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## YELLOW FEVER AT RIO DE JANEIRO

During the second quarter of 1928 yellow fever reappeared at Rio de Janeiro, Brazil, in all probability imported from the northern States. The first case occurred on May 12, 1928, in a soldier stationed at the army barracks located in a suburb of the city.<sup>1</sup> Up to October 6, 1928, there had been reported in Rio de Janeiro 119 cases of yellow fever, with 66 deaths. From that date until January 12, 1929, there were reported only 7 cases, with 4 deaths; but beginning in January the prevalence began to increase, and during the period January 13 to June 1, 1929, there have been reported 615 cases, with 351 deaths.<sup>2</sup>

Following are extracts from a report relative to the new epidemic increase and the control measures employed, received by the Director of the International Office of Public Hygiene from the Director General of Public Health of Brazil and transmitted by the Director of the International Office under date of April 15, 1929:

The yellow-fever curve is rising during the summer months. The conditions most favorable to the development of the disease are naturally those most favorable to the life and multiplication of the mosquito carriers. The constant high degree of temperature and frequent rains constitute the greatest obstacles to the campaign against the dangerous insect carrier of yellow fever. The development of the mosquito, which normally takes, on an average, 12 days, is reduced to 7 or 8 days, which makes the combat more difficult, because if a shelter of larvæ is overlooked in the weekly visit the mosquitoes necessarily reach winged form before the next periodic visit.

In addition to this, the mosquitoes, driven out of houses, lay their eggs in the eaves of the houses, in the hollows of trees, on the ground, and in any more or less remote place where there is a small quantity of water.

The difficulties are redoubled in a large city like Rio de Janeiro, having a large residential section, of which the great majority of the homes are surrounded by flower gardens, parks, and vegetable gardens. And it must not be forgotten that the present population has already lost its former immunity; about 80 per cent of the inhabitants of the city are susceptible to yellow fever, which shows that the infected

<sup>1</sup> See Public Health Reports for Nov. 23, 1928, p. 3079.

<sup>2</sup> See p. 1734.

mosquito, however rare, will almost certainly find a nonimmune individual to whom it will transmit the disease.

The work is indeed difficult. We have in service about 6,000 men in charge of weekly visits in the entire Federal district. Besides, the streets and sections where a case of yellow fever has been reported receive two visits a week to permit of the discovery of any focus which may have escaped the investigation and which should be found before the complete development of the mosquito. These visits are made by a review squad, made up of other elements than those in the regular squads.

The epidemic increase of the summer was foreseen, since it must necessarily have resulted from conditions favorable to the development of the disease. In six months of prophylactic work we have not had the time necessary to exterminate the disease. The recrudescence during the summer months was inevitable, considering the duration of the life of the mosquito in the adult state, on an average, 60 days. Another epidemiological condition important in the propagation of the disease is the existence of abortive forms, which are unnoticed by the most cautious practitioners, and serve, therefore, to maintain the disease and transmit it to other individuals.

The localization of the cases of yellow fever in the present outbreak reaches the most remote places, suburban sections and rural zones, where the centers of population are to-day very dense. During the current month [April, 1929] the number of cases has increased in the S. Christovão and Andarahy sections.

Cases are again appearing in a part of the commercial center, especially the small foreign commerce center, which is explained by the fact that the inhabitants of the remote districts and suburbs work in these places.

In spite of the unfavorable conditions which we have had to face, the cases of yellow fever have been limited, if we consider the figures, obviously small for a population of about 2,000,000 people. An average of 1 case per day in January, 2 cases in February, and 6 in March, including suspected cases, is indeed unimportant, considering the percentage of nonimmune persons which form almost the entire cosmopolitan population of Rio de Janeiro. It must be stated that in the present figures not only the confirmed cases but also those merely suspected are considered. In the total deaths are included all the cases of clinical diagnosis, whether or not confirmed by autopsy.

The danger of ship contamination is almost nonexistent, even if ships come alongside the quay, because there are no houses or mosquitoes in this zone, the service here being very strict, consisting not only of destruction by police of foci in the larval stage but also of a campaign against adult mosquitoes.

The small boats are also under control along the quay and are kept under careful supervision. Tourist boats have also come alongside the quay without being disturbed by mosquitoes and without a case of yellow fever up to the present time. Specific prophylactic service is intensively carried on in the entire urban, suburban, and rural districts and in the neighboring towns of the State of Rio, with about 6,000 men carrying on the daily work of hunting the larva shelters and in the work of disinfecting in the campaign against the adult insect in sections where houses have become foci. Disinfection is carried on with sulphur or with insecticides, which are brought to the interior of the houses by powerful dusting machines. There have actually been 400 houses disinfected per day in this manner at Rio.

The formula used by the Public Health Service of Brazil contains petroleum, carbon tetrachloride, and pyrethrin, and it was used only after careful experiments had been made to ascertain its efficacy.

In addition to these measures, two others complete the prophylactic campaign—the isolation of cases and the sanitary supervision of sections where there have been cases, following the radius of action of the mosquito carrier.

The campaign against yellow fever extends to-day regularly to all sections of the country where the disease has been reported. The campaign in the northern part of the country is conducted with the collaboration of the Rockefeller Foundation, under the technical and administrative guidance of the Director of the National Department of Public Health.

Doubtless the reduction in cases at this period of great danger confirms the efficacy of the fight against the disease. The Brazilian Sanitary Service hopes to overcome yellow fever again at Rio de Janeiro, having in this prophylactic campaign the advantage of having before reduced the fear of yellow fever, it having been proved that, with the application of prophylactic measures, the disease does not spread in epidemic form in an extended radius.

The Government is willing that financial resources shall not be spared and that the sanitary campaign shall come to an end only after it is certain that yellow fever is no longer present in Brazil.

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## CURRENT STUDIES OF UNDULANT FEVER<sup>1</sup>

By H. E. HASSELTINE, *Surgeon, United States Public Health Service*

For the purposes of this discussion the term "undulant fever" includes Malta fever and all other febrile disturbances caused by infection with a member of the bacterial genus *Brucella*.

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<sup>1</sup> Read at the Twenty-seventh Annual Conference of State and Territorial Health Officers with the United States Public Health Service, Washington, D. C., June 3, 1929.

Twenty-four years ago undulant fever made its début into American medical circles when Craig reported the first case of Malta fever in the United States. The formal presentation of this newcomer contained the following statement: "I am convinced that a careful study, by use of the Widal test and the agglutination reaction with *micrococcus melitensis*, of many of the cases of obscure continued fevers which are prevalent in this country will result in the demonstration that Malta fever is by no means a rare disease in the warmer portions of the United States, and that many of the so-called anomalous cases of typhoid fever are in reality instances of infection with the organism of Malta fever." At that time fevers were so prevalent that the newly reported stranger was soon forgotten and remained so until Ferenbaugh and Gentry recognized the disease in the goat-raising sections of Texas. Even then it was considered only of passing interest.

In 1918 Evans called attention to the fact that the organism causing Malta fever was practically identical with that causing infectious abortion of cattle. This announcement caused numerous laboratory investigators to repeat the work of Miss Evans and, when it was confirmed, there was speculation as to infection of human beings with the organism of infectious abortion until Keefer (in 1924) reported a case of Malta fever which was proved by positive blood culture to be due to the *abortus* organism. Since 1926, each year brings an increasing literature on the subject and a large number of case reports.

On the whole, the reaction of the medical profession in seeking information on the disease has been most encouraging. Nearly every day brings an inquiry on some phase of the subject. With a view to assembling the information that might be of help to the medical profession and to health authorities, I have undertaken the collection of data on the clinical and epidemiological aspects of the disease.

As to the amount of undulant fever in the United States, we have no reliable statistics, as the disease is not reportable in some States, and has been reportable only a short time in others. Laboratory workers have conducted serological surveys by applying the agglutination test using *Br. abortus* as antigen, to all serums submitted for Wassermann or Widal tests. The results have been quite variable in different States, owing partly to a difference in the agglutination titer that each laboratory requires before a positive result is recorded, and partly to a true difference in prevalence. These results vary from 0.6 per cent to 7.5 per cent according to the titer considered necessary for a positive report.

In a questionnaire sent to different State laboratories Hardy collected information up to January 1, 1929. In 1928, 40 States reported 560 positive results; in 1927, 18 States reported 194 positive; in 1926, 7 States reported 34 positive. These figures represent persons whose

serum showed agglutinins for *Brucella melitensis* or *Brucella abortus*. An attempt to ascertain the number of clinical cases reported in the literature since January 1, 1927, has been only partially completed, but has revealed over 300 cases reported. How many cases have passed unrecognized, or recognized and not reported, we do not know.

