UNITED STATES TREASURY DEPARTMENT

PUBLIC HEALTH REPORTS

ISSUED WEEKLY

BY THE UNITED STATES
PUBLIC HEALTH SERVICE

VOLUME 50 :: :: NUMBER 1

JANUARY 4 - - - 1935

IN THIS ISSUE

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1935

For sale by the Superintendent of Documents, Washington, D. C. - - - - - - - - - Price 5 cents
Subscription price, $2 a year

PUBLIC HEALTH REPORTS

VOL. 50 JANUARY 4, 1935 NO. 1

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

A Preliminary Study

By A. J. LANZA, *Assistant Medical Director*, WILLIAM J. MCCONNELL, *Assistant Medical Director*, and J. WILLIAM FEHNEL, *Chemist*, *Metropolitan Life Insurance Co.*

INTRODUCTION

In 1929 the Metropolitan Life Insurance Co. was approached by officials representing the asbestos industry in the United States, who were desirous of ascertaining whether 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 problem (in the metal mining and certain other industries) in Great Britain, the United States, the British Dominions, and 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 (combined silica) 44.1 percent, magnesia 43 percent, and water 12.9 percent, 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 5 to 15 and even 20 or 25 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 length 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. It might well be assumed that similar variables would influence the occurrence of asbestosis.

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

The industrial health service of the Metropolitan Life Insurance Co. undertook the following investigation during the period from October 1929 to January 1931, which included:

1. A study of dust conditions in asbestos mines and mills in Canada and in fabricating plants along the Atlantic Seaboard in the United States.
2. Physical examinations of asbestos workers, including X-ray films.
3. A study of dust exhaust systems designed to eliminate asbestos dust.

Data on the fabricating plants only are included in this study and are designated in the report as plants A, B, C, D, and E. This is a preliminary report. A more extensive study of the asbestos industry is now under way.

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 a small rotary fan, run by a motor, the quantity being measured by a flow meter.

Samples of dust brushed from beams and pipe lines near the ceiling also were secured for chemical analysis. All samples were shipped to the industrial hygiene laboratory of the Metropolitan Co., and the dust counts were made according to the methods accepted by the United States Bureau of Mines and the United States 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).

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 tissu ; 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.[1]

Total particles per cubic foot and the corresponding weight of the dust in milligrams were obtained. Since no relationship between the dust counts and the corresponding weights was found, the weights are not included in the tabulations in this report.

Table 1 shows by plants and by departments the maximum and minimum dust counts in million particles per cubic foot of air. Although the counts in plant A are of particles 10 microns and under,

[1] Samples collected by both the impinger and the electric precipitator were counted. In addition, microphotographs of these dust particles were enlarged by being projected upon a screen with a lantern-slide projector, the enlarged particles being measured with a ruler. Knowing the entire enlargement (by actual 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.

and in the other plants are for all particles, 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.

TABLE 1.—*Maximum and minimum dust counts in million particles per cubic foot of air, by plant and department*

Department	Plant A		Plant B		Plant C		Plant D		Plant E	
	Number of samples taken	Million particles under 10μ per cubic foot of air	Number of samples taken	Million particles per cubic foot of air	Number of samples taken	Million particles per cubic foot of air	Number of samples taken	Million particles per cubic foot of air	Number of samples per cubic foot of air	Million particles per cubic foot of air
Preparation	5	½–1½	9	½–8	8	2 –43			3	30 –92
Card room	3	½–1⅜	5	¾–4¾	6	3½–15½			3	26 –76
Ring frame spinning room	2	½– ¾								
Mule spinning	2	⅜–2½	5	½–1	3	1¼– 4½			3	5½–10
Twisting	10	⅛–1¾		(1)	8	[2]1 – 3¾			3	6½– 7
Weaving	4	½o–2	4	½– ⅝	6	3¾–10	15	1 –7	3	10½–19¾
Felt department	6	½–4½								
Sheet insulating	3	½–4								
Molded brake band and clutch							2	½– ⅜		

[1] Included in spinning.

[2] Includes broad loom weaving.

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 comprises about 25 percent of the material used. On special jobs the percentage of asbestos may be 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 percent of the total material used. In the molded brake-lining and clutch division of plant D, asbestos comprises about 25 percent of the total material. The actual exposure to asbestos dust, therefore, is considerably less than is indicated in the dust counts in table 1.

CONDITIONS IN THE PLANTS

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

Asbestos is received in bags. These are emptied upon the floor and the asbestos is shoveled into pug mills and run for about 5 minutes in order to crush and open the fibers. From there the asbestos is shoveled into trucks and wheeled to hoppers with either horizontal or vertical openers, similar to those used in 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 to the openers, and the material is fed into these in the same manner as is the new material. After being discharged from the opener, the material is put onto a vibrating or shaking screen, where the long fibers are picked off by a suction hood and blown into a bin to be used again in textiles. The short fibers 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 fiber, the cotton, or the waste material are taken 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 taken by a mechanical conveyor 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. The third machine is not equipped with an oil spray. The object of the oil spray is primarily to entrap the small asbestos fibers and hold them enmeshed in the cotton fiber 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 percent of oil at this point; but by the time it reaches the looms, the oil had diminished to less than 1 percent.

