PLAINTIFF'S EXHIBIT

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(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

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. [redacted]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.

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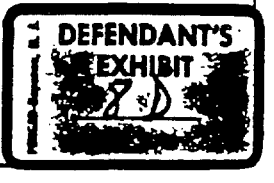
* "Asbestosis Bodies in Sputum and Lung, Kenneth M. Lynch, M.D. and W. Atmar Smith, M.D., Charleston, S.C., Journal of American Medical Association, August 30, 1930, Vol. 95, No. 9, pp. 659-661.

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 Britian, 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 - Sally Choyce

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

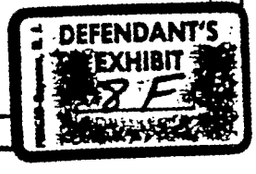
A. A study of dust conditions in asbestos mines, mills, and fabricating plants.

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:

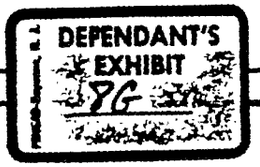
Both the impinger (1) and the electric precipitator (2) were used for the collection of air samples, for dusts, at the breathing levels and in close proximity to the workmen. Both of these forms of apparatus are adapted for this work, as they are transportable, easily set up and adjusted for the taking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



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, exterpolation 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 per cent. 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.

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.

TABLE I

Dept.	PLANT A		PLANT B		PLANT C		PLANT D		PLANT E	
	No. Samples Taken	Million Particles under 10 μ 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	½ to 1½	9	½ to 8	8	2 to 43	—	—	5	30 to 82
ard room	5	½ to 1 2/5	5	¾ to 4¾	6	5½ to 15½	—	—	5	28 to 78
ing frame pinning room	2	½ to ¾	—	—	—	—	—	—	—	—
ule spinning	2	2/3 to 2½	5	1/5 to 1	5	1¼ to 4½	—	—	5	5½ to 10
visting	10	1/3 to 1¾	—	Included in spinning	8	1 to 5¾ (includes broad loom weaving)	—	—	5	6 1/3 to 7 (includes broad loom weaving)
eaving	4	1/20 to 2	4	1/3 to 4/5	6	5¾ to 10	15	1 to 7	5	10½ to 19¾
elt Dept.	6	1/3 to 44½	—	—	—	—	—	—	—	—
Sheet in-sulating	5	1/3 to 4	—	—	—	—	—	—	—	—
Molded brake bend and clutch	—	—	—	—	—	—	2	½ 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.

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.

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

	Positive	Doubtful	Negative
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

	Positive	Doubtful	Negative
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.

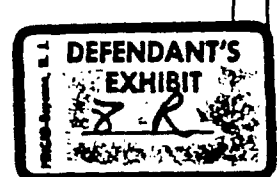


TABLE V

By Departments

	Positive	Doubtful	Negative
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

ASBESTOSIS (BY STAGES)