May I take this opportunity to urge upon all of you that, if you have not already done so, you place undulant fever on the list of reportable diseases in order that we may have official statistics as to its prevalence? And may I suggest that each State health department issue a leaflet of information to the physicians of their respective States giving a brief description of the disease?

Among other diseases that may be confused with undulant fever are typhoid and paratyphoid fever, tuberculosis, rheumatism, malaria, influenza, focal infections, sinusitis, appendicitis, and tularæmia. Hoping to be able to give the profession at large assistance in recognizing the disease, I have undertaken to obtain a clinical history of a large number of cases. While my study has not progressed sufficiently to warrant a report, I will offer the following sketch of the salient points for consideration in the diagnosis of undulant fever.

The first symptom usually is a general feeling of weakness in the afternoon, accompanied by headache, or general aching. The patient then discovers that he has fever, which is usually preceded by chilliness, or a definite rigor, followed by a hot stage and a rather profuse perspiration, particularly noticed upon awakening. If questioned closely, most patients will report that the sweating is limited to the upper half of the body. It is quite common for patients to feel so well in the morning that they want to go to work; but if they do, they are ready to quit early in the afternoon. When seen in the febrile stage the patient appears only slightly ill; and if his temperature is taken, it will be surprising to find it  $1^{\circ}$  to  $3^{\circ}$  higher than the general appearance of the patient would indicate. The daily variation is usually marked, being at or near normal in the morning, and going to  $102^{\circ}$  or  $104^{\circ}$  in the afternoon. If taken every four hours, a rather irregular curve within each day is frequently noted. The blood picture usually shows a slight anemia, a slight leucopenia, and a considerable increase in the lymphocytes, with a corresponding reduction of the polymorphonuclears. After the fever has risen by irregular steps to a maximum of  $103^{\circ}$  to  $104^{\circ}$ , occasionally higher, it usually declines by lysis, and remains normal for a period varying from three days upward. Then another febrile wave begins, usually not quite so severe, nor quite as long as the first, followed by an afebrile period slightly longer than the first. Some cases have but one febrile wave, others have them irregularly over a period of one to three years. The general symptoms of the disease come and go with the febrile wave. Joint pains, especially upon getting about after the fever has returned

to normal, are present in most cases. They vary from slight pain to moderate interference with the function of the joints. Anorexia, constipation, insomnia, and increased irritability are present in a majority of cases.

Among complications reported, the most prominent are orchitis, or epididymitis, and abdominal conditions caused by a localization of the infection in the viscera, particularly in the female reproductive organs. Prostatic symptoms have been noted in the male, and abortion in pregnant women has been attributed to the infection on frequent occasions.

It is too early to know whether the infection may cause any late sequelæ, such as sterility, which the disease is known to cause in a portion of the domestic animals that it attacks.

We know comparatively little of the pathology of the infection in human beings. Whether the infection with the types of *Brucella abortus* differs in its pathology from that of *Brucella melitensis* is not yet known.

For diagnosis, blood serum should be submitted for an agglutination test against *Brucella abortus* as well as against the typhoid organism. The first specimen should be taken about a week after the onset of fever; if negative, a second specimen should be forwarded. I recommend the drawing of 10 cubic centimeters of blood, under sterile precautions, placing it in a suitable sterile container, allowing it to clot, and then forwarding the whole specimen to the laboratory. In the laboratory the serum may be separated for serum tests and the clot planted in proper media in an effort to grow any organism that may be present. The diagnosis should always be confirmed by a positive agglutination test, and preferably by blood culture, if possible. A positive agglutination test in 1:80 dilution, or higher, in a case presenting clinical symptoms of undulant fever warrants a diagnosis even in the absence of positive blood culture.

The obtaining of blood cultures is important in order that the strain of the infecting organism may be determined. At the present time it appears that there are at least three strains of the *Brucella* genus that may cause undulant fever. These are known as the caprine, bovine, and porcine strains. Huddleson has assigned to each a specific name, the caprine strain being *Brucella melitensis*, the bovine strain *Brucella bovis*, and the porcine strain *Brucella suis*. If this nomenclature should be accepted, it will avoid some confusion; for the adjective used to specify a strain does not necessarily denote its source. Apparently cattle can be infected with all three strains, so a human case contracted from cattle may be due to any of the three strains. There is no record of swine being infected naturally with the bovine or caprine strains. Close study of the bovine and porcine strains of *Brucella abortus* show distinct difference

in cultural, biological, and pathogenic characteristics, which seem likely to result in these two being ultimately considered two distinct species.

Several methods of differentiating these strains have been devised. McAlpin and Slanetz accomplish it by chemical studies of the metabolic activities of the organisms; Huddleson, by the use of certain growth-inhibiting dyes; and Theobald Smith and others by the use of guinea pigs and the study of the pathology in the test animals. Fortunately, all three of these methods seem to check reasonably closely; and if your laboratories are not able to do differential tests I can assure you that any, or all, of the above-mentioned workers will be glad to have your cultures forwarded to them for type differentiation.

Huddleson gives the following results of his work on the typing of the different strains:

Of 96 cultures from cattle, 86 were bovine, 8 porcine, and 2 caprine.

Of 20 cultures from swine, 20 were porcine.

Of 2 cultures from horses, 1 was bovine, 1 porcine.

Of 15 cultures from goats, 15 were caprine.

Of 46 cultures from man (excluding laboratory infections and cases from the Southwest United States), 21 were bovine, 25 porcine.

Huddleson also reports that in monkeys the porcine strain is the most highly pathogenic, the caprine strain is next, and the bovine is the least pathogenic of the three. It was not easy to infect monkeys with the bovine strain; but when infected and subsequently recovered, these animals showed immunity to all three strains.

On the epidemiological side I find that but little organized investigation of cases has been done by State departments of health. To the questionnaire sent out by Hardy, 12 States replied that epidemiologic studies had been made. I have not visited all the States but find that in some the epidemiologic studies have not been made by the epidemiologist, or representative of the bureau of communicable diseases, but by the attending physicians or laboratory workers who have been consulted in establishing the diagnosis. These attempts, made by well-meaning individuals whose experience in epidemiology is limited, have frequently lacked information on vital points. I hope to be able to visit each State that has an undulant-fever problem and confer with the epidemiologist regarding work on this disease.

The fact that British soldiers and sailors seemed to get Malta fever from goat's milk has probably led us to believe that most undulant fever is contracted through milk. While a goodly proportion of cases in the United States have no other reasonable explanation, there are

many cases, especially in rural districts, that may be due to contact with infected animals. For instance, a farmer may have infectious abortion present in his cows and he uses a portion of the milk produced on his place. There is a tendency to attribute such farmer's attack of undulant fever to infected milk. But the fact that in rural sections the fever attacks about five males to one female suggests that the men are more exposed to infection than the women. This is brought up not as an argument that the disease may not be milk-borne, but to emphasize the point that we should consider all possible sources of infection, particularly when two or more exist in connection with any individual case.

There are many cases occurring in urban communities, and in persons following occupations that do not bring them in contact with animals, that seem to offer no possible explanation other than milk-borne infection. In support of this mode of spread, the strongest trio of cases of which I have knowledge included a physician on the staff of a tuberculosis sanatorium and two women patients who had been bed patients for approximately a year. Milk was produced from a herd belonging to the institution and used in the raw state. The physician was not concerned with the administrative side of the institution and did not come in contact with the farm animals, nor did he do laboratory work. The physician developed typical undulant fever, and the two patients showed a definite febrile exacerbation without evidence of increased activity of their tuberculosis. The blood of all three gave positive agglutination tests against *Br. abortus*.

In the group whose occupation does not furnish a clue to the probable method of acquiring the infection, it is found that practically all have used unpasteurized milk within a sufficiently recent period to account for the disease. In the large cities where a high percentage of the milk supply is pasteurized the disease is comparatively rarely reported. A relatively small group of cases acquire the infection in the laboratory. I think all of the cases reported in the District of Columbia can be traced to probable laboratory infection.

The ideal method of prevention of the disease is the elimination of the infection from livestock, a goal which veterinarians and the Department of Agriculture hope can be reached by separation of infected from noninfected herds. It appears that after one or two abortions cows develop sufficient resistance to enable them to carry their calves to full term. These calves do not harbor the infection for any great period, and if transferred to noninfected environment before reaching the age of sexual maturity will be found to be free from the infection. The segregated infected animals may become free from infection. Frequent observation and testing by competent veterinarians is required to determine this.



