

(COPY)

December 21, 1934

Dr. A. J. Lanza,
Metropolitan Life Insurance Co.,
1 Madison Avenue,
New York City.

Dear Dr. Lanza:

I am handing you herewith the galley proof of your preliminary study entitled "Effects of Inhalation of Asbestos Dust upon the Lungs of Asbestos Workers". I am also enclosing copy of Mr. Hobart's letter to me of December 15th which contains his comments as to certain aspects of the study that he considers undesirable from the industry viewpoint. I trust that you will give his comments and suggestions, as well as those mentioned in my letter of December 10th, your most serious consideration. I am sure that you understand fully that no one in our organization is suggesting for a moment that you alter by one jot or tittle any scientific facts or inevitable conclusions revealed or justified by your preliminary survey. All we ask is that all of the favorable aspects of the survey be included and that none of the unfavorable be unintentionally pictured in darker tones than the circumstances justify. I feel confident we can depend upon you and Dr. McConnell to give us this "break" and mine and Mr. Hobart's suggestions are presented in this spirit.

Sincerely yours,

Vandiver Brown
Attorney

VB:T
Enclosure

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EFFECTS OF THE INHALATION OF ASBESTOS DUST
UPON THE LUNGS OF ASBESTOS WORKERS

Industrial Health Service
Policyholders Service Bureau
Metropolitan Life Insurance Company
New York

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FOREWORD

The authors wish to express their sincere thanks to all those who aided in making this study possible, especially the officials and employees of the asbestos companies who cooperated to the fullest extent and gave every facility for securing data. Also to Dr. Pancoast of the University of Pennsylvania for his interest and valuable advice in interpreting X-ray films and to Dr. V. Meriwether, United States Public Health Service, Surgeon in Charge of the United States Bureau of Mines Cooperative Clinic in Picher, Oklahoma, whose interpretation of all the X-ray films listed in this report is followed. Also to Dr. W. Atmar Smith, and Dr. Kenneth Lynch of Charleston, S.C. for information and advice, particularly as to the pathology of asbestosis;* to Dr. W. W. Wild of Charleston, S.C.; to Dr. Joseph H. Wyatt of Newark, N.J., and Dr. H. H. Fellows of New York, for assisting in interpreting X-ray films; to Dr Paul O. Snoke of Lancaster, Pa.; to Dr. R. H. Stevenson of Danville, Quebec, Canada; to Dr. L. U. Gardner and Mr. Donald E. Cummings of Saranac Laboratory, who have undertaken the study of the pathological effects of the inhalation of asbestos dust through animal experimentation, with the aid of a grant from the Metropolitan Life Insurance Company.

* "Asbestosis Bodies in Sputum and Lung, Kenneth M. Lynch, M.D. and W. Atmar Smith, M.D., Charleston, S.C., Journal of American Medical Association, August 30, 1930, Vol. 95, No. 9, pp. 659-661.

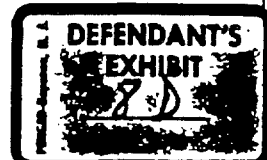
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INTRODUCTION

In 1929 the Metropolitan Life Insurance Company was approached by firms representing the asbestos industry in the United States with the request that a hygienic study be made of that industry. The officials representing these firms were desirous of ascertaining if asbestos dust was an occupational hazard in their establishments and, if so, what was the nature of this hazard and what should be done to prevent or control it.

About this time several articles had appeared in English medical journals describing a pneumoconiosis due to asbestos dust. While in one or two isolated instances the occurrence of this type of pneumoconiosis had been described in American journals, the industry itself appeared to be quite uninformed of the existence of any such occupational disease.

The hazard of silica dust with special reference to the lungs has long been appreciated and a great deal of study and research has been applied to the silica dust problem, primarily in the metal mining industry, in Great Britain, the United States, the British Dominions, as well as in other countries. The nature of the effects of silica dust expressed in the term "silicosis", with the resultant extraordinary predisposition to pulmonary tuberculosis, is well known. These effects have been associated with the inhalation of dust containing free silica in varying amounts. The effects upon the pulmonary tissue of dusts containing combined silica - silicates - are still a fertile field for investigation, but evidence is accumulating that certain of these dusts produce pathological results quite distinct from true silicosis.



The name asbestosis has been applied to the pneumoconiosis caused by asbestos dust and it will be so used in this report. Chemically, the asbestos of commerce is a hydrated magnesium silicate consisting primarily of

Silica 44.1%
Magnesia 43.0%
Water 12.9%

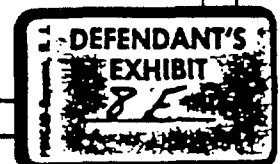
while ferrous iron and nickel are present in small quantities. This commercial variety of asbestos most commonly encountered is designated as chrysotile and is one of the four varieties of the mineral serpentine, in which it usually occurs in seams.

It should be borne in mind that silicosis (and presumably asbestosis) develops very slowly, taking from five to fifteen and even twenty or twenty-five years to become established. This rate of progress is influenced mainly by the dosage of silica which the lungs receive and this dosage, in turn, depends upon three variables: the amount of silica in the dust, the quantity of dust in the air, and the extent of exposure. Individual idiosyncrasy might be included as a fourth variable; however, little is known of the variations in susceptibility in those exposed to dust.

There does not appear to be present, in places where asbestos is mined or fabricated in North America, the clear-cut clinical picture which is so unescapable in communities with a true silicosis hazard, such as hard rock mining communities. It might be thought that some of the asbestos plants are of too recent origin for the typical effects of a silicate dust to become manifest, but this theory would not apply to the older mines or to all of the fabricating plants.

