, analysis was confined to clean glass and asbestos fibers. The fibers were categorized into two groups with aspect ratios <5, and >5. Counts were made on the number of fibers for which the angle between the fiber axis and the center line of the deposit (in effect the axis of the channel) was within discrete ranges between 0 to 90 degrees. For fibers with aspect ratios <5, all types showed little tendency for preferred orientation; for such low values even a positive result would be of limited importance since fibers within this category and of respirable diameter ($<3.5\mu$) would be too short to respond strongly to the interception mechanism. Of the fibers with aspect ratios >5, the glass fibers, of good cylindrical form, showed a marked tendency to align parallel to the direction of air flow; the asbestos fibers showed a reduced tendency to align with the air flow, the tendency being small for fibers of poor cylindrical shape. For all types, fibers with particles attached to their surfaces showed little or no tendency to exhibit the preferred orientation.

The fact that even clean asbestos fibers exhibited less tendency than the more regular glass fibers to align with the direction of air flow indicated that the asbestos fibers in industry, being less regular and less

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clean, would tend to have random orientation. Calculations then made on the deposition of randomly-orientated rectilinear fibers in airway models pointed to the respiratory brinchioles, especially the bifurcations in them, as preferred deposition sites for long fibers.

These data have several implications for the inhalation of glass fibers. If the fibers are clean they will in general achieve deeper penetration of the fine airways than asbestos fibers, expecially as a consequence of their presentation, end-on, to bifurcations and their resulting minimal collision cross-sectional areas. This deeper penetration, although it may amount to only a millimeter or so, can make the difference between deposition on the ciliated epithelium and subsequent efficient clearance on the one hand, and deposition beyond the ciliated epithelium and permanent retention on the other. If the fibers have particles attached to their surfaces, as may occur for instance in the case of fibers generated by the fragmentation of fiber-composites, assymetry of the resulting fiber shape militates against deep penetration and this in turn promotes clearance.

Influence of fiber length on pulmonary penetration was investigated in an experiment in rats using two aerosols of amosite asbestos, one comprising short fibers and the other both long and short fibers. [Timbrell and Skidmore, 1968] It is difficult to produce an aerosol containing only long fibers. The data obtained from the short-fiber experiment show that the fibers recovered from the rat lungs were only slightly shorter than those in the aerosol. The notable feature of the length distributions, shown in Figure 4-4 for the aerosol fibers and the lung fibers in the long fiber experiment, is that the two distributions are similar up to about 6 μ fiber length, beyond which there is a decrease in the number of lung fibers compared with the aerosol fibers. This indicates that interception begins to be effective at about 6 μ, and then increases with increase in fiber length with consequent earlier deposition and more efficient clearance.

Data also were obtained which show that the ratio of numbers of long fibers to aerosol fibers was only slightly greater in the short-fiber than in the long-fiber study. This result is in sharp contrast to the gravimetric data. Although the two aerosols had been arranged to have the same mass concentration of respirable dust as measured by the Casella Type 113A sampler, the weight of dust recovered from the lungs in the short-fiber experiment was almost twice that in the long-fiber study. This feature of the results exemplifies a characteristic of fibrous aerosols which has important implications. The feature arises from a correlation, common in fibrous materials, between fiber diameter and fiber length, related to the fact that, in general, thin fibers are more likely to break than thick fibers and thus have their lengths reduced. In the experiment the long fibers tended to be deposited higher in the respiratory tract than short fibers and consequently were more readily cleared. The long fibers also tended to be the thickest and, since fiber weight is proportional to length and to diameter squared, individually they were heavier than the short fibers, hence the major influence of the clearance of a small number of long fibers on the weight of dust retained in the lungs.

This correlation also implies cooperation between dimensional and aerodynamic deposition mechanisms. An increase in fiber length thus

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increases not only the deposition by interception but also the deposition by sedimentation and impaction. A further implication is that fiber-counting methods which only take account of fiber length are much less sensitive than direct gravimetric methods for the measurement of variations in gravimetric dust-load of lungs or in mass concentrations of fibrous aerosols.

Fiber Shape

For inhalation experiments in rats using the five UICC reference samples of asbestos, [Timbrell et al, 1968] it has been our practice to arrange for the aerosols to have the same mass concentrations of respirable dust as measured by the Casella 113A size-selective sampler, and also to equalize duration of exposures of the animal groups. Yet the lungs exposed to the amphibole samples have repeatedly retained substantially more dust than those exposed to the chrysotile samples. Two factors determining these results are the solubility of chrysotile in lung fluids and the contrast between the curly shape of chrysotile fibers and the rectilinear shape of the amphiboles illustrated in Figures 4-5 and 4-6 respectively.

In an attempt to ascertain the contribution made by fiber shape, experiments were carried out in hollow casts of mammalian (porcine) lungs, models which enable deposition and retention to be examined in the absence of dissolution and clearance mechanisms. A cast was mounted in an artificial thorax to which suction was applied to draw an aerosol of one of the UICC samples into the bronchus. The dust which penetrated the open respiratory bronchioles, and which in vivo would reach the alveoli, was collected on a filter. For equal exposures to amphibole and chrysotile samples the former gave 5 times as much dust on the filter, indicating that the difference in fiber shape made a significant contribution to the results obtained from the animal experiments. Most of this contribution can be attributed to the influence of fiber shape on deposition by the dimension-related interception mechanism which is particularly important at bifurcation in lung airways. In relation to this mechanism the significant feature of chrysotile fibers is that because of their curly shape they are effectively three-dimensional objects, in contrast to the amphibole fibers which are straight and effectively one-dimensional. This feature, in conjunction with the effect of the curly shape in causing chrysotile fibers to adopt random orientation in lung airways, makes interception a much more efficient deposition mechanism for these fibers than for amphibole fibers which show a tendency to align with the air streamlines and to be presented end-on to bifurcations. Chrysotile fibers are thus less likely than amphibole fibers to penetrate beyond the ciliated airways and to be retained in the lungs. These experiments indicated therefore that the shape of fibers is an important factor in the pulmonary penetration and retention of these particles.

In a further experiment in rats to investigate the influence of fiber shape, an aerosol of straight glass fibers was passed through an electric furnace to produce a mixture of straight fibers, curved fibers, spheres, and aggregates. [Timbrell and Skidmore, 1971] The lung dust, illustrated in Figure 4-7, was then compared with the aerosol for these different particle categories. The data obtained indicated only a very limited influence of

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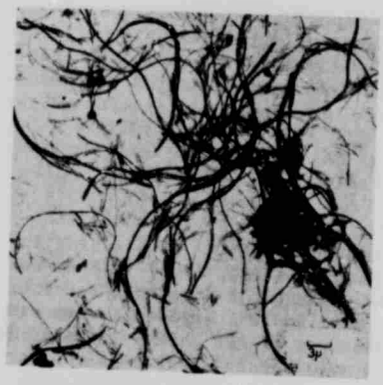


FIGURE 4-5. ELECTRON MICROGRAPH OF CHRYSOTILE ASBESTOS



FIGURE 4-6. ELECTRON MICROGRAPH OF AMPHIBOLE (AMOSITE) ASBESTOS

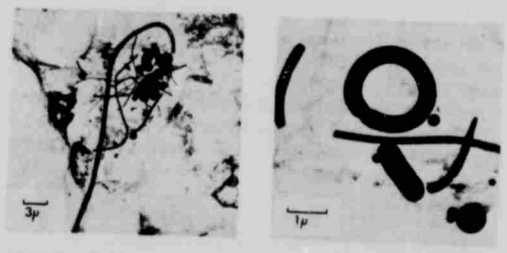


FIGURE 4-7. ASHED SECTIONS OF LUNG FROM RAT EXPOSED TO GLASS FIBERS

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fiber shape. The contrast between this result and the marked influence of fiber shape observed in both the animal and the lung cast experiments using amphibole and chrysotile asbestos requires an explanation. The glass fibers were shorter than the asbestos fibers with the consequence that, since fiber shape affects the dimension-related interception mechanism only if the fibers are long, fiber shape had less influence on lung deposition in the glass experiment than in the asbestos studies. Furthermore, the curved glass fibers differed in conformation from the curly chrysotile fibers: these glass fibers were typically confined to single planes and effectively two-dimensional objects, in contrast to the chrysotile fibers which were effectively three-dimensional. As a consequence, the difference between the penetration of straight and curved or curly fibers was less in the glass experiment than in the amphibole-chrysotile investigations. These studies thus indicated the nature of the conformation is an important factor in the pulmonary penetration and retention of non-rectilinear fibers.

Pleural Mesotheliomas

Epidemiological and pathological studies have shown that asbestos can cause bronchial tumors and pleural mesothelioma in man. [Wagner et al, 1971] An indication of a relationship between these cancers and fiber size came from an observation [Timbrell, 1972a and Timbrell et al, 1971] of a correlation between differing mesothelioma risk [Harington et al, 1971] and differing fiber diameters and lengths of amphiboles in the North West Cape and Transvaal mining regions of South Africa. Measurements made by electron microscopy showed that, on average, N.W. Cape crocidolite fibers were three times thinner and three times shorter than both Transvaal crocidolite and amosite fibers.

These results indicate that N.W. Cape fibers in comparison with Transvaal fibers are: (a) 27 times lighter, giving a proportionately greater number of fibers per ton of asbestos fiberized; (b) likely to remain airborne and available for inhalation 9 times longer; (c) more capable of penetrating to the periphery of the lung by virtue of their smaller aerodynamic diameters and shorter lengths. These multiplicative factors suggest that there could be a marked difference between the fibers in the two areas if penetration to the deeper alveoli adjacent to the pleura is an important factor in the production of mesothelioma.

The major influence of the size and shape of fibers on their ability to penetrate to target sites in the lungs indicates that the application of test materials by intrapleural inoculation is an unrealistic route of entry in comparison with human inhalation exposure. Nevertheless, the fact that this procedure has yielded mesotheliomas similar to those induced by asbestos in man shows that it is a step towards understanding the mechanism of cancer induction by asbestos fibers. We have used the procedure in a series of experiments using SPF Wistar rats inoculated with asbestos or other materials. [Wagner et al, 1973] Mesotheliomas were observed in animals with samples of asbestos as well as with nonasbestos fibrous materials, including glass fibers. The results of these experiments suggest that chemical properties are unlikely to be the main factor

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producing mesothelioma. But they do support the hypothesis that asbestos causes cancer not because it is asbestos but because it can separate into fibers.

Supplementary studies on fiber-cell preparations indicated that "significant" fibers were those less than 0.5 μ in diameter and greater than about 10 μ in length. Use of this definition in a semi-quantitative analysis [Timbrell, 1973] of the results obtained from our own experiments and similar studies carried out by Stanton and Wrench [1972] using a number of materials gave a correlation between carcinogenicity of a material and the estimated number of "significant" fibers it contained. These results suggest the benefits likely to be gained from the use of precise methods of characterizing the test materials.

One important item of data is the number of fibers/g of material. The problem is that the material usually contains both fibers and non-fibrous particles of irregular shape. As a consequence, when a small sample is viewed in the light or electron microscope, the diameter and length frequency distributions of the fibers can be measured but it is not possible to determine precisely the number of fibers/gram. Some techniques recently developed to overcome this difficulty involve, in effect, the uniform dispersion of a known weight of the material as a thin layer. [Timbrell, unpublished] The number of fibers/g can then be determined from the number in small microscope fields of known area and the total area of the layer.

These techniques have been applied to samples of Johns-Manville glass microfiber Code Nos. 110 and 100 used in one of our intrapleural inoculation experiments. Injection of 20 mg of the Code 110 sample yielded no mesotheliomata in the rats, whereas injection of 20 mg of Code 100 produced mesothelioma in 12% of the animals. Further details of the experiment are given by Wagner, et al. [This Symposium]

Figures 4-8 and 4-9 show electron micrographs of the two materials. Figure 4-10 shows the diameter distribution of the Code 110 sample obtained from transmission electron microscopy (TEM) and the length distribution from light microscopy (LM). Figure 4-11 gives the diameter and length distributions of the Code 100 sample, both obtained from transmission electron microscopy.

Table 4-1 gives a comparison of data for the two materials. For the Code 110 sample, the number of fibers/g obtained by LM was 840 million compared with 1.46 billion by TEM this indicates that most of the fibers were thick enough to be visible in the LM. In contrast, for the Code 100 sample the LM gave 300 billion fibers/g compared with 1.35 trillion given by TEM, indicating that a high proportion of the fibers were invisible in the LM. The TEM clearly provides the relevant data in both cases.

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TABLE 4-1

COMPARISON OF DATA FOR CODE 110 AND 100
GLASS MICRO-FIBERS

Parameter	Code 110		Code 100	
Mean diameter μ	1.9	(TEM)	0.12	(TEM)
Mean length μ	26.0	(LM)	2.7	(LM)
Number of fibers/g	840 million 1.46 billion	(LM) (TEM)	300 billion 1.35 trillion	(TEM) (TEM)
Number of fibers /animal	29 million	(TEM)	27 billion	(TEM)



FIGURE 4-8. ELECTRON MICROGRAPH OF CODE 110 GLASS MICRO-FIBER



FIGURE 4-9. ELECTRON MICROGRAPH OF CODE 100 GLASS MICRO-FIBER

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FIGURE 4-10. DIAMETER AND LENGTH DISTRIBUTIONS OF CODE 110 GLASS MICRO-FIBER

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FIGURE 4-11. DIAMETER AND LENGTH DISTRIBUTIONS
OF CODE 100 GLASS MICRO-FIBER

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A notable feature of the data given in Table 4-1 is that the carcinogenic Code 100 sample contained 1000 times as many fibers/g as the noncarcinogenic Code 110 sample. Also notable is the large number of fibers (27 billion) in the 20 mg of Code 100 sample associated with the 12% mesothelioma risk.

There also were many more "significant" fibers in the Code 100 sample than in the Code 110 sample. Examination of Figures 4-10 and 4-11 show that in both samples only a small percentage of fibers were less than 0.5 μ in diameter and longer than 10 μ in length, but the 1000 fold variation in fibers/g ensured that the Code 100 sample contained a much larger number of "significant" fibers than the Code 110 sample.

These techniques are now being applied to many other test materials in an attempt to ascertain which parameter of the fibers (diameter, number or surface area) is the one directly connected with tumor formation.

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AERODYNAMIC CO

Introduction

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AERODYNAMIC CONSIDERATIONS; WHAT IS A RESPIRATORY FIBER OF FIBROUS GLASS?

Robert L. Harris, Jr.

Introduction

DR. CRAFT: Our next speaker is Dr. Robert Harris, formerly Chief of the Engineering Section of the Division of Occupational Health, United States Public Health Service, a predecessor organization to NIOSH. In 1968, he became the Director of the Bureau of Abatement and Control of the National Air Pollution and Control Administration. He is now an Associate Professor of Environmental Engineering, Department of Environmental Sciences and Engineering, School of Public Health, the University of North Carolina. He serves as the Deputy Director of the occupational health studies group.

The mathematical models for estimating lung deposition of compact dust particles such as those of quartz or coal are based on the behavior of spheres in air. Dr. Harris will report a model for estimating lung deposition of straight fibers based on the behavior of rods in air. Calculated deposition values for rods of various equivalent aerodynamic diameters and lengths for each of three tidal volumes and for each of three respiratory system compartments will be reported.

Presentation

DR. HARRIS: Almost a decade ago, Dr. Timbrell addressed the matter of respirability of fibers. [1965] He suggested that the deposition of fibers which penetrate the nose is related largely to their falling speeds. His experiments showed that the falling speed of a fiber is primarily related to its diameter and only secondarily related to its length.

Following Timbrell's suggestion that further attention should be given to the matter, an analytic model for deposition of straight cylindrical fibers in the human respiratory system was developed. [Harris, 1972 and Harris and Fraser, 1974] Models for deposition in the respiratory system of compact particles are generally based on the behavior of spheres. [Findeisen, 1935; Landahl, 1950a, 1950b; and Task Group on Lung Dynamics, 1966] The model offered here to represent simple fibers is based on the behavior of right cylinder rods of high aspect ratio.

An analytic model for deposition of particles in the respiratory system requires mathematical expressions for:

(a) Respiratory system architecture
(b) Respiratory system airflow
(c) Particle behavior.

For the model described here the regular dichotomous model of Weibel [1963] was used to describe system architecture. Respiratory system airflow was based on Weibel's model and the respiratory cycle described by the Task Group on Lung Dynamics, I.C.R.P. [1966] The respiratory compartments (nasopharynx, tracheobronchial, and pulmonary spaces) and tidal volumes (750 cc, 1450 cc, and 2150 cc) used by the Task Group were

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adopted; the sequence of the respiratory cycle was altered to show the pause following exhalation rather than inhalation.

Mathematical expressions for particle behavior relating to the deposition mechanisms settling, impaction, diffusion, and interception for the various air flow regimes in the system were obtained from a number of published reports or were derived as necessary.

Orientation of rods relative to the gravitational field and to the walls and bifurcation carinas of airways is fundamental to the description of deposition probability. Theory on the periodic motion of prolate ellipsoids of revolution in a viscous fluid was published about 50 years ago. [Jeffery, 1922] Prolate ellipsoids of revolution of high aspect ratio approach the shape of long thin right cylinder rods. Mason and co-workers at McGill University have shown the theory of periodic motion to be applicable to rods in viscous fluid with low velocity gradients. [Trevelyan and Mason, 1951]

From our experiments, we have concluded that the theory is applicable as well to rods in viscous fluids having velocity gradients such as those in lung airways, one to three orders of magnitude greater than those of Mason's experiments.

The theory predicts, and experiments demonstrate, that at any time in their periodic motion a greater fraction of rods present will have their axes near parallel to streamlines (< 10 degrees) than at large angles to streamlines (> 10 degrees). The greater its axis ratio, the more likely is a rod to be closely aligned with a streamline at any instant. The fraction so aligned is a function of axis ratio and is independent of velocity gradient.

The idealized systems identified here have been used to develop a digital computer program which consists essentially of materials balanced for rods in each airway generation based on airway dimensions, flow characteristics, and deposition mechanisms. The program yields values for the fractions of rods deposited in each respiratory system compartment by equivalent size (falling speed), length, and tidal volume. Table 5-1 lists results for unit density rods of various lengths and aspect ratios for a tidal volume of 1450 cc. Similar tables have been developed for tidal volumes 750 cc and 2150 cc as well. [Harris, 1972] For a rod with a density different from one, the equivalent diameter is proportional to the square root of P where P is density.

| | FRA
EACH RE |
|---|---|
| Rod length μ | Rod diam. μ |
| 0.5 | 0.03 |
| 1.0 | 0.03 |
| 3.0 | 0.03 |
| 3.0 | 0.10 |
| 3.0 | 0.20 |
| 10 | 0.03 |
| 10 | 0.10 |
| 10 | 0.20 |
| 10 | 0.40 |
| 25 | 0.03 |
| 25 | 0.10 |
| 25 | 0.20 |
| 25 | 0.40 |
| 25 | 1.0 |
| 25 | 2.0 |
| 50 | 0.03 |
| 50 | 0.10 |
| 50 | 0.20 |
| 50 | 0.40 |
| 50 | 1.0 |
| 50 | 2.0 |
| 50 | 4.0 |
| 100 | 0.03 |
| 100 | 0.10 |
| 100 | 0.20 |
| 100 | 0.40 |
| 100 | 1.0 |
| 100 | 2.0 |
| 100 | 4.0 |
| 200 | 0.03 |
| 200 | 0.10 |
| 200 | 0.20 |
| 200 | 0.40 |
| 200 | 1.0 |
| 200 | 2.0 |
| 200 | 4.0 |

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TABLE 5-1

FRACTION OF UNIT DENSITY RODS DEPOSITED IN EACH RESPIRATORY COMPARTMENT - TIDAL VOLUME 1450 CC

Rod length μ	Rod diam. μ	Equiv. diam. μ	Fraction Deposited			
			Naso-pharynx	Tracheo-bronchial	Pulmonary spaces	Fraction Exhaled
0.5	0.03	0.068	0.008	0.024	0.365	0.603
1.0	0.03	0.074	0.016	0.022	0.329	0.632
3.0	0.03	0.083	0.049	0.030	0.288	0.633
3.0	0.10	0.245	0.049	0.029	0.289	0.633
3.0	0.20	0.448	0.048	0.029	0.309	0.613
10	0.03	0.092	0.151	0.069	0.264	0.517
10	0.10	0.277	0.150	0.069	0.272	0.510
10	0.20	0.519	0.147	0.068	0.300	0.485
10	0.40	0.959	0.329	0.067	0.307	0.296
25	0.03	0.098	0.325	0.057	0.275	0.344
25	0.10	0.300	0.327	0.021	0.299	0.353
25	0.20	0.566	0.323	0.030	0.309	0.338
25	0.40	1.06	0.492	0.048	0.274	0.186
25	1.0	2.40	0.709	0.099	0.152	0.040
25	2.0	4.36	0.850	0.107	0.039	0.004
50	0.03	0.102	0.545	0.021	0.227	0.207
50	0.10	0.315	0.549	0.025	0.214	0.211
50	0.20	0.599	0.543	0.034	0.219	0.204
50	0.40	1.13	0.660	0.047	0.190	0.104
50	1.0	2.59	0.795	0.082	0.104	0.018
50	2.0	4.79	0.904	0.075	0.019	0.001
50	4.0	8.73	1.000	-	-	-
100	0.03	0.106	0.793	0.023	0.116	0.069
100	0.10	0.330	0.793	0.025	0.114	0.069
100	0.20	0.631	0.782	0.031	0.124	0.063
100	0.40	1.20	0.830	0.035	0.105	0.029
100	1.0	2.77	0.891	0.051	0.053	0.004
100	2.0	5.19	0.955	0.038	0.007	<0.001
100	4.0	9.59	1.000	-	-	-
200	0.03	0.111	0.949	0.012	0.035	0.005
200	0.10	0.345	0.948	0.013	0.035	0.004
200	0.20	0.661	0.945	0.014	0.037	0.004
200	0.40	1.26	0.956	0.014	0.028	0.002
200	1.0	2.94	0.971	0.016	0.013	<0.001
200	2.0	5.5	0.990	0.009	0.001	<0.001
200	4.0	10.4	1.000	-	-	-

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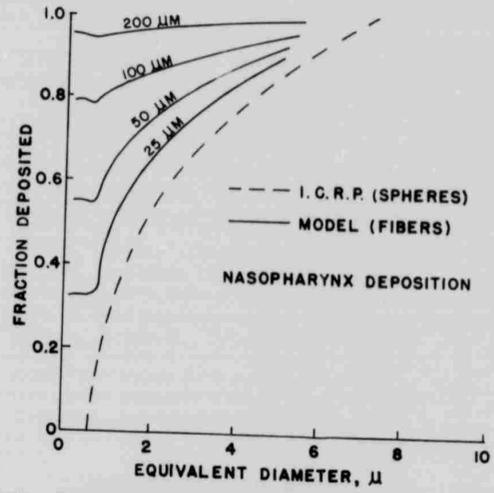


FIGURE 5-1. FRACTIONS OF RODS OF VARIOUS LENGTHS AND EQUIVALENT DIAMETERS WHICH DEPOSIT IN THE NASOPHARYNX. TIDAL VOLUME 1450 cc

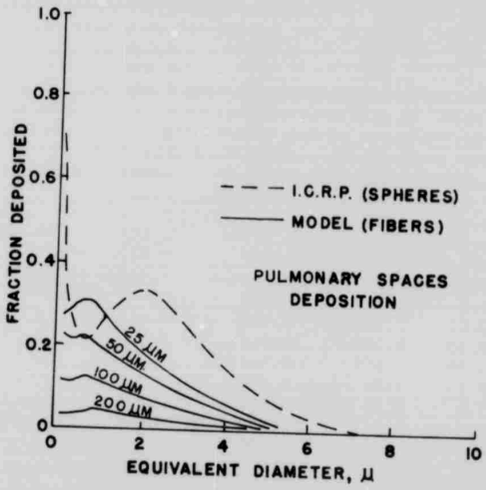


FIGURE 5-2. FRACTIONS OF RODS OF VARIOUS LENGTHS AND EQUIVALENT DIAMETERS WHICH DEPOSIT IN THE PULMONARY SPACES. TIDAL VOLUME 1450 cc

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A high probability of deposition of long fibers in the nasopharynx is expected and is a characteristic of the model results. The increase in predicted deposition with increasing rod length is illustrated in Figure 5-1. The I.C.R.P. (International Commission on Radiological Protection) curve in the figure is that proposed for spheres. [Task Group on Lung Dynamics, 1966] One may speculate that in mouth breathing much of the protective element represented by fiber deposition on nasal hairs is lost.

Pulmonary spaces deposition for rods of various lengths as predicted by the model is depicted in Figure 5-2. The results suggest that a substantial fraction of rods can penetrate to, and be deposited in, pulmonary spaces. Approximately 1 to 3% of rods 200 μ in length, but of small diameter, may deposit in pulmonary spaces.

The model is unvalidated; actual data on deposition of fibers of specific lengths and aerodynamic sizes have not been found. The results, however, are qualitatively consistent with findings of fibers 200 μ and greater in length in pulmonary spaces. The model is based on idealized systems of respiratory architecture and airflow; actual configurations may have deposition patterns departing somewhat from those predicted.

Many natural fibers are not well represented by the right cylinder rods used in the model. Natural fibers often are not straight, may be irregular in cross-section, and may be agglomerates of two or more fibers while airborne. Glass fibers, however, appear to be better represented by rods than do many natural fibers. A straight glass fiber (density approximately 2.5) having an actual diameter of about 1 μ generally has an equivalent diameter of about 4 or 5 μ. Fibers of smaller actual diameter have lower equivalent diameters. It is suggested that single straight fibers of glass up to 200 μ long with actual diameters of 1 μ and less may have a significant fraction of their number deposited in pulmonary spaces, and may thus be considered respirable.

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DEPOSITION OF FIBROUS GLASS IN THE HUMAN RESPIRATORY TRACT

Morton Lippmann
Daryl E. Bohning
Richard B. Schlesinger

Introduction

DR. CRAFT: Our next speaker is Dr. Morton Lippmann. He is Director of the Aerosol Technology Laboratory and Associate Professor, Department and Institute of Environmental Medicine, New York University Medical Center, New York City. Dr. Lippmann, like Dr. Harris, is also a former employee of a predecessor organization of NIOSH. He is an active member of several professional societies and the author of a very impressive list of publications. He will be presenting some experimental deposition data obtained using a hollow bronchial cast of the human airway, and comparing this data to predicted depositions for the same airways.

Presentation

DR. LIPPMANN: The only airborne glass fibers that represent a potential inhalation hazard are those that are deposited in the respiratory tract. Furthermore, the hazard potential is strongly dependent on the sites of deposition within the component anatomic regions of the respiratory tract. Obviously, fibers which deposit in the upper airways and on ciliated bronchi and are carried by mucous to the larynx and swallowed within one day do not contribute toward the development of chronic lung disease. On the other hand, fibers that deposit on airway surfaces that are not rapidly cleared by mucociliary action, or fibers moving on the mucous layer which become lodged at obstructions or airway junctions and are retained there for extended periods, are of considerable concern with respect to tissue reactions and disease progression.

While the potential inhalation hazard from airborne fibers is dependent on a number of other factors, including retention times, chemical composition, surface properties, etc., the respiratory tract deposition pattern is one of the most important. This has been demonstrated by Timbrell. [1972] He found that amphibole asbestos fibers had five times the penetration through a hollow bronchial cast of a pig's lung as chrysotile. A similar ratio was found for alveolar retention in vivo in rat lungs following exposure to the same fiber aerosols. Unfortunately, there are virtually no experimental data on intrabronchial fiber deposition in man or in experimental animals. The only data available are based on deposition calculations. The basis for, and utility, of such calculations have been discussed by Harris and Timbrell in the two preceding papers of this symposium.

Timbrell's calculations indicate that the bifurcations of the airways are preferred deposition sites for long asbestos fibers. The airway bifurcations also receive the highest deposition density with spherical particles. This was demonstrated experimentally by Schlesinger and Lippmann, [1972] using the hollow bronchial casts of human airways, and by Bell, [1974] using smooth wall tubing bifurcation models. Lippmann and

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Schlesinger also showed that the bifurcations having the most deposition were the same ones which have been reported to exhibit the highest incidence of primary bronchial carcinoma.

While calculations are extremely useful in elucidating the factors affecting regional deposition, one must be cautious in equating calculated deposition to in vivo deposition. The calculated deposition values can only be as good as the assumptions made. Some of the critical assumptions concern the airway anatomy, the air flow profiles within the airways, and the form of the equations used to calculate deposition by impaction, sedimentation, diffusion, and interception.

Systematic examinations of the validity of some widely used assumptions are in progress at the Institute of Environmental Medicine. This paper describes the discrepancies between experimental measurements of deposition in faithful reproductions of human airways, and predictions based on the commonly used Findeisen [1935] and Landahl [1963] relations for impaction and sedimentation. We are also exploring the influence of airway branching pattern on deposition efficiency. Most of the recent model calculations have assumed symmetric airway bifurcations, e.g., the ICRP Task Group Model for spherical particles, [1966] and the fiber deposition calculations of Harris and Fraser. [1974] We are testing the effect of airway asymmetry by repeating some of the Harris calculations, changing only the airway anatomy. For this purpose, we have selected the asymmetric model of Phalen [1974] to replace the Weibel Model A anatomy used by Harris. This comparison is still in progress, and the results will be reported in a separate publication.

Predictive Value of Deposition Equations

Our investigation of the accuracy of the relations used to predict deposition in a given airway was made possible by the availability of accurate experimental data on the deposition of spherical particles on the larger bronchial airways for a variety of particle sizes and flowrates. These data are being generated in an ongoing study of intrabronchial deposition patterns, and this analysis represents their first utilization. In this study, we are measuring the deposition at the bifurcations and along the lengths of airways from the larynx down to 2 mm diameter bronchi in hollow casts of human lungs. The tests cover a range of aerodynamic particle diameter form 8 μ to 0.2 μ, and inspiratory flowrates of from 60 liters/minute to 15 liters/minute.

The experimental measurements of deposition efficiency as a function of airway size and generation number are still in progress. Based on the limited amount of data analyzed to date, we can show that the predictions of deposition by impaction and sedimentation according to the formulations of Findeisen and Landahl differ significantly from the experimental measurements. This is illustrated in Figure 6-1 which is a plot of the experimentally determined collection efficiency of individual airway segments from the trachea to the sub-segmental bronchi in a hollow cast of a human lung versus the calculated deposition in the same segments. This plot was obtained using the impaction and sedimentation equations of

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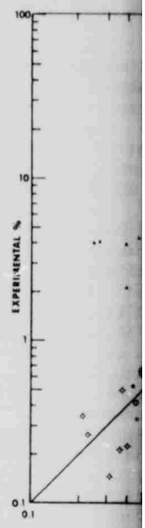


FIGURE 6-1. PREDICTE
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Each point represents
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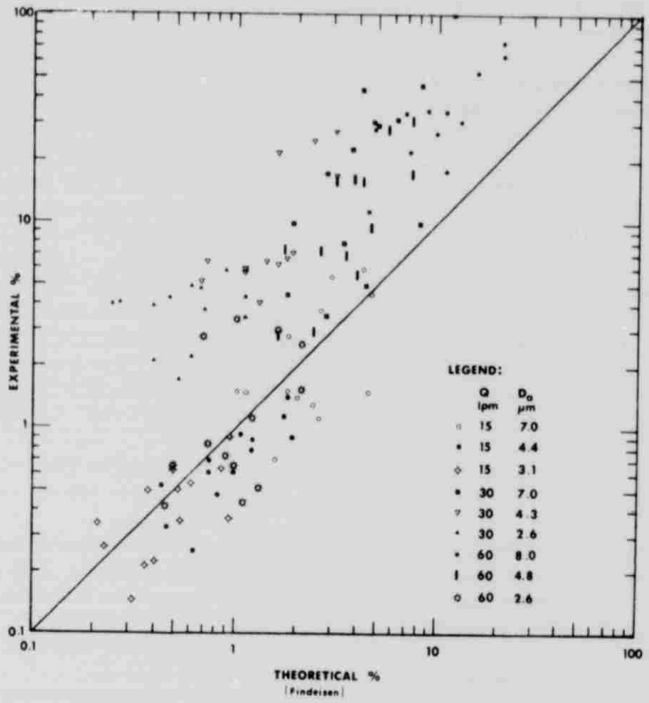


FIGURE 6-1. PREDICTED AND EXPERIMENTALLY MEASURED DEPOSITION ON A SPECIFIC AIRWAY BIFURCATION AND ALONG ITS DAUGHTER TUBES, FOR A SPECIFIC PARTICLE SIZE AND INSPIRATORY FLOW

Each point represents both the predicted and experimentally measured deposition on a specific airway bifurcation and along its daughter tubes, for a specific particle size and inspiratory flow. It includes data for the tracheal bifurcation and the three succeeding branching levels. Within these larger airways, there were no apparent differences in the ratio of experimental to theoretical values attributable to airway generation.

The experimental efficiencies may be in error to the extent that the assumed internal flow distributions are incorrect. The known flowrate in the trachea was divided among the major bronchi in proportion to their cross-sections. Similarly, a comparable division of flow was assumed for subsequent bifurcations. The adequacy of this assumption is currently being evaluated. We are measuring the flow leaving each terminus of the hollow cast at each total flow used. The results of the flow measurements and any changes these results necessitate in the deposition efficiency values will be reported in a separate publication at a later date.

The theoretical values plotted on Figure 6-1 were calculated for each specific airway on the basis of Findeisen's formulations for impaction and

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sedimentation collection, using the measured airway diameters, branching angles, and angles of inclination. For the particle sizes and flowrates used in these tests, the calculations indicated that impaction was the dominant mechanism.

The predicted dominance of impaction over sedimentation was confirmed by the experimental data, which represent the summation of the collections within the bifurcation regions and the associated downstream airway lengths. In almost every case, the bifurcation region had a greater concentration of particles than the length regions.

Similar deposition calculations were made using Landahl's formulations for impaction and sedimentation. The impaction formula included the correction proposed by Johnston and Muir, [1973] which leads to higher predicted values than the uncorrected form. The results were remarkably similar to those obtained using the Findeisen relations. The range and mean of the ratio between the Landahl and Findeisen predictions were 1.09 - 1.25(1.16) at 15 liters/minute, 1.03 - 1.11(1.07) at 30 liters/minute, and 0.96 - 1.14(1.02) at 60 liters/minute. A plot of the Landahl predictions vs. the experimental data in the manner of Figure 6-1 would look very much like Figure 6-1. Thus, neither the Findeisen nor the Landahl relations are accurate for predicting upper bronchial deposition efficiency. If they were, the ratio of experimental to theoretical deposition would be unity, and the data points in Figure 6-1 would fall close to the 45 degree line. The scatter among the data probably results from the non-ideal configurations of the airways. The ribbed structure of the airway walls, the diameter variations along the airway lengths and any curvature of the length segment axis are all factors which could affect deposition, but have been neglected in the calculations.

As shown in Figure 6-1, the calculated deposition is in reasonable agreement with the experimental at 15 liters/minute for all three particle sizes. All of the 30 and 60 liters/minute data, with the exception of the 2.6 μ - 60 liters/minute data, indicate that the Findeisen relations underestimate the deposition. The discrepancy is, in some cases, as much as a factor of 10. Different behavior at different flowrates may reflect changes in flow patterns. At 15 liters/minute, the flow is almost certainly laminar, while at 60 liters/minute, it would be turbulent in the larger bronchi. Further work is needed to clarify the effect of flowrate.

Summary

The deposition sites for fibrous particles within the lungs are not known. There have been no experimental measurements of deposition efficiency at various airway depths using fibrous particles in vivo, in excised lungs, or in hollow casts. The only data available are based on calculation, which are based on anatomic models and predictive deposition formulas of unknown accuracy.

We have used deposition data obtained in a hollow bronchial cast of a human lung to test the predictive value of the commonly used Findeisen and Landahl relations for deposition by impaction. We found that both

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relations are inadequate, especially at flowrates $\geq$ 30 liters/minute where they tend to grossly underestimate deposition in the larger bronchi.

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DR. CRAFT: We shall now open the program for general questions and discussion. Any questions regarding carcinogenic aspects directed to Dr. Timbrell should be held until a more appropriate session. Questions you have for him now are to be addressed more specifically to the physical aspects, the aerodynamic considerations.

DR. KUSCHNER: This question sort of overlaps the problem of carcinogenesis, but relates actually to particle movement. Dr. Timbrell has beautifully presented the way in which fiber dimension is a determinant of deposition and also the way in which fiber dimension is a determinant of mesothelioma induction when placed in the pleural cavity, but there is a link in the chain that's missing there. Do we know anything about how fiber dimension affects translocation?

DR. TIMBRELL: Our present evidence is that there is very little transportation of fibers in the tissue. For example, if a fiber is to reach the pleura it must do so during inhalation. We have very little evidence for transportation from the major airways to the small ones or through the tissue.

DR. KUSCHNER: How does it do that?

DR. TIMBRELL: I say we have no evidence for it happening. Therefore, I don't have to explain how it happens.

DR. PUNDSACK: I have a question for you, Dr. Timbrell. I was not clear on one of the experiments that you described in which I believe you took glass fiber and treated it in a furnace or purposely distorted it. My question really concerns the experimental method that you used. What was the experiment and the conclusion that you drew from that experiment?

DR. TIMBRELL: This experiment was intended to detect the influence of fiber shape. We always have liked to use glass fiber because it is uniform in diameter. We didn't have much success with this experiment, and I tried to explain the reasons--that the fibers were probably not curved enough; also that the fibers were too short. The cloud contained fibers up to, I think, 130 μ. But fiber shape only shows an influence when the fiber is long. So that first, the fibers were too short to show the influence, and second, they weren't curly enough.

But I think that this, of course, suggests that glass fiber may penetrate more readily into the bronchial tree than chrysotile. The chrysotile fiber is a curly one. It's like a stretched coil. And when you consider interception, what you have to think about is the size of this circumscribing envelope. In the case of chrysotile, that is large.

In looking at our pictures of glass fibers, we had the impression that when they were curved they were curved in single planes, and the envelope of a single plane is much less than that around a curly fiber. So I think the evidence is that glass fiber, in general, can be expected to penetrate more readily into the bronchial tree than chrysotile, but less readily than the amphiboles, except, perhaps, in industrial situations

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where the fibers can have particles attached to them. In that condition, the fiber is less likely to align part of the axis of the airway. It is likely to have an overtation at an angle to the axis. This means that there is a very large increase in the collision cross-section. It is much more likely to be deposited at bifurcations in the upper respiratory tract.

DR. GROSS: With regard to Dr. Kuschner's question and Dr. Timbrell's answer, I think Dr. Timbrell had in mind the translocation towards the pleura. We do, of course, recognize translocation of particles both fibrous and non-fibrous, to the satellite lymph nodes of the lung, where we have found a considerable range of fibers.

MR. PARRILLO: This question is for any of the doctors on the panel. Have there been any studies on the aerodynamic considerations of particles involved when fiber glass is sanded, as in boat manufacturing?

DR. LIPPMANN: I can't provide any real answer to that except to point out that work is going on at the Institute of Environmental Medicine using commercial fiber glass plastic mixtures for inhalation studies. Professor Laskin is doing inhalations with these, and I am sure he has size data on the aerosols, with which he simulates industrial aerosols. They have grinding machines and cutting machines to make freshly generated fiber glass plastic particle aerosols which are then put in the animal chambers, and these, I know, have been characterized by them. I don't know the results of this study. But that's a source for you.

MR. PARRILLO: Those are regular spheres, not fiber.

DR. LIPPMANN: Dr. Kuschner is familiar with that work. There is fibrous glass in the material, but apparently there are very few, if any, resulting airborne fibers when these are broken down by mechanical processes.

MR. CARSTENS: In manufacturing processes, has there been any experimentation made similar to plastic fibers that conjugate spinning where you get a curled fiber in the spinning process?

MR. SMITH: The first ones we made were curved, but they are coarse fibers and the curl was in there on purpose. We have made what we call a curly wool which has been used, if I recall correctly, by Mine Safety and Appliance for air filters for respirators. With regard to the really fine fibers--fibers that are down to respirable range--I know of no successful work on making those curl. They want to be straight. If it became necessary to do some work on it that might be a different thing.

DR. TIMBRELL: Actually, that type of fiber could be an extremely useful experimental material. We have explained the difference in the penetration of amphibole compared with chrysotile on the basis of the curl in the chrysotile fiber, but we can't eliminate the possibility that some of the difference is also due to the solubility of the chrysotile. It is extremely difficult to estimate the size of these two factors, the shape of the fiber and the solubility. So this type of experimental material could be very, very useful indeed.

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UNIDENTIFIED PARTICIPANT: I wonder if any of you gentlemen have done any experimental work on the effects of the dielectric property of the fiber either on the orientation of the particles as they are penetrating the lung or on the site of deposition, that is, whether they are ionized, and the degree of ionization of the particles.

DR. TIMBRELL: I know of no conclusive evidence on this, but I would suspect that the dielectric properties and their influence would be minimal because the respiratory tract, of course, is very humid, and I think that charge, excepting the possible building up of a droplet around the particle, might not be very important. But it's just a guess.

DR. PUNDSACK: I would agree with Dr. Timbrell that probably the dielectric properties, as such, would have relatively small influence, because there are some differences in dielectric properties, of various asbestos minerals that have never been related to their biological effect.

DR. HARRIS: Just on a qualitative basis, in the analytic model we did inquire of persons who have done experimental work on the matter of charges and lung deposition. Their qualitative response was that they would not expect this to have major influence on the deposition.

DR. SOBONYA: What is the relative importance of the nasal hairs in the nasopharynx compared with configuration in filtering out some of these longer fibers? Some people trim these hairs for various reasons. Does that mean they are more susceptible to breathing the fibers into the lungs?

DR. HARRIS: One would calculate that they would be.

DR. SOBONYA: But does it mean the hairs are that good a filter?

DR. HARRIS: For long fibers, yes.

DR. TIMBRELL: But there could be another factor in the deposition of long fibers in the nasal passages, and that is that there is a correlation between fiber damage and fiber length if a fiber usually has a large diameter. So if there were a long fiber in nasal passages, because it has a large diameter, it would have a large aerodynamic size. This would assist impaction and sedimentation. Length has two effects; first, in terms of interception, and second, in terms of an increase in impaction.

DR. HARRIS: One further thing that one might consider too, is that when a fiber of substantial size is deposited, it itself acts as a receptor for other fibers. In the work which we have done, we have not considered this secondary effect which might be very significant after the material starts to collect.

DR. SCHEEL: I would like to ask this question of the experimenters as opposed to the manufacturers. Are we talking, in your experiments, about noncoated fibers as opposed to fibers with binders?

DR. TIMBRELL: I think that whether a fiber is coated or not would have little influence on its aerodynamic properties. The same way as the dielectric factors might be expected to be minimal. But, of course, I

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think that whether a fiber is coated or not could have a large influence on what happens when the fiber gets to the tissue. But aerodynamically, I don't think that it would make very much difference, unless, of course, the coating is so thick that it is thicker than the original fiber.

DR. SCHEEL: From the industrial people, do you have any idea as to the thickness of the coating or the uniformity of it?

DR. PUNDSACK: Again it depends on the type of fiber that you are talking about. In the case of fiber made for the purpose of thermal insulation with a phenolic formaldehyde binder on it, the coatings over the entire length of the fiber can vary in thickness. The coating tends to coalesce and to bind the fibers together at the points of intersection, so that there are nonuniformities in the thickness of that coating. In the case of material made for reinforcement of plastics, the coating would be quite thin compared to the diameter of the fiber.

From a practical point of view, I think that you raise a point that is of some interest. That is, in the case of thermal insulation, the fibers are those coated with phenolic formaldehyde. It is possible for the shape of the fiber perhaps to be altered somewhat in terms of having, in effect, a particle attached to it in the form of the binder, which, from a practical point of view, would affect its thermodynamic behavior.

DR. SCHEEL: One other question on the process. In your descriptions this morning of the density measurements with regard to pounds of fiber glass per cubic foot or per cubic inch, I think it would help in understanding the whole process if you could go from the loose wall insulation type density to the fiber glass pipe insulation density as a stepwise increase in concentration both of binder and fiber glass.

MR. SMITH: I don't know that I can truthfully answer your question because I'm not sure exactly what the question is. I think I said earlier as an illustration, that house insulation could have a binder content of 4 to 5%. Its density would be down in the range of .6 to .7 pounds per cubic foot.

Pipe insulation is made in about three different processes by three different manufacturers. For instance, I can't recall at this moment what Johns-Manville's density and binder content are. I can give you an approximation of our own product. The binder content would be of the order of 7%, and the density would be approximately 6 pounds per cubic foot.

DR. SCHEEL: The point that I am making here is that with the binder and the increase in density the aerodynamic diameter of particles changes very significantly because the binder then creates not a single spicule type product, but will create a cross-linked product which has a three-dimensional aerodynamic diameter. I don't think we want to go away with the impression that people are exposed to single spicules.

DR. TIMBRELL: I would concur with that, certainly. If the binder does produce aggregation in the cloud, then this would certainly protect the man. These would be deposited probably in the nasal passages,

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certainly high up in the respiratory tract. It would be a very efficient protective measure, I should imagine.

MR. CARSON: I would like to ask the speakers what they think from their studies would be the most important parameter to measure in fibrous glass aerosols and to give us some idea of what the true occupational hazard is. Also, tell us whether it's the respirableness of the fiber, the diameter, or the length.

DR. TIMBRELL: It all depends whether we are talking about control or whether we are talking about the collection of information to try to elucidate the disease. I think that it doesn't really matter very much what parameter you use for control purposes. If you reduce dust levels in terms of one parameter, you are almost certainly going to reduce levels in terms of any other.

If you want information on the aerosols to help show what is happening relative to disease, then I think one can't be satisfied with use of one parameter. You would want information on fiber diameter, fiber length, and information collected by the use of, for example, the MRE (Medical Research Equivalent) sampler. This would be very useful indeed. So, it all depends what the purpose of the sampling is.

DR. HARRIS: I would agree with that. I think until the mechanisms of injury are well established or agreed upon, one can't be certain what parameter is involved, perhaps the three things Dr. Timbrell mentioned: length, diameter, and then configuration. Otherwise, departing from an ideal, the parameters would be the data which one might accumulate to identify the mechanisms and correlate with biologic intelligence.

DR. FRASER: I would like to ask Dr. Lippmann whether the data presented were collected using lung casts, the rigid casts. If so, what is the state of the inside of these casts? Is it moist, or is it a dry surface? And finally, was this done with a pulsating flow or with a steady state flow?

DR. LIPPMANN: Time did not permit description of the experimental details, actually it's not very complicated. The cast is not completely rigid, but we could consider it so in the context of your question. It is coated with a thin layer of silicone oil so that particles which strike that surface stick essentially where they strike, and with no rebound. The data all refer to constant inspiratory flowrates. There was no cyclic flow.

UNIDENTIFIED PARTICIPANT: Dr. Timbrell, I believe in your analysis you have assumed that the fiber diameter and fiber length are kind of co-related. This is probably true based on a given generic type of fiber. I wonder whether you have determined from the standpoint of different kinds of fiber there might be a different relationship. In other words, the parameter might be different in fiber glass as compared with other types of fiber.

Secondly, to minimize carcinogenic effects, one would avoid using fibers less than half a micron in diameter and keep the length above 200 μ.

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Based on our information, 90% of the fiber really is above that fiber diameter criteria. Are we prepared to say that we are safe?

DR. TIMBRELL: We haven't used in our analysis the fact that there is usually a correlation between fiber diameter and fiber length. We have done what calculations we have on interception on the basis that the fiber is very thin and therefore has no aerodynamic size. Then we have calculated the influence of length. It's not correct to say that the analysis is based on this correlation, although there usually is one, and we found this in the amphiboles. I think that probably with glass fiber one might not expect this correlation. It all depends on how the fiber has been treated. I should imagine that it would continue to break up indefinitely. The fiber could become shorter and shorter, in which case you wouldn't expect a correlation.

To answer your second question, I was talking about carcinogenesis. If one wants to protect the respiratory tract, then you keep the diameter up to about 3 μ, which makes the fiber virtually nonrespirable.

There is evidence that some of the glass fibers being made have a very, very high proportion of fibers less than half a micron in diameter. If I remember correctly, something like 99% of the code 100 fibers are less than this diameter.

We have to do more work on this threshold. We don't know it precisely, and possibly it wouldn't be sharp anyway. It would be sort of a graded factor. But we have been able to explain the results of our injection experiments on this basis, namely, that there were two factors involved, a factor of diameter, and a factor of length. We can alternate fibers of asbestos using magnetic fields, and this made it possible to orient the fiber and present it to the membranes of cells. When we did this with fibers greater than half a micron in diameter the cell morphology was destroyed. But when we did it with fibers less than half a micron in diameter we could implant the fiber in the cell without destroying the morphology and presumably without immediately killing the cell.

This was the first factor. Then we did work in fiber cell preparations and found that if a fiber was longer than 10 μ, the fiber seemed to be capable of finding anchorage in the cells and in the tissue.

It seems that you have to satisfy both these criteria, that the fiber diameter must be less than half a micron and the fiber must be longer than 10 μ. This evidence suggests that we may have to reduce this last threshold, so that it may be nearer 5 than 10.

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SESSION II

ENVIRONMENTAL MEASUREMENTS OF AIRBORNE FIBROUS GLASS

Chairman: Jeremiah Lynch

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CYCLONE SAMPLING OF FIBROUS GLASS AEROSOLS

Lawrence W. Ortiz
Harry J. Ettinger

Introduction

MR. LYNCH: This afternoon's presentations will go into the matter of measurement of concentrations of airborne fibrous glass, both for the purpose of determining actions that need to be taken in order to obtain control and for the estimation of exposure, so as to obtain data that will be useful in correlating the exposure with the health effect. In addition, some of these presentations will give us some information on the different levels that have been observed in industry.

Our first paper this afternoon will be presented by Mr. Lawrence Ortiz for Dr. Harry J. Ettinger, who could not be here. Mr. Ortiz is a staff member of the Industrial Hygiene Group of Los Alamos Scientific Laboratory.

Mr. Ortiz received his Master of Science degree from New Mexico State University, and his background is principally in chemistry. His experience with the physics of aerosols relates to a broad range of environmental problems.

Presentation

MR. ORTIZ: Increasing interest in the aerodynamic behavior of extreme shaped particles and fibrous aerosols is related to the potential biological effects resulting from long-term human exposure to specific fibers, such as asbestos and fibrous glass. It is well documented that inhalation of asbestos can result in pneumoconiosis and neoplastic diseases, [Criteria for a Recommended Standard for Occupational Exposure to Asbestos, 1972] and the concept of lung deposition and clearance [Hatch and Gross, 1964] indicates that these hazards are related to aerosol aerodynamic size distribution, or "respirable" fraction, [ACGIH, 1973a] as well as aerosol concentration. Since this "respirable" material constitutes a long-term exposure hazard to man, a need exists for an accurate means of measuring human exposure to fibrous dusts in terms of the aerodynamic properties of fibers, and/or estimating the fraction that is capable of penetrating to, and being retained in, the deep lung.

While it is currently assumed that fibrous glass is a material of relatively low toxicity, comparable to a nuisance dust, limited recent work indicates that fibrous glass may be a potential carcinogen, [Stanton and Wrench, 1972 and Stanton, 1972] emphasizing the need to measure the "respirable" fraction of this airborne material. A critical related need is the development of a model defining lung deposition and clearance for fibrous materials, such as exists for particulates. [ACGIH, 1973a and ICRP Task Group on Lung Dynamics, 1966] However, a preliminary estimate of lung deposition for fibrous materials can be made on the basis of equivalent aerodynamic size for fibers and compact particles, [Timbrell, 1965 and Stoeber, 1972] using these deposition models. This introduces a potentially significant deficiency since this would include limited consideration of interception as a collection mechanism. However, this is

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acceptable as a first approximation of those fibrous materials constituting a hazard due to deposition and long term retention in the lung.

The present standard for fibrous glass as defined by the ACGIH Threshold Limit Value, is in terms of mass of fibrous material less than 5 to 7 μ in diameter. [ACGIH, 1973b] This apparently is an effort to limit consideration to the "respirable" fraction; however, the rationale for this criterion is not clearly defined. An initial draft of the NIOSH criteria document for fibrous glass suggested a maximum concentration [ACGIH, 1971] in terms of total mass, possibly because of the uncertainties existing regarding the practicality of "respirable" sampling for fibrous material. Based on experience in "respirable" sampling for coal dust and silica, the use of cyclone presamplers seemed potentially useful in defining that fraction of the fibrous material whose aerodynamic diameter is less than 5 to 7 μ.

While previous data [Bien and Corn, 1971; Balzer, personal communication] indicated that cyclone samplers do not reproducibly sample and aerodynamically separate fibrous material, these studies did not attempt to calibrate the 10 mm cyclone for the size selective sampling of fibrous aerosols. Because of the apparent need for a sampler to separate total fibrous glass from the "respirable" fraction, a study was initiated to reevaluate the practicality of using the 10 mm nylon cyclone for the size selective sampling of fibrous materials. Our preliminary approach was directed at the calibration of the 10 mm cyclone sampler against various polydisperse, aerodynamically sized, test fibrous glass dust.

Technique

Test aerosols are produced from bulk materials in the following manner: large quantities of bulk fibrous glass are blendorized as a thin aqueous slurry, reducing individual fiber lengths but leaving fiber diameters largely unaffected. Aerosols prepared from the bulk materials treated in this fashion contain fibers with a range of well-defined diameters, and corresponding multiple lengths. The actual test aerosols are produced using a generator modeled after the device described by Timbrell and associates, 1968 (Figure 7-1). Operating principles include a gear-driven mechanical infusion device to control the slow feeding of a compact plug prepared from the blendor ground fibrous glass into the path of fast moving rotor blades. Fibers shaved from the end of the feed plug are exhausted into an aerosol chamber for sampling. Test aerosols produced by this type of generation technique are polydisperse, composed of both fiber singlets and loosely bound fibrous agglomerates, variable in total mass output, and aerodynamically related to the distribution of individual fiber diameters which compose the feed material.

Air sampling standards, based on the concept of "respirable" dust, relate the mass fraction of an aerosol capable of penetrating to and depositing in the nonciliated portion of the lung. Figure 7-2 shows the ACGIH curve relating "respirable" fraction to particle aerodynamic diameter. [ACGIH, 1973a] Figure 7-3 illustrates a calculated extension of this concept, permitting determination of the "respirable" fraction for polydisperse aerosols having a given mass median aerodynamic diameter

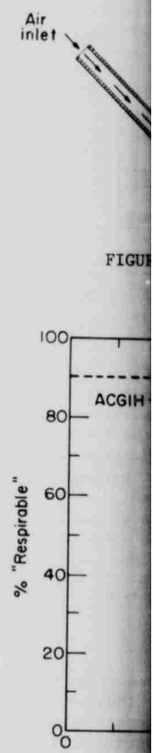


FIGURE 7-2. RESPIRABLE

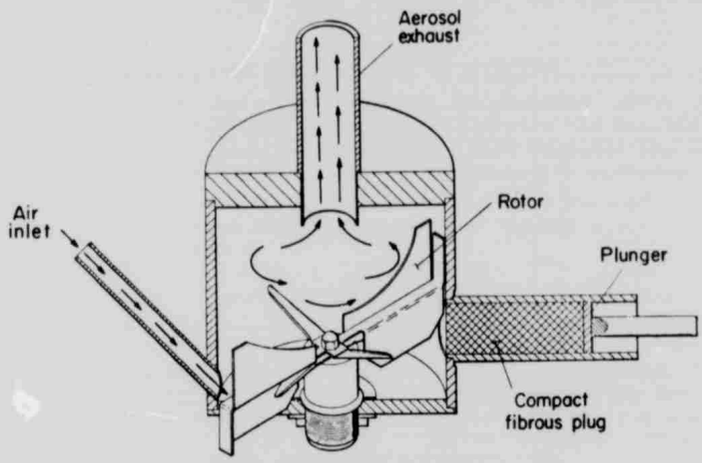


FIGURE 7-1. FIBROUS AEROSOL GENERATOR

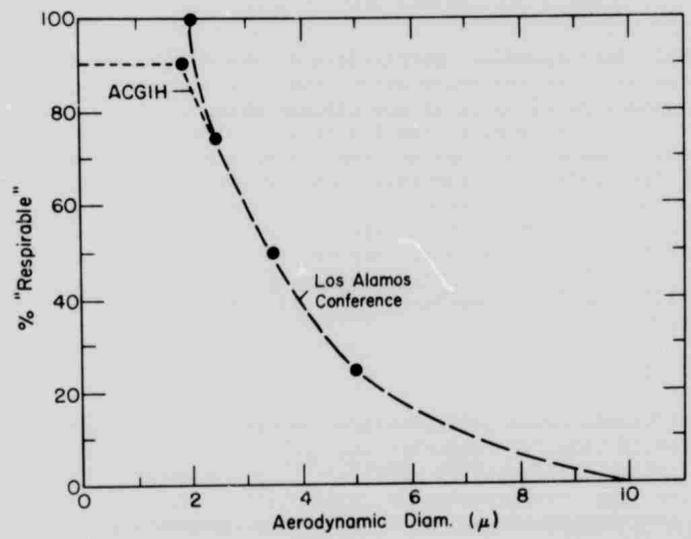


FIGURE 7-2. RESPIRABLE FRACTION AS A FUNCTION OF PARTICLE AERODYNAMIC DIAMETER

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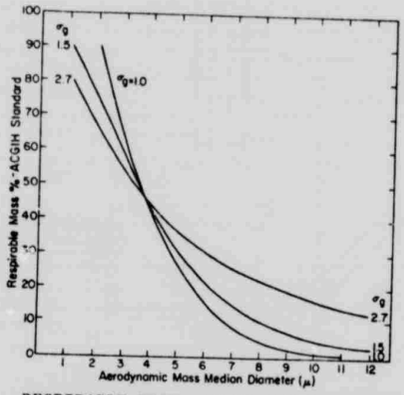


FIGURE 7-3. RESPIRABLE MASS--ACGIH STANDARD--AS A FUNCTION OF AERODYNAMIC MASS MEDIAN DIAMETER FOR POLYDISPERSE AEROSOLS

(mmad) and corresponding geometric standard deviation (GSD). Figure 7-3 shows a family of curves which define the "respirable" mass fraction of various compact particulate or non-fibrous aerosols, as a function of mmad, for different GSD's ranging from 1.0 to 2.7 using the ACGIH definition for "respirable" mass fraction of the "respirable" fraction for any log-normally size distributed aerosol, when these size parameters are known. Cyclone sampler calibration was accomplished by aerodynamically characterizing the test fibrous aerosol with the Andersen Cascade Impactor (mmad, GSD), and incrementally varying the cyclone sampling flowrate until the mass of fibrous material passing the cyclone stage matched the corresponding ACGIH "respirable" fraction for an aerosol with these size parameters.

Results

Aerodynamic size characterization of three separate test fibrous glass aerosols using the Andersen Cascade Impactor indicate that aerosols produced with the modified Timbrell type generator are log-normally distributed and aerodynamically consistent. During the experimental program, variations in the aerosol size parameters for these three different test aerosols were: mmad $\pm$ 10%, and GSD $\pm$ 5%. A fourth test aerosol, produced from a material labeled as Swedish Wool, caused major difficulties in providing reproducible aerosol generation and aerodynamic characterization and is discussed separately. Impactor data was obtained by weighing the fibrous mass collected on each impaction stage. A log probability plot of cumulative mass collected on successive impactor stages versus impactor effective cut-off diameter (ECD) for the appropriate stage defined the aerodynamic size parameters for the test aerosol. The

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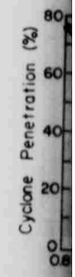


FIGURE 7-4.
AA-12 FIBROUS

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"respirable" mass fraction for a given test aerosol was calculated on the basis of this experimentally measured mmad and GSD, and the relationships detailed in Figure 7-3.

Experimental cyclone penetration data obtained for each of the test aerosols represent the average fibrous mass penetrating three cyclone samples operated simultaneously at various sampling points within the aerosol chamber. The mass fraction penetrating these cyclones was obtained by measuring the fibrous mass collected on preweighed membrane filters downstream of each cyclone and relating this mass to the average challenge mass as determined by open face filter samples operating simultaneously within the aerosol test chamber.

Cyclone calibration results are detailed in the following three graphical plots (Figures 7-4 to 7-6) showing cyclone penetration versus sampling flowrate. Analysis of these data, in conjunction with aerosol size characteristics in Figure 7-3, indicate the proper sampling flowrate to best approximate the ACGIH respirable dust definition for a given aerosol. Figure 7-4 summarizes these data for O/C AA-12 fibrous glass which produced an aerosol mmad=3.9 $\pm$ 0.4 μ, and GSD = 1.8 $\pm$ 0.1, and based on Figure 7-3, has an ACGIH defined "respirable" mass fraction of approximately 43%. The recommended cyclone sampling flowrate necessary to approximate this cut off is 1.5 liters/minute. Figure 7-5 shows similar data for O/C AAA-10 fibrous glass which provided an aerosol mmad = 3.1 $\pm$ 0.1 μ, and GSD = 1.8 $\pm$ 0.1, which should result in an ACGIH "respirable"

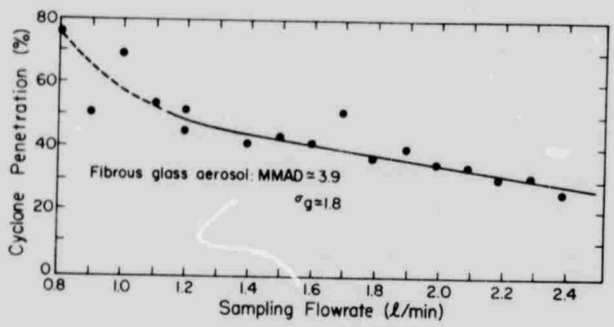


FIGURE 7-4. CYCLONE PENETRATION VS. SAMPLING FLOWRATE: O/C TYPE E, AA-12 FIBROUS GLASS

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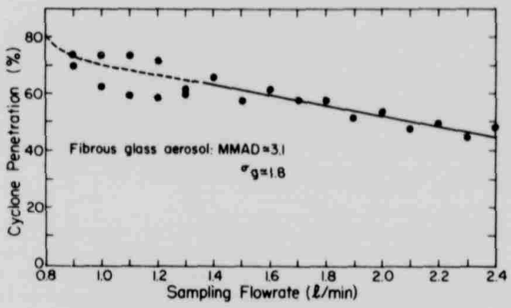


FIGURE 7-5. CYCLONE PENETRATION VS. SAMPLING FLOWRATE: O/C AAA-10 FIBROUS GLASS

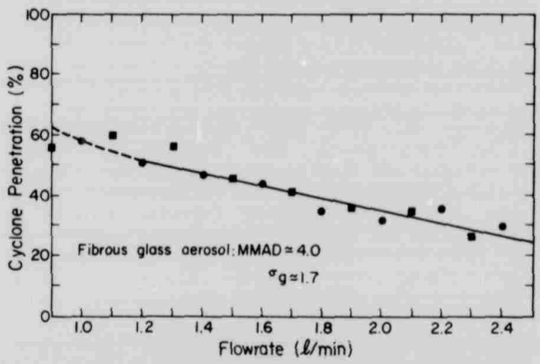


FIGURE 7-6. CYCLONE PENETRATION VS. SAMPLING FLOWRATE: TYPE O/C AA 10 FIBROUS GLASS

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mass fraction of approximately 54%. These experimental data indicate a cyclone sampling flowrate of 1.9 liters/minute, which is appropriate to approximate this "respirable" mass fraction. Figure 7-6 shows similar results for fibrous glass O/C AA-10 which has a mmad of 4.0 ± 0.3 μ, and GSD 1.7 ± 0.1 and a calculated ACGIH "respirable" mass fraction of approximately 41%. The recommended cyclone sampling flowrate to approximate this "respirable" fraction was approximately 1.7 liters/minute.

These results were obtained against test aerosols which were consistent in aerodynamic size throughout the entire test sequence. Major difficulties were encountered, with another test aerosol produced from a commercial hearing protection product marketed as "Swedish Wool." The aerosol generated from this particular material was characterized by fluctuations in total aerosol mass output and aerodynamic size. This aerosol varied in mmad from 3.3 to 5.6 μ, and required a different analytical procedure to define the proper flowrate to approximate the ACGIH "respirable" dust definition. Cyclone calibration tests conducted against this aerosol were normalized by calculating the ratio of the experimentally measured and predicted cyclone penetration at a given flowrate to that predicted on the basis of aerodynamic size measured immediately before and after each cyclone calibration run, and the relationships detailed in Figure 7-3. Figure 7-7 summarizes these data presented as a ratio of penetrations Pex/Pth (experimental cyclone penetration/theoretical penetration) plotted against cyclone sampling flowrate. The correct sampling flowrate for data normalized in this fashion occurs at Pex/Pth = 1.0. Assuming a linear fit using least squares regression analysis, Figure 7-7 indicates that a cyclone sampling flowrate of approximately 1.5 liter/minute best approximates the correlation between experimental data and theory for this aerosol. Although this graph exhibits considerable scatter, the correlation coefficient, assuming a linear fit, is 0.74.

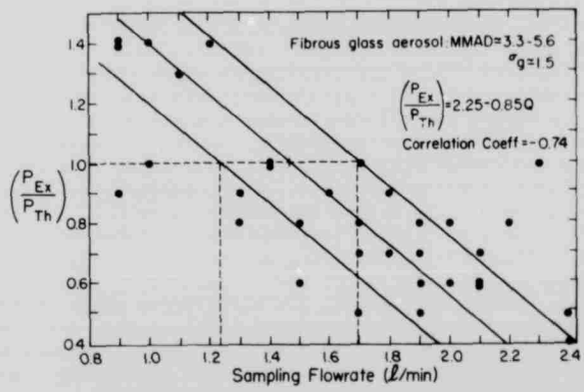


FIGURE 7-7. RATIO OF EXPERIMENTAL TO PREDICTED CYCLONE PENETRATION VS. SAMPLING FLOW RATE: SWEDISH WOOL FIBROUS GLASS

Table 7-1 summarizes these results, indicating the sampling flowrates recommended for the 10 mm nylon cyclone range from 1.5 to 1.9 liters/minute. These data suggest that cyclone collection efficiency as a function of sampling flowrate may not be as precisely defined for fibers as for particulates. However, the magnitude of the potential error in estimating the "respirable" fraction, due to this range of appropriate cyclone sampling flowrates must be considered in terms of variations inherent in field sampling. For example, if the O/C AAA-10 aerosol were sampled at 1.5 liters/minute rather than the appropriate 1.9 liters/minute flowrate, the "respirable" fraction would be overestimated by only 15%. This would be the maximum error introduced by sampling at one of the two recommended flowrate extremes, 1.5 and 1.9 liters/minute, for two aerodynamically different test aerosols. A more realistic estimate of the error associated with measuring the "respirable" fraction for different fibrous aerosols is to assume that each of the separate test aerosols is sampled at a fixed flowrate of 1.7 liters/minute.

Table 7-2 indicates that the maximum error associated with sampling three of the four test glass aerosols at 1.7 liters/minute instead of the experimentally obtained recommended flowrate previously listed is only a 4% overestimate of the ACGIH defined "respirable" mass fraction. This type of comparison is not possible for the test aerosol produced from "Swedish Wool" because of large variations in the apparent aerodynamic size. While Table 7-2 indicates that a fixed sampling flow of 1.7 liters/minute for the 10 mm nylon cyclone may provide an adequate measure of the "respirable" mass fraction of challenge fibrous glass aerosols as defined by impactor characterization, additional cyclone calibration data should be obtained prior to reaching any final conclusions regarding the size selective sampling of fibrous aerosols.

Summary

Experimental observations concerning the sampling of fibrous dusts with the 10 mm nylon cyclone sampler indicate that: 1) the cyclone, operating at various flows and sampling the same fibrous dust appears to have a reproducible and distinct aerodynamic cutoff; 2) the shallow slope of each fiber sampling curve suggests that the collection mechanism within the cyclone body is probably not entirely due to centrifugal impaction. There is some evidence that suggests fiber-fiber interception as a significant collection mechanism within the cyclone itself. Electrical charge accumulation on the generated aerosol as well as the cyclone inner walls may also enhance this suspected mechanical interception collection mechanism; 3) a fixed cyclone sampling flowrate of 1.7 liters/minute would provide an adequate approximation of the "respirable" mass fraction (as defined by the Andersen Impactor) for the fibrous aerosols tested. This observation is consistent with recommended sampling flow for particulates, [Aerosol Tech Comm, 1970] and 4) this preliminary evidence suggests the practicality of using this sampler as a field instrument for the size selective sampling of fibrous glass dusts.

TABLE 7-1
CYCLONE PERFORMANCE FOR FIBROUS GLASS AEROSOLS

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TABLE 7-1

CYCLONE PERFORMANCE FOR FIBROUS GLASS AEROSOLS

Test Aerosol Designation	Impactor Characterization No. of Tests	MMAD μ	Geometric Std. Deviation	% Respirable by ACGIH Curve approx.	Recommended Sampling Flowrate 10mm Nylon Cyclone liter/minute
O/C Type E, AA-12	12	3.9 $\pm$ 0.4	1.8 $\pm$ 0.1	43	1.5
O/C AAA-10	10	3.1 $\pm$ 0.1	1.8 $\pm$ 0.1	54	1.9
O/C AA-10	11	4.0 $\pm$ 0.3	1.7 $\pm$ 0.1	41	1.7
"Swedish Wool"	32	3.3 - 5.6	1.5 $\pm$ 0.1	Variable	1.5

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TABLE 7-2

COMPARISON OF RESPIRABLE MASS FRACTION OBTAINED USING RECOMMENDED CYCLONE SAMPLING FLOWS VS CYCLONE SAMPLING AT 1.7 LPM

Test Aerosol Designation	ACGIH Respirable Mass Fraction (%)	Recommended Cyclone Sampling Flowrate liter/minute	Predicted Respirable Mass Fraction at 1.7 liter/minute (%)	Relative Difference (%)
O/C Type E, AA-12	43	1.5	40	-3
O/C AAA-10	54	1.9	58	-4
O/C AA-10	41	1.7	41	0
"Swedish Wool"	Variable	1.5	-	-

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ENVIRONMENTAL DATA; AIRBORNE CONCENTRATIONS FOUND IN VARIOUS OPERATIONS

J. LeRoy Balzer

Introduction

MR. LYNCH: Dr. Balzer has a doctorate from the University of California at Berkeley, and is widely known for his work on asbestos and other fibers.

PRESENTATION

DR. BALZER: Few articles have been published concerning glass fiber concentrations in the environment; generally, the data published refer to exposures in the insulating or sheet metal trades, or in fibrous glass manufacturing plants.

Air samples collected on membrane filters from three specific areas were available at the University of California at Berkeley: (1) Occupational exposure to fibrous glass; (2) Ventilation systems lined with fibrous glass; (3) Ambient air samples from sites around the state of California.

Mass sampling was not appropriate for enumerating fibrous glass exposures, so methods for counting individual glass fibers were chosen. Using phase-contrast and polarizing microscopes, fibers as small as 0.5 μ were seen, although not identified by fiber type. Previous studies have shown that analytical methods for identifying fibers have been limited to fibers having diameters greater than 2.5 μ. [Fowler et al, 1971; Balzer et al, 1971]

Data from 120 membrane filter samples, 40 from each specified area, were examined by light and electron microscopy. Since amorphous mineral fibers (fibrous glass and mineral wool) are not crystalline in structure, it is possible by electron diffraction of the individual fibers to distinguish them from noncrystalline materials. Morphological characteristics of fibers such as asbestos help in this classification. Presence or absence of resins and uniformity of the fibers also assist in identification of fibrous glass and mineral wool fibers.

Sampling Methods

Filters used for this study included 37 mm and 47 mm diameter 0.8 μ nominal pore size Millipore and Nuclepore membrane filters. The 47 mm filters were mounted in 47 mm diameter open metal Millipore and Gelman aerosol filter holders. Gelman air sampling vacuum pumps, capable of flow-rate through the filter of 1-2 cu ft/minute were used. The 37 mm filters were all Millipore and mounted in plastic cassettes. Air samples were taken at flow rates between 0.1 and 2 cu ft/minute. Selected filters were weighed before and after sampling to determine the gravimetric concentrations of all particulate matter; this was reported previously. These samples were collected in workplaces, ventilation systems, and ambient air sites over a 3 1/2 year period, January 1968 to June 1971.

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Analytical Methods

The Nuclepore filter is handled differently from the Millipore filter for light microscopy. The Nuclepore filter is made of polycarbonate which is crystalline and which has two refractive indices, 1.616 and 1.584. A filter segment is mounted on a glass slide and cleared with a refractive index medium of 1.585. A polarizer is placed in the microscope light beam in front of the condenser and rotated until the Nuclepore filter looks transparent in the microscope.

Enumeration of fibers greater than 2.5 μ in diameter on Millipore filters was by the standard phase-contrast optical microscopy method developed by Edwards and Lynch. [1968] A Siemens Elmscope I or IA electron microscope operated at 100KV was used for counting and sizing the fibers on a grid prepared for each sample. The fibers were counted and sized directly on the microscope viewing screen at magnification of 200X. This magnification and the field of view resemble those of a light microscope, but the electron microscope provides much higher resolution. For identification of fibers by morphology, the electron microscope is used at 20,000X magnification. For selected area electron diffraction, the microscope is set at 20,000X magnification. The beam current is increased until diffraction rings, spots, or lines appear. If the fiber is amorphous, like glass, no diffraction pattern will be seen, only the faint diffused diffraction rings of silicon oxide.

This electron microscopic technique, including the counting and statistical methods, was developed by Mr. Jack Murchio of the School of Public Health, University of California at Berkeley. [National Technical Information Service Report, 1973]

Results

One hundred and twenty samples were collected for detailed examination; 40 from an inventory of 200 occupational exposure samples, 36 from 300 ambient air samples, and 37 from over 600 ventilation system samples lined with fibrous glass.

Ambient air samples were selected from those collected in Berkeley, San Jose, Sacramento, the Sierra Mountains, and Los Angeles. Ventilation samples represented 13 air transmission systems in the San Francisco Bay Area. [Balzer et al, 1971] The occupational exposure samples were collected from sheet metal and asbestos workers during the application and installation of fibrous glass insulating materials in northern California. [Fowler et al, 1971]

The electron microscopic technique for counting fibers collected on membrane filters is inadequate for counting fibers greater than 2.5 μ in diameter. Even with the silicon pseudo-oxidereplication technique, larger-diameter fibers can become lost when the Nuclepore filter is dissolved in chloroform. The larger-diameter fibers tend to roll to and off the edge of the grid. This phenomenon has been observed by light microscopy with vertical illumination. Large fibers can also be lost in the electron beam. Consequently, the electron microscope is used for counting fibers $\leq$ 2.5 μ

TABLE 8-1

SUMMARY OF FIBER CONCENTRATIONS BY COUNTED LIGHT AND ELECTRON MICROSCOPY

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TABLE 8-1

SUMMARY OF
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(CONCENTRATION IN FIBERS PER LITER)

Sampling Site	Glass Fibers				Other Fibers			
	No. of Samples	Arithmetic Mean	Standard Deviation	Range	Arithmetic Mean	Standard Deviation	Range
Ambient Air	36	2.57	2.98	none seen 9.0	7.70	15.97	0.1-14.6
Ventilation Systems	37	0.87	0.48	none seen 2.0	0.40	0.54	0.01-2.2
Occupational Exposure	40	405.90	532.0	0.5-2407	222.50	53.30	none seen 2859.0

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in diameter. The fiber count results are derived from a combination of the number of fibers greater than 2.5 μ diameter (light microscope), and the number of fibers less or equal to 2.5 μ diameter (electron microscope)--1/3 of the total fibers.

