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AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

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10 October 1968

MEMBERS OF AIHA-ACGIH RESPIRATOR COMMITTEE:

Recently, Dr. Irving J. Selikoff of the Mount Sinai School of Medicine gave a talk concerning the asbestos health problem to the New England Section of AIHA. Inasmuch as the asbestos health problem is a respiratory health problem, I thought that you would be interested in what Dr. Selikoff had to say. I am enclosing a copy of notes which I took during Dr. Selikoff's talk and which later were typed.

Very truly yours,

W. H. Revoir

W. H. Revoir, Chairman
Respirator Committee
AIHA-ACGIH

WHR/m
Enc.

cc - E. D. Palmes, Ph.D.
Mr. V. E. Rose

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NOTES WITH REGARD TO ASBESTOS HEALTH PROBLEM GIVEN BY DR. IRVING J. SELIKOFF OF MOUNT SINAI SCHOOL OF MEDICINE OF CITY UNIVERSITY OF NEW YORK TO NEW ENGLAND SECTION OF AMERICAN INDUSTRIAL HYGIENE ASSOCIATION ON 19 SEPTEMBER 1968 AT FRAMINGHAM, MASSACHUSETTS

You may be interested to know that Dr. H. Hyman of the Harvard School of Public Health in Boston, Massachusetts, initiated the investigation of the asbestos health problem a few years ago.

Industrial medicine did not recognize the asbestos health problem as important previously. Why? The disease asbestosis was first reported in 1924. The asbestosis victim also had TB. The first case of lung scarring caused by asbestos without TB being present was reported in 1927. A case of lung cancer caused by asbestos was first reported in 1935. The victim also had asbestosis.

Why were cases like these not reported before? Disease caused by asbestos has a long latent period from time of exposure to the onset of the effects of the disease. The disease rarely occurs in less than 20 years from time of exposure. Not many people worked with asbestos until the 1930's. Therefore, it is only recently that sufficient medical data could be obtained that asbestos is a health problem.

Even pneumoconiosis has a long latent period from time of exposure to onset of effects. A long period of latency complicates the medical problem.

In the mid 1960's there were 632 asbestos workers in local unions of insulation workers in the New York City area. We have studied these men for 20 years. Statistics indicate that during this period 203 of these men should have died had their experiences been the same as the general population. Instead, 255 of these men actually died during the 20-year period. There were excess deaths among these men due to cancer. It was expected that 36 would have died of cancer, but 95 actually died of cancer instead. It was thought that 6 should have died specifically of cancer of the lung and pleura, but 45 died of this type of cancer. Also, deaths of these men due to cancer of the stomach, colon, and rectum were excessive. Whereas it would be expected that 9 would die of cancer of the stomach, colon, and rectum, actually 29 died.

Therefore, industrial exposure to asbestos results in theliomas.

During the last 6 to 7 years, three additional observations made pertaining to the asbestos health problem are important.

(1) Pleural calcification is common in a part of Finland where asbestos is mined. The incidence of pleural calcification in people in other parts of Finland is 1 case per 1,000 people. In the mentioned area we would have expected 47 cases of pleural calcification among the population but 94 cases were actually found.

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Pleural calcification is common among asbestos workers. With an exposure of 20 years to asbestos, 30% to 50% of these workers develop pleural calcification. Pleural calcification is a characteristic of exposure to asbestos. Pleural calcification does not cause much bodily harm.

(2) When asbestos fibers are inhaled and deposited in the lungs, there is a reaction. Asbestos bodies are formed. An asbestos body is a foreign body in the lungs composed of an iron-containing protein surrounding an asbestos fiber. It is known now that such bodies may form in the lungs due to other fibers also. There are billions of asbestos bodies in the lungs of an asbestos worker.

500 consecutive autopsies of people who died in hospitals in Capetown, South Africa, showed that almost half of the people had asbestos bodies in the lungs although these people had no occupational history of exposure to asbestos. A similar prevalence of asbestos bodies among hospital deaths is found in all parts of the world. Autopsies indicate that about 25% of the population have asbestos bodies in their lungs. Most of the asbestos bodies have asbestos fibers as their base. Thus, inhalation of asbestos fibers is widespread.

(3) In recent years a rare type of tumor was first noted in South Africa. During 1956-1960, autopsies resulted in the finding of 47 cases of mesotheliomas in people who had at one time or another lived in an asbestos mining area. In 45 of these cases, a definite contact with asbestos was determined although in some cases the contact was extremely brief and meant an exposure to only a very small amount of asbestos.

