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EFFECTS OF INHALATION OF MINERAL WOOL DUST ON RATS

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AMERICAN ROCK WOOL CORPORATION

TORRANCE, CALIF.

CS024 0534

EFFECTS OF INHALATION OF MINERAL WOOL DUST & FIBERS

By

William P. Helm

June 3, 1947

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CS024 0538

TABLE OF CONTENTS

	Page
Introduction	1
The Problem	3
Definitions of Pneumoconiosis, Fibrosis, Silicosis and Asbestosis	3
Physiology of the Inhalation of Dusts	5
Action of Silica Dust on the Lungs	5
Particle Size	6
Safe Dust Concentrations in Air	7
Permissible Asbestos Dust Concentrations	7
Free Silica vs. Silicates	8
Experiment At The Laboratory of Physiology at Harvard	9
Statement by Dr. R. R. Sayers	10
"Hazards of Exposure to Glass Wool"	10
Mechanical Irritation To The Lungs of Workers Using Glass Wool	11
Asthma In Glass Wool	11
Fiber Glass Not Cause of Pneumoconiosis	11
Rock Wool Not Cause of Pneumoconiosis	12
Results of X-Ray Analysis of the Lungs of Mineral Wool Workers	13
Statement by Mr. F. A. Van Atta	13
Summary	15
Conclusions	16

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CS024 0539

INTRODUCTION

Mineral Wool is an inert, fireproof insulating material consisting of very fine, interlaced fibers giving the appearance of loose wool or cotton. Mineral wools consist primarily of silicates of calcium, aluminum, and magnesium with varying texture and size of fiber depending upon chemical compositions and manufacturing procedure used. Mineral wools may be reclassified as: 1/

Rock Wool, made from natural rock with or without mineral additives.

Slag Wool, made from iron, copper, or lead blast furnace slags alone or mixed with fluxing materials.

Glass Wool, made from silica sand, soda ash, and limestone with or without scrap glass or other materials.

In addition there is another similar type of wool that is classified as a mineral wool but is made from waste rock materials from manufacturer's processes and not from a natural rock.

The fundamental principle of the manufacture of the fibers is to impart a high velocity to droplets of the materials in the molten state, and the combination of the drag of the air, surface tension and viscosity of the molten minerals draws the droplets into fibers.

The principle use for mineral wools (with the exclusion of glass wools) is for insulation of ceilings and wall spaces of homes. However, mineral wools have been entering the industrial field with increasing popularity for insulation in stoves, refrigerators, industrial ovens, refrigerated trucks, as a soundproofing material in radio studios etc., and many other industrial applications.

The dusty nature of mineral wools caused many to complain of irritation to the nose and throat when handling the material. Also cases of dermatitis were frequent among workers in contact with the material or in an atmosphere containing the dust from mineral wools. These complaints led to investigations into the possibility of such mineral wool dusts promoting silicosis or pneumoconiosis.

1/ Bureau Mines Information Circular 6988

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In a letter to the author Mr. L. Erskine, Asst. Industrial Hygienest, Engineer, U. S. Department of Labor, states:

> "The Division has been interested in the glass and rock wool industries on the part of labor. Dermatitis, and a suspicion that the sharp dusts liberated in manufacturing and handling operations may be injurious to the lungs, has called for comment in this country and abroad."

It is the intent of this report to present the comments, findings, and experimental results of various agencies on the affects of breathing mineral wool dusts. in the hopes that recent reports might show complete enough data to reach a definite conclusion as to the health hazard of mineral wool dust.

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CS024 0541

EFFECTS OF INHALATION OF MINERAL WOOL DUST & FIBERS

The Problem

Most mineral wool products when fiberized are coated with a thin film of light mineral oil. This oil serves very well to reduce the amount of dust and "fly wool" encountered in the manufacture, handling, and installation of mineral wool. Refinements in manufacturing procedure such as adequate ventilation and exhaust systems serve also to reduce the amount of dust in the air in the manufacturing plants. However, in instances of installation such as in attic spaces and in some parts of manufacturing plants where ventilation is inadequate there are still slight amounts of dust present even with oiled products. In the case of the use of an unoiled wool for some uses this dust concentration in the air is quite high and is very objectionable causing irritation of the mucous membranes of the nose and throat as well as a violent itching and scratching sensation to the skin.