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 plant B depended on natural humidity. 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 2 buildings, 1 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 percent. One plant, C, had a humidification system in the twisting department and also in the weaving department, where the relative humidity was 76 percent with a dry-bulb temperature of 69° F., as compared with a relative humidity of 44 percent in the weaving room of plant A.

Aside from plant E, 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 granite dust, established by the United States Public Health Service (7), namely, about 10 million particles (10 microns and under) per cubic foot. However, we are not justified in assuming that because available information suggests that asbestosis is a milder disease clinically than silicosis, the threshold of permissible dust counts is higher. Asbestosis appears to be pathologically different from silicosis, and the experience so far does not warrant an attempt to define a standard of dustiness for asbestos dust.

DUST CONTROL

Various measures, such as oiling, humidification, and local exhausts, tended to reduce the dust. Nevertheless, it was evident that they were only partly successful. 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 percent and with further alterations will probably be 75 percent effective. In this case such a reduction seemed quite satisfactory. It is neither practicable nor economically desirable to install such equipment as will make the air entirely dust free. 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 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 a needless expenditure of money.

PHYSICAL EXAMINATIONS

X-ray films were made of 126 persons (108 men, 18 women) working in asbestos plants in the United States. All but five of these were given physical examination. The cases were selected more or less at random from among those having more than 3 years of employment in the industry. It was soon obvious that the early diagnosis of asbestosis must rest to a large extent on X-ray pictures of the chest. As in the early stages of silicosis, the clinical symptoms of the asbestos workers were usually indefinite and inconclusive. The interpretations of these films are based on the readings of one competent roentgenologist, but they have been reviewed 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, taking into account the physical examination and the age of the individual, and were classed as positive only when there was no major disagreement. All examiners were guided by their experience in silicosis and other types of pneumoconioses. 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 the 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.

The cases of asbestosis were 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 [2] were actively engaged in factory work, and it was not practicable to make any distinction 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 cases were found.

Of the total of 126 X-ray examinations, 4 were diagnosed as second-degree asbestosis, 63 as first degree, 39 as doubtful, and 20 as negative.

There is a definite increase in the percentage diagnosed as positive in relation to the years of exposure, as shown in table 2. Small numbers make it impossible to determine how far the factor of age enters into this increase.

TABLE 2.—*Classification of cases by years of exposure*

Years of exposure	Percentage positive	Number		
		Positive	Doubtful	Negative
Over 15 years	87	13	[1] 1	[1] 3
10 to 15 years	58	7	3	2
5 to 10 years	50	30	25	3
Under 5 years	43	17	10	12
Total examined		67	39	20

[1] Part time. [2] 1 not continuous.

[3] One asbestos worker (second stage) retired of his own accord, because of old age, 10 years previous to this study, but is still active about his garden.

Of the 64 persons given physical examination and diagnosed as having positive asbestosis, only 8 were entirely free from symptoms, while 10 out of 37 with doubtful and 7 out of 20 with negative diagnoses were free from 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 statements of subjective symptoms. 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 a community with a silicosis hazard is noteworthy.

The incidence of tuberculosis (based upon X-ray films) is given in table 3; 40 of the 67 examined had less than 15 years of employment in the industry.

TABLE 3.—*Incidence of tuberculosis*

	Positive	Doubtful	Negative
Tuberculosis (healed)	7	1	2
Tuberculosis (active)	[1] 1	0	1
Total examined	67	39	20

[1] This case was diagnosed as probably active on the X-ray findings.

Table 4 gives information as to physical signs of chest trouble.

TABLE 4.—*Chest findings*

	Positive	Doubtful	Negative
Physical signs of chest trouble	[1] 33	[2] 9	[3] 7
No physical signs	31	28	13
Not examined	3	2	0
Total	67	39	20

[1] At least 6 of these showed no evidence associated with asbestosis.
[2] 2 of these not associated with usual signs found with asbestosis.
[3] 6 of these not associated with usual signs of asbestosis.

What significance, if any, can be attached to the presence of "asbestos corns" on the hands of workers appears doubtful, as 18 percent of the positives (12 out of 64) and 15 percent of the others (doubtful, 7 out of 37, negative, 2 out of 20) showed such corns.

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

Many workers 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.

INSURANCE CLAIMS

To throw 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 companies carrying group insurance and having available figures.