In the New York area from 1963-1968, 13 of 113 deaths of asbestos workers have been due to mesothelioma. This means that about 1/10 of the asbestos workers in this area are dieing of mesothelioma. who died

Newhouse and Thompson report 76 cases of mesotheliomas at the London Hospital, most of which have been observed in recent years. 31 people of the 76 people having mesotheliomas had worked with asbestos. 9 of the 76 people had been exposed to asbestos only because they lived in a household with an asbestos worker. 11 of the 76 people merely lived within 1/2 mile of an asbestos factory.

Lieben of Pennsylvania has reported 42 cases of mesotheliomas from 1958-1963. 10 of the people had definite industrial exposure to asbestos. 3 of the people had lived in households with asbestos workers. 8 of the people lived or worked within 1/2 mile of an asbestos factory. 10 of the people had a questionable contact with asbestos. 11 of the people either had no contact with asbestos or information pertaining to asbestos contact was missing.

- The data cited indicates that cases of mesothelioma are rising. Also, the data indicates that a small dose or exposure to a low concentration of asbestos can lead to a serious disease - mesothelioma.

Exposures to asbestos 25 to 35 years ago are important.

In 1942, two girls from Wellsley College worked in a Boston shipyard during the summer for 6 weeks applying asbestos insulation in ships. One of these girls died recently of mesothelioma. The other girl has calcified plaques in the pleura.

Recent information indicates that there is a dose-response relationship for cancers due to asbestos. This means that if appropriate industrial hygiene practices can be applied to operations involving the use of asbestos, then we can reduce the risk of death due to cancer caused by asbestos.

Animal tests indicates that if the asbestos concentration in air is low, the number of mesotheliomas in exposed animals is less than for animals exposed to high concentrations of asbestos in air.

In an area in Finland surrounding an asbestos plant no lung cancers have been found among people living in the vicinity of the plant although 1/3 of the deaths of workers in the asbestos plant are due to lung cancer. The people outside the plant in this case who were only lightly exposed to asbestos did not develop lung cancers. This indicates that sound industrial hygiene practices, which significantly reduce the amount of asbestos in the air of the work areas, should solve the asbestos health problem.

A report from Dresden, Germany, shows that the use of practices that result in decreasing the amount of airborne asbestos in work areas of an asbestos factory results in decreasing the number of deaths due to cancer.

In an area in Bulgaria, many deaths due to lung cancer are reported for both workers in an asbestos mine and people living in the area but not working in the mine. The soil in this area contains much asbestos. The area is devoted to farming and thus tilling of the soil releases asbestos to the air where it can be inhaled by people living in the area.

708 people of a total of 1,500 people in an asbestos plant have asbestos bodies. Thus, 47.2% of these people have asbestos bodies. More blue collar workers than white collar workers in the plant have asbestos bodies.

All workers in a shipyard where asbestos insulation is applied to ships being constructed were found to have asbestos bodies.

Many construction workers have asbestos bodies since asbestos is used as insulation in the construction of buildings.

Industrial hygiene work involving the development of engineering controls for limiting the concentration of airborne asbestos in work areas should be concentrated on the shipyards and on building construction.

Exposure of workers in shipyards and in building construction to airborne asbestos fibers involves many, many people. People in these trades who work side by side with people using asbestos also are exposed to asbestos. It is estimated that 5 million people in building construction and 350,000 people in shipyards are exposed to airborne asbestos.

In the United Kingdom, workers handling asbestos are supposed to wear air-supplied helmets for respiratory protection.

There is a long latent period from time of exposure to asbestos to development of cancer. This latent period is greater than 20 years.

In 1910, only 30,000 tons of asbestos was used annually in the world. In 1930, the annual use of asbestos had increased to 500,000 tons. Over 4½ million tons of asbestos were used in the world in 1967. Thus, we can expect that in 20 to 30 years the number of cases of mesotheliomas and other types of cancer will be vastly greater than at present.

What about a TLV for airborne asbestos?

As yet, we do not know the relationship between concentration of airborne asbestos and disease caused by airborne asbestos.

Two actions many years apart are important - the exposure to asbestos and the onset of disease due to asbestos.

We must rely more on applying sound industrial hygiene practice on the jobs where asbestos is handled than on relying upon a TLV for airborne asbestos.