The siliceous nature of the dust coupled with the recent wide spread investigations into occupational lung diseases of workers in the dusty trades has led to tests to determine if the dust from mineral wools presented a health hazard from the standpoint of pneumoconioses or lung diseases.

Definitions of Pneumoconiosis, Fibrosis, Silicosis, & Asbestosis

Before presenting any data it might be well to define for purposes of explanation the following terms: Pneumoconiosis, Fibrosis, Silicosis, and Asbestosis.

Pneumoconiosis: A strict definition of pneumoconiosis refers specifically to the various changes in the lungs caused by the inhalation of dust. The generally accepted interpretation of the word involves the concept merely of "dusted lung". The kind of dust inhaled determines the type of injury noted in the lungs.

Fibrosis: Fibrosis is the pathological process which underlies lung disease caused by dust. This consists of a fibrosis or a replacement of the elastic tissue of the lungs by an impervious scar tissue.

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CS024 0542

Silicosis: Undoubtedly the most important kind of lung disease falling under the general classification of pneumoconioses is silicosis. A definition of silicosis is as follows: 2/

> "Silicosis is a chronic disease of the lungs resulting from prolonged inhalation of fine particulate silica."

The International Silicosis Conference in 1930 defined Silicosis as a "pathological condition of the lungs due to inhalation of free silica (SiO_2).

Asbestosis: Asbestosis is defined and discussed in the National Safety Council Health Practice Pamphlet #4 as follows:

> "Asbestos is the only silicate at present recognized as causing pathology, and even its reaction is definitely different from that due to silica alone (Drinker). Its behavior is the exception to the rule that silicates are inert, at least in uninfected tissues. Asbestos, when inhaled, produces fibrous tissues in the lungs of both men and animals. Asbestos is made up of hydrated silicates of magnesium with variable amounts of iron, calcium, sodium, potassium, and aluminum replacing portions of the magnesium. The property common to all of this group is the fibrous structure. It has been shown that the presence of fibers of asbestos are necessary for the production of asbestosis and this fact, couples with the observation that fine asbestos dust is analogous in its inert reaction on the lungs to that of other silicate dusts leads to the conclusion that asbestosis is the result of physical irritation of the lung tissue and not of chemical action as in the case of silicosis."

The above discussion links mineral wool to asbestos on two counts, i.e., both are of a fibrous nature and both are silicates, though mineral wool contains mostly only aluminum, calcium, and magnesium silicates.

2/ National Safety Council Health Practices Pamphlet #4, 1939.

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CSG24 0543

Physiology of the Inhalation of Dusts

Respiratory System:

The body has a well developed protective mechanism against dust particles which are inhaled. The nostrils contain fine hairs which, together with the moisture of the nose and throat, catch great numbers of dust particles. The larynx, with its moist lining and delicate coughing mechanism, helps to trap and reject many particles. Many of the dust particles which pass on into the trachea (wind pipe) and bronchi (branches) are caught by the moisture, and are whipped toward the mouth by the cilia (microscopic hairs which line these tubes). In this way much of the dust which enters the breathing apparatus does not remain there, but is moved toward and out of the mouth by the normal body protective mechanism.

Action of Silica Dust on the Lungs:

Silicosis is produced by the inhalation of only the very minute silica particles. These particles of dust to reach the lung must be five microns 2/ in diameter or less. 3/ Any silica particles larger than this probably cannot reach the lung aveoli or air cells.

In earlier years it was believed that the physiological action of quartz on the lung was caused by the hardness of the material, but tests using silicon carbide, aluminum oxide, and diamond dusts, all harder than silica, had practically negative physiological effects when inhaled.

The theory of action of silica on the lungs as discussed in the National Safety Council Health Practices Bulletin #4 is:

> "Where dust is inhaled and is passed through the upper respiratory tract, it reaches the terminal air sacs of the lungs where the exchange of gases between the blood and air takes place. At this point, as the dust is

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2/ National Safety Council Health Practices Pamphlet #4 states: "With the exception of such fibrous materials as asbestos, the dust particles must be smaller than 10 microns in their longest dimension to reach the inner recesses of the lung where the damage is caused."

3/ Occupational Health Pamphlet Number Three (NS) State of California Department of Public Health.