The Industrial Health Service of the Metropolitan Life Insurance

Approximately - Baldy Chrysotile



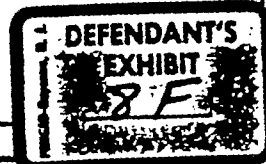
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Company undertook the following investigation during the period from October, 1929 to January, 1931. This investigation included:

- A. A study of dust conditions in asbestos mines, mills, and fabricating plants.
- B. Physical examinations of asbestos workers, including X-ray films.
- C. A study of dust exhaust systems designed to eliminate asbestos dust.

Asbestos fabricating plants located along the Atlantic Seaboard were included in this study and are designated in the report as plants A, B, C, D, and E. Asbestos mines and mills in the Province of Quebec are designated as F, G, H, and I.

The Canadian portion of the investigation was conducted jointly with the Industrial Hygiene Division of the Department of Public Health of the Medical School of McGill University. The results will be summarized in a report which will follow the present one dealing with the investigation in the United States.



DUST STUDIES

Apparatus and Methods of Sampling:

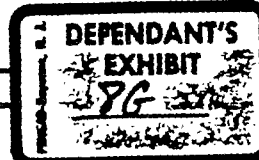
Both the impinger (1) and the electric precipitator (2) were used for the collection of air samples, for dusts, at the breathing levels and in close proximity to the workmen. Both of these forms of apparatus are adapted for this work, as they are transportable, easily set up and adjusted for the taking of the air samples at the proper level, and are extremely efficient.

The impinger collects the dust by aspirating the air and then impinging it onto a flat surface covered by a liquid in a container. The liquid used was distilled water, containing 50 percent alcohol to prevent the solution of some of the dusts, particularly any silica which might be present. The impinger was actuated by an electrically driven rotary pump and the rate of air sampled was measured by means of a resistance type of flow meter with an inclined manometer for measuring the pressure difference. Each sample represents the dust collected from a volume of 100 cubic feet of air.

The electric precipitator is designed upon the Cottrell precipitator principle used for recovering dusts and fumes commercially. This principle depends on electrifying the dust particles by making them pass through an electrostatic field and thus causing them to settle out upon a sheet of celluloid. The electrostatic field is set up by means of a transformer operating from the lighting lines on 120 volts, alternating current. Air is drawn through the apparatus by means of a small rotary fan, run by a motor, the quantity being

(1) "Comparative Tests of Instruments for Determining Atmospheric Dusts", Public Health Bulletin No. 144, Treasury Department, Washington, D.C.

(2) "Determination of Suspensoids by Alternating-Current Precipitators", Philip Drinker and Robert M. Thomson, Journal of Industrial Hygiene, Vol. VII, No. 6, June, 1925.



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measured by means of a flow meter.

Samples of dust brushed from beams and pipelines near the ceiling also were secured for chemical analysis. All samples were shipped to the Industrial Hygiene Laboratory of the Metropolitan and the dust counts made according to the methods accepted by the U.S. Bureau of Mines and the U.S. Public Health Service.⁽¹⁾ Particles in size up to 360 microns in the greatest diameter were counted.

Chemical analyses were made for total (free and combined) silica according to the accepted standard method of Hillebrand.⁽³⁾

Percentage Frequency of Particle Size:

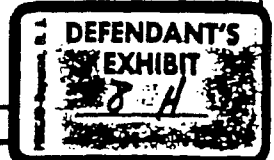
In the first plant studied (A) only particles 10 microns and under in size were counted. It has been demonstrated repeatedly that no silica particles exceeding 10 microns - a micron equals one-millionth part of a meter or one-thousandth part of a millimeter - in greatest diameter, enter the lung tissue, and for this reason the larger particles usually are not considered when determining dust counts. Later, as it appeared from some of the published articles⁽⁴⁾ (English) that asbestos particles much larger in size were found in lung tissue, it was decided to count all particles and make differential counts of all those 10 microns and under.

Both samples collected by the impinger and the electric precipitator were counted. Microphotographs of these dust particles were further enlarged by being projected upon a screen by means of a lantern slide projector, the enlarged particles being measured with a ruler. Knowing the entire enlargement (by actual

(1) "Comparative Tests of Instruments for Determining Atmospheric Dusts", Public Health Bulletin No. 144, Treasury Department, Washington, D.C.

(3) "The Analysis of Silicate and Carbonate Rocks", W.F. Hillebrand, Bulletin 422 U.S. Geological Survey, Washington, D.C.

(4) "Pulmonary Asbestosis", W. E. Cooke, British Medical Journal, December 3, 1927.



measurement), it was easy to calculate the original particle size.(5) By the use of Hazen's (6) logarithmic probability paper, the logarithms of the function to be measured - in this case microns - are plotted as ordinates against the probability of occurrence as abscissae. Since the line is straight, extrapolation and interpolation of any desired region is facilitated.

The analyses are reported in total particles per cubic foot and also in the corresponding weight of the dust in milligrams. There is no indicated relationship between the dust counts and the corresponding weights. Consequently, the weights are not included in the tabulations in this report.

Table No. 1 shows by plants and by departments the dust counts in million particles per cubic foot of air. The plants included are all in the United States and are mainly of a textile nature. It will be noted that the counts in plant A are of particles 10 microns and under, while in the other plants all the particles were counted. Nevertheless, direct comparison is possible, as in no sample taken were the number of particles 10 microns and under less than 94 percent of the whole, which makes it evident also that in regard to dust control we are concerned only with the smaller sizes.

It is also important to note that whereas the dust in the preparation room is practically all due to asbestos, the contrary is the case in the other departments.

In the carding, spinning and weaving rooms asbestos is about 25 percent. of the material used. On special jobs the percentage of asbestos may be

(5) Green, H. "A Photomicrographic Method for the Determination of Particle Size of Paint and Rubber Pigments", Journal Franklin Institute, 1921, 192, 637.
(6) Whipple, G.C. - Vital Statistics, John Wiley & Sons, N.Y., 1925, p. 451.