The concentration of fibers in the ambient air environment is 2.57 and in the ventilation systems is less than 0.5 fibers/liter (Table 8-1). The occupational environment has a glass fiber concentration of up to 2,400 fibers/liter, with a mean of 405 fibers/liter (2.4 and 0.41 f/cc). Since these were combined light and electron microscopic results, the data in Table 8-2 is presented to illustrate the ambient air results separately. It should be noted that the light microscopic count might include some electron microscope-sized fibers and vice-versa.

TABLE 8-2

COMPARISON OF LIGHT AND ELECTRON MICROSCOPY-GLASS FIBERS (CONCENTRATION IN FIBERS PER LITER)

	No. of Samples	Mean Concentrations
Ambient air	LM 38	2.5
	EM 39	0.8

The diameter of glass fibers measured from ambient air and ventilation systems had a mean fiber diameter of 4.3 μ and 3.7 μ respectively, whereas the occupational samples had a higher average fiber diameter of 6.5 μ, and a range of 0.3 μ to 25 μ, (Table 8-3). Figure 8-1 reports frequency distribution by fiber diameter. Ventilation systems had the greatest number of fibers less than 2.2 μ in diameter, while the occupational samples had the greatest number larger than 2.2 μ in diameter.

The mean diameter of glass fibers found in these samples is generally greater than 3 μ. However, samples from the ventilation system, having the lowest fiber concentration, 0.37 per liter, have the greatest number of fibers less than 3 μ in diameter, approximately 60% (Figure 8-1). About 15% of the fibers from occupational environments are less than 3 μ, and approximately 40% of ambient air fibers are less than 3.1 μ in diameter. In the samples, the presence of fibers less than 0.5 μ in diameter indicates the existence of some microfibers.

The length of fibers was greatest for fibers from the occupational environment--a mean of 103.6 μ. The ambient air and ventilation systems were similar for fiber lengths of 61.8 μ and 51.3 μ respectively (Table 8-4).

Sampling Site

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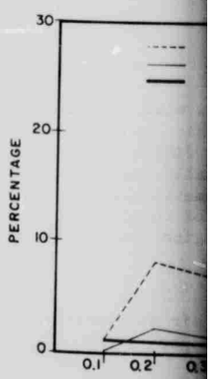


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TABLE 8-3

FIBER DIAMETERS
BY SAMPLING SITE AND FIBER TYPE
μ

Sampling Site	Arithmetic Mean	Range	Geometric Mean	Geometric Standard Deviation
Glass Fibers				
Ambient Air	4.3	0.1-17.7	2.2	2.7
Ventilation Systems	3.7	0.1-17.7	1.3	3.8
Occupational Exposure	6.5	0.3-25.0	8.4	3.4
Other Fibers				
Ambient Air	1.3	0.05-17.7	0.3	4.7
Ventilation Systems	4.3	0.05-25.0	1.5	3.7
Occupational Exposure	0.87	0.05-25.0	0.07	11.4

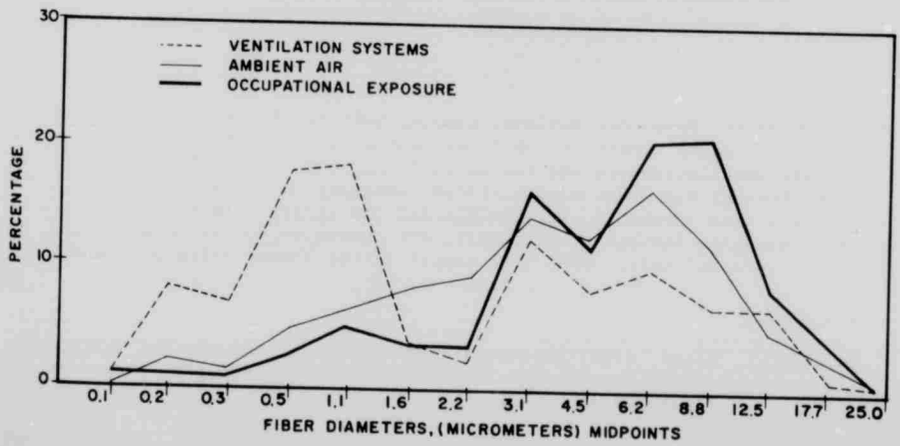


FIGURE 8-1. DISTRIBUTION OF GLASS FIBERS BY DIAMETERS AND SAMPLING LOCATION

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TABLE 8-4

FIBER LENGTHS
BY SAMPLING SITE AND FIBER TYPE
μ

Sampling Site	Arithmetic Mean	Range	Geometric Mean	Geometric Standard Deviation
Glass Fibers				
Ambient Air	61.8	1.2-160.0	16.0	5.0
Ventilation Systems	51.3	1.2-160.0	11.0	6.4
Occupational Exposure	103.6	1.2-160.0	60.0	2.5
Other Fibers				
Ambient Air	23.7	1.2-160.0	17.0	1.5
Ventilation Systems	66.9	1.2-160.0	15.0	6.0
Occupational Exposure	40.4	1.2-160.0	2.2	8.0

Results from occupational samples indicate fibers with the greatest mean diameter and length of the three sample types. This would suggest that for ventilation systems and ambient air, the larger fibers settle out. The mechanical and other manipulations necessary to install fibrous glass materials are probably responsible for the larger diameter and length of fibers becoming airborne. Certainly, the concentration of total fibers in the occupational environment is a result of the installation processes.

Conclusions

(1) Glass fiber concentrations in the ambient air and ventilation system samples are less than three fibers per liter (0.003 fibers /cc).

(2) The ventilation duct samples would suggest that fibrous material, specifically glass fibers, are being removed by passage through systems which are lined with fibrous glass.

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that fibrous removed by ous glass.

(3) Glass fiber concentrations in the occupational environment are generally less than one fiber/cc.

(4) Identification of fibers with diameters less than 2.5 μ is possible using electron microscopy.

(5) Glass fibers are one of the fibrous materials found in the ambient air and account for approximately 1/3 of the total fibrous material.

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SAMPLING STRATEGY, AIR SAMPLING METHODS, ANALYSIS, AND AIRBORNE CONCENTRATIONS OF FIBROUS GLASS IN SELECTED MANUFACTURING PLANTS

Morton Corn

Introduction

MR. LYNCH: The paper will be presented by Dr. Morton Corn, Professor in the Department of Occupational Health, Graduate School of Public Health, University of Pittsburgh. Dr. Corn's many accomplishments in aerosol technology and industrial hygiene are certainly well known to you all.

Presentation

DR. CORN: The strategy of sampling to evaluate the concentration of an airborne substance is closely coupled to the purpose of the sampling program. Several purposes are possible; the following four are, perhaps, the most common.

a. To determine, in a relatively quick and efficient manner, the range of airborne concentrations occurring in practice. Thus, a guide or "marker" to existing conditions is desired.

b. To evaluate the effectiveness of pollutant control techniques, including equipment and work practices. Achievement of this purpose implies the completion of measurements before and after implementation of control techniques.

c. To determine adherence or lack of adherence to a standard for the airborne substance, i.e., this purpose fulfills a regulatory function, that of determining compliance with a legal standard.

d. To determine the exposure of men employed in different job categories for purposes of epidemiological studies to assess the possible effects of the agent on the health of individuals exposed to it during production or product usage.

The professional efforts required to fulfill these four purposes of sampling are different. Roach [1966] was one of the first to clearly see that a relationship exists between the sampling strategy and the behavior of the agent in the human body following inhalation. His remarks are related to purpose d and assist us in fulfilling that purpose. At present, there are no guidelines for fulfilling purpose a. In practice, precision of sampling results has engendered a feeling of security; lack of precision has led to additional sampling. Purpose b is fulfilled, in general, by accumulating sufficient samples before and after installation of controls to perform a standard test of the difference of mean concentrations associated with the two conditions. Guidelines are beginning to appear for the minimal ingredients of a sampling strategy to ensure adherence to a legal standard, expressed as a single result for the entire work period or a series of short term or "grab" samples. [Leidel and Busch, 1973]

The results presented here are derived from sampling data obtained to fulfill purpose a. That is, we desired a "fix" on airborne concentrations

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of fibers in three manufacturing facilities representing different types of efforts in the industry. An investigation is now in progress to determine airborne fiber concentrations and total suspended particulate matter concentrations in approximately 20 fibrous glass facilities to fulfill the requirements of purpose d. In this case, we are determining exposures as they relate to job categories and job areas, as extracted from employment histories of men involved in the epidemiological study. The goal of this approach is to obtain an exposure index for each man, one which recognizes and properly accounts for his different exposures throughout the entire period of his employment in a variety of jobs in the plant.

The results reported here for airborne concentrations of total suspended particulate matter and fibers are based on analysis of samples collected during 1972-73 in a brief survey undertaken to fulfill purpose a. These results will be published elsewhere. [Corn and Sansone, 1974] Results associated with the more extensive epidemiological survey are not ready for presentation at this time. In this article we describe our sampling and analytical methods and indicate where we have altered these methods for use in the current epidemiological investigation.

Three large manufacturing facilities, designated as Plants A, B, and C, were surveyed; a total of 125 air samples were obtained. Plant A produces insulation in a variety of forms, including thermal insulation, molded pipe, acoustical tile, rigid and flexible round duct, furnace filters, and bonded mat. Plant B is characterized by a wide variety of products, including tube filters, several forms of insulation, and woven and non-woven textile products. The products of Plant C are all molded, fiber glass reinforced plastics in a variety of shapes and sizes. The three plants are owned by three different companies. Thus, results reported here are not associated with a single corporate approach to fibrous glass manufacturing, but rather represent a sampling of manufacturing practice and airborne particulate concentrations in this industry.

Sampling and Analytical Methods

Both environmental and personal samples were collected. Environmental or area samples are those collected from areas of work environment which are thought to be representative of the airborne concentrations of total suspended particulate matter (TSPM) that may reach the breathing zones of employees. Personal samples are those which are collected from within the breathing zones of the employees. The latter are obtained by having the worker wear a filter with holder and pump while he performs his work tasks. Particular attention was paid to not permitting personal samples to be contaminated with dust from worker garments.

In general, personal samples were obtained when it was observed that a worker was in close proximity to one or more processing operations for extended periods of time. In such cases, personal samples can be expected to yield a higher estimate of exposure to airborne particulate matter than an environmental sample obtained at what one hopes to be a representative, fixed location. Enviromental samples were obtained in large plant areas where employees appeared to traverse the entire floor area in order to

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micrometer with 10 μ
mounted in the microscope
the area of the micros
the lengths and diameters

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perform their work tasks. All environmental samples were obtained with the filter placed at breathing level, at a location in the work area believed to represent contaminated air conditions. The reproducibility of sampling results was estimated by obtaining multiple samples at most locations.

In order to obtain a filter sample which could be validly analyzed for fibers by optical microscopy, that is, filters not overloaded with particles, it was necessary to change filters in a sampler every 2 hours. Thus, personal samples do not reflect full-shift time-weighted average exposures of the men because four 2-hour samples, representing 8 hours of exposure, were not obtained on any men. We believe that by selecting men in close proximity to operations which were dusty, we obtained average 2-hour exposures which would be higher than time-weighted average exposures to airborne dust or fibers. The limited scope of this survey and the large sizes of the plants involved precluded obtaining time-weighted average exposures.

Samples of total suspended particulate matter (TSPM) were collected on 37 mm diameter Millipore Type AA membrane filters. The filters have a pore size of 0.8 μ and have been shown to retain, with 100% efficiency, particles as small as 0.01 μ diameter. [Megaw and Wiffen, 1963]

Before use, the filters were desiccated and weighed to $\pm$ 0.01 mg. After weighing, the filters were placed in completely enclosed plastic filter holders. For sample collection, one part of the filter holder was removed, which allowed the filter to be used in the open-face mode. To prevent leakage into the filter holder, a cellulose band was allowed to shrink tightly around the circumference of the filter holder, thus effectively preventing contamination of the sample.

Samples of air were drawn through the filter at a flowrate of 2 liters/minute by means of a pump calibrated for volume of air flow with the filter and sampling line in place. The filter was oriented face down during sampling; air flowrate was checked periodically. After collection the filter was sealed in its holder until analysis could be performed.

The sample and filter were desiccated and reweighed in the laboratory. The gain in weight represented the total suspended particulate matter collected. Airborne concentrations of total suspended particulate matter, expressed as mg/cu m, were calculated from sample weight and the sampled volume of air.

A pie-shaped portion of the 37 mm membrane filter was rendered transparent by mounting it in a solution of equal parts by volume of dimethyl phthalate and diethyl oxalate. These samples were analyzed for fibers by optical microscopy using phase contrast illumination. A Reichert phase contrast objective with 40X magnification and a numerical aperture of 0.65 was used. The eyepiece had a magnification of 10X; total nominal magnification was 400X. This arrangement of apparatus yielded resolution to 0.56 μ. It was possible to discern particles of 0.2 μ size. A stage micrometer with 10 μ divisions was used to calibrate a Porton graticule mounted in the microscope eyepiece. By this means we accurately defined the area of the microscopic field of evaluation and were able to measure the lengths and diameters of the fibers observed. The number of fields

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evaluated with each sample depended on the fiber concentration sampled. In most cases, 40 or more microscopic fields were evaluated to obtain a fiber count and, hence, an airborne fiber concentration.

The airborne concentration of fibers was calculated using the equation:

$$\text{No. of fibers per milliliter} = \frac{A}{(F)(Q)(a)} (N \pm \sqrt{N}) \quad \text{[Roach, 1966]}$$

where: A = Effective filter area (860 sq mm)
F = Number of fields evaluated
Q = Air volume sampled (cc)
a = Area of one microscopic field = 7140 square microns
N = Number of fibers counted

The statistical error of the count was estimated from the square root of the count. [Herdan, 1960]

The sensitivity of this technique, that is, the lowest fiber concentration detectable by optical microscopy, was 0.01 fibers/cc.

In some cases a second portion of the sample was ashed at low temperature in oxygen plasma. The remains of the filter and sample were examined with phase contrast illumination using the optical arrangement described above.

All evaluation procedures adhered to the guidelines offered by the Occupational Safety and Health Administration (OSHA Standard 29 CFR 1910.93).

One half of each 37 mm diameter membrane filter used to obtain an air sample was placed in a small glass vial. The filters were ashed in a low temperature oxygen plasma furnace for 4 to 8 hours at temperatures which did not exceed 200C. The ashes of each sample were mixed in the vial with a solution of 5% collodion in amyl acetate. The suspension was further agitated by ultrasonic vibration and then made up to a volume of exactly 1.0 cc. One tenth of a cubic centimeter of suspension was removed by means of a graduated syringe and placed on a clean distilled water surface in a beaker. The spreading of this suspension on the surface was limited to the inside of a partially submerged 15 mm diameter teflon ring. After the solvent evaporated, three electron microscope grids were lifted from beneath the film; the grids were covered with the collodion film and the sample. After the film dried on the electron microscope grids, the grid edges were scored with a needle; the coated grids were stored for electron microscope evaluation.

The collodion coated grids with sample were placed in a Phillips Model 75 electron microscope. Sample fields were photographed at a nominal magnification of 2,000X; calibration was achieved with a 28,800 lines/ inch reference grid. The final magnification of photographic prints was 6,130X.

The resolving power of the electron microscope used is approximately 50 to 100 Angstrom units. The sensitivity of our techniques, that is, the

lowest detectable airborne
0.6 fibers/cc.

Results and Discussion

Results of measurement
in tabular form as TSPM (mg/
according to fiber length in
microscopy were classified by
length; percent of fibers
fibers > 10 μ length and < 3.
analysis of samples were p
and fibers > 1 μ length. Wit
by plant areas. A descript
not presented here because pr
this subject. Results of s
Plant B areas in Table 9-2, a

Discussion

In general, the most
absence, with few exceptions,
The sites where such fibers w
Plant, in Plant B in the Fibe
Plant C in the large Prefor
highest concentration of fibe
was associated with an envir
Molding Department. At this t
occurrence of larger numbe
and particularly greater than
Perhaps insight into the
processing will clarify the r

In Plant A, total susp
from 0.7 to 6.0 mg/cu m: the
upper quartile value was 4.1
All concentrations derived fr
below the 10 mg/cu m thre
dustiest areas of Plant A were
Department. The percentage o
3.5 μ diameter varied greatly
collected in the same area
associated with more than 50%
[Megaw, 1963] collected in
less than 5 μ length ranged f
the latter occurring in the A
values of this parameter were

Total suspended partic
from less than 0.1 mg/cu m to
values were 0.5 mg/cu m and 1.
0.8 mg/cu m. The highest dust

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lowest detectable airborne fiber concentration by electron microscopy, is 0.6 fibers/cc.

Results and Discussion

Results of measurements of airborne particulate matter are presented in tabular form as TSPM (mg/cu m) and fiber concentrations (fibers/cc) according to fiber length in Appendix. Samples analyzed by phase contrast microscopy were classified by length as follows: $< 5\ \mu$ length; $> 5\ \mu$ length; percent of fibers $> 5\ \mu$ length and $< 3.5\ \mu$ diameter; percent of fibers $> 10\ \mu$ length and $< 3.5\ \mu$ diameter. Results of electron microscopic analysis of samples were placed into two groupings: fibers $< 1\ \mu$ length and fibers $> 1\ \mu$ length. Within tabular presentations, results are grouped by plant areas. A description of processing operations in plant areas is not presented here because previous speakers on the program have addressed this subject. Results of sampling in Plant A are presented in Table 9-1, Plant B areas in Table 9-2, and Plant C in Table 9-3 (see Appendix).

Discussion

In general, the most interesting result of these surveys was the absence, with few exceptions, of airborne fibers less than $1\ \mu$ in length. The sites where such fibers were detected were in Plant A in the Bonded Mat Plant, in Plant B in the Fiber Production and Microfiber Areas, and in Plant C in the large Preform Area and the Custom Molding Department. The highest concentration of fibers less than $1\ \mu$ length (1.30 ± 0.92 fibers/cc was associated with an environmental sample obtained in the Plant C Custom Molding Department. At this time we cannot offer an explanation for the occurrence of larger numbers of airborne fibers greater than $1\ \mu$ length, and particularly greater than $5\ \mu$ length, but so few less than $1\ \mu$ length. Perhaps insight into the nature of breakage of fibrous glass during processing will clarify the reasons for this observation.

In Plant A, total suspended particulate matter concentrations ranged from 0.7 to 6.0 mg/cu m: the lower quartile value was 1.3 mg/cu m and the upper quartile value was 4.1 mg/cu m; the median value was 2.6 mg mg/cu m. All concentrations derived from environmental or personal samples were below the 10 mg/cu m threshold limit value for fibrous glass dust. The dustiest areas of Plant A were the Filter Factory and the Reconditioning Department. The percentage of fibers greater than $5\ \mu$ length and less than $3.5\ \mu$ diameter varied greatly from area to area, and even among samples collected in the same area; only one area, the Bonded Mat Plant, was associated with more than 50% values of this parameter for all samples [Megaw, 1963] collected in a single area. The concentrations of fibers less than $5\ \mu$ length ranged from none detected to 0.19 ± 0.05 fibers/cc, the latter occurring in the Aeroflex Section. The lower and upper quartile values of this parameter were 0.02 and 0.07 fibers/cc, respectively.

Total suspended particulate matter concentrations in Plant B ranged from less than 0.1 mg/cu m to 5.2 mg/cu m. The lower and upper quartile values were 0.5 mg/cu m and 1.4 mg/cu m, respectively; the median value was 0.8 mg/cu m. The highest dust concentrations of 5.2 mg/cu m and 4.7 mg/cu m

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were associated with personal samples obtained on personnel in the fiber production area. The concentrations of fibers less than 5 μ length ranged from 0.01 to 1.34 fibers/cc, the latter value occurring in the Filter Tube Finishing Area, where the second highest concentration of 1.12 fibers/cc was also measured. Both of these concentrations were associated with personal samples. The lower and upper quartile values of the concentration of fibers less than 5 μ length were 0.02 and 0.80 fibers/cc, respectively; the median value was 0.04 fibers/cc. Five samples from this plant were associated with detectable concentrations of fibers/cc less than 1 μ length. These samples were obtained in the Fiber Production and Microfiber Areas and were associated with concentrations from 0.32 ± 0.32 to 1.06 ± 0.61 fibers/cc. As was found in Plant A, the percentage of fibers greater than 5 μ length and less than 3.5 μ diameter was a highly variable parameter, both between and within plant areas.

Total suspended particulate matter concentrations in Plant C ranged from 0.2 to 6.8 mg/cu m. The lower and upper quartile values were 1.5 mg/cu m and 3.8 mg/cu m, respectively; the median value was 2.8 mg/cu m. The highest TSPM concentrations of 6.8 cu m and 5.4 mg/cu m were obtained in the Custom Molding Department and Small Preform Area, respectively. The concentrations of fibers less than 5 μ length ranged from none detected to 0.17 fibers/cc. Ashing of samples increased the content of fibers less than 5 μ length and, hence, the airborne concentrations of these fibers derived from analysis of ashed samples. Ashing of samples may result in greater visibility of these fibers in the microscope or the large fibers may be degraded by the ashing process. Only three samples obtained in this plant were associated with detectable concentrations of fibers less than 1 μ length. These samples were obtained in the Large Preform Area, the Panel Department and the Custom Molding Department. As in Plants A and B, fibers less than 3.5 μ diameter were associated with lengths which showed great variability when grouped in the categories of percent of fibers greater than 5 μ or percent of fibers greater than 10 μ.

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Introduction

MR. LYNCH: The n
who is an engineer with
Investigations in NIOSH.
been in field studies for
his Master of Science degr

Presentation

MR. DEMENT: The
goals. First of all, we w
glass fiber exposure
therefore, we wanted to ch
producing respirable glass

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Study Methods

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ENVIRONMENTAL ASPECTS OF FIBROUS GLASS PRODUCTION AND UTILIZATION

John Dement

Introduction

MR. LYNCH: The next paper will be presented by Mr. John M. Dement, who is an engineer with our Division of Field Studies and Clinical Investigations in NIOSH. Mr. Dement was with EPA for several years and had been in field studies for 3 years prior to his going to Harvard to receive his Master of Science degree in industrial hygiene.

Presentation

MR. DEMENT: The NIOSH environmental studies had several primary goals. First of all, we were most interested in the pulmonary effects of glass fiber exposure rather than the related dermatitis problems; therefore, we wanted to choose operations for study which had potential for producing respirable glass fibers.

As several previous speakers have pointed out, glass fiber operations are usually classified by the industry as textile or wool operations. Textile fibers are generally formed as continous filaments and are usually greater than 3.0 μ in diameter. In contrast, fibers formed by wool forming methods may be as small as 500 Angstroms in diameter and shorter than 1.0 μ in length. Due to these considerations and after review of previous Public Health Service data collected and reported by Johnson et al, [1969] it was decided that the present study should be confined primarily to those operations producing or using wool type glass fibers.

Study Methods

During the present study, lasting from approximately June 1972 to September 1973, investigations were made in four facilities producing standard insulation products such as home insulation and in six facilities producing or using small-diameter glass fibers. In this presentation, small-diameter fibers are those less than 1.0 μ in diameter. One facility producing plastic reinforced products was also surveyed. Fairly complete industrial hygiene surveys were made in each facility, including air samples for glass fibers, free silica in the glass batch operations, phenol and formaldehyde from the associated resin binders and solvent vapors where appropriate. However, only the glass fiber levels found in these facilities will be reported here.

Samples for airborne fibrous glass were collected in all facilities for simultaneous evaluation of total airborne dust concentrations in terms of mg/cu m and airborne fiber concentrations in terms of fibers/ml.

Two samplers were placed on each worker or at each stationary sample location. One sampler collected dust for laboratory evaluation by fiber counting and sizing and the other collected a total airborne dust gravimetric sample. No attempt was made to take respirable dust samples

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due to difficulties encountered by Bien and Corn, [1971] in calibration of these instruments for fibrous aerosols.

All samples were four to six hours in duration. Total dust gravimetric samples were collected at a flow of 2.0 liters/minute using 37 mm diameter Mine Safety Applicances Co. PVC filters, tared and reweighted to the nearest 0.01 milligram using a "Cahn Gram Electrobalance."

Samples for fiber count and sizing were collected on Millipore type "AA" membrane filters also at a flow of 2.0 liters/minute. Laboratory counting and sizing was done in two ways. For those samples collected in operations involving a preponderance of large fibers, counts and sizing were done at 430X magnification using dry objectives; however, for those operations where most fibers were less than 1.0 μ in diameter, counts and sizing were made at 1000X magnification using oil immersion objectives. All samples were mounted for counting using the present asbestos method. Using these techniques, nearly all airborne glass fibers may be counted using optical microscopy.

In addition to optical fiber sizing, a transmission electron microscope was used to size airborne fibers collected in operations producing small diameter fibers. Air samples were prepared by the method described by Fraser, 1953, and fibers sized from micrographs at a total magnification of approximately 16,000X.

For the sake of clarity, sample results were grouped as large fiber and small fiber operations.

Large Fiber Operations

Table 10-1 shows a summary of the airborne fiber concentrations found in the four facilities producing conventional insulation products such as home and appliance insulation and thermal pipe insulation. Concentrations were found to be extremely low with the highest average concentration being about 0.1 fibers/ml. The highest single concentration seen was 0.83 fibers/ml.

Table 10-2 shows a summary of the total airborne dust concentrations found in these same four facilities. The highest mean total dust concentration of 2.73 mg/cu m was observed in plant "C." The highest single concentration observed was 14.5 mg/cu m also in plant "C."

The airborne fiber diameter distributuions by operation for these same four facilities are shown in Table 10-3. The smallest count median diameter was 1.1 μ in two operations of plant "C." From this table, one can see that a large percent of the airborne fibers in this facility were less than 3.5 μ in diameter, ranging from 35 to 98%. From these data, and considering the results shown by Drs. Timbrell and Harris earlier in this symposium, one can conclude that a large fraction of airborne fibers in these operations could be considered respirable.

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no. samples

SUMMARY
FIB

ALL OPERATIONS
mean
range
no. samples

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TABLE 10-1

SUMMARY OF FIBER CONCENTRATIONS (fiber/ml) IN
LARGE FIBER INSULATION PRODUCTION FACILITIES

	PLANT			
	A	B	C	D
ALL OPERATIONS				
mean	0.06	0.11	0.13	0.08
range	0.01-0.13	0.0 -0.47	0.04-0.26	0.01-0.83
no. samples	47	48	21	49

TABLE 10-2

SUMMARY OF TOTAL DUST CONCENTRATIONS (mg/cu m) IN
FIBER INSULATION PRODUCTION FACILITIES

	PLANT			
	A	B	C	D
ALL OPERATIONS				
mean	0.83	1.28	2.73	0.34
range	0.1-3.8	0.3-4.8	0.2-14.5	0.1-1.0
no. samples	44	48	21	33

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TABLE 10-3

SUMMARY OF AIRBORNE FIBER DIAMETER DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN WOOL INSULATION MANUFACTURING FACILITIES
(Large Diameter Glass Fibers)

OPERATION	INSULATION PLANT A	B	C	D
Centrifugal Formed Building Insulation				
Count Median Diameter, μ	2.3	1.1	---	1.3
Percent <1.0 μ	10	46	---	30
Percent <3.5 μ	70	93	---	90
Centrifugal Formed Appliance Insulation				
Count Medium Diameter, μ	---	1.1	---	---
Percent <1.0 μ	---	46	---	---
Percent <3.5 μ	---	91	---	---
Flame Attenuated Insulation				
Count Median Diameter, μ	2.8	---	1.3	---
Percent <1.0 μ	4	---	35	---
Percent <2.5 μ	60	---	98	---
Pipe Attenuated Insulation				
Count Median Diameter, μ	2.1	---	1.4	2.0
Percent <1.0 μ	3	---	16	15
Percent <3.5 μ	80	---	88	85
Scrap Reclamation				
Count Median Diameter, μ	4.3*	1.3	1.9	2.1
Percent <1.0 μ	2	34	17	14
Percent <3.5 μ	35	87	70	80

*Scrap reclamation operations in this plant include scrap from fibrous glass textile operations.

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Table 10-4 shows the airborne fiber lengths also for the same four facilities. The smallest count median length observed was 19 μ and the largest was 70 μ. One interesting thing to note is the small fraction of airborne fibers less than 5 μ in length, ranging from 2 to 7%.

TABLE 10-4

SUMMARY OF AIRBORNE FIBER LENGTH DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN WOOL INSULATION MANUFACTURING FACILITIES
(Large Diameter Glass Fibers)

OPERATION	INSULATION PLANT A	B	C	D
Centrifugal Formed Building Insulation				
Count Median Length, μ	37	19	---	24
Percent <5.0 μ	2	7	---	4
Percent <50 μ	60	81	---	74
Centrifugal Formed Appliance Insulation				
Count Median Length, μ	---	28	---	---
Percent <5.0 μ	---	7	---	---
Percent <50 μ	---	68	---	---
Flame Attenuated Insulation				
Count Median Length, μ	70	---	33	---
Percent <5.0 μ	<1	---	3	---
Percent <50 μ	40	---	60	---
Pipe Insulation				
Count Median Length, μ	39	---	25	30
Percent <5.0 μ	<1	---	4	---
Percent <50 μ	60	---	71	68
Scrap Reclamation				
Count Median Length, μ	60*	23	60	30
Percent <5.0 μ	<1	5	2	6
Percent <50 μ	42	70	42	64

*Scrap reclamation operations in this plant include scrap from textile operations.

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Figure 10-1 shows a typical photomicrograph of airborne fibers from a large fiber insulation operation. The fibers are relatively large in both diameter and length; however, a few small fibers can be noted.

Figure 10-2 shows another photomicrograph of airborne fibers from these operations; however, it shows more fibers less than 10 μ in length.

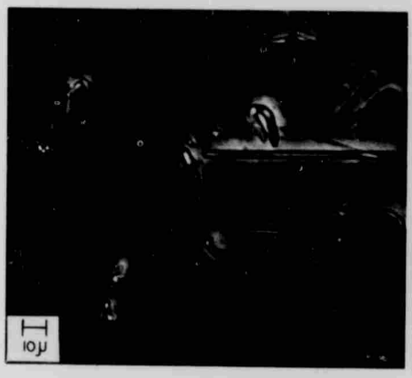


FIGURE 10-1. PHOTOMICROGRAPH OF AIRBORNE FIBERS IN A LARGE INSULATION FACILITY.



FIGURE 10-2. PHOTOMICROGRAPH OF AIRBORNE FIBERS IN A LARGE FIBER PRODUCTION FACILITY

Small Fiber Operations

As previously mentioned, six facilities producing or using small diameter or so called "microfibers" were included in the present studies. These included two facilities producing these fibers, two facilities making fibrous glass paper and two facilities fabricating aircraft thermal insulation. The fiber diameters for the products produced in these facilities ranged from less than 0.1 to approximately 2.0 μ with a large majority of the fibers being less than 1.0 μ in diameter.

Table 10-5 shows the mean airborne concentrations observed in these six facilities. Mean airborne fiber concentrations for these facilities ranged from 1.0 to 21.9 fibers/ml with the single highest concentration being 44.1 fibers/ml. In the bulk fiber handling operation, four of the six facilities had a mean concentration in excess of 5.0 fibers/ml. These concentrations are clearly orders of magnitude higher than those observed in the four conventional insulation products facilities.

TABLE 10-5

AIRBORNE FIBER CONCENTRATIONS (Fibers/ml) IN OPERATIONS PRODUCING OR USING SMALL DIAMETER GLASS FIBERS

PLANT

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TABLE 10-5

AIRBORNE FIBER CONCENTRATIONS (Fibers/ml) IN OPERATIONS PRODUCING OR USING SMALL DIAMETER GLASS FIBERS

OPERATIONS	PLANT							
	BULK FIBER PRODUCTION		PAPER MANUFACTURE		AIRCRAFT INSULATION FABRICATION			
	C*	E	F**	G**	H	I**		
Bulk Fiber Handling								
Mean	1.0	9.7	5.8	21.9	1.2	14.1		
Range	(0.1-1.7)	(0.9-33.6)	(4.7-6.9)	(8.9-44.1)	(0.4-3.1)	(3.2-24.4)		
Number of Samples	5	54	2	3	13	3		
Fabrication and Finishing								
Mean	---	5.3	1.9	10.6	0.8	2.1		
Range	---	(0.3-14.3)	(1.6-2.1)	---	(0.2-4.4)	---		
Number of Samples	---	24	2	1	15	1		

*In addition to the large wool insulation on operations, Plant C had several lines producing small diameter fibers.
**Only limited surveys were conducted in these facilities.

Shown in Table 10-6 are the mean total airborne dust concentrations for three of these facilities. All operations had concentrations less than 1.0 mg/cu m with the single highest observed concentration being 2.0 mg/cu m.

TABLE 10-6

TOTAL AIRBORNE DUST CONCENTRATIONS (mg/cu m) IN OPERATIONS PRODUCING OR USING SMALL DIAMETER GLASS FIBERS*

OPERATION	PLANT		
	BULK FIBER PRODUCTION		AIRCRAFT INSULATION FABRICATION
	C	E	H
Bulk Fiber Handling			
Mean	0.4	0.7	0.6
Range	(0.1-1.1)	(0.2-2.0)	(0.2-1.4)
Number of Samples	5	25	8
Fabrication and Finishing			
Mean	---	0.3	0.4
Range	---	(0.1-0.7)	(0.2-0.9)
Number of Samples	---	13	10

*Samples for total dust were not collected in facilities in which limited surveys were conducted.

Table 10-7 shows the airborne fiber diameter distributions by operation as determined by optical microscopy for these six facilities. A large majority of all airborne fibers are seen to be less thant 0.5 μ in diameter, ranging from 40 to 85%.

Airborne fiber length distributions for these same facilities are shown in Table 10-8. It can be seen that 5 to 45% of the airborne fibers were less than 5.0 μ in length. This is in obvious contrast to the 2 to 7% observed for the four large fiber insulation operations.

The transmission electron microscope was used to size both airborne fiber diameters and lengths for the bulk fiber handling operations of the two fiber production facilities. Figure 10-3 shows the airborne diameter distributions obtained. Count median diameters were 0.15 and 0.18 μ for the two operations.

AVERAGE AIRBORNE FIBE
MICROSCOPY IN OPERATI

FACILITIES

Bulk Fiber Production

Plant C
Plant E

Paper Manufacture

Plant F
Plant G

Aircraft Insulation Fabrication

Plant H
Plant I

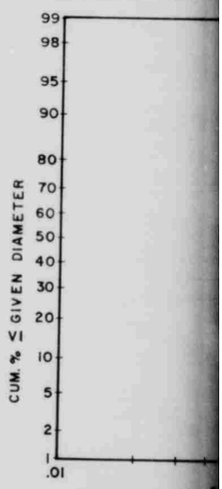


FIGURE 10-3.
SMALL DIAME

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TABLE 10-7

AVERAGE AIRBORNE FIBER DIAMETER DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN OPERATIONS PROVIDED OR USING SMALL DIAMETER GLASS FIBERS

FACILITIES	PERCENT OF FIBERS - UPPER CLASS INTERVAL, μ							
	BULK FIBER HANDLING				FABRICATION AND FINISHING			
	0.5	1.0	1.9	3.8	0.5	1.0	1.9	3.8
Bulk Fiber Production								
Plant C	85	96	100	100	---	---	---	---
Plant E	72	88	95	98	76	93	98	100
Paper Manufacture								
Plant F	40	62	75	89	55	83	94	100
Plant G	84	96	100	100	53	86	97	100
Aircraft Insulation Fabrication								
Plant H	48	72	92	97	46	73	89	97
Plant I	75	91	98	99	69	92	98	100

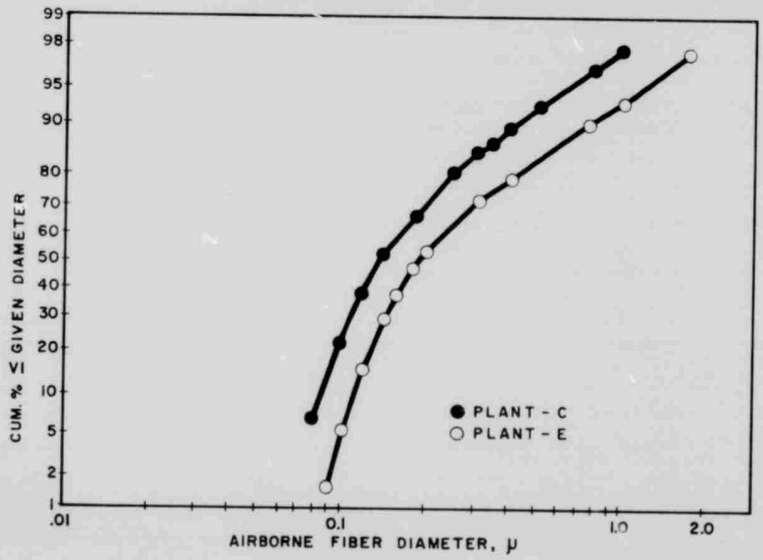


FIGURE 10-3. AIRBORNE FIBER DIAMETER DISTRIBUTION, SMALL DIAMETER FIBER PRODUCTION

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TABLE 10-8

AVERAGE AIRBORNE FIBER LENGTH DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN OPERATIONS PRODUCING OR USING SMALL DIAMETER GLASS FIBERS

FACILITIES	PERCENT OF FIBERS - UPPER CLASS INTERVAL, μ							
	BULK FIBER HANDLING				FABRICATION AND FINISHING			
	5	11	22	48	5	11	22	48
Bulk Fiber Production								
Plant C	10	20	40	80	---	---	---	---
Plant E	23	39	54	87	23	38	32	70
Paper Manufacture								
Plant F	8	27	52	73	5	22	54	86
Plant G	33	52	77	86	30	34	60	86
Aircraft Insulation Fabrication								
Plant H	14	31	50	94	18	37	66	91
Plant I	45	64	73	87	38	61	84	97

TABLE 10-9

AIRBORNE FIBER AND TOTAL DUST CONCENTRATION IN A FIBROUS GLASS REINFORCED PLASTICS OPERATIONS

OPERATION	FIBER CONCENTRATION Fibers/ml	TOTAL DUST CONCENTRATION mg/cu m
Fibrous Glass Laminating		
Mean	0.07	1.1
Range	(0.02-0.10)	(0.1-5.7)
Number of Samples	7	7
Cutting and Grinding		
Mean	0.03	3.6
Range	(0.02-0.05)	(1.2-4.9)
Number of Samples	3	3

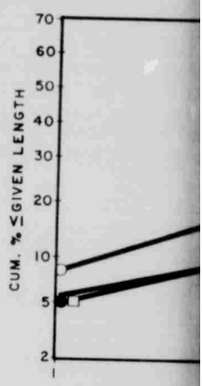


FIGURE 10-
SMALL DIA



FIGURE 10-5. ELECTRON M
AIRBORNE FIBERS IN A SMA
PRODUCTION FACILITY

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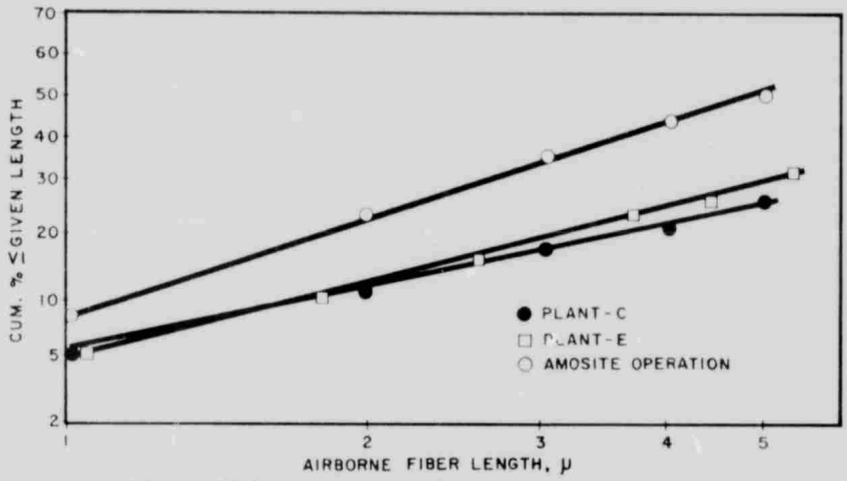


FIGURE 10-4. AIRBORNE FIBER LENGTH DISTRIBUTIONS, SMALL DIAMETER FIBER PRODUCTION.



FIGURE 10-5. ELECTRON MICROGRAPH OF AIRBORNE FIBERS IN A SMALL FIBER PRODUCTION FACILITY

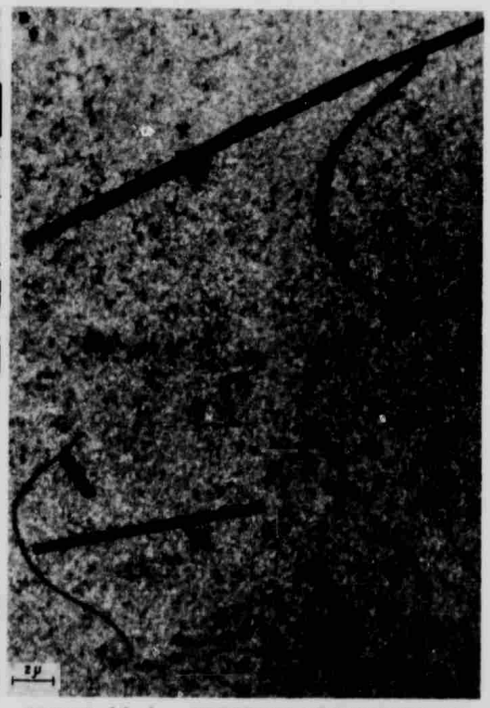


FIGURE 10-6. ELECTRON MICROGRAPH OF AIRBORNE FIBERS IN A SMALL FIBER PRODUCTION FACILITY

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The resulting length distributions are shown in Figure 10-4. As can be seen, 25 and 30% of the fibers were found to be less than 5.0 μ in length. A fiber length distribution of a typical amosite asbestos insulation operation is also shown for comparison. It can be seen that length distributions for the two fibrous glass operations approach that found for the asbestos operation. Figures 10-5 and 10-6 show typical electron micrographs of airborne fibers in these operations demonstrating fibers less than 1.0 μ in length.

Plastics Operation

In the one fibrous glass plastics operation which was surveyed, bath fixtures were made by a spray lay-up process using polyester resin and chopped fibrous glass. Potential for glass fiber exposure exists at the spraying operation and at the finish grinding operation.

Table 10-9 shows the mean fiber and total dust concentrations obtained.

Fiber concentrations even in the cutting and grinding operations were very low. Total dust concentrations, consisting of mainly polyester resin, were higher at the grinding operation.

TABLE 10-10

SUMMARY OF AIRBORNE FIBER DIAMETER AND LENGTH DISTRIBUTIONS IN A FIBROUS GLASS REINFORCED PLASTICS OPERATION

Upper Class Limit μ	Percent of Fibers < Upper Class Limit
Diameter	
1.0	<1
3.5	35
5.0	50
Length	
5.0	0
20	18
50	25

Median Diameter = 5.0 μ
Median Length = 35 μ

The airborne fiber d
10-10. The count median f
less than 5.0 μ in length

Summary and Conclusion

The data which ha
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make them potentially more

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1. Bien CT, Corn M:
dust. Am Ind Hyg Ass

2. Fraser DA: Absolu
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3. Johnson DL, Healey J.
Am Ind Hyg Assoc J 3

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The airborne fiber distributions for the operation are shown in Table 10-10. The count median fiber diameter was found to be 5.0 μ. No fibers less than 5.0 μ in length were observed.

Summary and Conclusion

The data which have been presented show that respirable fiber concentrations in the small fiber operations are many orders of magnitude higher than those found in standard insulation operations. In addition, the very small diameters and short lengths of these airborne fibers tend to make them potentially more respirable.

All worker population studies to date concerning the health aspects of glass fiber exposure have been done on worker populations exposed to low fiber concentrations in large fiber insulation operations. No worker studies have been done with populations exposed to small diameter fibers at concentrations found in this study. In fact, there does not presently exist such a population for study as exposure durations of these populations are extremely short.

Recent animal studies by Dr. Stanton of the Cancer Institute and Dr. Pott of the University of Dusseldorf have shown small glass fibers to be carcinogenic when injected into the pleural cavity of experimental animals. In view of these studies, it would appear prudent that exposures to respirable glass fibers be kept at an absolute minimum through the use of appropriate engineering controls and material handling methods.

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2. Fraser DA: Absolute method of sampling and measurement of airborne particulates. Arch Ind Hyg Occ Med 8:412, 1953

3. Johnson DL, Healey JJ, Ayer HE, Lynch JR: Exposure to fibrous glass. Am Ind Hyg Assoc J 30:545-50, 1969

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MR. LYNCH: At this time we will have questions for the speakers.

DR. GROSS: I have a comment to make on the paper presented by Mr. Ortiz, a question for Dr. Corn, and a comment on the statement made by Mr. Dement. Mr. Ortiz used the statement "potentially lung-damaging fibrous glass dust." That statement implies a conclusion for which I do not know a single datum of support. If he has such data, I would be very interested to hear it.

My question to Dr. Corn is that in doing fiber counts on human lungs in conjunction with the mineral dust content of the same lungs I also do not find any correlation whatsoever. I wonder if he would comment on the coincidence of his not finding correlation between mineral dust, total mass, and fiber count, and my not finding any correlation in human lungs.

Mr. Dement referred to the application of flow injected fiber glass dust, that is, the results obtained with pleurally injected fiber glass dust as compared with inhaled fiber glass dust. I again believe that this implies a conclusion which has not yet been proven; that is, that the results obtained by intrapleurally injected fiber glass dust are applicable to the results which are obtainable when the same glass dust is inhaled.

DR. CORN: The question you are asking is really a very fundamental one in the whole field of particle physics, which we have attacked on different occasions. We ask this same question in reference to air pollution in the following way: could you measure the weight of suspended particulate matter in the air of a city and reach conclusions about the number of particles and their size? Now we are asking: can you weigh the dust in a lung or in a plant, and can you reach conclusions about the nature and size of the particles? That's really the same question.

What it requires to get an affirmative answer is that there be a constant shape factor relationship between all particles you are examining. If the shape factor which relates one property of particles to another property of particles is not constant for the population, the answer to the question is inevitably no.

So I think what we are saying is that in different lungs with people exposed to different dusts you have different shape factors. In the atmosphere from day to day with variable winds from different directions. with different sources, we have variable shape factors from day to day. Therefore, the answers to these questions which would so simplify our problems of evaluation are no, it doesn't work.

MR. CARSTENS: I have something related to the last question that Dr. Corn just answered. In the area of cotton dust we depend strictly on personal reaction to a weight of sample collected by a specific method rather than based on the particle size. We do know particle size. I would suggest that this type of method be used in other areas such as fibrous dust. In that relation I would like to ask Dr. Corn how he collected his area samples and his personal samples.

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DR. CORN: Both the environmental and the personal samples were collected with personal sampling kits. They were either worn by the man or they were suspended in the area and immovable, at 2 liters/minute.

MR. CARSTENS: Were these the nylon cyclones?

DR. CORN: Some used nylon cyclones, some used the filter without a cyclone in the open face mode to get the total sample. We collected both with and without cyclone.

MR. CARSTENS: Have you run a comparison between that method and, say, the elutriator, which is used in cotton dust sample?

DR. CORN: No, we have not. One of the questions we are asking is, do these fibers behave the same in an elutriator as they do in a cyclone or an impactor? I think this is a very fundamental question about fibers and these are the kinds of sampling devices being compared.

MR. CARSTENS: From personal experience, I don't think they do. Mr. Ortiz, what liquid does he use to make up his aerosol? Is he using dust or a liquid produced aerosol?

MR. ORTIZ: To produce the aerosol you take bulk fibrous glass and grind it as an aqueous slurry. Then we dry it and generate it from a compact.

MR. CARSTENS: Then it is injected into the chamber as a dried material rather than aerosol?

MR. ORTIZ: A dry aerosol, yes.

MR. STEINFURTH: I think that if the doctors in this room would get involved with the public a little more, instead of all working on scientific methods, they would uncover situations that are quite unbelievable. We had a family who had a home built with baseboard radiant heat, so they installed a fiber glass duct system for air conditioning. The whole family became sick. They had to have their pet dog put away. It was full of cancer, full of fiber glass. They had to perform bronchial washings on the little boy. The husband, who worked at a government job, had to retire. The daughter and the wife were very, very ill. The doctor bills were extremely high. There were tests run on the people's home by several agencies, including Mount Sinai, NASA, and the duct work corporation that made the material and which has since sold the duct work operation. It was one bad mess.

I am not going to mention the people's name because there is going to be litigation involved. But I want you to know that in my files at the union there are many, many more like this that have no connection with our union whatsoever. They have no place to call, so they call me. Somehow, the word got around that I am interested in this type of problem because I represent our union members. I am also representing nonunion members. The way it is now I am representing maintenance people, and others that don't belong to our union. But somehow or other, eventually they get to me.

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In the testing that was done at this family's house, cracks were deliberately made in the masonry between the fireplace and ceiling in an attempt to blame it on the building insulation in the attic and ceiling. But in all cases, the duct work fibers that came out were in the small micron diameters that were traceable directly to the duct work system.

MR. LYNCH: I think that we would all like to see the data you have referred to in detail; the environmental and the medical data, together with the methods that were used, should appear in the open literature so that information can be applied in a scientific forum such as this to allow us to prevent occurrences of this kind.

MR. TILLENY: With respect to respirable sampling, I certainly agree with Dr. Corn that if one has to weigh a sample, one must know something about the percentage of background and fiber in order to make an estimate of the fiber content. But the use of a mechanical device to separate the respirable fraction from the nonrespirable fraction doesn't preclude the examination of the respirable fraction. One can still separate them, which I think is a very meaningful separation with respect to an inhalation hazard, and then analyze what penetrates in terms of what fraction is fiber and what fraction is background aerosol.

MR. BIERBAUM: Mr. Ortiz, you mentioned that in attempting to evaluate the cyclone for the separation of respirable fibers, you looked at four different diameter-size fibers, is that correct?

MR. ORTIZ: That's correct.

MR. BIERBAUM: What would happen if the cyclone were used in an environment where you are looking at a widely varying size distribution? What's going to happen to the workability of the cyclone in that situation? Can you even evaluate that? You do it on a single-sized fiber, but what happens in a multi-sized fiber situation?

MR. ORTIZ: I think what you are asking is whether we have done any field testing. The answer is no. But the test fibers we used have discrete size ranges of diameters. They are not just one diameter. For example, the Owens-Corning AAA-10. Owens-Corning assigns a nominal fiber diameter of .7 μ, but yet it does contain fibers extending from about .06 μ diameter all the way to 2 μ diameter. To further answer your question, yes, we do need to field test these samples.

MR. LYNCH: I have a question for Mr. Dement. You mentioned that you counted the fine fibers using 1,000X phase contrast and then these same samples were also examined by electron microscope. Were you able to obtain any data that compared the number of fibers found per cubic centimeter of air by the two methods, and did they agree?

MR. DEMENT: If I understand the question correctly, you are asking if we do comparison counts?

MR. LYNCH: Yes. In other words, did the electron microscope verify that the 1,000X phase contrast was finding all the fibers?

MR. DEMENT:
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DR. BALZER: O
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MR. DEMENT: We did not do comparison counts. We did size distribution in the hope of learning what our length of resolution was for optical microscope and to have an estimate of what percent of the fibers we were counting in that way. From our estimates, we feel that we can adequately count a 0.1 μ fiber.

You have to realize that in order to count something you don't have to resolve it in the conventional sense. You simply have to distinguish it for its background. Other investigators have found 0.1 μ fibers are easily counted with optical microscopy, similar to what we use. From the size distributions, we estimate that we are counting 90 to 95% of fibers that are there.

DR. BALZER: One of my concerns is that you are not identifying those fibers below 1 μ. Morphologically, you cannot distinguish them from other fibers. In the manufacturing of fiber glass it might be all right, but when you get into an occupational exposure and try to count those less than 1 μ, you are going to be including a lot of other fibers other than fibrous glass in the count.

MR. DEMENT: I agree. That's true of the present asbestos standard as well. In fact, I doubt that you could distinguish an asbestos from a fiber glass particle unless you went to a spectrographic method. Phase contrast is useless for that.

DR. GROSS: There is a question that I want to address to LeRoy Balzer. This has reference to the distinction between glass fibers and the nonglass fibers on the basis of the absence of a diffraction pattern. I don't believe filamentous diatom fragments have a diffraction pattern, and fipolites, that is, fibers derived from the skeleton of plants, could have a diameter of less than 2 μ in size and a length of around 10 μ.

DR. BALZER: I really can't comment since I didn't do the electron microscopy. I know that this is something we observed. In fact, we did some selective ashing of plant material you had sent to us. We looked at them under the microscope to try to differentiate. We felt that was one of the risks we had to take for those particular two items, especially in the occupational environment where silicate type insulating materials might also be applied at the same time.

DR. TIMBRELL: Coming to a previous item of discussion, the identification of glass fiber as against asbestos, for instance, there is one very simple way of doing it, and that is using the magnetic properties of asbestos. All types of asbestos, the amphiboles and chrysotile, align the magnetic field. All you have to do is put them in a liquid suspension, apply reasonable field, and they will act like compass needles. The glass fibers, of course, won't.

MR. LYNCH: I have a question for Mr. Dement. I think we are aware that, with asbestos, it is some years before we would anticipate seeing human cancer if any were occurring. Have the plants in which you were finding fibers that were in the respirable range been in business that long?

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MR. DEMENT: In the four plants that we surveyed making conventional insulation products, we did find fibers less than 1μ in diameter. The concentrations were obviously very low. In the six plants that we surveyed we found high concentrations but we couldn't find anybody that had more than 15 years experience. In fact, our medical team has done chest X-rays of pulmonary functions to create just a baseline for these people. We don't have a population sufficient for study yet.

DR. KONZEN: I want to clarify a point that John Dement made. The flame blown process or the flame attenuated process that produces the fine fiber was first used in large scale commercial production, in 1940, in the plant where the mortality study was done. So, there has been a period of time since 1940 in which people have been exposed to fine diameter fiber manufactured by the flame attenuated process.

MR. DEMENT: Dr. Konzen, what do you define as fine diameter? As I understand it, the flame attentuation process can be used to make fibers 7 μ and above. Just because the process is flame attenuation doesn't necessarily mean that a fine diameter fiber will be produced.

DR. KONZEN: I think that when you have flame attenuation you are going to have a range of fibers, as Mr. Ortiz has attested. I think I would like Mr. Smith to answer the question with regard to what would have been the reasonable nominal diameter of the flotation fiber which was produced during World War II.

MR. SMITH: There is no question that the flotation fiber we made in World War II was about 1 μ in average diameter. It had to be in order to fill the needs of that particular special application.

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Introduction

MR. LYNCH:
Medical Director of

Presentation

DR. KONZEN:
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RESULTS OF ENVIRONMENTAL AIR-SAMPLING STUDIES CONDUCTED IN OWENS-CORNING FIBERGLAS MANUFACTURING PLANTS

Jon L. Konzen

Introduction

MR. LYNCH: This paper will be given by Dr. Jon Konzen, who is the Medical Director of the Owens-Corning Fiberglas Corporation.

Presentation

DR. KONZEN: Some industrial hygiene sampling has been carried out for many years in Owens-Corning's manufacturing plants. In 1970, a reorganization of the industrial hygiene program resulted in a more comprehensive survey program. The first survey after 1970 at each plant location was designed to be a thorough industrial hygiene appraisal of the facility. It took approximately 18 months to complete a survey of all plant locations. Thereafter, annual audit surveys were carried out at each location until 1973. Since that time audit surveys have been carried out semiannually. Since 1970, as new plants were added to the corporation they received an initial or baseline survey shortly after they were placed on stream.

The airborne fibrous glass exposures quoted in this paper were obtained as part of these post-1970 industrial hygiene sampling surveys. We do not have a total airborne dust concentration and simultaneous fiber count for all samples. We do have sufficient frequency and number of samples to give us a reasonable characterization of fibrous glass exposure in the plants.

The sampling was carried out in the six plants which produce wool insulation products, in the five plants which produce continuous textile filaments and in the four noncorrosive products plants which produce fibrous glass reinforced plastic tanks and pipe.

Materials and Methods

Total dust samples were collected at a nominal flowrate of 2 liters/minute on a 37 mm fiber glass filter using an open-faced filter cassette.

Samples for fiber count and fiber sizing were collected on 37 mm membrane filters at a nominal flowrate of 2 liters/minute with an open-faced cassette. The fiber concentrations were determined by counting a minimum 100 fibers or 20 fields in a cleared segment of the filter sample. The fiber diameter was determined by sizing approximately 50 fibers into 11 size classes whose mean diameter ranged from 0.95 to 30.1 μ. Fiber as a percent of total suspended particulate was determined by counting about 500 particles. A particle was defined as a fiber if it had a minimum aspect ratio of 3:1 or greater. All microscopy was carried out by the phase contrast technique at 400X magnification.

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Both personal and breathing zone samples were collected but not necessarily simultaneously. The personal samples were taken with the sampler attached to the man's clothing in his breathing zone. Breathing zone samples were taken about 5 1/2 feet above the floor in areas where men worked.

Airborne Concentrations By Operations

The airborne concentration of fibrous glass will be reported by manufacturing and fabrication processes. The six "wool" plants produce thermal and acoustical insulation products by the centrifugal or rotary manufacturing process. The products range from lighter density home insulation through material compressed into heavy density board. It was noted early in our sampling program that airborne concentrations of fibrous glass were not markedly different regardless of the process used or product produced.

In the rotary or centrifugal process, molten glass flows by gravity from a conventional glass melting furnace into the rotary fiberizing unit. The fibrous glass exits at the bottom of the fiberizing unit. A water-base phenolformaldehyde resin is applied to the newly fiberized glass as it is directed down onto the continuous conveyor located on the bottom of the collecting or forming hood. Air is used to aid in directing the glass onto the conveyor belt. The fibrous glass then passes into the oven where it is compressed to the required density and the resin binder is cured. The wool pack emerges from the oven in a continuous ribbon. As the ribbon travels down the line it is cooled, trimmed to the required dimensions, a backing of kraft paper or foil is applied to some products and it is removed manually at the end of the line and packed for shipment. Removing the product from the line is the first operation where men handle the fibrous glass. Some products, usually the heavier density board type of product, may undergo further fabrication operations such as film facing or painting operations prior to packing.

The reconditioning operation utilizes trimmings and offware which is nodulated and pneumatically conveyed and bagged for use as pouring and blowing wool.

Table 11-1 indicates the average total dust concentration, the percent of fiber as a fraction of the total suspended particulate and the percent of total fibers sized less than 3.5 μ in diameter in the wool forming, packing, fabrication and reconditioning areas of the six wool plants.

The bonded mat operation has been further identified since it is carried out in wool plants but utilizes a modified textile process. Multiple streams of glass flow out of a textile type bushing and are attenuated by air or steam and collected on a chain conveyor.

As the newly formed mat is conveyed down the line, binder is applied by a dip trough and it passes into an oven where it is dried and cured. As the mat emerges from the oven it is automatically trimmed and rolled for shipment.

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Wool Forming

Wool Packing
& Fabrication

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TABLE 11-1

AIRBORNE FIBROUS GLASS FOUND IN SELECTED AREAS OF SIX WOOL PLANTS

	Total Dust Concentration mg/cu m		Fiber Concentration No. Fibers/ml		% Fiber/ Total Particulate		% Fibers <3.5μ Diameter	
Wool Forming	1.66	(59)	.15	(63)	.34	(67)	87.1	(52)
Wool Packing & Fabrication	2.02	(259)	.16	(246)	1.58	(257)	73.9	(206)
Reconditioning	1.09	(37)	.11	(37)	1.14	(38)	68.9	(30)

( ) denotes number of samples.

Table 11-2 characterizes the airborne fibrous glass found in the bonded mat operation.

TABLE 11-2

AIRBORNE FIBROUS GLASS FOUND IN BONDED MAT OPERATIONS

	Total Dust Concentration mg/cu m		Fiber Concentration No. Fibers/ml		% Fiber/ Total Particulate		% Fibers <3.5μ Diameter	
Bonded Mat	1.12	(13)	.15	(18)	.22	(18)	86.6	(13)

( ) denotes number of samples.

TABLE 11-3

AIRBORNE FIBROUS GLASS FOUND IN
FLAME ATTENUATED MANUFACTURING PROCESS

	Total Dust Concentration mg/cu m	Fiber Concentration No. Fibers/ml	% Fiber Total Particulate	% Fiber <3.5 Diameter
Flame Attenuated	1.33 (35)	.38 (8)	.40 (8)	89.3 (5)

( ) denotes number of samples.

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The flame attenuated process is also located in wool plants. In this process, fibers of pencil lead diameter size flow continuously from a textile type bushing and are immediately further attenuated by a horizontal blowing burner. The fibrous glass is sprayed with the phenolformaldehyde binder, gathered in the collecting chamber, and deposited on a conveyor which moves the material into the oven. The remainder of the flame attenuation process is similar to that for other wool products. Some fibrous glass matezzals, which are 6produced by the flame attenuated process, have an average diameter of 1 to 4 μ with others ranging up to 6 to 9 μ, in diameter. The flame attenuated products presently account for less than 1% of the glass fiber market. Table 11-3 characterizes the airborne fibrous glass found in the flame attenuated manufacturing operation.

Textile type products, in contrast to wool, are produced by an entirely different process. Continuous filament textile fibrous glass is produced by melting glass marbles in a small electric furnace located above each forming position. Another method of producing textile glass consists of conveying the molten glass from a large central furnace to each forming position. Molten glass streams flowing from bushings are mechanically pulled into continuous filaments which are drawn over a sizing applicator. The filaments are collected into strands and the strands are wound onto rotating forming tubes.

The strands from the forming tubes are twisted and plyed on standard textile twist frames to produce yarn. Rovings and mat which are not twisted and plyed are also fabricated for reinforcement applications. Table 11-4 characterizes the in-plant environment in the textile forming and textile yarn fabrication areas.

TABLE 11-4

AIRBORNE FIBROUS GLASS IN TEXTILE FORMING AND TEXTILE YARN FABRICATION AREAS

	Total Dust Concentration mg/cu m	Fiber Concentration No. Fibers/ml	% Fiber/ Total Particulate	% Fibers <3.5μ Diameter
Textile Forming	2.99 (18)	.13 (17)	.20 (6)	98.0 (2)
Textile Yarn Fabrication	1.19 (228)	.37 (205)	1.50 (190)	76.9 (161)

( ) denotes number of samples.

Staple fibrous glass is manufactured in small volume at only one textile plant by a modified textile process. The molten glass flowing from the bushing is attenuated by a stream of air, sprayed with a lubricant and either collected on a small revolving drum and gathered on a spool for later twisting into yarn or collected directly into a bin for bulk shipment.

Table 11-5 cha
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AIRBORNE FIBROUS GLASS

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AIRBORNE FIBR

	Total Dus Concentrati mg/cu m
NCP	3.49 (43)

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SUMMARY

1. The data c
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Table 11-5 characterizes the airborne fibrous glass found in the staple forming and staple yarn fabrication areas.

TABLE 11-5

AIRBORNE FIBROUS GLASS IN STAPLE FORMING AND STAPLE YARN FABRICATION AREAS

	Total Dust Concentration mg/cu m	Fiber Concentration No. Fibers/ml	% Fiber/ Total Particulate	% Fibers <3.5μ Diameter
Staple Forming	5.49 (10)	.07 (10)	.35 (2)	N/A
Staple Fabrication	2.25 (7)	.11 (7)	.20 (1)	N/A

( ) denotes number of samples.

In the noncorrosive product plants, fibrous glass reinforced plastic items such as storage tanks and pipe are produced. These are fabricated by spray-up, filament winding and also by hand lay-up methods. The hand lay-up methods are primarily used in assembly operations. The reinforced plastic items are then drilled, ground or sanded as necessary to install needed fittings and connections.

Table 11-6 characterizes airborne fibrous glass exposure in non-corrosive product plants.

TABLE 11-6

AIRBORNE FIBROUS GLASS IN NON-CORROSIVE PRODUCT PLANTS

	Total Dust Concentrations mg/cu m	Fiber Concentration No. Fibers/ml	% Fiber/ Total Particulate	% Fibers <3.5μ Diameter
NCP	3.49 (43)	.12 (38)	.25 (40)	72.3 (33)

( ) denotes number of samples.

SUMMARY

1. The data characterizing airborne exposure to fibrous glass in Owens-Corning plants were collected as part of our routine industrial hygiene sampling program.

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2. The airborne concentrations of fibrous glass average less than 0.4 fibers/ml.

3. Less than 2% of the total airborne particulate is fibrous.

4. Approximately 80% of the fibrous airborne material is below 3.5 μ in diameter.

5. The airborne concentration of respirable fibers in manufacturing operations is remarkably consistent, without apparent influence from geographical location of the plant, time of year, climatic conditions, method of manufacture or nominal diameter of the parent material.

6. There is no apparent relationship between total dust concentration and fiber concentration.

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MR. LYNCH: We shall now entertain questions for discussion, not only of the papers presented this afternoon, but also on anything presented throughout the entire day.

MR. PARRILLO: Mr. Dement, in sampling, you discussed two filters, one obviously open-faced, for your counting. For the total dust are you using open-faced or closed-faced filters, or does it matter? Would you expect a difference if it doesn't matter?

MR. DEMENT: For the total dust concentrations we used the closed-faced filter with only the small plug. The Millipore cassette was used with the small plug removed, the idea being that if you want to collect a representative sample of total airborne dust you want high velocity.

MR. CARSTENS: This question is for Dr. Konzen. In your textile glass, isn't the fiber coated just immediately after coming through the spinnerette or shortly thereafter?

DR. KONZEN: In textile glass the material is pulled out of a precious metal dye and it passes immediately over a sizing roller. The primary reason for this sizing is to make it compatible with further fabrication operations such as reinforcement applications.

MR. CARSTENS: Therefore, at no time do any of the workers come in contact with particles which are virgin glass, uncoated. Is that correct?

DR. KONZEN: I think that is a reasonable statement.

DR. ARMITAGE: Dr. Konzen, in talking about the textile glass, you were referring to the continuous rolling process. Much chop roving is actually used in thermal plastic reinforcement. Was air sampling done in the neighborhood of the chopping operation?

DR. KONZEN: Yes, it was, and this was included in the samples that were reported on noncorrosive products. Again, we don't show a large increase in airborne fibers. I think our counts were quite compatible with those found by Mr. Dement when he sampled spray-up type of operations which incorporate chop roving.

MR. LYNCH: One of the points that might be causing some confusion is the criteria we are using for the definition of the upper limit of respirability for fibers. We have been talking about some μ fibers that are less than 1 μ in diameter. Dr. Konzen mentioned a 3.5 μ size. Given Dr. Timbrell's independence of length, and given his relationship of 3 to 1 -- 3 times fiber diameter -- and considering the density of glass, what would we set then as an upper limit for a respirable fiber? Would somebody care to respond to that?

UNIDENTIFIED PARTICIPANT: Is it possible that this value would vary with the age of the individual, a child versus an adult?

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MR. LYNCH: I think it would vary with a lot of things. What I am seeking is that we use either a 7 μ or 10 μ unit density sphere, depending upon whether we use the Los Alamos or the Johannesburg criteria for spheres. Now, given the information that we have available, would we say it to be 3 μ, or 3.5 μ?

DR. GROSS: I don't believe that you can set a 100% limit of respirability. It is possible to find particles in human lungs which are well over 100 μ in diameter. These are nonfibrous particles.

I am reminded of an event that took place around 1865. Rudolph Burkholt at that time represented one school of thought which held that the inhaled black dust in the lung was of blood origin; the French school of thought, held also by the English, maintained that the black dust in the lung was inhaled. It wasn't until Dr. Burkholt in Berlin was sent a section of lung which contained a large particle of charcoal, identifiable as charcoal from the wood cells in the charcoal, that Burkholt admitted that the black dust in the lung was truly inhaled.

What I am pointing out is that occasionally you do find very large particles in the lung tissue. There is no 100% cut-off on respirability.

DR. TIMBRELL: The question was asked whether this limit would depend on the age of the individual. I remember Dr. Gross presenting some figures at the last inhaled particles conference in London where he showed that a child had fibers in the lung which were thinner than those in the adult lungs. I don't think it is really necessary to be specific about this limit. I think we only want an approximate value. After all, we are satisfied with the limit which is enforced. We say for instrumental purposes we will accept the largest particle of unit density as being so many microns. This has the advantage of not only being accurately comparable with human lungs, but it enables us to make instruments which are accurately reproducible.

I think this is the important point. It would be valueless to have instruments which vary from one to the other and give different values for the respirable dust. I think it would be satisfactory to take as the upper limit 3 μ or 3.5 μ.

Also, I think we have to remember that the density of the glass can vary. The glass that we use to derive this figure was a particularly low density glass, about 2.25. Glass usually has a specific gravity of 2.5.

The point I want to make is that there is no real need to be more specific than that.

MR. DEMENT: I have a question for Dr. Konzen. I noticed in your textile operations, the size distributions showed many fibers smaller than 3.5 μ. I think you showed something like 98%. What is the lower limit of that size distribution?

DR. KONZEN: You have to take into consideration the optical microscopy system that we are using. The lowest mean diameter of the cell that was used was about 0.9, and I think the lower range on that was around

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0.8. Anything below that we would not be seeing. It would be very unlikely that we would see many fibers below this, since the process by which they are manufactured is a dye-pulling process.

MR. DEMENT: Was this a so-called beta fiber?

DR: KONZEN: These could include beta, although the beta numbers would be small in comparison to other forming operations, since beta is a very low volume product.

DR. SCHEEL: I have two questions, one for Dr. Corn and one for Dr. Konzen. I will ask Dr. Corn first. Have you looked at the total composition of the dust, not just the fibers? How much organic are we talking about in this 2% fibers and 98% others?

DR. CORN: I can't comment. We did not do that analysis.

DR. SCHEEL: One of the things I should like to emphasize again is that we don't lose sight of the total picture when we talk about the biologic effects of dust in this particular situation. We are talking about coating fibers with plastic materials, and these are organic in nature. When the particles and fibers are coated, the solubility of the fiber changes greatly, and the biologic effect also changes. I think we should pay some attention to this.

I was amazed by the data this afternoon about the low concentration of fibers. I think it would be worthwhile to talk about process changes in the industry over the last 10 years which have made this condition as it is now, as opposed to the condition that existed before.

DR. KONZEN: In the textile operations this process has not changed appreciably since it was put into commercial production in the late thirties or early forties. The flame attentuated process, at least in our operations, has not changed appreciably since the early forties. The process by which insulation wool was manufactured, the so-called steam blown insulation, began to be supplanted by the rotary process as early as 1954 when the first machine was installed in Newark, Ohio. Subsequent installations took place over a period of years.

From my analysis of the situation, there has always been a small background of fine diameter fibrous glass in our fibrous glass plants. I have no idea what the concentration was years ago so I can only speak from the time that I began to look into this situation in a systematic manner, in about 1970.

MR. LYNCH: One thing that has occurred to me is that there may be a contradiction between the conclusion arrived at by Ettinger and presented by Mr. Ortiz, and some earlier work done by Bien and Corn on the usefulness of the cyclone as a respirable mass sampler for fiber glass. Accepting the fact that we must look to the question of biological effect to decide what we want to measure and it turns out that we want to measure respirable mass, is the cyclone a suitable device? Dr. Corn, would you care to comment on that?

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DR. CORN: I wasn't sure you wanted to get into that type of a discussion here. These findings by Ortiz and Ettinger have been known to us for some time. A member of our student body is pursuing a thesis in which he is looking into why their results, where the standard of comparison was clearly stated by Mr. Ortiz to be the impactor, yield a different conclusion from the standard of comparison. In our study, this was the settling in liquid media. The conclusion is that cyclones aren't good for fibers. We are now looking at this by using air elutriation rather than liquid elutriation as the standard for performance of the cyclone to see if we repeat our former finding; or if indeed it leads more to what the impactor would yield.

The problem is, what is your standard of reference? The Los Alamos standard of reference is the impactor and our standard of reference is an elutriation device. I think therein lies the difficulty. We are not quite sure what is happening.

I'd like to comment on a point that has not yet been mentioned. We have certain repeat samples. I think everyone should appreciate that this is a highly fluctuating environment; from day to day we get a 2 to 1 variation on the same man. I do not want to give the impression that this is the range for a different man. On any given day if you went back, you would get the same results with the same man. We are talking about a typical air sampling situation.

MR. CAPLAN: I wonder if anyone can make any comments about the effectiveness of the relative collection efficiency by open-faced versus closed-face sampling and also tell us which is the recommended method.

DR. CORN: There is a gentleman in the audience who has been working on this problem for a doctoral thesis for the better part of two years. Specifically, Mr. Hammad has written a thesis on errors in sampling due to changes in entries of samplers. Mr. Hammad, if you would like to respond to this I would be pleased to turn it over to you. Do you think that with the sizes of particles we are sampling, there is a bias due to open face or closed face mode?

MR. HAMMAD: I don't think today we can say there is a large effect in the range, that in this particular case it would make any difference. But I think there would be some difference if we were looking at samples for total dust. If we were looking for samples for respirable dust, it wouldn't really make any difference. If we were going to have samples for total dust, then we should look at small entries and effects because of the loss due to inertia.

I think there is some effect, and it might be also about 20% losses due to inertia, using a very small amount.

DR. CORN: Outside of the respirable range when it gets big?

MR. HAMMAD: Yes, about 30 μ.

MR. CAPLAN: I wanted to clarify that. Does that mean, whether you are looking for total dust or respirable dust, one should use the small

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nozzle with the cover on it? As a general rule then, would you say that a good way to sample for both the respirable and the total dust, would be to leave the cover on?

MR. HAMMAD: No, I think you should take the cover off.

MR. CAPLAN: Take it off?

MR. HAMMAD: Yes. If you have a very small nozzle, there will be lots of effects due to inertia. If you use the face velocity very low with an open face, you have losses due to sedimentation, and you would have losses on both sides. What we really have to do is to reach a happy medium, to get fast velocity hard and the sampling entries as big as possible. Now if you have an open face mode with very low face velocity you would have losses due to sedimentation. If you had a sampling nozzle with very high face velocity and small entries, you would have losses due to inertia.

DR. CORN: Mr. Caplan, if you recall, we examined this because of a theory in the literature by Davis. There was nothing on the market that adhered to the guidelines for proper sampling. I think the conclusion of Yehia's work is that in the respirable range it has no appreciable effect. You could sample either way. When you get up into large particles, sampling pollen, sampling things totally soluble in the upper respiratory tract, he suggests using a large entry to minimize the inertia trying to get in that small entry hole. For most of the work we do, it turns out we get very excited over what, in experimentation, turns out to be insignificant.