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CS024,0544

a foreign matter, it is ingested by what are known as "scavenger" cells which carry it through the walls of the air sacs to the lymph drainage canals located outside the air sacs. This results in a rapid increase of tissue cells which narrows the lymphatic channel, hampering elimination, as it is along these canals that foreign substances are removed. When silica is being removed the particle may kill the scavenger cell by which it is being transported, with the result that all dust particles are not removed from the lung. Instead, they may remain along the course of the drainage canal. Apparently, these small particles of silica dust are toxic in this location and the tissue cells in the immediate vicinity are altered and replaced by what is known as scar or fibrous tissue. The ultimate result is larger areas of tissue or lung volumes in which capillary action is definitely reduced-through which a normal exchange of gases between the blood and inhaled air. This produces a shortness of breath in the affected individual in proportion to the amount of scar tissue in his lungs."

Particle Size

The particles inhaled into the respiratory system must be small enough to float in air or be carried by air currents. Research has shown 2/ that dust particles must be smaller than 3 microns in diameter to promote silicosis.

The naked eye can discern objects 50 microns in diameter and sometimes particles 20 microns can be seen if they are of a shiny nature. It is obvious then that the dusts of size small enough to reach the lungs (3-10 microns) are essentially invisible to the naked eye.

It has been shown that in silicotic lungs dust particles under three microns greatly outnumbered those of larger dimensions; that 70 percent of particles in silicotic lungs were found to be less than 1 micron and that the largest particles did not exceed 10.5 microns; that the most representative particle size in the lungs of men who have died of silicosis (complications of tuberculosis, bronchitis, asthma, etc.) is one micron.

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C5024 0545

Safe Dust Concentrations in Air

The particle size distribution in the air is of little value in attempting to control pneumoconioses since all atmospheric dusts contain a majority of particles small enough to penetrate into the lungs. The logical standard for control of dust in atmosphere to eliminate pneumoconioses is to determine concentrations or amount of dust in air. In the case of dust the concentrations to be for those particles 10 microns or smaller which size range is that which will reach the lungs.

The following maximum permissible safe limits were determined by the United States Public Health Service by extensive tests, and were obtained from a representative of the Dept. of Industrial Safety, State of California.

More than 50% free silica in dust - 5 million particles

From 10-50% free silica in dust 10 million particles

Less than 10% free silica in dust 50 million particles

Number of particles refers to number per cubic foot of air. Concentrations based on an 8 hour working day; size less than 10 microns.

Permissible Asbestos Dust Concentrations

As previously stated (see asbestosis) asbestos when inhaled, produces fibrous tissues in the lungs. Fine asbestos dust particles have been shown to produce only the inert reaction on the lung characteristic of other silicate dusts. 2/ The fibrosis caused by breathing asbestos, therefore, has been contributed not to the asbestos dust, but to the fact that the asbestos fibers are the cause of a physical irritation resulting in asbestosis.

The United States Public Health Service made a study of the asbestos textile industry and they recommended that the dust concentrations be kept at less than 5 million particles per cubic foot of air if asbestosis is to be avoided.

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CS024 0516

No standard 2/ has been set up to determine the safe number of asbestos fibers allowable in air, but by keeping the fine dust at less than suggested standards, the concentration of injurious fibers will be in the safe range.

Free Silica vs. Silicates

Mineral wool is composed mostly of aluminum, calcium, and magnesium silicates as is the mineral wool dust. Silicosis has been attributed definitely to silica particles in the lung, and with the exception of asbestos dust, the silicate dusts have not been shown to cause a disabling lung condition such as is produced by free silica (SiO_2). Experiments on animals breathing silicate dusts and in some cases the dust was injected into their peritoneal tissues have shown that silicate dusts are inert and do not cause the characteristic nodulation of the lungs produced by free silica. 2/ In the examination of the lungs of men exposed to silicate dusts containing no free silica the nodular appearance of the X-Ray and actual nodules in the lungs, both of which are characteristic of silicosis, have not been found when the exposure was limited to silicates. In many cases of silicosis supposedly contributable to silicates it has been found that there was an unsuspected presence of free silica in the dust.

On the other hand, it has been reported 2/ that high concentrations of clean, washed mica (150 million particles per cubic foot) have promoted pneumoconiosis, and 14.5% of 221 tremolite talc miners showed fibrosis associated with a chronic cough. The talc workers were of 10 or more years exposure and the reported dust concentrations were as high as 1300 million particles per cubic foot of air.

It may be possible than that some silicate dusts promote a disabling lung condition, and before any definite conclusions are drawn, studies of the affects silicate dusts should be made.