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higher, but in general the figure quoted is approximately correct, the other material consisting principally of cotton. In the insulating departments of plant A, asbestos is but 5 per cent. of the total material used.

In the molded brake lining and clutch division of plant D, asbestos is about 25 per cent. of the total material.

The actual exposure to asbestos dust, therefore, is considerably less than is indicated in the dust counts in Table I.

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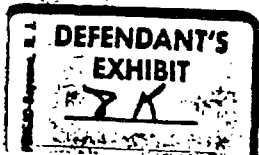


TABLE I

Dept.	PLANT A		PLANT B		PLANT C		PLANT D		PLANT E	
	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air	No. Samples Taken	Million Particles per cu.ft. of air
reparation	5	1/2 to 1 1/2	9	1/2 to 8	8	2 to 4 1/2	—	—	3	30 to 82
ard room	3	1/2 to 1 2/5	5	3/4 to 4 1/2	6	5 1/2 to 15 1/2	—	—	3	28 to 78
ing frame pinning room	2	1/2 to 3/4	—	—	—	—	—	—	—	—
hule spinning	2	2/3 to 2 1/2	5	1/5 to 1	3	1 1/4 to 4 1/2	—	—	3	5 1/2 to 10
rlating	10	1/5 to 1 1/2	—	Included in spinning	8	1 to 5 1/2 (includes broad loom weaving)	—	—	3	6 1/3 to 7 (includes broad loom weaving)
aving	4	1/20 to 2	4	1/3 to 4/5	6	5 1/2 to 10	15	1 to 7	3	10 1/2 to 19 1/2
elt Dept.	6	1/3 to 44 1/2	—	—	—	—	—	—	—	—
sheet in- sulating	3	1/3 to 4	—	—	—	—	—	—	—	—
welded brake band and clutch	—	—	—	—	—	—	2	1/2 to 3/5	—	—

CONDITIONS IN THE PLANTS

The processes in these plants were very similar to cotton mills in general. For illustration, the process in the preparation room of plant B is detailed at length:

Asbestos is received in bags and these are emptied upon the floor and the asbestos is shoveled into pug mills and run for about five minutes in order to crush and open the fibres. From there the asbestos is shoveled into trucks and wheeled over to hoppers with either horizontal or vertical openers, similar to those used in the textile mills. After passing through the opener, the material is discharged into trucks. Waste manufacturing material is first run through a garnet machine and discharged into trucks which are wheeled over to the openers and the material is fed into these in the same manner as the new material. The material, after being discharged from the opener is put onto a vibrating or shaking screen where the long fibres are picked off by a suction hood and blown into a bin to be used again in textiles. The short fibres falling through the screen are either sold as such or used in making an asbestos cement.

The cotton is received in bales, opened, and also run through vertical openers. It is discharged into trucks. The filled trucks from the vertical and horizontal openers containing either the asbestos fibre, the cotton or the waste material are transported to separate storage bins. As needed, these materials are weighed and dumped onto the floor in proportion to the mixture desired and the resulting mixed materials are then passed through



a mixing picker. As this mixture is discharged from the picker, it is sprayed with a light mineral oil. The material is then passed onto a mechanical conveyor and taken to the storage bin in the card room. Two of these mixing pickers discharge onto this conveyor system, while a third machine is equipped with a suction device which conveys the material to a storage bin in the card room. The object of the two systems is to facilitate the handling of two grades of material at the same time. This third machine is not equipped with an oil spray. The object of the oil spray is primarily to entrap the small asbestos fibres and hold them enmeshed in the cotton fibre throughout the processes of carding, spinning, and weaving. Incidentally, it is apparent that it also diminishes the amount of dust. The material is saturated with about 4 per cent. of oil at this point, but by the time it reaches the looms the oil has diminished to less than 1 per cent.

While mineral oil was used in the preparation room of plant C, it apparently was not as efficacious as in plant B. In plant E oil was not used, nor was there any attempt at humidification or any system of dust exhausting. Carding machines were fed by hand. In plant A there was a humidifying, ventilating and heating system, while in plant B natural humidity sufficed for the process. In plant E the carding machines were equipped with a dust exhaust system, but it was not efficiently used. In plant C carding was done in two buildings, one of which was equipped with an air conditioning system, and in both buildings the machines had exhaust equipment.

Two plants, B and C, had artificial humidity installations in their spinning rooms. In the former the temperature was 76°F., relative humidity 78 per cent.

One plant, C, had a humidification system in the twisting department

and also in the weaving department where the relative humidity was 76 per cent. with a dry bulb temperature of 69°F., as compared with a relative humidity of 44 per cent. in the weaving room of plant A.

Aside from plant E, which was unnecessarily dusty, it would appear that the dust hazard was not excessive except in the preparation rooms of plants B and C. The dust counts are interesting too, when considered in the light of the permissible standard for siliceous dust, established by the United States Public Health Service,* namely 10 million particles (10 microns and under) per cubic foot. However, we are not justified in assuming that because asbestosis is a milder disease, clinically, than silicosis, the threshold of permissible dust counts is higher. Asbestosis is pathologically different from silicosis, and the experience so far does not warrant an attempt to define an arbitrary standard of dustiness.

DUST CONTROL

Various measures, such as oiling, humidification, and local exhausts, tended to reduce the dust. Nevertheless, it was evident that they were only partially successful. It also is apparent that, if it is expected to control dustiness in these plants, final reliance must rest upon properly constructed exhaust equipment. In plant C in one department an experimental installation was set up. In spite of some obvious faults, this equipment, on the basis of comparative dust counts, reduced the dust by 50 per cent. and with further alterations will probably be 75 per cent. effective. For all practical purposes, this is quite satisfactory. It is neither practicable nor economically desirable to install such equipment as will make the air entirely dust free, even if this

* "Health of Workers in Dusty Trades (Granite Industry", Public Health Bulletin No. 187, U.S. Public Health Service, Washington, D.C., 1929.

were possible. The normal defensive mechanism of the body takes care of a fair amount of atmospheric pollution. It is when the body is exposed to an excessive amount of dust over a long period of time that this defense mechanism breaks down.