We do not know the relationship between the size of airborne asbestos fibers and disease caused by these fibers. We do know that small asbestos fibers, invisible to the naked eye and even invisible with the aid of an optical microscope, are much more numerous than asbestos fibers visible to the naked eye and visible with the use of an optical microscope.

The current TLV for asbestos is based upon an excellent study made in the 1930's in a textile plant. At that time, few of the plant workers had been exposed to asbestos fibers for longer than 20 years. Also, there was not much disease in the textile plant.

The current TLV for asbestos fibers in air of 5×10^6 fibers per cubic foot of air is temporary. NOTE: in 1969 will be 2 mppcf.

We need much more information pertaining to the relationship of concentration of airborne asbestos fibers to diseases caused by asbestos fibers before we can establish a reliable TLV for airborne asbestos fibers.

What about the toxicity of airborne glass fibers that are used in insulation materials?

Insulation materials containing glass fibers were first used in the early 1930's. It was not until 1945 that large quantities of insulation materials containing glass fibers were used in the construction industry. There has been insufficient time of exposure of large numbers of workers to make a good study of the toxicity of airborne glass fibers. Some animal tests indicate airborne glass fibers are not toxic like airborne asbestos fibers.

Asbestos has properties requiring its use in many jobs and we cannot stop using it.

What are the health problems associated with airborne asbestos?

Pleural calcification caused by airborne asbestos does not result in a health problem.

Asbestosis is due to exposure to very high concentrations of airborne asbestos. We do not get as many cases of asbestosis as we did many years ago because we now employ better engineering controls to limit airborne asbestos concentrations which we did not do many years ago.

Mesothelioma is due to exposure to low concentrations of asbestos. There is a long latent period from time of exposure to the onset of the disease of mesothelioma - this period is greater than 25 years.

There is a wide range of neoplasms caused by inhalation of airborne asbestos - lung cancer, mesothelioma, stomach cancer, cancer of the colon, cancer of the mouth.

Mesothelioma is a pleural fibrosis that may attain a thickness of one centimeter or more and the consistency of shoe leather. The lungs become inelastic and this results in a respiratory insufficiency which causes death. The time period that elapses from clinical detection of mesothelioma to death is only one year.

There is no difference between the different types of asbestos with regard to causing neoplasms. Different types of asbestos fibers may result in different periods of latency from time of exposure to onset of the neoplasm but they all will cause neoplasms.

Crocidolite asbestos at first was reported to be the only type of asbestos causing neoplasms. Evidence now indicates that the other types of asbestos cause neoplasms also.

Crocidolite asbestos was not used in the United States until 1930. From 1930 to 1935, only 500 tons of crocidolite asbestos was imported into the United States. Many large asbestos firms in the United States never used crocidolite asbestos yet workers in these plants have died from mesotheliomas. Therefore, crocidolite asbestos is not the cause of mesotheliomas in the United States.

Asbestos bodies are found in the lungs of people exposed to asbestos for the first time 2 months previous to the detection of asbestos bodies.

The presence of asbestos bodies in the lungs only signifies an exposure to asbestos - the presence of asbestos bodies in the lungs does not necessarily mean that the person has a disease due to asbestos.

All people who have died from mesothelioma were found to have had asbestos bodies in their lungs. Only 25% of the normal population has asbestos bodies in their lungs.

We have no data concerning mesotheliomas in youngsters.

Smoking cigarettes has a very serious effect upon the incidence of lung cancer of asbestos workers. A study of 370 asbestos workers living in 1963 showed that 87 did not smoke cigarettes while 283 did. Since 1963, none of the 87 non-smokers have died of lung cancer while 27 of the smokers have died of lung cancer. The expected death rate of smokers expected was only 2.8.

The combination of smoking cigarettes and exposure to airborne asbestos is extremely bad from the standpoint of ~~possible~~ frequency of death due to lung cancer.

The high death rate due to lung cancer of people who smoke cigarettes and who are exposed to asbestos fibers indicates that there is a multiple factor in the cause of lung cancer. Certainly! Surface adsorption into asbestos

It is concluded that workers exposed to asbestos fibers should not smoke.