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SUMMARY OF FINDINGS AND CASE HISTORIES ON THE EFFECTS OF ROCK WOOL AND GLASS WOOL DUST ON THE LUNGS

Experiment at the Laboratory of Physiology, Harvard.

One of the first recorded experimental works on rock wool and its physiological effects on the lungs was performed at the Laboratory of Physiology, Harvard School of Public Health.4/ This experiment was performed in 1935.

The increasing popularity of rock wool insulation prompted the experiment to determine if the siliceous nature of dust from rock wool made it injurious to the lungs.

The experiment was performed on three groups of cats breathing air under the following conditions:

Group 1 - Air containing any fumes that could be evolved by passing air over rock wool at room temperature.

Group 2 - Air passed over rock wool so heated that the resultant air temperature was 67 to 82° C.

Group 3 - In addition to same exposure given to Group 1 this group was further exposed for 1 hour daily to a dusty atmosphere of very finely ground rock wool.

All these groups received the exposure indicated for two months continually.

The atmospheres of Groups 1 & 2 were so controlled to determine if the occupants of a house using rock wool insulation would be subjected to any toxic effect of fumes or vapors evolving from rock wool. The results of the analysis of these cats showed no significant changes in the lungs, liver and kidneys.

Three out of the eight cats subjected to dusting (Group 3) showed a varying number of small cellular lesions in the lungs in which silicate particles could be detected. Except for the presence of large amounts of silicate particles retained in smaller air passages of the lung tissues, bronchi, bronchioles, and alveolar ducts the remaining five cats showed no abnormalities of the lungs. The absence of wide spread inflammatory changes in the lungs of all these animals where silicate particles were in intimate contact with tissues suggests that no highly irritating substance was present.

4/ "Rock Wool in Relation to Health", Journal of Industrial Hygiene, Vol. 17, No. 6, November, 1935, Fairhall, Webster, and Bennett.

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CS024 0518

The conclusions drawn by this experiment are: 4/

> "The chemical data thus obtained entirely deny the existence of any hygienic hazard in connection with contact with commercial rock wool of the type investigated. The histological examination of lungs, kidneys, and liver revealed no constant or significant pathological change. In the dusted cats there was evidence of inhalation of considerable quantities of silicate particles and accompanying changes in the lungs were those one might expect such foreign matter to produce."

This article then does not definitely say that in the case of the three cats (Group 3) that the foreign body reactions (cellular lesions) noted in their lungs were of a serious nature. The exposures were not long enough to definitely state whether or not these lesions would eventually develop a fibrosis.

Statement by Dr. R. R. Sayers, Chief, Division of Industrial Hygiene, United States Public Health Service, 1939

Dr. Sayers stated to J. R. Thoenen, United States Department of Interior: 1/

> "From some experiments we have performed, using mineral wool fibers, we have found that particles remain in the tissue, producing an inert foreign body reaction similar to that of asbestos, and we have been considering dust from mineral wool as potentially harmful, that is, capable of producing a diffuse pulmonary fibrosis, when inhaled.
>
> "We have been considering dusts that produce an inert reaction less harmful than pure silica and more dangerous than dusts which are absorbed from the tissues, such as limestone."

Mr. Thoenen adds: "The results of these experiments indicate there is a definite danger from inhaled mineral wool dust, but its action is mechanical rather than pathological."

"Hazards of Exposure to Glass Wool"

In an article "Hazards of Exposure to Glass Wool", Journal of American Medical Association, May 1, 1943, the following statement is made:

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"Although Schulz (Gasmaske 11:37, 1939) mentions an asbestosis-like condition of the lung of a man working with glass wool, it may be doubted that any specific pneumoconiosis is likely to arise."

Mechanical Irritation To The Lungs of Workers Using Glass Wool

The Journal of American Medical Association, September 1945, published an article from the Archives des Maladies Professionnelles, Paris, Vol. 6, 1944-1945, in which it was stated that manifestations of mechanical irritation of the lungs of workers using glass wool were observed. Neither clinical nor roentgenologic observation disclosed pulmonary changes, but the glass wool had been used by these workers for only six months; and even if danger of silicosis existed, several years would have to elapse for lung changes to be perceptible by X-Ray.

Asthma and Glass Wool

Occupational Medicine, May 1946, presents a case history from Archives des Maladies Professionnelles, 6:392, 1944-1946. A woman aged 44, had worked for four years in a shop which produced fireproof clothing. During the first year asbestos was used for this clothing, but after 1942 glass wool was used. The woman suffered from attacks of asthma provoked by deep inspiration. Respiration was retarded, inspiration required great effort, and her face was red after severe coughing. Tests showed no tuberculosis. X-Rays showed micro-nodular images on the lung.