The application of exhaust equipment to textile machinery involves considerable difficulty, especially where the construction of the plant is such that it is not possible to apply down draft suction to the looms.

It is desirable to install exhaust apparatus in any plant on an experimental basis first, and then check its efficiency by dust counts. This practice will result in saving the needless expenditure of much money. C

PHYSICAL EXAMINATIONS

It was soon obvious that the diagnosis of asbestosis must rest mainly on X-ray films of the chest. As in the early stages of silicosis, the symptoms of the asbestos workers were usually indefinite and inconclusive. The following tables are based on physical examination, together with X-ray films of 126 persons actually working in asbestos plants in the United States. No Canadian results are included here. These cases were selected more or less at random from among those having more than three years of employment in the industry. The interpretations of these films all are based on the readings of one competent roentgenologist, but they have been studied by several others experienced in this field. The differences of opinion were of a minor nature, and there was general agreement as to interpretation. The films were read conservatively, the X-ray findings took into account the physical examination and the age of the individual, and only those were classed as positive about which there was



no major disagreement. All concerned were guided by their experience in silicosis and other types of pneumoconiosis, hence the films classed as negative for asbestosis were further subdivided into "doubtful" and "negative". Only time can tell whether the individuals classed as doubtful are progressing toward a definite asbestosis. Particular attention was focussed on the presence or absence of indications of tuberculosis and, in this respect, all the reviewers of these films were in accord. With the unhappy experience of silicosis in mind, it was felt that a great deal of care should be devoted to ascertaining, if possible, whether or not asbestosis predisposes to tuberculous infection.

Table II exhibits the diagnostic classification of these 126 individuals, with their symptoms and specific occupations. Five of these persons were not physically examined.

The cases of asbestosis are divided into two classes, first stage and second stage. The first stage embraces those who show by X-ray examination definite lung pathology sufficient in extent to warrant a diagnosis of pneumoconiosis, but who have no definite symptoms. The second stage cases exhibit more extensive lung pathology and also definite symptoms.

All these individuals were actively engaged in factory work, and it was not practicable to make any distinction between them on the basis of working ability or disability.

Had any individuals been found who exhibited extensive pulmonary involvement and marked physical disability or total disability, they would have been classified as third stage, but no such were found.

One asbestos worker (2nd degree stage) retired of his own accord and because of old age ten years previous to this study, but is still active about his garden.

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It will be seen from Table II that of a total of 126 X-ray examinations four were diagnosed as second degree asbestosis; sixty-three as first degree; thirty-nine as doubtful; and twenty as negative.

Table II also shows that only eight individuals out of sixty-four with positive asbestosis were entirely free of symptoms, while ten out of thirty-seven with doubtful and seven out of twenty with negative diagnoses were free of symptoms.

Dyspnoea and cough were the symptoms most complained of, but none of these cases exhibited the urgent or evident type of "short wind" seen in true silicosis. Several of those classed as negative stated that they were "short-winded" and were so recorded, but too much emphasis should not be placed on these statements of subjective symptoms. Table III shows the occurrence of dyspnoea and cough.*

TABLE III

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
Dyspnoea	40	16	8
Cough	39	20	10
Not examined	3	2	0
Total	67	39	20

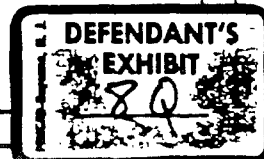
The incidence of tuberculosis (based upon X-ray films) as revealed in Table IV is both interesting and important.

TABLE IV

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
Tuberculosis (healed)	7	1	2
Tuberculosis (active)	1**	0	1
Total Examined	67	39	20

*In Tables III to XI, inclusive, the division into positive, doubtful, and negative refers to the cases so classified in Table II.

**This case was diagnosed as probably active on the X-ray findings.

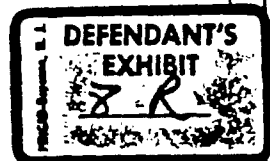


During the progress of the study, physicians who were practicing in the communities where asbestos workers lived were questioned, and stated that they did not find an unusual amount of tuberculosis among these workers. The contrast between this state of affairs and that found in communities with a silicosis hazard, is noteworthy.

The following tables classify results of examinations by occupation in industry and by years of exposure. It is to be remembered, however, that these individuals are classified according to the department in which they were working at the time of examination or where they had spent most of their time. Many had changed from one department to another and from one plant to another during their years of employment in the asbestos industry. Since only the amounts of dust collected at the time of this survey in the various departments are known, there is no way of knowing the average amount of dust in the atmosphere inhaled by these people over the years of their employment. Consequently, it is not possible to correlate individual cases with definite amounts of dust exposure.