Any such program as that outlined above is going to take at least several years to accomplish. In the meantime, what should the health officer do to protect the health of his community? We may require pasteurization of milk in urban centers, but we will not get a very effective pasteurization in rural sections. We may get certain families to boil it. Those who seem to be exposed to the infection as a result of their occupation must be taught the danger to which they are subjected, just as physicians and nurses have learned of the dangers to themselves arising from their work. In order to do this we should have more detailed epidemiological knowledge of the individual cases. We have no definite proof of transmission from one person to another. Amoss has reported the recovery of *Brucella abortus* from the stools of patients suffering from the infection. In view of this we must recognize the possibility that the disease may be disseminated through contact infection.

Among known facts on which more data are desired to explain certain phases of the question are the following: There are at least four cases in males to one in females. We know there is not such a disproportion in the sexes in the general population. Are males more susceptible than females? Or does the theory of greater exposure by reason of occupation explain it? The latter seems to be the most rational explanation, but health officers, like our good friends from Missouri, like to be shown.

The disease is relatively rare in children below 12 years of age. In this group we would expect to find many cases if the disease is milk-borne. Does the disease show symptoms in the child so variant from those found in adults that we fail to recognize it? Is the child actually less susceptible? Huytra and Marek in their *Pathology and Therapeutics of the Diseases of Domestic Animals* state that surviving calves born of infected mothers rapidly eliminate the *Br. abortus* and that it seems difficult, or impossible, for the organism to maintain itself in sexually immature animals. The susceptibility of young cattle increases with the approach of sexual maturity. This suggests that possibly children are less susceptible than adults; but here again we are willing to see more positive proof.

One of the most frequent questions asked is, Can undulant fever organisms cause abortion in women? There have been cases reported as being due to *Br. abortus* infection. Knowing that abortion frequently occurs in infectious diseases, it seems quite likely that the *Brucella* organisms which seem to have a preference for the genital tract may occasionally cause human abortion. This question needs more laboratory work on cultures from human abortion cases before it can be answered definitely.

Why isn't undulant fever more prevalent in view of the probable widespread presence of *Br. abortus* in milk? I can't answer this

question directly. However, if you can tell us why the milk-borne epidemic of typhoid fever in Montreal caused only about 1 person out of every 160 in Montreal to take typhoid, and why all of us who drank typhoid-infected water in the days before filtration and chlorination did not take typhoid fever, I will say that you will probably find that your answer will apply to the above question on undulant fever. We know that the *Br. abortus* is not present in large numbers in milk—50,000 per cubic centimeter being an exceptionally high number. We know that under laboratory conditions it takes *Br. abortus* a week to achieve any such multiplication as *B. typhosus* does in less than one day.

We also know that the bovine strain of *Br. abortus* is less pathogenic for animals than the goat or swine strain. We believe that resistance to disease is a variable factor among persons, and differs in any particular individual at different times. We know from experience that seed and soil are not always the only factors necessary to insure reproduction. When we find the accessory factors that determine the incidence of infectious disease and learn how to control these factors so that the proper combinations for the production of disease will not occur, our communicable disease problem will be much simplified, if not completely solved.

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## A STUDY OF LEAD POISONING IN A STORAGE-BATTERY PLANT

By LEONARD GREENBURG, *Sanitary Engineer, United States Public Health Service*, and A. A. SCHAYE and HERMAN SHLIONSKY, *Yale School of Medicine*

The president and officers of the company at whose plant the present investigation was made, realizing the possibility of the existence of a health hazard to their workers engaged in storage-battery manufacture, appealed to the State health commissioner of Connecticut for assistance in the study of the problem at their factory. The State health commissioner in turn enlisted the aid of the Surgeon General of the United States Public Health Service, who detailed Sanitary Engineer Leonard Greenburg to conduct the study here described. During the progress of the study it was found advisable to make medical examinations and blood studies of the workers in the plant. These were carried out by Mr. A. A. Schaye and Mr. Herman Shlionsky, both at that time third-year medical students of the Yale Medical School. Laboratory facilities and supplies for all of the work were generously donated by the department of public health of the Yale Medical School, for which we acknowledge our thanks and appreciation. To Dr. C.-E. A. Winslow, professor of public health, Yale Medical School, we owe our grateful appreciation for his constant aid and guidance in this study.

Throughout the course of our study we have received the unfailing assistance of the officers and staff of the plant, of Dr. Millard Knowlton, epidemiologist of the Connecticut State department of health, and of Dr. William T. Nagle, plant physician, to whom we also express our sincere thanks.

The factory at which our studies were made is devoted chiefly to the manufacture of storage batteries for automobile and radio use. A very minor amount of attention is devoted to the manufacture of radio trickle chargers, a process with which we need not concern ourselves.

The chief constituent of storage batteries is lead, in the form either of metallic lead or of powdered lead oxides. In many of the storage-battery plants of this country lead poisoning in a greater or lesser degree is encountered. The objects of the present study were to determine whether or not a lead-poisoning hazard existed in the plant under consideration; should such a hazard be found, to determine its exact extent; and, lastly, to propose such structural changes and remedial measures as would serve, in so far as possible, to eliminate this hazard.

#### THE PROCESS OF BATTERY MAKING AS EMPLOYED AT THE PLANT UNDER STUDY

There are three chief forms of storage batteries in use at the present time: The Edison alkaline cell, having nickle and iron electrodes; the Planté cell, composed of a metallic lead positive plate and a pasted negative plate; and the Faure cell, composed of plates made of lead grids into which has been pressed a paste composed of lead oxides. It is this last type of cell which is manufactured at the plant under consideration.

Briefly, the method of manufacture consists in casting the lead plates, two of which are cast simultaneously in a mold, forming a single grid. The grids are then placed in a power press where the excess metal is trimmed from the edges and the plate is straightened. The plate is then transported to the pasting room for the next operation.

The oxides commonly used for battery making are litharge and red lead, and the making of the paste from these ingredients consists in the opening of the kegs, the compounding of the necessary quantity of the dry powders, the addition of suitable liquids (usually acid or ammonium sulphate), and the mixing of this batch until a uniform paste is formed. The paste is then transported to the pasting room. Here, by means of a flat wooden paddle, the lead oxide paste is rubbed into the spaces of the grid. The plates, as they are now called, are allowed to dry and are transported to the forming room, where they are placed in lead-lined tubs and joined together to form a positive

and negative circuit. Sulphuric acid is added and the plates are subjected to the action of the electric current. This process, known as forming, serves a double purpose. It changes the lower oxides of the positive plate to a chemically higher lead oxide (the brown peroxide) and at the same time the negative plate is reduced to form spongy metallic lead. After being formed, the plates are passed through a pair of rubber rolls, which removes the excess acid, and are allowed to dry and cool. They are then passed on to a saw table where they are cut in two, for, as noted earlier, each grid consists of what finally becomes two separate finished plates. These finished plates are then taken to the assembly room where they are placed in racks. Here groups of positive and negative plates are made by fusing together the proper number of plates along with a binding post. This operation is accomplished by means of an oxygen-gas blow torch. The lugs at the corner of the plates are first melted down and then a sufficient quantity of lead is added from a stick of this metal held in the left hand of the worker. The groups of plates are removed from the burner's bench and passed to the assembly bench, where wooden separators are inserted between the plates. The assembled groups are then placed in the battery jars and sealed in place by means of hot tar or pitch. The connectors between the various groups of plates are then burned in place and the battery is given a final inspection, filled with acid, charged, and sent to the storage or shipping room.

#### THE METHODS USED IN THE PRESENT STUDY

The plan of this study was arranged with the purpose in mind of determining the actual quantity of lead in the air of the workroom and of finding the effect of this quantity of lead on the condition of the worker as determined by a rather complete physical examination.

Sanitary surveys of each workroom were made at the outset of our study, using a form which we described in an earlier publication<sup>1</sup>. It will be noted by reference to this form that provision is made for noting all of the salient features of the workroom which might have a bearing on the problem at hand. These surveys served to acquaint us with the problem in practically all of its details.

Sampling of the atmosphere for lead analyses was done by means of the Greenburg-Smith impinger apparatus.<sup>2,3</sup> The impinging tube and plate were in one piece and made of Pyrex glass. The sampling

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<sup>1</sup> Winslow, C.-E. A., and Greenburg, Leonard: A Useful Factory Inspection Form. Pub. Health. Rep., Vol. 37, No. 1, January 6, 1922, pp. 9-12.