DR. SCHEEL: I want to ask one question about this work. When we compare sampling particles such as pollen or organic materials and we use silica standards which have a density of 2.5, are we talking about the same ball park? This to me, especially in this fiber glass area, is a real problem in terms of sampling operations.

DR. CORN: We talk only about equivalent diameters. I acknowledge my debt to Vernon Timbrell. In my classes, I show some of his work; I show the fiber, the bulky particle, and ask the students what the size of the particle is. Next to it is that little sphere, and that is the size of the particle. Always reduce to the aerodynamic equivalent diameter if you work with pollen or other materials and then you are on the same baseline. I think that contribution was a very major one.

MR. CAPLAN: We have been talking about the larger particles and perhaps the micron size particles. I wonder if anyone has done any checking on the penetration of these very small fibers through the Millipore filter, particularly at high velocity, which you would be doing with the cover on. In other words, for the respirable size you are closing it off, so you have a high velocity. Apparently most of the dust will be collected right at the center spot of the filter, obviously going through at a high velocity. Has anybody studied the penetration through that filter of fibers that are submicron--in the range of 0.1 or 0.2 μ? There was some work done several years ago at Schenectady indicating that

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submicron particles, if the velocity is high enough, will actually penetrate the filters. Has anyone done any studies on that?

MR. LYNCH: The study in Schenectady--was that on the membrane filters or Millipore?

MR. CAPLAN: On the membrane filter and the AA filter, and I think it was with dioctyl phthalate. I believe that at a high velocity it was something like 30 or 40 cm/second going through the filters, with pentration as much as 80%. These were the dioctyl phthalate spheres.

MR. LYNCH: In the case of the asbestos samples, we know, of course, that they do not penetrate the filter. We have studies to that effect by backup filtering.

Was there another comment on this question?

DR. LIPPMAN: I believe Paul Caplan is referring to the Fitzgerald and Detwiler report which nobody else was ever able to reproduce, including Fitzgerald and Detwiler. It was an anomaly that couldn't be explained. There is very little evidence of penetration for small particles through membrane filters, except for the 5 μ pore filters where the possibility exists, and I don't know the data on that filter. But for AA and Millipore, it would be very difficult to conceive of any particles getting through in a large percentage, no matter what their size.

DR. GROSS: I should like to clarify something for Dr. Scheel regarding organic material, and particularly the binder associated with fiber glass. We have investigated the biologic effect of various binders, and find it has no measurable biologic effect.

UNIDENTIFIED PARTICIPANT: Has anybody looked at the ends of these fibers? What do they look like? Are they sharp, do they vary with different processes, and would this have any effect on end direction in the lung?

MR. LYNCH: Perhaps Dr. Balzer would like to comment on that as well as someone on the panel.

DR. BALZER: In our work with the electron microscope at 20,000X magnification, most of the fibers are fairly square on the end. They are not spheres. Some of the mineral wool tends to have a shattering effect, but most of the things we see are fairly square on the end. The fibers may be slightly rounded or chipped, but nothing like a sphere or point.

I want to reply to Paul Caplan on a point. The document that Mario Cooper and Del Leon produced in the technical archives has in it some breakthrough experiments for various size filters for asbestos. It confirms that at 5 μ, you do have some breakthrough, but nominal breakthrough.

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DR. TIMBRELL: There seems to be some difficulty in characterizing performance of cyclones. I think it might be useful to mention what we discovered about the MRE sampler. We looked at the elutriated characteristics of the MRE sampler, narrow spectrum at the inlet and narrow spectrum at the outlet just before the filter. This gave us the aerodynamic size distributions of the test cloud and the diluted cloud. We did this for various types of asbestos spheres, and we also did it with fiber glass. What we found was independent of the shapes of any of these fibers. The MRE characteristic was very similar to what you get with perfect spheres. We did this with respirable particles, using the USICC samples. This is a particularly useful characteristic because it means that length up to 200 μ has no influence on the elutriated characteristic. It only depends on the aerodyamic size of the particles. I think this could be a very useful characteristic for using the MRE sampler as a standard against which you will compare the cyclone.

MR. LYNCH: You did this with your aerosol spectrometer?

DR. TIMBRELL: Yes. I assure you that the curve is very close to the designed curve, and there is an accurate cutoff at about 7 μ. This doesn't mean that the MRE sampler is an ideal sampler for estimating what fiber glass would get into the lung because, of course, the lung would take account of not only the aerodynamic size of the glass fiber, but also of the length. It is useful that the MRE sampler characteristic is well defined. It depends purely on the aerodynamic size of the fibers.

DR. FRASER: In the textile forming operation, is it true essentially that 98% or almost 100% of the fibers are less than 3.5 μ? I have difficulty in visualizing the forming operation as being a source of fibers. I wonder if any of the industry people would care to comment on the various operations as a source of fibers, because there are rather great differences in these operations. In some cases it is easy to see where they might produce fibers, while in others it's difficult to see where the fibers might be coming from.

DR. KONZEN: This is not an area in which we would expect a high fiber fly, for the simple reason that the vast majority of fibers made by textile operations are very uniform because they are dye-drawn.

MR. CARSON: I am wondering if anyone on the panel or in the audience knows how much of the material in what we are calling total dust samples is actually glass and/or how much is something else? Granted, we have to look at how much of it might be fibers by our assumed 3:1 aspect ratio. I am wondering how much of it is glass. If there are glass particles present, it may be a different kind of biological hazard.

DR. CORN: Our samples are virtually all glass dust, except for whatever binder is associated. That is what you are dealing with. There is very little dirt that we can see. Morphologically, it is glass. We did

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not do what Dr. Scheel suggested, which was to get rid of the binder and check the difference in weight; or get rid of the organic and see what was there. However, I think you would find that there is a very small percentage of the sample.

MR. CARSON: How much bearing do you think this should have on whether we go to a count of total fibers or to either a total mass or respirable mass of fibers?

MR. LYNCH: I think to a great extent we have been hearing data which was collected in fiber glass manufacturing establishments where you find glass in the air. Any standard we have set or any sampling methods upon which a standard is based, would be used in addition for fiber imports plastic operation, where a great deal of the respirable dust would be other than glass.

DR. POSNER: What is the chemistry of the glass that is used for making fibers? Does it vary with the textile and the wool fibers?

DR. KONZEN: This is an area in which I have no very little expertise. The textile glass and the wool glass do vary in some respects in their chemistry. But basically they are all glass products or made from silica and the other materials that were mentioned this morning.

To keep things in perspective, the amount of fibrous glass, as opposed to nonfibrous particulate, in the plant should be noted. In approximately 1,500 samples, we have found that over 98% of the suspended particulate is nonfibrous. This is in a manufacturing plant. If you go out on a job site, a variety of other materials would be found.

MR. DEMENT: Didn't you base your 98% on particle count and not particle weight?

DR. KONZEN: That's right.

MR. DEMENT: In our samples we have often noted very fine bits of particulate matter, most of it being binding material, which on a particle count basis would be fairly high, but on the mass basis would be relatively low.

In a number of our microfiber operations there weren't any binders added to the products. There we saw fiber concentrations averaging approximately 15 in one operation with dust concentrations of about a milligram per cubic meter. If you subtract what might be air pollution background, a tenth of a micron would give you some idea of what the actual glass content was.

DR. KONZEN: Again, these are satisfactory results.

MR. MAITRA: I
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MR LYNCH: Dr
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MR. MAITRA: Dr. Gross mentioned something about studies with binder. I believe we who are manufacturers, as well as the end users, should associate the fibrous problem along with the binder. I wonder if Dr. Gross would discuss in some detail the type of study he did.

MR LYNCH: Dr. Gross has offered to do that tomorrow in connection with our discussions at that time of the biological effects of the materials.

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SESSION III

BIOLOGIC EFFECTS

Chairman: A. Walter Hoover

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EFFECTS OF INHALED FIBROUS GLASS IN THE LOWER RESPIRATORY TRACT OF GUINEA-PIGS

Susan Botham

Introduction

DR. HOOVER: The first paper this morning will be given by Dr. Susan K. Botham from Sussex, England. Dr. Botham formerly was affiliated with Dr. P. F. Holt at the University of Reading where they performed animal experiments involving fibrous glass.

Presentation

DR. BOTHAM: Reichmann [1949] pointed out similarities in X-ray pictures between changes resulting from inhalation of glass wool and asbestos, although Gardner had suggested earlier [1940] that glass wool was not respirable.

In 1951, Vorwald et al reported that glass wool administered to guinea pigs by intratracheal injection had not resulted in fibrosis within one year, but they described fibers that after a year in the lungs had become coated with a smooth layer of Perls-positive material as occurs with asbestos.

Now that glass fiber of a diameter comparable to asbestos is available, a comparison was made at the University of Reading of the rate of this coating reaction of the lung to different kinds of inhaled asbestos and glass fiber. As a further comparison, some of the glass fiber material was fused and then finely powdered before administration to guinea pigs by inhalation. [Botham and Holt, 1968, 1971a, and 1973]

Materials and Methods

The guinea pigs were exposed to a dust-laden atmosphere in the apparatus that has been fully described by Holt et al. [1964] It consists of a Micro Hammer Mill from which asbestos or glass fibers are ejected into a circulating jet of air. An extractor fan draws this air stream through an elutriator into a chamber in which the experimental animals are housed. The dust concentration is regulated by alteration of the volume of air entering the elutriator.

Finely powdered glass dust was generated by the mutual grinding action of fused glass spheres in a rotating rubber-lined tumbler box. This dust was similarly drawn through the elutriator into the dusting tunnel housing guinea pigs.

Groups of guinea pigs were exposed to one of these dust-laden atmospheres for 24 hours and were killed at intervals after dust inhalation. The 18 animals that inhaled glass fibers were killed during the succeeding 18 months, but the first 9 of these were sacrificed during the first month after dusting, while the 10 animals that inhaled glass powder were all killed within a month for comparison of early stages of the

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lung reaction. The lungs were fixed in buffered neutral 4% formol saline and paraffin-wax sections were prepared and stained with Perls' reagent alone or with Perls' and Harris's haematoxylin and eosin. Photomicrographs of the sections were taken with a Leitz Ortholux instrument. These lungs were compared with those of guinea pigs killed at similar times after inhalation of one of four types of asbestos. [Botham and Holt, 1971b]

Results

After milling, the glass fibers ranged up to 0.5 mm in length with a maximum diameter of 3.0 μ, but such large fibers were rare, most were of submicron diameter.

The particles of glass powder had a maximum diameter below 10 μ. Neither form of glass gave a positive reaction with Perls' reagent.

Clearance of Extracellular Fibers

Many glass fibers were deposited in bronchioles; very few reached the alveoli initially. In the animals killed at the end of the dust-inhalation period and a day later, many extracellular fibers were found in all generations of bronchioles. They were generally shorter than 20 μ although examples up to 40 μ were recorded. Extracellular fibers became progressively more scarce in animals killed in the following three weeks and were not found a month or longer after dust inhalation.

Clearance of Intracellular Fibers

Detached alveolar macrophages containing one or several glass fibers appeared in the larger bronchioles in the first week after dusting, but were mostly cleared during the following 3 weeks. Very occasionally, these intracellular fibers were seen in bronchioles of animals killed up to 14 months after dusting. By 18 months, fibers in free macrophages had all been coated in Perls-positive material.

During the first week after dust inhalation, some small giant cells containing fibers were also found in bronchiolar debris together with some desquamated epithelial cells and red blood cells. In older animals, most bronchioles were open but some debris consisting of macrophages and giant cells occurred, together with eosinophils at over 8 months after dust inhalation.

Penetration of Fibers Into Alveoli

Extracellular glass fibers were not found in the alveoli of any guinea pigs, but fibers partly ingested by alveolar macrophages were found in some alveoli of the animals killed at the end of the dust-inhalation period. They were not seen in subpleural alveoli until 2 days later than this. In guinea pigs killed later than a week after dust inhalation, some

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macrophages containing fibers had foamy cytoplasm and had occasionally formed giant cells.

In animals killed during the first week, many macrophages containing fibers became detached from the interalveolar septa and were common in alveolar ducts and spaces. By a month after dust inhalation, there were fewer such cells, but their cytoplasm now was often foamy and vacuolated. In the nine animals killed during the last 17 months, macrophages containing fine glass fibers occasionally were seen detached in alveoli.

Red blood cells were seen in alveoli throughout the lungs of guinea pigs killed during the first week after dust inhalation, but only occasionally in older animals.

There was no particular concentration of long fibers in alveoli near respiratory bronchioles. Although in the nine older guinea pigs thickening of interalveolar septa was general, even in the final animal this thickening rarely led to complete obliteration of the alveolar spaces.

Reaction to Staining with Perls' Reagent

In the first guinea pig, the cytoplasm of some macrophages gave a diffuse reaction, while granules were more deeply stained. Sometimes these cells contained partly ingested fibers. In the second animal, some Perls-positive material stained in the form of a very thin coating around some fibers in macrophages in the interalveolar septa. In the next four animals increasing numbers of these coated intracellular fibers were found. Sometimes the coating was incomplete, with short parts of the fiber being unaffected, although completely intracellular. By the fifth day after dusting began, the coating on some fibers was uneven, appearing banded. The coating on some fibers retained in the lung for 2 weeks developed a finely beaded outline.

After a month, many fibers throughout the lungs were enclosed in coatings that were either smooth or corrugated in outline; these structures are glass fiber bodies. Their further development was traced in older animals through a stage where corrugations of the coating extended to the fibers resulting in a beaded appearance. Next the exposed fiber apparently fragments. In the older animals, many macrophages containing large Perls-positive granules were seen. These were possibly the remnants of fragmented glass fiber bodies. As it was not always possible to detect a fibrous core, this could not be verified with the light microscope. However, an electron microscope examination, made by Dr. J.M.G. Davis, of the lungs of the final animal revealed only a few uncoated particles, mostly shorter than 1 μ.

Only one glass fiber body was formed in most macrophages or giant cells, other fibers generally remaining uncoated. These fibers usually were shorter than the coated fibers. The longest glass fiber body found was 80 μ, but this was exceptional, the majority measuring under 30 μ. Most were straight, although some slightly curved forms were found.

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No association of collagen fibrils with fragmenting glass fiber bodies was found in sections treated with Perls' and Van Gieson's reagents, and there was little fibrosis of the lungs as indicated by collagen increase. The only instance of a glass fiber in lymphoid tissue occurred in the animal killed 14 days after dust inhalation.

By examining 100 glass fiber bodies selected at random from Perls' stained sections from the last 10 animals, it was possible to classify these bodies into five forms representing progressive developmental stages, as had previously been shown with the four types of asbestos. If the five forms were randomly distributed, they would have had an equal chance of occurring in each animal, the results, however, did not show this:

TABLE 12-1

DIFFERENTIAL COUNT OF GLASS FIBER BODIES: 100 GLASS FIBER BODIES CLASSIFIED FROM EACH HISTOLOGICAL SECTION

Time (in months) after dusting began	Percentage of each form of glass fiber body*					$\sum \frac{(O-E)^2}{E}$
	1	2	3	4	5	
1	54	28	15	2	1	96.5
2	55	29	12	4	0	101.3
3	55	19	17	4	5	85.8
6	18	34	21	24	3	25.0
8	26	33	20	17	4	23.5
10	33	26	24	11	6	24.6
12	23	21	23	20	13	3.4
14	7	19	12	23	39	30.2
18	4	9	10	25	52	76.3

*Form 1, smooth coat: 2, corrugated: 3, partly beaded: 4, completely beaded: 5, fragmented

An examination of the Chi squared values for each animal showed that at 12 months only, after glass fiber inhalation, each form of glass fiber body has an equal chance of occurring in any animal. This implies that some forms predominate; it can be seen from the table that in younger animals the smooth-coated forms are more numerous, while in the older, fragmenting forms are more common, thus confirming the developmental sequence of a glass fiber body. Coating of all potential glass fiber

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Inhalation of Powdered

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bodies did not begin immediately after fiber inhalation, many examples of early stages in their formation being found even after fibers had remained a year in the lungs.

Inhalation of Powdered Glass

The clearance of inhaled powdered glass particles followed a similar pattern; first extracellular and then intracellular particles appeared in bronchiolar debris. The particles were found in free macrophages but very few giant cells were formed. Some red blood cells were also seen in this debris.

Isolated small, extracellular glass particles were found in the alveoli even in the animal killed 1 month after exposure to dust. Macrophages containing fine glass particles were found in the interalveolar septa of all animals. Intracellular Perls-positive material appeared as fine granules in macrophages in interalveolar septa of all animals. Initially, few cells were found to contain both glass particles and Perls-positive material but, by 1 week, few macrophages containing glass did not also contain Perls-positive cytoplasm or granules. After this time, the Perls-positive granules were almost exclusively found in cells also containing glass in the final three animals. Such cells often had been shed and were seen in alveolar and bronchiolar debris in animals killed within a week after dust inhalation.

Even a month after inhalation of powdered glass, there was no accumulation and deposition of a Perls-positive material around intracellular glass particles.

Discussion

The initial deposition of inhaled glass fibers occurred in the bronchioles. While some fibers were removed by ciliary action, others were ingested before removal by phagocytes, as occurred following inhalation of asbestos fibers of a similar size. Other fibers reached the alveoli and were ingested by phagocytes that were retained in the septa or shed. In either case, some giant cells resulted from their fusion and the interalveolar septa became thicker.

In guinea pigs, the fate of inhaled and retained asbestos and glass fibers has been found to follow a similar sequence resulting in morphologically indistinguishable structures composed of a ferro-protein coating around an ingested fiber. If a comparison between the Table and similar data for the four types of asbestos is made, the development of a glass fiber body is slower than that seen with chrysotile but more rapid than other types of asbestos. The time elapsing before an asbestos or glass fiber body fragmented is related to the chemical composition of the enclosed fiber. The remarkable feature is that glass fiber fragments at all, since the glass is a fused silicate unlike the layered structure of asbestos, from which it is thought that cations may be leached to leave a fragile silica lattice.

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Inhaled particles of glass powder also penetrated to the alveoli and were ingested by phagocytes. These cells often contained Perls-positive material but it was not deposited around the powdered glass particles within the same period as occurred following inhalation of glass fibers of the same chemical composition.

Inhalation of asbestos and glass resulted in a diapedesis of red blood cells into alveoli and a subsequent appearance of Perls-positive material in alveolar phagocytes. The amount of this material was greater following asbestos inhalation than glass fiber, which in turn was greater than that following glass powder inhalation.

From a comparison of the effects of these three dusts on alveolar macrophage cultures, Beck et al [1972] showed that long glass fibers, like long asbestos fibers, greatly increased the permeability of the cell membrane while glass powder did not. This change in membrane permeability, as demonstrated by increase in lactic dehydrogenase activity in the supernatant fluid, appeared to be related to the process of ingestion of the fiber; as it was a slow process, the increased permeability might be sustained for many hours and substances leak from these cells. This factor might affect capillary permeability and together with the chemical composition of the fiber influence the amount of Perls-positive material in alveolar regions, since Szentei [personal communication, 1970] showed that haemolysis of red blood cells was greater with chrysotile than glass fiber or powder.

Thus, increased diapedesis following fiber inhalation and greater haemolysis by chrysoltile could affect the development of a Perls-positive coating around ingested fibrous dust.

Fiber length may also result in several macrophages being close enough to form giant cells thus bringing sufficient Perls-positive material near to a fiber for a coating to be formed. Fewer giant cells were seen after glass powder inhalation; this also resulted in fewer blocked bronchioles and a greater clearance of intracellular Perls-positive material and powdered glass in individual phagocytes.

Stanton and Wrench [1972] found that when glass fiber of a diameter comparable to asbestos was applied to the pleura of rats, a relatively high incidence of mesotheliomas occurred. Davis [1972] also recognized that when dusts are finely ground before intrapleural injection, they produced small distinct lesions, while long fiber samples resulted in larger, more cellular lesions.

Thus, similarities in the effects of inhaled glass fiber and asbestos dusts in the lungs of guinea pigs may be due to similar fiber dimensions, while their chemical differences affect the speed at which changes occur.

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2. Botham SK, Holt PF
Pathol 96:443-53,

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lungs of guinea pig

4. Botham SK, Holt
chrysotile and croc
105:159-67, 1971b

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guinea pig lungs.

6. Davis JMG: The fib
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Summary

Guinea pigs were kept for 24 hours in an atmosphere that contained a high concentration of glass fiber particles of sub-micron diameter or finely powdered glass of the same chemical composition.

Many fibers and particles were cleared in bronchiolar debris within a week after dust inhalation but other particles were retained in alveolar phagocytes. Meanwhile, red blood cells escaped from capillaries into alveoli and an iron-containing material subsequently appeared in alveolar macrophages.

Some intracellular glass fibers became coated with this material within a few days of inhalation, but by the end of the month no powdered glass particles had become coated. Subsequent development of glass fiber bodies followed the sequence demonstrated for asbestos bodies, but was slower than that recorded for chrysotile though more rapid than for other asbestos varieties.

Inhalation of powdered glass resulted in less severe diapedesis. Fewer giant cells formed and so red blood cells and intracellular glass particles were cleared more readily as junctions between respiratory and terminal bronchioles remained open.

The similarities between the effects of inhaled asbestos and glass fiber and differences between glass fiber and powder are considered to be due to the morphology of the inhaled particles.

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PATHOLOGIC
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Introduction

DR. HOOVE
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Presentation

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PATHOLOGICAL ASPECTS OF THE INJECTION OF GLASS FIBER INTO THE PLEURAL AND PERITONEAL CAVITIES OF RATS AND MICE

J. M. G. Davis

Introduction

DR. HOOVER: The next paper will be presented by Dr. J.M.G. Davis, who is head of the Pathology Branch, Institute of Occupational Medicine, Edinburgh, Scotland.

Presentation

DR. DAVIS: The pathological effects of fibrous asbestos have been known for a number of years and strict dust control measures have been enforced for the industrial processing of this material. Until recently, however, it has been assumed that other fibrous minerals, including glass, are harmless, and no dust standards have been laid down for their use other than a general standard of 15 mg/cu m which applies to all 'nuisance' dusts in the United States.

Clinical studies have so far given rather conflicting results, thus Wright [1968] reported that no abnormalities could be found in the chest X-rays of more than 1,400 workers from a factory processing fibrous glass. Gross et al [1971] examined the lungs of 20 workers from a similar factory and found no pathological lesions that could be attributed to the inhalation of glass fiber. However, Murphy in 1961 had reported a case in which the industrial inhalation of glass had caused sufficient lung damage to necessitate a right lower lobectomy.

Animal experimental work on the pathological effects of glass fiber on lung tissue have used both intratracheal injection and inhalation. Schepers [1955], Gross et al [1970], and Botham and Holt [1973] all reported that the presence of glass fiber in the lungs resulted in a marked cellular response consisting mainly of dust containing macrophages and giant cells. However, this cellular reaction did not progress to fibrosis as occurs with asbestos dust. Damage to the bronchial tree would be expected after intratracheal injection, but Schepers reported that in his studies there were marked bronchial changes after the inhalation of glass fiber. These changes included epithelial hyperplasia, epibronchial cellular infiltration, bronchiectasis and bronchiolectasis. It also was noted by Gross that while glass fiber inhalation did not result in fibrosis of the lung parenchyma in his experiments, pleural fibrosis was found, as well as fibrosis within the satellite lymph nodes.

This indicated the desirability of studying the effects of glass fiber injected directly into the pleural cavity, and a number of studies were undertaken in different laboratories. Stanton and Wrench [1972] found that thick glass fiber was not carcinogenic when injected into the pleural cavities of rats. However, when glass of smaller diameter was used, significant numbers of mesotheliomas developed. In a later paper, Maroudas et al [1973] extended these observations to suggest that fiber length was as important as fiber diameter and that to produce neoplasia glass fibers must be more than 20 μ in length. Wagner [1973] also undertook

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intrapleural injections of glass fiber into rats but produced no tumor with his glass fiber sample, although one mesothelioma was found after the injection of glass powder. These results are difficult to correlate with Stanton's work, but Wagner reported that the fiber length in his material averaged 20 μ. It may be that this sample contained too few fibers long enough to be carcinogenic.

The present study involved the injection of glass fiber into both the pleural and peritoneal cavities of mice. Two samples of a boron silicate glass fiber were used. One had an average diameter of 0.05 μ and the other 3.5 μ. Each of these samples was treated with two different methods to produce the material for injection. First, samples of the original felted mass of glass fibers were saturated with distilled water and gently ground in a hand mortar and pestle. The supernatant fluid was then poured off and concentrated by centrifugation. The resulting glass suspension consisted mainly of long fibers several hundred microns in length (Figure 13-1). As an alternative, glass fibers were ground down in a mechanical mortar and pestle. This treatment greatly reduced fiber length and few fibers in these samples exceeded 20 μ (Figure 13-2). In the first series of experiments, all four glass samples were suspended in distilled water at a concentration of 20 mg/ml and doses of 10 mg were injected into the pleural cavities of separate groups of 25 Balb/C mice. The animals were killed at intervals from 2 weeks to 18 months after injection and the tissues subjected to light microscope and, in some cases, electron microscope examination. All the glass samples produced granulomas within the pleural cavity, but these differed markedly in their size and structure. It was found that short fiber samples of both large and small diameter glass produced very small compact granulomas which never formed adhesions between the lungs, heart and chest wall. Within the granulomas the glass particles were surrounded by large numbers of macrophages and fibroblasts, but the dust was closely packed and masses of glass particles were clearly visible between the cells (Figure 13-3). The granulomas eventually fibrosed but since these lesions were so small initially, the amount of collagen produced in response to a 10 mg dust dose was also very small. With the long fiber samples, however, the results of intrapleural injection were different. With these materials very large granulomas were produced, which often filled a large part of the pleural cavity and formed firm adhesions between the lung, diaphragm, heart, and chest wall. Within the granulomas the glass fibers were not packed closely together, but remained widely separated. They were not usually visible at all by low power light microscope examination, but could be seen at higher magnification (Figure 13-4). A small amount of collagen was present within the granulation tissue from about 2 weeks after injection. After this time, however, collagen production was gradual but continuous until in older animals the lung lobes were often firmly bound to the chest wall by masses of old fibrous tissue that still contained dust fibers. The degree of fibrosis produced by these samples of long glass fiber appeared as severe as that produced by similar doses of chrysotile or crocidolite asbestos. [Davis, 1971]

Of the 100 mice used in this study, 52 were allowed to survive for more than one year. After this time, a number of deaths occurred, but 37 of the mice were still alive 18 months after injection. The experiment was then terminated and all the animals were killed for examination. No tumors



FIGURE 13-1.
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FIGURE 13-1. ELECTRON MICROGRAPH OF FIBERS SEVERAL HUNDRED MICRONS IN LENGTH WITH AVERAGE FIBER DIAMETER OF 0.05 (μ) MAGNIFICATION X 10,000

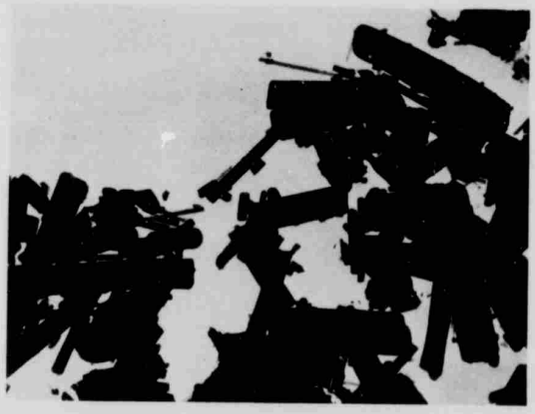


FIGURE 13-2. ELECTRON MICROGRAPH OF FIBERS MILLED TO AVERAGE LENGTHS LESS THAN 20 (μ). MAGNIFICATION X 10,000

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FIGURE 13-3. PART OF A GRANULOMA PRODUCED IN THE PLEURAL CAVITY OF A MOUSE 2 WEEKS AFTER THE INJECTION OF A SAMPLE OF FINELY GROUND GLASS FIBER (AVERAGE DIAMETER 0.05 μ). MAGNIFICATION X 4,500

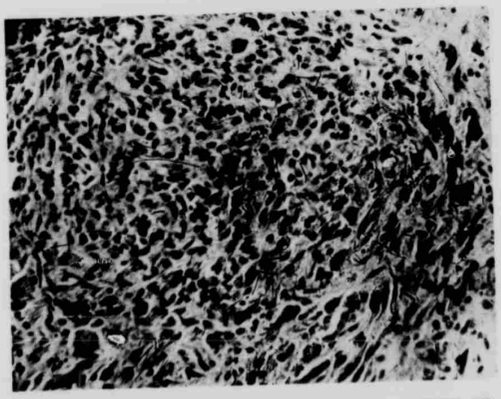


FIGURE 13-4. PART OF A GRANULOMA IN THE PLEURAL CAVITY OF A MOUSE 2 WEEKS AFTER THE INJECTION OF A SAMPLE OF GLASS FIBER THAT HAD BEEN PRODUCED BY GENTLE GRINDING IN ORDER TO RETAIN A HIGH AVERAGE FIBER LENGTH. MAGNIFICATION X 450

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were found within the pleural cavity of any animal during the course of this study, but in a parallel experiment involving the injection of chrysotile or crocidolite asbestos into the pleural cavities of mice, only two mesotheliomas were found in a total of 150 mice. It would appear, therefore, that the pleural cavity of the mouse is very resistent to tumor production by any type of mineral fiber.

Since evidence was available that mesotheliomas might develop more readily in the peritoneal cavity than the pleural cavity, a further series of experiments was undertaken in which long fiber samples of the smallest diameter glass fiber (average diameter 0.05 μ) were injected into the peritoneal cavity of 25 Balb/C mice. The dose level remained at 10 mgs of glass. In this study 18 rats were also given intraperitoneal injections of the fine glass fiber, but the dose level was increased to 25 mg for these animals. For this study the animals were left for their full life span or until there were signs of tumor development. Three peritoneal tumors were found in the rats and three in the mice. In structure, these tumors appeared identical to those produced in the peritoneal cavities of rats and mice by the injection of crocidolite asbestos. [Davis, 1974]

The earliest discernible tumor stage consisted of large numbers of very small pedunculated nodules scattered over the surfaces of the viscera, the diaphragm, and the body wall. These nodules normally contained a central core of reticulin or collagen around which were arranged layers of pleomorphic connective tissue cells. The surfaces of the nodules were covered with a single layer of epithelial cells very similar to normal mesothelium. In later stages some of the tumor nodules remained distinct and became quite large but often the tumor spread over the peritoneal surfaces as a relatively uniform sheet. For the most part, the cells found in the tumor sheets were of the same type as those found in large nodules, pleomorphic connective tissue cells in the central region, and epithelial cells on the surface. In some advanced tumors, the cells adopted a spindle cell pattern very similar to a fibroma or a fibrosarcoma. In this form the tumors were locally invasive, although at earlier stages no signs of invasion could be detected in any animals. Electron microscope studies showed that there was only slight difference in ultrastructure between the different tumor types (Figures 13-5,6,7,8) and it is suggested that the tumors arise from undifferentiated mesenchymal cells in the submesothelial tissues. These cells may retain their normal pleomorphic pattern or give rise to either epithelial cells on free surfaces or spindle cells in deeper layers.

From this group of studies it would appear that in the pleural or peritoneal cavities glass fiber behaves in a very similar fashion to asbestos. Thus, long fiber samples produced massive fibrosis, while short fiber samples produced only small discrete granulomas with minimal fibrosis. (Long fiber samples produced neoplasms in the periotoneal cavity but not in the pleural cavity.) Within animal lung tissue however, Shepers, Gross, and Holt suggest that glass fiber does not produce fibrosis, and this was also true of the human case reported by Murphy. Glass fiber may not, therefore, produce a typical fibrotic pneumoconiosis in human beings even if widespread industrial use of small diameter fiber (less than 3 μ) does occur in the future.

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FIGURE 13-5. ELECTRON MICROGRAPH OF AN AREA FROM A SMALL TUMOR NODULE PRODUCED IN THE PERITONEAL CAVITY OF A RAT 15 MONTHS AFTER THE INJECTION OF A SAMPLE OF GLASS FIBER. MAGNIFICATION X 24,000



FIGURE 13-6. AN AREA OF CELL CYTOPLASM FROM A TUMOR NODULE PRODUCED IN THE PERITONEAL CAVITY OF A MOUSE 14 MONTHS AFTER THE INJECTION OF GLASS FIBER. MAGNIFICATION X 44,000

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FIGURE 13-7. AREAS OF THE SURFACE MEMBRANES OF SEVERAL CELLS ON THE PERIPHERY OF A TUMOR NODULE PRODUCED IN THE PERITONEAL CAVITY OF A RAT 16 MONTHS AFTER THE INJECTION OF GLASS FIBERS. MAGNIFICATION X 32,000

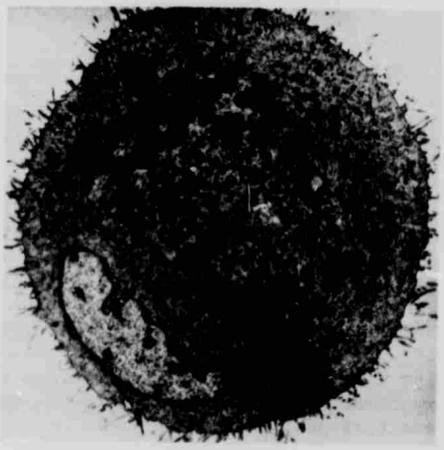


FIGURE 13-8. ELECTRON MICROGRAPH OF A LARGE TUMOR CELL FROM RAT PERITONEAL ASCITES FLUID 18 MONTHS AFTER INJECTION OF FINE FIBER GLASS. MAGNIFICATION X 5,500

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The problem of neoplasia is a different one, however, and the possibility of human tumors caused by glass fiber must be carefully examined before it is possible to decide upon an effective threshold limit value for fine glass fiber in the human environment.

Since the behavior of glass fiber and asbestos in the pleural and peritoneal cavity is very similar, it is important to consider why the presence of glass fibers in the lungs does not apparently result in lung fibrosis of the type caused by asbestos. The reasons for this may be found in the reaction of glass fiber with lung macrophages. Heppleston [1967] has shown that fibrogenic mineral dusts probably stimulate fibrosis by causing the deaths of the macrophages which form the initial cellular response. Such evidence presently available from tissue culture experiments suggests that both chrysotile and glass fiber increase the permeability of macrophage cell membranes to a similar degree during the process of phagocytosis [Beck et al, 1972] but Szentei [1970] found that glass was much less haemolytic than chrysotile. If glass fiber does have a significantly lower cytotoxicity for lung macrophages than the various asbestos types, then it is probable that after glass inhalation few macrophages will be killed (at least initially). Since lung macrophages are continually removed from the lung via the bronchial tree, it is likely that the number of dead macrophages in the lung at any one time is never enough to stimulate fibrosis. Within the body cavities, there is little ability to move an injected mass of dust or the cells that accumulate around it. In these conditions even glass may produce sufficient macrophage death to cause fibrosis.

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THE EFFECTS OF INTRATRACHEAL INSTILLATION OF GLASS FIBER OF VARYING SIZES IN GUINEA PIGS

Marvin Kuschner
George Wright

Introduction

DR. HOOVER: This paper will be presented by Dr. Marvin Kuschner, Dean of the School of Medicine, State University of New York, Stony Brook, New York.

Presentation

DR. KUSCHNER: The senior author of this paper is properly George Wright, who secured the materials that were furnished us by the J.M. Research and Engineering Center and who performed all the intratracheal installations himself.

This is a very preliminary report, one that can hardly be termed more than work in progress. The insights gained from some of the studies I shall describe to you should permit us to plan a proper experiment, one which utilizes inhalation rather than the exploratory intratracheal efforts. There are many more questions we can ask of this experiment, such as questions relating to the handling, translocation, and disposition of glass fibers of various lengths and diameter within the lung. Another series of questions that will require additional investigative efforts relate to the mechanisms of tissue damage which we suspect are dependent upon the role of endogenous mediators.

Groups of 30 guinea pigs each were exposed to a variety of types of asbestos, crocidolite, chrysotile, and a synthetic fluoro-amphibole with varing spectra of lengths and diameter. Similarly, groups of guinea pigs were exposed to a variety of glass fibers, differing in lengths and diameters. Exposure was by means of intratracheal instillation of a fiber suspension in 1/2 ml of distilled water. The number of injections in each group varied from 2 to 6 and the total amounts injected from 3 to 25 mg. The differences were dependent upon the difficulty encountered in injecting very fine fibrils of asbestos or very thin glass fiber (and I shall define these more precisely presently). Suspensions of these fine materials produced a jelly unless very diluted and, on injection, balled up in the bronchioles and did not give the proper dispersion.

These preliminary experiments cannot be considered exploratory of dose response relationships or of dose dependent differences among a variety of species of fiber. Rather, they should be looked on as examining the presence or absence of a significant biological response to any particular fiber. Pigs were sacrificed 6 months, 1 year, and 2 years after the last injection. (Injections were given 2 weeks apart.) Although our major interest today is in glass fiber, I should like to show you some of the asbestos results to provide a background for the glass work.

AA-1

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First, I'd like to compare two samples of crocidolite prepared from the UICC sample, which is characterized as short and thin. (Table 14-1) In this sample only 2% are longer than 10 μ and all the fibers are below 1 μ in diameter. Indeed, the thickest is 0.62 μ. Twenty-five mg were introduced in two injections. Figure 14-1 is an electron micrograph of this sample. At 6 months, the reaction is primarily a macrophage reaction. Figure 14-2 is a low power photograph of the lung showing the presence of intra-alveolar macrophages. Figure 14-3 is a high power photograph in which fiber may be seen in a multi-nucleate giant cell. Figures 14-4,5,6 and 6a is a whole mount of the lung after 2 years and illustrates the absence of any massive fibrosing lesion. Indeed, in Figures 14-5 and 14-6 which are low power and high power, respectively, of the 2-year lung, one may see that except for the presence of macrophages in the alveoli, there are no significant alterations in pulmonary structure. Figures 14-7 and 14-8 are low power and high power respectively of the lymph node in a 2-year sacrifice animal and demonstrates the absence of fibrosis and the presence of refractile spectules within macrophages invoking little other reaction.

Table 14-2 lists the characteristics of the next sample which has a similar distribution of diameters, but in which more than 80% of the fibers are longer than 10 μ. The animals received only 4 mg. Figure 14-9 is a micrograph of this long fibered asbestos sample. Figure 14-10 is the whole mount of a lung after 2 years and broad areas of alteration can be recognized. Figures 14-11 and 14-12 show extensive fibrosis involving the region of the respiratory bronchi extending out into the proximal alveoli with fiber present in the interstition. Thus, two samples of crocidolite of comparable diameter but differing in length produced quite different effects.

Next, I should like to compare two samples of very thin asbestos, namely, chrysotile fibrils, measuring 0.03-0.05 mg in diameter. Table 14-3 lists the characteristics of a short fibril with only 1.6% over 10 μ and none over 20. The animals received 6 mg with four injections. Figure 14-13 is an electron micrograph of this short fibril sample. Figure 14-14 is the 2-year whole mount and demonstrates the absence of significant reaction. In Figure 14-15, the details of the architecture of the lung are preserved without alteration.

Table 14-4 gives the characteristics of a long fibril sample. These animals received half the dose of the preceding group, 3 mg in six injections. Somewhat less than one-quarter of the fibrils were over 10 μ in length. Figure 14-16 is an electron micrograph of these long fibrils. Figure 14-17 is the 2-year whole mount of the lungs of an animal receiving long fibrils and demonstrates widely distributed areas of abnormality. Figure 14-18 - These are seen to be areas of fibrosis again in the area of the respiratory bronchioles and proximal alveoli. Figure 14-19 demonstrates that these animals showed no significant alteration in their hilar lymph nodes. The last two asbestos groups I wish to show are derived from a synthetic fluoroamphibole. Again, these are thin fibers with diameters less than 1 μ.

SHORT F
GROUP 11 SAMPLE BY WE

Length (μ)	% of Tot
0.40-00.99	17.6
1.00-01.9	44.4
2.00-04.9	29.8
5.00-09.9	6.2
10.00-19.0	1.6
20.00-29.0	0.4
	100%

Minimum 0.6
Maximum 22
Total Measured 245

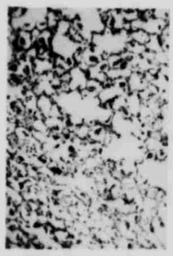


FIGURE 14-2



FIGURE 14-6

TABLE 14-1

SHORT RESPIRABLE ASBESTOS
GROUP 11 SAMPLE BY WEBSTER SOUTH AFRICAN CROCIDOLITE

Length (μ)	% of Total	Diameter (μ)	% of Total
0.40-00.99	17.6	0.03-0.04	1.2
1.00-01.9	44.4	0.05-0.09	7.5
2.00-04.9	29.8	0.10-0.19	43.3
5.00-09.9	6.2	0.20-0.39	32.4
10.00-19.0	1.6	0.40-0.59	4.4
20.00-29.0	0.4	0.60-0.99	1.2
	100%		
Minimum 0.6		0.04	
Maximum 22		0.62	
Total Measured 245		252	

MICROGRAPH
OF FIBERS



FIGURE 14-1

TISSUE EFFECTS

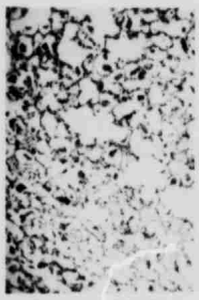


FIGURE 14-2



FIGURE 14-3



FIGURE 14-4

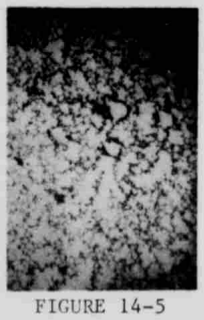


FIGURE 14-5



FIGURE 14-6



FIGURE 14-6a

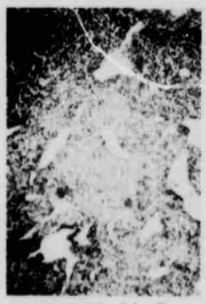


FIGURE 14-7



FIGURE 14-8

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TABLE 14-2

LONG RESPIRABLE ASBESTOS
GROUP 14A SAMPLE 4106-22-4 (CROCIDOLITE)

Length (μ)	% of Total	Diameter (μ)	% of Total
3.00-004.9	4.2	0.05-0.0	5.3
5.00-009.9	13.6	0.10-0.14	27.8
10.00-019.0	28.3	0.15-0.19	15.0
20.00-029.0	23.0	0.20-0.29	30.9
30.00-059.0	24.1	0.30-0.39	13.1
60.00-129.0	6.8	0.40-1.49	7.9
	100%		100%
Minimum 3.6		0.05	
Maximum 127		1.20	
Total Measured 191		620	

MICROGRAPH
OF FIBERS



FIGURE 14-9

GROUP 1

Length (μ)
0.10-00.69
0.70-00.99
1.00-01.9
2.00-04.9
5.00-09.9
10.00-19.0
Minimum 0.3
Maximum 18.0
Total Measure

TISSUE EFFECTS



FIGURE 14-10



FIGURE 14-11



FIGURE 14-12

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14-9

TABLE 14-3

SHORT RESPIRABLE ASBESTOS
GROUP 19 SAMPLE 4173-46-1 (JEFFREY FIBRILS)

Length (μ)	% of Total	Diameter (μ)	% of Total
0.10-00.69	10.4	0.03-0.05	99.9%
0.70-00.99	19.1		
1.00-01.9	34.7		
2.00-04.9	27.6		
5.00-09.9	6.6		
10.00-19.0	1.6		
	100%		

Minimum 0.3
Maximum 18.0
Total Measurements 550

MICROGRAPH
OF FIBERS



FIGURE 14-13

TISSUE EFFECTS



FIGURE 14-14

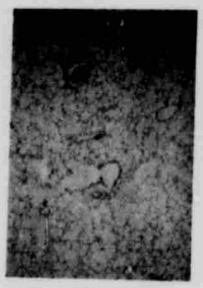


FIGURE 14-15

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TABLE 14-4

LONG RESPIRABLE ASBESTOS
GROUP 22 SAMPLE 4173-46-2 (JEFFREY FIBRILS)

Length (μ)	% of Total	Diameter (μ)	% of Total
0.10-00.99	11.8	< 0.10	99.9%
1.00-01.9	20.0		
2.00-04.9	2[illegible]		
5.00-09.9	20.5		
10.00-19.0	17.7		
20.00-39.0	5.9		
	100%		

Minimum 0.3
Maximum 34
Total Measured 220

MICROGRAPH OF FIBERS



FIGURE 14-16

GROUP 24 SA

Length (μ)
0.06-0.09
0.10-0.39
0.40-0.99
1.00-01.9
2.00-04.9
5.00-19.0

Minimum .
Maximum 12.
Total Measu

TISSUE EFFECTS



FIGURE 14-17



FIGURE 14-18



FIGURE 14-19



FIGURE

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TABLE 14-5

SHORT RESPIRABLE ASBESTOS
GROUP 24 SAMPLE 4106-31-3 SYNTHETIC FLUOROAMPHIBOLE

Length (μ)	% of Total	Diameter (μ)	% of Total
0.06-0.09	0.5	0.03-0.09	46.0
0.10-0.39	52.2	0.10-0.19	40.9
0.40-0.99	37.2	0.20-0.39	9.6
1.00-01.9	5.8	0.40-0.99	2.9
2.00-04.9	3.6	1.00-2.95	0.6
5.00-19.0	0.7		
	100%		100%
Minimum .08		0.01	
Maximum 12.0		2.10	
Total Measured 444		313	

MICROGRAPH OF FIBERS

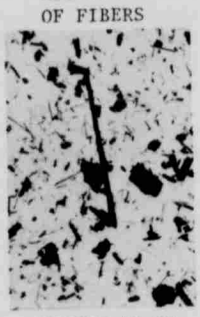


FIGURE 14-20

TISSUE EFFECTS



FIGURE 14-21

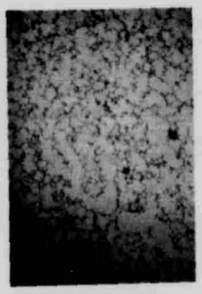


FIGURE 14-22



FIGURE 14-23

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Table 14-5 is a short fiber of this type with only 0.7% over 5 μ. The animals received 12 mg in three injections. Figure 14-20 is a photo micrograph of these fibers with occasional long coarse fibers and some blocky fragments. Figure 14-21 shows the normality of the lung after 2 years. This is emphasized by Figure 14-22 in which there is no significant alteration and Figure 14-23 demonstrates that the fiber has been translocated to the lymph node where there is a macrophage reaction that is fibrosis.

The dimensions of long fibers of similar composition and comparable diameter are shown in Table 14-6. Here 16% of the fibers are over 10 μ and that is enough to make a considerable difference. The animals received the same dose (12 mg in three injections) as did the previous group. The electron micrograph of these fibers is shown in Figure 14-24. Figure 14-25 - The whole mount of the lung (2 years) demonstrates extensive lobular fibrosis. Figure 14-26 shows its location in the same vulnerable site, the respiratory bronchioles and the peribronchiolar alveoli. The indications then would seem to be that thin and very thin fibers of asbestos below 10 μ in length do not produce fibrosis.

Now we will turn to glass fibers. First, let us look at two groups of thin glass fiber, most of the diameters being below 1 μ. Table 14-7 illustrates a short thin group with only 7% of the fibers greater than 10 μ. The animals received two injections of 12.5 mg each, i.e., a total of 25 mg. Figure 14-27 is the electron micrograph of these glass fibers. Figure 14-28 is a whole mount of the lung after one year demonstrating the absence of architectural alteration at that time. In Figure 14-29, one may see that there are alveoli filled with macrophages but no fibrosis. In Figure 14-30, one may see that the fibers have reached the lymph node where they are present within macrophages, again without fibrosis. Figure 14-31 is the whole mount of a lung after 2 years and again the absence of evident alteration in pulmonary architecture is noted. In Figure 14-32 one may see that the pulmonary architecture is indeed preserved but there are aggregates of alveoli containing macrophages. Figure 14-33 is a high power photomicrograph of fiber-bearing macrophages in alveoli without alteration of the pulmonary paranchyma. Figure 14-34 again shows the presence of a macrophage reaction to fiber in the lymph node without fibrosis.

The next group is of a comparable fiber size but with only 7% of the fibers less than 10 μ. This is characterized in Table 14-8. These animals received three injections of 4 mg each. Figure 14-35 is a micrograph of these fibers. Figure 14-36 demonstrates the presence of some interstitial reaction to these long fibers which can be seen easily within the interstitium in Figure 14-37. Figure 14-38 demonstrates that this reaction, as in asbestos, involves the areas of the respiratory bronchioli and proximal alveoli. All of the three foregoing figures are of the lung at 6 months. In Figure 14-39, the lung at 1 year may be seen and there is apparently coarsening of the texture. Figures 14-40 and 14-41 demonstrate the presence of peribronchiolar interstitial fibrosis. Figure 14-42 shows some lymph node reaction. Figure 14-43, at 2 years again, shows coarsening of the texture of the lung which, on higher magnification (Figures 14-44 and 14-45), again involve the proximal portions of the respiratory lobule.

LONG
GROUP 23 SAMPLE 41

Length (μ)	% of
0.5-01.49	8
1.5-02.9	21
3.0-04.9	27
5.0-09.9	27
10.0-19.0	12
20.0-39.0	3
	10
Minimum 0.8	
Maximum 34	
Total Measured 200	

FIG

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TABLE 14-6

LONG RESPIRABLE ASBESTOS
GROUP 23 SAMPLE 4106-30-1 SYNTHETIC FLURO AMPHIBOLE

Length (μ)	% of Total	Diameter (μ)	% of Total
0.5-01.49	8.2	0.03-0.04	44.7
1.5-02.9	21.5	0.05-0.14	25.3
3.0-04.9	27.0	0.15-0.39	20.9
5.0-09.9	27.5	0.40-0.99	5.6
10.0-19.0	12.5	1.00-2.00	3.5
20.0-39.0	3.5		
	100%		100%
Minimum 0.8		0.03	
Maximum 34		1.60	
Total Measured 200		340	

MICROGRAPH
OF FIBERS



FIGURE 14-24

TISSUE EFFECTS



FIGURE 14-25

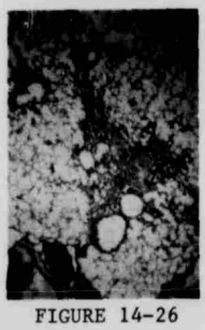


FIGURE 14-26

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TABLE 14-7

SHORT-THIN GLASS FIBERS
GROUP 2 SAMPLE 4106-4-3 (12.5 mg x 2)

Length (μ)	% of Total	Diameter (μ)	% of Total
1.0-1.9	14.1	0.05-0.14	5.9
2.0-2.9	35.2	0.15-0.29	16.4
3.0-4.9	35.2	0.3 -0.59	35.4
5.0-9.9	8.5	0.6 -0.99	26.2
10.0-22.0	7.0	1.0 -1.99	16.1
Minimum 1.1		0.06	
Maximum 22		1.98	
Total Measured 71		305	

MICROGRAPH
OF FIBERS



FIGURE 14-27

TISSUE EFFECTS



FIGURE 14-28



FIGURE 14-29



FIGURE 14-30



FIGURE 14-31

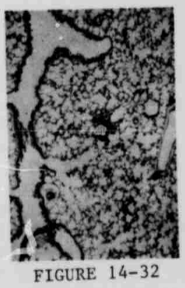


FIGURE 14-32



FIGURE 14-33



FIGURE 14-34

GROU
GROU

Length (μ)
3.0-04.9
5.0-09.9
10.0-19.0
20.0-39.0
40.0-59.0
60.0-99.0
Minimum 4.0
Maximum 80
Total Measur



FIGURE 14-36



FIGURE 14-40

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TABLE 14-8

LONG-THIN GLASS FIBERS
GROUP 10 SAMPLE 4106-19-2 (12.5 mg x 2)
GROUP 12 SAMPLE 4106-19-2 (4.0 mg x 3)

Length (μ)	% of Total	Diameter (μ)	% of Total
3.0-04.9	0.7	0.05-0.14	51.4
5.0-09.9	7.0	0.15-0.19	19.6
10.0-19.0	37.5	0.20-0.29	11.9
20.0-39.0	36.0	0.30-0.39	6.1
40.0-59.0	14.8	0.40-0.59	6.2
60.0-99.0	4.0	0.60-1.49	4.8
Minimum 4.0		0.05	
Maximum 80		1.25	
Total Measured 405		210	

MICROGRAPH OF FIBERS



FIGURE 14-35

TISSUE EFFECTS



FIGURE 14-36



FIGURE 14-37



FIGURE 14-38



FIGURE 14-39

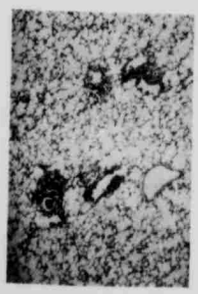


FIGURE 14-40



FIGURE 14-41



FIGURE 14-42



FIGURE 14-43

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FIGURE 14-44



FIGURE 14-45



FIGURE 14-46

Figure 14-46 is i
the larger fibers

Now a set
characterized in T
the fiber length
shown in Figure 14-
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reaction consistin
refractile materia
with still no react
14-52. The lymph
macrophage reactio
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Another very
contrast to the pre
length. An electro
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Figure 14-46 is illustrative of the paucity of change in the lymph node as the larger fibers are retained in the lung.

Now a set of very thin glass fibers of short diameter are characterized in Table 14-9. Here the diameters are less than 0.3 μ and the fiber lengths are less than 5 μ. A microphotograph of the sample is shown in Figure 14-47. Figure 14-48 is the whole mount of the lung after 1 year with no architectural alteration. Figures 14-49 and 14-50 show the reaction consisting simply of otherwise unaltered alveoli containing refractile material. Figure 14-51 is a whole mount of the lung at 2 years with still no reaction as emphasized by the microphotograph shown in Figure 14-52. The lymph node is shown in Figures 14-53 and 14-54 and there is a macrophage reaction with no fibrosis, the macrophages containing refractile material.

Another very thin fiber is characterized in Table 14-10 but here, in contrast to the preceding group, some 50% of the fibers are over 10 μ in length. An electron micrograph of these long thin glass fibers is shown in Figure 14-55. Figure 14-56 is the whole mount and this micrograph, as well as Figures 14-57, 58, and 59, show coarsening of the texture of the lung with interstitial fibrosis about the respiratory bronchioles and involving the proximal alveoli.

Finally, here are two samples of relatively thick glass fibers with diameters around 2 μ. Table 14-11 lists the characteristics of a group with 88% of the fibers below 10 μ in length. Figure 14-60 is an electron micrograph of these fibers. Figure 14-61 is a whole mount which shows some mild focal alteration of pulmonary structure. The photomicrograph of this is shown in Figure 14-62. At 2 years, Figures 14-63, 64, and 65 show some interstitial fibrosis and this perhaps may be attributed to the small, but significant, percentage of fibers (12% over 10 μ). Some of these longer fibers actually can be seen in Figure 14-65. Figure 14-66 shows the lymph node containing fiber at 2 years.

In Table 14-12, there are given the characteristics of a long thick fiber with 75% of the fibers over 10 μ in length. An electron micrograph of this sample is shown in Figures 14-67 and 14-68. Figures 14-69 and 14-70 show the lung at 6 months with focal areas of interstitial fibrosis. In Figure 14-71, a whole mount of the lung after 2 years, some coarsening of texture is seen as further demonstrated by Figures 14-72 and 14-73. A ferruginous body is seen in Figure 14-74. Figures 14-75 and 14-76 show fibers within lymph nodes with accompanying macrophage reaction.

Under the conditions of this experiment, there are certainly striking quantitative differences between asbestos fiber and glass fiber; but there is the strong suggestion that there are qualitative similarities. These similarities tend to support the contention that it is the physical characteristics of fiber which determine tissue damage. Indeed, one puzzling aspect is the apparent disappearance of long glass fiber from the lung. One is led to wonder whether durability of length may contribute to the quantitative differences.

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TABLE 14-9

SHORT-VERY THIN GLASS FIBERS
GROUP 13 SAMPLE 4106-23-7 (12.5 mg x 2)

Length (μ)	% of Total	Diameter (μ)	% of Total
0.1 -0.39	40.4	0.03-0.04	2.9
0.40-0.69	30.8	0.05-0.09	49.1
0.70-0.99	12.5	0.10-0.14	35.4
1.0 -1.4	8.6	0.15-0.19	5.7
1.5 -1.9	2.9	0.20-0.29	6.9
2.0 -4.9	4.8		
Minimum 0.1		0.03	
Maximum 4.1		0.29	
Total Measured 104		175	

MICROGRAPH
OF FIBERS



FIGURE 14-47

TISSUE EFFECTS



FIGURE 14-48

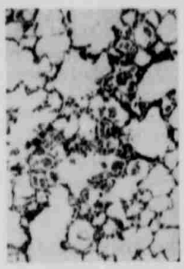


FIGURE 14-49



FIGURE 14-50



FIGURE 14-51

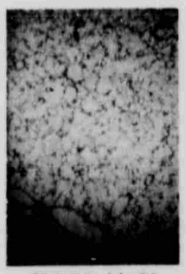


FIGURE 14-52



FIGURE 14-53



FIGURE 14-54

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MICROGRAPH
OF FIBERS



FIGURE 14-47



FIGURE 14-51



-54

TABLE 14-10

LONG-VERY THIN GLASS FIBERS
GROUP 16 SAMPLE 4106-24-2 (2 mg x 6)

Length (μ)	% of Total	Diameter (μ)	% of Total
1.0-02.9	9.2	0.03-0.04	5.6
3.0-04.9	15.8	0.05-0.09	58.1
5.0-09.9	25.0	0.10-0.14	25.6
10.0-19.0	30.0	0.15-0.19	8.8
20.0-29.0	9.2	0.20-0.29	1.6
30.0-59.0	10.8	0.30-0.59	0.3
Minimum 1.2		0.03	
Maximum 59		0.41	
Total Measured 120		774	

MICROGRAPH
OF FIBERS



FIGURE 14-55

TISSUE EFFECTS



FIGURE 14-56

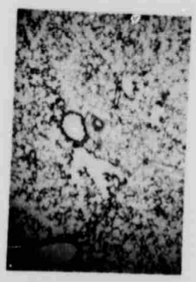


FIGURE 14-57



FIGURE 14-58



FIGURE 14-59

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TABLE 14-11

SHORT-THICK GLASS FIBERS
GROUP 1 SAMPLE 4106-6-1 (12.5 mg x 2)

Length (μ)	% of Total	Diameter (μ)	% of Total
1.0-02.9	22.4	0.40-0.59	3.4
3.0-04.9	33.5	0.60-0.99	5.8
5.0-09.9	31.5	1.00-1.49	15.2
10.0-19.0	10.5	1.50-1.99	28.6
20.0-29.0	2.1	3.00-4.95	17.6
Minimum 1.0		0.50	
Maximum 23.0		4.80	
Total Measured 143		119	

MICROGRAPH OF FIBERS



FIGURE 14-60

GROUP 17

Length (μ)	%
3.0-04.9	
5.0-09.9	
10.0-19.0	
20.0-29.0	
30.0-59.0	
60.0-99.0	
Minimum 4.)	
Maximum 92	
Total Measured	

TISSUE EFFECTS



FIGURE 14-61



FIGURE 14-62

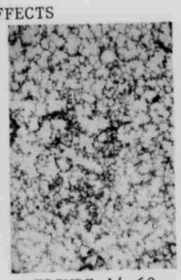


FIGURE 14-63

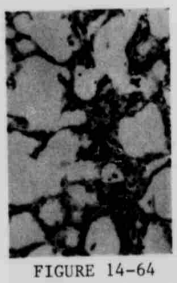


FIGURE 14-64



FIGURE 14-69



FIGURE 14-65

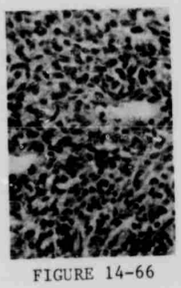


FIGURE 14-66



FIGURE 14-73

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MICROGRAPH OF FIBERS



FIGURE 14-60

TABLE 14-12

LONG-THICK GLASS FIBERS
GROUP 17 SAMPLE 4106-19-1A (12.5 mg x 2)

Length (μ)	% of Total	Diameter (μ)	% of Total
3.0-04.9	9.9	0.20-0.59	4.1
5.0-09.9	14.6	0.60-0.99	18.0
10.0-19.0	28.9	1.00-1.49	35.2
20.0-29.0	20.1	1.50-1.99	22.7
30.0-59.0	22.1	2.00-2.95	14.8
60.0-99.0	4.4	3.00-5.95	5.2
Minimum 4.0		0.20	
Maximum 92		5.20	
Total Measured 294		689	



FIGURE 14-67

MICROGRAPH OF FIBERS



FIGURE 14-68

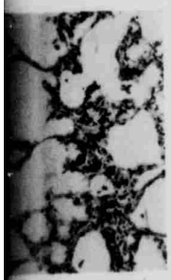


FIGURE 14-64

TISSUE EFFECTS



FIGURE 14-69



FIGURE 14-70



FIGURE 14-71



FIGURE 14-72



FIGURE 14-73



FIGURE 14-74



FIGURE 14-75



FIGURE 14-76

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It must also be conceded that the conditions of the experiment, that is, intratracheal injection, are highly artificial. (Indeed, as artificial as intrapleural instillation.) It produces an uneven dose and therefore an extraordinarily high local dose. It also produced aggregates which lodge unnaturally in larger air passages. Obviously, an inhalation experiment of improved design incorporating the use of carefully characterized fiber is now required.

THE EFFECTS

Introduction

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Presentation

DR. GROSS: The
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THE EFFECTS OF FIBROUS GLASS DUST ON THE LUNGS OF ANIMALS

Paul Gross

Introduction

DR. HOOVER: The next paper will be presented by Dr. Paul Gross, who is known to all of you. Dr. Gross formerly was Director of the Research Laboratories of the Industrial Hygiene Foundation, and now is having a very active retirement as the Distinguished Research Professor of Pathology at the Medical University of South Carolina.

Presentation

DR. GROSS: The results of animal experiments and conclusions derived from them can be misleading and completely erroneous if the experiments are not properly designed. For example, the rat would not be a suitable test animal for testing the capability of a putative inhaled irritant to cause bronchitis because chronic bronchitis is endemic in this species. Similarly, the occurrence of tumors in response to injections would be of dubious significance if these tumors occur spontaneously in the test animal employed. Extrapolation of results obtained in experimental animals to man may be received with justifiable skepticism when the route of administration of a suspected toxic material to the test animals has been totally different from that of man; so different, that the tissue responding to the challenge was not the same as in man. A like skepticism would be appropriate if the dosage employed has been an astronomical multiple of the probable human dosage.

When we planned our study on the pulmonary response to fiber glass dust, a number of problems required consideration. One of these was concerned with the size of the fiber glass particles that were to be inhaled by the test animals. Inasmuch as the average diameter of commercially available fiber glass was about 9 μ and inasmuch as very few particles larger than 3 μ in diameter reach the air spaces when inhaled, such commercially available fiber glass was deemed too coarse and unsuitable for conversion into a dust to be inhaled by test animals.

Information obtained from human lungs provided clues regarding the optimum size of fiber glass to be used in animal experiments. [Gross et al, 1970b and 1971] We previously had studied the mineral fiber content of the lungs of Pittsburghers and found that 75% of the fibers deposited in the lungs of these men and women ranged between 1.5 μ and 2.5 μ in diameter. Less than 6% of the fibers were thicker than 4 μ. [Gross et al, 1971] In order to be certain that the inhaled glass fiber particles would reach the air spaces and would be deposited there, we asked the fiber glass industry to provide us with glass fibers specially produced for this investigation.

A second problem in the design of the fiber glass study was concerned with the amount or concentration of the dust to which the animals were to be exposed. The dust exposure should be of such magnitude that the lung clearance mechanism is overwhelmed and significant dust accumulations occur

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in the lung tissue. The dust accumulations are necessary if tissue reactions to their presence are to be studied.

Materials and Method

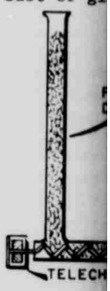
We obtained specially produced fiber glass that had an average diameter of about 1 μ. One-third of the shipment consisted of uncoated fibers; one-third, of fibers coated with a phenol-formaldehyde resin; and the remainder, with a starch binder. These fibers were ball-milled until the fiber lengths were predominantly less than 50 μ.

In preliminary short-term pilot studies, we found that rats would tolerate fiber glass concentrations as high as 2,000 mg/cu m and yet show disproportionally small dust accumulations in their lung tissue. Considering that the threshold limit value (TLV) for fiber glass dust in occupational surroundings had been set at 15 mg/cu m, this tolerated dust concentration was a very high exposure indeed. However, inasmuch as a 2-year exposure was contemplated, it was agreed that the dust concentration in the exposure chambers was to be around 100 mg/cu m, about 10 times as high as the present TLV. [Threshold limit values of ACGIH, 1972]

For dispersing the dust throughout the inhalation chamber we employed a squirrel-cage blower situated at the bottom of a d-shaped system of sheet-metal tubes, 10 cm in diameter (stove pipe) (Figure 15-1). An L-shaped glass reservoir of fiber glass dust was inserted into the vertical lucite tube. A spiral wormfeed mechanism in the horizontal limb of the "L" slowly fed the dust into the system, allowing the flake-like agglomerates to drop into the squirrel-cage blower. The latter caused turbulent refluxing of the dust, breaking up the agglomerates and allowing ultrafine particles to float upward into the inhalation chamber. An injection of air at a rate of 1 liter/minute through a 1/4 inch tube inserted into the horizontal limb of the sheet metal ensemble assisted in the transport of dust particles into the inhalation chamber. The spent chamber air was pulled at a rate of 1 liter/minute through a 10-inch furnace-type filter containing a preweighed filter paper. The average daily dust concentration in the chamber air was calculated from the increase in the weight of the filter paper divided by the volume of air that had passed through the filter. There were three chambers of similar construction, each with a capacity of 1.02 cu m. Each held 30 rats and 30 hamsters housed in 20 cages which were rotated daily. Exposures were for 6 hours daily, 5 days per week, for 24 months.

Periodically, the character of the dust in the chamber air was determined from photomicrographs taken with phase-contrast optics. The dust was obtained by pulling chamber air through a millipore filter at a rate of 1 liter/minute for 6 hours. It was found that 70 to 76% of the dust particles were fibrous and 24 to 30% non-fibrous. The fibers had an average diameter of 0.5 μ within a very narrow range and an average length of about 10 μ. The range of lengths was approximately 5 to 20 μ. In one chamber, the animals were exposed to uncoated glass fiber dust; in the

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INJECTED

FIGURE 1

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second chamber, to dust of glass fibers coated with resin; and in the third, to dust of glass fibers coated with starch binder.

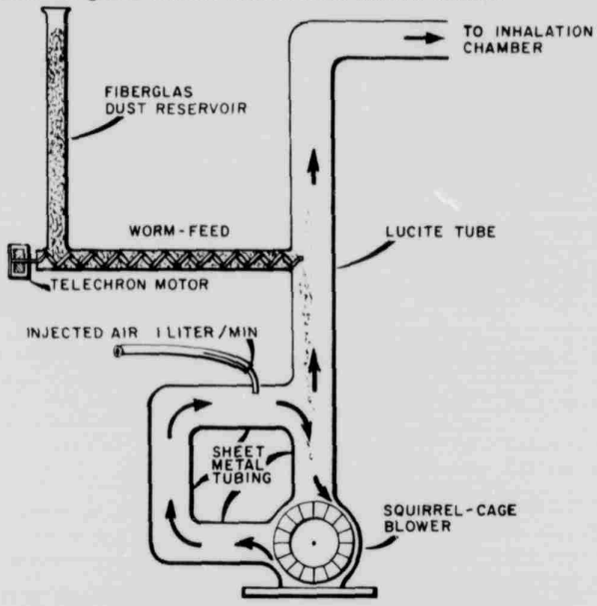


FIGURE 15-1. DIAGRAM OF DUST-DISPENSING APPARATUS

The animals were allowed to live out their lives after the completion of the dust exposure except for five rats and five hamsters from each chamber that were killed for sampling after 6 and 12 months exposure respectively. The same three dusts were also injected intratracheally into an additional 150 rats and 60 hamsters according to the schedule in Table 15-1. These animals were not sampled and all were allowed to live out their lives. A group of 20 rats and 20 hamsters from the same shipment as that of the experimental animals served as laboratory controls. They were kept in room air. All animals had access to food and water ad libitum. Altogether, there were 240 rats and 150 hamsters either exposed to, or injected intratracheally with dust of glass fibers.

All animals, except a few that had been cannibalized, were autopsied and the lungs distended with buffered formalin at a pressure of 12 cm water. Paraffin sections stained with hematoxylin and eosin (H & E), as well as with Gordon and Sweets silver impregnation technique, were studied and photographed. Many of the sections were also subjected to microincineration. By photographing a selected field of tissue stained with H & E, again after decolorization and silver impregnation, and finally after microincineration, information was obtained on the cell-stroma relationship of the tissue reaction and its relationship to the dust particles.

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TABLE 15-1

SUMMARY OF GLASS FIBER DUST ADMINISTRATION TO ANIMALS

Dust Administration	Type of Dust	No. of Rats	No. of Hamsters	Dose Injected mg	Chamber Dust Concent. mg/cu m	Number of Rats Killed for Sampling 6 mos	12 mos	No. of Animals Surviving 2 yrs
Inhalation	Uncoated	30			135	5	5	4
			30		135	5	5	7
	Coated with resin	30			106	5	5	8
			30		106	5	5	6
	Coated with starch binder	30			113	5	5	3
			30		113	5	5	13
Intratracheal Injections	Uncoated	15		3 x 3.5				0
		30		10 x 3.5				1
			12	3 x 3.5				3
	Coated with resin	30		3 x 3.5				0
		30		10 x 3.5				0
			12	1 x 3.5				1
			12	2 x 1.75				2
			12	3 x 3.5				4
	Coated with starch binder	15		3 x 3.5				0
		30		10 x 3.5				2
			12	3 x 3.5				9
Controls	none	20	20					
Total		260	170					

Results

Inhalation S
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Controls	none	20	170
	20		
Total	260		

Results

Inhalation Series: Grossly no abnormality attributable to the inhaled fiber glass dust was observed. Occasional patches of pneumonia and other complications of endemic murine chronic bronchitis were present. It is possible to summarize the microscopic findings on the lungs of test animals which inhaled the fiber glass dust as follows:

1. Whether the glass fibers were coated with resin or starch binder, or whether they remained uncoated resulted in no detectable difference in the pulmonary tissue reaction. It was not possible to determine, from the sections, which kind of glass fiber had been inhaled.
2. The tissue reaction evoked by inhaled fiber glass dust was that of a non-fibrogenic or so-called "inert" dust. It consisted of a macrophage reaction associated with minimal focal reticulin stromal increase but no collagen production.

In the lungs of all rats exposed to fiber glass dust, macrophagic accumulations were found in alveoli clustered around respiratory bronchioles and alveolar ducts. Such foci were much less numerous in rats that lived longest after completion of the dust exposure. The macrophages were well preserved even though often crowded into solid masses. Upon incineration of the section, these cells were found filled with acid-insoluble ash (glass). In contrast to the finding in coal miners' lungs and in the lungs of animals that had inhaled carbon dust where atelectasis of alveolar ducts and alveoli around dust accumulations is the rule (Figure 15-2), no atelectasis was associated with the presence of fiber glass dust (Figure 15-3). Not only was the alveolar architecture preserved, but the alveolar wall structure as indicated by its reticulin stroma had undergone little or no change. Focally, scattered alveoli showed slight arborization of the axial reticulin stroma and some alveoli showed hypertrophy and hyperplasia of type II cells (Figure 15-4).

In a few rats exposed to the higher dust concentrations (Table 15-1) there were several foci of collagenous fibrosis in the lungs. These were associated with cholesterol needle spaces, lipidic macrophages, and cellular debris (Figure 15-5). This is an all-too-familiar lesion in the lungs of rats inhaling large amounts of non-fibrogenic dusts [Gross et al, 1952; Gross and Nau, 1967; Gross et al, 1973], and is the result of alveolar stasis.

Significant transport of glass particles to satellite lymph nodes was not observed until after 1 year of dust exposure, although small clusters of large pale phagocytic cells filled with glass dust were noted in the lymphoid tissue after 6 months of exposure. Manifest enlargement of satellite lymph nodes occurred in only a few rats. In the enlarged nodes, the phagocytic cell accumulations were extensive and had been infiltrated and partially replaced by a coarse network of thick collagenous fibers. The insoluble ash after incineration suggested that nearly one-half of the volume of the enlarged nodes was occupied by the translocated glass dust, much of which was extracellular. In most of the other rats, the dust in the satellite nodes was intracellular and fibrosis was not evident (Figure 15-6).