W. H. Revoir
9/29/68

NOTES TAKEN AT FIBROUS DUST SEMINAR AT MELLON INSTITUTE, PITTSBURGH, PENNSYLVANIA ON 22 NOVEMBER 1968 SPONSORED BY INDUSTRIAL HYGIENE FOUNDATION AND MELLON INSTITUTE

Part I - FIBROUS GLASS HEALTH RESEARCH

Opening Remarks by W. Clark Cooper, M.D., School of Public Health, University of California

Airborne glass fibers are not known to be a medical hazard at this time. There is a suspicion that airborne glass fibers may be a medical hazard. More time may be needed to study the airborne glass fiber problem to permit a decision to be made whether or not these fibers are hazardous.

Statistical Studies of Health of Fibrous Glass Workers by Industrial Hygiene Foundation - H. Michael D. Utidjian, M.D., Department of Epidemiology, University Of Pittsburgh

The Industrial Hygiene Foundation made a statistical study of the health of employees of a glass fiber manufacturing plant operated by the Owens-Corning Fiberglas Corporation at Newark, Ohio. The plant has been in operation for 30 years. More than 2,000 workers are employed at the plant. The study was made in the period from August through October 1968.

Employees were classified with regard to degree of exposure to airborne glass fibers. The exposure classifications used are - high, intermediate, low. Also, the employees were classified by age. The age classifications used are - under 30 years, 30 to 49 years, 50 years and over. A sample of 232 employees was used in the study.

Employees were subjected to answering a questionnaire with regard to health. Employees also were subjected to spirometry tests to determine the breathing capacity of their lungs. In addition, chest X-rays of the employees were studied.

The purpose of the questionnaire was to determine gross effects of exposure to airborne glass fibers on the health of employees of the glass fiber manufacturing plant. A study of the questionnaires indicates that there is no clear relationship between the degree of exposure to airborne glass fibers and the health of persons exposed. Only age seemed to have an effect upon the health of the employees. Coughing - this increases with age but there is no increase due to high exposures to airborne glass fibers. Bronchitis - there is no clear pattern of relationship between bronchitis and degree of exposure to airborne glass fibers and there is no relationship between bronchitis and age. Breathlessness - results are the same as for bronchitis. Chest illness in last 3 years - results are the same as for bronchitis. Total history of chest illness during life - there is a slight increase in chest illness with age.

The results of the spirometry tests indicate that there is a slight decrease in the breathing capacity with age but there is no relationship between breathing capacity and degree of exposure to airborne asbestos fibers. Smokers were found to have a greater decrease in breathing capacity than non-smokers.

The study of the chest X-rays showed that there was nothing unusual in the X-rays to suggest a radiologic lung pattern that could be associated with exposure to airborne glass fibers.

It is conlcuded that there is no definite relationship between the health of a worker and his degree of exposure to airborne glass fibers. The study showed that health deterioration increased only with age.

A question was asked if any workers in the plant wore dust respirators. Dr. C. S. Bishop, physician at the plant who was present, stated that only workers in a batch house used dust respirators.

Statistical Studies of Health of Fibrous Glass Workers by National Insulation Manufacturers Association - L. J. Cralley, Ph.D. of U. S. Public Health Service

A study is now being made of the health profile of glass insulation workers and glass textile workers. The health records of 20 to 25 thousand workers are being studied. A substantial number of these workers have had long periods of exposure to airborne glass fibers. In addition, a study is being made of the concentrations of airborne glass fibers for various operations in the glass insulation and glass textile fields.

Workers are exposed to much lower concentrations of airborne glass fibers than concentrations of airborne asbestos fibers. Glass fibers generally are much larger in size than asbestos fibers. Glass insulation employs glass fibers having diameters 6 microns and greater. Glass fibers used in glass textiles are smaller than glass fibers used in glass insulation. Recently, micro-fine glass fibers having diameters less than 1 micron have been introduced for use in the manufacture of glass textiles.

Pathological Studies of Respirable Fibrous Glass Dust with and without Phenol-Formaldehyde and Textile Binders - Paul Gross, M.D., Industrial Hygiene Foundation

Finely ground flake glass of respirable size deposited in the lungs of animals was found to behave like inert dust particles.

In the study, rats and hamsters were exposed to uncoated glass fibers, glass fibers coated with phenol-formaldehyde resin, and glass fibers coated with a textile binder (starch). Thirty rats and thirty hamsters were used in each phase of the study.