TABLE V
By Departments

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
Weaving	22	13	5
Carding	13	4	1
Spinning	9	3	3
Warping	1	0	2
Spooling	3	5	2
Trucking	1	1	0
Loom fixing	3	1	0
Packing	1	0	0
Winding	3	4	1
Picker	2	0	2
Twisting	3	5	0
Other	6	3	4
Total examined	67	39	20



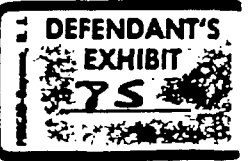


TABLE II

ANALYSIS OF FINDINGS
First Period Stage of Development
From Period

Second Period Stage of Development

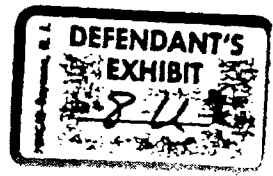
Case No.	Date of Observation	No. of Observations	Sex	Age	Findings	Stage of Development	Location	Significance	Application
1	Carling	49	M	39	Vertical striations both lungs; generalized mottling.	Stage, subacute at base	1 inch	Hyperemia, cough (dry), slight exertion.	—
2	Carling	100	M	40	Vertical striations and mottling, both lungs.	(not observed, but not worked for post 30 yrs.)	—	Hyperemia, exertion poor, exertion exertion frequently vesicular before breakfast.	—
3	Splinting	41	M	36	Thin mottling both lungs. Allium obscure enlarged.	Stage at base	2 inches	Hyperemia, history of chest trouble.	—
4	Carling	20	M	31	Thin mottling enlarged; general baricentric and some mottling both lungs.	Stage enlarged	1 1/2 inches	Hyperemia, history of chest trouble.	—
First Period									
1	Spooling	4	F	19	Allium obscure enlarged; early mottling both lungs.	Stage of subacute	2 inches	Hyperemia, cough (dry) slight exertion.	—
2	Treating	6	M	40	General striated spots; thin mottling both lungs.	Stage, subacute at base	2 inches	Exertion, history of chest trouble.	—
3	Splinting	12	M	23	Consistent mottling both lungs.	—	1 1/2 inches	Hyperemia, history of chest trouble.	—
4	Carling	5	M	42	Uniform mottling both lungs.	Stage at base; increased vesicular.	2 inches	Hyperemia (allium) cough (dry) exertion.	—
5	Carling	5	M	26	Thin mottling, particularly right side.	—	2 inches	Hyperemia (allium) cough (dry) exertion.	—
6	Carling	15	M	32	Thin mottling both lungs.	—	1 1/2 inches	Cough (dry) pronounced on deep inspiration.	—
7	Carling	4	M	31	Generalized mottling throughout both lungs.	Stage at base.	1 1/2 inches	Hyperemia.	—
8	Carling	12	F	36	Thin mottling both lungs; allium obscure enlarged.	—	1 inch	Hyperemia cough (dry) exertion.	—
9	Spooling	4	F	29	Perforated striations; thin mottling in both lungs.	—	1 1/2 inches	Hyperemia (allium) cough (dry) exertion.	—
10	Spooling	4	M	10	Small amount of mottling generalized throughout both lungs.	—	2 inches	Swelling of side around middle.	—
11	Spooling	4	F	11	Consistent thickening and early mottling both lungs.	—	1 1/2 inches	Cough (dry)	—
12	Treating	5	F	33	Perforated striations; general baricentric to both lungs.	—	2 inches	Hyperemia.	—
13	Carling	12	M	27	Thin mottling both lungs; heart enlarged.	—	2 inches	Hyperemia.	—
14	Carling	4	M	23	Thin mottling and mottling in both lungs.	—	1 1/2 inches	Hyperemia.	—
15	Carling	9	F	34	Thin mottling and early mottling in both lungs.	Stage enlarged.	2 inches	Hyperemia.	—
16	Carling	4	M	29	Thin mottling and early mottling in both lungs.	Stage enlarged.	2 inches	Hyperemia.	—
17	Carling	4	M	25	Thin mottling and early mottling in both lungs.	Stage enlarged.	2 inches	Hyperemia.	—

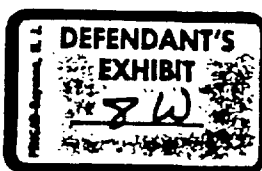
18	Swelling	4	2	4	peribronchial thickening and mottling, particularly bronchi	dry rales posteriorly at base.	2 inches	(expiratory) crackle of air around mucus	---
19	heaving	4	4	55	Considerable early mottling in both lungs.	scattered rales throughout breath sounds.	1 inch	(expiratory) cough (dry) history of chest trouble.	small rales right upper lobe.
20	heaving	3	11	2	peribronchial thickening and early mottling both lungs.	low crackle.	not taken	not questioned.	---
21	heaving	4	4	24	peribronchial thickening and mottling both lungs.	heart enlarged heart murmurs increased white sounds	2 inches	none	---
22	heaving	13	4	41	infiltrate in both lungs.	scattered rales.	1/2 inches	(expiratory) (slight)	heart enlarged
23	heaving	15	2	42	peribronchial enlargement both lungs mottling.	distended ribs.	1 inch	(expiratory) cough on inspiration.	---
24	heaving	16	4	39	peribronchial thickening and mottling both lungs.	distended left apex.	2 1/2 inches	(expiratory) (slight) cough (with expiratory rales).	small rales left apex.
25	heaving	16	2	53	General mottling both lungs, faintly right apex.	distended ribs, apex distended at breath sounds.	1 inch	(expiratory) cough on inspiration.	small rales left apex.
26	heaving	15	4	7	fine mottling especially on left side.	distended right upper lobe.	2 inches	(expiratory) cough (dry)	small rales left apex.
27	heaving	4	7	45	heavy and dense shadow both bases.	---	1 1/2 inches	(expiratory) (slight) cough (with expiratory rales) mottling of ribs around middle.	small rales left apex.
28	heaving	4	4	26	Some peribronchial thickening and mottling both lungs.	not noted.	---	---	---
29	heaving	5	4	33	peribronchial thickening and some mottling.	distended breath sounds.	1 1/2 inches	Cough (dry) poor expiratory markings.	---
30	heaving	6	4	42	fine mottling in both bases, particularly right.	slight distended at bases.	2 inches	no fact might mottling of ribs around middle.	---
31	heaving	3	4	21	Considerable thickening and mottling both lungs.	---	2 inches	none	---
32	heaving	5	4	46	Some thickening and mottling in both lungs, particularly right.	---	1 1/2 inches	(expiratory) cough (dry).	---
33	heaving	3	4	23	peribronchial thickening and mottling both lungs.	---	3 inches	slight cough (with expiratory rales).	---
34	heaving	7	4	21	Shadow enlarged peribronchial thickening both lungs.	---	2 inches	slight cough (dry) mottling of ribs around middle.	---
35	heaving	8	4	33	Considerable peribronchial thickening and mottling both lungs.	heart enlarged distended left base.	1 1/2 inches	Cough (with expiratory rales).	---
36	heaving	7	4	32	distal shadow above several scattered spots in both lungs some mottling.	---	1 1/2 inches	(expiratory) (slight) cough dry.	---
37	heaving	8	4	36	Shadow enlarged fine mottling both lungs.	heart enlarged.	2 1/2 inches	(expiratory) cough (dry) history of chest poor appetite.	---
38	heaving	5	4	39	Shadow enlarged fine mottling both lungs.	---	2 inches	(expiratory) cough (dry) mottling of ribs around middle.	---
39	heaving	4	4	23	Shadow enlarged rather general mottling both lungs.	---	2 inches	(expiratory) (slight) shadow none.	---
40	heaving	3	4	33	Scattered spots in both lungs shadow fine mottling both lungs.	---	2 inches	(expiratory) (slight) cough (dry).	---
41	heaving	23	4	29	distal shadow enlarged small amount thickening and mottling both lungs.	---	3 inches	(expiratory) (slight) shadow none.	---
42	heaving	3	4	37	fine mottling in both lungs.	distal at bases distended at breath sounds.	1 inch	none	---