<sup>2</sup> Katz, S. H., Smith, G. W., Myers, W. M., Trostel, L. J., Ingels, M., and Greenburg, Leonard: Comparative Tests of Instruments for Determining Atmospheric Dusts. Pub. Health Bul. No. 144, U. S. Public Health Service, 1925.

<sup>3</sup> Greenburg, Leonard: Studies on the Industrial Dust Problem. III. Comparative Field Studies of the Palmer Apparatus, the Konimeter, and the Impinger Methods for Sampling Aerial Dust. Pub. Health Rep., Vol. 40, No. 31, July 31, 1925, pp. 1591-1603.

bottles were also of Pyrex glass and the stoppers were coated with paraffin to preclude the possibility of any leaded rubber contaminating the sample. The samples were of various sizes, usually between 60 and 90 cubic feet, and were taken at positions which made them representative of the general conditions of the workroom or of the air breathed by the worker. The dust samples were taken to the laboratory and analyzed for lead by the method of Fairhall, as modified by the workers of the United States Public Health Service in their studies on tetraethyl lead.<sup>4</sup> In all, we obtained and analyzed 16 such samples.

In recording our studies on the workers themselves, we utilized the accompanying forms. These forms consist of four pages. The first half-page is given over to an industrial history, an important portion of which relates to the worker's previous employment, the object being to ascertain the bearing of his former employment on his present physical condition. This is followed by a page and a half of personal and family history. Then follows one page of physical examination data, and, lastly, a portion of a page devoted to laboratory and blood findings. These forms were devised with the object well in mind of finding those facts which might have a bearing on lead poisoning.

The physical examinations and histories took an average time of 45 minutes each. At the beginning of the study a longer time was required, and toward the close somewhat less.

At the time of our study the plant was not engaged at full production, but on certain days of the week it was possible to find each workroom nearly completely manned; for example, during the early portion of the week the pasting department worked full time, while later in the week no work was conducted there. It was thus possible to obtain representative conditions in each shop. We were able to perform physical examinations and blood studies on 56 workers.

The blood studie consisted of a determination of hemoglobin by the Dare method, a white and red cell count, and a differential count. For each worker we made two smears. These were stained with Wright's and Unna's stain, respectively, and examined under the high-power oil immersion lens for stippled cells. In those cases in which stippled cells were not found, the search was continued for at least one hour before the slide was considered negative. The actual ratio of stippled cells to the red cells was obtained from the number of fields counted for stippled cells and the average number of red cells per microscope field. We have expressed our results in terms of stippled cells per 10,000 red cells.

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<sup>4</sup> Leake, J. P., and others: The Use of Tetraethyl Lead Gasoline in Its Relation to Public Health. Pub. Health Bul. No. 163, U. S. Public Health Service, 1928.

**INDUSTRIAL HISTORY**

(Page 1)

Name..... Occupation..... No.....  
 Address..... Age..... Race.....  
 Birthplace..... How long in United States..... Number of years employed.....  
 Room in which employed..... Number hours per day..... Number days per week.....  
 History of previous employment in this plant:

Position	Date of employment	Hours per day	Room
1.....	.....	.....	.....
2.....	.....	.....	.....
3.....	.....	.....	.....

History of previous employment in other plants:

Trade	Dates	Hours of labor	Lead exposure
1.....	.....	.....	.....
2.....	.....	.....	.....
3.....	.....	.....	.....

Do you wear gloves?..... Mask?..... Wash daily?..... Bathe weekly?.....

**PERSONAL AND FAMILY HISTORY**

General health.....  
 Lead poisoning.....  
 Hours of sleep..... Restful or disturbed..... (Cooperation).....  
 Nervous system.—Memory..... Drowsiness..... Headache.....  
     Dizziness..... Nervousness.....  
 Mouth.—Soreness..... Metallic taste.....  
 Teeth.—Care..... Last visit to dentist.....  
 Gums.—Tender..... Bleeding.....  
 Gastrointestinal.—Bowel movements: Number per day.....  
     Constipation..... Use of laxatives.....  
     Abdominal pain.....

(Page 2)

Name..... No.....  
 Appetite..... Breakfast anorexia..... Salivation.....  
 Nausea..... Vomiting..... Eructations.....  
 Distention.....  
 Cardiovascular.—History of blood pressure.....  
     Palpitation..... Dyspnea.....  
 Respiratory.—Colds..... Cough.....  
     Sputum.....  
 G. U.: Frequency..... Nocturia..... Hematuria.....  
     Hesitancy..... Dribbling..... Anuria.....  
     Urgency.....  
 Muscular.—Weakness..... Tremors..... Tingling.....  
 Joints.—Arthritis.....  
 Sexual.—Married..... Potency.....  
     Number of years married..... Birthplace of wife..... Lead exposure of wife.....  
     Number of children before working at factory..... After working at factory.....  
     Miscarriages before working at factory..... After working at factory.....  
 Family history.—Father..... Mother.....  
     Familial diseases.....  
 Habits.—Alcohol..... Tobacco..... Sunlight.....  
 Food.—Milk..... Vegetables.....  
 Health history at present.....  
 Health history before working at this plant.....  
 Past diseases.....



It was impossible to obtain 24-hour specimens of urine from the workers, which, in our opinion, would be beyond criticism, and for this reason we have refrained from studies on the urinary excretion of lead.

### THE MECHANISM AND DIAGNOSIS OF LEAD POISONING

Chiefly as the result of the work of Doctor Aub and his colleagues<sup>5</sup> at the Harvard Medical School, the mechanism of lead poisoning is now quite clear. Lead may be taken into the body by three routes—ingestion, inhalation, and absorption through the skin. The latter method is an uncommon one; in fact only in certain cases such as, for example, with lead tetraethyl does absorption take place in this manner.

The composition as well as the concentration of the lead compounds to which the worker is exposed, as well as the route of absorption, plays an important rôle in determining the time of onset of disease, for it is the composition which determines the solubility of the lead in the gastric juice and other fluids of the body. Doctor Aub and his colleagues present the following table showing the solubility of various lead compounds:

*The solubility of various lead compounds in blood serum*

No.	Substance	Solubility in serum at 25° C. <sup>1</sup>	Solubility in H <sub>2</sub> O <sup>1</sup>
1	PbCO <sub>3</sub> .....	0.0333	0.0017
2	PbSO <sub>4</sub> .....	.0437	.044
3	PbCrO <sub>4</sub> .....	.....	.00001
4	PbO.....	1.1520	.0171
5	Pb.....	.578	.....

<sup>1</sup> In grams per liter.

The diagnosis of chronic lead poisoning is a matter depending on one's standards. For example, the Harvard group of workers depend for a diagnosis of early or mild poisoning on "one typical sign of lead intake and two signs of generalized intoxication." They consider the blue line on the gums, commonly called the lead line, and lead in the excretions as indicative of lead intake; and pallor, stippling of the red blood cells, or mild secondary anemia as signs of general intoxication. The United States Public Health Service workers in their study<sup>6</sup> of lead poisoning in the pottery trades divided the symptoms into two groups—major and presumptive. According to the standards of these workers a positive diagnosis depends on the

<sup>5</sup> Aub, J. C., Fairhall, L. I., Mihot, A. S., and Reznikoff, P.: Lead Poisoning. Medicine Monographs, VII. The Williams & Wilkins Co., Baltimore, 1926.

<sup>6</sup> Newman, B. J., McConnell, W. J., Spencer, O. M., and Phillips, F. M.: Lead Poisoning in the Pottery Trades. Pub. Health Bul. No. 116, U. S. Public Health Service, 1921.



presence of two or more major symptoms, one major symptom with two or more presumptive symptoms, and finally, a presumptive diagnosis "would be suggested" by three or more of the presumptive symptoms. In each case the diagnosis would be rendered clearer by the presence of the minor symptoms usually associated with those of major or presumptive character.

It is necessary to present the standards for the diagnosis of lead poisoning proposed by a third group of workers. Dr. Charles Badham and H. B. Taylor, of Australia, in 1925 published an important study on lead poisoning.<sup>7</sup> Concerning the diagnosis of this disease, they say:

For the accurate diagnosis of early or mild chronic lead poisoning there should be present in every case a definite sign of a lead intake determined by the presence of a blue line or lead in the urine in an amount exceeding 0.05 milligrams per liter, together with one or the other of the following signs of intoxication:

(i) The presence of stippled red cells to the number of over 500 per million red cells, with no other blood changes and with or without symptoms of lead poisoning.

(ii) The presence of stippled cells in number less than 500 per million red cells, with symptoms of lead poisoning.

(iii) The presence of stippled red cells with other blood changes such as changes in size or shape of red cells, polychromasia, reduction of hemoglobin to 80 per cent or less, and reduction of red cells below 4,500,000, with or without symptoms of lead poisoning.