Third Degree Stage of Asbestosis

None Found

Second Degree Stage of Asbestosis

Case No.	Dept. of Plant	Yrs Exposure	Sex	Age	X-ray	Chest Findings	Chest Expansion	Symptoms	Complications
1	Carding	45	M	59	Marked fibrosis both lungs; generalized mottling.	Rales, dullness at bases	1 inch	Dyspnoea; cough (dry); excessive rate	—
2	Sewing	40	F	63	Marked fibrosis and mottling, both lungs.	(not examined, has not worked for past 10 yrs.)	—	Dyspnoea; expectation; poor eating appetite; frequently vomits before breakfast.	Pulled thr. left apex
3	Spinning	25	M	36	Fine mottling both lungs. Hilum shadows enlarged.	Rales at bases	2 inches	Dyspnoea, history of chest trouble.	—
4	Carding	20	F	51	Hilum shadows enlarged; general haziness and dense mottling both lungs.	Heart enlarged	1½ inches	Dyspnoea, history of sinus trouble.	Heart disease
					First Degree Stage of Asbestosis				
1	Spooling	4	F	19	Hilum shadows enlarged; early mottling both lungs.	—	2 inches	Dyspnoea, cough (dry); slight cyanosis.	—
2	Trucking	6	M	60	Several calcified spots; fine mottling both lungs.	Rales posteriorly at bases	2 inches	Cyanosis, leaden hue	—
3	Spinning	12	M	23	Considerable mottling both lungs.	—	3½ inches	Dyspnoea; history of chest trouble; asbestos corns.	—
4	Weaving	5	M	42	Uniform mottling both lungs.	Rales at bases; increased voice sounds.	2 inches	Dyspnoea (slight); cough (with expectoration); scaling around nails.	—
5	Carding	5	M	26	Fine mottling, particularly right side.	—	2 inches	Dyspnoea (slight)	—
6	Loom fixing	15	M	32	Fine mottling both bases.	—	2½ inches	Cough (dry) pronounced on deep inhalations; leaden hue; asbestos corns.	—
7	Weaving	4	M	31	Generalized mottling throughout both lungs.	Rales at bases.	1½ inches	Dyspnoea	—
8	Sewing	12	F	36	Fine mottling both bases; hilum shadows enlarged.	—	1 inch	Dyspnoea; cough (with expectoration).	—
9	Spooling	4	F	29	Peribronchial thickening; fine mottling in both lungs.	—	1½ inches	Dyspnoea (slight), cough (dry).	—
10	Spinning	6	M	34	Small amount of mottling generalized throughout both lungs.	—	2 inches	Scaling of skin around nails.	—
11	Spinning	4	F	41	Considerable thickening and early mottling both lungs.	—	1½ inches	Cough (dry)	—
12	Twisting	5	F	33	Peribronchial thickening; general haziness in both bases.	—	2 inches	None	—
13	Sewing	11	M	27	Fine mottling both lungs; heart enlarged.	—	2 inches	Nervousness.	—
14	Winding	4	M	23	Some thickening and mottling in both lungs.	—	2½ inches	None	—
15	Loom fixing	9	M	54	Some thickening and early mottling in both lungs.	Heart enlarged.	1½ inches	Cough (dry); cyanosis (slight); leaden hue; asbestos corns.	—
	Shuttle	4	M	29	Some thickening and mottling in both lungs	—	2 inches	None. Asbestos corns	—
—	Twisting	4	M	45	Conspicuous bronchial thickening and peri, [illegible]	Increased voice sounds; diminished [illegible]	3 inches	Dyspnoea (slight); asbestos corns	—