Glass batt material was used to generate the aerosol used in the study. The glass utilized had a diameter of 1 micron. The batts were cut into pieces and ball milled. The material from the ball mill was in the form of a finely divided powder. 60% of the product was fibrous while 40% was non-fibrous. The fibrous material was chiefly 5 to 10 micron in length. The material from the ball mill was fed continuously by means of a screw type feeder into an air stream which passed through a blower having a squirrel cage type impeller and then into the animal inhalation exposure chamber. The concentration airborne matter in the animal inhalation exposure chamber was 100 milligrams of matter per cubic meter of air. The animals were exposed to the airborne glass matter for 6 hours per day, 5 days per week, for a period of 1 year.

A gross examination of the lungs of animals killed after 1 year exposure to the airborne glass matter showed that the lungs were normal.

Microscopic examination of lung tissue of the animals exposed to the airborne glass matter for 1 year indicates that none of the 3 types of glass (uncoated, coated with phenol-formaldehyde resin, coated with textile binder) had any distinguishing effect upon the lungs. An increase in connective tissue in the lungs usually is due to irritation - there was no increase in the connective tissue in the lungs of the exposed animals. The glass matter was found to have penetrated through the alveoli walls. Air spaces in the lungs were not affected by the glass matter. Collections of glass were found in the lymph nodes in the lungs and in cells of lung tissue - this collected glass seemed to have caused no effects in the lungs. Reactions in the lungs of the exposed animals were similar to that of inert soot or coal dust.

It is concluded that exposure to airborne glass matter results in reactions similar to that of inert dust. Coatings on glass fibers of phenol-formaldehyde resin or textile binder (starch) do not alter the benign characteristics of glass.

Examination of the lungs of animals exposed to the airborne glass matter for a 2-year period showed that neoplasms were not formed.

A study of the effects on animal lung tissue of an aluminum silicate fiber made by the Carborundum Corporation showed that this fiber produces lung effects like those of the glass fiber.

It is known that animals exposed to airborne asbestos fibers for less than 6 months develop a fibrosis in the lungs. Some people have said that this is due to the fibrous nature of asbestos. Since animals exposed to airborne glass fibers do not develop a fibrosis in the lungs, we must conclude that the fibrous shape is not a significant factor in causing adverse physiological effects in the lungs.

Physiological Studies of Health of Fibrous Glass Workers - Benjamin J. Lambiotte, M.D., Industrial Hygiene Foundation

Pneumoconiosis is a diagnosable disease of the lungs produced by dust.

Let us propose the following hypothesis: Inhalation of airborne glass fibers will cause significant effects on lung tissue.

The hypothesis would be proven to be true if it is found that long term workers exposed to high concentrations of airborne glass fibers have significant deterioration of their lung functions. The hypothesis would be proven to be untrue if it is found that long term workers exposed to high concentrations of airborne glass fibers do not have significant deterioration of their lung functions.

The Industrial Hygiene Foundation is making a study of the effects of inhalation of airborne glass fibers on lung tissue to prove or disprove the mentioned hypothesis. Effects of inhalation of airborne glass fibers on the mechanical function of the lungs is to be determined by spirometry tests on workers who have been exposed to airborne glass fibers. The effects of inhalation of airborne glass fibers on the membrane walls

of the alveoli of the lungs will be determined by measuring the diffusion of carbon monoxide gas through the alveoli membrane walls of workers who have been exposed to airborne glass fibers. The study involves both older workers who have been exposed to high concentrations of airborne glass fibers for many years and younger workers who have been exposed to low concentrations of airborne glass fibers for only a few years. The results of this study will be available for use by the Threshold Limits Committee of the American Conference of Governmental Industrial Hygienists in 1969.

Radiological Studies of Health of Fibrous Glass Workers - Jon L. Konzen, M.D Owens-Corning Fiberglas Corporation

The chest X-rays of the sample of workers in the glass fiber manufacturing plant used in the study reported today by Dr. H. Michael D. Utidjian were reviewed to determine if the lungs of these workers showed micronodulation or a diffuse fibrosis pattern.

The chest X-rays showed that 33 of the 232 workers had a minimal of micronodulation in their lungs. It could be explained that this minimal micronodulation was due to matters other than that of glass fibers.

Micronodulation in Lungs

Number of Cases Found

Age of Workers Exposed to Glass Fibers	Workers with Low Exposures to Glass Fibers	Workers with Intermediate Exposure to Glass Fibers	Workers with High Exposures to Glass Fibers
Under 30 years	3	2	6
30-49 years	4	3	4
50 & more years	4	4	3

The table pertaining to micronodulation in the lungs of workers exposed to airborne glass fibers shows no pattern of relationship of micronodulation to either degree of exposure or to age.