44	Curling	9	11	33	Thickening and rattling throughout both lungs.	Diminished right basal diaphragmatic breath sounds.	1 1/2 inches	Dyspnea (slight); cough (with expectoration); infection base.	Healed the.
45	Swelling	10	12	32	Some thickening and rattling, particularly at bases.	—	1 1/2 inches	Dyspnea; cough (dry); slight expectoration; history of chest trouble.	Healed the.
46	Twisting	7	7	31	Peribronchial thickening and rattling both lungs.	—	1 1/2 inches	Dyspnea (slight).	—
47	Spitting	8	8	30	Peribronchial thickening and rattling both lungs.	—	1 1/2 inches	Dyspnea	—
48	General distention of digits.	15	15	61	Right hilum enlarged; generalized rattling both lungs.	Rattled right; diminished breath sounds.	1 inch	Dyspnea; cough (dry).	—
49	Spitting	6	6	48	Peribronchial thickening in both lungs; generalized rattling.	Bullness at bases.	2 1/2 inches	Cough (with expectoration); history of chest trouble.	—
50	Curling	9	9	27	Right hilum enlarged; considerable peribronchial thickening throughout.	—	2 inches	Cough (dry); swelling of skin around navel.	—
51	Curling	4	4	24	Some thickening and rattling both lungs.	—	2 1/2 inches	Cough (dry); history of chest trouble.	—
52	Picker	5	5	42	Peribronchial thickening and rattling both lungs.	Diminished at bases; some dullness.	2 inches	Dyspnea (slight); poor appetite; infection base.	Same as such.
53	Curling and twisting	8	8	24	Considerable peribronchial thickening and rattling both lungs.	—	3 inches	Cough (with expectoration).	—
54	Eleventh thoracic	9	9	44	Rattling throughout both lungs; some relieved spots.	Bullness at base; diminution of breath sounds.	1 1/2 inches	Expectoration; some dyspnea	—
55	Twisting	12	12	35	Right hilum enlarged; considerable rattling both lungs.	Diminished at right base.	2 inch	Dyspnea; cough (dry); poor appetite (normal); swelling of skin around navel.	—
56	Forming (airing)	16	16	44	Considerable rattling both lungs.	Diminished breath sounds.	2 inches	None	Healed the.
57	Swelling	8	8	41	Considerable peribronchial thickening and rattling both lungs.	Increased valve sounds at bases.	1 inch	Loose weight; infection base; cough (dry).	—
58	Swelling	9	9	34	Some rattling and peribronchial thickening both lungs.	Diminished right apex.	2 inches	Cough (dry); swelling of skin around navel; infection base.	—
59	Swelling	12	12	32	Peribronchial thickening and rattling, particularly at bases.	Slight dullness at bases.	2 inches	Dyspnea; cough (dry); swelling of skin around navel.	Healed the.
60	Swelling	10	10	50	Enlarged hilum; involvement, slight peripheral.	Diminished breath sounds.	2 1/2 inches	Dyspnea; cough (with expectoration); lost weight; history of chest trouble.	Healed the.
61	Twisting	15	15	37	Further marked rattling at bases.	—	2 1/2 inches	Dyspnea (slight); cough (with expectoration); history of chest trouble.	—
62	Curling	5	5	44	Generalized rattling both lungs.	Diminished posteriorly; some dullness.	2 1/2 inches	Dyspnea; cough (dry); poor appetite (normal); history of chest trouble.	Healed the.
63	Swelling	22	22	34	Right hilum enlarged; rattling at bases.	—	1 inch	Dyspnea; cough (dry); slight swelling of flange.	Healed the.

Continued on next page.