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18	Carding	4	M	41	Peribronchial thickening and mottling, particularly bases.	Dry rales posteriorly at bases.	½ inches	Dyspnoea; swelling of skin around nails.	---
19	Weaving	6	M	55	Considerable early mottling in both lungs.	Scattered rales; diminished breath sounds.	1 inch	Dyspnoea; cough (dry); history of chest trouble.	Healed tbc. right apex.
20	Weaving	3	M	47	Peribronchial thickening and early mottling both lungs.	Not examined.	Not taken	Not questioned.	---
21	Weaving	4	M	34	Peribronchial thickening and mottling both lungs.	---	2 inches	None	---
22	Weaving	11	M	41	Increase in both lungs.	Heart enlarged; heart murmurs; increased voice sounds	1½ inches	Dyspnoea (slight)	Heart enlarged
23	Carding	15	M	42	Peribronchial thickening both lungs; mottling.	Scattered rales.	1 inch	Dyspnoea; cough on inhalation.	---
24	Picking	18	M	39	Peribronchial thickening and mottling both bases.	Dullness left apex.	2½ inches	Dyspnoea (slight); cough (with expectoration).	Slight tbc. left apex; tuberculosis of sacculus (right shoulder)
25	Winding and stock room	16	M	53	General mottling both lungs, density right apex.	Dullness rt. apex; diminished breath sounds.	1 inch	Dyspnoea; cough on inhalation.	Tbc. right apex. Probably active.
26	Weaving	7½	M	39	Fine mottling especially on left side.	Dullness right upper lobe.	2 inches	Dyspnoea; cough (dry)	---
27	Spooling	4	F	45	Heavy and dense shadows both bases.	---	1½ inches	Dyspnoea (slight); cough (with expectoration); swelling of skin around nails.	Heart disease (mitral regurgitation).
28	Weaving	8	M	46	Some peribronchial thickening and mottling both lungs.	Not noted.	---	---	---
29	Weaving	5	M	33	Peribronchial thickening and some mottling.	Diminished breath sounds.	1½ inches	Cough (dry); poor appetite morning.	---
30	Spinning	6	M	38	Fine mottling in both bases, particularly right.	Slight dullness at bases.	2 inches	Has lost weight; swelling of skin around nails.	---
31	Carding	3	M	21	Considerable thickening and mottling both lungs.	---	2 inches	None	---
32	Picker	5	M	46	Some thickening and mottling in both lungs, particularly right.	---	1½ inches	Dyspnoea; cough (dry).	---
33	Carding	3	M	23	Peribronchial thickening and mottling both lungs.	---	3 inches	Slight cough (with expectoration).	---
34	Winding	7	M	21	Shadows enlarged; peribronchial thickening both lungs.	---	2 inches	Slight cough (dry); swelling of skin around nails; asbestos corns.	---
35	Spinning	8	M	33	Considerable peribronchial thickening and mottling both bases.	Heart enlarged; dullness left base.	1½ inches	Cough (with expectoration).	---
36	Weaving	7	M	30	Hilum shadows show several calcified spots in both lungs, some mottling.	---	1½ inches	Dyspnoea (slight); cough dry.	---
37	Carding	8	M	36	Shadows enlarged; fine mottling both lungs.	Heart enlarged.	2½ inches	Dyspnoea; cough (dry); history of colds; poor appetite.	---
38	Carding	5	M	29	Shadows enlarged; fine mottling both lungs.	---	2 inches	Dyspnoea; cough (dry); swelling of skin around nails; asbestos corns.	---
39	Grinding	4½	M	21	Shadows enlarged; rather general mottling both lungs.	---	2 inches	Dyspnoea (slight); asbestos corns.	---
40	Carding	3	M	23	Calcified spots in both hilum shadows; fine mottling both lungs.	---	2 inches	Dyspnoea (slight); cough (dry).	---
41	Spinning	3½	M	25	Hilum shadows enlarged; small amount thickening and mottling both lungs.	---	3 inches	Dyspnoea (slight); asbestos corns.	---
42	Assorting	3	M	39	Fine mottling in both lungs.	Rales at bases; diminished breath sounds.	1 inch	None	Heart disease (mitral regurgitation).
	Loom fixer	8	M	44	Hilum shadows enlarged; some mottling throughout.	Increased voice sounds.	2 inches	Dyspnoea; cough (with expectoration);	---