The author, S. Tara, states: "This case does not permit generalizing conclusions, but it does raise several questions. Was the glass wool the cause of the asthma? It appeared after two years work with the glass wool, and asthmatic attacks diminished after contact with the glass wool ceased. Then there is the question whether the conditions observed by the X-Ray were caused by glass wool or perhaps by asbestos dust to which the woman had been exposed for a year. However, as a rule asbestosis does not produce micronodular images so rapidly."

"Fiberglass Not Cause of Pneumoconiosis"

The Journal of the American Medical Association, June 16, 1945, published an article entitled, "Fiberglass Not Cause of Pneumoconiosis". This article is in reply to a question asked by a physican concerned over the cases of persistent coughs (7 months) with ship yard workers working in compartments where glass wool insulation is being applied. Some of these men showed physical signs of bronchitis, others did not. X-Ray films

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CS024 0550

were normal except for a number of Ghon shadows on the lungs of the man with the most persistent cough.

The article states:

> "Unpublished animal experiments at the Saranac Laboratory have demonstrated that "Fiberglas" is not inhalable in appreciable quantities. After exposures of 3 years with the maximum atmospheric concentrations that could be maintained, the lungs of guinea pigs and white rats contained practically no fiber, and no scar tissue developed. The fiber used for insulation varies from 3-10 microns in diameter, which is too coarse to penetrate the lungs. A specially prepared fiber, 3 microns or less in diameter, was also excluded from the lungs, apparently because it carries an electrostatic charge which causes it to adhere and mat on surfaces with which it comes in contact. Annual X-Ray examinations of employees manufacturing this product have revealed no signs of pulmonary reaction. While Fiberglas is irritating to the skin and upper respiratory membranes it does not cause pneumoconiosis."

"Rock Wool Not The Cause of Pneumoconiosis"

In reply to a question from a doctor asking for the diagnosis, prognosis, and treatment of a condition observed in several of his patients apparently caused by the inhalation of rock wool dusts and other fine particles while installing rock wool insulation in ships for refrigeration insulation, the Journal of the American Medical Association, May 19, 1945, published the following article entitled, "Rock Wool Not The Cause of Pneumoconiosis."

> "Information on the chronic pathologic effects of inhaled rock wool is practically non-existant. Fibers of glass wool are not inhalable, and it is assumed that rock wool is likewise excluded from the lungs because of the size and physical properties of the fiber. X-Ray films of persons exposed to this material tend to confirm the inference, for none of those observed in any way resemble the reaction to the known fibrous dust asbestos. If there is any change noted at all it is limited to an exaggeration

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CS024 0551

of the normal linear markings due to particles and broken fragments of the fiber; such damages are rarely associated with symptoms or disability. Diagnosis would depend on a history of exposure over periods measured in years, the demonstration of a uniformly altered X-Ray pattern in a considerable group of workmen and a physical examination for evidence of disability. The prognosis should be good, and no disability or increased susceptability to pulmonary infection is likely. Treatment is entirely prophylactic."

Results of X-Ray Analysis of the Lungs of Mineral Wool Workers

Eighty-four workers exposed to mineral wool for periods ranging from 7 to 29 years showed no X-Ray evidence of silicosis or fibrosis 5/ of the lungs. This includes 10 years of exposure to slag wool. The exaggerated linear findings exhibited were shown to be a characteristic of that community for the age group under study. The duration of exposure for the sample group under study was long enough to produce any pulmonary changes if rock wool were capable of producing a fibrosis. The free silica content of finished rock wool was found to be a trace to 0.5%.

Statement By Mr. F. A. Van Atta, Industrial Department National Safety Council

In a letter to the author, Mr. F. A. Van Atta, Industrial Department, National Safety Council states:

"Rock wool specifically is not very easily inhaled. When it is inhaled it produces a foreign body reaction in the lungs and may produce a diffuse pneumonitis according to some work by the Division of Industrial Hygiene of the United States Public Health Service. This result was obtained on experimental animals and I have not seen reports of actual pneumonitis or other systemic effects in men actually employed in the industry. I have

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5/ "Negative Chest Findings in a Mineral Wool Industry", Carpenter and Spalyar, Industrial Hygiene Digest, February, 1946.