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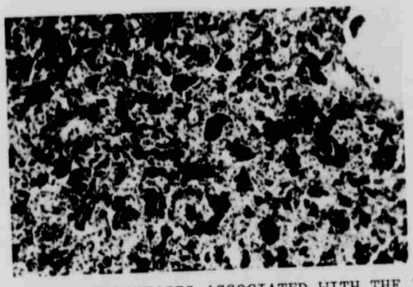


FIGURE 15-2. SEVERE ATELECTASIS ASSOCIATED WITH THE INHALATION OF CARBON DUST. THE MASSES OF CARBON REPRESENT CASTS OF THE STRONGLY CONTRACTED LUMENS OF ALVEOLI AND ALVEOLAR DUCTS. HEMATOXYLIN AND EOSIN. X 60



FIGURE 15-3. CHARACTERISTIC APPEARANCE OF FIBER GLASS DUST DEPOSITS IN THE LUNG OF A RAT SHORTLY AFTER COMPLETION OF 24 MONTHS OF EXPOSURE TO 95 mg/cu m FIBER GLASS DUST BY INHALATION. THE DUST IS CONTAINED IN WELL-PRESERVED, CLUSTERED MACROPHAGES. THERE IS NO ATELECTASIS AND NO FIBROSIS. HEMATOXYLIN AND EOSIN. X 190



FIGURE 15-4. A HIGHER MAGNIFICATION OF A REGION OF FIBER GLASS DUST DEPOSITION, SHOWING FOCAL HYPERPLASIA AND HYPERTROPHY OF TYPE 2 CELLS WITHOUT EVIDENCE OF FIBROSIS. HEMATOXYLIN AND EOSIN. X 390



FIGURE 15-5. CH
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FIGURE 15-6. SATEL
MACROPHAGES FILLED
HEMATOXYLIN AND EOS



FIGURE 15-7. FOC
A RAT THAT HAD IN
SUBJACENT ALVEOLI
AND EOSIN. X 70

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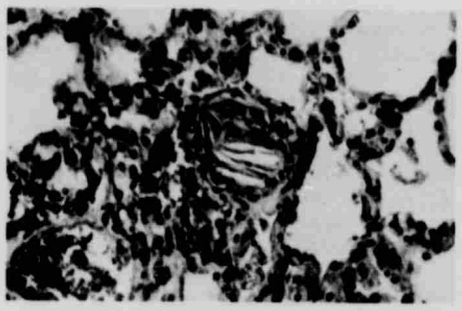


FIGURE 15-5. CHOLESTEROL-NEEDLE DEPOSIT WITH SURROUNDING CELLULAR INFILTRATION AND COLLAGENOUS FIBROSIS. THESE FOCI ARE RELATIVELY FEW AND WIDELY SCATTERED. HEMATOXYLIN AND EOSIN. X 390

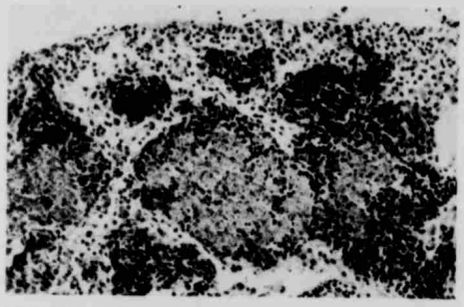


FIGURE 15-6. SATELLITE LYMPH NODE CONTAINING AGGREGATES OF WELL-PRESERVED MACROPHAGES FILLED WITH FIBER GLASS DUST. THERE IS NO FIBROSIS. HEMATOXYLIN AND EOSIN. X 190

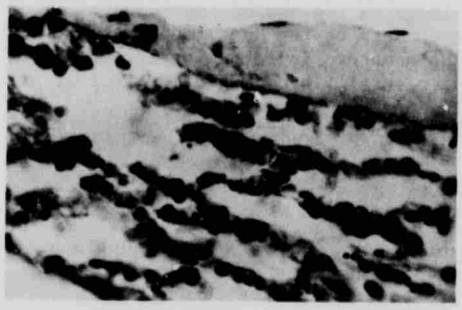


FIGURE 15-7. FOCAL BLAND THICKENING OF THE VISCERAL PLEURA. FROM A RAT THAT HAD INHALED FIBER GLASS DUST FOR 24 MONTHS. NOTE THAT THE SUBJACENT ALVEOLI ARE NORMAL AND CONTAIN NO MACROPHAGES. HEMATOXYLIN AND EOSIN. X 700

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The lungs of the hamsters that had inhaled glass dust showed grossly no stigma of pneumoconiosis. Microscopically, the tissue reaction to the fiber glass dust was similar to that observed in rats except that the macrophage accumulations were more numerous and more dense. Plentiful ferruginous bodies were seen in the air spaces. Calcific laminated alveolar microliths in a few hamsters represented an unusual sequel to the severe alveolar clearance failure previously found in this animal species. [Gross et al, 1967; Gross and de Treville, 1968] There was less lymphatic transport of fiber glass dust in hamsters than in rats. The satellite nodes were not enlarged, although clusters of dust-containing pale cells were evident in the lymphoid tissue. There was no stromal reaction to the presence of the phagocytic cells.

The pleura of a few rats and hamsters was focally thickened by bland, acellular collagenous fibrous tissue. Although an occasional dust-containing macrophage was noted in some of the subpleural alveoli, no glass fibers could be seen in the thickened pleura after microincineration (Figure 15-7).

Intratracheal Injection Series: Bronchial polypoid inflammation secondary to the technique of injection was noted soon after the injection in rats but not when they were killed 1 year or more later. Hamsters examined 6 months or later after the injections failed to show bronchial inflammation. The sequellae of severe alveolar clearance failure, i.e., the foci of cholesterol needle deposits with their associated septal fibrosis in rats, and the calcific alveolar microliths in hamsters, were not found in the animals injected intratracheally with fiber glass dust. Otherwise, the results of the intratracheal injections were similar to those observed in the animals that had inhaled the dust, but the number and size of the dust foci in the lungs of the injected animals were smaller than those in the lungs of animals in the inhalation series. There was no collagenous fibrosis of alveolar septa and only slight arborization of alveolar reticulin fibers in regions of macrophage accumulations. There was no fibrosis secondary to the dust deposits in the lymphoid tissue of either the rats or the hamsters injected intratracheally with fiber glass dust. Focal bland pleural fibrosis similar to that encountered in a few animals of the inhalation series was also observed, but with somewhat greater incidence in the injected animals.

Comments

A non-fibrogenic dust, formerly also designated as a biologically "inert" dust, has been defined as a dust that evokes a pulmonary tissue reaction having the following features [Gross et al, 1970a]:

1. The stromal proliferation is minimal and consists essentially of reticulin fibers.
2. The alveolar architecture remains intact.
3. The tissue reaction is potentially reversible.

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The pulmonary reaction to fiber glass dust has all of these features and therefore, this dust is appropriately categorized as a non-fibrogenic or nuisance-type dust.

The fact that none of the animals exposed to or injected intratracheally with fiber glass dust developed a lung or pleural tumor, stands out conspicuously against the background of experimental cancers produced by the implantation in the chest or the injection into the chest or abdominal cavity of large amounts of glass fiber dust (as well as other materials such as aluminum oxide, magnesium hydroxite, barium sulfate, and silica). Those who wish to ascribe a cancerogenic potential to inhaled fiber glass dust by extrapolation from the experimental production of chest tumors in rats, may find the occurrence of pleural fibrosis in some of the present experimental animals relevant to their viewpoint. However, to use the latter finding as support for this extrapolation would ignore important differences in the character of the pleural inflammation produced by the injection, or implantation of fiber glass dust in the chest cavity on the one hand and, on the other hand, the pleural inflammation found in some animals in which the dust was present in the air spaces of the lung. In the first instance, the inflammation involved both pleurae. It was violent, very cellular, extended into the skeletal muscle of the chest wall, and eventuated in cancer. Whereas, in the second instance, only the visceral pleura was affected. It was a self-limited, hypocellular, bland inflammation which healed without tumor production.

Although the presence or absence of focal atelectasis in relation to dust deposition may, at first glance, seem of minor significance, atelectasis is, nevertheless, of importance since the rate of clearance is greatly influenced by it. Collapse of alveolar walls around dust agglomerations tends to sequester them and thereby prevent their clearance. Furthermore, a dust that so affects nearby septa as to cause atelectasis, may be considered to have more biological activity than a dust that has no such effect. Lignite and steam-activated carbon dusts have been shown to be non-fibrogenic but nevertheless to cause focal atelectasis. [Gross and Nau, 1967] Inasmuch as fiber glass dust deposited in the lungs of rats and hamsters is not associated with either fibrosis or atelectasis, its biological activity as far as the lung is concerned, is less than that of lignite dust or steam-activited carbon dust.

The observation that dust foci in the lungs of animals that survived longest after the dust exposure were less numerous and smaller than those during, or shortly after the exposure is highly significant. It supports the observation made in the preliminary study that after a short-term exposure to 2000 mg/cu m of fiber glass dust, disproportionately few dust foci were found in the lungs of rats. It underscores the conclusion that the inhaled dust of glass fibers is subject to rapid removal by the pulmonary clearance mechanism. The rapid clearance of fiber glass dust is best explained by the failure of fiber glass dust to cause atelectasis which, in turn, is a reflection of the lack of noxiousness of fiber glass dust when in contact with the alveolar surface.

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AN INVESTIGATI
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Introduction

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AN INVESTIGATION OF THE IRRITANT PROPERTIES OF INHALED BETA-CLOTH FIBROUS DUST BOTH ALONE AND IN COMBINATION WITH LOW CONCENTRATIONS OF Cl 2 OR TRICHLORO-TRIFLUOROETHANE

Charles C. Haun

Introduction

DR. HOOVER: This paper will be presented by Mr. Charles C. Haun, who is the principal inhalation research toxicologist of the Toxic Hazards Research Unit, University of California, Dayton, Ohio.

Presentation

MR. HAUN: In 1969-70, at the request of the National Aeronautics and Space Administration, a series of experiments were conducted to evaluate the capability of Beta cloth dust to produce upper respiratory and nasal irritation or, more specifically, its ability to produce symptoms similar to the common cold. Beta cloth has been found useful in space flight programs, because of its nonflammability, for clothing and for flexibile ties to hold other materials in place. The Beta cloth uniforms and other items are woven from yarn consisting of bundles of glass fibers, approximately 2 to 3 μ in diameter and coated with a thin layer of Teflon. In use, the uniforms can be abraded resulting in the production of very small glass dust particles well within the respirable range. In space flight, small particles of Beta cloth dust can become suspended in the atmosphere indefinitely due to weightlessness and therefore may represent a potential source of respiratory irritation.

Since the actual space cabin exposures of men to the cloth dust could occur in conjunction with small amounts of other contaminants that could be irritating, the studies I'm reporting here were conducted using Beta cloth dust alone and, also, in the presence of chlorine or 1,1,2-trichloro 1,2,2-trifluoroethane (Freon 113). Why did we select chlorine and Freon 113 for this study? Chlorine is a recognized and, in this case, a selected model tissue irritant. Freon 113 is a common solvent contaminant in space cabins.

These exposures were conducted at the Toxic Hazards Division Facility of the Aerospace Medical Research Laboratories, Wright Patterson Air Force Base. We used Rochester Chambers with 20 cu ft/minute air flows while controlling temperature and relative humidity at desirable limits.

One to four rhesus monkeys were used in the 24-hour preliminary exposures to determine the threshold irritation levels of Cl 2 and Freon 113.

The next set of experiments consisted of exposure of groups of four monkeys for 7 days to the appropriately reduced threshold concentrations determined in the 24-hour exposures to establish the no-irritation levels of Freon 113 and Cl 2.

The third and final tests were to determine the 7-day irritating effect of Beta cloth dust alone and in combination with Freon or chlorine;

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examining the monkeys very carefully for any evidence of synergistic action. Groups of four monkeys were used in these tests. Groups of two monkeys each were used as controls in all 7-day experiments and were exposed to air only.

All monkeys used in this study had been carefully examined for any signs of upper respiratory tract disease before exposure. Symptomatology was recorded every hour during the course of exposure. At the conclusion of a test run, the animals were again examined for signs of ocular, nasal and oral mucosa irritation, then lightly anesthetized with sodium pentobarbital and submitted for immediate pathologic examination of both the upper and lower respiratory tract.

Beta fiber yarn coated with Teflon was ground in a ball mill as a water slurry using 1-inch steel balls. After 5 days grinding, the slurry was cleaned by acid treatment to remove iron originating from the mill and balls; then filtered and washed. Subsequently, sedimentation techniques were used to collect particles in the 3-7 μ size range. The dust particles were then dried for use in animal exposures.

The dust exposures were generated using an air elutriator. The reservoir was filled with dust and replenished when necessary. A constant speed timing motor coupled to a five-turn coiled rod produced a continuous delivery of glass fiber dust to the loop delivery system. Effluent air from the shaded pole blower motor carried the dust fibers through the piping to the exposure chamber. A variable-voltage transformer provided a means of controlling the blower motor over a wide range of speeds, thus ensuring the capability to maintain the desired exposure chamber dust concentration of the required particulate size. (Dr. Gross supplied the design information for the generator.)

Continuous measurement of the chamber dust concentration was made with a dust photometer coupled to a variable speed recorder. This instrument measures the forward scattering of light from particulate matter drawn continuously through a dark field illumination chamber. The dust concentration selected was 15 mg/cu m. This is the ACGIH threshold limit for nuisance dusts.

A Mast Ozone Meter was calibrated to measure 0-5 ppm Cl 2 and used for analysis of chamber concentrations. The Freon 113 delivery system consisted of a dual syringe feeder which supplied a precalculated flow of liquid to a glass helix. The vapors were then swept into the exposure chamber by 8 CFM carrier airflow passed through the glass evaporator. A gas chromatograph provided analysis of chamber concentration of Freon 113.

Results

In the 24-hour preliminary experiments, separate groups of male monkeys exposed to measured Freon 113 concentrations of 2,040 and 3,575 ppm showed no signs of irritation during exposure or at postexposure necropsy. On the basis of this information, 2,000 ppm appeared to represent a suitable level for purposes of the 7-day test. The 7-day exposure was then conducted. The mean measured concentration was 1,920 ppm Freon 113. No

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abnormalities were noticed during exposure and pathological findings were completely negative.

In regard to the chlorine exposures, definite signs of irritation were seen in monkeys exposed to selected concentrations of 5 and 1 ppm chlorine for 24 hours. The indications of irritation from the exposures, although differing in degree and onset, included lacrimation, salivation, emesis and frequent gasping. Gross examination of the respiratory tract showed hyperemia of the tracheal and bronchial mucosa.

On the basis of the preceding information, a dose level of 0.1 ppm Cl 2 was selected for the 7-day exposure. Accordingly, four monkeys were exposed to a mean concentration of 0.11 ppm for this time period. No symptoms indicative of irritation were observed during or immediately after exposure.

Groups of four monkeys each were exposed continuously for 7-day periods to each of these environments. The animals were observed hourly for signs of irritation throughout the exposure and their entire respiratory tracts examined at necropsy. There was no evidence of irritation from the Beta cloth fiber dust either singly or in combination with Cl 2 or Freon 113 at the levels tested. Histopathologic examination of the nasal passages and respiratory airways failed to show any differences between the exposed monkeys and their controls.

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RESULTS OF ANIMAL CARCINOGENESIS STUDIES AFTER APPLICATION OF FIBROUS GLASS AND THEIR IMPLICATIONS REGARDING HUMAN EXPOSURE

F. Pott
F. Huth
K.H. Friedrichs

Introduction

DR. HOOVER: The next paper has been written by Dr. F. Pott and Dr. F. Huth, who are from the Medical Institute for Air Hygiene and Silicosis Research, University of Dusseldorf, in the Federal Republic of Germany. The paper will be presented by Dr. Huth. Dr. Pott is in the audience and will participate in the question and answer session.

Presentation

DR. HUTH: Until 2 years ago, there was the general opinion that no other fibers except asbestos would lead to tumors. In 1972, Stanton and Wrench published data about rats with intrapleural implantation of 40 mg fiber glass of very small diameter resulting in 8 tumors out of 54 animals. Grained glass particles induced tumors at a significantly lower frequency. At the same time, we reported a result of 23 tumors within a collection of 40 rats following intraperitoneal injection of 100 mg fiber glass (type S + S 106). [Pott and Friedrichs, 1972] Fibrous magnesium hydroxide (mineralogically nemalite), as well as chrysotile, induced tumors. In contrast, dust with granular shape like biotite, haematite, sanidin, talc, as well as grained fibers of actinolite or pectolite led to tumors only singularly or were not followed by any tumor. Table 17-1 offers a survey on the injected dusts and on the rates of tumors. These results have been published before. [Pott and Friedrichs, 1973]

Newer Investigations and their Results

The experiments were made mainly with fiber glass of type MN 104 and type MN 112. The curves of distribution in length and diameter of 1000 fibers per sample were evaluated from microphotos. Of type MN 104 fibers, 50% measured < 0.2 μ in diameter and 50% of the fibers had lengths < 11 μ (Figure 17-1). The corresponding values for the fiber MN 112 were 1 μ in diameter and 28 μ in length (Figure 17-2). Both types of fibers were uncoated. Up to 25 mg of the fibrous dusts were suspended in 2 ml of 0.9% saline. The suspension was injected in Wistar rats by one or two portions intraperitoneally. Dosage and results are summarized in Table 17-2. The stated tumor rate of each group is not the final one since some of the animals are still alive after 2 years. Up to now 2 mg of fiber glass MN 104, as well as 2 mg UICC crocidolite, induced tumors in 10% of the animals. The tumors arose mainly in the abdominal cavity. Some tumors were observed in pleural or pericardial cavities. With the dosage of 2 mg only a slight fibrosis developed.

Histologically nearly 90% of the tumors turned out to be mesotheliomas (Figure 17-3). The remaining tumors were spindle cell sarcoma or polymorphocellular sarcoma. Microscopically, there did not exist any

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TABLE 17-1

TUMORS AFTER INTRAPERITIONEAL INJECTION OF DIFFERANT FIBROUS AND GRANULAR DUSTS

Dust		form	Dose ip mg	First animal with tumor after ... days	Tumor rate % from 40 rats
Chrysotile	A UICC	f	2	431	15.0
Chrysotile	A UICC	f	6	343	67.5
Chrysotile	A UICC	f	25	276	62.5
Chrysotile	A UICC	f	4 x 25	270	37.5
Chrysotile	A UICC	f	4 x 25	400	30.0
Chrysotile	A UICC (milled)	f	3 x 25	257	65.0
Palygorscite		f	4 x 25	249	62.5
Nemalite		f	2	762	2.5
Glass fibers (S+S 106)		f	10	350	10.0
Glass fibers (S+S 106)		f	4 x 25	197	57.5
Glass fibers (S+S 106)		f	4 x 25	546	5.0
Gypsum		s	4 x 25	569	2.5
Pectolite		s	4 x 25	579	2.5
Sanidine		s	4 x 25		
Talc		s	4 x 25		

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TABLE 17-1 (continued)

Gypsum	f	4 x 25	546	5.0
Pectolite	g	4 x 25	569	2.5
Sanidine	g	4 x 25	579	2.5
Talc	g	4 x 25	587	2.5
Actinolite	g	4 x 25	-	-
Biotite	g	4 x 25	-	-
Haematite (precipitated)	g	4 x 25	-	-
Haematite (mineral)	g	4 x 25	-	-
NaCl - Control	-	4 x 2ml	-	-

Abbr.: f = fibrous; g = granular

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difference between tumors following injection of fiber glass and those after injection of asbestos.

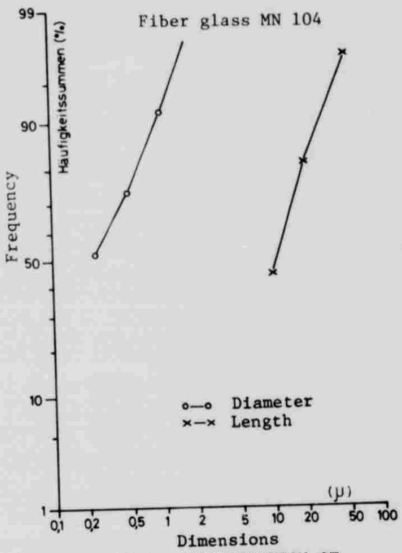


FIGURE 17-1. DISTRIBUTION OF DIAMETER AND LENGTH OF FIBROUS GLASS MN 104

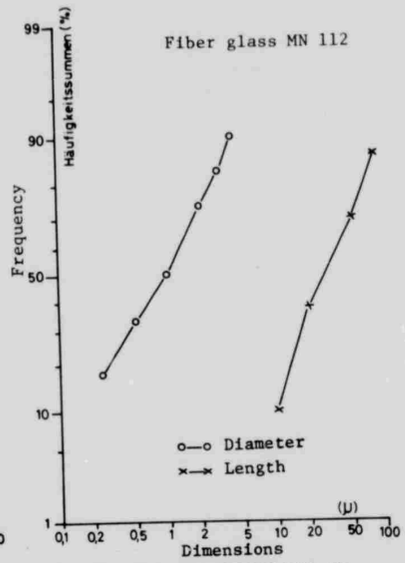


FIGURE 17-2. DISTRIBUTION OF DIAMETER AND LENGTH OF FIBROUS GLASS MN 112

NMRI mice developed comparable tumor rates following intraperitoneal injection of fiber glass, nemalite, UICC-chrysotile, and UICC-crocidolite. However, in some groups, the tumor rates did not reach the same degree as observed in the rats. Hamsters and guinea pigs did not develop tumors that could be related to the injection of fibers, neither those of glass nor those of chrysotile.

Conclusions

The carcinogenic properties of asbestos depend on its fibrous shape. The investigations did not reveal any co-carcinogenic effect of the chemical composition of the injected dusts. Non-asbestos fibrous dusts can induce tumors as it has been proven for asbestos. The main precondition is the small diameter of the fibers and the time the fiber can be effective within one area.

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Dust
Dose mg ip in 2 ml saline
Number of rats at the start
AFTER 24 MONTHS
Surviving rats
% of rats with tumor
HISTOLOGIC DIAGNOSIS
Mesothelioma
Spindle cell sarcoma
Polymorphocellular sarcoma
Carcinoma
Reticulocellular sarcoma
Survival time of first rat with tumor days

*Spontaneous tumor, not

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TABLE 17-2

TUMORS AFTER INTRAPERITONEAL INJECTION OF FIBROUS GLASS, CROCIDOLITE AND CORUNDUM IN RATS (unfinished experiment about 24 months)

Dust	Fiber Glass MN 104	MN 104	MN 104	MN 112	Crocidolite UICC	Corundum
Dose mg ip in 2 ml saline	2	10	2 x 25	20	2	2 x 25
Number of rats at the start	80	80	80	40	40	40
AFTER 24 MONTHS						
Surviving rats	38	21	-	9	17	25
% of rats with tumor	10.0	33.8	68.8	27.5	10.0	2.5
HISTOLOGIC DIAGNOSIS						
Mesothelioma	7	23	47	10	4	1
Spindle cell sarcoma	1	3	6	1	-	-
Polymorphocellular sarcoma	-	-	2	-	-	-
Carcinoma	-	1	-	-	-	-
Reticulocellular sarcoma	1*	-	2*	-	2*	1*
Survival time of first rat with tumor days	576	210	194	390	452	545

*Spontaneous tumor, not valued

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Problems of Dose Response Relationship of Fibrous Dusts

Regarding the carcinogenic effect of the fibrous shape in the dusts, there arise particular difficulties concerning the determination of injected doses being still effective. We have predicted that the fibrous shape is the only carcinogenic agent; in addition, all fibers should be cylindric. Therefore, the most important factor for a carcinogenic dose would be the amount of fibers (disregarding their weight). Of course, limits in total size of the fibers concerning carcinogenicity have to be expected. Beck and coworkers [1972] observed the contact of fibers and cells within cell cultures; according to their investigations, the effective length and diameter of the fibers ought to range between sizes that damage individual cells due to partial phagocytosis. With respect to cellular diameters, the effective diameter of fibers may be estimated to be 1 μ at maximum. Most of the used fiber glass measured less than this value. Animal experiments by Gross and coworkers [1970] with fibers around 1 - 3 μ in diameter led to the assumption that these fibers were not carcinogenic.

These experiments will hardly answer the open questions, since the dosage was rather low. Regarding the fibrous shape as the main tumorigenic factor, the dose is dependent upon the amount of fibers. That means, if 2 mg of fibers measuring 0.3 μ in diameter induced tumors in 10% of the animals, fibers with 3 μ in diameter have to be injected in a dosage 100 times higher to get an analogous rate of tumors. This concept requires constant lengths of the fibers.

However, the dose response relationship has to be regarded as even more complicated. Probably it means a simplification to assume that all those fibers proven as carcinogenic are of the same effectiveness. For instance, a specified diameter could be of maximal effect, since an increase or decrease of diameter could cause less tumorous alterations. Up to this day, the importance of irregularities in shape and diameter of the fibers remains uncertain. According to Edwards and Lynch, [1968] a fiber has been defined as a particle three times longer than it measures in diameter. It has to be clarified if this relation is a correct one regarding carcinogenicity of the fibers.

In addition, the length of the fibers includes a limiting effect. The upper limit should be the length of a fiber that still allows inhalation and deposition into the alveoli. The lower limit should be below 10 μ since experiments with grained chrysotile shorter than 5 μ in 99.8% of the specimen still caused tumorigenicity. That could be explained either by the remaining 0.2% of longer fibers or by a carcinogenic effect of fibers being shorter than 5 μ. Further experiments have to reveal the dose response relationships of the number of fibers, the length of the fibers, and the diameters of the fibers. Experiments were started with fibers measuring constantly 3 μ and 5 μ in diameter. In order to estimate the shape of fibers with different sizes in correlation to the counts of fibers per unit of weight, a nomogram has been developed (Figure 17-4). In the case of known length of fiber diameter, the amount of fibers/mg of dust can be calculated corresponding to the densities 1 or 2.3 for glass. According to the assumption that within one specimen the diameter and

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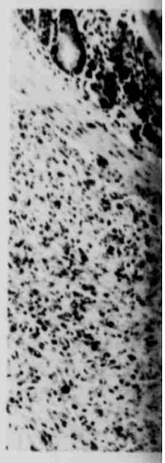


FIGURE 17-3:
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lengths of fibers are nearly constant, 1 mg of fiber glass measuring 0.5 μ in diameter and 10 μ on the length in average should contain 200 million fibers.

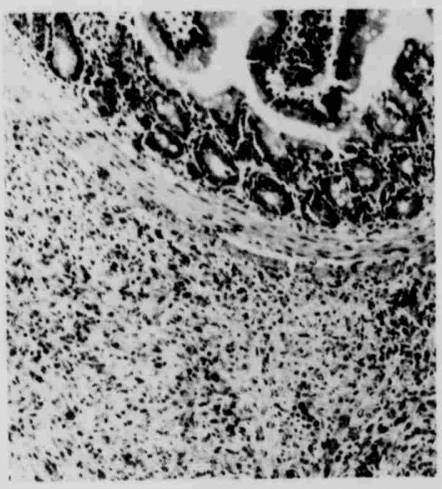


FIGURE 17-3: MESOTHELIOMA OF THE INTESTINAL WALL WITH PARTLY EPITHELIAL PARTLY SARCOMATOUS PATTERN FOLLOWING INTRAPERITONEAL INJECTION OF 10 MG FIBROUS GLASS MN 104. 125x

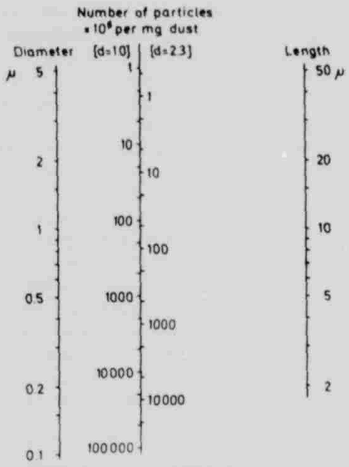


FIGURE 17-4: NOMOGRAM FOR ESTIMATION OF NUMBER OF PARTICLES PER 1 MG FIBROUS DUST WITH THE PROPORTIONS OF DIAMETER AND LENGTH IN CYLINDRICAL PARTICLES WITH DENSITY 1.0 AND 2.3

Significance of Experimental Results to Human Pathology

The importance of our previously published results, as well as those of Stanton and Wrench, [1972] has been called irrelevant concerning carcinogenicity in men. There were three essential arguments against the significance of the results:

1. The applied dose is much higher than those amounts that could reach effect within the human organism.

2. As a general principle, experimental models leading to tumors by foreign bodies are not of any significance for men.

3. Fiber glass has been produced for 50 years. Any increase of tumors in workers who inhaled fiber glass should have been proven in the meantime. Former investigations did not prove any severe damage to health from fiber glass.

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These arguments could be answered by the following observations: 2 mg of fiber glass, chrysotile, or crocidolite have been proven as carcinogenic within the referred tests with rats and mice. The lowest effective dose in animals has not been reached, therefore. However, the low dose injected during the referred experiments seems to be comparable with amounts incorporated by men.

Sparse tumors following application of non-fibrous inert dusts, as also observed by Wagner and coworkers, [1973] could be explained by a high dosage. In addition, huge amounts of granular dusts could contain a small portion of fibrous particles.

The results of injections of fibrous dusts in rats may not be compared with those observed by Oppenheimer and coworkers [1948] as well as Nothdurft [1955] after implantation of foreign bodies. These foreign bodies led to tumors only in larger pieces with flat surfaces; in granular condition, they were not able to cause tumorous alterations. Fiber glass and asbestos yet have been controlled simultaneously to granular dusts of comparable chemical composition and size of particles, as demonstrated before. After application of equal weights, there resulted enormous differences in tumor rates in favor of those after fibrous dusts.

Finally, we should like to comment on the argument concerning the negative epidemiological results in men. In this respect, one has to point out that the production of fiber glass measuring less than 5 μ did not start before 1961 in Germany. Since that year, there exists a progressive tendency to produce fiber glass with smaller diameters. Fiber glass less than 1 μ in diameter has been inhaled only in the past 10 years in Germany. Manifestation of mesotheliomas is expected no earlier than 20 years following inhalation of fibers with very small diameters. Consequently, no increase of frequency of tumors has been observed. The proof of significance of the results in animal experiments probably will not be possible before 1985.

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STUDIES OF THE CARCINOGENIC EFFECTS OF FIBER GLASS OF DIFFERENT DIAMETERS FOLLOWING INTRAPLEURAL INOCULATION IN EXPERIMENTAL ANIMALS

J. C. Wagner
Geoffrey Berry
J.W. Skidmore

Introduction

DR. HOOVER: The next paper will be presented by Mr. Geoffrey Berry, associate of Dr. Wagner, who is a statistician in the Medical Research Council, Pneumoconiosis Unit in Penarth, Wales.

Presentation

MR. BERRY: In addition to Dr. Wagner and myself, another name associated with this paper is Mr. Joe Skidmore. Dr. Timbrell, who spoke yesterday, has been responsible for the size determinations of the fibers which I shall mention.

For a number of years, we have been engaged in experiments in which rats have been inoculated intrapleurally with asbestos and have shown that mesotheliomas, similar to those seen in human cases, with the same spectrum of histological pattern, occur following this treatment. [Wagner and Berry, 1969] Our more recent experiments have led us to the tentative conclusion that a major characteristic of asbestos responsible for its carcinogenicity is its fibrous nature, and that the finer fibers are the more carcinogenic. [Wagner, Berry and Timbrell, 1973] Accordingly, we have carried out experiments involving fiber glass, even though we were not interested in this material per se, and in this paper the results are presented of an experiment using two samples with markedly different fiber diameter distributions.

Material and Methods

Samples of commercially available Johns-Manville micro-fiber Code Nos. 110 and 100 were obtained. Since these samples were composed mainly of very long fibers, it was necessary to reduce the fiber length for our experimental purposes. Normal grinding processes tended to destroy the fibrous nature of the glass and the fiber length was therefore reduced by cutting in a microtome. To do this, the fiber was embedded in a water soluble wax, and the block was cut into slices 5 μ thick which were transferred to warm water. Multiple washing was necessary to remove all the wax from the fibers.

The fiber diameter and length distributions were determined by electron microscopy. For the finer sample (Code 100) 99% of the fibers had diameters of less than 0.5 μ, and the median fiber diameter was 0.12 μ. For the coarser sample (Code 110) only 17% had diameters of less than 1 μ, and the median fiber diameter was 1.8 μ. Since the fibers took up a range of orientations in the wax block, the microtome cutting did not reduce all the fiber lengths to less than 5 μ. The coarse sample had a median length of 22 μ and 10% of the fibers were longer than 50 μ. The fine sample had a

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median length of 1.7 μ and only 2% of fibers were longer than 20 μ. Fuller details of the size distributions are given by Timbrell. [1974]

The experimental animals were barrier protected caesarian derived rats of the Wistar strain bred at the unit from stock originally given to us by Imperial Chemical Industries, Pharmaceutical Division, Alderley Edge, Cheshire. Ninety-six rats, 48 of each sex, were randomly allocated to one of three treatment groups, which were inoculated intrapleurally with one of the two samples of glass fiber or with saline solution only for the control group. The dose per treated rat was 20 mg in 0.4 ml saline. The method of inoculation was as given previously [Wagner and Berry, 1969] and as in our previous experiments the rats were allowed to live out their lives, or were killed when they appeared distressed. At death a full necropsy examination was carried out on each rat. The experiment started in February, 1971 when the rats were 10 1/2 weeks old and the last rat died in November, 1973.

Results

The mean survival and the number of mesotheliomas found at death are given in Table 18-1. Mesotheliomas occured only with the finer fiber glass (Code 100) and the four rats with this type of tumor died between 663 and 744 days after inoculation. The lung sections of the rats in the glass fiber groups were examined in more detail and the degree of hyperplasia in the mesothelial cells assessed on a 7 point scale, ranging from no change to a mesothelioma. Because of the wide difference in diameters of the fibers, which were visible on microscopic examination, it was not possible to make this assessment in ignorance of which sample had been injected. The results are given in Table 18-2. For the coarse fiber glass (Code 110) hyperplasia was observed up to the generalized category. However, with the finer fiber glass, in addition to the four mesotheliomas there were seven rats with marked hyperplasia and one in which there was a suspicion of malignancy. Only 1 rat injected with the finer fiber glass showed no signs of hyperplasia compared with 12 of those injected with the coarse fiber glass.

Discussion

Before summarizing, two points must be emphasized: First, the materials were applied by injection into the pleural cavity and therefore the difference between the carcinogenic effects reported have nothing to do with the different aerodynamic properties of the samples. The comparision is between the carcinogenicities of the materials given that are in contact with the pleura. Second, a fixed mass was injected, and the number of fibers in this fixed mass would vary 1000 fold between the two samples: 30 billion fibers for the finer fiber and 30 million for the coarse fiber. [Timbrell, 1974]

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TABLE 18-1

MEAN SURVIVAL AND NUMBER OF MESOTHELIOMAS FOUND AT DEATH

	No. of rats	Mean survival after injection (days)	No. with mesothelioma
Glass fiber 100	32	716	4
Glass fiber 110	32	718	0
Saline control	32	697	0

TABLE 18-2

DEGREE OF HYPERPLASIA IN MESOTHELIAL CELLS

	Glass fiber 100	Glass fiber 110
Nil	1	12
Occasional	3	5
Focal	12	11
Generalized	4	4
Marked	7	0
Suspicion of malignancy	1	0
Mesothelioma	4	0

The coarser fiber glass (Code 110) was used in one of our earlier experiments and no mesotheliomas were observed in 35 rats. [Wagner, Berry, and Timbrell, 1973] Thus, in total, no mesotheliomas have occurred in 67 rats injected with this material, and in contrast, the 4 mesotheliomas in 32 rats injected with the finer samples are significant ($p = 0.01$). In the earlier experiment a sample of non-fibrous glass powder was also used. This was a borosilicate and was all in the respirable range and all

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particles had projected diameters of less than 8 μ. Out of 35 rats injected, there was 1 which developed a mesothelioma.

Our results are not contradictory with those of Stanton and Wrench [1972] who applied samples of fibrous glass to the pleura of rats with a dose of 40 mg, double our dose. They found 4 mesotheliomas out of 91 rats exposed to coarse fibers, which did, however, include some fibers with diameters less than 0.5 μ, and 8 mesotheliomas out of 54 rats exposed to a fine sample, diameter range 0.06 - 3.0 μ.

Finally, it is of interest to compare our results with fiber glass with those obtained with various types of asbestos and other materials in our earlier experiments using identical techniques. [Wagner, Berry and Timbrell, 1973] All the asbestos samples we have used produced more mesotheliomas than the finer glass fiber (Table 18-3) and some of the asbestos samples are much more carcinogenic.

TABLE 18-3

PERCENTAGE OF RATS DEVELOPING MESOTHELIOMAS
AFTER INTRAPLEURAL INOCULATION OF VARIOUS MATERIALS

Material	Percentage of rats with mesotheliomas
SFA chrysolite	66
UICC crocidolite	61
UICC amosite	36
UICC anthophyllite	34
UICC chrysotile (Canadian)	30
UICC chrysotile (Rhodesian)	19
Glass fiber code 100	12
Ceramic fiber	10
Glass powder	3
Glass fiber code 110	0

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DISCUSSION

DR. HOOVER: We shall now have a question and discussion session.

MR. DEMENT: I have a question for Dr. Gross. In your inhalation experiments, you made a statement that your average fiber diameter was 1 μ and that most of your lengths were less than 50 μ. In the paper presented by Dr. Kuschner, he found that fibers longer than 10 μ produced fibrosis.

In your paper, you stated that most of your fibers were less than 50 μ. My question is, what percent of your fibers were actually longer than 10 μ?

DR. GROSS: The figures that you quoted refer to the length and diameter of the fibers before they entered the chamber. The length of the fibers in the chamber were in the range of 5 to 20 μ, and the diameter was within the very narrow range of 0.5 μ.

MR. DEMENT: Could you estimate what percent were greater than 10 μ?

DR. GROSS: The only information I have is that the range of the fibers in the chamber was 5 to 20 μ.

DR. CORN: I should like to ask the entire panel to think out loud. With all the caveats you have given us about the experiments performed by injection into the pleural and peritoneal cavities, what do they mean when the disparity between response of these experiments and the inhalatory route appear to be what they are? How should we weigh and interpret these findings? And certainly, Dr. Kuschner, you stressed the unrealistic nature of this mode of experimentation. I can't put these results in perspective.

DR. KUSCHNER: I think there is one link in the chain which is missing. We must have good data on what determines deposition and retention in the lung. We are beginning to hear some very sharp definitions of what the characteristics of a fiber placed in the pleura has to be in order to induce mesothelioma.

Now the question is, what is the intervening step? I think everyone would be willing to grant that certain fibers do induce mesothelioma when inhaled. We have to presume that they do so by getting into the pleura. It is conceivable that you have a curve of optimum particle size or fiber size that determines deposition and another curve of optimum size that determines carcinogenesis. It might be a quite different and altogether detached curve that determines translocation into the pleura. We still are missing one piece of information that would relate the intrapleural injection to the inhalation. Perhaps somewhere these three curves overlap and that is the area we should be looking at.

DR. GROSS: We were interested in that very point, namely, to what extent do inhaled glass fibers appear in the pleura? For that purpose, we subjected the lungs of our animals to microincineration in an attempt to visualize the presence of the glass fibers in or near the pleura. We were unable to demonstrate in our animals that any glass fibers were in or near the pleura.

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DR. DAVIS: I should like to agree that the important thing we have to discover about glass fiber is how much of it can reach the pleura after inhalation. Perhaps none of it can. In this case, we might not have trouble with pleural mesothelioma in human beings. We have shown what can happen with glass if you bypass all the essential routes and get it as far as the pleural cavity. We need information about whether it can get there on its own under normal circumstances.

DR. HUTH: I shall try to explain our idea about those things. We know that asbestos fibers can reach the pleura in men and can induce mesothelioma. By our experiments, we could prove that we get the same results by injection. Now, if you try to get glass pattern needles into the pleura by inhalation, you have to think about the long way they have to pass. Most of them take the way through the interstitium to the lymph nodes. Most of them get out by the normal way, of course. The other part reaches lymphatic tissue. But only some of them, and I can imagine that they are nearly undetectable by normal histological investigations, have to reach the pleura to induce that process. We could demonstrate by the electromicrograph the fight between an individual mesothelial cell and one fiber.

MR. BERRY: I can remember the same criticism being made about 10 years ago. It was said that the results of our experiments with asbestos had no meaning at all. Time has proved these experiments are of meaning, and that intrapleural inoculation is a valuable experimental technique. Of course, we don't claim that this is the only technique; we do inhalation experiments as well. Our opinion at the Pneumoconiosis Unit in Wales is that both types of experiments are useful. The results generally complement one another. Inhalation experiments are more expensive to carry out than injection experiments. That is one good reason for continuing with intrapleural inoculation or injection experiments by other routes.

Another reason is to disentangle different effects. With an inhalation experiment, there are several possible effects. One is the biological effect of the dust, given that we can get it to where we want it to get to. Another is the aerodynamic properties of the dust which determine how much of the dust reaches the target. By injecting the material directly into the target you can eliminate the second factor. Therefore, inhalation experiments are necessary. Studies of aerodynamic properties, from which one could predict what proportion of the dust would reach the target area, are important, too.

DR. GROSS: Mr. Chairman, may I make a comment on Dr. Huth's comment? Dr. Huth stated that glass fibers are difficult to visualize histologically. That is correct only insofar as the normal histological section is concerned. On microincineration, glass fibers become easily visible, particularly under dark field illumination. Our examinations were conducted on slides that had been subjected to microincineration.

I do not know the reason why glass fibers become easily visible. All I know is that they do become readily visible with microincineration.

I should like to comment also on Dr. Kuschner's presentation and ask a question. Which type of glass fiber is relevant to human experience?

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Our results with inhaled glass fibers are very similar to Dr. Wright's and Dr. Kuschner's results with short glass fibers in animals. However, our results differ markedly from the results that Doctors Wright and Kuschner obtained with the longer fibers.

I should like to propose an explanation for the difference in results, and I am wondering whether Dr. Kuschner will agree. Namely, with the longer fibers, as he has indicated, there is a tendency for the fibers to mat. Consequently, clearance of these fibers would be much more difficult than clearance with the short fibers. With the failure of clearance, prolonged irritation could be present and the fibrosis result.

DR. KUSCHNER: I think we would agree that there is difficulty in managing the long fibers in terms of intratracheal injection. I believe that Dr. Gross would agree, too, that the critical experiment is to do inhalation with fibers that are longer than the ones he used. There is some concern that they may actually have been in the category of short fibers comparable to our short fibers. The pictures he pointed out were remarkably similar to ours, especially the short fiber pictures.

I think the issue here is really what question you are asking. This relates to the problem of intrapleural inoculation as well. If you are asking whether a material in contact with tissue can induce malignant transformation, then you are entitled to use a variety of models to answer that question. If you are asking if a material which is inhaled is hazardous, then you are asking a different series of questions.

Now I think our experiment answers one kind of question, and that is that a long fiber is more fibrogenic than a short fiber. My own view of why this is so relates to what Dr. Huth showed you. The incomplete encompassing of a fiber by the macrophage results in the release of a number of substances from the macrophage. Dr. Botham referred to this in her reference to lactic dehydrogenase. There also are a whole variety of proteases that are released which induce tissue damage, and indeed these proteases may even alter cell surfaces. We can conceive of a variety of carcinogenic mechanisms. The fact that the fiber is long and incapable of being completely emcompassed by the cell, may be a very important determinant of the release of endogenous mediators of a whole variety of tissue alterations, from cell death to dissolution of elastic tissue to fibrosis to carcinogenesis.

DR. GROSS: The question of relevance is still up in the air. I think perhaps some indication of relevance can be obtained from the data derived from human lungs. The average length of the fibers we found in human lungs was in the neighborhood of 20 μ. In more than 100 cases of fibers found in human lungs, the vast majority of them were in the neighborhood of 5-10 μ. When we talk about the relevance of these experimental investigations to human experience, we are referring to animal experiments which are relevant to human lungs as those involving the shorter fiber length. These consist of fibers that under the optical microscope look like vitreous fibers.

DR. HUTH: To control the lung and the pleura for the content of small short fibers you have to do thousands of serial sections before you

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can tell there are no fibers within the pleura. You have to consider that there are different lymphatic pathways through the pleura through which the fibers could pass. There are a variety of individual characteristics that could cause deposition of those small and short fibers.

I would be very careful considering animal lungs or human lungs before I would say whether there are any fibers within the pleura. One rat lung needs 3,000 to 5,000 serial sections to be controlled before saying there are no fibers.

MR. BIERBAUM: Dr. Gross mentioned that he hasn't seen any fibrous glass in the pleura after animal inhalation studies, is that correct?

DR. GROSS: That is correct.

MR. BIERBAUM: Then we found that due to injection into the pleura of fibrous glass we found mesothelioma. Is that correct? Have we found asbestos in the pleura of the animals after the asbestos inhalation studies?

DR. GROSS: I should like to answer Dr. Huth first. I did not say that there were not any fibers in the pleura. I said I did not find any. Two different things.

The fact that I did not find any does have significance insofar as it implies that if any fibers were present, they were not sufficiently numerous to be detectible with an optical microscope. This is in contrast to the massive number of fibers which were injected into the pleura and which were necessary for the production of intrapleural or intra-abdominal tumors. Asbestos of course, would be much more difficult to detect because it has a finer diameter than glass. However, I did not make a particular search for it, so I am unable to answer that question. Maybe the other participants can.

MR. BIERBAUM: Isn't that a pretty critical point though? If we don't find fibers but we still find mesotheliomas, it seems likely that there is a connection there.

DR. DAVIS: I think the point here is that you can get a very high percentage of mesotheliomas by the injection techniques. The percentage produced by inhalation in experimental studies has been very, very small. Therefore, it has not been possible to show this transmigration of fiber out to the pleural cavity with any certainty.

MR. BERRY: I will just say we found very few mesotheliomas after inhalation. We did find quite a high proportion of other known lung tumors, varying from benign adenomas to malignant squamous carcinoma and adenocarcinoma. Although the mesothelioma rate is down with inhalation of asbestos, we produced lung cancer which, of course, agrees with the finding in man.

DR. DAVIS: Yes, it would appear that you get a very, very low incidence of tumors after inhalation in experimental animals. As far as we can see, this happens with humans as well. There must be a factor

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affecting, perhaps, the transport of the fiber out to the pleural cavity which doesn't happen with everybody, or other factors that we don't understand.

The relationship between the inhaled dose of fiber seems much more certain when you are dealing with bronchial carcinomas than when you are dealing with mesotheliomas.

DR. LIPPMANN: The intratracheal instillation technique allows convenient experimental delivery of fibers to target tissues appropriate to the induction of mesothelioma. The availability of this technique has stimulated the performance of a number of excellent papers in this session. But with asbestos there is another condition, bronchial cancer. Would any member of the panel be willing to speculate on the probability of bronchial cancer arising from the inhalation of fibrous glass? If this is an area of real concern and something we have to think about in terms of sampling of the appropriate particles, what kind of biological test system might be appropriate for such testing? We may be lacking this ability to test this condition as we have for mesothelioma.

DR. KUSCHNER: I will take a crack at that. It's a very dangerous question. I, myself, have a strong feeling that the carcinogenicity of asbestos is related to its fibrogenicity, at least with regard to the lung. This may not be entirely true of the pleura on the basis of what we heard today. But, within the lung, the striking feature is that one gets intense epithelial proliferation that accompanies the fibrosis in the standard honeycomb lung.

It is also curious that there is often a necessary additional condition. Most of the cases reported in asbestos workers have been associated with cigarette smoking. One wonders if the carcinogen is not really the cigarette in this instance and the asbestos simply provides a suitable substrate. Indeed, one might wonder if the form of the cancer produced by asbestos might not be different from what one sees in the ordinary cigarette smoking individual who is not exposed to asbestos.

In 1972, Jacob Cher did a study on the histologic types of cancer seen in asbestos workers as compared to a control group. He came to the conclusion that they were identical. They were very curious groups, because there was an excess of adenocarcinoma in both of these groups. I don't know how he chose his control group, but they curiously had only about 22% or 25% squamous cell carcinomas.

I still wonder if there isn't a real necessary relationship with asbestosis. That is the presence of fibrosis within the lung, and concomitant epithelial proliferation as the background, against which carcinoma develops in the asbestos worker who smokes.

DR. GROSS: Molly Newhouse, in her statistics, has clearly indicated that the asbestotic noncancer occurs only in those cases where the amount of exposure or the degree of asbestosis was more than moderate, thus conforming with Dr. Kuschner's contention.

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DR. DAVIS: I think the question basically was what sort of a test system do we need to check up on the possibility of glass fiber producing bronchiogenic carcinoma in human beings. I think the answer is much, much simpler than the mesothelioma problem. Inhalation experiments with asbestos have produced bronchogenic carcinoma in experimental animals. We know that asbestos produced bronchogenic carcinoma in human beings. Therefore, on the asbestos side we seem to have a perfectly satisfactory test system, straightforward inhalation studies.

Inhalation studies performed so far with glass fiber have not shown bronchogenic carcinoma. Maybe you can say there haven't been enough studies done at present. Perhaps that's true. but, I think the indication is that the test system will work. In other words, we need some more inhalation studies with glass fiber, and if these don't produce bronchogenic carcinoma, then I think this will prove true for human beings.

MR. SMITH: I have a question for Dr. Huth. If I understood you correctly, you told us that Germany did not start making fine fibers til about 1961 and that you felt it would be 1985 before the results on people would be meaningful. We started making them in 1940 or 1941. We produced the material for flotation fiber in substantial quantities in 1945, and that was an average of 1 1/2 μ.

DR. HUTH: May I ask directly, were the fibers in diameter below 1 μ?

MR. SMITH: Some were.

DR. HUTH: How could you measure that in 1941? By which instrument did you measure that?

MR. SMITH: I couldn't answer that at this moment, but I could find out and tell you.

DR. DENEE: I have a comment for the panel. In the light micrographs of the lung tissue it was stated that fiber glass fibers are very difficult to see and one has to relate to Transmission Electron Microscope (TEM) work and serial sections. I have been pioneering a technique for use with the scanning electron microscope whereby fiber glass fibers that are approximately 0.1μ, can be seen in sections on the order of 5 to 7 μ thick. It's through the use of back scatter electron imaging.

DR. UTIDJIAN: To settle this issue of whether inhaled glass fibers can reach the pleura or not, I am wondering whether it would be possible, by a partial digestion technique, to strip off the visceral pleura and do whole mounts rather than thousands of transverse sections. Also, would it be possible to ask the manufacturers of fibrous glass to produce a deeply pigmented glass for this purpose to aid in visualization?

DR. GROSS: Both are good suggestions.

DR. KUSCHNER: I think one of the other instruments that could be used is a labeled fiber, a radioactively labeled fiber in an instance where the label does not leach out.

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MR. SHECKLER: I should like to pose a question to Dr. Gross, Dr. Davis, and Dr. Kuschner. Comparisons have been made relating to asbestos and fibrous glass. Over a 35-year latent period, in the absence of significant chronic pulmonary effects to fibrous glass workers, what is your opinion as to the extrapolation of the fiber glass injection and implantation animal study results to these workers?

DR. KUSCHNER: I think that here again it depends on the question you are asking. If the question is whether glass can produce a fibrogenic effect, or as Dr. Davis pointed out, whether glass can produce mesothelioma in contact with suitable tissue, the answer is that it has to or it might be able to. It is a different question if one asks if the dosage and particle size to which humans are exposed under usual conditions of exposure, are comparable to asbestos. It probably is better related to epidemiological findings than to the experimental findings.

Introduction

DR. HOOVER:
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Presentation

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SOME CUTANEOUS EFFECTS OF FIBER GLASS EXPOSURE

Eldred B. Heisel

Introduction

DR. HOOVER: We now come to some papers on the cutaneous effects of fiber glass exposure. The first paper is presented by Dr. Eldred B. Heisel of Columbus, Ohio. Dr. Heisel is the author of several papers dealing with this subject.

Presentation

DR. HEISEL: For the past 20 years, we have consulted with persons with dermatologic problems from the Newark plant of the Owens-Corning Fiberglas Corporation. Our experience supported Siebert [1942], Sulzberger and Baer [1942], and Schwartz and Botvinick [1943], all of whom concluded that cutaneous reactions to glass fiber materials are mechanical and not due to sensitization. In addition, we [Heisel and Mitchell, 1957] felt that the degree of reaction possibly varied directly with the caliber of the fibers. About 15 years ago, we set about to prove or disprove this concept by using glass fibers of several diameters in patch test and rubbing experiments. In the early studies, coarse to medium commercially available fibers and very fine research fibers were tested.

A few years later, fire casualties in homes, hospitals, hotels, auto races and space ships sparked the urgency for wearable fireproof materials. At about the same time, complaints were being registered by persons wearing clothing laundered with glass fiber fabrics. An attempt was made to determine if glass fiber materials might possibly be a partial answer to the fire casualty problem and if clothing laundered with glass fiber fabrics was irritating. To do this, we [Heisel and Hunt, 1968] repeated the patch and rubbing experiments with two finer calibered, commercially available glass fibers, and we studied the cutaneous response to underwear laundered with fabrics made from these finer fibers and a fiber commonly used in the manufacture of glass fiber draperies.

Patch Testing and Rubbing Experiments

Patch Tests: Participating in the patch test studies were 104 white women. The first group of 50 was patch tested 1 to 3 times for 48 hours, at 3-week intervals, with finely cut and unbroken, plain and starch-sized glass fibers of 3 diameters. These were the finest, middle range and coarsest fibers encountered in such manufactured fiber glass products as heat insulators, textiles and sound absorbers. The second group of 54 was exposed to similar samples of more recently developed, finer calibered Beta and C glass fibers. In addition, 92 men who had worked in a glass fiber plant from 1 to 15 years were patch tested with plain and sized unbroken fibers.

Rubbing Experiment: Thirty of the first group of 50 women participated in the first rubbing experiment. In that there was no reaction to patch testing with the finest fiber tested, an extra coarse

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fiber used only in impingement type air filters was included. One-half cubic centimeter of cut particles of the medium, coarse and extra coarse glass fibers was rubbed vigorously into 5 cm areas on the volar aspect of one upper extremity for 60 seconds on 5 successive days. On the other extremity, unbroken fibers were applied in the same manner. The results were observed at 48 hours, 96 hours, 1 week and 1 month. At a later time the same experiment was done using the finer Beta and C fibers on 54 subjects.

Laundering Fiber Glass with Underwear Tests

Clinical Study: Beta, C and DE fiber fabrics were used as test materials in a double blind study in which 76 young, white women and 26 young, white men participated. Underwear laundered and tumble dried with hemmed lengths of glass fabrics equivalent in bulk to a pair of draperies was worn for two successive days without bathing by two-thirds of the subjects. One-third acted as controls. Neither the subjects nor the examining physician knew who was or was not a control subject.

Physical Analysis: Duplicate sets of underwear for physical analysis were laundered with those used in the clinical study. From these 1-inch square swatches were ashed at 880 F for 3 hours, mounted and examined microscopically.

One gallon aliquots of water taken from three successive rinsings were filtered after the washer had been used for laundering glass fabrics. The lint so obtained was ashed and examined as had been the swatches of underwear.

The fiber diameters of the glass fibers used in these studies are compared in Table 19-1. The first group of four were used in the early patch and rubbing experiments. In addition to patch and rubbing tests, Beta, C and DE fibers were examined in the laundering experiment.

Results

Patch Tests: Positive reactions to finely cut fibers consisted of small discrete red papules which were interpreted as being due to mechanial irritation. None of the unbroken fibers of the entire series of fibers tested produced any cutaneous reaction. When finely cut Beta, C and No. 333 glass fibers, the finest fibers studied, were applied to the skin, only 4 of 172 patch tests were minimally positive. Figure 19-1 reveals clearly that cut fibers of greater diameters produced larger numbers and more severe cutaneous reactions than did the finer calibered fibers. Patch tests with the coarsest fiber used, No. 37, were positive in 85% of 118 tests and 51% were greater than 2+ reactions. This is compared with 50% positive tests and 10% greater than 2+ reactions when a finer glass fiber No. 150 was used.

Rubbing Tests: Positive reactions in the rubbing tests varied from minimal erythema to marked eczematization. There were no cutaneous reactions when cut Beta fibers were rubbed into the forearms of 54 subjects. Only one objective and two subjective responses were elicited by C fibers. Figure 19-2 demonstrates again in these tests that the coarser

Fiber

Fine - #333

Medium - #1

Coarse - #3

Ex. Coarse

Beta

C

D-E

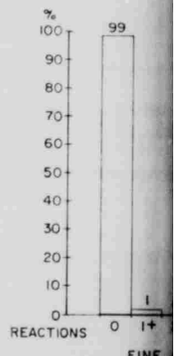


FIGURE 19-

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TABLE 19-1

DIAMETERS OF GLASS FIBERS

Fiber	Diameter inches	Diameter μ
Fine - #333	0.0003	0.762
Medium - #150	0.00035 - 0.00040	8.89 - 10.16
Coarse - #37	0.00070 - 0.00075	17.7 - 19.1
Ex. Coarse - #12	0.00150	38.1
Beta	0.00010 - 0.00018	2.54 - 4.57
C	0.00014 - 0.00023	3.55 - 5.8
D-E	0.00021 - 0.00035	5.3 - 8.89

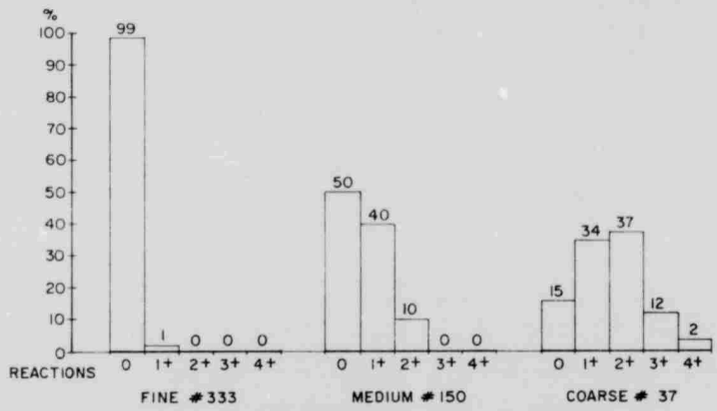


FIGURE 19-1. PATCH TESTS WITH FINE CUT FIBERS

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TABLE 19-2

UNDERWEAR LAUNDERED WITH FIBER GLASS

Fiber	Subjects	No Reaction	Reaction	% Total
Beta	80	80	0	0
C	72	71	1	1.4
D-E cont.	40	34	6	15.
D-E text.	39	20	19	48.7

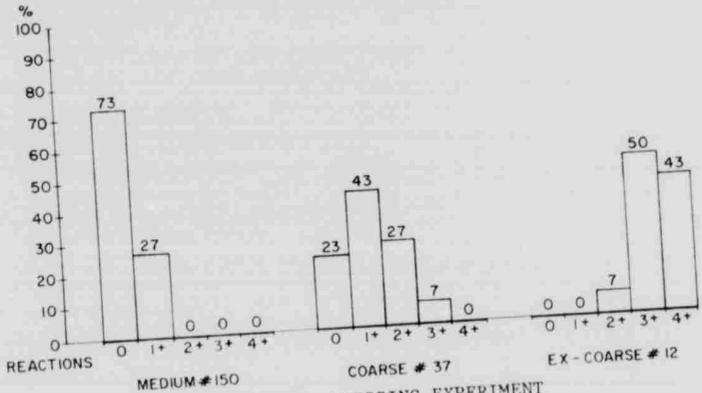


FIGURE 19-2. RUBBING EXPERIMENT

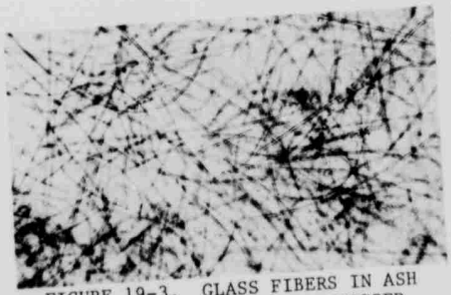


FIGURE 19-3. GLASS FIBERS IN ASH OF COTTON KNIT FABRIC LAUNDERED WITH GLASS FABRIC. (DE, CONTINUOUS DYED, X 126)

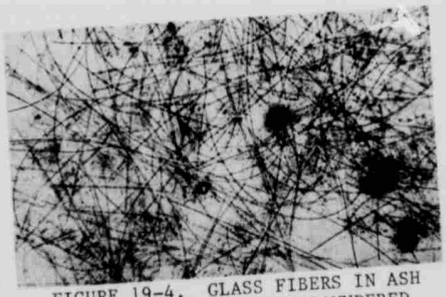


FIGURE 19-4. GLASS FIBERS IN ASH OF COTTON KNIT FABRIC LAUNDERED WITH GLASS FABRIC. (BETA, TEXTURED DYED, X 126)

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fibers produced greater numbers and more severe cutaneous reactions. Every subject responded adversely to No. 12, the coarsest fiber tested. Of these, 93% were severe responses and 43% were 4+ reactions. More than 3 times as many subjects had no reactions when tested with cut fiber No. 150, the finest fiber represented, than did those tested with a coarser fiber, No. 37. With neither of these fibers were there any 4+ reactions.

Sensitization: At 3-week intervals, 50 subjects were patch tested with 6 patches, 1 to 3 times. Thirty of the 50 were rubbing experiment subjects. These 30 were patch tested again 6 weeks later with unbroken glass fibers. In no case was there any reaction in these tests. Ninety-two employees who had worked from 1 to 15 years in a glass fiber factory were patch tested with unbroken fibers. Four had reactions consisting of one or two papules which were felt to be due to mechanical irritation from accidentally broken fibers. When exposed to uncut glass fibers, none of the test subjects or the glass fiber workers exhibited erythema, edema or vesiculation, common signs of cutaneous sensitization.

Laundering Experiment:

Clinical - In Table 19-2, one observes that D-E fibers, those used in the manufacture of draperies and textiles, effected cutaneous irritation much more often than did the finer calibered, essentially non-irritating Beta and C fibers. The textured D-E fabrics, which release lint more freely caused irritation 3 times more often than did the continuous fiber fabrics. One-third of the subjects who had subjective complaints exhibited objective signs of irritation. The complaints varied from a slight prickling sensation to extreme pruritus. Objective signs were largely secondary to scratching.

Physical Analysis - Microscopic examination of the residue from ashed swatches of underwear laundered with glass fiber fabrics disclosed an abundance of short glass filaments. Lint recovered from 3 successive rinses after laundering of glass fiber fabrics contained numerous glass fibers.

Serendipitous finding: One employee, when examined, presented a widespread, urticarial eruption which he stated had appeared every day during 10 months of employment. This urticaria disappeared soon after showering. One test subject developed localized urticaria within a few minutes of exposure to shredded glass fibers. Both exhibited marked dermographism.

Conclusions

In these studies more than 150 persons were exposed repeatedly to glass fibers of varying diameters. The fibers were used as patch tests, rubbed vigorously into the skin, and entrapped in underclothing. From our findings we felt the following conclusions were logical:

1. Cutaneous sensitization to fiber glass is difficult and probably is rarely encountered.

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2. Cutaneous reactions to fiber glass fibers are transitory, largely mechanical and vary directly with the diameter of the fibers.

3. Lint from glass fiber fabrics, like that of any fabric, laundered with clothing in modern automatic washers and dryers becomes entrapped on or in the clothing and further. Lint clings to the walls of laundry equipment even after several rinsings.

4. Fabrics or lint from fabrics made from glass fibers having diameters larger than 0.00021 inches will likely cause transient, mechanical, cutaneous irritation in a significant number of persons.

5. Fabrics or lint from fabrics made from glass fibers less than 0.00018 inches in diameter probably will not irritate the skin.

6. Fiber glass fibers, along with other fibers, may be coarse and harsh, or fine and soft.

7. A simple test for dermographism may be a useful preemployment examination test for persons contemplating working with glass fibers.

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Introduction

DR. HOOVER:
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Presentation

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THE CUTANEOUS AND OCULAR EFFECTS RESULTING FROM WORKER EXPOSURE TO FIBROUS GLASS

James Lucas

Introduction

DR. HOOVER: The next paper will be presented by Dr. James Lucas of the Medical Services Branch, Division of Technical Services, NIOSH, in Cincinnati.

Presentation

DR. LUCAS: The cutaneous effects of modern fibrous glass apparently were first appreciated by various company physicians shortly after the Owens-Corning Fiberglas Corporation [Health Aspects of Fiber Glass Materials, 1942] began production in 1933. While the company sustained steady and significant growth during the ensuing years, the magnitude of the problem was not apparent until the early 1940's when the country geared for the war effort and large amounts of this marvelously versatile material first came into usage. At that time the principal application was insulation and a major consumer was the Navy Bureau of Ships.

The first scientific account of fibrous glass dermatitis and other possible health effects was published in January, 1942, by Dr. Walter J. Siebert, a prominent St. Louis pathologist. Interestingly, his study was carried out at the request of the International Association of Heat and Frost Insulators and Asbestos Workers, Local Union No. 1 of St. Louis, Missouri. Dr. Siebert traveled to the Owens-Corning plant in Newark, Ohio, and personally examined approximately 200 production workers. [Siebert, 1942] The findings of this investigation retain their significance to this date. Dr. Siebert found only one worker with evidence of significant skin irritation. This individual had been employed only 4 days. One 22-year-old worker related to him that he had become accustomed to handling fiber glass and suffered no discomfort while continually on the job, but he noticed a mild skin irritation that lasted a few days after returning to the job following his two week vacation. "As long as I am working right along," he related, "I stay toughened up to it. But if I haven't been handling it for a while, like when I'm on my vacation, then it takes a few days to get over the itch when I start in again."

Significantly, Siebert did not see a single case of eye irritation during this inspection. He correctly attributed the ephemeral discomfort of inexperienced workers to mechanical irritation and attributed prolonged dermatitis problems to other causes.

The fact that nearly all glass fibers, regardless of ultimate use, are coated with binders, lubricants, or coupling agents which serve to bind individual fibers together, protect the fiber surface, or to serve as bonding agents when the fibers are eventually incorporated into resin matrices, led to some confusion in subsequent early reports. Schwartz, in fact, noted three out of seven fiber glass dermatitis patients with reactive patch tests, indicating allergic sensitization to a binder consisting

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of starch, polyvinyl alcohol, and a pyrazine compound. [Schwartz and Botvinick, 1943] However, Sulzberger and Baer in more extensive studies were unable to demonstrate allergic sensitization in either man or animals with various fiber glass materials. [Sulzberger and Baer, 1942] Despite the addition of these various coatings, allergic sensitization appears to be very rare, probably because most of the resin systems employed for this purpose are in the fully cured state prior to human exposure. The inertness of fiber glass itself is well attested to by its once widespread usage as a suture material. In view of what is now known regarding intracavitary injection of fibrous glass, it might be epidemiologically interesting to follow-up such a group of patients today.

Clinically, fibrous glass produces an eruption consisting of tiny erythematous papules. While numerous other morphologic types of dermatitis are described in the literature these appear to be largely secondary to the scratching or rubbing produced by the pruritus provoked by the epidermal irritation of the embedded glass specules. Secondary infection is a rare complication in our experience, although commonly mentioned in reference book description of the lesions.

In actuality, the name, Fiber Glass Dermatitis, is usually a misnomer. Pruritus without objective evidence of dermatitis is the much more common clinical presentation and pruritus or itching is the cardinal symptom. It apparently results from the release of chemical mediators occurring when the fibers pierce the epidermis. These substances, most likely kinins or histamine, are powerful vasodilators. With vasodilation, edema fluid collects and pruritus occurs. It should be recalled that itching and pain are physiologically very closely akin sensations. Dr. Heisel's observations on the relationship between fiber length and diameter and epidermal penetration, are now classic and clearly elucidated the observation that only certain fibers resulted in dermatitis. [Heisel and Hunt, 1968] In any event the clinical hallmark is pruritus, entirely out of proportion to the clinical signs of dermatitis. If the possibility of fibrous glass dermatitis is forgotten, and such is still frequently the case even among dermatologists, the patient is often subjected to an extensive and unrewarding medical work-up in search of some systemic cause for his itching. A simple potassium hydroxide (KOH) preparation of a superficial skin scraping, as first reported by Fisher a few years ago, is often sufficient to demonstrate the presence of fibrous glass fragments. [Fisher and Warkentin, 1969] The microscopic examination of a piece of clear Scotch tape, which had been used to strip the outer layers of the integument, also is often quite satisfactory in resolving puzzling cases of pruritus when due to fibrous glass. These fibers can easily be differentiated by their absolutely uniform diameter and refractile characteristics.

Fibrous glass dermatitis is by far the most important cause of mechanical irritant contact dermatitis and most certainly is not a problem limited to industrial users. Within the last 15 years, consumer products made of fibrous glass such as curtains, draperies, tablecloths, etc., have become increasingly important domestic household items. The problem stems not from the use of these items themselves, but rather, in their cleaning by laundering. When laundered, these items shed large numbers of glass

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specules which may cross-contaminate clothing. Many physicians have now observed and recognized family outbreaks of fibrous glass dermatitis of this origin since the original report of Madoff in 1962. This cause of pruritus should always be suspected when several family members begin nearly simultaneous complaints of intense itching. After the prevalence of this problem became known, it resulted in the promulgation of a Trade Regulation Ruling by the Federal Trade Commission in 1968 that glass fiber curtains and draperies be labeled with a warning tag affixed to the product. This label must caution that skin irritation may result from handling such products, and that irritation from body contact with clothing or other articles such as bed sheets, which have been washed with such products, or in a container previously used for washing them without thorough cleaning, may result in irritation. In several instances I have traced the source of clothing contamination to the use of commercially operated self-service coin laundries. Since the Federal Trade Commission ruling, it apparently has become common practice to launder fibrous glass articles in such public facilities rather than in the home washing machine. Unfortunately, there is sufficient fibrous glass carryover to thoroughly contaminate the next unsuspecting laundromat customer's clothing. Properly, fibrous glass items should be washed separate from clothing items in a basin or tub which can be repeatedly rinsed after use. Rubber gloves should be worn. Understandably, this method of laundering these bulky items has proved unacceptable to many modern homemakers, hence they go to the laundromat. The ingenuity of housewives never fails to astonish me.

In terms of the number of NIOSH requests for Health Hazard Evaluations and other forms of assistance in problems involving dermatitis, fibrous glass and cutting oils rank about equally and well ahead of epoxy systems, the rubber sensitizers, and the metals--nickel and chromium. Very few requests are received from primary producers, probably because these workmen either quickly "harden" or leave. Generally, the problem, as we see it, is noted within the reinforced plastics industry which uses large amounts of textile type fibrous glass. This industry contributes greatly to the estimated 33,000 end products incorporating fibrous glass and utilizes huge quantities, both in the form of short-chopped fibers and as yarns or cloth which are incorporated into various resin systems. [Possick et al, 1970] Major product groups include laminated electrical circuit board, vehicle bodies, sports equipment, boats, furniture, storage tanks, building panels, and bathroom tubs and shower stalls. Production of these items not only brings the worker into contact with fibrous glass, but also with a myriad of resins, hardeners, catalysts, dyes, and plasticizers.

The most commonly employed systems utilize polystyrene, vinyl, acrylic, amino, epoxy, polyester, and phenolic resins. The majority of these are primary irritants and many are common causes of allergic sensitization. Fortunately, confusion with other forms of dermatitis is not common, since fibrous glass dermatitis usually is easily differentiated by its prompt appearance in new workers, the marked disproportion between objective findings and complaints, and its transient nature. If the KOH preparation is to be useful in the industrial setting, careful skin cleansing should be carried out first since heavy skin contamination can cause false positive results.

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When fibrous glass has been identified as the offending agent in plants manufacturing such products, the source is usually not the basic fibrous glass stock, but rather the fly or dust generated by various sawing, grinding, sanding, or other finishing type operations. Once airborne, these fibers settle out on uncovered areas of the body or in between cuffs and collars and the skin. Large amounts may settle out on work place surfaces and may be secondarily carried to the face or eyelids where they may be rubbed into the skin. These are then the common sites for fibrous glass dermatitis. Prevention is difficult because these operations are often performed on only an intermittent basis and employees never harden to the irritation. Loose fitting or cloth gloves may trap fibers and aggravate the dermatitis. Shirts, blouses and other clothing should be loose fitting and changed daily. Brooms and air hoses should not be used in personal cleansing, but ample amounts of water are recommended. The results with various barrier creams are, in my opinion, equivocal.

The second portion of my assigned topic deals with the ocular effects of fibrous glass. I hope I can quickly put this topic in its proper perspective.

All the standard reference works briefly mention eye irritation from airborne fibrous glass. Yet this seems to be rare in actual practice despite the huge amounts of fibrous glass in use. In reviewing the actual literature following both Toxline and Medline computer searches, only the frequently cited paper by the Australian workers, Longley and Jones, [1966] was located. They reported only a single case, that of a 48-year-old woman who had worked 9 years in a factory making a variety of electric cables. She operated a machine which cut glass-covered cables 1 day per week. Other employees operated the machine daily on a rotation basis. The patient and the other employees had long noted the typical cutaneous effects of fibrous glass. Each time she operated the machine, her eyes became sore and she commented that the dust gave her "pink eyes." This was always relieved by washing the eyes with tap water and they felt normal the next day. After 8 months running the machine, she suddenly noted severe eye irritation which did not subside overnight and only gradually improved over the next week. At that time, she again operated the offending machine following which she developed intense pain, photophobia, and lachrymation. She was then seen by a ophthalmologist who reported finding refractile material adhering lightly to the conjunctiva. He felt that the keratitis was, in his opinion, due to fibrous glass as any material which could irritate the skin enough to cause dermatitis could, with equal facility, irritate the conjunctiva enough to cause conjunctivitis, and the cornea enough to cause keratitis. Three weeks later, the eye was still inflamed with an obvious opacity of the cornea. An abscess subsequently developed in the area of the opacity which was drained. Bacterial culture of the pus removed from the abscess was sterile. A breathing zone sample of the machine operator contained a total of 1.5 mg/cu m of glass.

While this case is suggestive, it certainly is not fully convincing as to the etiology of the keratitis, a not rare ocular problem of diverse etiology which often remains cryptic. The fact that other workers with identical exposure were not affected; that the condition did not develop for over 8 months; that the process apparently affected largely one eye;

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its duration and course; and finally the relatively low environmental levels of fibrous glass all suggest that another causal agent might have been responsible or that this patient had some very unusual susceptibility. The paper concludes with an appeal for other case reports of keratitis in which fibrous glass might be implicated. To the best of my knowledge, no similar case reports have been forthcoming in the 8 years since this initial report.

Eye irritation is an extremely common complaint in industry. It is no less common in that segment of the economy which deals with plastic laminates, because of the many irritating chemicals and solvents required in processing. Yet eye irritation reported from fibrous glass seems singularly rare while the problem of cutaneous irritation remains extremely common. This probably is attributable to the amazingly efficient flushing action of the tears and, to that fact, that in most jobs, at least in this country, which generate large amounts of airborne fibrous glass such as grinding or sanding, require the use of safety glasses or goggles. In any event, I believe we may reasonably conclude that the ocular hazards of airborne fibrous glass dust, if they occur, are remarkably rare and under ordinary conditions of exposure do not pose a problem.

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DR. HOOVER: The papers of Dr. Lucas and Dr. Heisel are now open for discussion.

MR. STEINFURTH: I have to agree with the statement made about the wives going to the laundromats. All asbestos workers' wives do not buy fiber glass drapes, but they do go to the laundromat with their husbands' clothes. They will not wash them at home. I come from Toledo, Ohio, which basically is the home of Owens-Corning, and I have been to their plant in Newark. I find that the ventilation in the factories varies. There is a great deal of difference from construction project to construction project. You run into not only poor ventilation, but extreme temperatures, which is another matter we have to put up with.

A question was asked as to how Owens-Corning measured the size of the fibers when they said they were down to 1 μ in 1941. I understand it takes an electron microscope to measure their sizes. It has been my impression that they couldn't go below 5 μ.

Also, in conclusion, it appears that I have more information in my files than NIOSH in many instances. They are more than welcome at any time to come over and look at my files, and maybe we could get together and agree on some things.

MR. PARRILLO: To either speaker: Since it is probably going to be quite some time, if ever, that a threshold limit value other than nuisance limit for fiber glass will be established, do you feel that in the near future there might be a limit established to help out with this mechanical irritation problem?

DR. LUCAS: I will try to answer that. I would doubt whether this is possible because what is in the air is not too much a determining factor of whether or not you get dermatitis. You get dermatitis from dust that has settled, and this depends upon the amount the length of time that the operation has been occurring, and many other factors. This could build up on work desk surfaces, over a very long period of time.

The problem is, of course, in intermittent operations. At least, that's where I see the problem. Where people work with daily exposures, they soon harden and there is no real dermatitis problem. I don't know that instituting some sort of limit in terms of air levels would be of value at all. Perhaps some sort of regulation or something dealing with work practices and housekeeping might be a more practical way to approach the mechanical dermatitis.