	ing							loaden hue; asbestos corns.	
44	Carding	7	M	33	Thickening and mottling throughout both lungs.	Dullness right base; diminished breath sounds.	1½ inches	Dyspnoea (slight); cough (with expectoration); leaden hue.	Nodes of the.
45	Weaving	10	M	32	Some thickening and mottling, particularly at bases.	—	1½ inches	Dyspnoea; cough (dry); slight cyanosis; history of chest trouble.	asthma.
46	Twisting	7	F	33	Peribronchial thickening and mottling both lungs.	—	1½ inches	Dyspnoea (slight).	—
47	Spinning	8	M	39	Peribronchial thickening and mottling both lungs.	—	1½ inches	None	—
48	Several different depts.	15	F	47	Hilum shadows enlarged; generalised mottling both lungs.	Scattered râles; diminished breath sounds.	1 inch	Dyspnoea; cough (dry).	—
49	Spinning	6	M	48	Peribronchial thickening in both lungs; generalised mottling	Dullness at bases.	2½ inches	Cough (with expectoration); history of chest trouble.	—
50	Carding	7	M	37	Hilum shadows enlarged; considerable peribronchial thickening throughout.	—	2 inches	Cough (dry); swelling of skin around nails.	—
51	Carding	6	M	34	Some thickening and mottling both lungs.	—	2½ inches	Cough (dry); history of chest trouble.	—
52	Picker	5	M	42	Peribronchial thickening and mottling both bases.	Râles at bases; some dullness.	2 inches	Dyspnoea (slight); poor appetite; asbestos corns.	Tumor on neck.
53	Carding and brushing	8	M	34	Considerable peribronchial thickening and mottling both lungs.	—	3 inches	Cough (with expectoration).	—
54	Elevator operator	9	M	48	Mottling throughout both lungs; some calcified spots.	Dullness at bases; diminished breath sounds.	1½ inches	Emaciation; some dyspnoea	—
55	Weaving	12	M	35	Right hilum enlarged; considerable mottling both lungs.	Râles and dullness at right base.	1 inch	Dyspnoea; cough (dry); poor appetite (morning); swelling of skin around nails.	—
56	Reeler (forming)	16	M	44	Considerable mottling both bases.	Diminished breath sounds.	2 inches	None	Aortic aneurysm
57	Weaving	8		41	Considerable peribronchial thickening and mottling both lungs.	Increased voice sounds at bases.	1 inch	Losing weight; leaden hue; cough (dry).	—
58	Weaving	9	M	38	Some mottling and peribronchial thickening both lungs.	Râles right apex.	2 inches	Cough (dry); swelling of skin around nails; asbestos corns.	—
59	Weaving	12	M	32	Peribronchial thickening and mottling, particularly at bases	Slight dullness at bases.	2 inches	Dyspnoea; cough (dry); swelling of skin around nails.	Nodes the.
60	Weaving	10	M	50	Marked hilum involvement, slight peripheral.	Diminished breath sounds.	2½ inches	Dyspnoea; cough (with expectoration); lost weight; history of chest trouble.	Nodes the. apex.
61	Twisting	15	F	37	Rather marked mottling at bases.	—	2½ inches	Dyspnoea (slight); cough (with expectoration); history of chest trouble.	—
62	Carding	5	M	44	Generalised mottling both lungs.	Râles posteriorly, some dullness.	2½ inches	Dyspnoea; cough (dry); poor appetite (morning); history of chest trouble.	Heart disease.
63	Weaving	12	M	36	Hilum shadows enlarged; mottling at bases.	—	1 inch	Dyspnoea; cough (dry); slight clubbing of fingers.	Nodes the.

26	Grinding	4	M	-?	Some generalized [illegible]			slight loss of appetite; lost [illegible]		
27	Carding	6	M	42	Some peribronchial thickening both lungs near bases.	—	1½ inches	Dyspnoea; cough (dry)	—	
28	Carding	1½	M	22	Fine mottling scattered in both lungs.	—	3 inches	None. Asbestos corns.	—	
29	Weaving	7	M	37	Considerable thickening in both lungs.	—	½ inches	Swelling of skin around nails.	—	
30	Weaving	9	M	45	Several haziness both lungs; fibrotic bands at bases.	Diminished breath sounds.	2 inches	Dyspnoea; cough (dry); slight emaciation; asbestos corns.	—	
31	Weaving	6	M	37	Small amount mottling at bases; right hilum enlarged.	Dullness at bases; diminished breath sounds.	1½ inches	Dyspnoea; cough (dry); poor morning appetite; lost weight.	—	
32	Weaving	10	M	45	Small amount thickening and mottling throughout both lungs.	—	1½ inches	Dyspnoea; poor morning appetite; swelling of skin around nails.	—	
33	Weaving	8	M	30	Some peribronchial thickening both lungs; very small amount [illegible]	Diminished breath sounds at bases.	2 inches	Dyspnoea; cough (dry); poor morning appetite.	—	
34	Weaving	6	M	37	Some peribronchial thickening; mottling in both bases.	—	2 inches	Dyspnoea (slight); cough (with expectoration); swelling of skin around nails.	—	
35	Spooling	6	M	53	Some peribronchial thickening and fine mottling both lungs.	Some dullness at bases.	2 inches	Dyspnoea; cough (with expectoration); clubbing of fingers and swelling of skin around nails.	—	
36	Weaving	9	M	32	Hilum shadows enlarged fine mottling throughout.	Slight dullness at bases; diminished breath sounds.	1½ inches	Dyspnoea; cough (dry); emaciation.	Pleural thickening.	
37	Spooling	5	M	58	Considerable peribronchial thickening and fine mottling.	Diminished breath sounds.	2 inches	Dyspnoea; cough (with expectoration); leaden hue.	—	
38	Twisting	5	F	19	Small amount peribronchial thickening fine mottling.	—	1½ inches	None.	—	
39	Carding	4½	M	21	Some mottling both lungs, hilum shadows enlarged.	—	2 inches	Slight cough (with expectoration).	—	