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CS024 0552

seen records of X-Ray examinations of men employed for a number of years in areas where rock wool batts were cut and packed without anything more significant than some exaggeration of the linear marking of the lungs. This should not, however, be taken as an indication that the possibility does not exist but perhaps as an indication that the hazard is relatively mild."

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SUMMARY

It can be seen by the preceding discussions that there are many and contradicting opinions on the hazards of mineral wool dust and fiber as obtained from experiments and personal observations. However, in the cases where the opinion was that a pulmonary condition similar to asbestosis existed due to mineral or glass wool dust and fibers several important facts were not ascertained. With reference to the article "Asthma and Glass Wool" (the only case found where definite pathology of lung was present) these facts are:

1 - Was there present in the atmosphere any free silica?

2 - Is there a possibility that this remote case of pathology was due to some disease (excluding Tuberculosis) other than a fibrosis caused by glass wool?

3 - Since the patient had been exposed to asbestos dust and fibers which has been definitely proven to cause a fibrosis of the lungs, would it be possible that the inhalation of glass wool dusts aggravated the already diseased lung causing the asthmatic cough?

4 - Was the concentration of dust and fibers unusually high?

With the exception of the above case, all other cases of a pathology of the lung from breathing mineral wool dust were obtained on animals in experimental work (see page 10) in which case dust and fiber concentrations may have been higher than those encountered in practice. It is felt that the concentrations of mineral wool dust and fibers may have more effect on the production of an asbestosis condition whereas in the case of silicosis it is not so much the concentrations but the chemical nature of the dust which produces the fibrosis.

Although Thoonen and Sayers (see page 10) state that there is a definite danger due to inhaled mineral wool dust, recent experiments (see "Fiberglas Not Cause of Pneumoconiosis" and "Rock Wool Not Cause of Pneumoconiosis") have shown that the dust and fibers of the size encountered are not in themselves inhalable.

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CS024, 0554

This conclusion was reached after the examination of the lungs of animals exposed to glass wool dust and fibers showed the presence of no scar tissue and practically no fibers. This fact, that mineral wool dust and fibers are not inhalable in quantities sufficiently large enough to cause asbestosis or fibrosis, has been borne out by the lack of a case of pathology of the lung definitely proven to be due to inhalation of such dusts. Furthermore, results of X-Ray analysis of workers exposed to mineral wool dust (see "Results of X-Ray Analysis of the Lungs of Mineral Wool Workers") showed no evidence of silicosis or fibrosis after exposures ranging from 7 to 29 years.

CONCLUSIONS

1 - The chemical nature (silicates) of mineral wool dust and fibers excludes it definitely as a source of silicosis; providing no free silica is present.

2 - The chemical nature and physical nature of mineral wool fibers places them in the same classification as asbestos fibers which have been definitely shown to produce a fibrosis by mechanical aggravation.

3 - That it may be possible that mineral wool fibers will produce a fibrosis if they can be inhaled into the lungs in sufficient quantities. This quantity is not known.

4 - Tests have proven that mineral wool dusts and fibers are not inhalable into the lungs in sufficient quantities to produce a fibrosis and therefore, do not present a hazard.

5 - No case of a pathology of the lung has been definitely attributed to inhalation of mineral wool dust.

6 - X-Ray examinations of workers in the mineral wool industry have shown no pathological changes in the lung which is perhaps the most conclusive argument against any possible danger from inhalation of mineral wool dusts.

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(signed) William P. Helm

June 3, 1947

CS024 0553

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CS024 0555

November 5, 1947

Mr. F. J. Stephens

Subject: Report #7 "Effects of Inhalation of Mineral Wool Dust & Fibers"

Several months ago, Mr. William P. Helm, Research Analyst at the Torrance Plant, sent us a copy of the paper "Effects of Inhalation of Mineral Wool Dust & Fibers" which he prepared in connection with special studies at the University of Southern California for an additional degree in Industrial Engineering and Management. Mr. Helm's paper was circulated to all members of the Staff.

A few days ago Mr. Helm sent us fourteen copies of the paper, which copies were prepared by Coast Insulating Products. In transmitting these extra copies to us Mr. Helm stated, "Coast was able to use this paper to advantage in their sales efforts, and obliged us by preparing some copies for ourse.

Since these additional copies were made available to us, and because we believe that this exellent treatise should become a part of our permanent technical records, we are releasing it as a Blue Back Report.

C. L. Neumeister

CLN:ms

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