DR. UTIDJIAN: I first want to comment and then ask a question of Dr. Lucas. My comment is, when I was in the Shaw Industrial Health Service in London some years ago, there was a fiber glass epoxy resin boat manufacturer where the resin was applied manually to a sheet of fiber glass. There was a phenomenon of ephemeral immunity to the itching. The irritation was very well established in that plant. I should like to ask Dr. Lucas what theories he may have about the mechanism of this

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DR. LUCAS:
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DR. UTIDJIAN:

DR. LUCAS:
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DR. HEISEL: It
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DR. LUCAS: The
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DR. TANAKA: I
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DR. HEISEL: A
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DR. LUCAS: Yes,
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tachyphylaxis, as it has been called. It is reminiscent of a situation where histamine release is also implicated.

DR. LUCAS: I am not sure whether your question refers to the dermatographism that can be produced by the spicules in certain individuals who have this propensity, or whether you are referring to the hardening that the vast majority of people go through after a transient episode of dermatitis.

DR. UTIDJIAN: The hardening which sloughs off during vacation periods.

DR. LUCAS: I don't think the answer is known exactly. It seems likely that it is probably due to epidermal thickening.

DR. HEISEL: It seems to me that there are several things. One, that they may be exposed to so much of the material that there is no longer an ability to respond. Two, a great number of these people block out their pruritis psychogenically. You can block out things if you wish. It seems to me that these two factors are quite important in what is commonly called a hardening process.

DR. LUCAS: The whole process of hardening is very poorly understood, and one has to question whether it occurs to many of the substances, for instance, many of the sensitizers. Based, on modern immunologic theory, it would be most unlikely to occur. Yet, these people do adapt and are able to work without problems. I think what Dr. Heisel said has a lot of merit.

Unfortunately, it is very difficult to study hardening because you don't know whether the person might have changed his work practices. It is a very poorly understood phenomenon. I have never heard a really good discussion on it.

DR. TANAKA: I have two questions. The first one is, what is the fate of imbedded fibrous glass in the skin? The second one is, what is the treatment of choice of this contact?

DR. HEISEL: After we did our early experiments, we did biopsies on five patients and we actually couldn't find the fibers. Now, it may be similar to some of the other workers in that they were there but we just couldn't see them. We used regular microscopes and polarized light and were unable to find the fibers. It is my feeling that they were very superficially imbedded and that they are exfoliated. Actually, the best therapy is a good bath. You wash off what you can. In our laundering experiments in which some patients had excruciating pruritus, I mean enough that they had to get up in the middle of the night and take their clothes off, therapy was a minimal problem. There was only one person that we really had to treat. We simply treated him with baths and with corticoid cream. In about 3 or 4 days, he was completely clear and had no problem.

DR. LUCAS: Yes, treatment is really totally directed at symptomatic relief. I think you simply go as far as is necessary to obtain relief. Bathing or a simple shake lotion often affords relief. You just go as far as necessary with individuals.

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DR. HOOVER: If there are no more questions on the dermatologic aspect, are there any questions you wish to ask of the previous speakers on animal experimentation?

DR. PUNDSACK: I should like to direct a question to Dr. Huth on the paper presented by Drs. Pott and Huth, for clarification. As I understand, your injections were done with rats. I also thought you said that you did experiments with guinea pigs and with hamsters and that these did not show any effect. If that is the case, then my question is, what is the significance of that difference in behavior between the various animal models that were used?

DR. HUTH: We got tumors with rats and mice. We didn't get any tumors with hamsters and guinea pigs. That is a well known problem for those experiments. You have to search for the right animal which quickly develops the tumor you want to prove. But, you should discount that problem as to why hamsters and guinea pigs don't develop tumors. Some people are sensitive to some agents and other people are not. There are individual characteristics which can be explained, but not here and not by me.

DR. PUNDSACK: I have one other point. Although fiber glass, as such, has not been made in Germany for a long length of time, I believe that mineral wool has been produced in Germany for quite some time. In fact, it was in World War I when asbestos shipments to Germany were cut off that mineral wool was really developed commercially in Germany. It might be possible to do epidemiological studies on some of those worker populations. It would be possible, I think, to go back and obtain old samples of mineral wool produced back in the 1920's or 1930's from old installations and actually determine fiber size distribution in those products to which the workers presumably were exposed.

DR. HUTH: I have to think about the advice concerning the data and perhaps some people would like to start the epidemiological studies.

DR. HEISEL: Most of the contact I have had with fiber glass and the positive cutaneous reactions we have had, have been on materials that were put on the glass in the form of sizing. We have never had a positive reaction to the glass itself. I was wondering whether consideration had been given to the possibility that the coatings on the fiber glass was responsible for some of the responses in the lung rather than the glass itself.

I noted, in the work from Wales, that they used commercially available fibers. I would feel fairly certain that numbers 100 and 110 would be covered with some kind of sizing. Is that correct? I just wondered if they took this into consideration.

UNIDENTIFIED PARTICIPANT: I have a question for the panel. This deals primarily with the technique of lung fixation. Since most of the members on the panel have had experience with pulmonary pathology, I would like to know what they think is the best technique for fixation of lungs for evaluation?

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There is o tumor, mesotheliom be noncarcinogeni of attempting to g of lung tumors in adenomas. Attempt a substance will spontaneously deve

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DR. GROSS: With regard to the last question, I think some of us on the panel have clearly indicated that the inhalation technique is the proper and best technique to use.

With regard to the question of attempting to find whether the coating on glass fibers has an effect on the lungs, I think that was answered by my own investigation in which animals were exposed to uncoated glass, to glass coated with phenol formaldehyde resin, and with glass fibers coated with starch binder. There was no detectible difference in the reaction of the lung tissue to these three types of dust.

Now I should like to pose another comment. I have been greatly concerned with the validation of the intrapleural technique to demonstrate the carcinogenic potential of fibrous glass or any other fibrous material. I have been greatly concerned with the fact that a number of investigators have found that an occasional rat will respond to a nonfibrous dust with the development of what was called mesothelioma. The development of a mesothelioma with such an inert dust as barium sulfate and calcium sulfate is of great concern in the evaluation of the development of mesotheliomas in the experimental animal used.

There is one other comment, and that is the case of a malignant tumor, mesothelioma, developing in response to dust that has been proven to be noncarcinogenic. This development is similar to the discredited model of attempting to gauge the carcinogenicity of a substance by the formation of lung tumors in mice, so-called adenoma. Mice readily develop pulmonary adenomas. Attempts have been made to gauge carcinogenicity by showing that a substance will produce a greater number of adenomas in mice than would spontaneously develop. This technique has been thoroughly discredited.

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SESSION IV

EPIDEMIOLOGIC STUDIES

Chairman: Pierre Decoufle

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HUMAN EPIDEMILOGIC STUDIES WITH EMPHASIS ON CHRONIC PULMONARY EFFECTS

Michael Utidjian
W. Clark Cooper

Introduction

DR. HOOVER: I should like to introduce to you the Moderator and Chairman for session IV, Dr. Pierre Decoufle, who is the Chief of the Illness and Injury Surveillance Branch of NIOSH.

The first paper will be presented by Dr. Michael Utidjian of Tabershaw/Cooper Associates.

Presentation

DR. UTIDJIAN: The original plan of the Industrial Health Foundation (IHF) survey was to study a stratified random sample of the current work force at the Newark, Ohio plant of Ohio Corning Fiberglas (OCF) in 1968. To make the study population as representative as possible, with respect to age and crude intensity of exposure, the entire work force was classified by management into approximate exposure categories termed as high, medium, and low, and also into three age groups, under 30, 30 to 49, 50 and over. These two categorizations thus generated a Latin square of nine subgroups. The statistical designer of the study, the late Dr. Antonio Ciocco of the University of Pittsburgh, selected 30 as the optimum number of subjects in each cell of the 3 x 3 square, thus giving 30 x 9 = 270 as the size of the sample as originally planned, or slightly more than 10% of the total work force of some 2,400 at the time. From the total number of employees available in each of the nine age x exposure categories, a selection was made by application of random number tables to the employees' company badge numbers. It was then found that there were only 21 employees available in the "youngest, least exposed" category, but that every one of the remaining eight cells had a full complement of 30. It was therefore decided to proceed with the reduced number of 261 subjects. Between the time of the drawing up of the sample and the actual execution of the study in the field however, there was a further loss of available subjects due mainly to resignations and drafting for military service. There had also been one death from causes other than pulmonary disease. Finally, 232 of the originally possible sample of 261 were investigated. However, the greatest deficit was in the youngest age group. The highest exposure/oldest "cell" was intact with the full 30 employees.

All 232 employees examined were subjected to a questionnaire based upon a modification of the British Medical Research Council "Bronchitis" questionnaire. The modifications were chiefly semantic, and basically the same information was sought. The questionnaire was administered by three Industrial Health Foundation physicians and Dr. Jon Konzen, Medical Director of Ohio Corning Fiberglas. Each employee was next weighed and his height measured and then spirometry was performed by Dr. Ben Lambiotte, then of the IHF, using a standard Collins' type spirometer. The best of 3 measurements was taken on all subjects, and Vital Capacity, deviation of observed Vital Capacity from that predicted from age and stature tables, F.E.V. 1.0, and V.C./F.E.V. 1.0 ratios were calculated.

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Recent (ie within a year) chest X-rays of all 232 subjects studied were reviewed by Dr. Jon Konzen of OCF and Dr. Robert T. P. de Treville, then President of the IHF. Radiological abnormalities classified as "Possible Dust Patterns" (exaggerated linear markings) were recorded. Finally, at a somewhat later date in 1968, more sophisticated and detailed pulmonary function studies were performed at a well-equipped cardiopulmonary unit in a nearby community hospital by Drs. Henry L. Hook and George Morrice, Jr., on a sub-sample of 30 employees, all from the 50 and over age category, 15 from the highest and 15 from the lowest exposure categories. This more detailed evaluation consisted of comprehensive general medical history taken by an internist, and a special respiratory questionnaire. A comprehensive general physical examination was performed. Chest fluoroscopy was conducted with special attention to diaphragmatic motion, cardiac silhouette, vascular tree and evidence of air-trapping or parenchymal lesions. A standard 12-lead resting electrocardiogram was taken. Ventilatory function testing consisted of measurement of vital capacity: 1, 2 and 3-second forced vital capacities; mid-expiratory flow rate; maximal voluntary ventilation and residual volume determinations. These measurements were determined using the Collins 13.5 L, 3-speed spirometer. The pulmonary diffusing capacity for carbon monoxide was determined by a steady-state method, employing the end tidal sample of carbon monoxide. A modification of the technique of Bates as recommended by Wright and Gaensler was chosen because of its relative simplicity and because no arterial puncture was required. The test was performed in a sitting position at a constant tidal volume.

Results

The results of all phases of the IHF studies outlined above were essentially negative, in the sense of failure to demonstrate any significant difference in the prevalence of chronic respiratory illness or impairment as between the highest and lowest exposure categories of workers. Naturally, abnormalities were recorded but at the rate and of a nature to be expected in a population of this character. As was to be expected, increasing age was an influential variable, but increasing exposure category was not. It is recognized that this was essentially a cross-sectional and "survivor-group" study, but such can only be improved upon by a deliberate prospective study with exhaustive follow-up of all those who leave the industry at various stages in their employment history. However, it is felt that if a gross hazard to cardiopulmonary health were to be associated with occupational exposure to fibrous glass dusts at the levels encountered in this, the longest established plant in the U.S., some indication of this would have emerged even from a study with these limitations.

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THE PREVALENCE OF RADIOGRAPHIC ABNORMALITIES IN THE CHESTS OF FIBER GLASS WORKERS

Ahmed N. Nasr
Theodore Ditchek
Paul A. Scholtens

Introduction

DR. DECOUFLE: Our second paper will be presented by Dr. Ahmed Nasr of the Health and Safety Laboratory of Eastman Kodak Company.

Presentation

DR. NASR: This study was done at the University of Michigan Medical Center in collaboration with Doctors Theodore Ditchek and Paul A. Scholtens.

This investigation deals with the findings in a chest X-ray survey that has been conducted on the total working population of a factory manufacturing glass fibers. As will be evident from the duration of employment of the workers, this factory is one of the oldest manufacturing fiber glass on a commercial scale. The reports of McCord [1967] and of Wright [1968] describe the nature of the industrial operation and occupational exposure. In addition to fibrous glass, the workers are exposed to other inhalants. These include resins and plastics and their precursors, compounds that are used for coating glass fibers.

In a previous communication, one of us [Nasr, 1967] reviewed the pulmonary hazards from inhalation of glass fibers. Most of what has been reported in the literature dealt with few individual case reports. In the majority of those patients in whom exposure to glass fibers allegedly caused lung disease, the clinical and pathological manifestations were predominantly those of bacterial infection, and there was no evaluation of the environment for the presence of noxious inhalants. Because of the paucity of these cases and the tenuous nature of the association between exposure to glass fibers and the incidence of lung disease, it was concluded that fibrous glass is a relatively inert material, with no fibrogenic or other significant toxic properties. That conclusion was based also on the reported results of animal experiments. In order to substantiate, or perhaps refute, that conclusion, the present investigation has been undertaken.

Materials and Methods

We obtained one 14 x 17-inch postero-anterior X-ray film of the chest for every worker in the factory. The total number of workers was 2,394. The radiographs were taken during a one-year period ending on June 30, 1967. Thirty-six out of the 2,394 radiographs (1.5%) were found unsuitable for radiological diagnosis because of their technical quality, and were excluded from the study. The remaining 2,358 radiographs included 173 radiographs of male batch-house workers and 157 radiographs of female fiber glass workers. Since the female fiber glass workers were relatively few in number, and the batch-house workers were exposed neither to glass

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fibers nor to the compounds used for their coating, both of these groups were excluded from this investigation. Thus, the analysis of data was limited to 2,028 male fiber glass workers, ie, approximately 91% of the total factory population.

We were also supplied the name, age, duration of employment and exposure category of the workers. The degree of exposure of each worker to glass fibers was classified as low, average or high. This classification was assigned every individual worker by both the factory production manager and the factory physician, based on several industrial hygiene surveys that were conducted over the years in various areas of the factory. For example, clerks, machine operators and fabricators were classified as having low, average and high exposures, respectively.

On visiting the factory and discussing the various manufacturing processes with the manager and the physician, we learned that job transfers had been frequent between the two categories of exposure designated "average" and "high," while a man employed in a job of "low" exposure usually remained in this category. Thus, in the analysis of our findings, we combined the two exposure categories "average" and "high" into one category of exposure which we shall refer to as "production workers," while the "low" exposure group will be designated "office workers."

The radiographs were arranged in random sequence, divided into five equal groups, and the groups assigned at random to five radiologists who read the films without knowledge of the worker's name, age, duration of employment or exposure category. Diagnosis of radiographic abnormalities was made according to a classification adapted from the one used by the Radiology Department, the University of Michigan Medical Center, and from the International Classification of Radiographs of the Pneumoconioses. [1958] The adapted classification was as follows:

(A) Negative chest

(B) Abnormal chest of minor or no clinical importance:
- (1) Congenital rib anomaly
- (2) Congenital lung anomaly
- (3) Acquired bone lesion
- (4) Pleural scarring, minimal
- (5) Calcified lymph nodes
- (6) Calcified parenchymal scarring

(C) Abnormal chest:
- (1) Mediastinal lesion
- (2) Pleural thickening or extensive pleural scarring
- (3) Pleural effusion
- (4) Pleural calcification
- (5) ? Solid solitary pulmonary lesion
- (6) Solid solitary pulmonary lesion
- (7) Increased lung markings
- (8) Interstitial infiltrate/generalized fibrosis
- (9) Prominent hilar areas
- (10) Hilar adenopathy
- (11) Bullous emphysema

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(12) Generalized emphysema
(13) ? Nodularity
(14) Pneumoconiosis, pinpoint nodularity
(15) Pneumoconiosis, micronodular: 1 to 2 mm
(16) Pneumoconiosis, nodular: 2 to 6 mm
(17) Pneumoconiosis, conglomerate nodularity
(18) Other abnormalities
(19) Abnormal aorta
(20) Abnormal heart

In the analysis of our findings, we combined diagnostic category "A" (negative chest) and diagnostic category "B" (abnormal chest of minor or no clinical importance) into one single group. This was considered appropriate since diagnostic category "B" included only such radiographic abnormalities as minimal pleural or calcified parenchymal scarring, congenital rib or lung anomaly, etc. This group will be referred to as "normal chest" while diagnostic category "C" will be referred to as "abnormal chest." However, a worker who was found to have both a "B" and "C" abnormality was counted only once, as having a "C" abnormality, ie, "abnormal chest."

Results

Age of the Study Population: Table 22-1 shows the age distribution of the study population. As would be expected, the majority of the workers (approximately 92%) were in the range of age 20 to 59 years. It is also obvious from Table 22-1 that the distribution of workers was approximately equal among the four decennials of this range. The table also shows that the median and the mean ages do not differ greatly within each age group, indicating that the frequency of the various ages is approximately symmetrical around the mean age in all groups.

Duration of Employment: Table 22-2 shows the distribution of duration of employment. Approximately two-thirds of the workers had been employed for 10 years or longer.

Nature and Distribution of the Radiographic Abnormalities: The overall prevalence of all radiographic abnormalities of the chest in the 2,028 workers was found to be 16.22%, ie, 329 workers were diagnosed as having an abnormal chest radiograph, while 1,699 workers had no radiographic abnormalities. Table 22-3 shows the distribution of these abnormalties in the 329 workers. Because of the scarcity of some of the abnormalities, the 20 diagnostic categories mentioned in "Materials and Methods" were condensed to only 14 in Table 22-3. Table 22-4 shows the prevalence of the various abnormalities in the total study population, ie, 2,028 workers, and the proportion they contribute in 383 abnormalities diagnosed in the 329 workers who were found to have an abnormal radiographic chest abnormality in the study chest radiograph.

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TABLE 22-1

AGE DISTRIBUTION

Age Group	No. of Workers	% of Total Study Population	Age	
			Median	Mean
<20	32	1.58	19	18.7
20-	546	26.92	24	24.3
30-	427	21.06	35	34.9
40-	406	20.02	45	45.1
50-	493	24.31	54	54.2
>60	124	6.11	62	61.7
Total	2,028	100.00		

TABLE 22-2

DISTRIBUTION OF DURATION OF EMPLOYMENT

Duration of Employment Category (Years)	No. of Workers	% of Total Study Population	Duration of Employment	
			Nominal	Mean
<5	713	35.16	2	2.3
5-	44	2.17	5	5.9
10-	249	12.28	14	13.4
15-	357	17.60	16	16.5
20-	274	13.51	21	21.8
>25	391	19.28	27	27.5
Total	2,028	100.0		

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Abnormal aorta
Abnormal heart
Other +

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Tables 22-3 and 22-4 show that the most prevalent population is "increased lung markings," with "abnormal aorta" ranking second, while "emphysema" and "abnormal heart" are third in order of frequency.

TABLE 22-3

DISTRIBUTION OF RADIOGRAPHIC ABNORMALITIES

Radiographic Chest Abnormality	Total No. of Cases	No. of Cases Among 281 Workers*	No. of Cases Among 42 Workers*	No. of Cases Among 6 Workers*
Mediastinal lesion	2	2	0	0
Pleural effusion	3	0	1	2
Pleural thickening or calicification	27	13	11	3
? Solid solitary pulmonary lesion	26	23	3	0
Solid solitary pulmonary lesion	11	7	3	1
Increased lung markings	93	76	14	3
Prominent hilar areas or hilar adenopathy	20	12	7	1
Emphysema, bullous or generalized	38	25	10	3
Intersitial infiltrate, generalized fibrosis	18	15	3	0
? Pneumoconiosis	17	15	2	0
Pneumoconiosis	9	6	3	0
Abnormal aorta	61	47	11	3
Abnormal heart	38	23	13	2
Other +	20	17	3	0
Total No. of cases	383*	281	84	18

*281 workers with one abnormality each, plus 42 workers with two abnormalities each, plus 6 workers with three abnormalities each

+Rare conditions of extrapulmonary origin, eg, calcified thyroid adenoma, multiple shot in soft tissue

TABLE 22-4

PREVALENCE OF RADIOGRAPHIC ABNORMALITIES OF THE CHEST IN THE TOTAL STUDY POPULATION AND THEIR PROPORTION IN THE TOTAL NUMBER OF ABNORMALITIES

Radiographic Chest Abnormality	Total No. of Cases	% Prevalence in 2,028 Workers	% of Total No. of Abnormalities (383*)
Mediastinal lesion	2	0.10	0.52
Pleural effusion	3	0.15	0.78
Pleural thickening or calcification	27	1.33	7.05
? Solid solitary pulmonary lesion	26	1.28	6.79
Solid solitary pulmonary lesion	11	0.54	2.87
Increased lung markings	93	4.59	24.28
Prominent hilar areas or hilar adenopathy	20	0.99	5.22
Emphysema, bullous or generalized	38	1.87	9.92
Interstitial infilitrate, generalized fibrosis	18	0.89	4.70
? Pneumoconiosis	17	0.84	4.44
Pneumoconiosis	9	0.44	2.35
Abnormal aorta	61	3.01	15.93
Abnornal heart	38	1.87	9.92
Other +	20	0.99	5.22
Total	383+		100.00

*281 workers with one abnormality each, plus 42 workers with two abnormalities each, plus 6 workers with three abnormalities each

+Rare conditions of extrapulmonary origin, eg, calcified thyroid adenoma, multiple shot in soft tissue

Effect of Degree of Exposure: The study population comprised 1,832 "production" and 196 "office" workers, ie, approximately 90% and 10% of the total number of workers, respectively. The prevalence of radiographic abnormalities of the chest was found to be approximately the same in these two exposure categories: 16.84% in the office workers and 16.16% in the production workers. Since increased lung markings, abnormal aorta, abnormal heart and emphysema were the most prevalent, it seemed that age was a major determinant. This was further supported by the finding that

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ce	% of Total No. of Abnormalities (383*)
	0.52
	0.78
	7.05
	6.79
	2.87
	24.28
	5.22
	9.92
	4.70
	4.44
	2.35
	15.93
	9.92
	5.22
	100.00

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the prevalence of radiographic chest abnormalities was essentially equal in office workers and production workers in spite of the fact that office workers had a lower level and shorter duration of exposure. However, their age distribution, as shown in Table 22-5 was similar to that of the production workers. The duration of employment of both exposure categories is shown in Table 22-6. Nearly half of the office workers had been employed for 10 years or longer, as compared to almost two-thirds of the production workers.

TABLE 22-5

AGE DISTRIBUTION OF "OFFICE" AND "PRODUCTION" WORKERS

Age Group	Office Workers		Production Workers	
	No.	%	No.	%
<20	3	1.53	26	1.58
20-	51	26.02	496	27.07
30-	34	17.35	392	21.40
40-	36	18.37	370	20.20
50-	59	30.10	434	23.69
>60	13	6.63	111	6.0
Total	196	100.00	1,832	100.00

TABLE 22-6

DURATION OF EMPLOYMENT OF "OFFICE" AND "PRODUCTION" WORKERS

Duration of Employment	Office Workers %	Production Workers %
Less than 10 years	50.51	35.91
10 years or more	49.49	64.09
Total	100.00	100.00

Correlation of the Prevalence of Radiographic Abnormalities to Age and Duration of Employment in the Various Age Groups: Table 22-7 illustrates the prevalence of radiographic abnormalities of the chest according to age. The coefficient of regression of prevalence (percent) of radiographic abnormalities on mean age and mean duration of employment, for the age groups in the range of 20 to 59 years, was calculated according to

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the least squares method. It was found to be 0.7 % per year of age, and 1 % per year of duration of employment. The difference is not statistically significant ($P < 0.05$). The strength of the association between prevalence of abnormal chest radiographs and mean age, and between this prevalence and mean duration of employment, was found to be 0.99 and 0.96 respectively, calculated as correlation coefficients. Figures 22-1 and 22-2 illustrate the findings in Table 22-7.

TABLE 22-7

PREVALENCE OF ALL RADIOGRAPHIC ABNORMALITIES
IN THE VARIOUS AGE GROUPS

Age Group	No. of Workers	% of Total Population	Mean Age	Mean Duration of Employment (Yrs.)	% of Workers With an Abnormal Chest Radiograph
<20	32	1.58	18.7	1.0	3.12
20-	546	26.92	24.3	2.6	4.76
30-	427	21.06	34.9	11.6	9.37
40-	406	20.02	45.1	18.3	17.24
50-	493	24.31	54.2	22.6	25.76
>60	124	6.11	61.7	23.4	52.42
Total	2,028	100.00			

Nodular Opacities: Among a total of 2,028 workers, only 8 had micronodular opacities (1 to 2 mm) in their lung fields, and one worker was diagnosed as having pinpoint nodularity. Questionable nodularity was suspected in 17 workers. No workers had nodules greater than 2 mm in diameter, or conglomerate nodules. Table 22-8 reveals no consistent pattern in the prevalence of these nodular opacities according to age or duration of employment.

These nodular opacities were referred to in "Materials and Methods" and in Tables 22-3 and 22-4 as pneumoconiosis so that the readers would use a uniform and convenient classification of radiographic abnormalities in reporting their interpretations. As is well known, there are several diseases that cause nodular opacification in the lungs. It is unlikely that exposure to glass fibers is associated with a pneumoconiosis hazard since only 9 workers out of 2,028 had nodular opacities. Furthermore, none of the nine workers had nodules greater than 2 mm in diameter.

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% of Workers With an Abnormal Chest Radiograph
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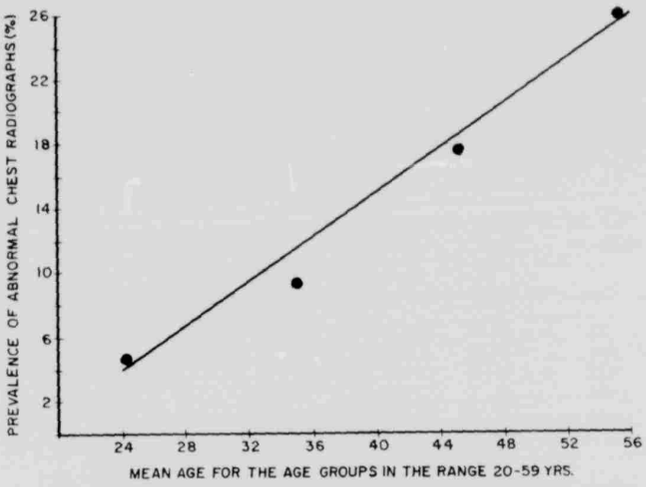


FIGURE 22-1. EFFECT OF AGING ON THE PREVALENCE OF RADIOGRAPHIC ABNORMALITIES OF THE CHEST

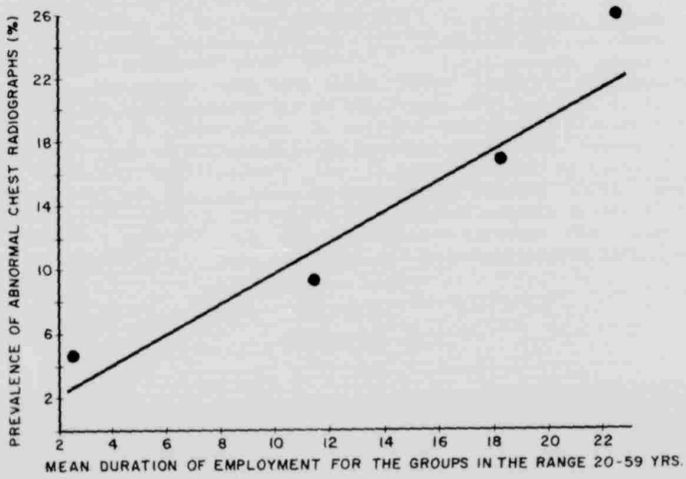


FIGURE 22-2. EFFECT OF DURATION OF EMPLOYMENT IN THE VARIOUS AGE GROUPS ON THE PREVALENCE OF RADIOGRAPHIC ABNORMALITIES OF THE CHEST

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TABLE 22-8

DISTRIBUTION OF NODULAR OPACITIES ACCORDING TO AGE AND DURATION OF EMPLOYMENT

Age Group	No. of Workers	Mean Age	Mean Duration of Employment	No. of Workers with Abnormality	
				Pinpoint or Micronodular	Questionable Nodularity
<20	32	18.7	1.0	0	0
20-	546	24.3	2.0	0	3
30-	427	34.9	11.6	2	2
40-	406	45.1	18.3	1	3
50-	493	54.2	22.6	4	7
>60	124	61.7	23.4	2	2
Total	2,028			9	17

Summary

Chest radiographs of 2,028 workers employed in a fiber glass factory were examined. Approximately 16% of the workers were found to have radiographic abnormalities. Increased lung markings, abnormal aorta, emphysema and abnormal heart were the most prevalent. Statistical correlation between the prevalence of all abnormalities and mean age, and between prevalence and mean duration of employment, in the various age groups, showed that the strength of the association is approximately equal. No difference in the prevalence of all abnormalities could be detected between office workers and production workers. All the radiologists made their diagnosis without knowledge of the worker's name, age, duration of employment and exposure category. One and the same classification of radiographic abnormalities of the chest was used by all readers.

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THE RESULTS AND SIGNIFICANCE OF HUMAN EPIDEMIOLOGIC STUDIES

George W. Wright

Introduction

DR. DECOUFLE: The next speaker will be Dr. George W. Wright. He is a consultant in occupational medicine and formerly Professor of Medicine of the School of Medicine of Case Western Reserve University.

Presentation

DR. WRIGHT: Prior to 1938, a small number of persons were exposed to airborne fibrous glass during pilot plant operations making glass wool. It was not until then that larger manufacturing operations began to employ a substantial number of workmen in this process. Now after 30 years, and based upon what we know of dust deposition, what pulmonary abnormalities should be looked for in order to determine whether glass fibers or particles in the air pose a hazard to health? Chronic bronchitis, pulmonary fibrosis, bronchogenic cancer and mesothelioma are the diseases that give us concern. Each of these can develop in the absence of any occupational influence. The question to be answered is whether there is an excess of any of these abnormalities not explainable by other causes in populations of employees exposed to the inhalation of particles or fibers of glass.

Looking for evidence bearing on this question, Wright, in 1968, reported the results of a chest radiographic study of the working population at the oldest existing large-scale fibrous glass manufacturing plant in the USA. The population studied was currently employed for more than 10 years. Many of them had been employed for 25 years. Those exposed in production of glass prior to its conversion into fibers were excluded.

Of the 1401 employees qualified for study, 11 were excluded because of substantial prior exposure to free crystalline silica in previous foundry employment. One person was excluded because his large size precluded making a satisfactory chest roentgenogram. A technically adequate current chest film, and in many instances serial films over the preceding years, were described by the author without knowledge of the employment or exposure history. Each film was compared to a set of standard films representative of the variations in size and density of the branching vascular pattern commonly observed in normal, healthy persons with no history of exposure in a dusty trade. This comparison permitted classification of the films into five categories in terms of the appearance of the branching vascular pattern. In modern radiographic parlance, this embraced, though was not identical to, the concept of small irregular densities expressed in the UICC classification. Category I was a "barn door" normal or 0/-, and Category V was similar, though not identical, to a 1/1 of the UICC classification. Other conventional radiographic appearances were also recorded.

In this entire set of films, the radiographic pattern of asbestosis or diffuse interstitial lung disease was not seen in a single individual.

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In two persons, a pattern of discrete nodular densities similar to that of silicosis was observed. Further study demonstrated each was caused by sarcoidosis.

Individuals were ranked in two ways, each attempting to classify them in categories of total exposure to respirable airborne glass fibers. The first classification was in terms of severity of exposure estimated on the basis of duration of employment in specific job designations believed to be different in intensity of dustiness. This classification was performed by a committee having detailed knowledge of work conditions going back to the beginning of plant operations aided by contemporary measurement of the dustiness of various jobs. In addition, the total study population was separated into clerical as contrasted to factory workers. The latter presumably was more heavily exposed. Comparison of the frequency of various radiographic findings shown by each of these groups, demonstrated no difference relatable to total exposure to fibrous glass. Wright concluded: "The effect, as revealed in chest roentgenogram, of 10 to 25 years of exposure in an environment containing airborne particles of fibrous glass has been studied. No unusual pattern of radiologic densities was observed. The frequency of various radiologic appearances known to occur in the general population was no higher in those with greatest exposure than in those with the least."

The employees of this same fibrous glass manufacturing plant were re-examined radiographically in 1971 by Nasr, Ditchek and Scholtens. They classified the radiographs in a similar but not identical way and observed no difference in the prevalence of radiographic abnormalities of the chest between office and production workers. The ages of the two groups were similar. They also observed that the prevalence of radiographic abnormality was equally related to the age and duration of employment, thus showing no excess influence of exposure as measured by the latter.

Recently, J.W. Hill et al [1973] have compared the frequency of "radiographic pneumoconiosis" and other abnormalities in a group of fibrous glass manufacturing workers and an observed control sample, matched for age and size. They observed no radiological difference between the fibrous glass exposed group and the controls. They also report that Gilson, examining the same films using the UICC classification, likewise concluded that "there were no significant differences in the radiographic appearances of the two groups."

De Treville, Utidjian, Hook and Morrice [1970] studied the medical history and pulmonary function of a population of fibrous glass manufacturing employees, stratified by age and duration of employment, but randomly chosen within each cell. They found no clear relationship between the degree of exposure and evidence of disease or alteration of pulmonary function. In a group of fibrous glass manufacturing employees, J.W. Hill et al [1973] observed no difference between those exposed and the controls with respect to pulmonary function other than a slightly lower FVC in the controls. They also found a greater prevalence of cough and phlegm in the controls.

These radiographic, medical and pulmonary function observations of employees exposed for 10 to 25 or more years in fibrous glass manufacturing

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demonstrate no adverse health effects in terms of chronic bronchitis or pulmonary fibrosis.

Gross et al, [1971] reported autopsy examinations on 20 fibrous glass workers whose exposure history ranged from 16 to 32 years. The autopsies failed to show evidence of fibrotic disease attributable to such exposure. In contrast to this experience, populations exposed to other dusts causing disease, for example, free crystalline silica or asbestos, show discernable evidences of pulmonary abnormalities well within 10 to 20 years. One would expect, that if fibrous glass is a potent cause of chronic bronchitis or pulmonary fibrosis, some evidence of an excess of these abnormalities should be observable by now.

No study thus far referred to has been designed to investigate the full post-employment period and the possible carcinogenic effect of the exposure experienced by workers in fibrous glass manufacturing. A study of the mortality pattern of employees of the oldest USA glass wool plant has been carried out by NIOSH and will be reported at this symposium. A preliminary report of this study by Wagoner [1974] indicated that no excess of malignancy of any kind was observed. The only category of disease showing a possibly significant excess was in "other respiratory diseases of a non-malignant nature exclusive of influenza and pneumonia." Of the 17 cases in this category, the majority were said to be emphysema or corpulmonale. Only 5 of the 17 had any mention of fibrosis on the death certificate. In none of these could the likelihood of exposure to free crystalline silica or coal dust be excluded. The importance here is the absence of an excess of bronchogenic or GI cancer or mesothelioma.

It is clear that the health experience of those exposed for long periods of time to airborne fibers in the manufacturing of glass wool for insulation or other purposes is not in the same category as those employees exposed to another fibrous material, namely, asbestos. There are several possible explanations for this difference of human experience.

There is evidence that fibrous glass introduced directly into the lungs of experimental animals does not evoke the same severity of inflamatory and fibroginic response characteristic of asbestos fibers even though comparable amounts of the same sizes of fiber are used. [Kuschner, 1974 and Gross, 1974] The reason for this is not clear but may be related to the fact that glass is amorphous while asbestos is crystalline. They are of different chemical composition even though both are silicates. Hence, their surfaces might behave differently when in contact with living cells.

Measurements of the concentration of respirable size fiber in the work-place where glass insulation is made or used, demonstrates the level is far lower for glass than for asbestos which has been produced or used. In the period of exposure responsible for the disease now occurring, asbestos fibers of comparable respirable sizes were 10 or more times as numerous as glass fibers. [Johnson et al, 1969] The lung residue of employees manufacturing fibrous glass shows many times fewer fibers per gram of lung tissue than is observed in the lung residue of persons occupationally exposed to asbestos. [Gross et al 1971 and 1974] This may be due to the lower concentration of respirable fibers in the fibrous glass

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manufacturing workplace. Perhaps the glass fibers do not penetrate as deeply into the lung, or are more effectively cleared, than asbestos fibers. To the extent that numbers of fibers resident in the tissue influences their biological effect, the outcome of exposure to airborne fibrous glass could be expected to be less than for asbestos.

There are lower numbers of respirable fibers in the environment of fibrous glass manufacturing than in asbestos. There is also a greater difference between the fibrous glass and asbestos environments in terms of the proportion of various sizes of fibers present. The number of fibers longer than 20 and thinner than 1 μ in the air of fibrous glass manufacturing is far smaller than in occupational environments where asbestos is present. Animal experimentation has shown that fibers in this category are biologically more active than those that are shorter or thicker. [Kuschner, this symposium; Stanton, 1974; Wagner, 1973; Gross, 1974] Quite possibly, the low dose potential of fibers of this category in the environment of fibrous glass wool manufacture bears on the absence of a demonstrable excess of mesothelioma in the employees of this type of operation.

It is also possible that glass fibers, even of appropriate size, do not migrate to the mesothelial surfaces whereas those of asbestos do. The fact that pleura plaques have not been observed in fibrous glass exposed employees suggests that failure to reach the pleura is the case.

The longest period of exposure to fibrous glass has been in employees exposed to the insulation and textile varieties, characterized by airborne fibers. The majority have diameters exceeding 3 μ, though a small proportion are thinner. In the past 15 years, fibrous glass of smaller nominal diameters, down to and below 1 μ, have been manufactured on a commercial basis.[Pundsack, 1974] Recent experimental animal studies indicate that, when long glass fibers of this diameter are placed directly into the mesothelial space, massive fibrosis develops accompanied by an excess occurrence of tumors designated as mesothelioma or sarcoma. [Stanton, 1974 and Davis, this symposium] These experiments obviously do not mimic the normal route of human exposure to long very thin glass fibers. The question has been raised as to whether or not inhalation of such fibers by humans poses a carcinogenic threat. Clinical and mortality data are not available as yet for those employees who have been exposed to the varieties of fiber glass where the majority of the fibers are thinner than 1 μ.

Some, though scant, information bearing on the question does exist. Long, very thin glass fibers have been present in the air breathed by those manufacturing glass wool these past 25 or more years, without evidence of excess occurrence of mesothelioma. As noted earlier, the dose may have been lower than might occur where such fibers are characteristic of the specific product. It should be noted, however, that for a 6-year period of time during and after the second world war some employees in the population from the plant reported by NIOSH as showing no cases of mesothelioma were engaged in manufacturing a product composed primarily of fibers averaging 1.5 μ and thus including some 0.5 μ in diameter. [Smith, this symposium] It is not justifiable to assume that glass fibers entering the lung will have the same effect as those introduced into the pleura. It is quite possible

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Even though the airborne dust contained some fibers less than 1 μ in diameter, the epidemiologic evidence shows that employees working 25 or more years in fibrous glass wool insulation manufacturing have not developed irreversible disease attributable to that exposure. It is not possible to determine at this time whether the production of materials in which the majority of the fibers are thinner than 1 μ constitute a more severe potential hazard than appears to be the case with the substantially thicker fibers characteristic of glass wool. Until this is resolved, one can agree fully with Dement that all feasible precautions to minimize the airborne concentration of this size fiber should be used. As suggested by Pundsack, we need not wait another 25 years to obtain additional information on this important question. Employees making mineral wool over the past half a century constitute a population exposed for a long time to a substantial concentration of these very thin long fibers. It is encouraging to learn that this opportunity has been recognized and study of this population will be embarked upon promptly.

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THE EPIDEMIOLOGY OF GLASS FIBER EXPOSURE AND A CRITIQUE OF ITS SIGNIFICANCE

J.W. Hill

Introduction

DR. DECOUFLE: The next paper is to is to be presented by Dr. Joseph Hill, Senior Medical Officer, Pilkington Brothers, Ltd.

Presentation

DR. HILL: In a large and important study in 1966, Wright, in the USA, surveyed periodical chest X-rays and medical records of a group of 1401 employees with a minimum exposure of 10 years in glass fiber manufacturing. He reported "no injurious pattern of radiological densities" and made the significant observation that "the frequency of various radiological appearances known to occur in the general population was no higher in those with the greatest exposure than in those with the least."

Nasr, in a further study, found that there was no difference in the prevalence of radiographic abnormalities between office and production workers.

Hill et al in 1971 compared a group of 70 workers with a mean of 19.75 years exposure in the manufacture of fiber glass wool insulation with a control group matched to within 5 years or less in age, 1 inch in height, 7 lbs in weight and residing in the same geographical area, to minimize independent environmental factors (mainly the effects of air pollution). The importance of as good a match as possible was felt to be vital by the authors and its value will be evident later.

In this study, the first comparison was made by X-ray of the chest using two consultant radiologists, reading randomly mixed radiographs without identification and then reviewing the films as a series of either glass fiber workers or controls, with knowledge of age, etc. A further inspection of the films was then made by Gilson (M.R.C. England) using the UICC/Cincinnati classification.

Further comparison was made by questionnaire as to respiratory symptoms and by lung function as measured by peak respiratory flow, FEV1 and F.V.C.

No evidence of respiratory hazard was found in any of these observations and indeed the control group had a slight excess of complaints of phlegm. This study was accompanied by as accurate a statement as possible of the dust conditions prevailing in the relevant wool insulation manufacturing condition.

It is important to realize that the major part of the glass fiber industry is concerned with either the manufacture of continuous filament by a drawing process used for textiles or reinforcement, and having a very

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narrow distribution curve of fiber diameter which tends to be above the respirable range, or in the manufacture of wool products for insulation by either steam blowing or a combination of rotary and steam blowing processes, which produces a wool with a much wider range of fiber diameter, probably containing 1 - 2% of submicron size fibers and containing larger fibers within the theoretically respirable range.

Wool insulation production has cost benefits attached to the fiber diameter such as to deter manufacturers from going much below their present specifications. The production of submicron filaments is therefore a small and specialist part of the industry which experiences difficulties in providing researchers with these small fibers.

It is reasonably safe to assume, therefore, that the quantity and size of fibers in the respirable fraction of the product have not changed greatly since its first introduction in the late 1930's.

The epidemiological studies, which include evidence showing no alteration of crude morbidity or mortality rates in workers or pensioners, are all the more important for being based upon a lengthy experience, and in Hill's case, of study upon a relatively stable population. Upon present epidemiological evidence, which can be held to represent the real life situation, glass fiber would have to be classified as an inert dust and there can be no scientific basis on epidemiological grounds for moving from the accepted threshold values for inert dust.

As there is no human response to which a dose might be related, the choice of any figure below the present value would be a matter of fancy and invite the question as to why not 1 mg above or 1 mg below a chosen value and upon what evidence a decision to make any change relies?

We must then turn to the question of animal response bearing in mind that this is one order of extrapolation removed from the epidemiological observations, and giving due regard to the mode of administration, eg the physiological reality of modes such as inhalation which is the most realistic, intratracheal injection which bypasses the upper respiratory filter, and intrapleural implantation, which bypasses all the normal defense mechanisms of the lung.

In a well controlled inhalation experiment, Gross considered that exposure to high concentrations of respirable glass dust for 1 year produced only minimal lesions in the guinea pig, as did similar exposure to intratracheal injections in rats. The lesions consisted of small focal alveolar dust cell collections without fibrosis, and in a subsequent experiment he found a similar response to fiber glass wool, phenol formaldehyde coated glass fiber and fibers with a starch coating.

Indeed, he and others find glass fibers to be characterized by minimal tissue reaction and rapid lung clearance as compared with asbestos, when the physiologically realistic route of inhalation was used.

It is important to note that in his experiments he found a substance as inert as carbon producing alveolar atalectasis, that he found no lung or pleural tumors, and noted rapid lung clearance with a tissue reaction which

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was potentially reversible. Gross therefore felt he could describe glass fiber as an inert dust by inhalation.

Kuschner, in a preliminary report at this meeting, has found some thickening of the septa in the region of the respiratory bronchioles at 6 months and some coarsening of the tissue with thickening and fibrosis after 1 year using intratracheal technique. Botham describes a particular type of reaction in the alveoli of guinea pigs by the same technique in an experiment of relatively brief duration.

As intratracheal injection bypasses the normal upper respiratory filtration mechanisms, and has its own particular technical problems, it remains a valuable research tool but is an order less representative than inhalation where extrapolation to humans is concerned, and one must therefore attach the greatest weight in terms of extrapolation to Gross' experiments which are essentially negative.

When we consider intrapleural implantation we are dealing with an entirely different sort of question, namely, what is the reaction of a given material when in direct contact with a specific tissue? We must be conscious of the several logical steps between this and the description of a hazard.

At present, there is no information available whatsoever concerning the transferability of glass fiber to the pleura, beyond Gross' statement that using microincineration of the lung he was unable to find glass fiber in or near the pleura.

Morgan at Harwell (England), using irradiated asbestos, has shown both a large tissue reaction and rapid sub-pleural transfer with this material. Such a technique could be applied to glass fiber and it would be interesting to discover whether the large tissue reaction of asbestos does facilitate sub-pleural tissue transfer as one would theoretically expect, in contrast to the minimal tissue reaction of glass fiber which might favor lung clearance via the satellite lymph nodes and lumen.

Interested in the possibility of human observations, Hill, in an ongoing study, repeated the chest X-rays after 5 years, of 53 out of the original 70 members of the glass fiber subject group, many of whom have now retired.

These reveal evidence of pleural thickening in a proportion of cases, and therefore, recalling that the sample is of an older age group residing in an area noted for chronic chest illnesses, the original control group was reviewed. Preliminary observations show the percentage findings to be much the same as in those of the exposed group.

One may next inquire whether mesothelioma is found in association with glass fiber exposure.

Employees in the glass fiber insulation process studied are drawn largely from a relatively confined geographical area, and diligent inquiries of the pathologists drawing from this catchment zone, who are all senior and of long experience in the area, has revealed only a small number

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of mesotheliomas, each associated with asbestos. The author and his colleagues have no knowledge of any case arising in fiber glass employees, and this may be sharply contrasted to the case of asbestos.

Although it must be recognized that both misdiagnosis and the proportion of cases not going to autopsy might disguise a hazard, this accumulation of experience suggests that as regard the wool insulation process there is an absence of compatability between field observations and intrapleural implantation experiments in animals.

This is more striking when one reminds oneself that the employee population has been relatively stable and that the wool making operation has been continuous since the late 1930's. The direct comparison is with the incidence of asbestosis, bronchial carcinoma and mesothelioma in the case of asbestos, and this points clearly to the paradox between the effect of asbestos and glass fiber. The author believes, on the present evidence, that the answer will be found in a difference in tissue transfer, dependent upon the minimal tissue reaction and rapid lung clearance of glass fiber and in the differing quantities and size distributions presented for inhalation.

This paradox, however, must also cause us to examine the evidence on intrapleural implantation closely. Accepting that implantation techniques will produce a proliferative reaction, how is the diagnostic 'barrier' to be crossed and hyperplasia distinguished from neoplasia?

Occasional mitoses are of little significance, secondaries rare in mesothelioma and death a doubtful index in a small animal readily subject to lung dysfunction. Perhaps the real test is invasion of the outer quarter of the lung parenchyma.

Noting that many of the tumors are classified as benign and that both pleural and peritoneal mesothelium are notable for the labile nature of their morphological reaction, with what degree of confidence can a diagnosis of mesothelioma be applied?

Confusion between the fibrous type and fibrosarcoma, which occurs in these species per naturam would be important as an increased incidence by itself. In the latter case it might be subject to the same criticisms as the now discredited mouse adenoma, particularly as tumors have been found in control series. Perhaps the critical question is whether neoplasia can be induced by a physiologically realistic technique such as inhalation. This has yet to be shown.

Bearing these perspectives in mind, the necessary ingredients to further investigation should be:

(1) A definitive experiment determining the tissue transfer characteristics of varying size ranges of glass fiber.

(2) A dose related response type of inhalation animal experiment directed toward the sub-micron range of glass fibers.

(3) Rationalization of the methods of measurement and mode of

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expression of the quantities of fibers met within environmental samples in given ranges of diameters and lengths in such a way as to be readily correlated with findings of research workers.

(4) Further epidemiological effort directed toward cohort studies of suitable populations with careful attention to control groups, and the nature of the exposure.

(5) Special attention should be given to populations involved in the manufacture of microfiber.

(6) Autopsy follow up.

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THE LUNGS OF FIBER GLASS WORKERS: COMPARISON WITH THE LUNGS OF A CONTROL POPULATION

Paul Gross
Russell A. Harley
J.M.G. Davis

Introduction

DR. DECOUFLE: Our fifth paper, "The Lungs of Fiber Glass Workers: Comparison with the Lungs of a Control Population," will be presented by Dr. Paul Gross.

Presentation

DR. GROSS: Epidemiologic studies on populations exposed to respirable dusts serve to recognize health hazards and thereby prevent suspected or even unsuspected illnesses in the future. Such studies are advisable even though no indisputable case of pneumoconiosis has been reported in the world literature in connection with exposure to a particular dust and even though data from animal experiments indicate that the inhaled dust is nonfibrogenic and can be categorized as a nuisance-type dust. It is also advisable to conduct, if possible, a study of the lungs of people who had been occupationally exposed to this dust for most of their working life time. This is to supplement and reinforce the animal data.

We had the opportunity to study the lungs of 20 workers who had died from various causes in a local hospital. They had been exposed to fiber glass dust for 16 to 32 years in the oldest fiber glass factory on earth. Their ages ranged from 38 to 81. Although one would hesitate to label such a study as epidemiological, its purpose was to supply information on the response of human lungs to long-term exposure to fiber glass dust, and on the amount of fiber glass dust that is likely to be stored in the lungs under conditions of such long-term exposure.

For the purpose of comparison, the lungs of 26 Pittsburgh adults and the lungs of 14 adults from Charleston, South Carolina were studied. Charleston and its environs represented a clean sea coast region. In contrast, Pittsburgh, until about 20 odd years ago, was recognized as the "smoky" city.

Materials and Method

The lungs of the fiber glass workers were received fixed in formaldehyde solution in an unexpanded condition. Several of the cases were represented by one lung only. A brief occupational history accompanied each specimen. An attempt was made to quantitate the degree of dust exposure on the basis of slight, moderate, or severe, substituting the numbers one to three. The pertinent data regarding these 20 workers are given in Table 25-1.

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The lungs of 12 men and 14 women from Pittsburgh were recieved fixed in formaldehyde and unexpanded from the Department of Pathology, University of Pittsburgh. The Pathology Department had received them from various hospitals in Pittsburgh. These people were presumed not to have been occupationally exposed to asbestos. Their ages ranged from 35 to 89, with an average of 63.6 years. All lungs were examined for gross abnormalities and representative blocks (usually six to ten) were removed for microscopic examination. The satellite lymph modes were dissected in some of these cases and processed separately. The lungs from the people of Charleston were received from the Department of Pathology, Medical University of South Carolina. They were processed in a manner identical to that of the other lungs. By means of a commercial meat slicer, the lungs were cut into slabs 5 mm thick and dried in a vacuum oven. The tissues were then defatted in acetone and dried again. Aliquots of the dried lung tissues were then removed for digestion. Initially, the formamide method of digestion was used, but this was changed to the sodium hypochlorite method when the former was found to destroy the ferruginous bodies in which there had been considerable interest at that time. [Gross et al, 1971a] The sediments resulting from the digestion were washed. After proper dilution, the fibers were counted according to the procedure previously published. [Gross et al, 1970a]

The dimensions of the fibers were estimated from photomicrographs taken of 120 to 200 random fibers in each case. The lymph nodes were also dried, defatted, digested with sodium hypochlorite, and the sediment prepared for fiber counting. For Electron Microscopic (E-M) counting, a known volume of a standard suspension of latex spheres, measuring 1 μ in diameter, was added to an aliquot of sediment suspension. After transferring a small drop of this mixture to an E-M grid and allowing it to dry, the number of fibers per latex sphere were determined and from this determination, the number of fibers per gram of tissue was calculated. [Davis and Gross, 1972]

The blocks of tissue from the lungs and lymph nodes were embedded in paraffin and sections were cut and stained with hematoxylin and eosin as well as with Gordon and Sweets' silver impregnation method. During the microscopic study of the sections, attention was focused on the occurrence, extent, and character of septal thickening and on its relation to dust particles, if such were present.

Results

Tissue Damage: Foci of dust accumulation, characterized by large black macrophages and some black extracellular particles, were common in the lungs of the fiber glass workers as well as in the lungs from Pittsburgh. In the lung of one of the fiber glass workers, a former coal miner, moderate anthracosis and scattered silicotic nodules were present. It was not possible to determine from the sections whether or not any mineral fibers were present in these dust accumulations. The dust foci did not differ from those seen in the lungs of most city dwellers (with the exception of those seen in the lungs of the former coal miner).

TABLE 25-1

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS AND SATELLITE NODES OF MALE FIBER GLASS WORKERS

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TABLE 25-1

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS AND SATELLITE NODES OF MALE FIBER GLASS WORKERS

Case	Age	Diagnosis	Exposure Years	Mineral Dust % (a) Lung	Mineral Dust % (a) Nodes	Number of (b) Fibers in Lung Op.	Number of (b) Fibers in Lung E-M	Number of Fibers in Lymph Nodes (b) Op.	Number of Fibers in Lymph Nodes (b) E-M
1	71	Myocardial Infarction anthracosis	25	1.9	5.0	20	520	20.4	28,500
2	81	Lymphosarcoma anthracosilicosis	17	2.9	3.0	27	61,700	24.1	--
3	49	Coronary thrombosis apical emphysema	24	4.2	3.3	290	2,000	35.8	40,500
4	65	Peritonitis bronchopneumonia	22	1.7	2.2	72	81,000	14.7	26,700
5	54	Renal carcinoma Pulmonary metastases	32	4.0	--	230	--	--	--
6	45	Hodgkin's sarcoma bronchopneumonia	24	1.9	0.7	210	295	17.5	3,900
7	64	Mitral stenosis emphysema	22	2.7	3.0	100	46,500	34.1	--
8	47	Coronary thrombosis pulmonary fibrosis	24	2.6	1.4	72	1,770	4.8	4,600
9	61	Myocardial fibrosis congestive heart failure	17	0.9	1.0	200	1,520	16.6	41,600

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TABLE 25-1 (CONTINUED)

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS AND SATELLITE NODES OF MALE FIBER GLASS WORKERS

Case	Age	Diagnosis	Exposure Years	Mineral Dust % (a) Lung	Mineral Dust % (a) Nodes	Number of (b) Fibers in Lung Op.	Number of (b) Fibers in Lung E-M	Number of Fibers in Lymph Nodes (b) Op.	Number of Fibers in Lymph Nodes (b) E-M
10	66	Coronary thrombosis pulmonary fibrosis emphys.	31	1.5	4.0	28	--	41.5	--
11	46	Coronary thrombosis myocard. infarction	18	2.2	1.5	90	1,360	11.9	11,800
12	38	Coronary thrombosis myocard. infarction	16	2.2	2.0	42	--	20.1	51,700
13	59	Dissecting aortic aneurysm card. tamponade	21	1.3	6.0	24	1,420	26.9	--
14	50	Portal cirrhosis Massive G-I hemmorhage	28	1.5	6.2	100	1,660	8.2	125,300
15	59	Aortic stenosis myocardial infarction	23	3.0	1.0	99	460	9.8	--
16	55	Myasthenia gravis atelectasis	25	1.1	3.7	90	1,750	6.9	14,700
17	50	Coronary thrombosis myocard. infarction	21	0.8	1.4	70	2,500	23.9	--
18	62	Bronchopneumonia encephalomalacia	30	1.5	--	82	--	--	--

TABLE 25-1 (CONTINUED)

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS AND SATELLITE NODES OF MALE FIBER GLASS WORKERS

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		myocard. infarction							
18	62	Bronchopneumonia encephalomalacia	30	1.5	--	82	--	--	--

TABLE 25-1 (CONTINUED)

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS AND SATELLITE NODES OF MALE FIBER GLASS WORKERS

Case	Age	Diagnosis	Exposure Years	Mineral Dust % (a) Lung	Nodes	Number of (b) Fibers in Lung Op.	E-M	Number of Fibers in Lymph Nodes (b) Op.	E-M
19	67	Perforated duodenal ulcer, peritonitis	27	1.4	--	22	--	--	--
20	61	Bronchogenic carcinoma agranulocytosis	28	1.5	--	47	--	--	--
		SUMMARY OF 20 CASES							
Range	38 to 81		16 to 32	0.8 to 4.2	0.7 to 6.2	20 to 290	295 to 81,000	4.8 to 41.5	3,900 to 125,300
Average	57.5		23.6	2.0	2.8	95	14,600	19.8	34,930
		SUMMARY OF CONTROLS							
Range	35 to 89			0.3 to 6.1	0.04* to 5.3	15 to 380	850* to 6,750	0.5* to 334	28,400* to 65,100
Average	63.6	Pittsburgh Controls		2.1		105			
		Charleston Controls		0.5	1.9	15.9	2,260	65.5	46,100

*Charleston Controls.
a. Based on dry, defatted tissue.
b. X100 per gram dry, defatted tissue.

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We looked for alveolar thickening, as an important criterion of significant chronic lung damage from inhaled irritant particles. Small and larger foci of alveolar thickening were found in the lungs of both population groups. Some of the alveolar thickening was of a bland, acellular character. The silver stains clearly demonstrated that much of this type of alveolar thickening was caused by the thickening of axial reticulin fibers and the deposition of argentophilic material around alveolar capillaries, a condition designated as alveolar sclerosis (Figure 25-1). [Gross, 1963] This has been found unrelated to occupational exposure and to occur in women as well. The frequency of occurrence was about equal in both population groups (Table 25-2). There were also foci of bland, acellular alveolar septal thickening in which there was a proliferation of connective tissue fibers. Many were collagenous rather than reticulin in character. These foci were found with slightly greater frequency in the lungs of the fiber glass workers than in the control population. They are believed to represent foci of healed chronic pneumonitis (Figure 25-2).

Cellular alveolar thickening, often associated with varying degrees of lymphocytic infiltration and the presence of many macrophages, was found in foci of varying size in many lungs of both population groups. More was found in the lungs of the fiber glass workers. These are foci of active chronic pneumonitis (Table 25-2) (Figures 25-3 and 25-4).

TABLE 25-2

SIGNIFICANT PATHOLOGIC FINDINGS IN LUNGS

Pathologic Finding	Glass Workers %	Controls %
Alveolar mural sclerosis	65	70
Septal collagenous fibrosis	70	52
Chronic pneumonitis, active	55	40
Chronic bronchitis	40	35
Emphysema	65	70
Pulmonary vascular sclerosis	25	37



FIGURE 25-1.
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FIGURE 25-3. A
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FIGURE 25-1. ALVEOLAR SCLEROSIS CHARACTERIZED BY THE THICKENING OF RETICULIN FIBERS AND THE DEPOSITION OF ARGENTOPHILIC MATERIAL AROUND CAPILLARIES. X 700



FIGURE 25-2. HEALED CHRONIC PNEUMONITIS CHARACTERIZED BY FOCAL AVASCULAR COLLAGENOUS THICKENING OF ALVEOLAR SEPTA. X 390



FIGURE 25-3. ACTIVE CHRONIC PNEUMONITIS CHARACTERIZED BY THICKENING OF ALVEOLAR WALLS ASSOCIATED WITH LYMPHOCYTIC INFILTRATION AND THE PRESENCE OF MASSED MACROPHAGES IN THE AIR SPACES. HEMATOXYLIN AND EOSIN. X 390

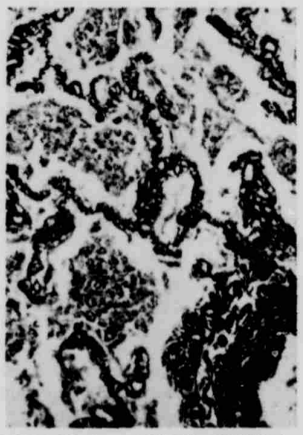


FIGURE 25-4. ACTIVE CHRONIC PNEUMONITIS SHOWING A SLIGHTLY DIFFERENT LEVEL. THERE IS PROLIFERATON AND THICKENING OF SEPTAL RETICULIN IF COLLAGENIZATION IS ABSENT. X 390

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Chronic bronchitis was found with nearly equal frequency in both population groups. Although no foci of bronchial epithelial metaplasia were found, there was one instance of bronchogenic carcinoma in the group of fiber glass workers. Emphysema, characterized by destruction of alveolar septa and enlargement of air spaces, was recognized in the sections of both groups with about the same frequency. The extent or the type of emphysema would not be determined from the sections. Sclerosis of pulmonary vessels was slightly more common in the control group.

The results of the histopathologic findings of the lungs of the two population groups (listed in Table 25-2) can be summarized as follows: chronic bronchitis, alveolar sclerosis, and emphysema occurred with about equal frequency in the two groups. Chronic pneumonitis, healed and active, was slightly more prevalent in the lungs of the fiber glass workers and sclerosis of the pulmonary vessels was slightly more prevalent in the control group. A bronchogenic cancer was present in the fiber glass worker group. According to his private physician, the worker who had developed a lung cancer had been a very heavy cigarette smoker for most of his life. The mineral fiber content of his lungs was less than one-half of the fiber glass workers' average fiber content and also less than one-half of the control group average fiber content.

With the exception of silicotic nodules in the satellite lymph nodes of two fiber glass workers, no fibrosis referrable to dust was noted in the satellite nodes examined.

Mineral Dust Content of Lungs

A comparative summary of a portion of the results is given in Table 25-3 and the data pertaining to the pulmonary mineral dust content of the individual cases of three groups of people are listed in Tables 25-1, 4, and 5. These data can be summarized as follows:

There was no relation between either the duration or the estimated severity of occupational exposure to the total mineral dust content or the mineral fiber content of the lungs of fiber glass workers.

The average number of optically visible mineral fibers in the lungs of fiber glass workers was similar to that of dwellers in an industrial city (Pittsburgh), but was considerably larger than that of city dwellers in a sea coast community (Tables 25-4 and 25-5).

The dimensions of optically visible mineral fibers from the lungs of fiber glass workers and the size-distribution of these fibers were very similar to those from the lungs of a control group (Pittsburgh), most of which were women (Table 25-4).

The content of Electron Microscopic (E-M) size fibers in the lungs of the people examined had no relation to the total mineral dust content, or the content of optically visible fibers in those lungs.

The presence of mineral fibers in the satellite lymph nodes indicated that these fibers are readily transported from lung to lymphoid tissue. The average total mineral dust content of satellite lymph nodes on a per-

TABLE 25-3

COMPARATIVE SUMMARY OF SOME RESULTS

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TABLE 25-3

COMPARATIVE SUMMARY OF SOME RESULTS

Items Compared	Glass Workers Ave.	Controls Ave.	P
Total dust	2.0 ± 0.9%	2.0 ± 1.1%	NS*
Fibers per gram of dry lung (thousands)	96 ± 76	105 ± 92	NS
Average diameter of fibers	2.3 ± 0.4 μ	2.1 ± 0.3 μ	NS
Average length of fibers	27 ± 6 μ	23 ± 6 μ	NS
Percent fibers ≤ 3 u in diameter	90 ± 9%	92 ± 9%	NS
Percent fibers 5 μ - 25 μ in length	67 ± 12%	74 ± 11%	NS
Distributional peak of fiber diameters	2.0 μ	1.5 μ	--
Distributional peak of fiber length	16 μ - 25 μ	16 μ - 25 μ	--

* NS, Not Significant

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TABLE 25-4

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS OF PITTSBURGHERS

Case	Age	Sex	Autopsy Diagnosis	Mineral Dust %	No. of Fibers in Lung X 1000*
1	57	F	Peritonitis, perforated duodenal ulcer	2.0	216
2	59	M	Anaphylaxis (penicillin)	1.6	87
3	77	M	Coronary thrombosis myocardial infarction	0.3	49
4	48	M	Hodgkin's disease	0.9	72
5	89	M	Myocardial infarction pulmonary embolism	3.3	170
6	69	M	Emphysema, hypertension uremia	6.1	102
7	72	F	Coronary occlusion, aorbic stenosis	0.9	168
8	56	M	Coronary thrombosis	4.2	52
9	70	F	Diabetes, pontine infarction	4.1	380
10	60	M	Encephalomyelitis, bronchopneumonia	2.1	83
11	45	F	Bronchopneumonia, fatty liver	0.9	296
12	35	F	Pulmonary embolism	1.7	183
13	47	F	Bronchopneumonia, hepatic cirrhosis	1.5	70
14	61	F	Coronary occlusion	2.9	255
15	68	F	Myocardial infarction, anthracosilicosis	1.9	52

TABLE 25-4 (CONTINUED)

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS OF PITTSBURGHERS

Case	Age	Sex	Autopsy Diagnosis	Mineral	No. of Fibers

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11	45	F	Bronchopneumonia, fatty liver	0.9	230
12	35	F	Pulmonary embolism	1.7	183
13	47	F	Bronchopneumonia, hepatic cirrhosis	1.5	70
14	61	F	Coronary occlusion	2.9	255
15	68	F	Myocardial infarction, anthracosilicosis	1.9	52

TABLE 25-4 (CONTINUED)

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS OF PITTSBURGHERS

Case	Age	Sex	Autopsy Diagnosis	Mineral Dust %	No. of Fibers in Lung X 1000*
16	63	M	Thrombocytopenia, massive hemorrhages	1.0	48
17	81	F	Uremia	1.7	78
18	42	M	Encephalitis	2.0	76
19	80	F	Ruptured aneurysm	1.7	80
20	75	M	Massive intracranial hemorrhage	2.7	15
21	59	M	Coronary thrombosis	2.3	66
22	69	F	Pulmonary embolism	1.9	15
23	70	F	Pulmonary embolism	1.8	21
24	74	F	Septicemia (E. coli)	1.3	21
25	81	F	Ruptured cerebral aneurysm	0.6	29
26	46	F	Gastric Ca. with metastases	2.0	63
Average	63.6			2.0	105
Ranges	35-89			0.3-6.1	15-380

* per gram dry, defatted tissue

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TABLE 25-5

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS AND SATELLITE NODES OF CHARLESTONIANS

Case	Age	Sex	Mineral Dust % of dry weight		Number Fibers * in Lung X 1000		Number Fibers * in Lymph Nodes X 1000	
			Lung	Nodes	Optical	E-M	Optical	E-M
1	45	F	0.3	1.1	8.0	855	20.0	28,400
2	71	F	0.9	4.1	6.0	6,750	53.0	37,200
3	79	F	--	0.3	53	1,260	38.0	55,800
4	78	F	0.7	4.5	34	1,040	55.0	65,100
5	54	F	0.3	0.1	5.0	--	102	--
6	60	F	0.7	0.3	20	1,390	334	43,800
7	60	F	0.7	1.3	20	--	238	--
8	86	F	0.7	2.0	19	--	53	--
9	48	F	0.9	0.2	0.4	--	0.5	--

TABLE 25-5 (CONTINUED)

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							238	--
8	86	F	0.7	2.0	19	--	53	--
9	48	F	0.9	0.2	0.4	--	0.5	--

TABLE 25-5 (CONTINUED)

DATA PERTAINING TO THE DUST FOUND IN THE LUNGS AND SATELLITE NODES OF CHARLESTONIANS

Case	Age	Sex	Mineral Dust % of dry weight		Number Fibers * in Lung X 1000		Number Fibers * in Lymph Nodes X 1000	
			Lung	Nodes	Optical	E-M	Optical	E-M
10	43	F	0.1	0.4	2.8	--	5.2	--
11	61	M	0.9	1.8	4.4	--	3.3	--
12	25	F	0.4	0.2	48.8	--	1.2	--
13	62	M	0.1	5.3	2.7	--	10.4	--
14	70	M	0.02	4.8	0.9	--	3.4	--
Ranges	25 to 86		0.02 to 0.9	0.1 to 5.3	0.4 to 53	855 to 6,750	0.5 to 334	28,400 to 65,100
Average	60.1		0.5	1.3	1.9	2,260	62	46,000

* per gram dry, defatted tissue

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gram-of-dry tissue basis was somewhat higher in fiber glass workers than in the control population. However, the average mineral fiber content (optically visible as well as E-M size) of the satellite lymph nodes was larger in the control group than in the fiber glass group.

In addition to this data, study of the mineral sediment remaining after digestion of the lung tissue showed that some fibers had irregular contours [Gross et al, 1970a] and some fibers were apparently derived from diatom fragments. [Gross et al, 1971b] Other work had suggested that many fibers may be phytoliths, derived from the ashes or smoke of burning plant products such as leaves, paper, wood, or coal. [Gross et al, 1971b]

Comments

It should be immediately apparent that the identity of the fibers counted and measured is unknown. Some of them are doubtlessly glass. Some may be of other vitreous material such as rock wool and more than a few may be asbestos fibers, inclusive of tremolite from cosmetic talc. Some of the E-M size fibers may also be diatom fragments. It is probable that a large proportion of the fibers are derived from the skeletons of plants (phytoliths). These have been demonstrated in the ashes of paper and wood. [Gross et al, 1971b]

In view of the fact that the dimensions of the fibers isolated from the lungs of the two population groups and the distribution of the dimensions are so similar, one is tempted to conclude that the bulk of the fibers in the two groups are therefore probably composed of the same material and derived from the same basic source. This conclusion could be flawed by the fact that the lung is an excellent size-selector and therefore, although the size range of the fibers is similar, they may be composed of different materials. An indication of this is seen in the wide variety and irregularity of shape of the fibers seen in the sediment of lung digestion. [Gross et al, 1970a]

Two conclusions seem inescapable, and these are: Whatever the composition of the fibers in the lungs of the fiber glass workers, their presence evoked no recognizable tissue response. A tissue response may have been present; but if it was present, it was so mild as to be unrecognizable. Fiber glass workers, exposed to fiber glass dust for 16 to 32 years, harbor fibers in their lungs similar to findings in the lungs of the control population with regard to quantity and dimensions.

It is interesting to consider that the quantity of mineral fibers found in the lungs from Pittsburgh, most of which were women, represents a "background" quantity, an amount that is (or has been) inherent in urban living and that has produced no discernable tissue reaction. Since fiber glass workers have no more than this "background" quantity of mineral fibers in their lungs even after 16 to 32 years of exposure to the dust of glass fibers, it would seem that either the fiber glass dust to which the workers had been exposed was not respirable or, as seems more likely, if it was respirable and had been deposited in the lungs, it was promptly removed by the pulmonary clearance mechanism, leaving only a "background" quantity of fibers.

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The data obtained from studies of the lungs of long-term fiber glass workers are in agreement with the conclusions derived from previous animal experiments [Gross et al, 1970b] that: Fiber glass dust inhaled and deposited in the lungs causes no harmful tissue reaction. Fiber glass dust deposited in the lungs is readily removed from the lung tissue by the pulmonary clearance mechanism.

Pathological examination of the lungs of long-term fiber glass workers as well as determinations of the pulmonary mineral fiber content disclosed that no discernable tissue reaction to fiber glass dust was present and that the average quantity and dimensions of the mineral fibers in the lungs of fiber glass workers were very similar to those of the mineral fibers in the lungs of a control population. Two conclusions are drawn:

Fiber glass dust, inhaled and deposited in the lungs, causes no harmful tissue reaction.

Fiber glass dust, deposited in the lungs, is readily removed from the lung tissue by the pulmonary clearance mechanism.

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THE COMPARATIVE PATHOGENICITY OF INHALED FIBROUS GLASS DUST

Gerrit W.H. Schepers

Introduction

DR. DECOUFLE: The next paper will be presented by Dr. Gerrit Schepers, who is Professor of Medicine at the State University Medical School in Hershey, Pennsylvania.

Presentation

DR. SCHEPERS: This paper will address itself precisely to the aspects mentioned in its title. This author has, over the past three decades, conducted numerous animal experiments with a variety of glass and glasslike materials, employing diverse techniques. Many of these studies have been published. [Schepers and Delahant, 1955; Schepers, 1955, 1958, 1959, 1960, 1961, 1962, 1965, 1969, 1971a, 1971b, 1971c, 1974; Schepers et al, 1958] Reference to the publication should be made for experimental methodology and protocol. Experiences with animals are synthesized in this paper.

In papers to be presented at this conference, subjects quite similar to the above will have been covered. It would, therefore, be redundant to recapitulate all that has already been placed in the literature. A contribution limited in emphasis to inhalation experiments seems likely to be able to complement other reviews. Some experimentation also has been personally conducted on a small number of primates. This may add more information since primates have not been mentioned by other conferees.

Clinical biopsy and necropsy investigations have also been accomplished on a number of human subjects who had been occupationally exposed to glass dusts of various kinds. All these items will be considered in this paper.

Finally, some comparisons will be made between the effects of inhaled glass and certain other fibrous minerals studied in comparable manner. There are many uncertainties concerning which environmental pollutants are truly injurious and to what extent they do cause harm to human lungs. An attempt will, therefore, be made to evaluate the biological action of fibrous glass against a numerical scale of pathogenicity recently devised for fibrous minerals. [Schepers, 1974] Studies by other authors have been reported for animal and human subjects. [Gross, 1968; Gross et al 1970a, 1970b, 1971a, 1971b, 1973; Timbrell, 1965; Wright, 1968; Von Koelsch, 1956; Bernabei, 1950; Bjure et al, 1964; Crawley, 1966; Desoille and Dhers, 1946; de Treville et al, 1970; Kahlau, 1947; Konzen, 1968; Lambiotte, 1968; Leder, 1945; Magaldi, 1951; Mauro, 1953; Milby, 1969; Murphy, 1961; Nasr, 1971; Roche, 1946; Steele, 1948; Tara, 1944-45; Trumper, 1946; Utidjian, 1968] These publications provide background data for conclusions stated at the end of this paper.

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Materials and Methods

Substances studied include glass fibers derived from various sources. They varied in caliber and length. Glass flakes also were studied. Comparisons were made between micronized fibers and longer fibers. Other studies were made on dusts and materials obtained from fiber glass plastic factories. One series of studies was made on volcanic glass (Perlite). The biological activity of the glass fibers was studied by cutaneous, subcutaneous, intravenous, intraperitoneal, and intratracheal injection and by inhalation techniques. Some glass materials also were consumed by animals along with their food. The propensities to stimulate tuberculosis and to induce lung cancer also were explored. Rats, guinea pigs, rabbits and monkeys served as experimental subjects. For each group of experiments appropriate control animals were used. The general methods of study have been described elsewhere. [Schepers and Delahant, 1955: Schepers et al 1958; Schepers 1955, 1958, 1959, 1960, 1961, 1962, 1965, 1969]

The components of average fibrous glass are in Table 26-1. The chemical composition of various types of glass from which the glass fibers or flakes are derived is summarized in Table 26-2. The slender fibers are of greatest interest since they are the most likely to get into the smallest recesses of the lungs. It is, therefore, important to note that approximately 20% of the fibers were of the 2 μ and smaller range. These varied from over 50 μ in length to less than 5 μ. Thirty percent of the fibers were 5 μ or shorter. Figure 26-1 shows typical fibers matched against red blood corpuscles to dramatize their relative diameters.