The chest X-rays did not show any cases of a diffuse fibrosis pattern in the lungs of workers exposed to airborne glass fibers.

Expert radiologists stated that all the chest X-rays of the workers, including those with the minimal of micronodulation in the lungs, are normal for the general population. Even those showing the minimal of micronodulation are normal for such micronodulation is found in normal chest X-rays of the general population.

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Summary of Portion of Seminar Pertaining to Fibrous Glass Health Research by W. Clark Cooper, M.D., School of Public Health, University of California

The serious effects of asbestos fibers on the health of humans undoubtedly may result in an increase in the use of glass fibers.

Many people are asking questions about the effects of inhalation of airborne glass fibers on the health of people exposed to these glass fibers.

Studies carried out up until the present of the possible effects of inhalation of airborne glass fibers on the health of people indicate that glass fibers do not have adverse effects on the health of people.

Perhaps we have not had a chance to study a sufficient number of people exposed to airborne glass fibers for long periods of time to make a decision on the effects of glass fibers on the health of people exposed to them.

Almost all reports on the effects of airborne glass fibers on the health of people are negative - that is, these reports indicate that glass fibers do not have any adverse effects upon the health of people exposed to them. Many medical research people find it difficult to accept these negative findings.

We must realize that airborne glass fibers vary widely in size. We should examine the lungs of people exposed to airborne glass fibers to determine the size of glass fibers deposited in the lungs.

Undoubtedly, we will hear more about the possible effects of airborne glass fibers on the health of people exposed to them in the years to come.

Industry must continue to monitor the work environment with regard to airborne glass fibers and industry must continue to monitor the health of workers exposed to airborne glass fibers.

PART II - FIBROUS ASBESTOS HEALTH RESEARCH

Opening Remarks by Lewis J. Cralley, Ph.D., U. S. Public Health Service

The asbestos fiber industry is much older than the glass fiber industry. Asbestos differs from glass chemically. Even different types of asbestos differ chemically. In addition, oils and metals are attached to asbestos fibers during processing of these fibers. The exposure parameters of concentration and fiber size differ for airborne asbestos fibers and airborne glass fibers. We wish to learn how to use a very valuable material, asbestos, safely.

Asbestos Exposure in the U. S. Textile Industry from 1930 to Date - Joseph L. Goodman, M.D., Raybestos-Manhattan, Ind.

Chrysotile is the type of asbestos chiefly used in the United States. The history of the use of fibrous asbestos in the United States is about 60 years in time.

During the 1920's, asbestos textile plants used only very crude methods to eliminate asbestos dust. An exhaust opening in the ceiling of the building generally was the means used to liminate asbestos dust. Asbestos textile plants were extremely dusty during the 1920's.

During the 1930's and 1940's, asbestos textile plants used floor exhaust systems to eliminate asbestos dust. The floor exhausts were connected by ducts to sack type cloth filters located in a dust room or dust house. Workers often had to crawl through the ducts using brushes to clean the ducts.

Currently, local exhaust hoods and vents are connected by ducts to Wheelabrator type tubular-shaped cloth bag filters located in a dust room or dust house. The tubular-shaped cloth filters are suspended with open ends attached to an inlet manifold either at top or bottom of the filter housing. A lower manifold also serves as a receiving hopper for the dust. As air enters the filter unit, it impinges on a baffle plate causing larger dust particles to fall into the hopper. The air then passes through the tubular-shaped cloth filters and the dust particles are deposited on the inner surfaces of the cloth. Since the accumulation of deposited dust on the cloth surfaces gradually increases the resistance offered by the cloth to air flow, it is necessary to vibrate the tubular-shaped filters at intervals to detach deposited dust particles. A shaking device is used for this purpose. Local exhaust hoods are used on carding machines. Overhead exhaust vents and hoods are used on weaving machines. Also, aprons in front of weaving machines are used to direct airborne asbestos dust particles to the exhaust vents and hoods.

Many people in an asbestos textile plant wear dust respirators.

Wet weaving is being tried as a means of reducing the amount of asbestos dust. Also, spraying of water sometimes is used to wet airborne asbestos dust particles - the wetted asbestos dust particles fall to the floor.