There were 2,099 lives involved, with a total exposure of 7,019 life-years. The death claims are so small in number that reliable conclusions cannot be reached 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, as compared with 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 6 of 36 death claims and 8 of 10 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 the hospital and sanatorium records, including available X-ray films, were investigated, with the following results:

Death claims:

1. A typist, not exposed to dust; died of pulmonary and intestinal tuberculosis.
2. Colored male, age 27; worked in asbestos 1 year and 8 months; died of pulmonary tuberculosis following a hemorrhage; was in sanatorium 10 months. Also had a four plus Wassermann. No evidence of asbestosis.
3. Colored male, age 32; worked in asbestos plant 1 year and 7 months; died of pulmonary tuberculosis after 1 month in hospital. Cavitation both lungs; no evidence of asbestosis.

4. Colored male, age 34; worked in asbestos plant 2 years and 6 months. His physician believes this was a case of uncomplicated tuberculosis. Was not inmate of hospital or sanatorium. No information could be obtained on the other two cases.

Total and permanent disability claims:

1. Male, employed in asbestos plant 3 years. His physician states that he first came under his observation with an old established case of tuberculosis about 2 years after asbestos employment started. Also had tuberculosis of the kidney and cervical glands.

2. Male, employed in asbestos plant 8 months. His physician states finding of old fibroid tuberculosis with tubercle bacilli in sputum. No X-ray available; but according to physician, asbestos bodies were found in sputum.

3. Male, 10 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 reexamined 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.

5. White male, 13 years in asbestos plant. Is now in sanatorium. An interesting case. His physician states that 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 deaths in death-claims cases appear to be due to uncomplicated tuberculosis; three of them were Negroes, who were probably tuberculous at the time their employment in the asbestos plant commenced.

Of the 8 disability claim cases, 1 was uncomplicated tuberculosis and 2 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.[3]

CONCLUSIONS

1. Prolonged exposure to asbestos dust caused a pulmonary fibrosis of a type different from silicosis and demonstrable on X-ray films. Clinically, from this study, it appears to be of a type milder than silicosis.

2. Cases of definite cardiac enlargement were frequently found to be associated with asbestosis.

[3] Personal communication.

3. A predisposition to tuberculosis due to asbestos dust was not indicated in this study.

4. Asbestosis as observed in this series of cases had not resulted in marked disability in any case.

5. It is not known how much asbestosis may add to the mortality of pneumonia and acute nontuberculous pulmonary infections.

6. It is not practicable as yet to establish standards for the asbestos dust content of air.

7. The amount of dust in the air in the asbestos plants studied can be substantially reduced.

RECOMMENDATIONS

It is recommended—

1. That the industry seriously face the problem of dust control in asbestos plants.

2. That new employees be examined physically, including X-ray examination of the chest, and rejected for employment if they show tuberculosis or pneumoconiosis.

3. That employees be examined physically, preferably every year, but at least every 2 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.

ACKNOWLEDGMENTS

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. F. V. Meriwether, of the United States Public Health Service, surgeon in charge of the United States Bureau of Mines Cooperative Clinic in Picher, Okla., whose interpretation of all the X-ray films listed in this report is followed. Appreciation is also expressed to Dr. W. Atmar Smith, and Dr. Kenneth Lynch, of Charleston, S. C., for information and advice, particularly as to the pathology of asbestosis;[4] 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; and to Dr. L. U. Gardner and Mr. Donald E. Cummings, of the Saranac Laboratory, who have undertaken the study of the pathological effects of the inhalation of asbestos dust

[4] Asbestosis bodies in sputum and lung. By Kenneth M. Lynch, M. D., and W. Atmar Smith, M. D. Jour. Am. Med. Assoc., Aug. 30, 1930, vol. 95, no. 9, pp. 659-661.

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ENDEMIC TYPHUS IN ALABAMA[1]

By J. N. Baker, M. D., James G. McAlpine, Ph. D., and D. G. Gill, M. D., D. P. H., *Alabama State Department of Health, Montgomery, Ala.*

I. INTRODUCTION

In a preliminary report made in June of this year before the Conference of State and Provincial Health Authorities, held in Washington, the authors discussed the epidemiological aspects of endemic typhus as it occurs in southern United States. In that report it was noted that there had been a rapid increase in this disease in certain Southern States, as is shown in table 1. That other countries have experienced a similar rise in incidence is evidenced by the figures in table 2.

The distinction between epidemic typhus and the endemic typhus of this country was first recognized in 1898 by Brill. He (1) found in the United States a type of fever which, resembling typhoid, gave a negative Widal reaction. In further studies he (2, 3) demonstrated its similarity to typhus, but showed that it was milder in character and less contagious, only one case as a rule being found in a household. Also he reported that it was most prevalent during the fall of the year instead of late winter or spring. In 1912 Anderson and Goldberger (4) proved that Brill's disease was immunologically identical with Mexican typhus, or tabardillo. Naturally this led to the belief that it was louse borne.

[1] Read before the laboratory section of the American Public Health Association, Pasadena, Calif., Sept. 3, 1934.