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DEFENDANT'S EXHIBIT

TABLE II

Doubtful Asbestosis (Classified as Negative)

Case No.	Dept. of Plant	Years of Expo-sure	Sex	Age	X-ray	Chest Findings	Chest Expansion	Symptoms	Complications
1	Reading	5	M	23	Small amount mottling; more fibrosis than normal.	--	2 inches	--	---
2	Trucking	3	M	18	Small amount thickening both lungs.	--	2½ inches	Slight cough (dry).	---
3	Reading	7	M	21	Some fine mottling; more fibrosis than normal.	--	2 inches	Cough (dry); swelling of skin around nails; asbestic corns.	---
4	Twisting	5⅓	M	33	Hilum shadows enlarged; more fibrosis than normal.	--	1½ inches	Dyspnoea (slight).	---
5	Spooling	6	F	30	Beginning mottling at bases; some peribronchial thickening.	--	1½ inches	Dyspnoea; cough (with expectoration); poor morning appetite, emaciation.	---
6	Spooling	5	M	21	Some peribronchial thickening; small amount mottling.	--	2½ inches	--	---
7	Reeling	4	M	22	Some early mottling and thickening both lungs.	--	2 inches	--	---
8	Legs flying	5	F	37	Hilum shadows enlarged; some mottling both lungs.	--	2 inches	Dyspnoea (slight); cough (dry); underweight; clubbing of fingers.	---
9	Seaming	1½	M	22	Hilum shadows enlarged; some peribronchial thickening.	Not examined.	--	--	---
10	Seaming	8	F	44	Calcified spots left hilum; some mottling both lungs.	Not examined.	--	--	---
11	Seaming	4	M	23	Hilum shadows enlarged; small amount mottling.	--	¾ inches	Swelling of skin around nails.	---
12	Seaming	6	M	24	Some thickening both lungs.	--	1½ inches	None.	---
13	Twisting	8	F	37	Hilum shadows enlarged; some mottling	--	1½ inches	None.	---
14	Spooling	23 (part time)	F	37	Hilum enlarged (right); small amount mottling both lungs.	Heart enlarged, mitral murmurs.	1 inch	Dyspnoea.	[illegible]
15	Spooling	6	M	22	Some thickening and mottling both bases.	--	2 inches	Cough (dry).	---
16	Carding	3	M	29	Some peribronchial thickening; early mottling right lung.	--	1½ inches	Cough (with expectoration).	---
17	Twisting	9	F	26	Hilum shadows enlarged; peribronchial thickening both lungs.	--	2 inches	Slight cough (with expectoration); emaciation.	---
18	Twisting	5	F	32	Some peribronchial thickening and mottling both lungs.	--	1¼ inches	None.	---
19	Carding	8	M	41	Some peribronchial thickening and early mottling both lungs.	Suggestion of fullness at bases.	2 inches	Cough (dry); emaciation.	---
20	General	10	M	42	Considerable peribronchial thickening and early mottling at bases.	--	2½ inches	None. Asbestic corns.	---
21	Seaming	7	M	31	Hilum shadows enlarged; some mottling both lungs.	--	2½ inches	Dyspnoea; swelling of skin around nails.	---
22	Spinning	13	F	33	Some fine mottling both lungs; hilum shadows enlarged.	--	1½ inches	Dyspnoea (slight); cough (dry).	---
23	Winding	4	M	28	Fine mottling both bases; right hilum enlarged.	--	2 inches	None	---
24	Winding	6	M	30	Fine mottling both lungs; hilum shadows enlarg	[illegible]art enlarged.	2 inches	Cough (dry); emaciation; swelling of skin around nails; asbestic corns.	---