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TABLE IX

Physical Abnormalities (Classified as Acquired)

Case No.	Depth of Fluct	Time of Appearance	Sex	Age	History	Chest Findings	Chest Expansion	Symptoms	Significance
1	Anding	5	M	23	Small amount mottling more fibrous than normal.	--	2 inches	--	--
2	Overling	3	M	18	Small amount thickening both lungs.	--	2 1/2 inches	Slight cough (dry).	--
3	Anding	7	M	21	Some fine mottling more fibrous than normal.	--	2 inches	Cough (dry) swelling of aorta around aortic valve.	--
4	Retaling	3 1/2	M	13	Alveolar shadows enlarged more fibrous than normal.	--	1 1/2 inches	Dyspnea (slight).	--
5	Spotting	4	F	10	Beginning mottling at bases some peribronchial thickening.	--	1 1/2 inches	Dyspnea severe (40) expectoration poor morning expecto. excitation.	--
6	Spotting	5	M	21	Some peribronchial thickening small amount mottling.	--	2 1/2 inches	--	--
7	Anding	4	M	22	Some early mottling and thickening both lungs.	--	2 inches	--	--
8	Long fibrous	5	F	37	Alveolar shadows enlarged some mottling both lungs.	--	2 inches	Dyspnea (slight) severe (dry) severe slightly clashing of trachea.	--
9	Spotting	1 1/2	M	22	Alveolar shadows enlarged some peribronchial thickening.	Not examined.	--	--	--
10	Spotting	8	F	64	Calcified spots left hilum some mottling both lungs.	Not examined.	--	--	--
11	Spotting	4	M	23	Alveolar shadows enlarged small amount mottling.	--	1 1/2 inches	Swelling of aorta around aorta.	--
12	Spotting	4	M	24	Some thickening both lungs.	--	1 1/2 inches	--	--
13	Spotting	4	F	37	Alveolar shadows enlarged some mottling.	--	1 1/2 inches	--	--
14	Spotting	2 1/2	F	37	Alveolar shadows (right) small amount mottling both lungs.	Heart enlarged, aorta normal.	1 inch	Dyspnea.	--
15	Spotting	4	M	22	Some thickening and mottling both lungs.	--	2 inches	Cough (dry).	--
16	Spotting	3	M	29	Some peribronchial thickening early mottling right lung.	--	1 1/2 inches	Cough (with expectoration).	--
17	Spotting	9	F	26	Alveolar shadows enlarged peribronchial thickening both lungs.	--	2 inches	Slight cough (with expectoration) expecto.	--
18	Spotting	5	F	32	Some peribronchial thickening and mottling both lungs.	--	1 1/2 inches	--	--
19	Spotting	8	M	41	Some peribronchial thickening and early mottling both lungs.	Suggestion of calcification at bases.	2 inches	Cough (dry) expecto.	--
20	Spotting	10	M	42	Considerable peribronchial thickening and early mottling at bases.	--	2 1/2 inches	Some. Abnormal expecto.	--
21	Spotting	7	M	31	Alveolar shadows enlarged some mottling both lungs.	--	2 1/2 inches	Dyspnea swelling of aorta around aorta.	--
22	Spotting	1 1/2	F	33	Some fine mottling both lungs Alveolar shadows enlarged.	--	1 1/2 inches	Dyspnea (slight) cough (dry)	--
23	Spotting	7	M	28	Some mottling both lungs Alveolar shadows enlarged.	--	2 inches	--	--
24	Spotting	4	M	10	Some mottling both lungs Alveolar shadows enlarged.	Heart enlarged.	2 inches	Cough (dry) expecto. swelling of aorta around aorta.	--

TABLE II
Locality for Abnormalities

Case No.	Age	Sex	Time of Onset	Duration	Chief Findings	Chief Complaint	Diagnosis	Disposition
1	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
2	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
3	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
4	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
5	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
6	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
7	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
8	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
9	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
10	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
11	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
12	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
13	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
14	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
15	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
16	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
17	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
18	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
19	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...
20	4	F	12	12	Plastic adhesion both bases.	1 1/2 in.	Cough (dry)	...

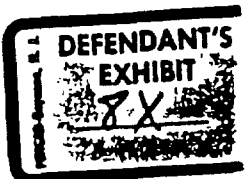


TABLE VI

Years of Exposure

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
Over 15 years	13	1(part time)	3 (1 not
10 to 15 years	7	3	2 cor-
5 to 10 years	30	27	3 tin-
Under 5 years	17	11	12 uous)
Total examined	67	39	20

The following tables are also of interest, but, like the two previous, are of limited value and little in the way of definite conclusions can be based upon them.

TABLE VII

Age Groups

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
50 and over	12	3	2
40 to 49	13	6	4
30 to 39	17	12	6
20 to 29	24	16	8
Under 20 years	1	2	0
Total examined	67	39	20

TABLE VIII

Sex

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
Male (Total examined 108)	59	32	17
Female (" " 18)	8	7	3

TABLE IX

Chest Expansion

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
1 to 2 inches	28	13	7
2 to 3 inches	31	21	12
3 inches plus	5	3	1
Not examined	3	2	0
Total	67	39	20

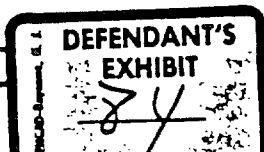


TABLE X

Chest Findings

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
Physical signs of chest trouble	33*	9**	7***
No physical signs	31	28	13
Not examined	3	2	0
Total	67	39	20

What significance, if any, can be attached to the presence of "asbestos corns" on the hands of workers appears doubtful, as indicated in Table XI:

TABLE XI

	<u>Positive</u>	<u>Doubtful</u>	<u>Negative</u>
Asbestos corns	12	7	2
Not examined	3	2	0

Each roentgenologist who reviewed the X-ray films called attention to the fact that these films indicated a very unusual incidence of enlargement of the heart. It is probable that this is a compensatory enlargement due to the additional work put upon the heart in efforts to pump blood through the fibrosed lungs. In the first silicosis studies, published in this country in 1917,**** Dr. S.B. Childs of Denver, who interpreted the X-ray plates of this study, commented as follows:

"The heart in miners' consumption (silicosis) casts a larger shadow than normal and in the early stages is probably caused by its increased musculature. The shadow of the heart remains larger than normal in the second and also in the third stage."