The components of fiber glass plastic dust are summarized in Table 26-3. In this material the polymerized resin predominates (61-65%) and the glass fibers constitute only about a quarter of the dust. Fiber glass plastic with two types of fillers were studied, namely calcium carbonate and calcium sulfate. It is known that there are plastic reinforced glass materials in which other fillers (eg China clay) are used. There also may be trace quantities of pigments and quartz. The plastic is a polymer derived from the chemicals listed in Table 26-4.

The total number of animals studies is given in Table 26-5. As is evident the majority of studies involved inhalation experiments. Before reviewing the results obtained by the latter modality, it may be worthwhile to briefly review certain results of the other studies.

Reactions of Animals to Miscellaneous Routes of Exposure

The types of experiments, substances, numbers and types of animals, duration of the studies and the prevalence of lesions are summarized in Table 26-6. Altogether, 71 animals were exposed to fibrous glass substances through techniques other than intratracheal injection or inhalation. Attention may be principally directed to the final column, which summarizes the prevalence and severity of all lesions evoked by these techniques in all affected organs. These are all highly artificial techniques but do bring to light that if the components of fiber glass materials are introduced intravenously or intraperitoneally, moderately severe tissue reactions may be observed in multiple organs such as the

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TABLE 26-1

COMPONENTS OF AVERAGE FIBROUS GLASS

Silica Oxide	52 - 54
Calcium Oxide	16 - 18
Aluminum Oxide	13 - 16
Barium Oxide	6 - 10
Magnesium Oxide	3 - 5
Sodium Oxide	1/2 - 1
Iron Oxide	1/4 - 1/2
Potassium Oxide	1/4 - 1/2
Water	1/2 - 1

TABLE 26-2

FIBROUS GLASS

AVERAGE SIZE DISTRIBUTION AS AEROSOL

Diameters μ	%	Lengths * μ	%
10 +	60	50 +	39
5-9	7	20-50	12
2-4	3	5-20	19
2-	20	5-	30

*Lengths of only fibers with 2 μ or less diameter.
Smallest diameters; 0.2 μ

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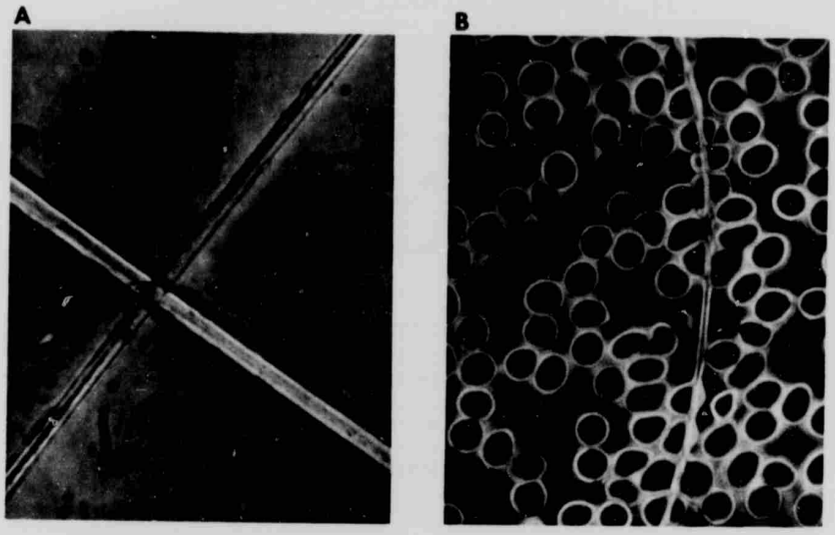
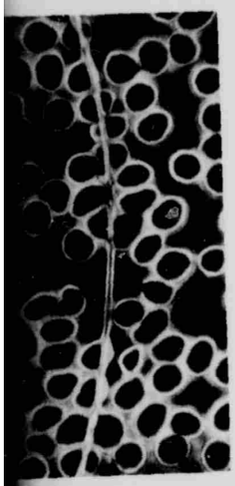


FIGURE 26-1. OPTICAL APPEARANCE OF TYPICAL GLASS FIBERS. PHASE CONTRAST MICROSCOPY. (X 1170)

A. Two fibers crossing. Caliber 5 and 6 (u) respectively.

B. To better illustrate fiber size a 2 (u) fiber has been incorporated in a human blood smear. Fiber caliber is readily apparent if one recalls that the average erythrocyte diameter is about 7 (u).

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TABLE 26-3

FIBER GLASS REINFORCED PLASTIC

Components	Percent
Glass Fibers	24 - 28
Resin	61 - 65
Fillers *	6 - 11
Pigments	0 - 1
Quartz	1/4-1/2

* CaSO 4, CaCO 3, China Clay

TABLE 26-4

COMPONENTS OF FIBER GLASS PLASTIC

Maleic anhydride
Phthalic anhydride
Ethylene glycol or
Propylene glycol
Styrene monomer *
Benzoyl peroxide
Retardants ?

*Polymerizes to styrene

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TABLE 26-5

TOTAL ANIMALS STUDIED

Route / Objective	Animals Test	Controls
Injections - Toxicity	71	120
Tracheal - Pathology	376	231
Inhalation - Pathology	400	708
Inhalation - Tuberculogenicity	681	515
Inhalation-Oncogenicity	700	148
Totals	2228	1788

TABLE 26-6

ANIMAL EXPERIMENTS
MISCELLANEOUS ROUTES OF EXPOSURE

Route And Dose	Species	Substance & Amounts	Animal No.	Months	Average Lesions All Organs
Venous 1% x 20	Rabbit	Glass wool 20-50μ	12	18	++
	Rabbit	Flake glass - Milled	5	10	++
	Rabbit	Resin - Milled (1000 mg each)	5	17	++
Peritoneal 5% x 1	Guinea pig	Glass wool 20-50μ (1000 mg)	20	12	++
Ingestion 100 mg/day	Guinea pig	Glass wool 20-50μ (35 grams)	20	12	0
Subcutaneous 5% x 1	Rats	Fiber glass (50 mg)	5	12	+++
Cutaneous Infriction 1% x 20	Rabbit	Flake glass (100 mg)	2	12	0
Ocular Instillation 1% x 20	Rabbit	Flake glass (100 mg)	2	12	++
Controls	Rat	Room air only	50	18	+
	Guinea pig		50	24	0
	Rabbit		20	24	+

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Controls
120
231
708
515
148
1788

al	Months	Average Lesions All Organs
	18	++
	10	++
	17	++
	12	++
	12	0
	12	+++
	12	0
	12	++
	18	+
	24	0
	24	+

lungs, kidney and liver, provided enough time is allowed to elapse. However, since control rabbits not subjected to dust injections had a slight number of comparable lesions in the same organs, the contributary role of the injected dust is commensurately reduced. So, in final analysis, one may summarize to say that the reaction to the injected glass wool, fiber glass and resin was slight. Attention may also be drawn to the relatively large doses used (1000 mg).

It is noteworthy that ingestion of glass fibers in large quantity (35 grams) over a period of 100 days had induced no lesions in any organ by the end of 12 months.

Subcutaneous injection of fiber glass did, however, produce a quite marked local tissue reaction. Many of the lesions looked exactly like fibrosarcomatous tissue. However, none of these lesions behaved in the manner of a neoplasm. The observation was not continued beyond 12 months so that our assessment of these lesions must be moderated by this fact.

Flake glass had no effect on the skin when repeatedly rubbed into it in quite a vigorous manner. On the other hand, the flake glass caused moderately severe conjunctivitis and occasional minimally severe focal keratitis. These experiments were cut off at the end of 12 months, so that our observations must be regarded with some reservation.

Reactions of Animal Lungs to Intratracheal Injection of Fibrous Glass

Tables 26-7, 26-8, 26-9 and 26-10 summarize the experiments performed on rats, guinea pigs, rabbits and monkeys. Attention may be specially directed to the two final columns in each table summarizing mortality, prevalence and severity of lung lesions.

Rats

Since 22% of control rats died during the 24 months of observation, the relatively high mortality following intratracheal injection of suspensions of fiber glass plastic dust (70%, 45%) does not appear to be so wholly bad. However, the controls were continued for 24 months, whereas the experimental dust studies were cut off at 12 months. Of the control rats only 4% had died by the end of 4 months. This suggests that the mortality from intratracheally injected fiber glass plastic is really significant. That it is neither the resin nor the fillers (CaSO 4 or CaCO 3) which produced the lethal outcome is revealed by the 9% and 2% mortality following the intratracheal injection of milled vycor and flake glass; the 6% mortality following injection of milled resin plus a filler without glass but the 40% mortality following the intratracheal injection of 37.5 mg of fibrous glass. Clearly the glass in fibrous form was the factor in causing the high mortality for rats.

The lesions found in the lungs of these animals should again be discounted against the mild prevalence of lesions in controls. On this basis milled vycor glass and fiber glass evoked no significant tissue

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TABLE 26-7

INTRATRACHEAL EXPOSURE OF RATS
TO 37.5 mg* FIBROUS GLASS

Substance	Animal No.	Months	Mortality Percent	Average Lung Reaction
Vycor glass - milled	16	12	9	+
Fiber glass + CaSO 4	10	12	70	++
Fiber glass + CaCO 3	10	12	45	+++
Plastic + CaCO 3 (milled)	10	12	6	++
Fiber glass only	10	12	40	+
Flake glass (milled)	10	12	2	++
Glass wool 20 - 50μ **	21	20	19	+++
Controls	56	24	22***	+

* As 5% aqueous suspension, 3 doses at weekly intervals
** Three varieties ranging from less than 3μ to 8μ in caliber
*** By the end of 12 months only 4% of the control animals had died.

TABLE 26-8

INTRATRACHEAL EXPOSURE OF GUINEA PIGS
TO 75 mg* FIBROUS GLASS

Substance	Animal No.	Months	Deaths Percent	Average Lung Reaction
Glass wool: Milled	24	18	4	0
Glass wool: 20-50 μ	20	12	16	++
Fiber glass 20-50 μ	20	12	12	+
Fiber glass + CaSO 4	10	12	10	++
Fiber glass + CaCO 3	10	12	0	+++
Plastic + CaCO 3 (milled)	10	12	50	+++
Fiber glass only	10	12	0	+
Perlite	160	12	3	+
Flake glass (milled)	10	12	0	++
Controls	150	24	0	0

* As 5% aqueous suspension in 3 weekly doses

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Mortality Percent	Average Lung Reaction
9	+
70	++
45	+++
6	++
40	+
2	++
19	+++
22***	+

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Deaths Percent	Average Lung Reaction
4	0
16	++
12	+
10	++
0	+++
50	+++
0	+
3	+
0	++
0	0

reactions within 12 months and the reactions to fiber glass plastic dust with a calcium sulfate filler and to the resin with calcium carbonate, but no glass, may be rated as slight. The moderately severe response to the intratracheally injected suspensions of fiber glass plastic with calcium carbonate filler is clearly in part a factor of the presence of the calcium carbonate filler rather than the resin or the glass.

Guinea Pigs

None of the control guinea pigs had died even after 24 months (Table 26-8). This then focuses attention on the high mortality (50%) following the injection of the milled polymerized plastic resin with its calcium carbonate filler. In this respect guinea pigs, therefore, differed markedly from rats which showed a low mortality. This also contrasts with the zero mortality by the end of 12 months following the intratracheal injection of the fiber glass plastic with calcium carbonate filler. Apparently fine milling of the resin is the lethal ingredient. The fiber glass without the plastic or filler caused no deaths. It is not clear why 45% of the rats died after receiving the fiber glass plastic (CaCO 3 filler) whereas no guinea pigs died after receiving a considerably larger dose of the dust (75 mg). The 16%, 12%, and 10% mortalities associated with intratracheal injection of long glass fibers (glass wool 20-50 μ) and fiber glass (20-50μ) and the fiber glass plastic with calcium sulfate filler, clearly point to increased fiber length as a significant factor in determining the lethal outcome.

Lung tissue lesions also are noteworthy in the guinea pigs. None of the controls had any abnormalities even at the end of 24 months. It may be pointed out here that this was a specially bred colony of albino guinea pigs derived from a single pair with no cross contamination with any other strains or colonies. The absence of pulmonary lesions in the controls was, therefore, expected. This fact lends greater significance to the moderately severe lesions encountered after injection of glass wool, fiber glass plastic with CaSO 4 filler and milled flakeglas. The marked severity of the lung reaction to fiber glass plastic dust with CaCO 3 filler is rather clearly a correlate of the resin and filler rather than an effect of the fibrous glass only, which induced a slight tissue reaction only.

Rabbits

The epizootic or spontaneous mortality among the control rabbits was high (50% in 24 months). Time, of course, is a factor in this matter, so that comparisons of the high mortalities among the test groups should be made with that of controls at contemporaneous cut off points. Viewed this way the mortality of 50% and 45% respectively in rabbits which received 300 mg of fiber glass plastic with CaSO 4 and CaCO 3 fillers, gains greater significance. Rather clearly, the polymerized resin and the fillers may have played a significant role with respect to the lethal outcome. Obviously, calcium sulfate is more poisonous for both rats and rabbits then it is for guinea pigs. The reason for this was not uncovered. However, the high, relatively rapid lethal effect of the injected glass fibers (40% mortality in 8 months) again is in contrast to the zero effect on guinea

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TABLE 26-9

INTRATRACHEAL EXPOSURE OF RABBITS
TO 300 mg (as 5% suspension*) FIBROUS GLASS

Substance	Animal No.	Months	Mortality Percent	Average Lung Reaction
Fiber glass + CaSO 4	5	9	50	+
Fiber glass + CaCO 3	7	18	45	++
Plastic + CaCO 3	5	12	20	+
Fiber glass only	5	8	40	++
Flake glass (milled)	5	12	2	++
Controls	20	24	50 **	+

* In 3 doses at weekly intervals
** Mortality at the end of 12 months was 11% and at 18 months it was 37%.

TABLE 26-10

INTRATRACHEAL EXPOSURE OF MONKEYS
TO 500 mg* FIBROUS GLASS

Substance	Animal No.	Months	Mortality Percent	Average Lung Reaction
Fiber glass + CaSO 4	5	12	0	++++
Controls	5	18	0	+

* As 5% aqueous suspension in 3 weekly doses

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GLASS

ortality Percent	Average Lung Reaction
50	+
45	++
20	+
40	++
2	++
50 **	+

18 months it

ality cent	Average Lung Reaction
0	++++
0	+

pigs and conformant to the high mortality in rats. That it was the fibrous form of the glass rather than the glass as a chemical which brought about the death of these rabbits is revealed by the low mortality in the group which received the milled flake glass intratracheally.

The pulmonary tissue reactions were relatively insignificant and in distinct contrast to the lethal outcome. There was a mild pulmonary reaction only to the fiber glass plastic with calcium carbonate filler, to the glass fibers and to the milled glass flakes.

Monkeys

Only one experiment (Table 26-10) was conducted, namely intratracheal injection into 5 Macacus rhesus monkeys of 500 mg of fiber glass plastic dust with the calcium sulfate filler. The animals were kept alive for 18 months and then killed, since none had died. A relatively severe chronic pneumonitis was found in all animals (Figure 26-2). The response indicated that the monkey is inclined to react much more severely to this type of fiber glass plastic dust than did the other three species. This matter will again be broached when we review the response of the monkey lung to inhaled fiber glass plastic dust.

Pulmonary System Dimensions and Retention of Dust Particles

For general understanding, one should first consider the dimensions of the respiratory tract at various levels. It has been contended that glass fibers and flakes are too large and too coarse to be respirable. As is clear from the schematic representations of Figure 26-3, the dimensions of the respiratory tract are sufficiently wide to permit any of the glass particles of the sizes mentioned in Table 26-2 to traverse to the ultimate reaches of the alveolar parenchyma. Whether the particles would settle out rather too soon along the respiratory tract to reach the alveoli may be evaluated from these charts. It appears theoretically likely that in a mix of particles such as that shown in Table 26-2 there would be enough small particles that would get through. This can be debated at length. The proof surely lies in the results obtained by inhalation experiments.

Inhalation Experiments

These studies are summarized in Tables 26-11, 12 and 13. The large number of animals used and the long periods of observation render possible relative decisive evaluations of the differential effects of various fibrous glass dusts on different species. The inhalation experiments were continued for relatively longer periods than were the others already discussed. This may have had an influence on the mortality trends and on the genesis of specific lesions.

Mortality trends are clearly brought out in Table 26-11. The relatively low death rates for guinea pigs is in striking contrast to the higher mortality rates for rats and monkeys. There is, however, an excess mortality of 12% in guinea pigs exposed to glass wool and glass cotton for

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44 months. The spontaneous death rates were relatively high for the rabbits and rats. When these rates for rabbits and rats are discounted against the death rates among the test animals, the picture is not quite so gloomy. Thus, the excess mortality for rabbits in the experiment with inhaled fiber glass plastic dust actually is lower than among undusted controls.

For rats, the excess mortality in the glass wool - glass cotton experiment is corrected to only 10%. The mortality following exposure of rats to fiber glass plastic dust was less than that of the controls when the filler was calcium sulfate and about the same as the controls when the filler was calcium carbonate. It again defies immediate explanation why inhalation of the fiber glass plastic dust with calcium sulfate filler proved so much less harmful for rats and rabbits than when the substance was introduced intratracheally (Tables 26-7 and 26-9). It seems reasonable to postulate that the CaSO 4 can be trapped by pulmonary phagocytes and thus evacuated if the material is inhaled in small quantities daily, whereas a large slug of the chemical is likely to be largely absorbed after intratracheal injection and thus work its lethal effect.

The mortality figure of 20% for the monkeys is of course skewed by the small number of animals in the study. However, it is significant that even one of the five animals died within 6 months after dust exposure was started. The significance of this will be more fully evident when the pulmonary histology is discussed.

The prevalence of pulmonary lesions is summarized in the final column of Table 26-11. Since none of the 300 control guinea pigs showed pulmonary tissue abnormalities even after 36 months, the high prevalence of tissue reactions (57%) in the glass wool - glass cotton experiment is particularly important. In the fiber glass plastic experiments, the guinea pigs were sample killed at 2 monthly intervals throughout the inhalation phase. The finding of significant lesions in 22 of the 40 animals in the CaCO 3 experiment signifies virtual 100% prevalence in the group which inhaled the dust for the longest period. Likewise, the 14 guinea pigs of the CaSO 4 experiment with significant lesions represent animals which remained in dust for the full 15-month period.

Similar interpretations apply for the numbers of rabbits and rats with significant lesions. Some of these animals were also sample killed during the course of the experiment so that the final numbers of animals represent more nearly all the animals surviving to the end of the experiments. The only exception to this statement is the small number of rats with significant lesions listed for the fiber glass plastic with calcium sulfate experiment. In this case, the experiment was aborted after only 9 months, so that the lack of lesions corresponds to the equivalent 9-month stage in the other experiments involving rats. The high prevalence of significant lesions in the monkeys deserves special emphasis. One animal died relatively early in the experiment but grossly did not show much pulmonary disease. Exposure of the remaining four was aborted after only 8 months. Yet all these four animals showed significant pulmonary pathology.

Substance

Glass wool *
and
Cotton **

Fiber glass +
CaCO 3 ***

Fiber glass +
CaSO 4 ****

Perlite *****

Controls

* 0.145
** 0.03 m
*** 4.6 mg
**** 3.8 mg
***** 2.3 mg
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TOTAL ANIMALS

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TABLE 26-11

FIBROUS GLASS

INHALATION EXPERIMENTS

Substance	Species	Animal No.	Months	Mortality Percent	Pulmonary Lesions
Glass wool * and Cotton **	Guinea pigs	100	44	17	57
	Rats	50	28	60	16
Fiber glass + CaCO 3 ***	Guinea pigs	40	24	4	32
	Rabbits	18	18	36	10
	Rats	30	18	6	15
	Monkeys	5	8	20	4
Fiber glass + CaSO 4 ****	Guinea pigs	63	15	0	14
	Rabbits	18	15	17	11
	Rats	20	9	55	3
Perlite *****	Guinea pigs	40	24	6	9
	Rats	30	18	8	4
Controls	Guinea pigs	300	36	5	0
	Rabbits	90	24	40	10
	Rats	310	24	50	2
	Monkeys	8	24	0	7 *** ***

* 0.145 mg/cu m
** 0.03 mg/cu m
*** 4.6 mg/cu m : 338 million particles /cu ft
**** 3.8 mg/cu m : 283 million particles /cu ft
***** 2.3 mg/cu m : 194 million particles /cu ft
****** Exclusive of mite lesions, there were no pulmonary lesions of relevance.

TOTAL ANIMALS : Test 400 - Control 708

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FIGURE 26-2. MONKEY LUNG. REACTION TO FIBER GLASS PLASTIC WITH CALCIUM SULPHATE FILLER DUST ADMINISTERED INTRATRACHEALLY. (X1)

Photograph of midplane sagittal section through both lungs. Demonstrates bilateral apical pneumonitic consolidation and focal infiltrates and minimal emphysema in the lower lobes. Animal was killed at the end of 12 months following intratracheal injection of a 5% aqueous suspension of 1500 mg of fiber glass Ca S04 filler dust administered in 3 equal doses at weekly intervals.

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Pulmonary Histopathology: Inhalation Experiments
Glass Wool and Glass Cotton

Guinea Pigs

The pulmonary tissue reactions developed progressively over the 40 months of exposure. The characteristic lesions involved the bronchial epithelium, peribronchiolar structures and pulmonary parenchyma, particularly in the subpleural region. The bronchi and bronchioles showed crypt formation and papillary outgrowths. There was bronchiolar stenosis and bronchiolectasia, as shown in Figure 26-4. Around the bronchioles focal alveolar epithelization occurred. This apparently was an extension of bronchiolar epithelium into alveolar zones, but may also represent metaplasia of alveolar epithelium. The parenchymal lesions were of two kinds (Figure 26-5). There was considerable accumulation of phagocytes filled with glass fibers. In other lesions, these glass filled koniophores occur as isolated or multinucleated cells and there also was infiltration of alveolar walls by koniophores. The pleura was focally thickened through infiltration by cells filled with glass particles cells. There was no pleural fibrosis or mesothelial proliferation.

Probably the most outstanding abnormality found in the rats which died was pneumonia with alveolar focal suppuration in about one-third of the cases. This probably was a non-specific lesion since it was also found in control undusted rats. The excess mortality of 10%, therefore, appears attributable to the inhaled glass wool fibers. In animals that were killed, pneumonic lesions were seen in only one case and an abscess was found in another. Specific lesions were not identifiable though there was abundant macrophage accumulation in subpleural alveolar spaces of animals which survived longest. The bronchiolar distortion, bronchiolectasia and the alveolar epithelization described for guinea pigs was not seen in the rats. However, it should be kept in mind that the exposure of the guinea pigs continued through to 44 months, whereas there were no rats left in the study beyond the 28th month of exposure to the glass wool.

Fiber Glass Plastic Dust

Guinea Pigs

The histologic changes in the lungs were remarkably uniform in this species. They evolved progressively and were fully established by the end of 18 months. In the tracheobronchial lymph nodes the lesions caused by the fiber glass plastic came to maturity somewhat earlier, being fully evoked by about 10 months.

In the lungs, the earliest defined reaction consisted of koniophore accumulation within alveolar spaces (Figure 26-6a). These koniophores became progressively confluent (Figure 26-6b) and became grouped in relation to the small bronchioles (Figures 26-6c and d). At this stage outgrowth of bronchiolar epithelium into adjacent alveoli could also be observed and some of the alveolar spaces were minimally distended (emphysema) and others somewhat collapsed (atelectasis). The characteristic appearance of the fully developed guinea pig reaction is shown in Figure 26-7a, which displays numerous parabronchiolar macules of

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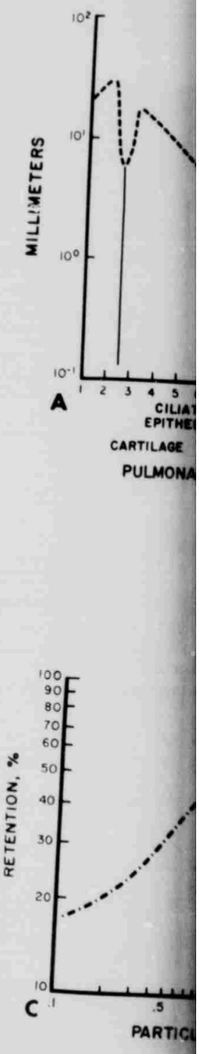


FIGURE 26-3. DIMENSIONS OF THE RESPIRATORY TRACT WHICH MAY INFLUENCE PENETRATION AND RETENTION OF PARTICULATE AEROSOLS AND THE INFLUENCE OF PARTICLE SIZE AND RESPIRATORY RATE ON THE RETENTION OF INHALED PARTICLES

A. Diameters

The graph shows that there is no point along the respiratory tract where there is an anatomical cross sectional dimension less than 200 μ. Under physiological conditions (e.g. contraction of bronchiolar smooth muscle) this dimension may decrease to about 100 μ. Since the transition from ciliated to desquamative epithelium corresponds to the junction of bronchioles and alveolar ducts, and this also is where smooth muscle ends, this region would constitute the closest approximation of a "bottle neck." Particles up to 200 μ may, therefore, penetrate alveolar spaces.

B. Surface Areas

The lung surface increases progressively beyond the segmental bronchi. Air flow would, therefore, slow down proportionately and facilitate drop off and retention of particles. Because of the ciliated nature of bronchial and bronchiolar epithelia, particles lodged here are likely to be conveyed out of the respiratory tract shortly after deposition.

C. Particle Size

When breathing is "normal," retention of particles up to 10 μ in size is a direct factor of particle size. Particles less than 0.1 μ in diameter are still retained but behave differently than larger particles.

D. Breathing Rate

Rate of breathing does not affect retention of 5 μ particles to a significant degree. Smaller particles are retained more readily if breathing is slow. Extremely minute particles are retained less readily if breathing rate is accelerated.

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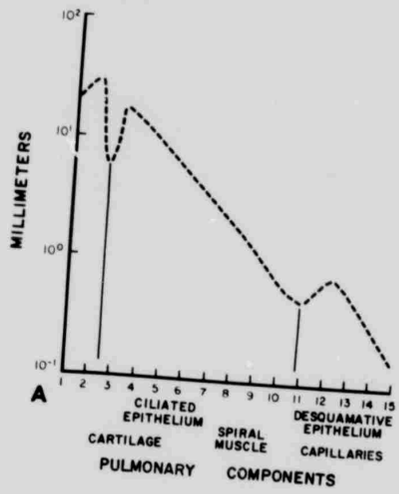
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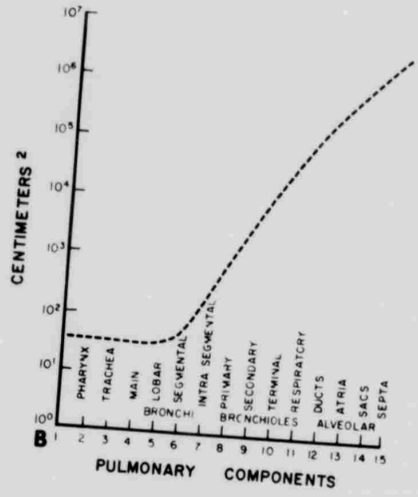
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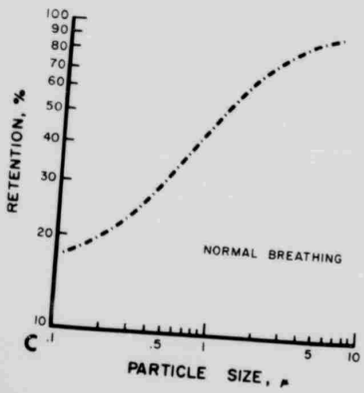
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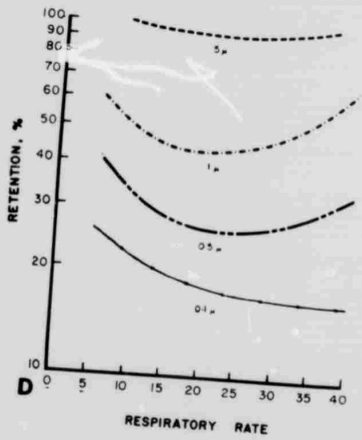
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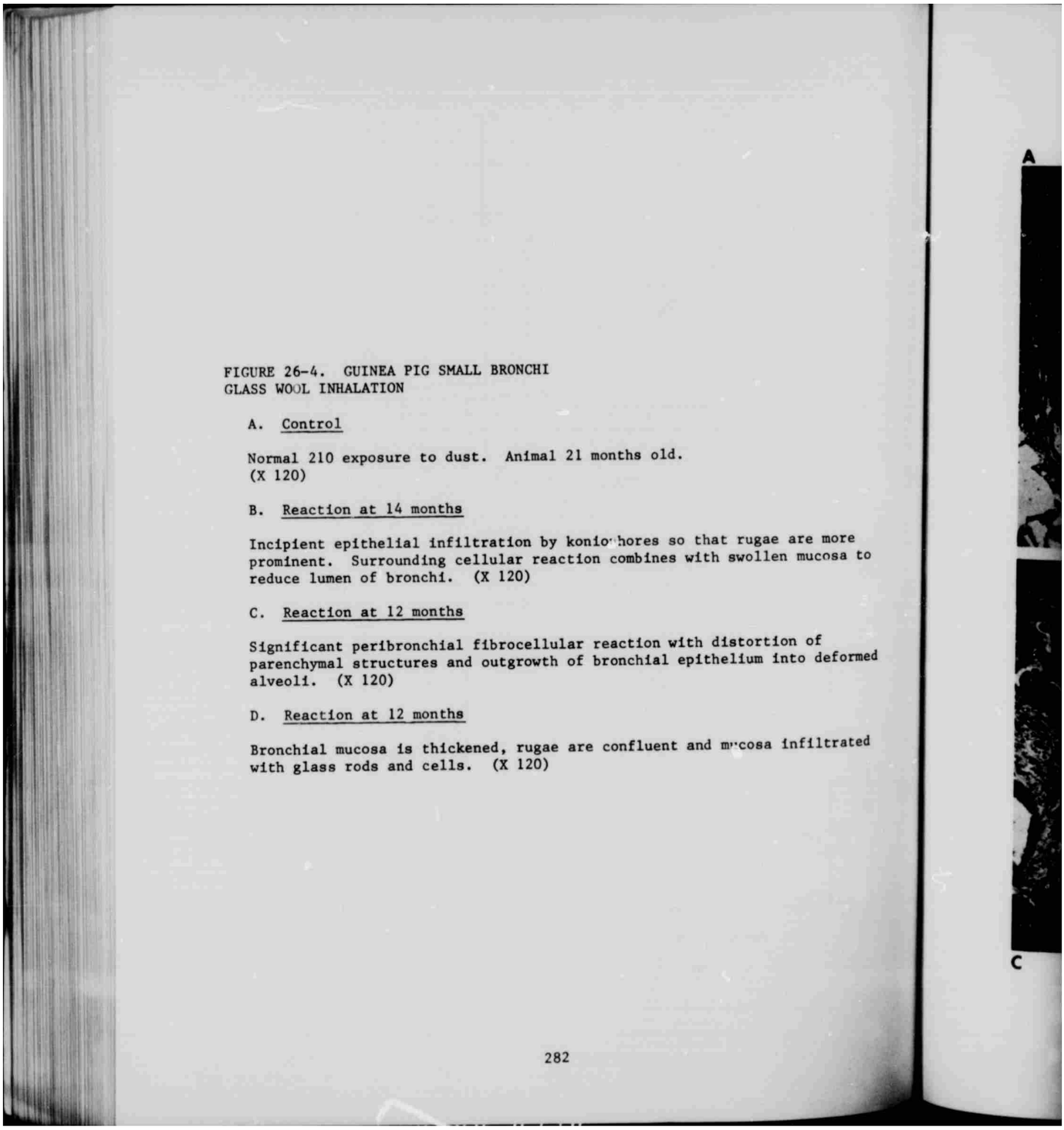


FIGURE 26-4. GUINEA PIG SMALL BRONCHI
GLASS WOOL INHALATION

A. Control

Normal 210 exposure to dust. Animal 21 months old.
(X 120)

B. Reaction at 14 months

Incipient epithelial infiltration by koniophores so that rugae are more prominent. Surrounding cellular reaction combines with swollen mucosa to reduce lumen of bronchi. (X 120)

C. Reaction at 12 months

Significant peribronchial fibrocellular reaction with distortion of parenchymal structures and outgrowth of bronchial epithelium into deformed alveoli. (X 120)

D. Reaction at 12 months

Bronchial mucosa is thickened, rugae are confluent and mucosa infiltrated with glass rods and cells. (X 120)

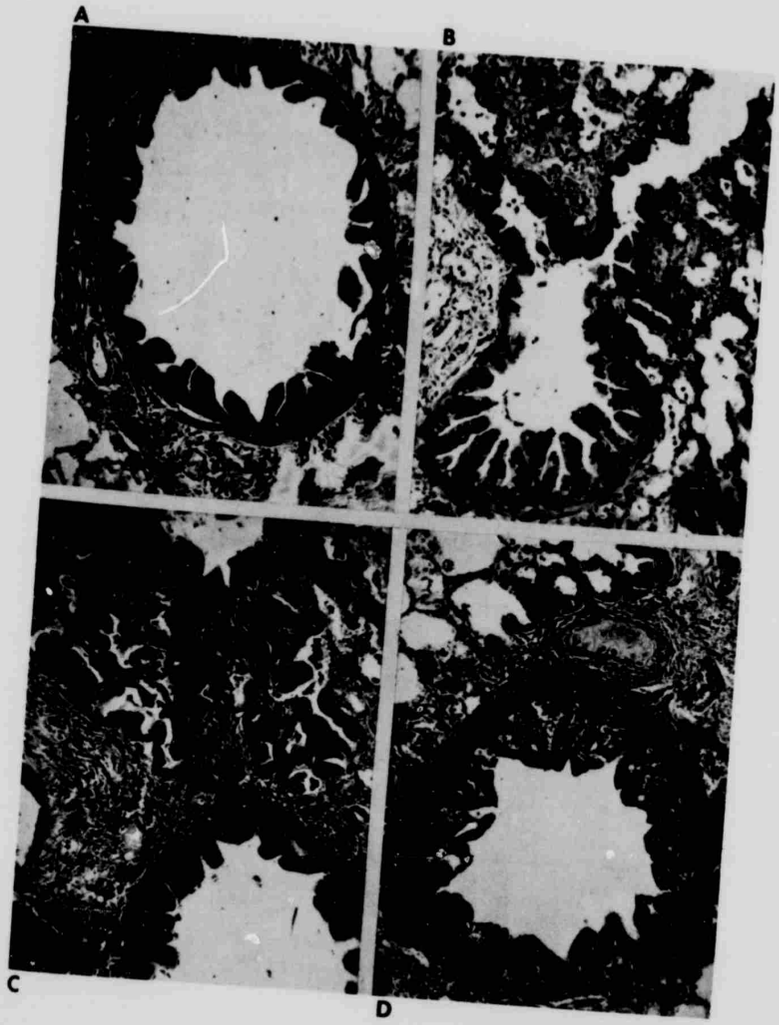
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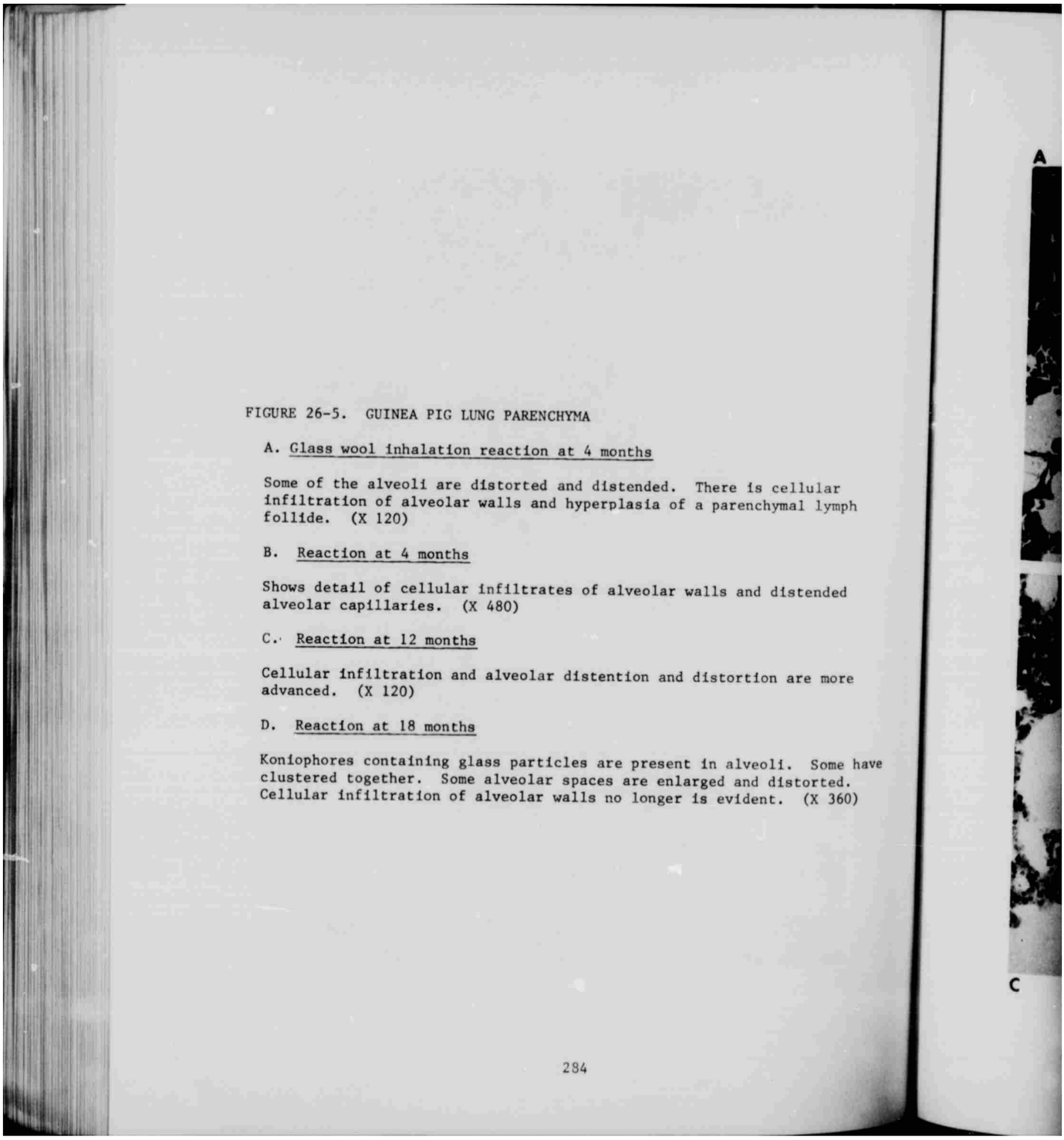


FIGURE 26-5. GUINEA PIG LUNG PARENCHYMA

A. Glass wool inhalation reaction at 4 months

Some of the alveoli are distorted and distended. There is cellular infiltration of alveolar walls and hyperplasia of a parenchymal lymph follide. (X 120)

B. Reaction at 4 months

Shows detail of cellular infiltrates of alveolar walls and distended alveolar capillaries. (X 480)

C. Reaction at 12 months

Cellular infiltration and alveolar distention and distortion are more advanced. (X 120)

D. Reaction at 18 months

Koniophores containing glass particles are present in alveoli. Some have clustered together. Some alveolar spaces are enlarged and distorted. Cellular infiltration of alveolar walls no longer is evident. (X 360)

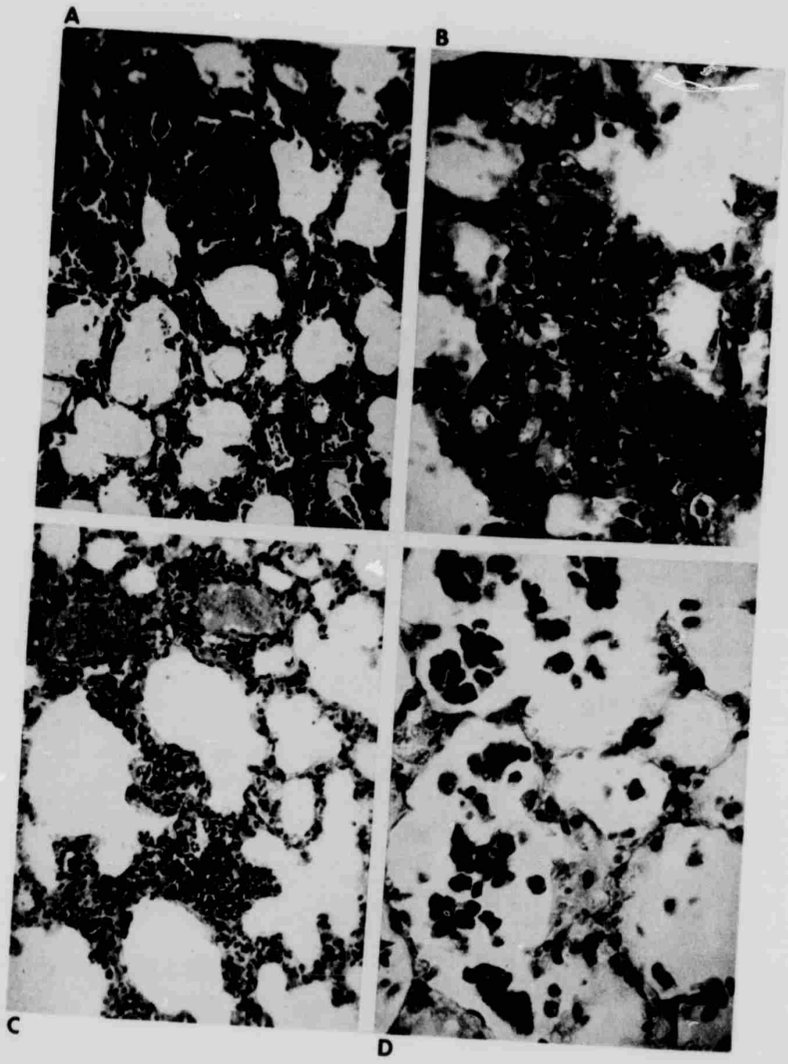
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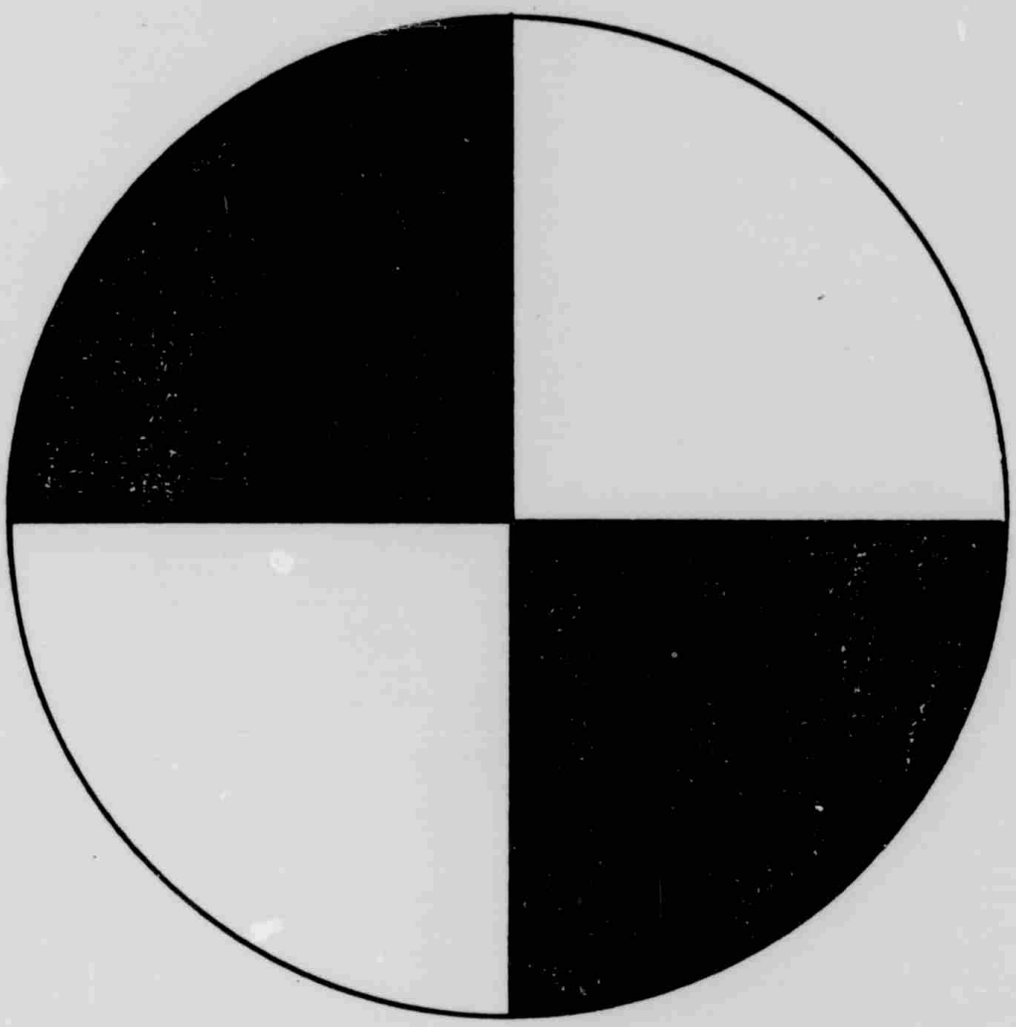
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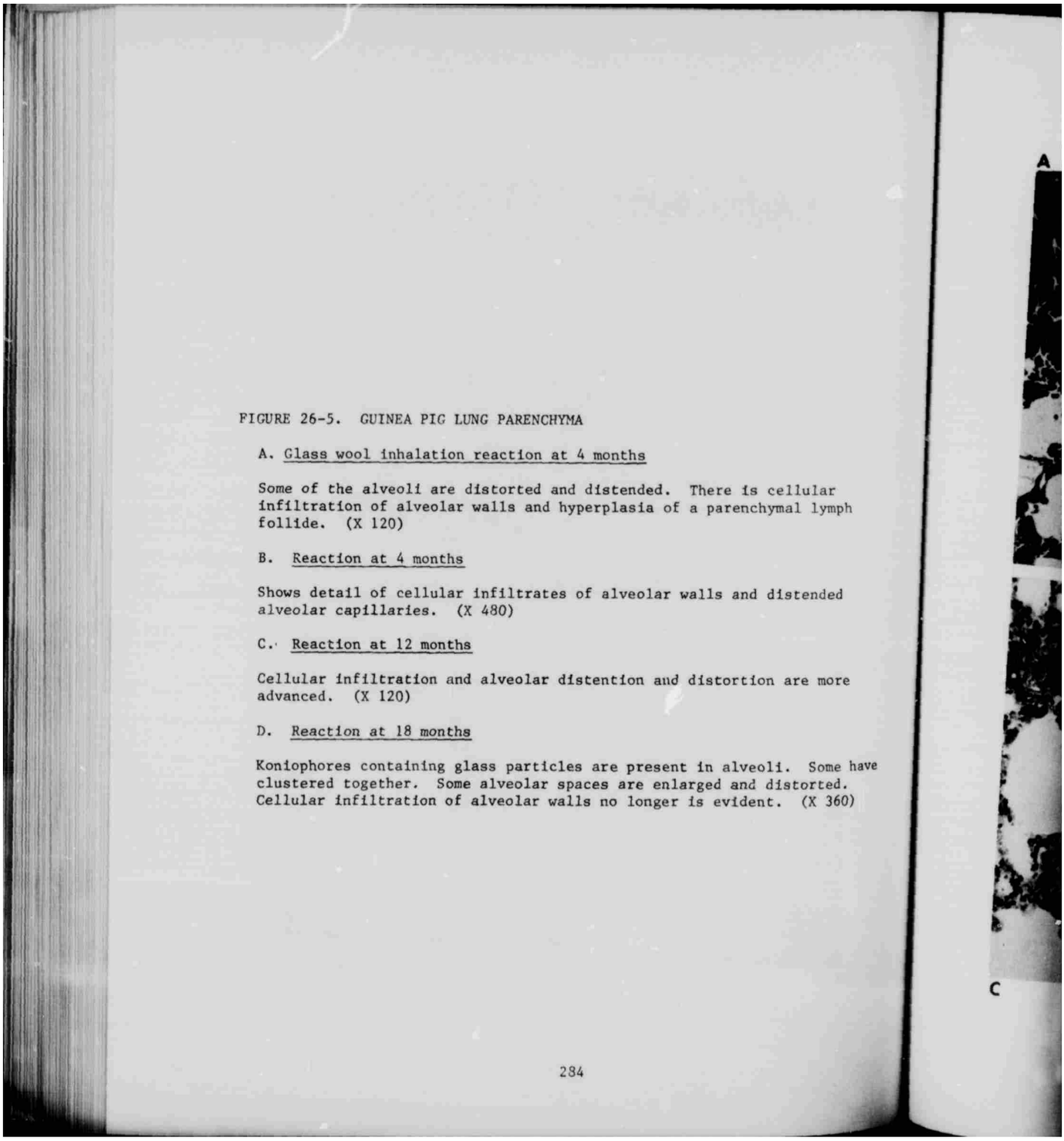


FIGURE 26-5. GUINEA PIG LUNG PARENCHYMA

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B. Reaction at 4 months

Shows detail of cellular infiltrates of alveolar walls and distended alveolar capillaries. (X 480)

C. Reaction at 12 months

Cellular infiltration and alveolar distention and distortion are more advanced. (X 120)

D. Reaction at 18 months

Koniophores containing glass particles are present in alveoli. Some have clustered together. Some alveolar spaces are enlarged and distorted. Cellular infiltration of alveolar walls no longer is evident. (X 360)

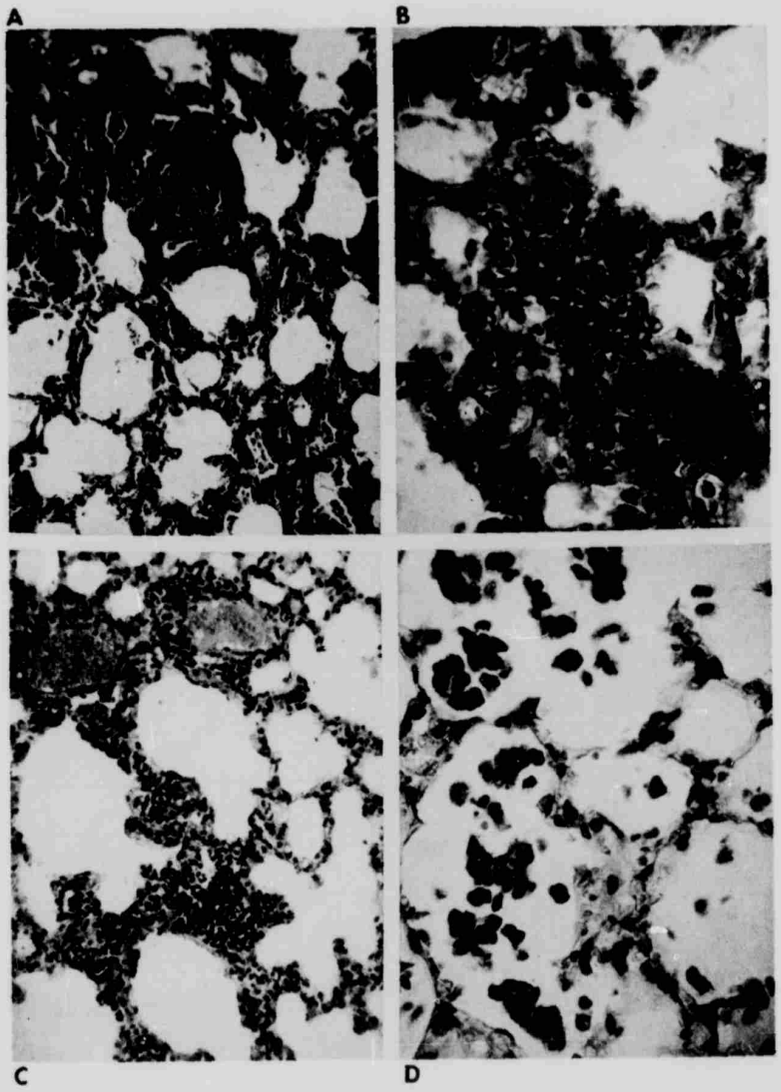
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FIGURE 26-6. GUINEA PIG LUNG. THESAUROTIC REACTION TO INHALATION OF FIBER GLASS PLASTIC DUST WITH CALCIUM CARBONATE FILLER. (X 260)

A. Dust Exposure 2 months

Discrete koniophores are abundantly present in alveolar spaces.

B. Dust Exposure 4 months

Some of the koniophores have begun to coalesce. Some of the alveolar spaces are over distended and some are smaller than normal.

C. Dust Exposure 12 months

Developing macule of lung parenchyma (paravascular). Confluent koniophores fill a group of alveolar spaces, leaving surrounding alveoli unaffected.

D. Dust Exposure 21 months

Parabronchiolar parenchymal macule. There is some breakdown of alveolar walls and minimal epithelization of marginal alveoli by bronchiolar columnar cells.

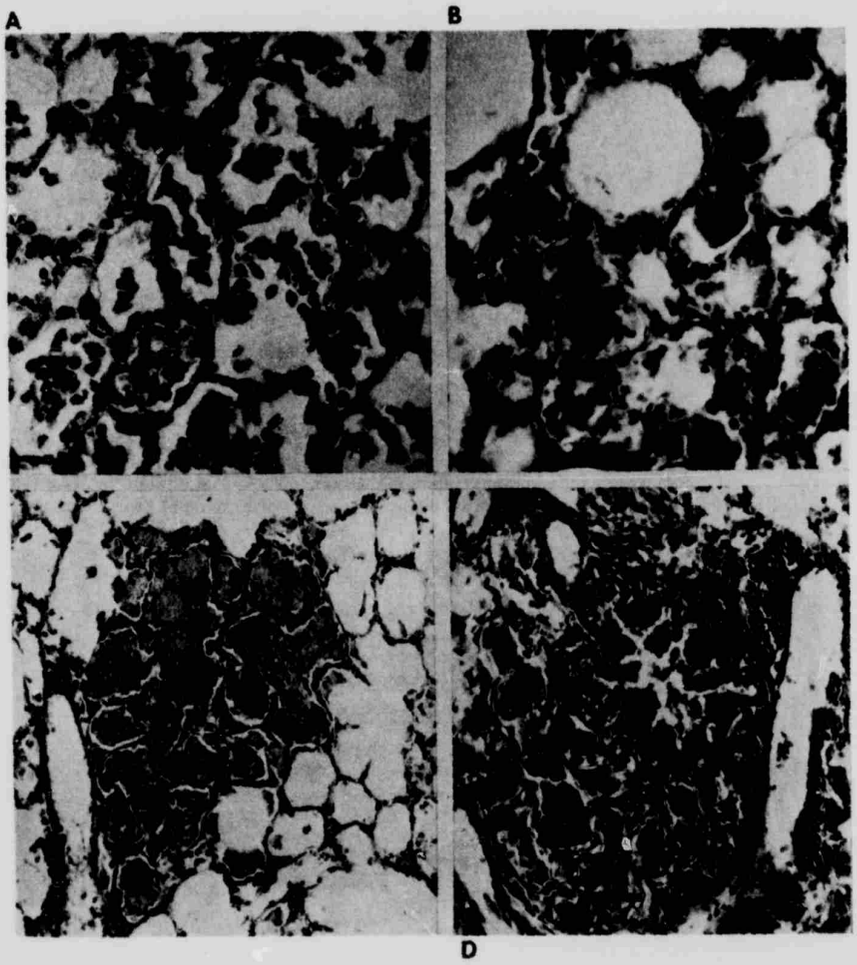
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FIGURE 26-7. GUINEA PIG LUNG. THESAUROTIC REACTION TO INHALED FIBER GLASS PLASTIC DUST WITH CALCIUM CARBONATE FILLER.

A. Dust Exposure 25 months

Demonstrates characteristic grouping of perabronchial thesaurotic macules, and minimal destructive emphysema. (X 35)

B. Dust Exposure 25 months

Detail of thesaurotic lesion showing pseudosyncytial confluent macrophages resulting in multinucleated particle filled protoplasmic masses. The alveolar walls show minimal infiltration by koniophores. Nuclear detail is well preserved. (X 450)

C. Dust Exposure 25 months

Silver stain of a thesaurotic macule demonstrating minimal reticulin reaction in alveolar mural and perivascular structures. (x 450)

D. Dust Exposure 24 months, then Normal Air for 3 months

The protoplasmic masses have again broken up into their component koniophores and are being evacuated via alveolar ducts (below). Some perivascular infiltration still persists, but mural infiltration has almost disappeared. (X 360)

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INHALED FIBER GLASS

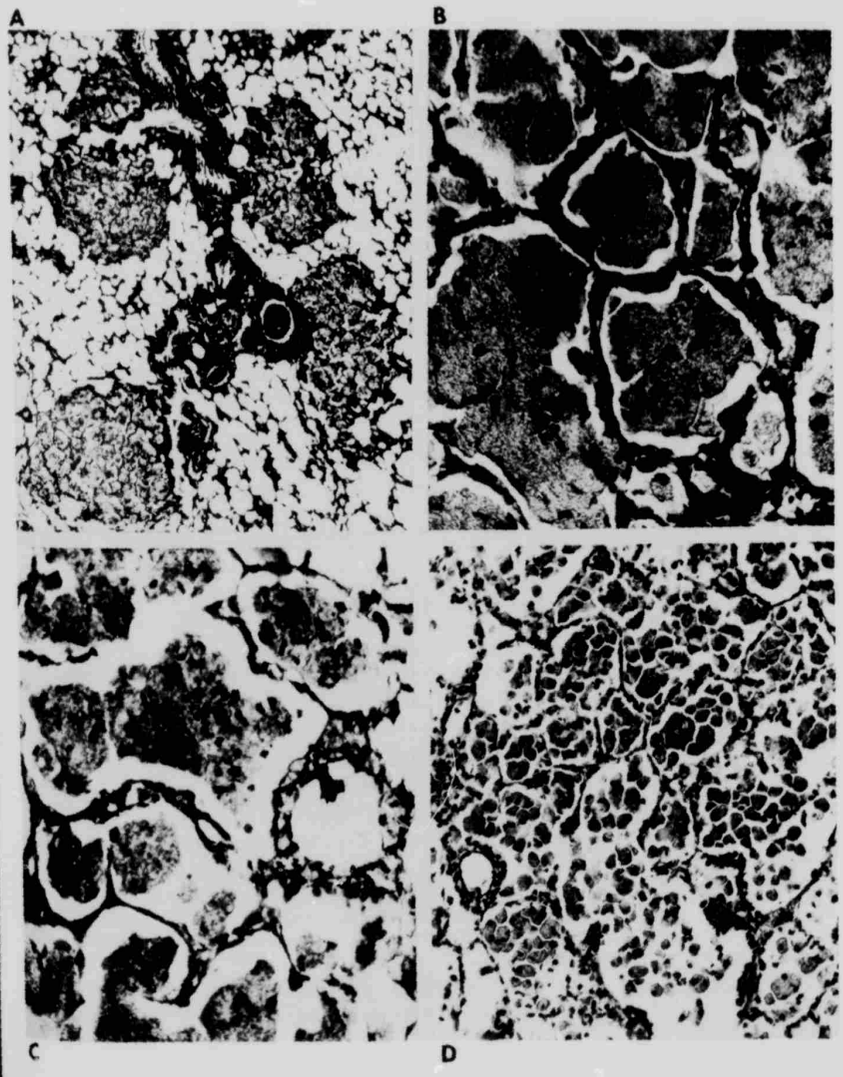
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alveoli are filled with syncytial masses of particle filled macrophages, somewhat resembling a bunch of cherries. Details of lesion are shown in Figures 26-7b and c. In the histologic preparation, the syncytial mass has undergone some differential shrinkage so that cleavage spaces are seen between the alveolar walls and the syncytiae. Also the multiple nuclei of the syneytiae are well preserved and seemingly healthy. There is only minimal to moderate infiltration of the alveolar walls. The silver stain shows only some loose reticulin. No collagen is perceptable. Under maximal optical microscopy, with phase contrast illumination, numerous glass fibers can be demonstrated.

These lesions were not permanent. After guinea pigs had been in normal air for 3 months following 25 months of exposure to the fiber glass plastic dust, the syncytial masses broke up, reverted to their original single koniophore condition and started being evacuated from the lung via the alveolar ducts and bronchioles (Figure 26-7d). At this stage, some minimal perivascular infiltration could also be observed. The alveolar walls survived almost intact in most areas.

Other lesions observed in the guinea pigs included some stenosis and distortion of alveolar ducts and bronchioles. This occured mainly after 18 months of exposure and was seen only in a group of animals with selectively higher dust exposure (485 million particles per cubic foot of air). Bronchiolar epithelial desquamation was noted at the beginning of the dust exposure only, but not later.

The lymphoid tissue reacted first by hyperplasia which progressively receded as exposure continued. Macrophage infiltration could be seen within 4 months and developed its maximal degree by the end of 10 months of exposure. This infiltration began as subcapsular foci which gradually expanded and eventually became confluent so that an almost complete subcapsular zone forms. Later foci of macrophages also developed in scattered loci throughout the gland substance gradually displacing the lymphoid tissue. A little pigment also appeared in the lymph node towards the end of the exposure period. This pigment may have been of endogenous orgin.

All these lymph node lesions receded progressively and relatively rapidly after the fiber glass plastic dust exposure was terminated. Since no permanent tissue damage resulted and the lesions proved transitory in the guinea pig, this biological reaction must be classified as a thesaurosis. The intrapulmonary lesions may be designated thesaurotic macules, in order to distinguish them from thesaurismotic lesions or pneumonoconiotic nodules or interstitial fibrosis.

Rabbits

The lungs of these animals reacted much less to the inhaled fiber glass plastic dust than did those of the guinea pigs. A specific lesion could not be defined.

Considerable pigmentation accumulated beneath the pleura between the seventh and tenth months. This then receded, despite continued exposure.

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It is surmised that the explanation may reside in transference of trapped dust to the lymph nodes via the pleural capillaries, for significant node infiltration by koniophores occurred principally after the 12th month.

Sparse discrete koniophores, more or less customarily applied to the alveolar surfaces were present in slight numbers throughout the exposure period (Figure 26-8a). These koniophores may have represented currently trapped dust which was being disposed of as fast as it was inhaled, so that there was no progressive accumulation of koniophores in the lung spaces or alveolar walls. Alveolar mural hyperemia also occurred during the first year of exposure. Mural infiltration by koniophores was most pronounced at the end of 4 months of exposure and decreased sharply thereafter.

Irregular distension of alveoli and alveolar ducts occurred progressively but not grossly. Towards the end of dust exposure there was decisive ductectasia and alveolar emphysema (Figure 26-8b). Perivascular and peribronchiolar changes did not occur and there was no damage to or infiltration of the bronchiolar, bronchial or tracheal epithelium, mucosa or submucosa (Figures 26-8c and d).

Rats

Once more, the lung reaction was dissimilar from that observed in guinea pigs and rabbits. Like the latter, there was significant subpleural pigmentation, but unlike the rabbit this subpleural pigmentation increased progressively and had reached a marked degree by the end of 15 months of exposure.

In the lung tissue the earliest lesions consisted of regional hyperemia, intra-alveolar exudation with minimal macrophage catarrh and moderate mural infiltration (Figure 26-9a).

Along with the dust filled macrophages, the rat lung characteristically displayed intra-alveolar lipophages. These increased in number and eventually formed lipophage islets. This cellular infiltration occurred to a moderate degree and, towards the end, focal lesions - some of which had the characteristics of granulomata - were discernable (Figure 26-9b). At this stage, the alveolar spaces were irregularly but sparsely the seat of emphysematous distention and distortion.

Progressive infiltration of lymph nodes occurred to a moderate degree without accompanying hyperplasia. Unfortunately the rat exposure component of the experiment did not include an elimination phase study so it is not yet known whether the lesions in the rat lungs represent transient or permanent change. However, the significant participation of tissue cells in the granuloma stage merits classification of the rat lesion as a thesaurismosis rather than a mere thesaurosis.

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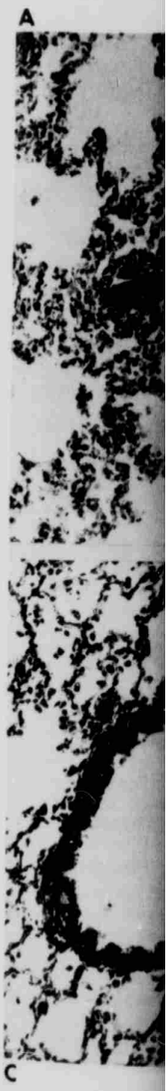


FIGURE 26-8. RABBIT LUNG. REACTION TO CONTINUOUS INHALATION EXPOSURE TO FIBER GLASS PLASTIC DUST WITH CALCIUM CARBONATE FILLER.

A. Dust Exposure 12 months

Koniophores are sparsely present in some alveolar spaces. Minimal perivascular lymphocytic infiltration. (X 100)

B. Dust Exposure 24 months

The predominant effect at this stage is moderate emphysema due partly to distended alveolar ducts and partly to confluence of alveoli through rupture of intervening walls. (X 60)

C. Dust Exposure 12 months

Cross section of small bronchus and surrounding pulmonary parenchyma. Isolated koniophores are demonstrable and occasional alveoli are confluent because of destruction of alveolar walls. Bronchial structures appear normal. (X 60)

D. Dust Exposure 24 months

Detail of mucosa of trachea. The only abnormality is sparse inflammatory cell infiltration of the submucosa. (X 360)

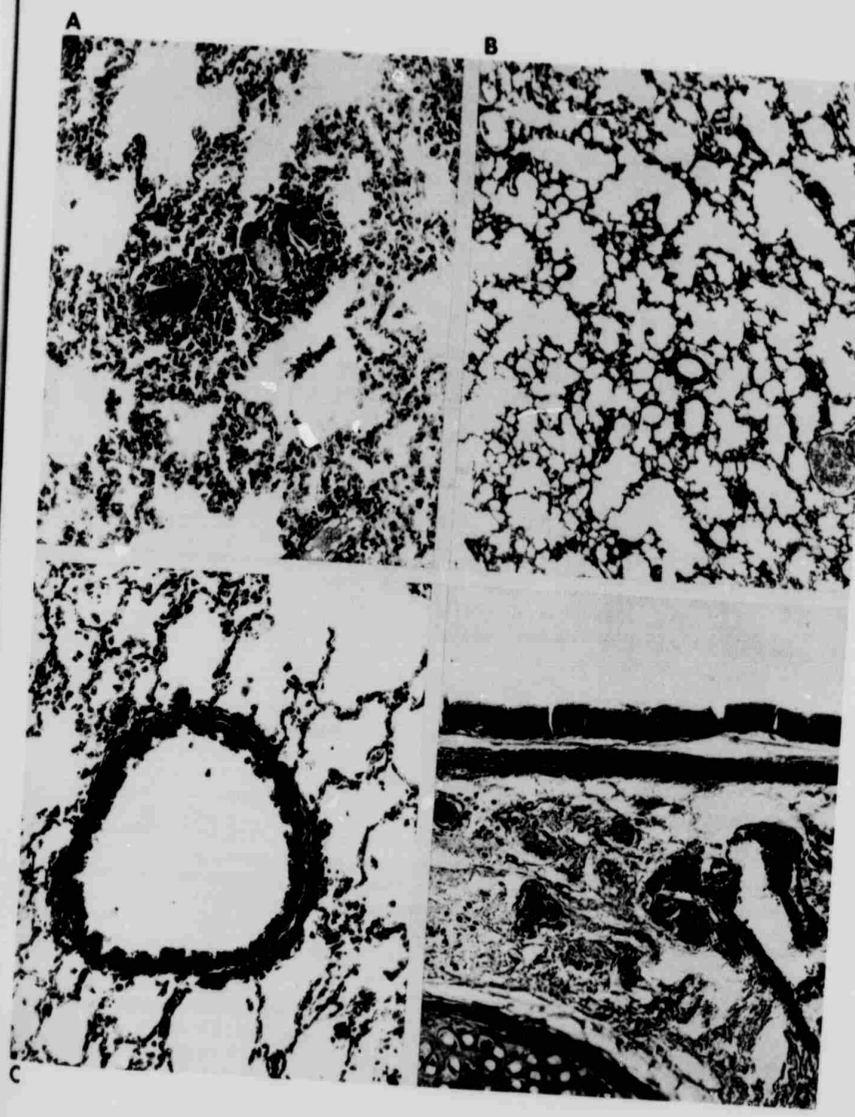
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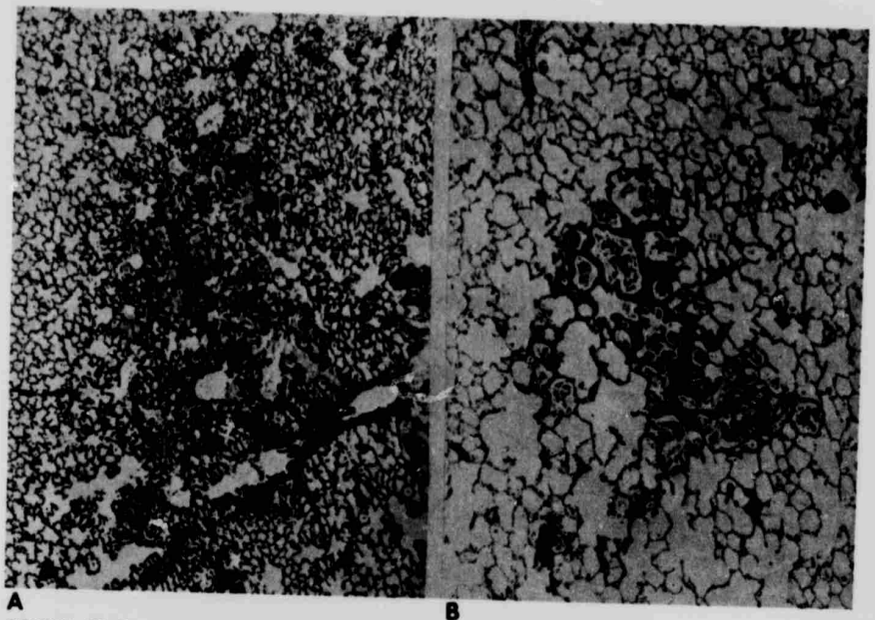


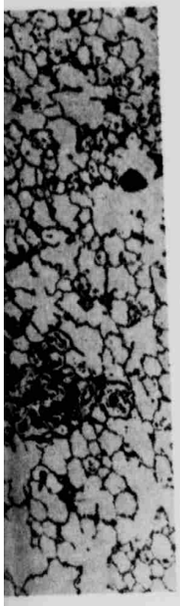
FIGURE 26-9. RAT LUNG. REACTION TO CONTINUOUS INHALATION EXPOSURE TO FIBER GLASS PLASTIC DUST WITH CALCIUM CARBONATE FILLER.

A. Dust Exposure 3 months
The parenchymal reaction still is somewhat vaguely defined, consisting of focal alveolar exudation and minimal koniophore entrapment. A few of the alveolar ducts are distended. (X 35)

B. Dust Exposure 15 months

The fiber glass dust macule now has the characteristics of both thesaurosis and thesaurismosis. The latter is represented by the mural infiltration which is about as defined as the accumulation of koniophores in intervening alveoli. Some alveolar spaces have become confluent. (X 60)

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Monkey Lung

As previously mentioned, one of the monkeys died at the end of 6 months. The cause of death was not determined by detailed necropsy so that a suspicion remains that the inhalation of the fiber glass plastic dust may have been a factor.

Gross section of the lung tissue did not reveal significant pathology. The parenchymal blood vessels appeared somewhat more prominent. There were two small "mite" lesions in one lung apex. Lymph nodes appeared somewhat enlarged but displayed no focal dust accumulation of the kind seen just 2 months later (Figure 26-10). Under the microscope, more abnormalities could be revealed (Figure 26-11). These will be discussed in conjunction with the histologic appearances at the 8-month stage of inhalation exposure to the fiber glass plastic dust. Although the inhalation exposure of the monkeys was relatively brief (8 months), rather significant pulmonary reactions now occurred. The lesions were universally distributed throughout the lungs and affected almost all the animals to an equally severe degree.

The most dramatic finding was the dense perivascular infiltration of the lung (Figure 26-11a). As compared to the wide collar of koniophores filling lymphatic sinuses around the blood vessels (Figure 26-11b), the lumen of the vessel appeared relatively small. This may be actual encroachment stenosis or may represent a mere optical illusion. The koniophore infiltration radiated into adjacent alveolar walls. Both around the blood vessels and in the alveolar walls a delicate argyrophilic network of reticulin fibers was well displayed (Figure 26-11c). No collagen was seen. Maximal magnification optical microscopy with phase contract illumination demonstrated numerous glass fibers and amorphous pigmented material in the cells which form the perivascular sheaths and alveolar mural infiltrates (Figure 26-11).

The dense infiltrating sheaths were not confined to the blood vessels. Similar infiltrates surrounded small bronchi and bronchioles (Figure 26-12a). In some of these lesions the bronchial lumen was decisively stenotic. The invading koniophores also congregated in relation to "mite" lesions, augmenting these in size and altering their appearance (Figure 26-12).

The larger respiratory channels of the monkey lung were not greatly affected. The epithelium remained intact and there was only minimal infiltration of the mucosa by inflammatory cells (Figure 26-12c). Intermediate and smaller bronchi, by contrast, showed significant reaction to the inhaled fiber glass plastic dust (Figure 26-12d). Koniophores filled with glass fibers and particulate material infiltrated both the mucosa and submucosa. Cuboidal epithelium lining these ducts was lost in some sectors or reduced to a thin single layered squame stretched over the luminal surfaces of the irregularly bulging infiltrated mucosa. Some of the ducts were distorted and distended distally to these zones of mucosal and submucosal infiltration with resultant focal emphysema (Figure 26-12d).

Some subpleural infiltration also was evident in most areas (Figure 26-11a). The lymph nodes reacted only to a moderate degree, showing slight

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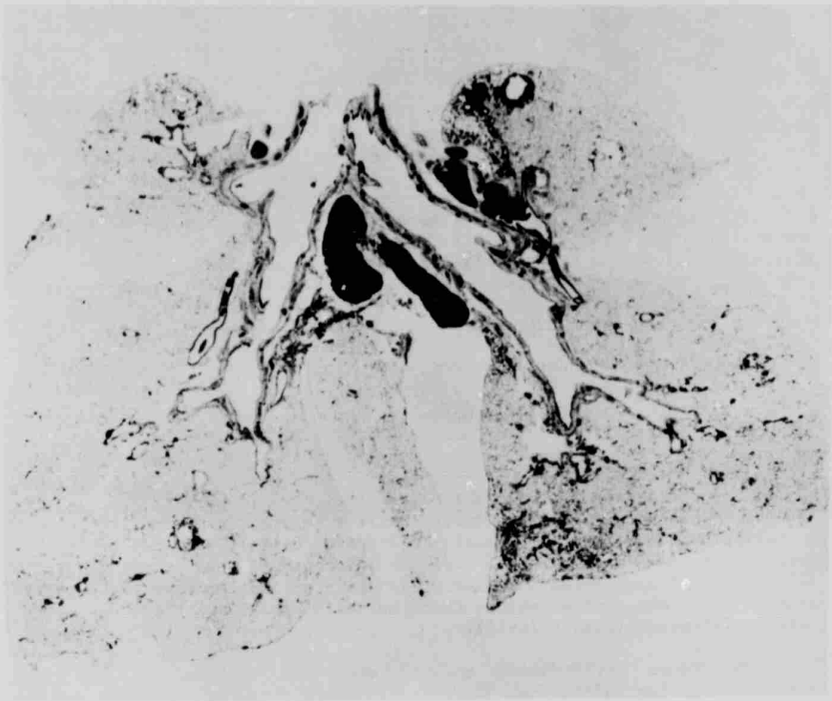


FIGURE 26-10. MONKEY LUNG. LACK OF GROSSLY PERCEPTIBLE REACTION TO INHALED FIBER GLASS PLASTIC DUST AT 6 MONTHS.