Measurement of asbestos dust concentrations in most asbestos textile plants show that these concentrations are below the present TLV for airborne asbestos.

The records indicate that the highest concentrations of asbestos dust occurred during the 1930's and that the largest numbers of deaths of asbestos textile workers due to asbestosis and lung cancer occurred during the 1950's.

The present trend in the asbestos textile field is a decrease in the number of deaths due to asbestosis and lung cancer with a decrease in the concentration of asbestos dust in the work area atmosphere.

<u>Statistical Studies of U. S. Asbestos Textile Workers - Philip E. Enterline, Ph.D., University of Pittsburgh</u>

A statistical study was made of men who were working in asbestos textile plants during the 1950's. The study involves a determination of what are the causes of death of these men.

A review of asbestos dust concentrations show that plants producing asbestos textiles have higher asbestos dust concentrations than do plants producing asbestos friction materials and that the asbestos-friction materials plants have higher asbestos dust concentrations than do plants producing asbestos building products. The incidence of lung cancer and asbestosis is found to be the greatest for workers in asbestos textile plants, next for workers in asbestos friction materials, and least for plants producing asbestos building products.

The death rate due to lung cancer increases with the time period from the initial exposure to asbestos dust. The death rate due to asbestosis does not increase as rapidly with time from the initial exposure to asbestos dust as that due to lung cancer.

The study indicates that if a person is exposed to a high concentration of airborne asbestos fibers for a short period of time, the risk of death due to lung cancer remains but the risk of death due to asbestosis does not remain.

Studies of Pathogenicity of Synthetic and Natural Asbestos and Brake Drum Dust - Paul Gross, M.D., Industrial Hygiene Foundation

Animals exposed to airborne synthetic chrysotile asbestos fibers do not develop adverse reactions in the lungs.

An investigation was made of the effects of exposure of animals to airborne synthetic chrysotile asbestos fibers containing various additives. The additives used were compounds of certain metals. The results of the investigation were negative - that is, the animals did not develop adverse physiological effects. These results do not agree with those of investigations of the effects of exposure of animals to airborne natural chrysotile asbestos fibers. The difference in the results of the exposures of animals to airborne synthetic chrysotile asbestos and to airborne natural chrysotile asbestos may be due to the fact that natural chrysotile asbestos contains insoluble metal additives while the synthetic chrysotile contained soluble metal additives.

Automotive brake linings contain asbestos fibers bonded together by plastic materials. There is a large amount of dust from brake linings in the urban atmosphere. The heat used in injecting asbestos fibers into the plastic materials of brake linings and the heat developed when brake linings are used results in causing the asbestos fibers to lose water of hydration and thus this asbestos differs chemically from the original asbestos. The asbestos which has lost the water of hydration is amorphous while the original asbestos was crystalline.

Rats and hamsters were exposed to dust from brake linings. A considerable amount of dust was found in the lungs of the animals. A microscopic examination of the lungs of these animals was made and adverse physiological effects were difficult to find.

The results for the exposure of animals to dust from automotive brake linings indicates that this dust produces reactions in the lungs which are similar to that produced by a biological inert dust.

Methodology Employed in Pathological Studies of Ferruginous Bodies - H. Michael D. Utidjian, M.D., University of Pittsburgh

A ferruginous body has a central core or shaft of fibrous form with a coating, often segmented or beaded in appearance, which is probably composed of ferritin or a ferritin-like protein material. Ferruginous bodies are found in the lungs of most people studied in autopsies. The fibrous central core may be an asbestos fiber or other types of fibers such as glass, natural textile fibers, or synthetic fibers. Also, the fibrous central core may be an elongated or fibrous particle of various minerals. A ferruginous body having an asbestos fiber as the central core is called an asbestos body.

Longitudinal lung sections were digested using domestic laundry bleach containing sodium hypochlorite. A gray sediment from the bleach solution is dissolved in a mixture of chloroform and ethanol and shaken with clean water in a separatory funnel. The lower layer of liquid in the separatory funnel contains the inorganic mineral residue from the digested lung tissue plus any ferruginous bodies that may have been in the lungs. The lower layer of liquid in the separatory funnel is run into an elongated glass tube, clean water is added, and the liquids are shaken. The material is then poured into a centrifuge tube and spun in a centrifuge. A dense material is deposited in the apex of the centrifuge tube. The liquid material is decanted from the centrifuge tube and discarded. The deposit in the apex of the centrifuge tube is removed from the tube and suspended in a few drops of distilled water to which is added a few drops of dimethyl sulphoxide as a preservative. This material is used to make smears on microscope slides for examination of ferruginous bodies.