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TABLE II

Narrative for Asbestosis

Case No.	Dept. of Plant	Years of Exposure	Sex	Age	X-ray	Chest Findings	Chest Expansion	Symptoms	Complications
1	Spinning	3	F	31	---	---	1½ in.	Cough (dry)	---
2	Carding	12	F	60	---	Diminished breath sounds.	1½ in.	Cough (dry)	---
3	Spooling	17	F	47	---	---	2½ in.	Slight cough (with expectoration); leaden hue swelling of skin around nails.	---
4	Weaving	4½	M	46	Pleuritic adhesions both bases.	Friction rub at base.	2 in.	Dyspnoea (slight)	---
5	Receiving	6	F	26	---	---	2 in.	None	---
6	Spinning	5	F	31	---	---	1½ in.	None	---
7	Spinning	6	M	24	---	---	2 in.	None	---
8	Spooling	4	F	29	---	---	1½ in.	None	---
9	Picker	3	M	27	---	---	2 in.	Dyspnoea (slight); cough (with expectoration; asbestos corn.	---
10	Jinning	3½	M	21	---	---	2 in.	Losing weight.	---
11	Picker	3	F	35	---	---	3 in.	Dyspnoea (slight); cough (with expectoration;	---
12	Waste	4	F	28	---	---	2½ in.	Slight cough (with expectoration).	---
13	Feeding	15	F	34	---	---	2 in.	None. Asbestos corns.	---
14	Grinding	3½	F	24	---	---	2½ in.	None.	---
15	Carding oiling	7 mos.	F	37	---	---	1½ in.	None.	---
16	Weaving	4	F	36	---	Slight dullness left side.	1 in.	Dyspnoea (slight); cough (with expectoration.	Pleuro-pericarditis
17	Carding	12	F	6	---	Rales (general) dullness; diminished breath sounds.	1½ in.	Dyspnoea; cough (with expectoration); emaciation; poor morning appetite.	Pooled the. (left apex) Chronic bronchitis
18	Weaving	18 mos. (intermittent)	M	45	---	Dullness at apices.	2½ in.	Dyspnoea; cough (dry); poor morning appetite.	Pooled the. (apices)
19	Weaving	3	M	39	---	Rales, dullness left apex.	2 in.	Dyspnoea; cough (dry); emaciation.	Tbc. left apex (possibly active)
20	Weaving	3	F	31	Density mid-section both lungs, calcified spots.	Rales (scattered)	2 in.	Dyspnoea (slight)	Case (?)

TABLE VI

Years of Exposure

	Positive	Doubtful	Negative
Over 15 years	13	1(part time)	3 (1 not
10 to 15 years	7	3	2 con-
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

	Positive	Doubtful	Negative
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

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

TABLE IX

Chest Expansion

	Positive	Doubtful	Negative
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

	Positive	Doubtful	Negative
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

	Positive	Doubtful	Negative
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."

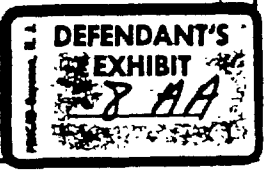
*At least six of these showed no evidence associated with asbestosis.
**Two of these not associated with usual signs found with asbestosis.
***Six of these not associated with usual signs of asbestosis.
**** "Miners' Consumption", U.S. Public Health Service Bulletin #85, January, 1917.

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.

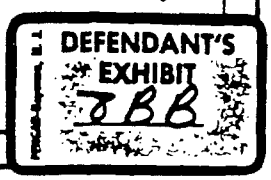
Total and permanent disability claims:

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.



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 (SiO_2).

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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