The lung parenchymal blood vessels are minimally increased in prominence. Two small mite lesions are in one lung apex. Lymph nodes are slightly enlarged. The pleura is minimally thickened at the base of one lung, opposite a restricted zone of apparent infiltration.

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hyperplasia of the medulla, progressive hyperplasia of the endotheloid cell system and cortical infiltration by dust filled macrophages. In its most extreme form, the latter was represented by a complete subcapsular zone of koniophores which occupied from one-third to half of the cross sectional area of the lymph nodes.

Blood Pressure Studies

A graphic portrayal is provided by Figure 26-13 of the results obtained when intraventricular pressures were measured in rabbits during the course of the experiment. A lack of response to right ventricular pressures to inhaled fiber glass plastic dust is shown in graph a. By contrast, four varieties of submicron amorphous silica dust elevated right ventricular pressures from 100% to 250% above normal levels.

Graph b shows a comparable lack of response of the left ventricular pressures to the inhaled fiber glass plastic dust. Again, by contrast, the inhalation of the four varieties of submicron amorphous dust elevated left ventricular pressures from 10% to almost 40%.

It should be emphasized that the above observations apply only for the rabbits. Nothing is known about the probable responses of rats, guinea pigs or monkeys. However, there was no evidence of right or left ventricular hypertrophy in the rats or guinea pigs. The right ventricle of the monkeys was moderately enlarged so that this is presumptive evidence that the perivascular infiltration, which reached such dramatic levels so swiftly in the monkeys, was probably associated with elevation of right ventricular pressure in the monkey. This of course needs experimental verification.

Tuberculogenicity Studies

This series of experiments was conducted in standard fashion, using the RiRv tubercle bacillus as the infecting agent and exploring the effects of concurrent dust exposure (simultaneous phase) and delayed dust exposure (reactivation phase).

The number of animals used in each phase of infection and dust exposure and the numbers used in the animals which received dust exposure only, are summarized in Table 26-12.

The control reaction is rated as Grade I with respect to severity and duration of the active phase of the infection. The disease produced in the guinea pig is a self limited mild focal infection which reaches its maximal stage between the second and eighth month after infection. There is no mortality associated with this infection. Typical maximal lesions in control animals are given in Figure 26-14. The lesion present was well circumscribed even when necrosis and caseation has supervened. The lesion healed into a localized fibrous scar with only limited distortion of surrounding parenchyma. Beyond the lung only the tracheobronchial lymph nodes were affected. There may be multiple tuberculous cortical

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TABLE 26-12

FIBROUS GLASS

TUBERCULOGENICITY

INHALATION EXPERIMENTS - GUINEA PIGS

Substance	Mycobacterium Tuberculosis RiRv Inhalation Infection Phase	Months	No.	Mortality Percent	Tuberculosis Grade	Tuberculosis Duration
Fiber glass + CaCO 3	None	25	65	0	0	0
	Simultaneous	24	25	4	II	III
	Reactivation	18	26	0	I	II
Fiber glass + CaSO 4	None	9	64	0	0	0
	Simultaneous	10	15	7	I	II
	Reactivation	10	27	0	I	I
Perlite Volcanic Glass	None	24	60	0	0	0
	Simultaneous	24	30	26	III	IV
	Reactivation	24	30	12	II	III
Quartz 1-5 μ	None	24	150	50	0	0
	Simultaneous	20	75	100	IV	IV
	Reactivation	18	70	100	IV	IV
Controls No dust	Infected	24	300	0	I	I
	None	24	215	5	0	0

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granulomata in these lymph nodes, but they all heal into relatively innocuous scars.

Mortality rates were slightly increased in the animals which were exposed to fiber glass plastic dust after infection (Table 26-12). This slight mortality cannot be ignored, but does contrast vividly with the moderately increased mortality seen after exposure of infected animals to Perlite (volcanic glass dust) and the 100% mortality after exposure to quartz dust.

The severity and duration of the tuberculous reaction was mildly to moderately increased in animals exposed to the fiber glass plastic dust with the calcium carbonate filler and only slightly prolonged after exposure to the fiber glass plastic with the calcium sulfate filler. By contrast, Perlite, which induced virtually no pathology in uninfected guinea pigs, (Table 26-12) provoked moderately to markedly increased tuberculosis in the infected animals. The infection was not always as severe as that seen after exposure to quartz dust, but almost reached such severity in some animals and lasted as long (Table 26-12). The histologic appearance of these lesions is shown in Figures 26-15 and 16. Attention was drawn to the greater severity of the lesions caused during the simultaneous phase experiment with fiber glass plastic dust. However, the augmented tuberculous lesion was more or less confined to the lungs with no perceptible involvement of the lymph nodes. More detail is provided in Figure 26-16 which shows that there was a more considerably expanded lesion, with poor zonular differentiation and central excavation within some tuberculous foci.

The thesaurotic lesion also underwent expansion as a result of the tuberculous infection. Typical examples are portrayed in Figure 26-17 which shows mural infiltration, interstitial cellular proliferation and confluence of lesions which contrast rather sharply with the thesaurotic macule developed when there was no tuberculous infection. The enhanced cellularity is a striking feature. There appeared to be less koniophore and syncytium formation. Foci of alveolar epithelization was clearly portrayed in Figure 26-17d.

Figures 26-18 and 26-19 summarize these experiments in a graphic manner. It is not fully understood why the fiber glass plastic with the calcium sulfate filler produced a significantly lesser reaction after infection with the RiRv tubercle bacilli. Possibly the lesser result is due to the slightly lower concentration of the aerosol in the CaSO 4 filler experiment. However, lesser aerosol concentration cannot be the sole factor, for in the perlite experiment the dust dose was even less, yet the tuberculous reaction was much worse. Perhaps the substitution of CaSO 4 for CaCO 3 makes a real difference. However, prior tuberculogenicity experiments with CaSO 4 and CaCO 3 separately showed neither to have the ability to stimulate tuberculosis in guinea pigs.

Finally, it may be mentioned that the virulence of the RiRv tubercle bacillus, low as it is, waxes and wanes through successive cultures. In the CaSO 4 fiber glass plastic experiment, the culture may have been in a low virulence phase.

lity ent	Tuberculosis Grade	Duration
	0	0
	II	III
	I	II
	0	0
	I	II
	I	I
	0	0
	III	IV
	II	III
	0	0
	IV	IV
	IV	IV
	I	I
	0	0

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FIGURE 26-11. MONKEY LUNG. REACTION TO INHALED FIBER GLASS PLASTIC DUST ($CaSO_3$ FILLER)

A. Lung Parenchyma (6 months)

Demonstrates almost universal involvement of pulmonary blood vessels by parietal koniophore infiltrates. Foci of distended alveoli and alveolar ducts are interposed between normal and semicollapsed alveolar spaces. Distinct subpleural koniophore infiltration. (X 35)

B. Lung Blood Vessels (6 months)

Demonstrates dense infiltration of perivascular lymphatics by koniophores. Distortion of surrounding alveolar spaces is evident. Infiltration of adjacent alveolar walls is clearly shown. (X 240)

C. Lung Parenchyma (8 months)

Bielschowsky silver stain shows minimal argyrophilic reticulin deposition in perivascular, peribronchial structures and alveolar walls. (X 60)

D. Detail of Macrophages (8 months)

Positive phase contrast microphotograph of perivascular koniophores showing numerous glass fibers, some lengthwise in the plane of view and others cross sectioned. Other particulate material in the koniophores may represent polyester resin and filler. (X 2000)

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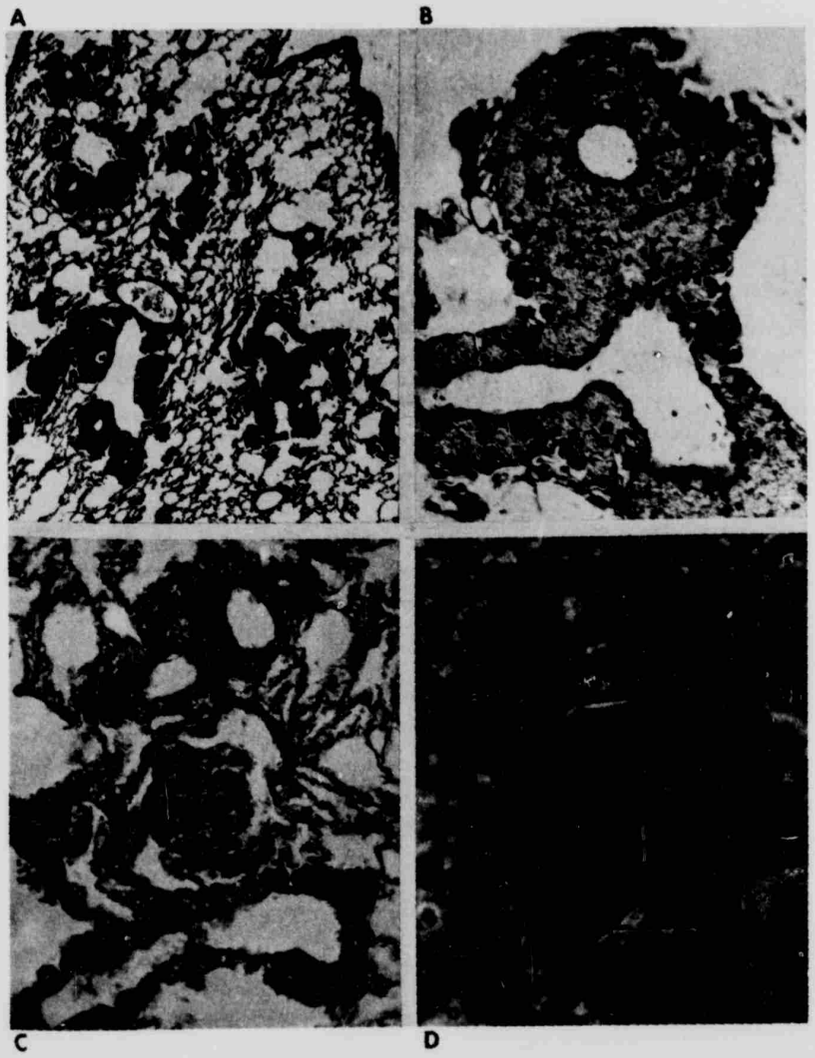
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FIGURE 26-12. MONKEY LUNG REACTION TO INHALED FIBER GLASS PLASTIC DUST WITH CALCIUM CARBONATE FILLER (8 MONTHS)

A. Bronchus

Accumulation of dust filled koniophores in peribronchial lymphatic spaces. The bronchial lumen is somewhat distorted and stenosed. (X 360)

B. Mite Lesion

"Mite" lesion augmented and altered by infiltration with dust filled cells grouped partly around blood vessels and partly in relation to bronchial structures. (X 35)

C. Trachea

The epithelium has remained normal. The mucosa has been minimally to moderately infiltrated by inflammatory cells. The mucosal glands and the muscularis mucosae appear normal. (X 360)

D. Bronchiole

Demonstrates massive infiltration of both mucosa and submucosa by dust filled phagocytes. (X 1000)

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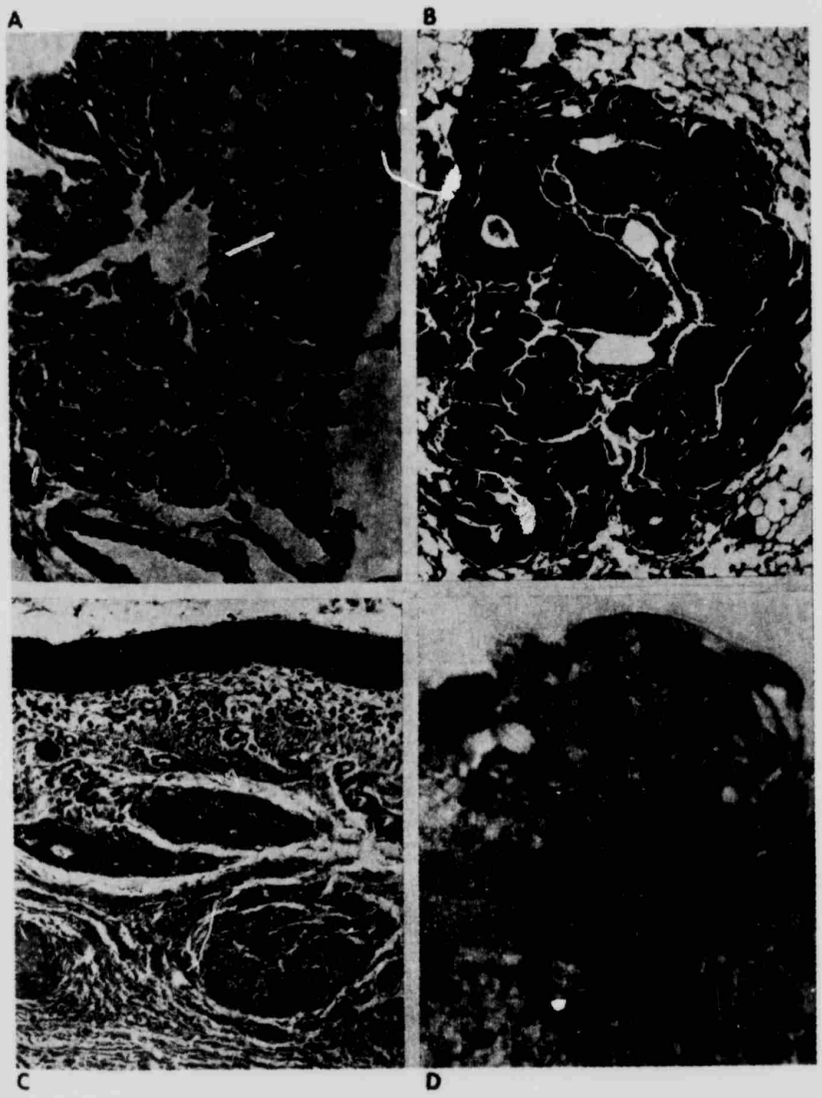
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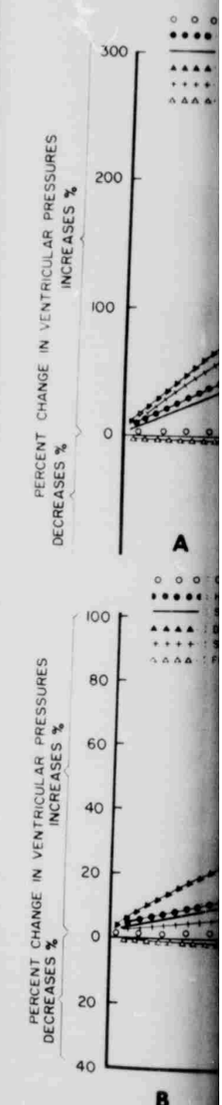


FIGURE 26-13. LACK OF HYPERTENSIOGENIC ACTION OF INHALED FIBER GLASS AS COMPARED WITH EFFECTS OF OTHER INHALED SILICEOUS COMPOUNDS ON RABBITS.

A. Right Ventricular Pressures

During 18 months of inhalation of fiber glass, the right ventricular pressure deviated very little from that of control rabbits not exposed to any dust. The right ventricular pressure increased progressively shortly after exposure to four varieties of submicron amorphous silica was commenced and remained elevated as long as exposure continued.

B. Left Ventricular Pressures

Demonstrates once more that inhaled fiber glass did not affect the left ventricular pressure during 18 months of inhalation whereas these pressures rose from 15% to 35% during exposure to four varieties ofsubmicron amorphous silica and in the case of hydrophobic silica continued to rise even after exposure was discontinued.

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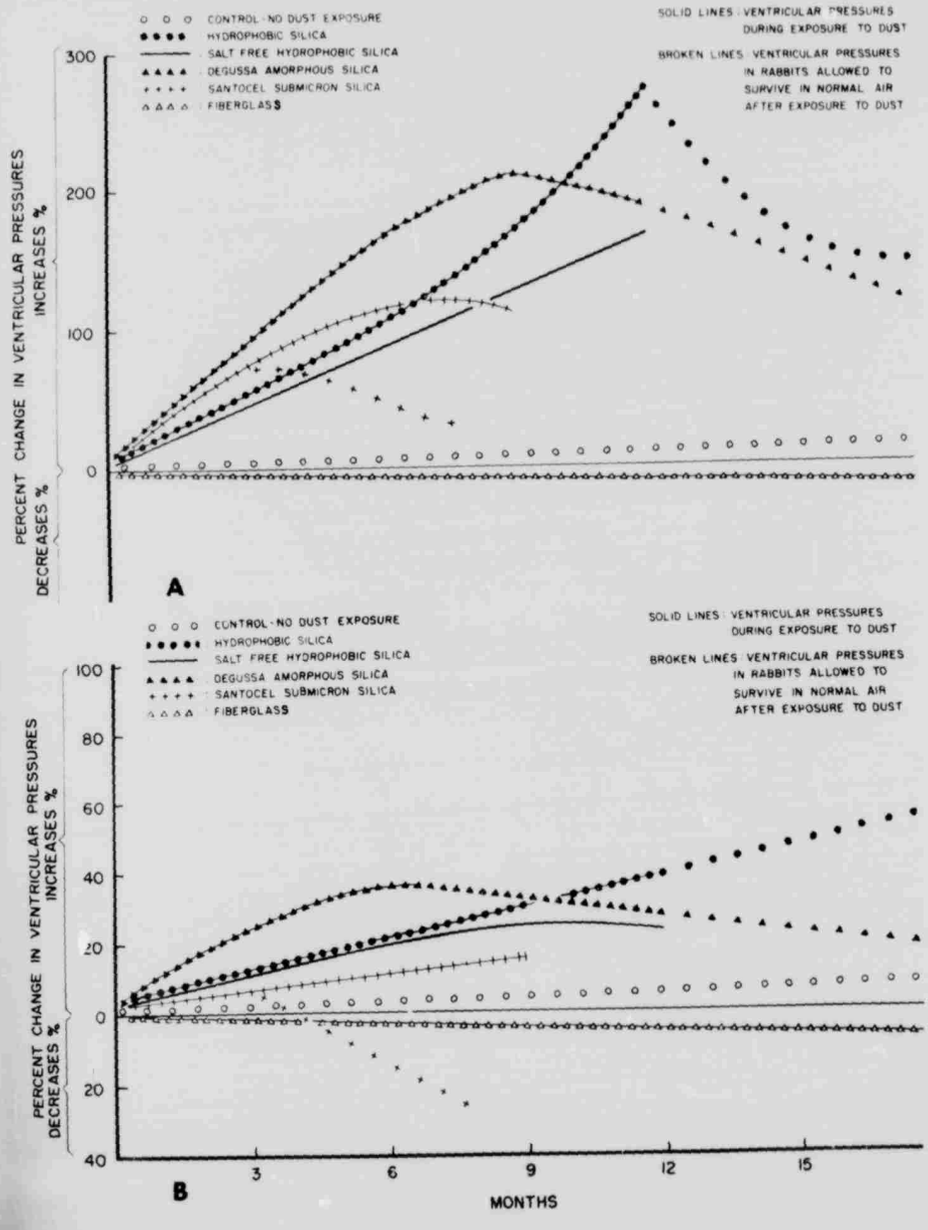

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FIGURE 26-14. GUINEA PIG LUNG. RESPONSE TO INHALATION INFECTION BY RiRv TUBERCLE BACILLI

A. Infection 4 months

Typical RiRv induced subpleural tubercle showing central necrosis and semicaseation and early capsule formation. (X 35)

B. Infection 15 months

Residual subpleural scar of a healed tubercle. Note dimple in pleural surface. (X 35)

C. Lung Tubercle at 4 months

There is abundant central necrosis and incipient collagenation. (X 35)

D. Tracheobronchial lymph node at 2 months
Fibrocellular tubercles have formed at multiple foci mainly in the cortex of the node. Tubercles undergo resolution or scarring beyond this stage and in subsequent months are scarcly detectable. (X 10)

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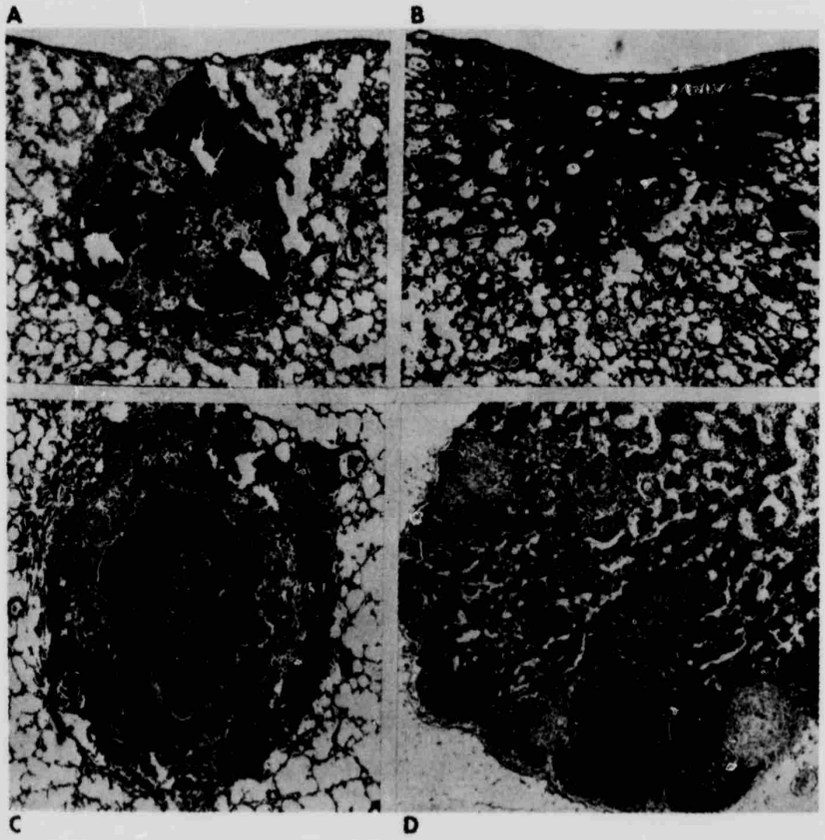
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FIGURE 26-15. GUINEA PIG LUNG. TUBERCULOGENIC PROPENSITY OF FIBER GLASS PLASTIC DUST WITH CALCIUM CARBONATE FILLER

Midcoronal plane section (1 X) of guinea pig lungs following inhalation infection with an aerosol of RiRv tubercle bacilli followed by continuous inhalation exposure to the fiber glass plastic dust.

A. Dust Exposure 4 months

Simultaneous Phase, Note confluent areas of mixed dust reaction and tuberculosis. Minimal lymph node reaction. No caseation or cavitation.

B. Dust Exposure 12 months

Reactivation after 2 months. There are areas of confluent reaction but lesions appear more discrete. Again minimal lymph node reaction, and no caseation or cavitation.

C. Dust Exposure 18 months

Simultaneous Phase. Several of the tuberculous lesions show central caseation and fibrous capsule formation. Many areas in the lung display only the characteristic guinea pig thesaurotic response to the dust.

D. Dust Exposure 12 months

Reactivation after 6 months. Only two areas of tuberculous involvement are discernable. Lymph node reaction is still minimal. Both basal lobes show pronounced thesaurosis and some thesaurismosis.



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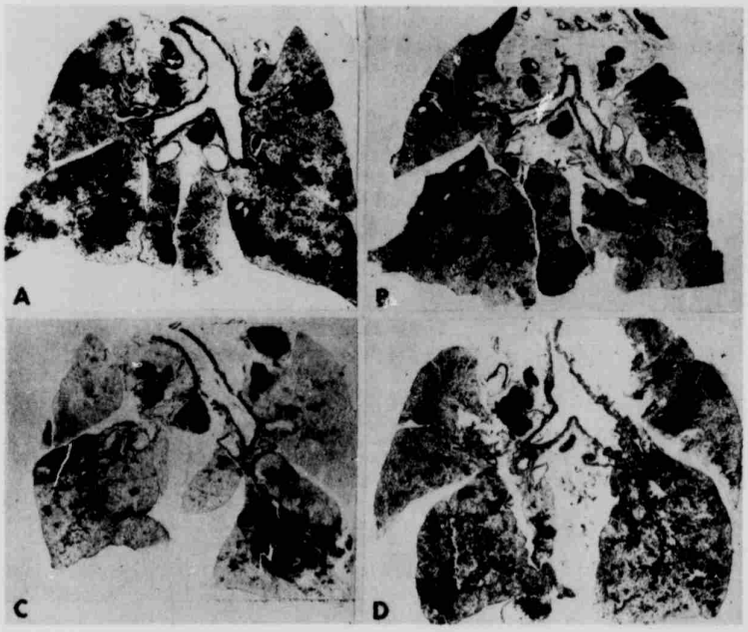
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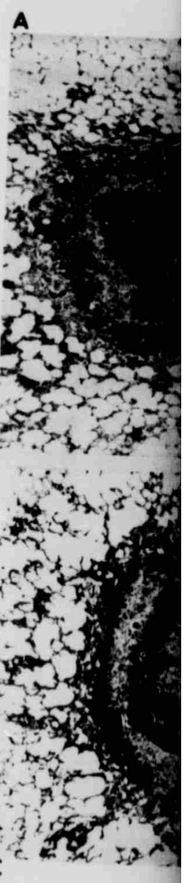


FIGURE 26-16. GUINEA PIG LUNG. TUBERCULOGENICITY OF FIBER GLASS PLASTIC DUST WITH A CALCIUM CARBONATE FILLER. EXPOSURE COMMENCED IMMEDIATELY AFTER INHALATION INFECTION WITH R1Rv TUBERCLE BACILLI

SIMULTANEOUS PHASE

A. Control Infection at 2 months. No Dust.

At this stage the tubercle is fully formed and displays a central necrotic zone, a surrounding palisade of epitheloid cells and a peripheral lymphocytic zone which shades off into the lung parenchyma. (X 35)

B. Tuberculous Lesion Augmented by 8 Months of Dust Exposure Following Infection.

The tubercle is several times larger than that of the control study, more irregular and shows caseation and incipient excavation. (X 35)

C. Infection then Dust for 4 months

The center of the efflorescent tubercle has undergone necrosis and shows a line of separation from the subcapsular cellular palisade zone. A small thesaurotic lesion is adjacent to the tubercle. (X 60)

D. Infection plus Dust for 15 months

Most of the necrotic center of this large lesion has been evacuated leaving a cavity which is unlikely to collapse in view of the broad capsular zone. (X 35)

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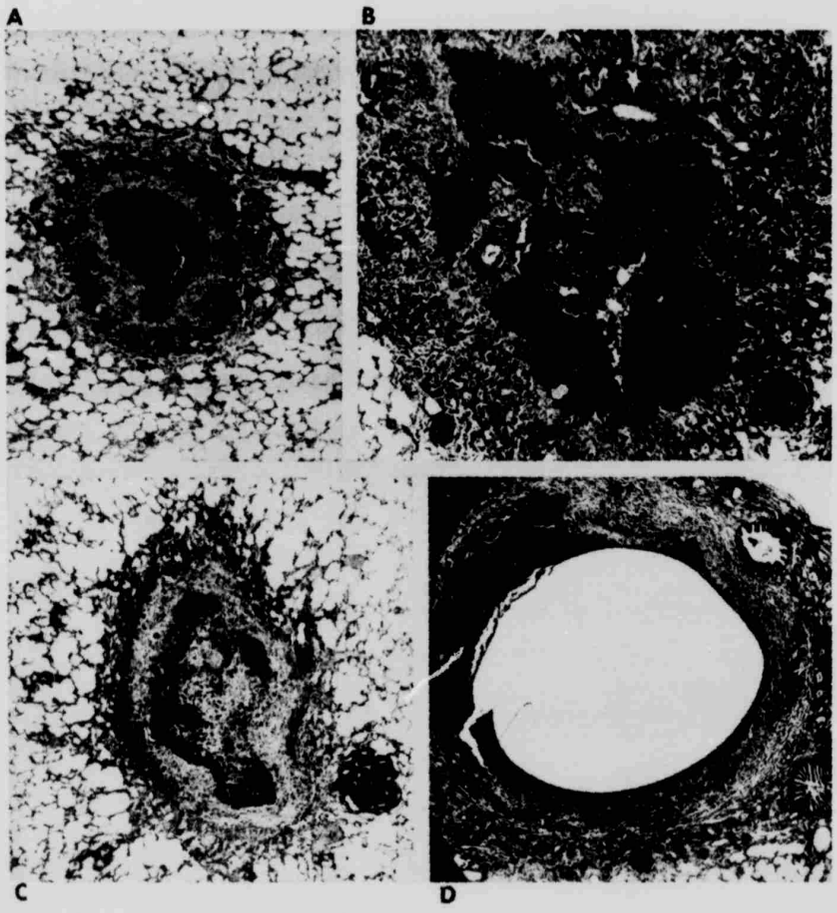
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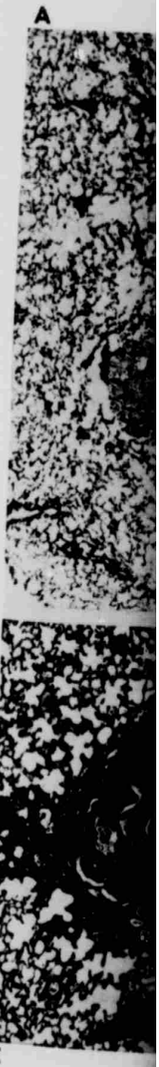


FIGURE 26-17. GUINEA PIG LUNG. THESAUROTIC REACTION TO INHALED FIBER GLASS PLASTIC DUST, WITH CALCIUM CARBONATE FILLER FOLLOWING INHALATION INFECTION WITH R Rv TUBERCLE BACILLI

SIMULTANEOUS PHASE

A. Dust Exposure 16 months No Tuberculous Infection

Typical parabronchial thesaurotic macule. Note precisely circumscribed lesion. (X 8)

B. Dust Exposure for 12 months. Infection with Tubercle Bacilli Preceded Dust Phase by 4 months

Demonstrates enlarged dust macules with loss of demarcation, loss of alveolar walls in the lesion, interstitial cellular reaction in alveoli between macules with a tendency to confluence. A small necrotic tuberculous focus is present. (X 35)

C. CONTROL: No Dust Exposure

The tubercle has partly healed through resorption of caseous material and interstitial cellular infiltration and fibrosis.

D. Dust Exposure Immediately After Infection

The degree of healing of the tubercle is about the same as that of the control. However, the residual tubercle is volumetrically about 16 times larger.

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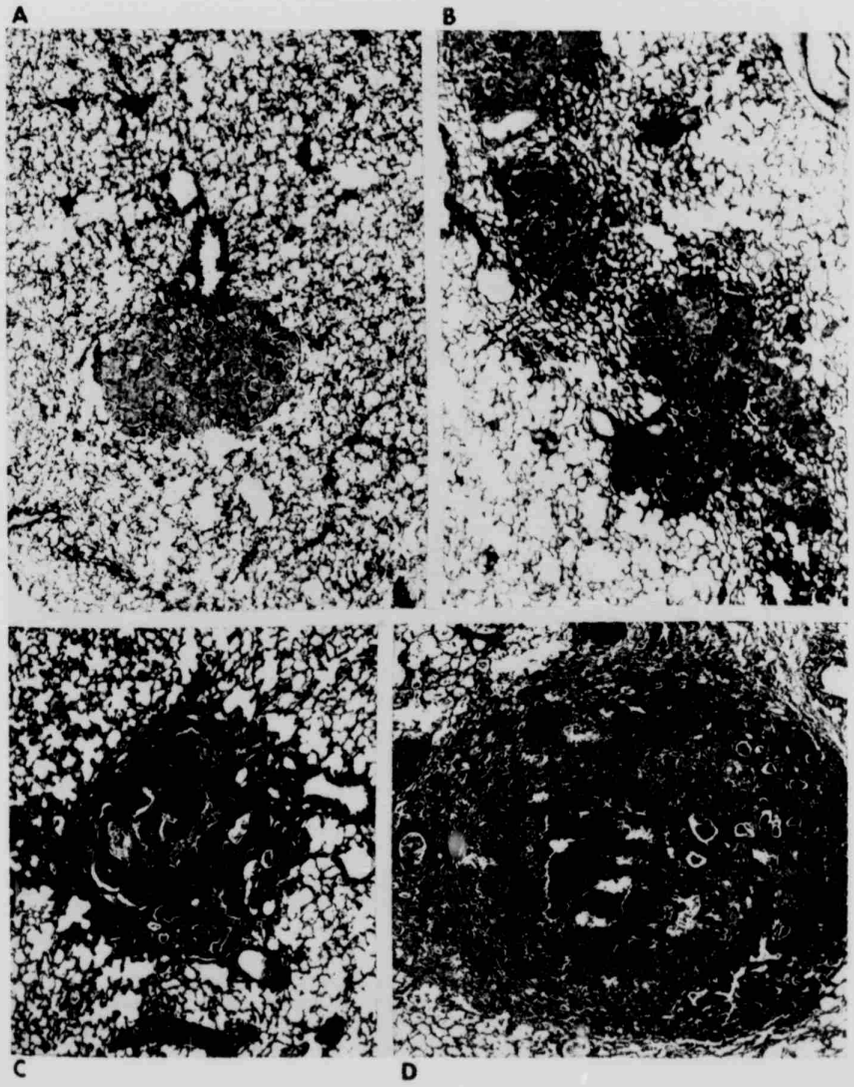
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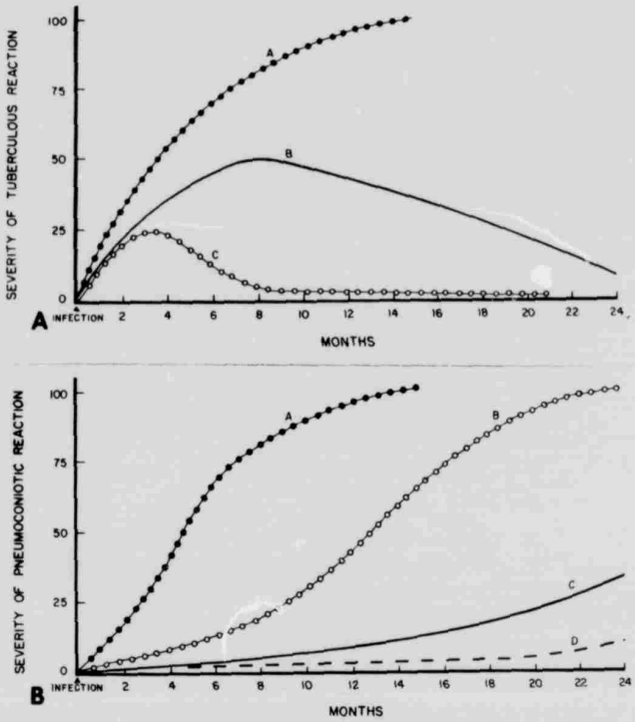


FIGURE 26-18. GUINEA PIG LUNGS. COMPARATIVE TUBERCULOGENICITY OF FIBER GLASS PLASTIC DUST WITH CALCIUM CARBONATE FILLER

SIMULTANEOUS PHASE

A. Tuberculous Reaction

The graphs demonstrate the differential responses of guinea pigs infected with RiRv tubercle bacilli and then either exposed to an aerosol of quartz dust (graph A) or an aerosol of fiber glass plastic dust (graph B) or not exposed to any dust (graph C).

B. Pneumoconiotic Reaction

Graph A shows the accelerated rapid progressive development of pneumoconiotic lesions in animals exposed to quartz dust following infection with RiRv tubercle bacilli. Graph B shows the more gradual development of the silicotic reaction when there has been no prior infection. Charts C and D show the much slower and substantially lesser reaction to the fiber glass plastic dust with or without prior tuberculous infection.

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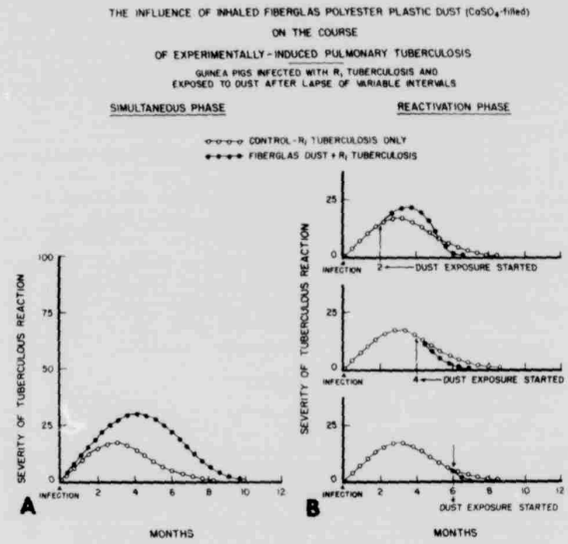


FIGURE 26-19. GUINEA PIG LUNG. LIMITED TUBERCULOGENIC ACTION OF INHALED FIBER GLASS PLASTIC DUST WITH CALCIUM SULPHATE FILLER

A. Simultaneous Phase

The tuberculous reaction is minimal and duration of active tuberculous reaction is about the same as in the control animals who were infected with RiRv tubercle bacilli but not exposed to dust.

B. Reactivation Phase

The tuberculous response in animals exposed to dust is not significantly more than that observed in control guinea pigs.

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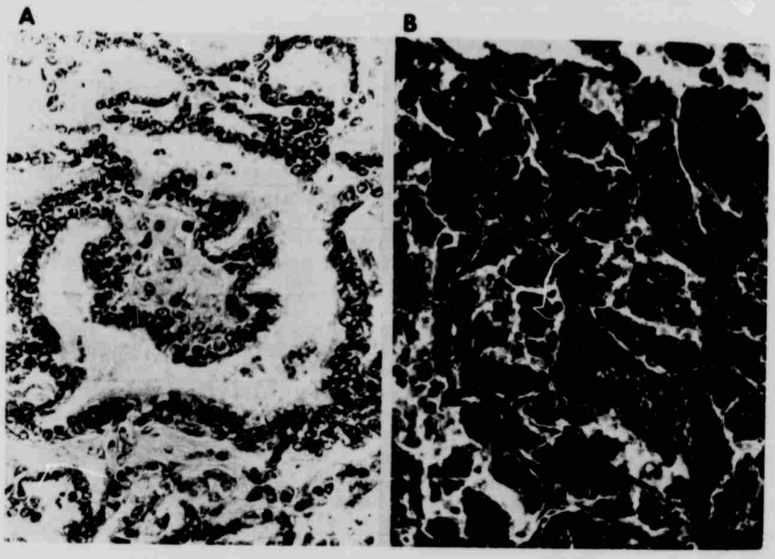


FIGURE 26-20. RAT LUNG. EXPERIMENTALLY INDUCED PULMONARY EPITHELIZATION AND CARCINOMA

A. Epithelization

Animal was exposed by inhalation to flux calcined diatomite for 12 months. Alveolar spaces became lined by cubodal epithelium. (X 240)

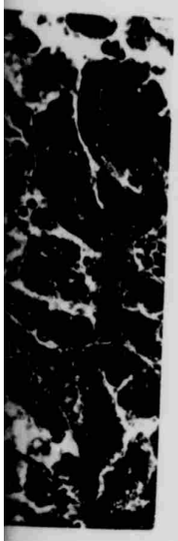
B. Adenocarcinoma

Detail of pulmonary carcinoma which developed after 14 months of exposure to beryllium sulfate. (X 480)

FIGURE 26-21. RA
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The graph shows
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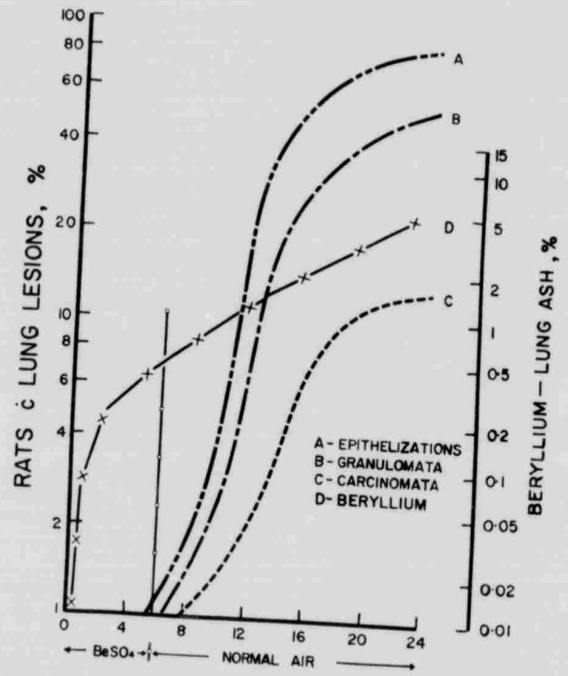


FIGURE 26-21. RAT LUNG. INHALATION EXPOSURE TO BERYLLIUM SULFATE AEROSOL CONCENTRATION 12 mg/cu ft

The graph shows the sequential appearance of epithelizations, granulomata and carcinomata in animals exposed to Be SO4 for 6 months and then transferred to normal air.

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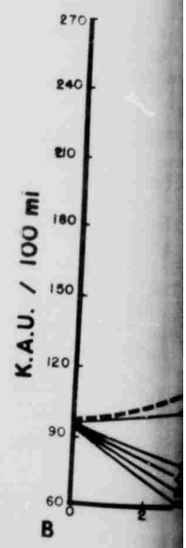
FIGURE 26-22. SERUM ALKALINE PHOSPHATASE LEVEL OF RATS

A. Exposure to Fiber Glass Plastic Dust and Beryllium Sulfate Aerosol

Fiber glass concentration was 4.6 mg/cu ft. $BeSO_4$ concentration was 12 gamma/cu ft. There is no discernable influence on the alkaline phosphatase.

B. Exposure to Beryllium Salts

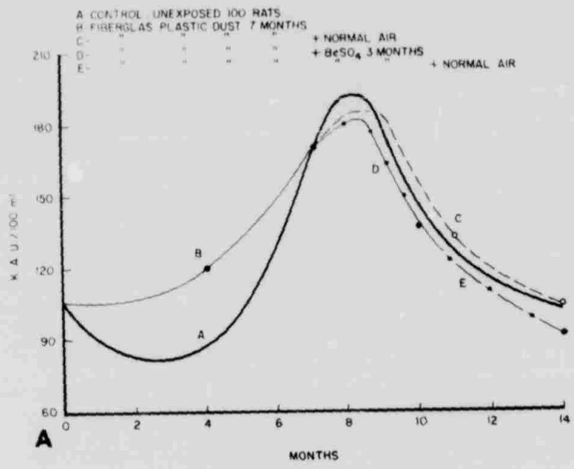
When the exposure was to $BeHPO_4$, BeO, BeF_2, $ZnMnBeSiO_4$ or $BeSO_4$ only, alkaline phosphatase was initally depressed and subsequently abnormally elevated.

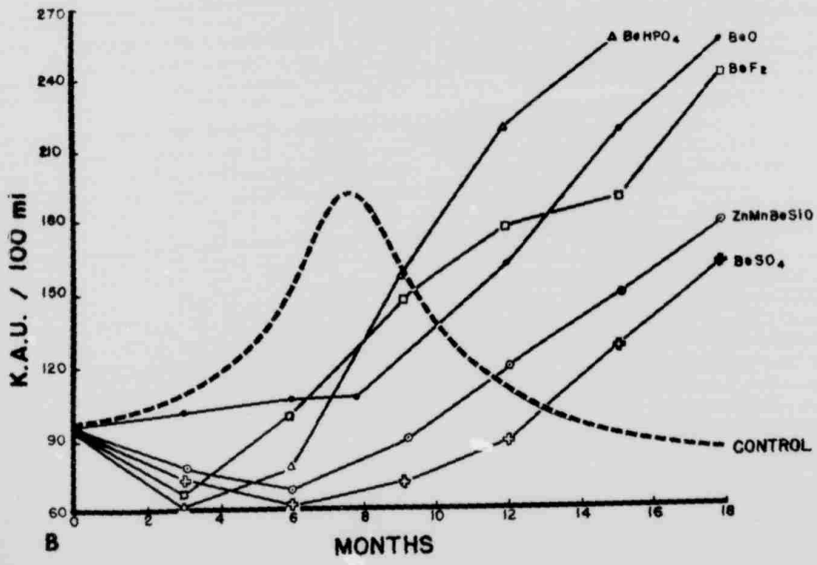


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preceded inhalation of ZnMnBeSiO 4. No tumors were observed either in the experiment with sequential exposure to BeO and fiber glass plastic dust. In the group of 94 rats which were exposed to fiber glass plastic dust and then BeSO4 and conversely, there were only 5 lung tumors, i.e. a percentile rate of only one-third that seen in the animals exposed only to the BeSO 4.

The sharply reduced occurrence of lung tumors in the animals which received inhalation exposure to both the fiber glass plastic dust and the beryllium compound prompts the inference that the fiber glass plastic dust possesses anticarcinogenic properties. However, consideration should be given to the fact that prior phagocytosis of fiber glass plastic dust particles may have left insufficient phagocytes to permit effective retention of ZnMnBeSiO 4 which is also a particulate aerosol dependent on phagocytes for its retention by the lungs. BeSO 4 is highly soluble, which would enable its penetration into pulmonary surface cells without significant interference by the fiber glass. That this is a possible explanation is suggested by the unmodified high prevalence of epithelization (64%) in spite of prior fiber glass plastic dust exposure, as compared with epithelization in rats exposed only to BeSO 4 (66%).

There also were some inequalities in the total periods of survival of animals. The average survival of rats in the experiments with the beryllium compounds only was appreciably shorter than the survival of rats in the combined exposure groups. Since oncogenesis in response to exposure to BeSO 4 and ZnMnBeSiO 4 is a direct function of elapsed time after exposure to the beryllium compound, this fact alone further dramatizes the low yield of lung tumors in the combined exposure experiments in which animals were kept alive longer so that they had more chance of developing lung cancer if they were going to do so. These facts all emphasize the fact that inhaled fiber glass plastic dust appears not to have an oncogenic effect on lung tissue.

A correlative study summarized in Figure 26-22a may shed light on this subject. In previously reported experiments, it was shown [Schepers, 1971a] that there is a decisive delayed progressive increase of serum alkaline phosphatase levels of rats after variable periods of exposure to BeSO 4, BeF 2, ZnMnBeSiO 4, BeO, and BeHPO 4 (Figure 26-22b). Serum alkaline phosphatase levels were, therefore, also determined in the groups of animals which had been exposed to fiber glass plastic dust only and to fiber glass plastic dust followed by inhalation of BeSO 4. The graphs of Figure 26-22a clearly show that the fiber glass plastic dust did not significantly disturb the alkaline phosphatase levels and the prior exposure of the rats to fiber glass plastic dust prevented the development of the customary late elevation of the alkaline phosphatase levels after exposure to beryllium sulfate. This finding suggests, therefore, that the inhalation of the fiber glass plastic dust may exert its retardant effect on oncogenesis not merely mechanically (e.g., preemption of alveolar phagocytes) but also in a biochemical manner.

Reaction of Human Lungs to Inhaled Fibrous Glass

Looking for glass fibers in human lung tissues is an unprofitable exercise unless one has a pretty good clue from the patients' work exposure

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that glass fibers may have been inhaled. First, it is widely held that particulate glass is harmless to human tissues so that the index of suspicion about fibrous glass as a factor in the pathogenesis of any pulmonary disorder is still low all round. Second, glass fibers are transparent when viewed by ordinary light microscopy and, therefore, not very likely to be recognized even when present. Third, the probability of finding a glass fiber in a plane of space at right angles to the optical axis of the microscope is extremely low with only 1 chance in 360 that the glass fiber will be in the horizontal plane, the chances of seeing fibers which are present are quite insignificant. This extremely low probability of identifying glass fibers in human tissue was further elucidated by the experience with the previously described animal experiments. It was only very rarely possible to find glass fibers in the tissues of the over 2000 test animals, even though it was known that the glass was there, since it had been experimentally introduced, and the presence of the glass could be demonstrated by chemical analysis of the lungs (Figure 26-23). Taking all these limitations into consideration, the discovery of even a few human cases with glass in the lung tissue is like finding the tip of a gigantic iceberg.

Figure 26-24 shows how clearly glass fibers can be visualized. In this case, there was severe peribronchial fibrocellular reaction. Most pathologists would be satisfied that the anatomical features constitute grounds for arriving at a diagnosis of non-specific peribronchial and interstitial fibrosis. To demonstrate this, the glass fibers in the adventitial sheath of fibrocellular tissue require examination of the section by phase contrast illumination at between 1000x and 2000x magnification. Many more fibers could of course be seen while examining the tissue with the microscope. Some glass fibers also lie within the dark shadows which represent nuclei and ferroproteinaceous material of phagocytic cells. No other pathogenic agent could be identified optically or chemically in this case, so that the likelihood of this being a fine glass fiber pneumoconiosis seems fairly good.

In the next case, the presence of the glass was slightly more readily demonstrated, however, again not until the tissue was scrutinized under high power and with the appropriate lighting system (Figure 26-25). The low power view (a) showed only the dense perivascular tissue reaction and provided no basis for a specific diagnosis beyond saying that the lung is the seat of a perivascular and interstitial fibrosis of undetermined etiology. In view b, irregular glass particles were inhaled along with glass fibers. Some of the fibers could be recognized in cross section. There were no fibers at right angles to the optical axis of the microscope. But the many "chunks" of glass provided the clue on how to detect the fibrous glass etiologic agent, i.e., to look for the materials likely to be inhaled along with glass fibers. Once these were seen, it takes only a little extra trouble to see the fibers in cross section and further search eventually will show a fiber sideview.

Glass fibers sometimes are rendered visible if there is pigmented material in the tissue section which is engulfed in the same phagocyte which has wrapped itself around a glass fiber. This is shown well in the third case (Figures 26-26a and b). In this instance, the low power view (a) shows only some fibrocellular reaction and pigment around sclerotic,

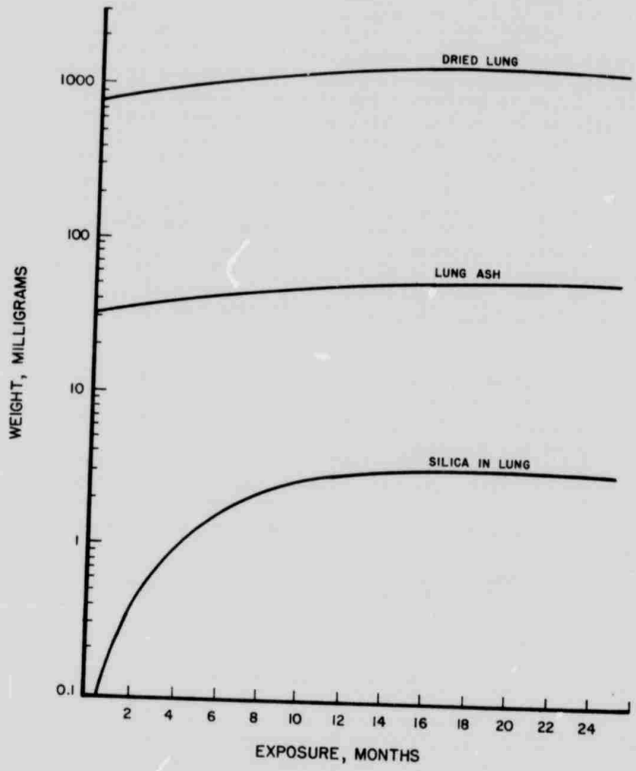


FIGURE 26-23. GUINEA PIG LUNGS. INHALATION EXPOSURE TO FIBER GLASS PLASTIC DUST

The graphs show the progressive accumulation of silica in the lungs of serially sacrificed animals, without corresponding significant change in the weight of the dried lung or lung ash.



FIGURE 26-24.
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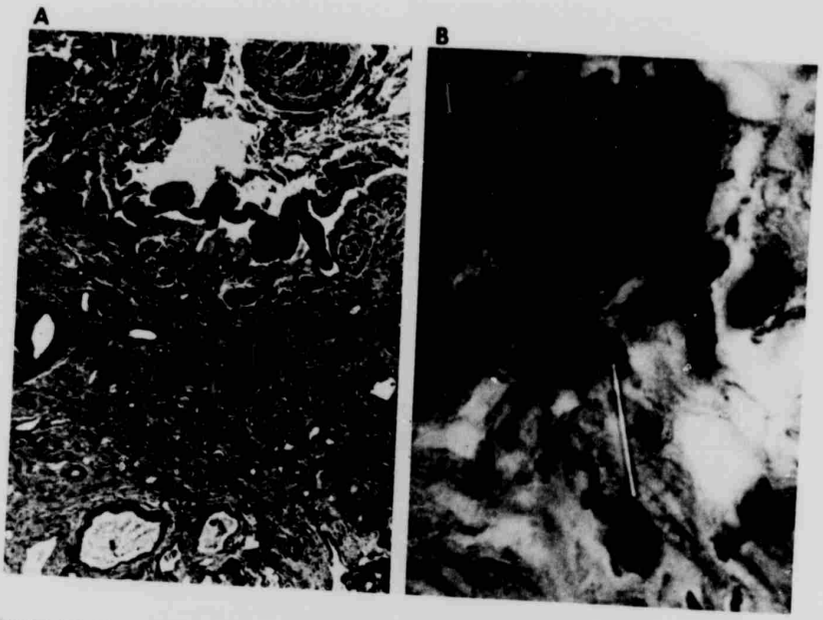


FIGURE 26-24. HUMAN LUNG. PULMONARY PATHOLOGY ASSOCIATED WITH GLASS FIBERS. NECROPSY SPECIMENS - MALE, AGED 54 WHO WORKED 20 YEARS AS AN INSULATION WORKER. DEVELOPED PROGRESSIVE PULMONARY AND CARDIAC INCAPACITATION. COURTESY, R. SMART, M.D.

A. Bronchus

Demonstrates marked luminal stenosis and distortion, focal epithelial polyposis, dense adventitial scarring, epithelization of deformed alveolar spaces. (X 75)

B. Phase Contrast Oil Immersion Photo

Two glass fibers are shown side view, one in the focal plane. Many others are seen in cross section. (X 2000)

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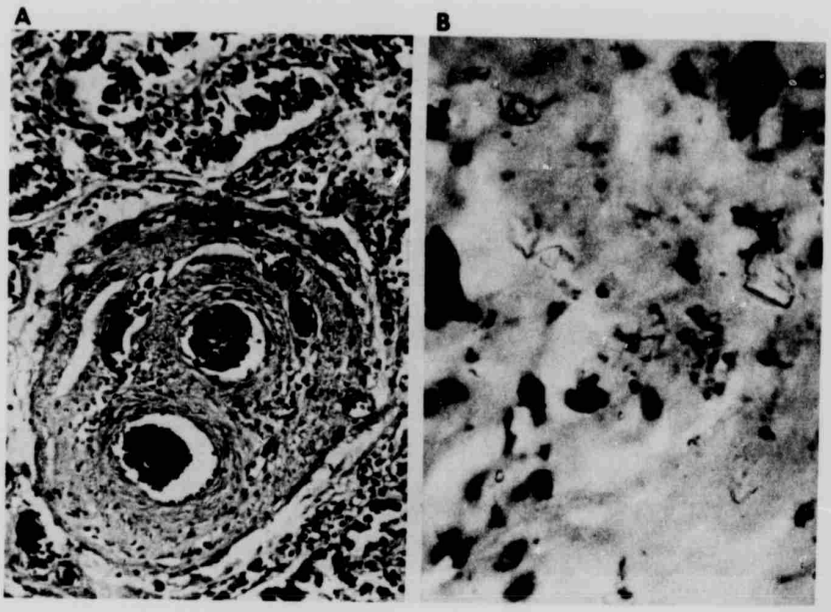


FIGURE 26-25. HUMAN LUNG. PULMONARY HISTOPATHOLOGY ASSOCIATED WITH GLASS PARTICULATES. MALE AGE 58, WHO DIED FROM CARDIOPULMONARY FAILURE FOLLOWING 32 YEARS EMPLOYMENT IN A GLASS UTENSIL FACTORY.

A. Pulmonary Blood Vessel

Myohypertrophy and dense circumferential scarring with sequential stenosis of pulmonary blood vessels. Interstitial mural infiltration and epithelization of surrounding alveoli. (X 75)

B. Perivascular Tissue

Fragments of glass are abundantly present. A few fibers are seen in cross section. There also are sparse opaque minute particles and a few larger chunks, none of which are birefringent. (X 2000)

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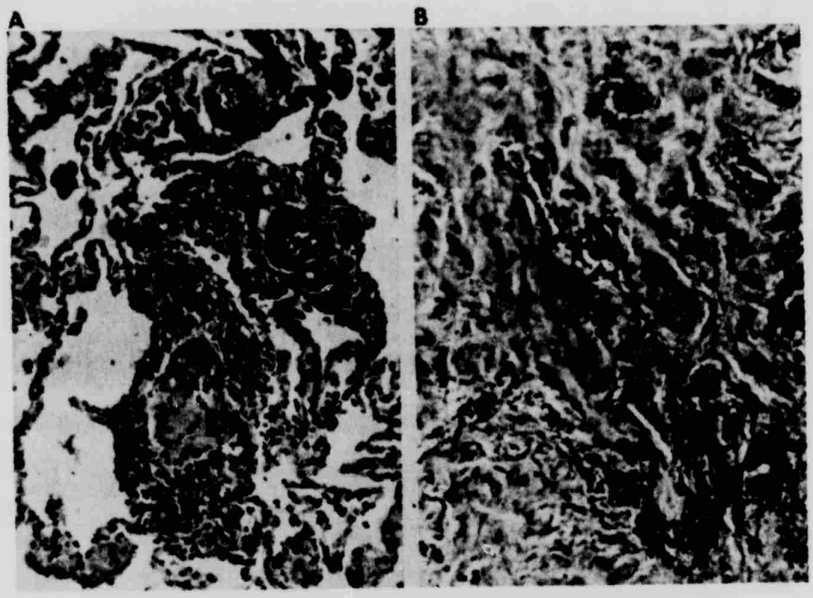


FIGURE 26-26. HUMAN LUNG. PULMONARY HISTOPATHOLOGY ASSOCIATED WITH FIBROUS GLASS. FEMALE, AGE 63, WHO DEVELOPED PROGRESSIVE PULMONARY DISABILITY FOLLOWING 25 YEARS OF SERVICE AS A MACHINIST IN A FIBER GLASS FABRIC FACTORY. BIOPSY SPECIMEN TAKEN 2 YEARS AFTER CESSATION OF EMPLOYMENT.

A. Lung Parenchyma

Several small blood vessels display irregular thickening of their walls and perivascular cellular infiltration and fibers. Alveolar walls are irregularly scarred. (X 200)

B. Detail of Perivascular Tissue

Positive phase contrast microscopy reveals numerous transparent fibers indistinguishable from glass. Particulate material represents carbon. No silica demonstrated. (X 1000)

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distorted, stenotic small pulmonary blood vessels. Ones first temptation in this type of case is to seek the explanation for the interstitial vascular and perivascular pulmonary tissue reaction in the presence of the pigment. On further petrographic analysis, however, the pigment proved to be merely innocuous carbon and no pathogenic agent could be seen. High power phase contrast microscopy subsequently revealed short glass rods in the perivascular cuffs of the damaged blood vessels (b). Two glass rods were visualized as lying almost at right angles to the optical axis of the microscope and several others were seen in oblique views. Numerous other fibers could be recognized only as minute transparent dots. Some of these were rendered visible by contrast with the opaque material clustered about them.

In the fourth case illustrating this human series, the presence of the glass fibers was revealed by virtue of the fact that the fibers are surrounded by ferroproteinaceous material of the same kind as that which surrounds the asbestos fibers in "asbestos bodies." Figure 26-27 demonstrates this feature well. Once more the low power view provided no clue. There was abundant coarse opaque material in the fibrocellular zones which surround small blood vessels of the lung. Again the pathologist would be at a loss to say just what this tissue reaction is due to and at the most would have to be satisfied with a diagnosis of subacute fibrocellular interstitial and perivascular pulmonary lesion of undetermined, possibly environmental, etiology. The high power analysis, however, provides a complete clue (b). Glass fiber, which is surrounded by a dense ferroproteinaceous coating rendered visible by Perl's stain, may be clearly seen. The free ends of the glass rod project beyond the main mass of ferroprotein around the body of the fiber.

In the fifth human case, a further diagnostic problem was illustrated. The low power view (Figure 26-28a) demonstrated severe intimal proliferation, almost to the point of occlusion, of a small pulmonary artery whose delineating elastic laminae had been clearly revealed by Weigert's technique. In the surrounding fibrous tissue there are no etiological clues. Without further study, the pathologist may be tempted to diagnose this as a case of degenerative vascular disease of the lung, perhaps idiopathic in etiology. Examination of the fibrous zone under high power, after applying Perl's stain, revealed typical ferruginous bodies (Figure 26-28b). The majority of these dark structures actually are asbestos bodies since delicate amosite fibers can be delineated within them. A minority of the ferroproteinaceous structures are, however, wrapped around glass fibers. In this case we are dealing with a mixed pneumoconiosis in whose pathogenesis asbestos plays a major role. Since the "asbestos bodies" are so much better known, it can easily occur that a case like this would be diagnosed as pure asbestosis and the contributory etiological role of the fibrous glass would be entirely missed.

By using the aforementioned techniques and criteria over the past 2 decades, this author has been able to identify more than a dozen cases of lung disease in whose pathogenesis inhaled fibrous glass played or may have played a primary or a contributory etiological role. Undoubtedly some earlier cases were missed, for the full realization that special techniques must be used did not come until several years had elapsed. There was originally also much skeptical rejection of the notion that there may be an



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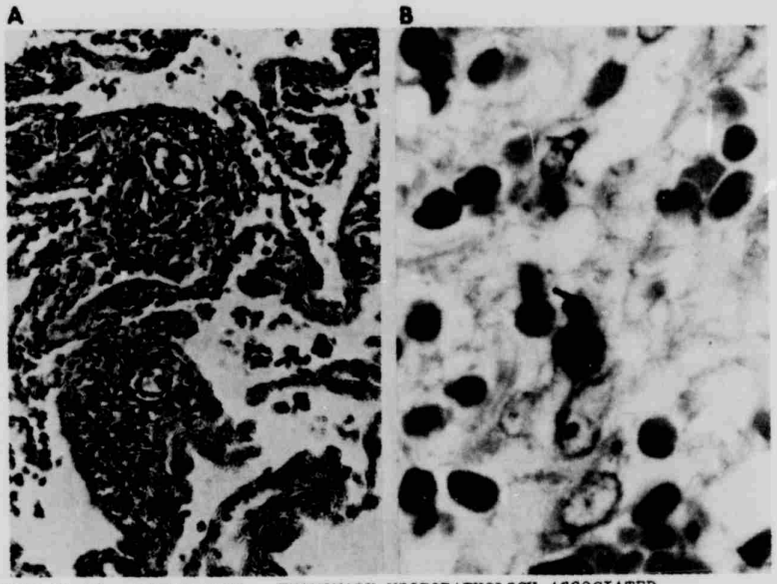


FIGURE 26-27. HUMAN LUNG. PULMONARY HISTOPATHOLOGY ASSOCIATED WITH MIXED DUST INCLUDING FIBROUS GLASS. MALE, AGED 61 YEARS, WHO DEVELOPED INCAPACITATING PULMONARY AND CARDIAC PROBLEMS. HE WORKED 40 YEARS IN A VARIETY OF TRADES INCLUDING FIBER GLASS INSULATION, ROCK WOOL POURING, TARRING AND PITCHING AND OTHER ROOFING TRADES

A. Lung Parenchyma

Shows wide sheaths of fibrocellular material around stenosed blood vessels. Several coarse glass fibers are shown. (X 200)

B. High Power Microscopy of Perivascular Tissue

A glass fiber is seen in the center of the field. The fiber projects at either end from a ferruginous body which has been rendered visible by staining for iron salts. (X 2000)

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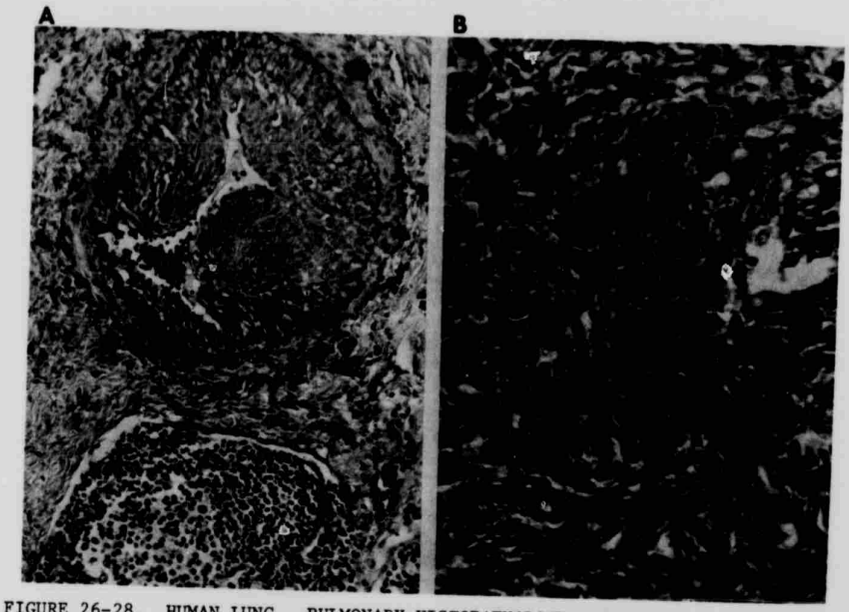


FIGURE 26-28. HUMAN LUNG. PULMONARY HISTOPATHOLOGY ASSOCIATED WITH GLASS AND OTHER PARTICULATES. FEMALE, AGE 49, WHO DIED FROM COR PULMONALE AFTER SEVERAL YEARS OF ILL HEALTH FOLLOWING 19 YEARS EMPLOYMENT AS A SYNTHETIC TEXTILE MACHINIST.

A. Small Pulmonary Artery.

The lumen is almost occluded by eccentric masses of fibrocellular intimal plaques. (X 100)

B. Adventitial Scar Tissue

Between irregularly distributed fibrocytes and collagen bundles there are numerous ferruginous bodies and fewer almost bare fibers. Asbestos fibers, although suspected, could not be conclusively demonstrated. Most of the ferruginous bodies had transparent cores consistent with glass or plastic textile filaments. (x 400)

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entity such as fibrous glass pneumoconiosis in man, since the earlier animal experimentation did not suggest the type of massive pathology found with exposure to silica and other known nocuous materials. Earlier epidemiological studies on populations of employees of fiber glass factories also gave a negative yield of symptomatic cases, radiological findings, or mortality.

Only slowly did cases emerge which convinced this author that fibrous glass can and does cause harm to human lungs under certain circumstances. The discovery that monkeys produced such prominent lesions so rapidly helped to provide stimulus for a search after human cases. The fact that the majority, even the overwhelming majority of human workers with fibrous glass, do not develop overt clinical disease should not lead to the conclusion that no human beings ever will be hurt. The five examples illustrated in Figures 26-24 to 28 dramatize how severely human lung tissue can be damaged.

Quite recently, several human cases have been identified clinically. In one patient, the occupational history was overwhelmingly convincing. The patient had worked his whole life in his father's insulating business and, therefore, could verify in a most convincing way exactly what materials he was exposed to and how. He remained quite well for about 15 years. Then he developed progressive upper and lower respiratory tract obstructive and restrictive ill health and peripheral neuropathy. He never smoked, and lived in a healthy rural area, played golf often for relaxation and displayed no allergies or hereditary illnesses. Diagnosis was finally established by bronchial biopsy, which showed abundant glass fibers in the mucosa. The neuropathy was a further clue and may provide an explanation why he and other exceptional fibrous glass industry workers became ill. The solvents used in gluing together bats of fiber glass wrapped around heating and cooling ducts, generally are very volatile, being of the methyl-butyl-ketone group. The vaporized solvent is inhaled by insulation workers and ultimately induces the peripheral neuropathy. On the way to the nerve sheaths the solvents dissolve into and injure any cells which contain lipids. Since the lining cells of the lung are of this type, extensive changes are produced in respiratory tract epithelial surface properties. This impairs defensive reactions to concurrently inhaled fibrous glass particles, facilitating their retention in the lungs and the accumulation of koniophores in lymph sinuses and mucosal tissues. This caused the pulmonary symptoms.

After the patient had been removed from the fibrous glass dust and solvent atmosphere for about a year, the majority of the extreme symptoms receded, suggesting that at least in part the pulmonary reactions were reversed. This is consistent with the fact that after cessation of exposure to methyl-butyl-ketone many induced lesions are reversed.

It will serve little purpose in a review of the present kind to discuss other examples of clinical cases since the diagnosis of these cases was established simply on the basis of biopsies, the finding of glass fibers in abnormal lung tissue and the lack of identification of other etiological agents. The underlying pathology has perhaps been sufficiently delineated by the preceding cases.

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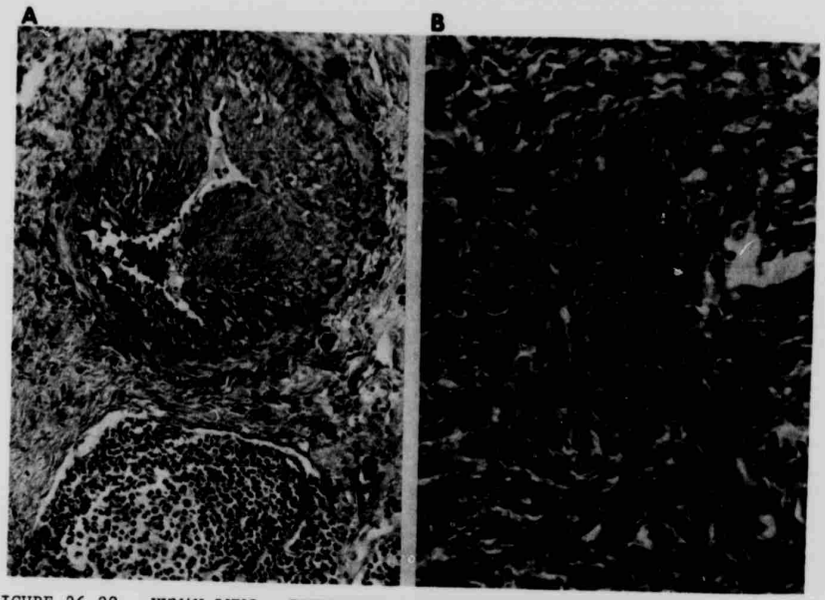


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Comparative Pathogenicity of Fibrous Glass

It remains now to assess the pathogenicity of fibrous glass in terms of the relative potential of different varieties of silicates to cause pulmonary damage. The physical appearances of seven of the principal varieties of industrially significant silicates are represented in Figures 26-29 and 26-30 in juxtaposition to typical fiber glass (Figure 29d). In Table 26-14 the silicates are arranged along a scale of zero to nine in ascending order of biological potency. Micronized glass and volcanic glass are given a zero rating. In other words, when glass is finely ground into particles 1 μ or smaller, it becomes biologically as inert as talc (Figure 26-29a), actinolite (Figure 26-29b) or tremolite without long rods (Figure 26-29c) or any of the biologically inert kaolinites or montmorillonite clays.

Perlite, flake glass, and short fiber glass of greater than 5 μ caliber, when inhaled, are classifiable with group 1 silicates such as vermiculite, pyrophyllite and biotite since they cause only a reversible thesaurosis.

When the fiber glass, rock wool or glass fibers are long (20 μ +) and of fine caliber ($<$ 5μ) or if the glass fibers are mixed with polyester resin plastic and its fillers, the biological reaction tends to be more than a simple thesaurosis and can be classified as a group 2 thesaurismosis.

When tuberculous infection has been combined with the inhalation exposure to fibrous glass or particulate volcanic glass, the resultant Grade 3 pulmonary tissue reaction is of a severity comparable to that which has been observed after exposure to sericite, antigorite, sillimanite or mica.

The tissue reactions caused by the ribbon or lathlike silicates, attapulgite and paligorskite, fall between the worst effects of the fibrous or particulate glass aerosols and the first group of asbestiform minerals (chrysotile, brucite and endellite). This group, which induces Class 4 lung tissue effects (Table 26-14) belong to the amphibole minerals of which chrysotile Class 5 also is representative. They consist of aluminosiloxane sheets rolled on themselves to form hollow fibers (Figure 26-30a). The true asbestos minerals, anthophyllite (Figure 26-30b), amosite (c) and crocidolite (d) respectively occupy spots 6, 7 and 8 on the ladder of relative pathogenicity (Table 26-14). These substances cause true pulmonary fibrosis which may have dire sequelae and complications. However, asbestos fibers alone are by no means as harmful as they can be when inhalation exposure is combined with exposure to silica. Glass fibers also appear to have this potentiating effect on silica.

Summarizing the foregoing, it is clear that, although fibrous glass morphologically resembles the asbestiform silicates, its biological action potential is considerably less, especially if the glass particles are reduced to their finest form. The one exception to this rule is fine glass fiber. Extremely slender glass fibers are injurious to lung tissue especially if the fibers also are relatively long. The combination of glass fiber inhalation exposure with tuberculous infection or silica

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exposure, have a potentiating effect on one another. Fibrous glass does not, however, have the capacity to cause lung cancer or act as a cocarcinogen with beryllium salts which are highly carcinogenic.