Instrumentation in Pathological Studies of Ferruginous Bodies - Martin N. Haller, Carnegie Mellon University

The electron microscope can be used to obtain electron diffraction patterns of mineral fibers. Different types of mineral fibers will result in different electron diffraction patterns.

Since different types of asbestos fibers have different mineral compositions there may be a different type of electron diffraction pattern for each type of asbestos fiber.

Unfortunately, the mineral composition of a specific type of fibrous asbestos varies depending upon the source of the asbestos. The minerals present in a specific type of asbestos have quantity ranges. Thus, different electron diffraction patterns may be obtained for asbestos fibers of the same type from different sources.

Electron diffraction patterns of asbestos fibers may not be specific enough to use electron diffraction methods of analysis to determine the types asbestos fibers that may be present in ferruginous bodies.

Basic Considerations in Pathological Studies of Ferruginous Bodies - John M.G. Davis, Ph.D., Department of Pathology, University of Cambridge, England

Electron microscope studies of ferruginous bodies indicate that ferruginous bodies are formed in the lungs by fibers being engulfed by cells. They are formed in giant cells.

Examination of ferruginous bodies using an electron microscope shows that the coatings of the fibrous core consists of granular materials.

Study of the coatings of the fibrous core of ferruginous bodies using an electron microscope shows that most ferruginous bodies contain a single layer coating while some have a multitude of layers in the coating.

An examination of the outside surface of the coating of a ferruginous body with the aid of an electron microscope shows the presence of fine filaments.

Ferruginous bodies shorter in length than 5 microns are not present in the lungs. Fibers less than 5 microns are taken up by macrophages in the lungs.

It is obvious that ferruginous bodies are an exception. Less than 1% of the fibers that are deposited in the lungs are cotaed to become ferruginous bodies.

Some fibers engulfed by giant cells get a coating to become ferruginous bodies but other fibers engulfed by giant cells do not get a coating and thus do not become ferruginous bodies.

Examination by the electron microscope shows that many ferruginous bodies, but not all, present in giant cells contain a membrane separating them from the cytoplasm of the giant cells.

Many fibers deposited in the lungs become coated with calcium. Whole areas in the lungs may become filled with calcified material.

The environment of a fiber deposited in the lungs is a most important factor in determining the type of coating the fiber will get if any.

It is thought that when a ferruginous body is formed, the fiber first becomes coated with acid polysaccharide and then colloidal iron attaches itself by impregnation of the acid polysaccharide coating.

We would like to learn more about the special chemical environment that is necessary to permit a small percentage of fibers deposited in the lungs to become ferruginous bodies.

Discussion of Papers Pertaining to Pathological Studies of Ferruginous Bodies - Paul Gross, M.D., Industrial Hygiene Foundation

We desire evidence that the presence of ferruginous bodies in the lungs are not associated with diseases of the lungs. There is scanty evidence today that there is not any definite association between the presence of ferruginous bodies in the lungs and diseases of the lungs.

Inasmuch as most of the asbestos used today is chrysotile, airborne asbestos fibers present in urban atmospheres should be chiefly chrysotile asbestos fibers. There is some evidence that the central cores of most ferruginous bodies may not be chrysotile asbestos fibers.

Summary of Portion of Seminar Pertaining to Fibrous Asbestos Health Research - Lewis J. Cralley, U. S. Public Health Service

There has been a great improvement in reducing the concentrations of airborne asbestos fibers in the atmosphere in asbestos textile plants and it is indicated that this is resulting in a reduction of deaths of asbestos textile workers caused by asbestosis and lung cancer. Much still remains to be done.

The results of animal studies indicate that asbestos dust from automotive brake linings may not produce adverse physiological effects in the lungs.

Ferruginous bodies are found to be present in the lungs of the majority of people. Much excellent work has been done with regard to identifying the central cores that make up the ferruginous bodies. However, much still remains to be done in making identifications of the central cores of ferruginous bodies. An excellent start has been made in determining how ferruginous bodies are formed in the lungs. There is an urgent need for more work to be done to determine the significance of ferruginous bodies in the lungs with regard to pathogenic effects, actual or potential.