The symptoms of mild lead poisoning are taken as abdominal pain, constipation, headache, debility, and tremor.

Two of the previously mentioned authorities demand evidence of lead absorption, either by the presence of a lead line, or lead excretion, while the third (Newman and others) requires only a history of lead exposure. The lead line is often lacking in persons who have absorbed even considerable quantities of lead and, as noted earlier, we have not made studies on the urinary excretion of lead. It is our feeling that the known exposure to lead in the plant under study was of such magnitude as to preclude any doubt as to the fact of lead absorption. This is further borne out by the fact that during the period from May, 1924, to June, 1926, there were 53 cases of acute lead poisoning in the plant under study. For these reasons we have not demanded any evidence of lead absorption, but have based our diagnosis of lead poisoning on the presence of stippled cells, plus either symptoms of lead poisoning or certain other marked blood changes which are known to be associated with the anemia of lead poisoning. Our standard, then, agrees very closely with that set up by the Australian workers, with the aforementioned exception that we have not required absolute evidence of lead absorption. In the

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<sup>7</sup> Badham, Chas., and Taylor, H. B.: Lead Poisoning—Concerning the standards which should be used in diagnosing this industrial disease, together with a new method for the determination of lead in the urine. Report of the Director General of New South Wales, for the year ended Dec. 31, 1925, pp. 52-78.

case histories we have noted the facts concerning the present as well as the previous employment of the workers, and in the discussion and evaluation of our results we have attempted to take these facts into consideration.

We have confined our study to those workrooms in which lead dust or fumes are generated to a more or less large extent, with one exception, namely, the battery charging room, the air of which we have studied as a control on the other workrooms. In a similar manner clinical studies have been made on the workers in the rooms having more contaminated atmospheres, but we have also included a number of other workers to serve as controls. For the reasons cited above, our routine plant studies have been confined to the casting, mixing, pasting, and battery assembly rooms.

#### CASTING ROOM

All the casting operations in this plant are done in a single workroom, of a 1-room brick building, 55 feet in length and 22 feet in width, with a ceiling height of 16 feet to the peak of the monitor roof. The floor of this building is made of cement and is in a satisfactory condition of maintenance.

In one corner of the room, a space 29 by 13 feet is partitioned off. This constitutes the lead storage room, where the lead pigs are kept before they are used.

The equipment in this shop consists of four pots—two large and two small ones—in which the lead is melted and from which it is ladeled into the molds. It is about the perimeter of the two large pots that the men are stationed, five men at each pot. At the two smaller pots a smaller number of men (three or four in all) cast the binding posts and smaller portions of the batteries which are required. At one side of the room there are two power presses by means of which the grids are trimmed of excess material and straightened.

Natural ventilation of the room is secured by means of three windows and four doors. Two of the doors open to the outside of the building, one opens into a storage room, and one into the pasting room (which will be described later). No artificial ventilation is provided in this room, the only provision for the removal of fumes being two 8-inch vertical tubes, one above one of the small melting pots and the other above one of the larger pots. Both of these tubes pass through the roof and terminate on the outside of the building. At the time of our visit both were blocked off and thus out of service.

The process of casting is exceedingly simple. In the case of the battery grids each man stands before one of the large lead pots (about 2 feet distant) and by means of a ladle removes a sufficient quantity of lead from the pot and pours it into a 2-piece mold which

is at his side. The cooling of the lead is accelerated by the application of water to the lead at the open side (top) of the mold; this is then opened by means of a lever and the grid is removed. Before closing the mold the caster daubs talc on the mold from a small sack held in one hand. This serves to dry the mold and to facilitate the removal of the casting. The mold is then closed by means of a lever, and the process is repeated.

In the case of the binding posts and other small parts the caster may or may not be seated at his work, and the mold, which is very much smaller, is clamped on a table or workbench. The casting process is essentially the same as that for the grids.

In the trimming operation the punch press operator places the grids under the press, and by means of a treadle the die of the press is released and descends on the grid, cutting off the excess material. The trimmings are placed in a wheelbarrow alongside the press and at a later time, when the wheelbarrow is more or less filled, the cuttings are removed and dumped on the floor near one of the big casting pots. This material is picked up from the floor and returned to the pot by means of a pitchfork. The finished grids are transported to the pasting room for the next operation.

There is no doubt that the dumping of the trimmings on the floor and the subsequent shoveling of them into the lead pots constitutes a source of lead dust; just how much lead is released by this procedure it is hard to say. We have not found it feasible to make lead determinations on this procedure.

A second possible source of lead in this room is the volatilization of lead directly from the surface of the lead pots. We have not attempted to make determinations on this point. In 1917 this question was investigated by Prof. E. B. Phelps<sup>8</sup> in a study of conditions at the United States Government Printing Office at Washington, D. C. Professor Phelps concluded from his studies that no volatilization of lead from the melting pots took place, but that lead oxide was disseminated from the surface dross on the lead pots when the metal in the pots was agitated.

The findings of Sommerfeld and of Lewin agree with those of Professor Phelps in that only when the temperature of the lead was raised to an excessively high degree or when the lead was agitated was it possible to recover it from the atmosphere. Certainly this is the case at the plant in question. Here each of the workers cast some 900 grids per day and at each casting the ladle is placed in the pot, an operation which slightly agitates the surface of the molten metal. During the course of the work day the dross is skimmed from the surface of the pot several times, a procedure which releases lead oxides

<sup>8</sup> Hamilton, Alice, and Verrill, C. E.: Hygiene of the Printing Trades. U. S. Bureau of Labor Statistics, Bul. No. 209, Washington, 1917, p. 21 et seq.

from the molten material, as does the addition of the lead pigs. And, finally, the shoveling of the lead scrap trimmings into the pots adds its quota of lead to the air.

Four samples of the air of this workroom were obtained, two near each of the large lead pots, in a position similar to that occupied by the casters. In each case it so happened that four men were at work at each lead pot.

The following table gives the pertinent data concerning these samples:

TABLE 1.—Lead content of the air of the casting room

Sample number	Position	Volume of air sampled	Lead found	Lead per cubic foot of air
		<i>Cu. ft.</i>	<i>Mg.</i>	<i>Mg.</i>
3	Near Pot 1.....	65	1.170	0.018
4	Near Pot 1.....	55	.283	.005
5	Near Pot 2.....	90	.904	.010
6	Near Pot 2.....	90	.373	.004
	Average.....			.009

It will be observed from Table 1 that the amount of lead in the air was found to range from 0.004 to 0.018 mg. per cubic foot. It seems fair to say that the men in this workroom under the conditions of the study were breathing an atmosphere containing an average concentration of 0.009 mg. of lead per cubic foot of air.

It is impossible to say what chemical form this lead was in. There is little doubt that some is metallic lead dust and lead oxides, but the proportions of each are unknown.

In all, we have made clinical studies on 12 men engaged in the casting shop. Nine of these were casters, two were trimmers, and one was the foreman. In Table 2 is presented a summary of the occupational and medical findings of these 12 workers.

In the last column of this table is our diagnosis of the workers' present condition. It will be observed that worker No. 2 is considered to be suffering from chronic plumbism; he had 8 stippled cells per 10,000, other blood changes (anisocytosis), and complained of a metallic taste in his mouth. Worker No. 7, with 27 stippled cells, metallic taste, and various other symptoms; worker No. 8, with 30 stippled cells and general muscular weakness; and worker No. 9, with 56 stippled cells, metallic taste, marked constipation, and transient tremors.

It may be of some interest to present these data in still another fashion. In Table 3 we have endeavored to bring out the relationship between the duration of exposure, the nature of the exposure, and the attacks of acute lead poisoning.

TABLE 2.—Occupational histories and clinical findings of casting-room employees

No.	Occupation	Years of employment	Previous lead exposure	Previously poisoned	Lead line	Stippled cells	Per cent hemoglobin	Red blood cells, in millions <sup>1</sup>	Other blood changes	Symptomatology			Chronic poisoning
										Gastrointestinal	Nervous	Neuromuscular	
1	Caster	2 5	+	2	—	—	—	—	++	—	+	—	—
2	do	7 5	+	1	—	8	—	—	++	—	+	—	—
3	do	5 5	+	—	—	—	65	—	++	+	+	—	—
4	do	1 5	—	—	—	—	—	—	++	+	+	—	—
5	do	6 5	+	—	++	—	—	—	++	+	+	—	—
6	do	3 5	+	1	++	1	—	—	—	+	+	+	—
7	do	8 5	+	2	—	27	—	—	—	+	+	+	—
8	Trimmer	1 0	+	1	—	30	—	—	—	—	+	+	—
9	Foreman	3 0	—	1	—	56	—	—	—	+	+	+	—
10	Trimmer	1 5	—	—	++	—	—	—	±	—	—	+	—
20	Caster	1 0	—	—	—	—	—	—	—	—	—	—	—
56	do	1 5	—	—	—	—	4.3	—	+	—	—	—	—

<sup>1</sup> Red blood cell count is given only when less than 4.5 million.