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FIGURE 26-29. DIFFERENTIAL MORPHOLOGY OF SILICATE MINERALS

A. Talc

The talc particles are extremely small and consist of flakes which can fragment to thin sheets close to the siloxane molecular configuration. (x 20,000)

B. Antigorite

The predominant particles occur as plates and ribbons. Occasional particles are more solid. (X 20,000)

C. Tremolite

The component particles are oblongate in form, cleavage being in all three planes of space. Occasional particles cleave predominantly in two planes, so that they assume a rod-like form. (X 200)

D. Fiber Glass

The glass fibers range from 0.5 to 5 μ in caliber. Since fiber glass is formed artifically as long filaments the "length" of fibers would be an accidental function. (X 100)

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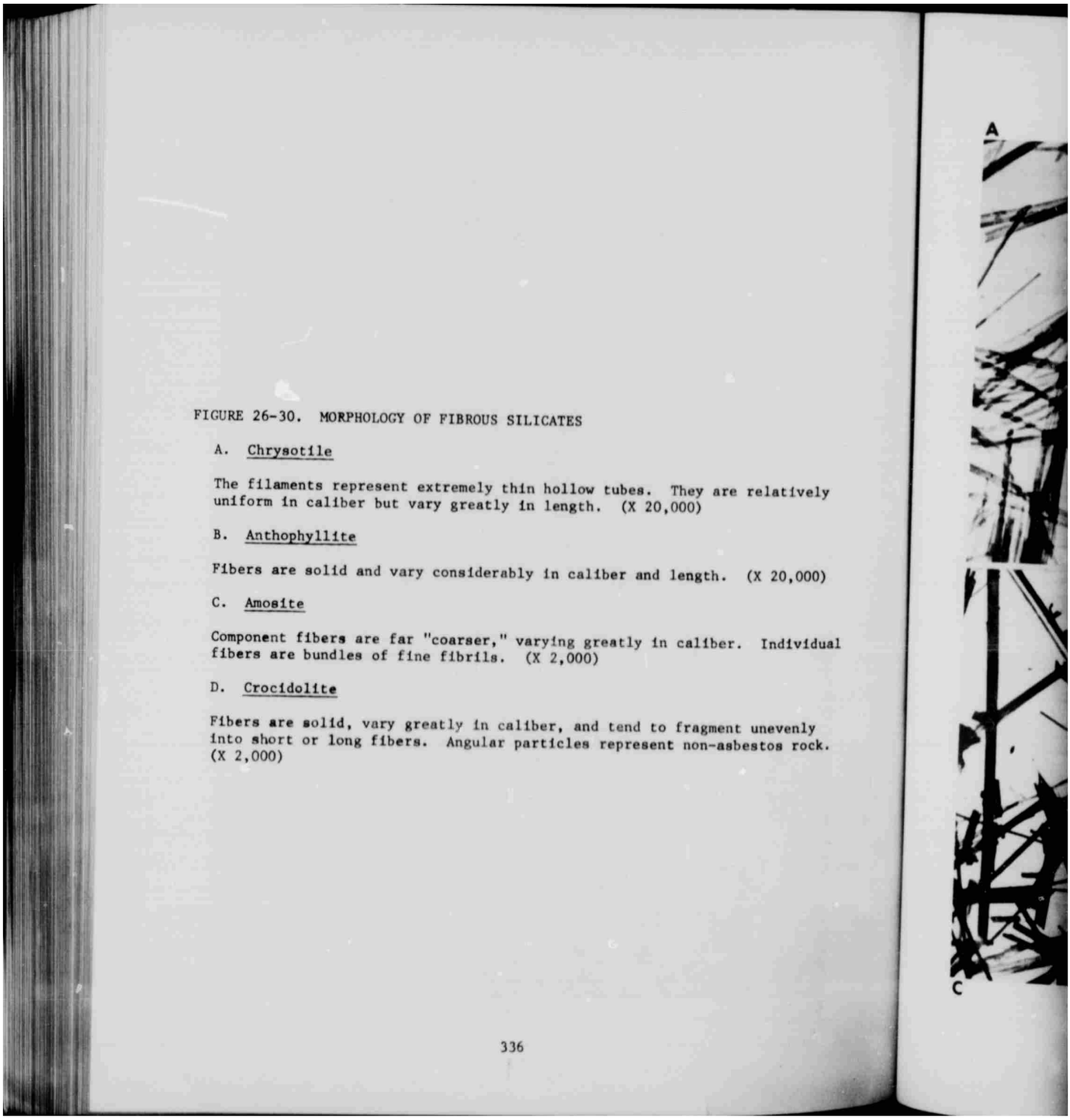


FIGURE 26-30. MORPHOLOGY OF FIBROUS SILICATES

A. Chrysotile

The filaments represent extremely thin hollow tubes. They are relatively uniform in caliber but vary greatly in length. (X 20,000)

B. Anthophyllite

Fibers are solid and vary considerably in caliber and length. (X 20,000)

C. Amosite

Component fibers are far "coarser," varying greatly in caliber. Individual fibers are bundles of fine fibrils. (X 2,000)

D. Crocidolite

Fibers are solid, vary greatly in caliber, and tend to fragment unevenly into short or long fibers. Angular particles represent non-asbestos rock. (X 2,000)

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TABLE 26-14

COMPARATIVE PATHOGENICITY OF SILICATES
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RATING	SUBSTANCE
0	Talc, Actinolite, Saponite, Kaolinite, Montmorillonite Micronized glass or micronized Perlite
1	Tremolite, Vermiculite, Pyrophyllite, Biotite, Perlite, Flake glass, less than 20 μ Fiber glass and of 5 μ + caliber
2	Tremolite with predominant long rods. Fiber glass, Glass wool, Rock wool 20 + μ long and 5 μ caliber. Fiber glass Plastic
3	Sericite, Antigorite, Sillimanite, Mica Fiber glass or Glass wool or Perlite plus tuberculosis
4	Attapulgite, Paligorskite, Brucite, Endellite
5	Chrysotile
6	Anthophyllite
7	Amosite
8	Crocidolite
9	Silica plus Asbestos or other fibrous minerals including fibrous glass

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TIMA'S HEALTH RESEARCH PROGRAM IN THE INSULATION INDUSTRY

Jon L. Konzen

Introduction

DR. DECOUFLE: Our next speaker is Dr. Jon L. Konzen.

Presentation

DR. KONZEN: Mr. Barnhart, Executive Director of the Thermal Insulation Manufacturers Association has described the organization. I am the Chairman of the Medical and Scientific Committee of TIMA. Our committee is comprised of a number of health and medical representatives from member companies.

TIMA member companies have cooperated in or sponsored health research into the health effects of fibrous glass beginning with Gardner's inhalation research at Saranac Lake in the late 1930's. We are continuing through the present time. This research has resulted in at least 35 papers or publications on the subject. [Gardner, 1940, 1942; Siebert, 1942; Sulzberger, 1942; Schwartz and Botvinick, 1943; Schepers and Delahant, 1955; Schepers, 1955, 1958, 1959a, 1959b; Heisel and Mitchell, 1957; Schepers et al, 1958; Gross, 1960; Nasr, 1967; Wright, 1968; Heisel and Hunt, 1968; Johnson et al, 1969; Utidjian, 1968, Gross et al, 1970a, 1970b, 1970c, 1971; 1973; Possick et al, 1970; Utidjian and de Treville, 1970; de Treville et al, 1970; Fowler et al 1971; Balzer et al, 1971a; 1971b, 1971c, 1971d; Cholak and Schafer, 1971; Nasr et al, 1971; Corn and Sansone, 1973; Enterline, 1973] The findings of many of these studies have been included in this symposium. A number of the studies were designed to delineate the health effects of fibrous glass exposure to man.

The animal research employed respirable size fibrous glass administered by inhalation and intratracheal injection techniques. It has not demonstrated malignant nor significant non-malignant pulmonary health effects.

Fibrous glass workers were first exposed to airborne fibrous glass over 35 years ago. During this period, fibrous glass workers were the subjects of several health evaluations. [Nasr, 1967; Wright, 1968; Utidjian, 1968; Utidjian and de Treville, 1970; Gross et al, 1970c, 1971; de Treville et al, 1970; Nasr et al, 1971; Enterline, 1973; Wagner, 1974] Human studies have demonstrated no significant chronic pulmonary health effects to date. However, research demonstrates that fibrous glass will, in some individuals, cause a transitory mechanical skin irritation. [Sulzberger, 1942; Heisel and Mitchell, 1957; Heisel and Hunt, 1968; Possick et al, 1970]

Industrial hygiene studies [Fowler et al, 1971; Balzer et al, 1971a; 1971b, 1971c, 1971d; Corn and Sansone, 1973], incorporating both light and electron microscopic analysis, were conducted in areas of fibrous glass manufacturing, fabrication, installation, ambient air and end use

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applications. These studies demonstrated low airborne concentrations of respirable fibrous glass.

Recent animal studies employing surgical implantation techniques [Pott and Friedrichs, 1972; Stanton and Wrench, 1972; Davis, 1972] suggest that the geometry of the fibers rather than the chemical composition determine their ability to cause adverse effects in animals. These studies, although not directly applicable to man, raise sufficient question to warrant additional research activity.

TIMA's Board of Directors has approved, in principle, the Medical and Scientific Committee's proposal for further fibrous glass health research studies. These studies are divided into eight projects. The first three are underway.

The first project is an epidemiological study of long-term effects of exposure to approximately 30,000 fibrous glass and mineral wool workers. We are including mineral wool workers in this study for two reasons: because it is also a man-made fiber and, because of the exposure longevity experienced in mineral wool manufacturing.

The purpose of this mortality study is to (1) determine the incidence of cause of death in man-made mineral fiber workers as compared to suitable control populations, with particular emphasis on malignant and other diseases of the respiratory system; (2) determine the relationship between level and duration of exposure and incidence of any disease; and (3) provide a basis for future monitoring of the health of man-made mineral fiber workers.

The second project is an industrial hygiene study to determine airborne fiber concentration in fibrous glass and mineral wool manufacturing facilities and in-field installations.

The purpose of this study is (1) to establish a valid methodology for determining concentrations and characterization of airborne fibers to which workers are exposed; (2) to identify the nature of the fiber; and (3) to determine the physical dimensions of airborne fiber within specific size ranges.

The third project involves the development and production of glass fibers of pre-determined diameters and length in the very fine diameter range to: (1) make available precisely sized fibers for animal studies; (2) facilitate control of the fiber dose administered to test animals; and (3) determine the precise fiber diameter and length combination that produce either negative or positive results in animals.

Five other projects are being developed for later implementation. These include: (1) animal studies to make comparisons of deposition patterns by inhalation as compared to intratracheal injection methods of introducing fibers into test animals; (2) investigate the mechanism of fiber migration in the lungs and gut; (3) intrapleural injection studies of specifically defined fibers by diameter and length to determine dose response and relationship to malignant disease in the pleura of animals; (4) to simulate the lifetime dosage experienced by man in animals by

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introduction of the fibers into the lungs and gut, and (5) epidemiological studies of currently employed manufacturing workers exposed to fibrous glass and mineral wool.

Summary

1. There have been recent animal studies which suggest that the geometry of the fiber rather than the chemical composition, determine their ability to cause adverse effects when these fibers are implanted in animals.

2. TIMA and its member companies have been sponsoring and cooperating in research into the health of fibrous glass workers over the last 35 years.

3. TIMA is sponsoring further health and environmental investigations of fibrous glass and mineral wool exposures.

4. Industrial hygiene studies sponsored by TIMA have demonstrated low concentrations of respirable sized fibers in fibrous glass insulation and textile manufacturing areas.

5. All the medical research reported to date has demonstrated no significant chronic health effects in man as a result of exposure to fibrous glass.

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MORTALITY PATTERNS AMONG FIBROUS GLASS PRODUCTION WORKERS --PROVISIONAL REPORT

David Bayliss
John Dement
Joseph K. Wagoner

Introduction

DR. DECOUFLE: Our last paper will be presented by Mr. David Bayliss, Assistant Chief of Biometry Branch of the Division of Field Studies and Clinical Investigations of NIOSH.

Presentation

MR. BAYLISS: The industrial production and commercial use of fibrous glass began in the United States slightly more than 30 years ago. As a result of the human carcinogenic and fibrogenic effects of asbestos, it was only natural that concern should be directed toward an evaluation of the pathogenicity of fibrous glass.

To date, experimental studies have produced conflicting results. [Pott and Friedrichs, 1972; Schepers, 1955, 1958, 1959, 1961; Schepers and Delahant, 1955; Schepers et al, 1958; Gross et al, 1970; Wenzel et al, 1969; Botham and Holt, 1971; Davis, 1972; Stanton and Wrench, 1972] This is not to be unexpected as many of these experimental studies suffered from one or more defects, such as insufficient lapse of time since onset of exposure, questionable mode of exposure (route of entry and size of fiber), inadequate sample size and lack of controls. Nevertheless, when fibrogenicity and carcinogenicity of fibrous glass was demonstrated, it was related more to the dimensions of the fiber than to its physio-chemical properties. [Pott and Friedrichs, 1972; Davis, 1972; Stanton and Wrench, 1972]

Only in recent years has sufficient time elapsed to make feasible studies among humans of the potential latent effects of occupational exposure to fibrous glass. Studies of employees in the fibrous glass industry by Wright, [1968] Gross et al, [1971] Utidjian and de Treville [1970] and Nasr [1971] indicated essentially negative results. However, it must be pointed out that each of those studies have a common limitation. They were cross-sectional prevalence studies conducted among current employees. There was no reference made to former employees, a group shown in many other industrial settings to manifest the majority of latent biological effects. With this limitation in mind and the potential carcinogenicity of fibrous glass posed by Stanton and Wrench [1972] and Pott and Friedrichs [1972], the National Institute for Occupational Safety and Health (NIOSH) undertook a study of the fibrous glass manufacturing industry.

Retrospective Cohort Study

Selected for the study were all white males initially employed during the period January 1, 1940, through December 31, 1949. All were employed

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in a major fibrous glass products construction manufacturing facility. Potentially eligible employees were found through an exhaustive search of company employment files. The study was subsequently limited to those individuals having achieved 5 or more years of employment in fibrous glass production, packing or maintenance activities. These restrictions were imposed to:

a) Eliminate effects of silica exposure present in the predecessor glass bottle plant operated at this facility. Glass bottle production ceased in the 1930's.

b) Minimize possible bias resulting from the unfortunate destruction of company employment files during a flood in or about 1960.

c) Minimize exposure, subsequent to 1940, to dust other than fibrous glass.

The final study cohort consisted of 1,448 white males.

Follow-up of all study cohort members was attempted from the time of termination of employment to June 1, 1972. Vital status information was obtained through records maintained by federal, state and local governmental agencies, including sources such as the Social Security Administration, and the vital statistics office of various states. For individuals not located through conventional data resources, use was made of state motor vehicle licensing agencies, R.L. Polk Directories, post office mailing correction service, voter and property tax records, marriage license records, the Soldiers' Relief and Veterans' Service Commission, the Veterans' Administration, city and county health departments, and area funeral homes. With this intense follow-up program, members of the study group were not lost to observation (Table 28-1). Death certificates were obtained for the known dead and cause of death were interpreted by a qualified nosologist according to the "Revision of the International Lists of Diseases and Causes of Death" in effect at the time of death. [1957] Causes of death were obtained for all individuals.

A modified life-table technique was used to obtain person-years at risk of dying by 5-year calendar time periods, 5-year age groups, duration of employment, and number of years since onset of initial employment. Comparison was made between the observed number of deaths in the study cohort and that number expected on the basis of age - calendar time cause-specific mortality rates for the general white male population of the United States.

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TABLE 28-1

STATUS AS OF JUNE 1, 1972 AMONG WHITE MALES WHO WERE INITIALLY EMPLOYED BETWEEN JANUARY 1, 1940 AND DECEMBER 31, 1949 AND SUBSEQUENTLY ACHIEVED 5 OR MORE YEARS OF EMPLOYMENT IN FIBROUS GLASS PRODUCTION, PACKING OR MAINTENANCE ACTIVITIES.

Status As Of June 1, 1972	Study Cohort Members No.	%
Known to be alive	1,072	74.0%
Known to be deceased	376	26.0%
Death certificate obtained	376	26.0%
Death certificate outstanding		
Not known to be alive or deceased	0	0.0
Total	1,448	

From January, 1940 to June, 1972, a total of 376 deaths were observed among the study cohort, whereas 404.24 would have been expected (Table 28-2). This deficit of overall mortality was anticipated. A certain level of good health was required of all individuals initially employed at this fibrous glass production facility through an extensive pre-employment medical screening program fully implemented in 1946. In support of this interpretation is the observation that the deficit of overall mortality among fibrous glass workers disappeared within 15 years since onset of employment. Further support for this interpretation is the significant deficit of deaths due to tuberculosis (0 Obs. vs. 4.69 Exp.; $p<0.05$.

Among the total study cohort, no excessive risk was demonstrated for all malignant neoplasms combined. In the category of respiratory malignancy, only 16 deaths were observed among these workers as contrasted with 20.23 expected.

For only one cause-of-death category was an excessive risk demonstrated. Nineteen "non-malignant diseases excluding influenza and pneumonia" were observed while only 10.04 were expected, an excess significant at $p<0.05$.

Is it possible that individuals who choose employment in the fibrous glass industry may have a predisposition to respiratory disease? To evaluate the role of selective factors, analyses were made of the non-malignant respiratory disease risk according to time interval since onset of initial employment in the fibrous glass industry. By examination of the mortality pattern in Table 28-3, it may be seen that an excessive risk occurred only after 10 years since onset of employment. This lack of an excessive non-malignant respiratory risk during the first 9 years is not

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TABLE 28-2

OBSERVED AND EXPECTED DEATHS ACCORDING TO CAUSE AMONG WHITE MALES WHO WERE INITIALLY EMPLOYED BETWEEN JANUARY 1, 1940 AND DECEMBER 31, 1949 AND SUBSEQUENTLY ACHIEVED 5 OR MORE YEARS OF EMPLOYMENT IN FIBROUS GLASS PRODUCTION, PACKING OR MAINTENANCE ACTIVITIES THROUGH JUNE 1, 1972.

Causes of Death	List Number*	Observed	Expected
Tuberculosis	001-019	0	4.69+
Malignant neoplasms	140-199	54	64.09
Digestive system	150-159	25	22.93
Respiratory system	160-164	16	20.23
Other & unspecified	140-149, 165-199	13	20.93
Vascular lesions affecting CNS	330-334	30	32.84
Diseases of heart	400-443	163	179.86
Non-malignant respiratory disease	470-527	25	19.96
Influenza & pneumonia	480-493	6	9.92
Other respiratory disease	470-475, 500-527	19	10.04+
Cirrhosis of liver	581	2	8.93+
Violent deaths	800-958	39	34.37
All other known causes		63	59.50
Unknown causes		0	-
Total		376	404.24

+ Significant at $p<0.05$
* 7th Revision of international lists of Diseases and causes of death

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ed	Expected
	4.69+
	64.09
	22.93
	20.23
	20.93
	32.84
	179.86
	19.96
	9.92
	10.04+
	8.93+
	34.37
	59.50
	-
	404.24

consistent with a self-selection of individuals having overt disease. When consideration was given to the risk of respiratory malignancy according to interval since onset of employment, no excessive risk was demonstrable even after 20 years of observation (Table 28-4).

TABLE 28-3

OBSERVED AND EXPECTED NON-MALIGNANT RESPIRATORY DISEASE DEATHS (LESS INFLUENZA AND PNEUMONIA) ACCORDING TO TIME SINCE ONSET OF EMPLOYMENT AMONG WHITE MALES WHO WERE INITIALLY EMPLOYED BETWEEN JANUARY 1, 1940 AND DECEMBER 31, 1949 AND SUBSEQUENTLY ACHIEVED 5 OR MORE YEARS OF EMPLOYMENT IN FIBROUS GLASS PRODUCTION, PACKING OR MAINTENANCE ACTIVITIES THROUGH JUNE 1, 1972.

Interval since onset of Employment (Years)	Observed	Expected
5 - 9	0	0.53
10 - 19	9	2.90++
20 - 29	10	6.11
(≥) 30	0	0.50
Total (≥) 5 years	19	10.04+
Total (≥) 10 years	19	9.51++

++ Significant at p<0.01
+ Significant at p<0.05

Industrial Hygiene Surveys

The apparent lack of an unusual malignant respiratory disease risk among individuals in the study cohort must be evaluated, however, in terms of the environment to which the workers were exposed. To evaluate this environment, industrial hygiene surveys were conducted in the study plant and in three other insulation plants producing similar products. These surveys determined total dust concentrations (mg/cu m), fiber exposures (fibers/ml) and airborne fiber diameter and length distributions.

Table 28-5 shows a summary of airborne fiber concentrations for the four insulation plants surveyed. An average fiber concentration of 0.08 fibers/ml was observed for plant D, the study plant. In addition, very low

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TABLE 28-4

OBSERVED AND EXPECTED MALIGNANT RESPIRATORY DISEASE DEATHS ACCORDING TO TIME SINCE ONSET OF EMPLOYMENT AMONG WHITE MALES WHO WERE INITIALLY EMPLOYED BETWEEN JANUARY 1, 1940 AND DECEMBER 31, 1949 AND SUBSEQUENTLY ACHIEVED 5 OR MORE YEARS OF EMPLOYMENT IN FIBROUS GLASS PRODUCTION, PACKING OR MAINTENANCE ACTIVITIES THROUGH JUNE 1, 1972.

Interval Since Onset of Employment (Years)	Observed	Expected
5 - 9	2	1.44
10 - 14	2	2.63
15 - 19	4	4.09
20 - 24	1	5.87
25 - 29	7	5.30
$\geq$ 30	0	.90
Total $\geq$ 5 years	16	220.23

TABLE 28-5

SUMMARY OF FIBER CONCENTRATIONS (FIBERS/ml) IN LARGE FIBER INSULATION PRODUCTION FACILITIES

	Insulation Plant			
Summary For All Operations	A	B	C	D*
Mean conc. (fibers/ml)	0.06	0.11	0.13	0.08
Range	0.01-0.13	0.00-0.47	0.04-0.26	0.01-0.83
No. of samples	47	48	21	49

* Study plant

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fiber concentrations were noticed for plants A, B, and C. Table 28-6 shows a summary of total airborne dust concentrations for these four plants. The study plant had an average concentration of 0.3 mg/cu m with the other plants being very similar.

TABLE 28-6

SUMMARY OF TOTAL DUST CONCENTRATIONS (mg/cu m) IN FIBER INSULATION PRODUCTION FACILITIES

	Insulation Plant			
Summary For All Operations	A	B	C	D*
Mean conc. (mg/cu m)	0.8	1.3	2.7	0.3
Range	0.1-3.8	0.3-4.8	0.2-14.5	0.1-1.0
No. of samples	44	48	21	33

* Study plant

In order to further evaluate these industrial environments in terms of respirable fiber concentrations, airborne fiber length and diameter distributions were determined. A summary of the resulting diameter and length distributions is indicated in Tables 28-7 and 28-8, respectively. The study plant had a median fiber diameter of 1.8 μ and a median fiber length of 28 μ. Again, the other three plants producing insulation products are seen to have very similar diameter and length distributions.

These data show that respirable fiber concentrations are extremely low in the study plant. It was difficult to compute a lifetime fiber exposure index for persons in the present study cohort since only limited historical dust measurements were available. In an earlier report of this plant, Wright included results of an environmental study by Kehoe done in 1963. Kehoe reported average dust concentrations of 2.24 mg/cu m with a median fiber diameter of 6 μ. Sixteen percent of the airborne fibers in the survey were less than 40 μ in length. Impinger counts averaged 0.22 mppcf (million particles per cubic feet) and less than 1% of the particles were fibrous. It thus appears that respirable fiber concentrations in the study plant were extremely low approximately 10 years prior to the present industrial hygiene survey.

Industrial hygiene surveys were also conducted in six facilities producing or making use of small diameter fibers. Table 28-9 shows the resulting airborne fiber concentrations found in these six facilities. Concentrations in these six plants are seen to be many orders of magnitude higher than those in the plant under study, with the highest single concentration being 44 fibers/ml. Tables 28-10 and 28-11 show fiber diameter and length distributions for these six plants. A large majority

TABLE 28-7

SUMMARY OF AIRBORNE FIBER DIAMETER DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN WOOL INSULATION MANUFACTURING FACILITIES (LARGE DIAMETER GLASS FIBERS)

Summary For All Operations Combined	Insulation Plant A	B	C	D*
Count median dia., μ	2.9	1.2	1.5	1.8
% $\leq$ 1.0μ	5	42	23	20
% $\leq$ 3.5μ	61	90	85	85

* Study Plant

TABLE 28-8

SUMMARY OF AIRBORNE FIBER LENGTH DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN WOOL INSULATION MANUFACTURING FACILITIES (LARGE DIAMETER GLASS FIBERS)

Summary For All Operations Combined	A	B	C	D*
Count median length, μ	52	23	39	28
% $\leq$ 5.0 μ	<2	6	3	4
% $\leq$ 50 μ	51	73	58	69

*Study plant

TABLE 28-9

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TABLE 28-9

AIRBORNE FIBER CONCENTRATIONS (Fibers/ml) IN OPERATIONS PRODUCING OR USING SMALL DIAMETER GLASS FIBERS

Operations	Plant							
	Bulk Fiber Production		Paper Manufacture		Aircraft Insulation Fabrication			
	C [a]	E	F [b]	G [b]	H	I [b]		
Bulk fiber handling								
Mean	1.0	9.7	5.8	21.9	1.2	14.1		
Range	(0.1-1.7)	(0.9-33.6)	(4.7-6.9)	(8.9-44.1)	(0.4-3.1)	(3.2-24.4)		
Number of samples	5	54	2	3	13	3		
Fabrication and finishing								
Mean	---	5.3	1.9	10.6	0.8	2.1		
Range	---	(0.3-14.3)	(1.6-2.1)	---	(0.2-4.4)	---		
Number of samples	---	24	2	1	15	1		

a) In addition to the large diameter wool insulation operations, Plant C had several lines producing small diameter fibers.

b) Only limited surveys were conducted in these facilities.

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TABLE 28-10
AVERAGE AIRBORNE FIBER DIAMETER DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN OPERATIONS PRODUCING OR USING SMALL DIAMETER GLASS FIBERS

Facilities	Percent of Fibers ≤ Upper Class Interval, μ							
	Bulk Fiber Handling				Fabrication And Finishing			
	0.5	1.0	1.9	3.8	0.5	1.0	1.9	3.8
Bulk fiber production								
Plant C	85	96	100	100	---	---	---	---
Plant E	72	88	95	98	76	93	98	100
Paper manufacture								
Plant F	40	62	75	89	55	83	94	100
Plant G	84	96	100	100	53	86	97	100
Aircraft insulation fabrication								
Plant H	48	72	92	97	46	73	89	97
Plant I	75	91	98	99	69	92	98	100

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TABLE 28-11

AVERAGE AIRBORNE FIBER LENGTH DISTRIBUTIONS AS DETERMINED BY OPTICAL MICROSCOPY IN OPERATIONS PRODUCING OR USING SMALL DIAMETER GLASS FIBERS

Facilities	Percent Of Fibers ≤ Upper Class Interval, μ							
	Bulk Fiber Handling				Fabrication And Finishing			
	5	11	22	48	5	11	22	48
Bulk fiber production								
Plant C	10	20	40	80	--	--	--	--
Plant E	23	39	54	87	23	38	32	70
Paper manufacture								
Plant F	8	27	52	73	5	22	54	86
Plant G	33	52	77	86	30	34	60	86
Aircraft insulation fabrication								
Plant H	14	31	50	94	18	37	66	91
Plant I	45	64	73	87	38	61	84	97

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of the airborne fibers are seen to be less than 0.5 μ in diameter and greater than 5 μ in length and, therefore, much more capable of deep lung penetration than airborne fibers in the study plant. Since commercial production of small diameter fibers began in the 1960's there is no facility that has been in operation sufficiently long to permit evaluation of the potential carcinogenic effects of small diameter fibers.

Case Control Study

Recent discussion at a symposium on occupational exposure to fibrous glass sponsored by NIOSH raised the possibility that a number of workers at the study plant may have been exposed to small diameter fibers. Subsequent communication with personnel of the study plant has indeed confirmed this. [1974] During the period, 1941-1949, this facility had a pilot operation that produced bulk fiber of smaller diameter than is now commercially produced. This fiber was formed by a flame attenuation process. In 1950, the flame attenuation process was moved to the production floor where fibers of larger diameter were produced than had been made in the pilot plant; however, this operation still had the potential for production of some small diameter fibers. [Personal communication, 1974] In order to evaluate the potential carcinogenicity of these smaller glass fibers among humans without waiting for an additional 10 or 20 years of prospective observation, a case-control study was deemed necessary. For each malignant respiratory disease death, a control was selected sequentially from an alphabetized list of all members of the study population matched on birth date plus or minus 6 months and on race and sex. By definition both cases and controls had been initially employed during the period, January 1, 1940 through December 31, 1949. Each of the 16 matched-pairs were distributed according to employment in the pilot plant or other than the pilot plant. Due to the uncertainty as to the fiber size after 1950, the study was limited to the pilot plant where small fibers were known to have been produced.

As seen in Table 28-12, the 16 malignant respiratory deaths exhibited a distribution of employment in the pilot plant that was different from that of the matched controls. Whereas 4 of the 16 malignant respiratory cases were exposed to this pilot plant operation, none of the controls were so exposed ($.10>p>.05$). Although these results are only of borderline significance, it is interesting to note their consistency with earlier conducted animal studies using small diameter glass fibers. [Pott and Fredrichs, 1972; Stanton and Wrench, 1972]

Summary and Conclusion

A study of mortality patterns among 1,448 workers occupationally exposed to low concentrations of large diameter glass fibers demonstrated a significant excess of non-malignant respiratory disease. This same study failed to demonstrate any excess risk of malignant respiratory disease even following 20 years since onset of exposure. However, a case-control study from among this same population did demonstrate an association of

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In view of the work of Stanton and Wrench [1972] and Pott and Friedrichs [1972], demonstrating carcinogenicity of small diameter glass fibers in laboratory animals and the possibility of human carcinogenicity posed by the present case-control study, further rapid in-depth investigation of the potential carcinogenicity of small diameter glass fibers is indicated. In the meantime, respirable exposure to small diameter glass fibers should be kept at a minimum by the use of good work practices and engineering controls.

TABLE 28-12

RESPIRATORY DISEASES AND RELATED DEATHS FOR CASES AND MATCHED CONTROLS BY EXPOSURE ASSESSMENTS

Employed In Pilot Plant	Malignant Respiratory Disease	Controls
Yes	4	0
No	12	16
	16	16

$X^2 = 3.0$, $(0.1>p>.05)$

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DISCUSSION

DR. DECOUFLE: We will now begin the discussion period.

DR. WILSON: Concerning the study just presented, I should like to ask Mr. Bayliss why no nonwhites were included in the study. Were there so few in the working group or were there other reasons?

MR. BAYLISS: Your first assumption is correct. There were very few nonwhites in the working group. Therefore, we wouldn't see very much.

DR. KUSCHNER: Mr. Bayliss, what were the five deaths from fibrosis? That's a very unusual category to appear not otherwise qualified as a cause of death.

MR. BAYLISS: What were the five deaths from fibrosis?

DR. KUSCHNER: What was the nature of the fibrotic reaction that caused the death in these patients?

MR. BAYLISS: We are trying to retrieve pathology reports on these people. We haven't received them as yet. We don't know exactly what it is that caused death. They were coded according to the diagnosis on the death certificate to a category 527 of the International Classification.

DR. DAVIS: I was very interested in Dr. Schepers' observation that macrophages can fuse to form a syncytial mass in the alveoli, and then he suggested separate again. I would like to ask if electron microscopy was used to check on the degree of initial fusion in the case of these cells.

DR. SCHEPERS: No, I didn't have an electron microscope at the time. That's why is wasn't done. But you are completely correct, it should be used.

MR. DEMENT: I have a question for Dr. Schepers. In your studies with monkeys, you stated that with your intratrachial injections you had acute reactions, but not with your inhalation studies. Could you tell us what the concentrations were in your inhalation experiments? Also, the size distribution of your fibers, and was the dust a pure fiber glass dust?

DR. SCHEPERS: The relative quantities were enormously different. The amounts injected into the lungs by intratracheal route were 500 mg each time for three times for a total of 1500 mg. This amount of silica will ordinarily kill a human being if he spends his whole lifetime in a siliceous industry. This was a gross amount of dust. The inhaled amount was at the rate of 3.4 mg/cu m of air, or 338 million particles per cubic foot of air.

The particular dust was a dust obtained from an Owens Corning Fiberglas factory where they made molded fiber glass. We obtained the dust from the factory shipped to us by the company. The dust was generated in the process of sanding, cutting, et cetera. The dust had the same characteristics as the dust that the human beings were exposed. That was the reason and the purpose of the study.

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MR. PARRILLO: This is a question to anyone on the panel. I'm mainly concerned with boat manufacturers in the state of New Jersey and I would like some information on the use of fiber glass. We have concerned ourselves these two days mainly with the manufacture of fiber glass. But these people lay it up in a polyester resin. It is hardened, dried, and hand sanded. Does anyone have any information on whether the sander still generates fibers? Are these particles of the respirable nature? What is the hazard involved in sanding of fiber glass?

Another reason I ask this question is that complaints don't usually come in about dermatitis or itchiness or any kind of reaction to polyester resin; but the men are greatly concerned with inhaling what they call glass dust. I was wondering if anyone had any information on that.

DR. KONZEN: Possibly I can shed some light on this. In our noncorrosive products operation, we manufacture reinforced plastic tanks which, in some ways, would be analogous to boat manufacturing where you drill and sand. We generally fit parts by all the same techniques that you would use in manufacturing boats. Our in-plant sampling has demonstrated airborne levels in the range of total dust concentrations of 3.49 mg/cu m of air, while the fiber concentration was 0.12 fibers per cc. The percent of fiber as opposed to total particulate was 0.25%; and of the airborne fibers, 72% of them were respirable.

I think the important point to make here is that there was only one-tenth of a milliliter of fibers in the air. This is a result of 43 samples in our noncorrosive products division operations. I believe Mr. Dement reported on similar operations and levels yesterday. This may be of help to you.

MR. CAPLAN: Dr. Schepers, I seem to recall one of your articles many years ago discussed the subject of nose irritation or throat irritation to the extent of nasal bleeding. Have you any comments on the possibility of irritation to the extent of bleeding from fiber glass?

DR. SCHEPERS: The technicians who were assigned the job of generating the dust for the purpose of dust inhalation studies were exposed to the dust. This is the way we found out that if you don't put on a face mask while you are making dust in order to experiment on a guinea pig, you can get a nose bleed from it. We presume glass particles got in there.

Answering Mr. Parrillo's question, when fiber glass is molded and sheet fiber glass is sanded or cut with a saw or in some other way abraded, glass fibers once more separate away from the polyester resin polymer and the fillers that are used in it. They can separate in the air before they go into the body. The body seems to have a capacity to dissect them again and selectively hold on to the glass fibers and some components of the polyester resin plastic dust, and to reject the other particles. How do we know that? We made chemical analyses of the lungs as the animals were exposed and found that the end composition of the lung was a silicate comparable to that of glass and very little of the other substances. The body rejected a lot of the polymer and retained the glass. When you look at these syncytial cells that I described under power phase constrast microscopy, the cells are filled predominantly with glass in far greater

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concentration than the glass that is present in the dust. So, there is a separation.

DR. HILL: If I might tease Professor Schepers, he should have had some industrial hygiene in his laboratory. I think one should certainly add the remark that in practice it would be, in England at least, quite exceptional to see nose bleeds or to have complaints of throat irritation in the ordinary manufacturing and handling of fiber glass. Would Dr. Konzen agree?

DR. KONZEN: Upper respiratory irritation is notably absent in our manufacturing facilities. I think that like any material, if you get it in a high enough concentration, you would tend to get an upper respiratory irritation which could be manifested as an epistaxis. I think we should again consider and keep in mind the airborne concentrations of fibrous glass in manufacturing facilities. In the limited studies carried out by Dr. Balzer in field installations where there was no mechanical ventilation, these were simply field installations and also involved fabrication operations in a small plant. In these operations we know of no irritation.

DR. HUTH: May I ask the experts of the panel if there is any experience of fibrous glass changing within the human organism during the course of decades after aspiration?

My second question is does fiber glass break down within the body to smaller pieces to when one could, perhaps, no longer speak of fiber?

DR. WRIGHT: I would have thought you could tell us. I think that Dr. Susan Botham touched on that today. Didn't you make some comments about the fibers breaking transversely?

DR. BOTHAM: Yes, I did. But you must remember that my work was in guinea pigs. As we already have had it pointed out today, is man a guinea pig, a rat, a rabbit? The fibers do, in fact, fragment transversely into submicron lengths as seen with asbestos fibers. This can take place in guinea pigs in a very short time, in terms of months after the inhalation and after they have been coated with a ferruginous material.

DR. BROOKS: I would like to ask a little different type of question of the panel. We have talked about carcinoma, and the possibility of fibrotic reactions. The question I would like to ask pertains to the possibility of sensitization of the individuals exposed to fibrous glass. Many times it is coated with phenol formaldehyde resins. In certain individuals, particularly patients who are atopic or patients with bronchial asthma, this may be a tremendous threat. There are some reports about patients who are exposed to fibrous glass who develop an asthmatic syndrome. There also are some reports, although not in the literature, that these individuals have become sensitized through the phenol formaldehyde resins.

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I should like to know from the panel of their thoughts about the potential hazard, not so much to the worker, but to the millions of patients who have asthma in the country, particularly the consumers.

LR. KONZEN: Well, I think there are several things that are important. One is that the phenol formaldehyde resin is put on the glass in order to protect the glass from itself. Glass is its own worst enemy. Therefore, the material is applied and it is cured to what is commonly known as an infusible, insoluble resin film. A high flash paraffinic mineral oil is also added to keep the dust down. The product without this on it and without proper cure doesn't perform very well. Therefore, Quality Control makes every effort to be sure that the pack is well cured or else they will get bleed through which, obviously, is undersirable. So, I think that there would be only a minimal likelihood that you would have uncured resin.

Mr. Smith, our Director of Quality Control, may have some other thoughts concerning that.

MR. SMITH: In order to have a product that is useful and beneficial the resins that are applied have to be cured. They do need to have some lubricant on the material to aid in handling and reduce dusting. If there are any small spots that are uncured, we would expect our selectors to get rid of those. You might have, on occasion, one in a million that was not removed.

DR. HILL: I think it could be said in amplification, and it should be, that unpolymerized resin -- in other words, uncured resin -- is a potential sensitizer. But by the time any of this glass has gone through curing and is polymerized you have reached the stage where, in fact, fully polymerized resin is not a skin sensitizer nor a pulmonary sensitizer. I think you could say, in general, that any dust can have an effect upon an asthmatic, whether the dust happens to be inert or otherwise. This quite well could be expected.

The only sort of sensitizations we see in the way of bronchospasm in workers like this are extremely exceptional. I think in 17 years I have only seen perhaps two, neither of which was scientifically provable. And one's suspicions lay very much in fact that they were the degradation products of either formaldehyde or one of the other similar substances in the manufacturing process. I would have thought that once it got as far as the consumer you are probably looking at the wrong track if you are looking at fiber glass in any specific sense for the cause of asthma. Certainly, if they are exposed to very high dust concentration at all, of course, that can precipitate asthma in an atopic individual.

DR. BROOKS: Just one comment. As I said, some of these patients that were studied have been found to be sensitized to phenol formaldehyde products. Whether it comes from the fibrous glass is something else. But I think it's a potential problem that we have not discussed so far, and perhaps it is something that should be watched out for. I think there are a lot of engineering mechanisms that should there be a problem could be changed.

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DR. UTIDJIAN: I would like to comment, if I may, on the earlier question on upper respiratory irritation by fibrous glass dust. Clark Cooper, in his draft criteria document, did find one Italian paper published in 1960 reporting a study of 13 insulation manufacturing workers who had handled plastic laminates with fibrous glass reinforcement for from 2 to 4 years. All were found to have both skin irritation and symptoms of upper respiratory tract irritation. All but one had normal chest X-rays, however, and the remaining one just had accentuation of bronchovascular markings.

There was a dust count of 64 particles per cubic centimeter, but no statement as to the size of the fibers or how many fibers were involved.

The other thing that I should like to comment on is Mr. Bayliss' paper. I note that, so far, an excess of chronic obstructive lung disease has emerged in the mortality study. I recall one very profound impression that I came away with from Newark, Ohio. I administered many of the histories myself and found extremely heavy smoking histories; heavy not only with respect to number of packs per day, but also the alleged age at which heavy smoking started. I remember at least a dozen men who assured me that they started to smoke a pack or even two packs a day before the age of 10 years. I was frankly incredulous, and therefore questioned some of the people who know this population. They said, "well, you know, a lot of these people are what they call in that area white Appalachians or poor white Appalachians." In fact they confirmed that heavy smoking does start at a very early age in that particular group.

Now I have no means of knowing as to how exceptional this is in industry. I know that smoking in U.S. in heavy industry is commonplace; but there may be some association here with the very early age of cigarette smoking.

Of course, as Mr. Bayliss has already pointed out, this may explain chronic obstructive pulmonary disease but it doesn't account for the lack of apparent excess in coronary heart disease or lung cancer.

MR. PARRILLO: Just to continue with Dr. Konzen about sampling, I have done some sampling in the boat areas that compare with the ones that you gave. I agree in plants like Johns Manville and Owens-Corning Fiberglas there are around 3 mg/cu m total dust in the type of operation of which you are speaking. However, in boat manufacturing and the sanding, with four or five men on the deck, I have measured as high as 8 mg/cu m. Perhaps this is total weight. Perhaps a fiber count should be done on such high readings that compare to what you have obtained.

The second question is, how do you feel about the mechanical irritation part? Do you think that personal hygiene and good housekeeping are sufficient to keep away this mechanical irritation?

DR. KONZEN: Number one, let's take care of the question of 8 mg/cu m of air. We, of course, run into an occasional level such as you have indicated, but we have not seen appreciable increase in fiber levels. I probably will go back and find two or three. But, in general, to satisfy yourself you could take some fiber counts. I think if our experience is

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anywhere near the run of the mill experience, the fiber count will not be over 1 fiber per cc. I would be quite surprised to see it over half a fiber per cc.

Second, I think good industrial hygiene practice would dictate that you would want to control dust, which in most cases is the cured polyester resin in boat building operations. There are effective dust collecting devices that can be placed on your sander that are not excessively expensive that would be amenable to a small boat operation such as you describe.

I have forgotten the third question you had.

MR. PARRILLO: Just about the dermatitis. I see it over and over again on the same men. I think there's pretty good housekeeping and pretty good personal hygiene, but there is no bagger on the sander like you describe.

DR. KONZEN: I think the importance here is that you may be dealing with nonfibrous glass and plastic particulate as well as fibrous glass. As you probably know, the old fashioned itch powder used to be nonfibrous glass. It was just glass powder. I think that in certain situations such as this, in addition to your industrial hygiene control, you may want to have the individual wear a long sleeved shirt, although he is going to be unhappy with this in the summertime. I think your best bet is reasonable industrial hygiene control.

MR. CAPLAN: Dr. Schepers, in your listing of orders of toxicity, or whatever that list was, I think you made some comment about a combination of fiber glass with silica dust. Are you suggesting here that they perhaps potentiate each other in fibrosis development?

DR. SCHEPERS: I do think they potentiate one another. The reason for this is an animal experiment in which this was tested. The net result was silicosis, far worse than was expected. This was not a large experiment. The other is the observation of what one sees in human beings. I have seen the lungs of quite a number of human beings who have worked in silica industries and also in glass fiber industries and also in asbestos industries. Almost invariably, when you see that they have been exposed to mixtures of dust, the summation of their effect seems to be greater than the individual separate effects that you might observe.

You will notice that I said that fiber glass, as far as I can tell, is of relatively low biological activity by itself, and there are comparable situations. For instance, if you combine beryllium oxide with silica, the toxicity of the beryllium is greatly enhanced. The beryllium oxide does not cause any significant mortality in animals exposed to it. Silica dioxide, for instance, also does not cause any great mortality at the start of any experiment, but only towards the end when silicosis begins to develop. If you put the two together, you potentiate them. You get a 100% mortality, no matter in which order you do it. I do not understand the reasons for it. I am just making the observation that it does occur.

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MR. CAPLAN: Is it possible there could be some potentiation of other things with fiber glass such as the polystyrene or the other plastics that are used in combination with the fiber glass in boat manufacturing?

DR. SCHEPERS: I don't think we have done enough work to give you a sensible answer. You know, everything is possible and you are really asking me to say what I know. I think my knowledge is kind of limited on that point. I wish somebody would study it.

MR. CAPLAN: Well, the point is that it is a very common occurrence. Fiber glass is often used in conjunction with some of the polymers. Has anyone done any work on the possible potentiation of the fiber glass with so many other polymers with which it is used?

DR. SCHEPERS: Well, I see what you mean. One big experiment is the experiment with the fiber glass polyester resin plastic, and that produces very interesting results. But I do not think that it is, by itself, more toxic than fiber glass alone or the plastic itself alone. In fact, when we injected the plastic alone, micronized it and injected into animals intravenously, they all died. It's a toxic substance when you introduce it that way. Any when you micronize the fiber glass, it's just little particles of silicate and nothing heppens to the animals. You can produce no lesion. Put the two together and you get a lesser effect. One is protective rather than additive. So you can have the result come out either way.

DR. KUSCHNER: We have ground fiber glass reinforced plastic with the design of producing particles of 1 μ in size with the grinding and elutriation and have exposed animals in a pilot experiment, hamsters and rats, at the levels of about 20 mg/cu m. The particles we saw in the EM were curious, irregularly shaped, roughly spherical particles with spikes sticking out of them. The glass sticks out of the remnants of the resin. This material in particle sizes as described simply are stored in macrophages and slowly eliminated from both these species without any other effect.

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SUMMARY OF SYMPOSIUM RESULTS

Irving Tabershaw

Introduction

DR. DECOUFLE: Any further questions or comments?

If not, I will call this part of the session to a close, and I want to thank each of the speakers for their fine presentations.

Dr. Irving Tabershaw of Tabershaw/Cooper Associates will now give a summary of the results presented here at the symposium.

Presentation

DR. TABERSHAW: I know it's incumbent on me as the last speaker to be brief and I promise to do so.

Let me first comment that I will not summarize the meeting. It would be somewhat presumptuous of me to gather all this information and capsulize it for you. At best, it could be superficial, and, in addition, it would obviously be redundant as a number of speakers before me have summarized parts of the conference. What I wish to do is to restate the problem at the beginning of this symposium, to comment on our findings or the state of our knowledge when we wrote the basic document as a contractor for NIOSH. I would like to react to some of the developments, and again comment on what are the next steps in regard to developing a standard.

The objectives of this symposium were spelled out clearly by Dr. Key and Mr. Rose. It was, first, to bring up to date the scientific knowledge applicable to the development of a criteria document by NIOSH for the promulgation of a Labor Department standard as defined by the Occupational Safety and Health Act of 1970.

A second purpose was to determine the gaps in pertinent knowledge needed to develop a proper standard and to advise on necessary research.

As a secondary objective, they both noted that this symposium was designed to provide a forum for free discussion, permitting advocacy, but without adversary confrontation; and also to test whether a symposium organized in this manner is an effective instrument for assisting in determining environmental levels, and to determine whether any modifications in this format would be helpful.

Mr. Rose pointed out that while fibrous glass is a relatively nontoxic substance in the list of 400-500 agents known or alleged to produce occupational disease in some 200,000 workers involved and the delay and difficulty in determining environmental levels and the problems of establishing suitable work practices for its control, was the reason for the selection of fiber glass as the first subject for the series of symposia planned by NIOSH.

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I might comment that the problem is generic. The reaction of this group, I believe, to the data presented may influence the future of other environmental levels, promulgation of levels, or of work practices.

It is obvious that the conference organizers directed their attention primarily at the difficulties in arriving at a suitable environmental level rather than at the efficiency of work practices, and again, this illustrates the continuing problem they face.

I might note that the present TLV -- this is the ACGIH's TLV -- is 10 mg/cu m as a time weighted average, or 30 million particles per cubic foot with glass less than 5 to 7 μ in diameter, the so-called nuisance dust level. The nuisance dust level of the OSHA interim standard is 15 mg/cu m or 50 million particles per cubic foot for total inert dust, or 5 mg/cu m for the respirable fraction.

I have a note here that the Russian standard is 3 mg/cu m, and the Bulgarian is 2 mg/cu m.

The major issue and still unanswered question regarding a suitable environmental level is whether or not fiber glass is to be considered an inert dust or has a significant carcinogenic -- and after this conference I will add fibrogenic -- potential.

I think Dr. Wright summarized pretty well that in actual practice it does not present the kind of problem that is presented by the experimental evidence; but obviously we cannot disregard the experimental data.

At the time that we wrote this document -- and "we" is really Dr. Clark Cooper, Dr. Utidjian, myself, and others--we had ample opportunity to review it with him and we knew that the eye and skin effects were not necessarily dependent upon airborne concentration. These are the only ones that had been proven. No clinical paper was presented at this meeting to indicate that there is any change in this point; and that essentially the effects, at least to skin and eye, are local and transient; that the epidemiologic evidence for chronic lung damage is negative. But this was based primarily, or almost entirely, on population exposures with less than 2 mg/cu m, and no fiber counts were available.

The question then, now, is this: is it justifiable to base a standard on experimental evidence of small fibers introduced into the pleural cavity or peritoneal cavity of animals with the hypothetical question whether these fibers can, in fact, reach the pleura or the peritoneum? This is still unanswered. I think Dr. Kuschner put it well when he pointed out that the problem was the translocation in the lung and that more research is necessary to demonstrate whether or not or how this actually occurs.

Now, as to the results of this symposium -- and this, of course, is an immediate reaction -- there obviously was no adversary confrontation here in that the symposium was conducted in good faith and with scientific responsibility. This, I might say, is quite refreshing, for those of you who have listened to some Labor Department hearings recognize that this is a plus-plus.

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If we didn't advance our knowledge, at least we reaffirmed the need for an accurate, rapid field method to determine the respirable fraction and its characterization by its geometry and number. The point was not addressed particularly, but it was obvious from Dr. Corn's work and others, that until this is done the total weight will not determine the potential hazard.

More information was given on the characterization of the fibers which produced malignancy, and I repeat, Dr. Kuschner's description of potential fibrosis in experimental animals.

No data was presented to support any environmental standard. Of course, the data presented here will have to be thoroughly reviewed, but I daresay that at the end of that time we still will not have any firm numbers. It raises the question of needed research to determine the number, if that's possible; and secondly, what whould be done in the interim, if anything?

Now, I might say that, in terms of the needed research, these again are quite obvious and were thoroughly covered by both Dr. Hill and Dr. Konzen. But it raises the question of how long will it take and how much will it cost, and at the end will you have a better answer?

The same goes for the determination of the respirable fraction in a total suspended particular mass. Obviously they both have to be done, and research must go on in these areas. These will answer some fundamental questions in industrial hygiene and in experimental pathology.

But what does one do in the interval?

One thing that struck me was that the microfibers being produced now are perhaps only 1% of the total fiber glass products produced. It was noted that fiber glass use is growing, and, therefore, the absolute exposure may be increasing more than the 1% indicates. Here, I think, is a point of control at the source and perhaps a responsibility of the industry. We are in the fortunate position here of at least anticipating that there may be a disease that is being produced occupationally. The criticism of industry and of the industrial hygienist and the occupational physician is that he is always putting out a fire, that he is not anticipating what might happen. Here we are with definite evidence that a fibrous particle can, under conditions when the geometry is correct and the site is correct, produce some adverse tissue reactions so that this perhaps is a point of control.

The next question is how to approach it with work practices, and I suspect that this will be NIOSH's next development. Work practices here again, I think, can be fairly well defined. The evidence is clear that, if you stay under 2 mg/cu m in mass, disease does not develop. I daresay that the continuing epidemiologic studies will indicate this to be true.

Dr. Bayliss' information that chronic pulmonary disease may be an end point is still to be worked out and still to be followed. But, nevertheless, it is obvious that you can control the degree and perhaps the severity -- or I should say the incidence and severity--of the disease if limits are placed on this as a "nuisance" dust, but not keep it at the level of 10 mg of 15 mg/cu m which is now accepted as nuisance dust.

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I was examining in my own mind whether some modifications might be made, and truthfully I cannot at this point say that I would have conducted the symposium any differently. I am sure that many of you in the audience will not agree with my conclusions; but please bear in mind they are simply reactions to the data presented now.

DR. DECOUFLE: Thank you, Dr. Tabershaw.

I think that brings the symposium to an end. Dr. May will have a few closing remarks.

DR. MAY: This has been a very long day, comprised of two full sessions, we are all tired, and thus I won't keep you longer than another minute.

I would simply like to say, on behalf of the National Institute for Occupational Safety and Health, that we appreciate your interest concerning occupational exposure to fibrous glass and your participation in this symposium. Special thanks to our speakers and to our session chairmen.

I would suggest that any comments, questions, and critique of the symposium be mailed to me at NIOSH, Rockville.

Again thank you all.

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APPENDIX

Tables 9-1, 9-2, 9-3

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TABLE 9-1

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy				Determined by Electron Microscopy		Comments
		length		%<3.5μ diameter length		length		
		<5μ	>5μ	>5μ	>10μ	<1μ	>1μ	
Recondition Department								
Personal: feeder end	3.3	0.02±0.02	0.04±0.03	50	50	ND	ND	
Personal	4.3	0.02±0.01	0.07±0.03	45	44	ND	ND	Sample obtained on group leader, who travels throughout department
Environmental: packing end	5.2	0.03±0.02	0.10±0.04	75	63	ND	ND	Among three packers
Environmental: packing end	1.2	0.07±0.03	0.05±0.02	21	21	ND	ND	Sample taken at location as above
Molded Pipe Plant								
Personal: cold end saws and packaging operations	3.7	0.05±0.03	0.07±0.03	33	11	ND	ND	Sample obtained on group leader, who moves throughout the department
Personal: saw operations	3.8	0.02±0.02	0.09±0.03	60	40	ND	ND	

TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample	TSPM Conc.	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy	Determined by Electron Microscopy

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								moves throughout the department
Personal: saw operations	3.8	0.02±0.02	0.09±0.03	60	40	ND	ND	

TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy, length <5μ	Phase Contrast Microscopy, length >5μ	Phase Contrast Microscopy, %<3.5μ diameter length >5μ	Phase Contrast Microscopy, %<3.5μ diameter length >10μ	Electron Microscopy length <1μ	Electron Microscopy length >1μ	Comments
Molded Pipe Plant								
Personal: cold end saws and packaging operations	2.6	0.05±0.02	0.09±0.03	43	29	1.00±0.71	ND	Group leader
Personal: press operation	4.4	0.09±0.04	0.04±0.02	29	0	ND	ND	
Warehouse								
Environmental	2.0	0.03±0.01	0.05±0.02	55	18	ND	ND	Light traffic
Environmental	1.0	0.02±0.01	0.02±0.01	50	17	ND	ND	Very light traffic
Flame Drawn Fiber Plant								
Environmental: furnace platform	1.8	0.04±0.02	0.05±0.02	55	18	ND	ND	
Environmental: furnace platform	0.07	0.02±0.01	0.02±0.01	60	0	ND	ND	

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TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Flame Drawn Fiber Plant								
Personal: roll-up man	4.1	0.07±0.04	0.11±0.04	20	20	ND	ND	
Personal: roll-up man	5.5	0.06±0.03	0.03±0.02	33	0	ND	ND	
Personal: roll-up man	2.3	0.05±0.02	0.02±0.02	33	33	ND	ND	
Personal: roll-up man	2.1	0.08±0.03	0.07±0.03	47	20	ND	ND	
Acoustic Tile Plant								
Environmental: Ross oven line near trim saws	0.8	0.04±0.02	0.07±0.03	55	18	ND	0.90±0.63	
Environmental: Ross oven line near trim saws	1.0	0.01±0.01	0.07±0.03	78	0	ND	ND	Repeat of above sample
Environmental: Ross oven line charging end	1.8	0.05±0.02	0.08±0.03	15	8	ND	ND	

TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length	%<3.5μ diameter length	Determined by Electron Microscopy: length

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TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Rigid Round Duct Section								
Personal: machine operator	2.9	0.07±0.04	0.04±0.02	17	0	ND	ND	
Personal: machine operator	2.7	0.05±0.02	0.04±0.02	22	11	ND	9.35±2.09	Repeat of above sample
Personal: finishing operator	1.3	0.05±0.03	0.04±0.02	29	0	ND	ND	
Flexible Round Duct Section								
Environmental: between two machine operations	2.6	0.11±0.04	0.07±0.03	31	8	ND	ND	
Environmental: between two machine operations	1.3	0.01±0.01	0.04±0.02	20	0	ND	ND	Repeat of above sample

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TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy				Determined by Electron Microscopy		Comments
		length		%<3.5μ diameter length		length		
		<5μ	>5μ	>5μ	>10μ	<1μ	>1μ	
Filter Factory								
Environmental: fore-hearth	4.6	0.10±0.04	0.05±0.03	33	11	ND	ND	
Environmental: fore-hearth	2.4	0.04±0.02	0.03±0.02	43	0	ND	0.43±0.43	Repeat of above sample
Personal: selector-packer	4.8	0.04±0.03	0.06±0.03	60	40	ND	ND	
Environmental: selector-packer station	0.7	0.02±0.01	0.01±0.01	33	33	ND	1.26±0.72	
Environmental: box-board line	6.0	0.18±0.06	0.15±0.06	40	27	ND	ND	Center of filter assembly line among three men working
Environmental: box board line	1.1	0.04±0.02	0.04±0.02	22	22	ND	ND	Repeat of above sample

TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy				Determined by Electron Microscopy		Comments
		length		%<3.5μ diameter length		length		
		<5μ	>5μ	>5μ	>10μ	<1μ	>1μ	

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[illegible] board line	1.1	0.04±0.02	0.04±0.02	22	22	ND	ND	Repeat of above sample

TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy, length		%<3.5μ diameter, length		Determined by Electron Microscopy, length		Comments
		<5μ	>5μ	>5μ	>10μ	<1μ	>1μ	
Bonded Mat Plant								
Environmental: hot end at operator's station	0.7	0.04±0.02	0.05±0.02	57	0	ND	ND	
Environmental: hot end at operator's station	0.7	0.01±0.01	0.02±0.02	67	33	ND	0.50±0.50	Repeat of above sample
Personal: roll-up man	1.8	0.07±0.03	0.10±0.04	58	8	1.16±0.82	1.74±1.00	
Personal: roll-up man	2.3	ND	0.06±0.03	75	0	ND	ND	Repeat of above sample
Textile Mat Section								
Environmental: forming end between two lines	1.5	0.05±0.02	0.06±0.03	36	0	ND	ND	
Environmental: forming end between two lines	1.4	0.19±0.05	0.11±0.04	23	5	ND	ND	Repeat of above sample

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TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Textile Mat Section								
Environmental: at roll-up station	3.0	0.05±0.03	0.10±0.04	45	22	ND	ND	
Environmental: at roll-up station	3.3	0.05±0.03	0.06±0.03	14	0	ND	ND	Repeat of above
Wool Plant								
Environmental:fore-hearth	2.7	0.10±0.03	0.03±0.02	18	12	ND	ND	
Environmental: fore-hearth	2.6	0.09±0.03	0.05±0.02	35	18	ND	0.36±0.36	Sample taken at same location as above
Environmental: paper pit, furnace end	2.8	0.04±0.02	0.03±0.02	38	25	ND	ND	
Environmental: paper pit, furnace end	0.9	0.01±0.01	ND	0	0	ND	ND	Sample taken at same location as above

TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of	TSPM Conc. mg/	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length	%<3.5μ diameter length	Determined by Electron Microscopy: length

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pit, furnace end						ND	ND	Sample taken at same location as above

TABLE 9-1 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT A

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy length <5μ	length >5μ	%<3.5μ diameter length >5μ	length >10μ	Determined by Electron Microscopy length <1μ	length >1μ	Comments
Wool Plant								
Personal: selector-packer	3.4	0.03±0.02	0.04±0.03	40	0	ND	ND	Making full-wall unfaced thermal insulation
Personal: selector-packer	4.9	0.09±0.04	0.14±0.05	63	8	ND	ND	Assistant group leader
Personal: selector-packer	2.3	0.05±0.02	0.08±0.03	25	19	ND	ND	Making appliance wool
Personal: selector-packer	1.8	0.02±0.01	0.01±0.03	58	33	ND	0.44±0.44	

*TSPM = Total Suspended Particulate Matter
ND = none detected

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TABLE 9-2

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Fiber Production Area								
Personal: glass furnace-fiber wind up	2.1	0.02±0.01	0.01±0.01	33	0	0.45±0.45	ND	
Personal: glass furnace-fiber wind up	1.1	0.05±0.02	0.04±0.02	46	8	ND	ND	Same man as sampled above
Personal: fiber forming-winding level	5.2	0.02±0.01	0.01±0.01	33	0	ND	ND	
Personal: fiber forming-winding level	4.7	0.03±0.02	0.02±0.01	33	17	1.06±0.61	0.35±0.35	Same man as sampled above
Personal: fiber forming-crucible level	1.1	0.03±0.02	0.04±0.02	43	14	ND	ND	
Personal: fiber forming-crucible level	1.4	0.01±0.01	ND	0	0	0.32±0.32	ND	Same man as sampled above

TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of	TSPM Conc. mg/	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length	%<3.5μ diameter	Determined by Electron Microscopy: length

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sampled above

TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy length <5μ	length >5μ	%<3.5μ diameter length >5μ	length >10μ	Determined by Electron Microscopy length <1μ	length >1μ	Comments
Cold Fiber Handling Operations								
Environmental: textiles	<0.1	0.02±0.01	0.01±0.01	20	20	ND	ND	
Environmental: rovings by creel storage between two rovings assignments	0.8	0.8±0.02	0.12±0.03	58	25	ND	ND	
Environmental: weaving room	<0.1	0.05±0.02	0.03±0.01	50	50	ND	ND	
Environmental: chopped strand	3.5	0.05±0.03	ND	0	0	ND	ND	
Environmental: chopped strand	0.8	0.02±0.01	0.05±0.02	50	25	ND	ND	Repeat of above sample

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TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy length <1μ	length >1μ	Comments
Hot Fiber Handling Operations								
Personal: chopped mat-quality control man	1.4	0.05±0.03	0.07±0.03	56	33	ND	0.53±0.53	
Personal: chopped mat-quality control man	0.5	0.04±0.02	0.08±0.03	63	25	ND	ND	Same man as sampled above
Environmental: ply mat left front of input end	0.7	0.03±0.01	0.02±0.01	37	13	ND	ND	
Personal: bonded mat-cold end: take-up rolls	1.1	0.12±0.05	0.10±0.05	45	33	ND	ND	
Environmental: bonded mat-cold end-band saw	0.6	0.03±0.01	0.02±0.01	50	19	ND	ND	

TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy length	%<3.5μ diameter length	Determined by Electron Microscopy length

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cold end: take-up rolls	1.1	0.12±0.05	0.10±0.05	45	33	ND	ND
Environmental: bonded mat-cold end-band saw	0.6	0.03±0.01	0.02±0.01	50	19	ND	ND

TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Hot Fiber Handling Operations								
Environmental: bonded mat-hot end	0.7	0.02±0.01	0.02±0.01	50	30	ND	0.19±0.19	Sample taken near bushings
Personal: helix operator	3.6	0.07±0.03	0.49±0.08	47	33	ND	ND	
Personal: helix operator	1.3	0.15±0.05	0.81±0.11	35	16	ND	Nd	Same man as sampled above
Microfibers Area								
Environmental: hot end	<0.1	0.03±0.01	0.01±0.01	11	0	ND	0.12±0.12	Sample taken near bushings
Personal: cold end, machine attendant	0.6	0.09±0.03	0.17±0.04	62	50	0.54±0.54	ND	
Personal: cold end, machine attendant	0.8	0.04±0.02	0.09±0.03	67	58	0.38±0.38	ND	Same man as sampled above

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TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Microfibers Area								
Personal: cold end, machine attendant	0.4	0.04±0.02	0.08±0.03	44	22	ND	0.57±0.57	
Chopped Mat								
Environmental: creel area	0.2	0.01±0.01	0.01±0.01	25	0	ND	ND	
Environmental: take-up rolls	0.2	0.02±0.01	ND	0	0	ND	ND	
Felts and Quartz								
Personal: Operator	0.5	0.19±0.07	1.23±0.16	82	55	ND	0.68±0.68	
Personal: Operator	1.0	0.05±0.02	0.11±0.03	65	41	ND	ND	Process down part of shift; same man as sampled above
Personal: Operator	1.3	0.12±0.05	0.76±0.12	84	64	ND	0.80±0.80	

TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample,	TSPM Conc.	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy	Determined by Electron Microscopy

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Personal: Operator	1.0	0.05±0.02	0.11±0.03	65	41	ND		0.68±0.68	
							ND		Process down part of shift; same man as sampled above
Personal: Operator	1.3	0.12±0.05	0.76±0.12	84	64	ND		0.80±0.80	

TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy			Determined by Electron Microscopy			
		length		%<3.5μ diameter length	length			
		<5μ	>5μ	>5μ	>10μ	<1μ	>1μ	Comments
Felts and Quartz								
Personal: Operator	0.9	0.05±0.02	0.46±0.06	79	66	ND	ND	Same man as sampled above
Filter Tube Production								
Environmental: between end trim and grinder-groover	0.7	0.08±0.02	0.09±0.02	52	24	ND	ND	
Environmental: hot end, near bushings	0.6	0.06±0.02	0.07±0.02	50	50	ND	ND	
Personal: socking station operator	3.4	1.34±0.23	3.16±0.36	62	39	ND	1.47±1.04	
Personal: socking station operator	2.1	0.81±0.14	1.88±0.21	66	33	ND	ND	Loose material observed inside holder; same man as sampled above

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TABLE 9-2 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT B

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Filter Tube Finishings								
Personal: saw operator	1.5	0.08±0.04	0.26±0.06	41	14	ND	ND	
Personal: saw operator	0.9	1.12±0.16	0.32±0.09	21	20	ND	ND	Same man as sampled above
Bonded Mat Area								
Environmental: hot end, between two heads	0.2	0.02±0.01	0.02±0.01	40	20	ND	ND	Process operating intermitently; preliminary runs on a new product
Environmental: cold end, take-up rolls	0.8	0.01±0.01	0.01±0.01	56	11	ND	0.14±0.14	

*TSPM = Total Suspended Particulate Matter
ND = none detected

TABLE 9-3

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations,	TSPM	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy	Determined by Electron Microscopy

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*TSPM = Total Suspended Particulate Matter
ND = none detected

TABLE 9-3

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy, length <5μ	Phase Contrast Microscopy, length >5μ	Phase Contrast Microscopy, %<3.5μ diameter length >5μ	Phase Contrast Microscopy, %<3.5μ diameter length >10μ	Electron Microscopy, length <1μ	Electron Microscopy, length >1μ	Comments
Mat Cutting Section								
Environmental: near mat cutting table	4.6	0.17±0.06	0.06±0.03	25	8	ND	ND	All samples obtained at this operation were taken at same location
Environmental: near mat cutting table	1.9	0.03±0.02	0.15±0.04	86	0	ND	ND	
(above sample ashed)		0.08±0.03	0.03±0.02	25	11			
Environmental: near mat cutting table	3.5	0.07±0.03	0.03±0.02	25	12	ND	ND	
Large Preform Area								
Environmental: preform operator station	1.6	0.10±0.04	0.10±0.04	50	0	ND	ND	
Environmental: preform operator station	0.8	0.12±0.04	0.03±0.02	18	18	ND	ND	

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TABLE 9-3 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy length <1μ	length >1μ	Comments
Large Preform Area								
Environmental: preform operator station	1.5	0.12±0.04	0.04±0.02	17	8	8.9±2.6	ND	
Environmental: preform operation station	1.1	0.05±0.03	0.11±0.04	58	33	ND	ND	
Small Preform Area								
Environmental: preform operator station	5.4	0.09±0.04	0.09±0.04	40	0	ND	0.89±0.89	
Environmental: preform operator station	2.3	0.03±0.02	0.09±0.04	63	37	ND	ND	
(above sample ashed)		0.12±0.04	0.10±0.04	46	23			
Environmental: preform operator station	0.3	0.06±0.03	0.12±0.04	69	31	ND	ND	
(above sample ashed)		0.06±0.03	0.14±0.04	70	29			

TABLE 9-3 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations, Type of Sample,	TSPM Conc.	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy length	%<3.5μ diameter length	Determined by Electron Microscopy length

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[illegible] operator station		[illegible]	0.12±0.04	69	31	ND	ND
(above sample ashed)		0.06±0.03	0.14±0.04	70	29		

TABLE 9-3 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Small Preform Area								
Environmental: preform operator station	1.8	0.05±0.02	0.02±0.02	33	17	ND	ND	
Panel Department								
Environmental	0.5	0.01±0.01	0.04±0.02	50	50	ND	ND	
Environmental	3.3	0.02±0.02	0.12±0.04	75	47	0.63±0.63	ND	
(above sample ashed)		0.15±0.05	0.06±0.03	29	14			
Environmental	2.8	0.03±0.02	0.01±0.01	33	0	ND	ND	
Environmental	3.2	0.09±0.04	0.07±0.03	42	25	ND	0.61±0.61	
Environmental	4.3	0.04±0.02	0.07±0.03	50	0	ND	ND	
Environmental	2.0	0.07±0.03	0.03±0.02	29	0	ND	ND	
(above sample ashed)		0.10±0.03	0.04±0.02	27	0			

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TABLE 9-3 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Panel Department								
Environmental	0.2	0.01±0.01	ND	0	0	ND	0.61±0.61	
Environmental	4.5	0.10±0.04	0.04±0.02	27	0	ND	0.61±0.61	
Custom Molding Department								
Environmental	3.2	0.04±0.02	0.09±0.03	27	30	ND	1.86±1.07	
(above sample ashed)		0.12±0.04	0.05±0.03	27	18			
Environmental	3.9	0.07±0.03	0.05±0.02	40	20	ND	0.67±0.67	Between sanding station and paramatic drilling station
Environmental	2.4	0.09±0.04	0.09±0.04	43	7	ND	ND	Wet belt sander station
Personal: operator, finishing area	2.4	0.11±0.04	0.04±0.02	17	8	ND	ND	Milling machine with local exhaust

TABLE 9-3 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	%<3.5μ diameter length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments

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finishing area — 0.0420.02 — 17 — 8 — ND — ND — Milling machine with local exhaust

TABLE 9-3 (CONTINUED)

RESULTS OF SAMPLING FOR TOTAL SUSPENDED PARTICULATE MATTER AND AIRBORNE FIBERS IN PLANT C

Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy				Determined by Electron Microscopy			
		%<3.5μ diameter length				length			
		<5μ	>5μ	>5μ	>10μ	<1μ	>1μ	Comments	
Custom Molding Department									
Personal: trimmer	4.7	0.08±0.03	0.05±0.03	30	10	ND	ND		
Personal: trimmer	6.8	0.04±0.02	0.12±0.04	50	0	ND	ND	Filing and using wire grinder	
(above sample ashed)		0.07±0.03	0.07±0.03	30	0				
Personal: operator-trimmer	2.6	0.06±0.03	0.06±0.03	50	20	ND	ND	Presses, files and drills	
(above sample ashed)		0.09±0.04	0.08±0.03	45	18				
Environmental	0.7	ND	0.10±0.03	100	38	1.20±0.92	0.65±0.65	Between operator and two trimmers	
(above sample ashed)		0.06±0.03	0.09±0.04	60	10				
Personal: operator-trimmer	3.2	0.10±0.04	0.09±0.03	33	7	ND	ND	Presses, files and drills	

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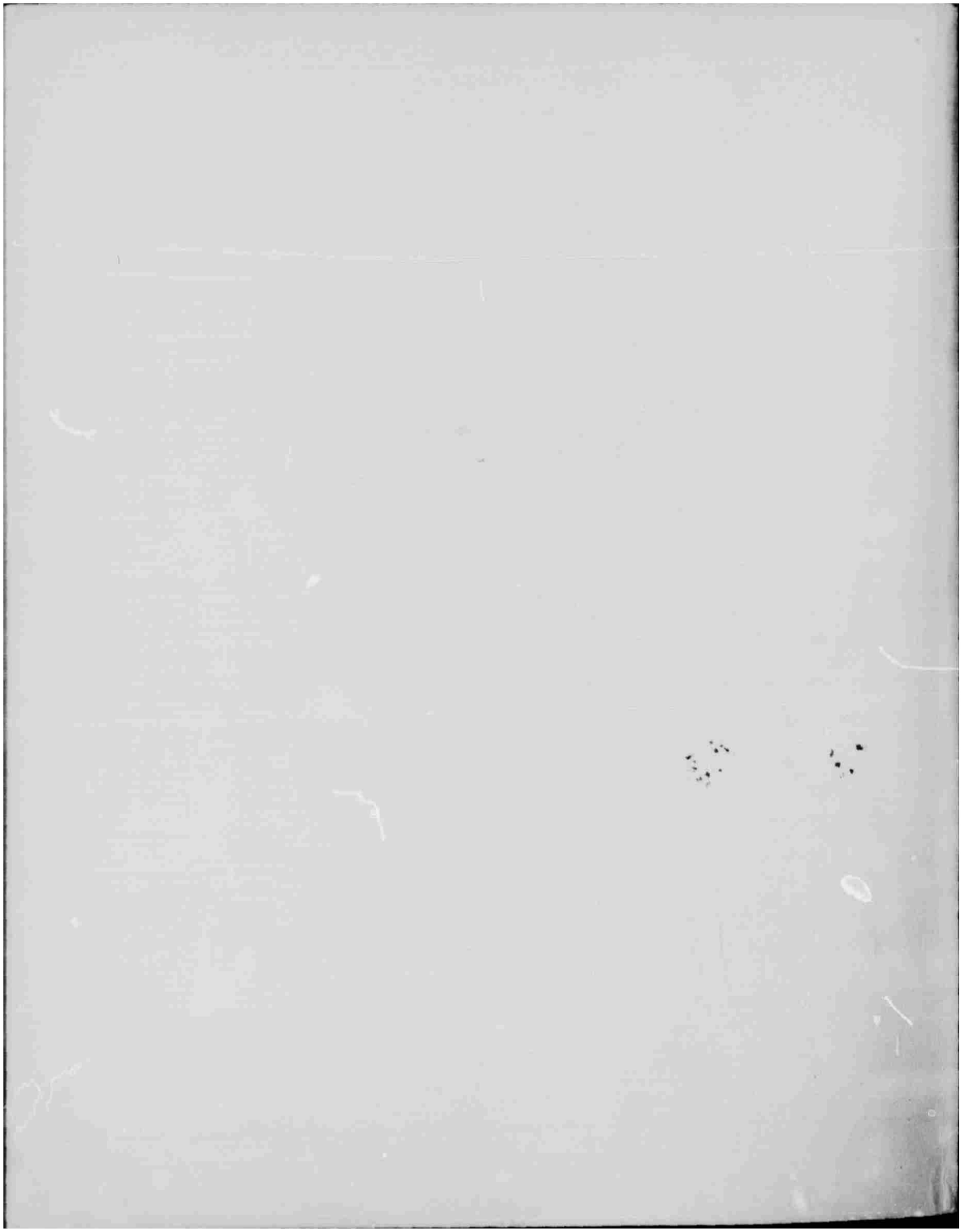
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Manufacturing Operations, Type of Sample, and Description of Sampling Site	TSPM Conc. mg/cu m	Fiber Concentrations (No./ml) Determined by Phase Contrast Microscopy: length <5μ	length >5μ	%<3.5μ diameter length >5μ	length >10μ	Determined by Electron Microscopy: length <1μ	length >1μ	Comments
Custom Molding Department								
Environmental	0.3	0.08±0.03	0.06±0.0	42	0	ND	ND	Between sanding and parametic drilling stations
Environmental: finishing operator	3.8	0.13±0.04	0.13±0.04	50	15	ND	ND	Near paramatic drilling rig
Environmental	1.9	0.09±0.03	0.08±0.03	40	20	ND	ND	Above drill jig
Environmental	1.5	0.07±0.03	0.08±0.03	55	9	ND	ND	Station at which operator trims premix to size and weight

*TSPM = Total Suspended Particulate Matter
ND = none detected

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