- *At least six of these showed no evidence associated with asbestosis.
- **Two of these not associated with usual signs found with asbestosis.
- ***Six of these not associated with usual signs of asbestosis.
- **** "Miners' Consumption", U.S. Public Health Service Bulletin #85, January, 1917.

DEFENDANT'S
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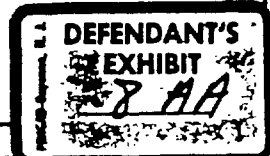
It is possible that not sufficient attention has been paid to the effects of the pneumoconioses upon the heart.

INSURANCE CLAIMS

In the effort to throw any possible light on the relationship between asbestosis and pulmonary tuberculosis, an analysis of death claims and total and permanent disability claims was made in regard to those companies carrying Group insurance and where the figures were available.

There were 2099 lives involved for a total exposure of 7019 life years. The death claims are so small in number that reliable conclusions cannot be arrived at from any subdivision of the figures. The same is true of the sickness claims under health insurance. The number of claims for respiratory diseases is high in two of the plants studied but low for the others, based upon the Metropolitan experience of 1927. However, during the latter part of 1928 and the first part of 1929 there was an epidemic of influenza.

→ The records of one establishment (plant B) showed that six of thirty-six death claims and eight of ten permanent and total disability claims were listed as due to pulmonary tuberculosis. This appeared to be an inordinate amount of tuberculosis from this plant. However, many of the employees were Negroes and also tuberculosis claims are generally high in this section of the country. Realizing the difficulty in diagnosing pneumoconiosis and the tendency to confuse it with tuberculosis, these claims were studied individually. The physicians who had treated the individuals were interviewed, and hospital and sanatorium records, including available X-ray films, were investigated with the following results.



Death claims:

1. Proved to be a typist, not exposed to dust; died of pulmonary and intestinal tuberculosis.
2. Colored male age 27; worked in asbestos one year and eight months; died of pulmonary tuberculosis following a hemorrhage; was in sanatorium ten months. Also had a four-plus Wasserman. No evidence of asbestosis.
3. Colored male age 32; worked in asbestos plant one year and seven months; died of pulmonary tuberculosis after one month in hospital. Cavitation both lungs; no evidence of asbestosis.
4. Colored male age 34; worked in asbestos plant two years, and six months. His physician believes this was a case of uncomplicated tuberculosis. Was not inmate of hospital or sanatorium.

No information could be obtained of the other two cases except from the death certificates which gave pulmonary tuberculosis as the cause of death.

Total and permanent disability claims:

1. Male, employed in asbestos plant three years. His physician states he first came under his observation with an old established case of tuberculosis about two years after asbestos employment started. Also had tuberculosis of the kidney and cervical glands.
2. Male, employed in asbestos plant eight months. His physician states old fibroid tuberculosis with tubercle bacilli in sputum. No X-ray available, but according to physician asbestos bodies were found in sputum.
3. Male, ten years employment. Two physicians who treated him at different times are now inclined to believe this man is not tuberculous, but has asbestosis.
4. White male, was re-examined at time of investigation. His physician reports well nourished, husky looking, good color, no cyanosis; no clubbing of fingers, diminished expansion, incessant cough; X-ray shows fine mottling disseminated through both lungs. No evidence of tuberculosis. Probably a second stage asbestosis.

DEFENDANT'S
EXHIBIT

BBB

4. Asbestosis as observed in this series of cases does not cause any marked degree of disability. However, it is possible for uncomplicated asbestosis to result fatally.
5. It is not known how much asbestosis may add to the mortality of pneumonia and acute pulmonary infections other than tuberculosis.
6. It is not practicable as yet to establish standards for the dust content of air and in view of the many low dust counts in this report, compared with the U.S. Public Health Service standard for silica dust, it is possible that asbestos may cause pneumoconiosis more readily than free silica (SiO₂).
7. The amount of dust in the air in the asbestos plants studied can be substantially reduced.

RECOMMENDATIONS

1. That the industry seriously face the problem of dust control in asbestos plants.
2. That new employees be examined physically and rejected for employment if they show tuberculosis or pneumoconiosis.
3. That employees be examined physically every year or certainly every two years, this examination to include an X-ray examination of the chest.
4. That the industry sponsor studies on known cases of asbestosis, as well as studies on effects of asbestosis on the heart and circulation.



5. White male, thirteen years in asbestos plant. Is now in sanatorium. An interesting case. His physician states he has extensive asbestosis and pulmonary tuberculosis; cavity in right lung; sputum loaded with tubercle bacilli and asbestos bodies; believes tuberculosis long antedated asbestosis. This patient is progressing in a satisfactory manner.

It was not possible to locate the other three cases of total and permanent disability who had been diagnosed as having pulmonary tuberculosis. On the basis of the information obtained the death claims appear to be due to uncomplicated tuberculosis; three of them were Negroes. It was more than probable that these men were tuberculous at the time their employment in the asbestos plant commenced.

Of the eight disability claim cases, one was uncomplicated tuberculosis and two were uncomplicated asbestosis who were put on disability because of a mistaken diagnosis of tuberculosis. In this same community we know of one death due to uncomplicated asbestosis in an individual with many years employment in the industry.*

CONCLUSIONS

1. Prolonged exposure to asbestos dust causes a pulmonary fibrosis of a definite type distinct from silicosis demonstrable on X-ray films. Clinically, it is of a type milder than silicosis.
2. There is associated with asbestosis in a large percentage of cases definite cardiac enlargement.
3. Asbestosis does not involve an unusual tendency to pulmonary tuberculosis.

*Personal communication. Details not yet published.



4. Asbestosis as observed in this series of cases does not cause any marked degree of disability. However, it is possible for uncomplicated asbestosis to result fatally.
5. It is not known how much asbestosis may add to the mortality of pneumonia and acute pulmonary infections other than tuberculosis.
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