TABLE 3.—Showing the relation between nature of exposure, duration of exposure, and acute lead poisoning

Nature of exposure	Acute lead poisoning	Duration of exposure in years
No previous lead exposure	{ Not poisoned	1.08, 1.5, 1.5, 1.5.
	{ Poisoned	2.5, 6, 8.1
Previous lead exposure <sup>2</sup>	{ Not poisoned	6.
	{ Poisoned	1, 1 3, 7, 1 5.5.

<sup>1</sup> Now diagnosed as suffering from chronic lead poisoning.

<sup>2</sup> All in pasting and mixing departments.

It appears from this table that the four workers (Nos. 56, 20, 10, and 4) who were not exposed to lead prior to working in the casting room and who had worked in the casting room for 1½ years or less at the time of our examination were found to be negative for chronic lead poisoning and never suffered an attack of acute lead poisoning. Three workers (Nos. 1, 7, and 9), who also had not been exposed to lead prior to working in the casting room but who were engaged for periods from 2½ to 8 years, suffered previously from acute lead poisoning, and two of these are at the present time considered to be suffering from chronic lead poisoning. Of the workers in this room who were previously exposed to lead at a prior occupation, one (No. 5), with an occupational history of six years' duration, has never had lead poisoning and is not now considered to be suffering from chronic lead poisoning. This man appears to be most resistant to lead poisoning. For three years he was a worker in the pasting room of this same factory, and since that time for an additional three years he has been employed in the casting room. We are unable to explain how he has resisted lead poisoning except to say that he may represent one of the well-known examples of a person who is particularly resist-

ant to this disease. Nothing in his history serves in any way to cast any light on his peculiar resisting powers. And, lastly, we come to four workers (Nos. 2, 3, 6, and 8), engaged for varying lengths of time from one to seven years, exposed to lead in a prior occupation, and who had at some time or other suffered from acute lead poisoning. Two of these (Nos. 2 and 8) are at the present considered to be suffering from chronic lead poisoning. Worker No. 3 may be considered to be a possibly questionable case.

We realize the rather meager nature of these data; but it would appear that between  $1\frac{1}{2}$  and  $2\frac{1}{2}$  years of exposure to this atmosphere of 0.009 mg. of lead per cubic foot of air with the lead in such a form as in this casting room is productive of lead poisoning. There remains little doubt that the continuous inhalation of an atmosphere such as the present one over a period of more than two and one-half years might be expected to produce lead poisoning.

The casting room which has just been under discussion adjoins the mixing and pasting room, and opens into it by means of a door which is seldom kept-closed. As we shall see later, the mixing operation gives rise to an excessively large quantity of dust, and it is the belief of the investigator that a certain significant amount of this dust finds its way into the casting room and serves to increase the hazard to the workmen who are engaged in the casting of lead. It is recommended that the design and construction of this door be altered so that it will remain open only at times when materials are being transported from one workroom to another. At all other times this door should be kept shut. The method of handling the trimmings and replacing these in the melting pots is undoubtedly the source of a considerable quantity of dust. Some new method should be devised for this purpose. It might be a very satisfactory procedure if these trimmings were allowed to accumulate throughout the course of the morning and were returned to the melting pots at noon when the workers were having lunch, and then again at the close of the workday. To one who has observed the operation of casting as conducted in the plant under consideration, there is no doubt that the lead pots themselves give rise to a certain amount of lead dust, probably in the form of lead oxides. There appears to be but one means by which this lead dust may be prevented from contaminating the general workroom atmosphere and that is by the installation of more suitable ventilation methods than are at present in vogue. It may be necessary to provide the lead pots with artificial exhaust ventilation; but before doing this we would suggest trying the less costly practice of hooding over the lead pots in so far as this is possible and venting these hoods through the roof of the building by means of suitable vertical flues. In order to prevent down drafts it is desirable to provide the top of these flues with suitable ventila-

tion cowls. The remainder of the precautions to be taken in this shop are of the nature of general attention to the sanitary and hygienic condition. The housekeeping of this workroom should be maintained in a satisfactory manner by proper wet sweeping methods, sweeping being done only when the workroom is completely free of workmen. The workroom should further be freed of all material held for future use and all finished products. Such incumbrances in the workroom merely constitute additional resting places for dust which may at some later time be stirred up and contaminate the atmosphere.

#### THE MIXING AND PASTING ROOM

The mixing and pasting operations at this plant are housed in a single room of a 1-story brick building which adjoins the casting room on one side and the converting room on the other. This room, which is 55 feet in width and 30 feet in length, is provided with a cement floor and a wooden roof, the peak of which is 20 feet above the floor level.

The room may be considered to be divided into two parts by an imaginary line joining the doors from the two adjoining workshops. On one side of the room all of the mixing operations are conducted, while on the other side the pasting process is carried on. In the portion of the room devoted to the mixing process there is provided a bucket elevator which raises the lead powders from the floor level and empties them into two dry-mixing machines. Here the proper proportions of the oxides are intermittently mixed, and it is from these machines that this material is delivered to the weighing scales. There are also provided three wet mixing machines in this part of the room. These merely consist of horizontal revolving tables and blades which may be lowered into the buckets and revolved in an opposite direction. On a raised platform in the corner of the room three tanks and a large ventilating fan (which will be described later) are mounted. Two of these tanks contain acid; the third contains a solution of ammonium sulphate.

The left side of the room (that part in which the pasting operation is conducted) is provided with six double workbenches, capable of accommodating 12 pasters. These are very light wooden benches at approximately waist level, provided with a central vertical partition on the opposite sides of which the two pasters are at work. In addition to his workbench, the paster is provided with two boards about 24 inches long and 10 inches wide by means of which he is enabled to turn over the grids, an operation which will be described later. He also has a paddle which is made of wood for use in pasting the grids.

The operation which is conducted in this room may be briefly described as follows: Kegs of lead oxide are wheeled into the mixing department by means of a hand truck. They are then opened and

dumped into the hopper or the bucket elevator which then delivers this material to the storage bins. The mix operator places a bucket on the platform of a scale beneath the outlet of the dry mixing machine and by means of a lever delivers the mixed oxides into the bucket until the correct weight is registered on the scale. The bucket, while still on the scale is wheeled over to the wet mixing machine. It is then drawn onto the table of this machine, a sufficient amount of a solution (sulphuric acid or ammonium sulphate) is placed in the bucket, the mixing blades are lowered and the power is turned on so as to rotate the table and the mixing blades, thereby completely mixing the batch.

After a sufficient amount of mixing has produced a homogeneous paste the power is shut off and the mixing bucket removed to the other side of the room and delivered to the pasting operators, or "pasters" as they are called.

Each of the pasters removes a fairly large ball of the paste from the bucket and places it on his workbench. One of the pasting boards is then placed on the bench and covered with a sheet of paper on top of which is placed a row of battery grids. By means of a wooden paddle a portion of the paste is removed from the supply on the worker's bench, placed on the surface of the grid, and rubbed into the interstices by means of the paddle. When this is accomplished, a sheet of paper is placed over this newly prepared surface and thereon is placed the second pasting board. By holding the upper and lower pasting boards tightly the operator is now in a position to turn over the grids. After this is done, the now uppermost board is removed, the sheet of paper beneath is then removed, and the worker proceeds as before to rub the paste into the grid once again. After this is done the excess paste is wiped off the edges of the grids and they are racked up ready for transport to the converting room.

Natural ventilation in this workroom is secured by means of windows, of which there are eight, and by means of three doors opening to the outer atmosphere and two opening to the two adjoining workrooms.

Artificial ventilation is provided by means of a 5-foot No. 70 American Blower Co. planing-mill exhauster, capable of exhausting 15,000 cubic feet of air per minute. This fan serves to ventilate (by exhausting air) both sides (mixing and pasting) of the workroom. On the mixing side of the room there is a 15-inch circular duct from which three 6-inch pipes branch off and are connected one with the hood over each of the wet-mixing machines. Similarly an 8-inch pipe serves the bucket elevator, and another 8-inch pipe, which later branches into two, serves the two dry-mixing machines. On the pasting side of the room there is provided an 8-inch circular duct from which 6-inch pipes branch off leading to the pasting benches. At each workbench these 6-inch ducts terminate in a narrow slot,



approximately 2 inches wide, in the vertical partition which separates the two sides of the bench. These 2-inch slots on each side of the workbench have been provided primarily for the purpose of removing the fumes of the ammonium sulphate which the workmen find so objectionable.

The mixing room differs from the casting room in that here practically every time the lead oxide is handled it becomes a source of poisonous dust which is capable of producing poisoning if the inhalation is continued over a sufficiently long period of time. The opening of the kegs of lead oxide and the dumping of these kegs into the bucket elevator is another source of lead dust. Then, later, when the lead oxides are mixed in the dry-mixing machine, additional dust is given off. In the weighing process an exceedingly large quantity of dust is produced; and lastly, at the beginning of the wet-mixing process, at a time prior to that when the batch is moist, a relatively large quantity of dust is continuously yielded up to the atmosphere. It is true that the exhaust ventilation provided for each of the mixing machines lessens the hazard to some degree, but this precaution is not sufficient to eliminate it completely.

In the pasting room the problem is brought about by a completely different state of affairs. Here the lead paste in its moist condition is relatively harmless, but it is permitted to dry on the wooden workbenches and on the floor, later to become pulverized and distributed to the atmosphere. The suction devices provided on the pasting benches undoubtedly remove some lead dust. It is impossible to say just how much; but it is to be noted that these exhausts have been designed primarily for the removal of the ammonia fumes and not for the removal of the pernicious lead dust.

Because of the fact that both of these processes take place in one room it is difficult to say exactly how much dust is contributed by each. To one experienced along these lines it appears, however, that the mixing room certainly contributed more than the pasting room to the total quantity of aerial dust. It is to be borne in mind that the mixing operation is carried on usually by one or two men, whereas the pasting operation is carried on by any number of men up to 12.

Seven samples of air were obtained in this room and analyzed for their lead content. Five of these (Nos. 7, 8, 9, 15, and 16) were obtained in that portion of the room devoted to pasting, whereas two (Nos. 10 and 14) were obtained in the mixing room. In Table 4 are tabulated the pertinent data concerning these samples.

It will be observed from this table that when no men were at work there was 0.0029 mg. of lead per cubic foot of air. When six men were at work the lead content was found to be 0.0344 mg. per cubic foot of air. In two other cases with pasters at work the results were 0.0402 and 0.0291 mg., respectively. When six men were at work and sweeping was carried on for 35 minutes the lead content was 0.0743 mg

TABLE 4.—Lead content of the air of the mixing and pasting room

Sample No.	Position	Vol. of sample	Lead found	Lead per cubic foot of air
		Cu. ft.	Mg.	Mg.
7	Pasting room, center, no work going on.....	75	0.221	0.0029
8	Pasting room, center, 6 men at work.....	75	2.582	.0344
9	Same as sample 8, sweeping for 35 minutes.....	75	5.572	.0743
15	2½ feet right of paster at breathing level.....	60	2.410	.0402
16	do.....	32	0.932	.0291
	Average.....			.0362
10	Center mixing room, no mixing going on, cleaning up work-room.....	75	2.969	.0396
14	Mixing dry batch.....	60	3.052	.0509
	Average.....			.0453

It would appear that there exists in this room a certain amount of lead dust which contaminates the atmosphere at all times. It is true that the presence of the investigator in the room may have aided in the stirring up of this dust in the atmosphere, for even with only this person in the room the lead content was found to be 0.0029 mg. per cubic foot of air. The effect of the ordinary occupation is disclosed by the three samples (Nos. 8, 15, and 16) which yielded an amount of dust varying from 0.029 to 0.04 mg. per cubic foot. The effect of sweeping the floor is very clearly brought out by sample No. 9, during the taking of which sweeping was carried on for a period of 35 minutes.

In the mixing room, with no work being carried on, but with cleaning up taking place, the lead content was found to be 0.0396 mg. per cubic foot of air, a relatively large quantity; and when the dry batch was being mixed a still larger amount, 0.0509 mg. per cubic foot, was obtained. It seems obvious from this that the dry mixing of these lead powders is the source of a considerable quantity of the lead dust in this room. The five samples taken in the pasting room (including even the one while sweeping was taking place) yielded an average of 0.0362 mg. of lead per cubic foot of air, as compared with 0.0453 mg. per cubic foot obtained in the mixing room. It would appear that the mixing process gives rise to more dust than does the normal process of plate pasting. It is our feeling that these results would present a more striking contrast if the pasting and mixing operations were conducted in separate rooms and could thus be compared on an absolute basis.

Practically all of the lead in this workroom must have been in the form of lead oxides.

Owing to the fact that work was at such a low ebb at the time of our studies, we were forced to be content with a rather small number of physical examinations of workers from this workroom. In all we have been privileged to examine seven pasters and but a single mixer.

A brief summary of the pertinent data relating to these examinations is given in Table 5.

TABLE 5.—Occupational histories and clinical findings of pasting and mixing room employees

No.	Occupation	Years of employment	Previous lead exposure	Previously poisoned	Lead line	Stippled cells	Per cent hemoglobin	Red blood cells, in millions	Other blood changes	Symptomatology			Chronic poisoning
										Gastrointestinal	Nervous	Neuro-muscular	
11	Foreman-paster.....	1.0	+	1	—	3			+	+	—	—	+
12	Paster.....	.5	—	—	—	4	55	3.8	—	—	—	—	—
13	do.....	.5	—	—	—	8			—	—	—	—	—
14	do.....	( <sup>1</sup> )	—	—	—				—	—	—	—	—
15	do.....	1.75	+	—	—				+	+	—	—	+
16	do.....	.75	+	—	—				—	—	—	—	—
17	Mixer.....	2.0	—	1	+	8			+	—	+	+	+
18	Paster.....	.70	—	1(?)	+	1	61	4.2	+	—	—	+	+

<sup>1</sup> Mixer, April to November, 1926

<sup>2</sup> Plate sticker, 1 month.

<sup>3</sup> Paster, January to June, 1926.

<sup>4</sup> Paster for 3 months and helper in casting shop.

<sup>5</sup> Employed 2 weeks.

It will be observed that of the 8 men examined, 4 had been previously exposed to lead in this or some other workroom of the plant for a shorter or longer period of time, varying from 1 to 8 months. Of these 4 men (Nos. 11, 15, 16, and 17), 2 had previously suffered from acute lead poisoning (Nos. 11 and 17), and at the time of the investigation these 2 were considered to be positive for chronic lead poisoning. The remaining 2 are questionable cases. Of the 4 men who have never been exposed to lead in any previous occupation, 1, employed 2 weeks, has 8 stippled cells per 10,000 red cells, with the presence of anisocytosis and no apparent symptoms, making his case merely suggestive of chronic lead poisoning; 1, employed 6 months, having 4 stippled cells, a low hemoglobin, and a low red-cell count, making his a positive case; 1, employed about 8 months, having a lead line, 1 stippled cell, nervous and certain other symptoms, a low hemoglobin, a low red-cell count, and other blood changes, making his a positive case; and, lastly, 1 man, employed 6 months, showing no significant blood changes and suffering occasionally from dizziness, slight nausea, and salivation, whom we regard as negative. Considering these 8 workers once again, irrespective of previous employment, 4 are positive, with periods of employment of 1 year, 6 months, 2 years, and 8 months, respectively; 2 are questionable, with periods of employment of 1¾ years and ¾ years, respectively; 1 is merely suggestive, with a period of employment of 2 weeks; and 1 man is negative, with a period of employment of 6 months.

It is interesting to note that of all of the workers whom we have examined there is only 1, No. 12, who is free from blood changes of any kind, and this man was employed for 6 months. From our

data it would appear that this occupation has produced certain blood changes in all the workers with the exception of this man. Even the man who was employed but 2 weeks showed marked blood changes, due undoubtedly to his employment in a leaded atmosphere. The question may be raised as to why worker 15 and worker 16 were diagnosed as only possible cases. They were so diagnosed because they showed no stippled cells. Number 15, for example, presented certain gastro-intestinal and neuro-muscular symptoms, in addition to which he suffered from anisocytosis and basophilia. Number 16 suffers from occasional spells of dizziness, headache, and nervousness.

In considering the remedial measures for the correction of existing conditions in this workroom, we have come to the conclusion that it is of prime importance that the pasting and mixing operations be completely separated. We would, therefore, urge the removal of the mixing operations from the room in which they are now conducted or the complete partitioning off of the mixing from the pasting room. This might well be accomplished by the installation of a partition completely across this room provided only with a door for the entrance and exit of workers and material, and provided, also, with glass panelling for the transmission of light. This partition will serve in a very large measure to prevent the contamination of the pasting-room atmosphere by dust from the mixing department. Wagener<sup>9</sup> has pointed out that when the making of paste and the pasting of plates were conducted in the same room in a certain factory, 20 of 27 men in that room suffered from lead poisoning, but after the two processes were separated, only 10 out of the same number were poisoned. He also discloses the results of certain changes in a factory in Cologne, where, in addition to separating the pasting from the mixing, the pasting tables were furnished with glass cabinets and exhausts. Before the introduction of these protective measures there were 33 cases of lead poisoning among 153 pasters. Later there were 9 cases among 194 pasters and the following year only 8 among 209 pasters.

It would be exceedingly helpful if the bucket elevator were re-arranged so as to open outside of the building at some point which might be protected from the wind by a type of semi-inclosure, perhaps made of ordinary boards. This bucket elevator should be so re-designed as to consist of a steel framework which will hold the keg of lead and which may be revolved, thereby tipping the keg upside down and emptying the material into the elevator hopper. It may be sufficient to place such a tipple over a chute or opening in the floor, surrounding this completely by a cabinet which is connected with the suction system. A handle protruding through this cabinet

<sup>9</sup> Hamilton, Alice: Lead Poisoning in the Manufacture of Storage Batteries. Bul. No. 165, U. S. Bureau of Labor Statistics, Washington, 1915, p. 11.

serves for revolving the keg from the outside. The keg is simply put into the cradle, the top removed, the door of the cabinet closed, and the handle turned, thereby dumping the keg.

The dry-mixing machines appear to be satisfactory as such, but when the powder is removed from these machines and weighed out a large amount of dust is liberated. This appears to be a very unsatisfactory procedure. The desideratum here would be to maintain these mixed dry powders in a completely inclosed system until such a time as they are converted into a paste. In the present instance this would necessitate the elevation of the dry-mixing machines, or perhaps some other provision so that the oxides could be delivered directly from these machines to the weighing or measuring device, and finally delivered to a completely inclosed mixer. After the proper charge has been delivered to a mixer of this type, the measured volume of solution may be run into the charge and the whole batch completely mixed until a homogeneous paste is obtained. It is possible, also, to connect such a mixer to the exhaust system by a small duct, so that when the batch is completely finished the mixer may be ventilated prior to its opening. In the light of the present investigation we recommend the complete abandonment of the three existing so-called wet-mixing machines. Should this, however, not be possible we would urge that the present mixers be entirely inclosed in a sheet-metal inclosure made by extending the present hoods to the floor level and provided with a door for the removal of the buckets.

The problem of dust prevention in the pasting room requires no such radical treatment as that outlined for the mixing room. Here, as noted earlier, and as shown by our dust determinations, a very large proportion of the dust is undoubtedly created by the sweeping of the workroom floor and the cleaning of the work places. For the alleviation of conditions in this portion of the workroom we recommend that all stored material which might serve as a collecting place for dust be removed in so far as possible; that the workroom floor be completely scraped and covered with a covering of smooth cement so as to present a new and nonadherent surface; and that all the workbenches be covered with sheet metal and have about their perimeter a raised ledge, approximately 1 or 2 inches high, so as to prevent the dropping of material from the workbench to the floor. Each workbench should further be provided with a glazed stoneware crock of a satisfactory size to be used as a receptacle in which the ball of lead paste should be kept by each of the workers. These crocks should be provided with covers, which should be replaced at the end of the workday and such other times as when work is not in progress.

As noted earlier, the proper maintenance of this room is an exceedingly important factor in the prevention of poisoning. The floor of this room should be scraped and washed down at the close of each

work day by means of a hose and a metal scraper. The cleaning of the workroom should be prohibited at all times when the workers are present.

**THE FORMING OR CONVERTING ROOM**

After the grids are pasted they are transported to the forming room or "converting room," as it is called. Here the grids are placed in a bath of sulphuric acid and connected by means of lead strips burned onto the lugs of the plates. When all of the positive plates have been connected in this manner and, similarly, all of the negative plates, the current is allowed to flow through the system.

The room in which this work is carried on is a very large one and need not be described here. Natural ventilation is quite ample for ordinary purposes, except that at certain times there is a noticeable amount of sulphuric acid mist present in the air of this room.

There are several sources of lead in this workroom. The plates themselves, composed as they are of lead and lead oxide paste, yield some dust when they are handled, and again just before being placed in the box, when the lugs on the corner of each grid are brushed with a steel or wire brush in order to brighten their surface and remove any adherent dross. And, finally, after they are placed in the bath the lead connector must be burned onto the lug of each grid. This is done by means of an oxygen-gas torch, the worker holding the torch in the left hand, leaning over the box and burning a small point on the side of the connector onto the lug.

TABLE 6.—Occupational histories and clinical findings of converting-room employees

No.	Occupation	Years of employment	Previous lead exposure	Previously poisoned	Lead line	Stippled cells	Per cent hemoglobin	Red blood cells, in millions	Other blood changes	Symptomatology			Chronic poisoning
										Gastrointestinal	Nervous	Neuromuscular	
19	Former	4.0	-	2	+(?)	20	P. ct. 65	-	+	-	+(?)	+	+
22	do	4.0	+	1	+	10	65	4.3	+	+(?)	+(?)	+	+
21	Plate sticker	.7	-	-	+	44	68	-	+	-	+(?)	-	+
23	do	.1	+	-	-	8	58	4.0	+	-	+(?)	-	+

<sup>1</sup> Paster for 1 year.

<sup>2</sup> Caster for 3 months in 1925; repairer for 6 months in 1926.

This whole procedure is somewhat limited in such a small plant as that under consideration. Due to this fact the "plate stickers," as they are called, are only intermittently engaged in the occupation of burning the connector onto the grids. There are only two such men engaged in this work; and while we have not made any analyses of the dust in this workroom, we have made physical examinations of

these two workers. The results of these examinations are presented in Table 6. Both of these workers are at the present time, in our opinion, suffering from chronic lead poisoning. One of the men has worked previously in other occupations in this plant for nine months, the other has been employed only as a plate sticker. The previous occupation of employee 23 may have an important bearing on his present condition, but employee 21, with 44 stippled cells and other blood changes and the presence of a lead line, certainly has indications of chronic lead poisoning due to the process in hand. Employees 19 and 22 also work in the forming room. They are engaged at various miscellaneous tasks, among which is the cleaning of the lugs on the grids. Both of these men are at present suffering from chronic lead poisoning.

The nature of the work in this room appeared, at the time of our shop inspection, to be productive of so little lead dust that we did not make lead determinations of the atmosphere. We are struck now by the fact that all four men appear to be suffering from chronic lead poisoning. This evidence forces us to raise the question as to the possibility of the sulphuric acid in the atmosphere being absorbed through the respiratory tract and altering the conditions in the body so that the lead is kept in the circulation, thus producing the characteristic symptomatology and blood findings. We regret that we have no further evidence on this question.

The grids, after treatment in the converting bath, are in a proper chemical state to form the finished battery and are now dried and taken to a circular saw where each grid is cut in two equal parts to form the individual plates of the battery. We have not made dust determinations in the neighborhood of this circular saw, for the work is highly intermittent; and while it appears that a large amount of lead dust is produced, the intermittent character of the operation introduces a factor of safety which aids the worker and makes it difficult to determine any relationship between the lead content of the air and the physical condition of the workmen. It seems obvious from our inspection of the task that the worker requires protection. The management has since made this operation automatic by means of a chain feed which continuously delivers the grids into the saw.

Only one worker was engaged to any extent on this task, and we have had the privilege of examining him. He was employed from April, 1925, to February, 1926, and for a while during March was on a night shift. He has never suffered from lead poisoning, but at the time of our examination disclosed a red-cell count of 3,500,000, a hemoglobin of about 60 per cent, 1 stippled cell per 10,000, and a questionable anisocytosis and poikilocytosis. It seems quite clear that this man is suffering from chronic lead poisoning.

As at present conducted, without the necessary inclosure and the use of exhaust ventilation, this plate-sawing operation is undoubtedly a hazardous one. We urge that improvements in this machine be made in order to render this operation harmless, and we would further recommend that the plate-sawing operation be conducted, if possible, in a partitioned-off portion of the converting room.

#### THE ASSEMBLY ROOM

After the grids have been cut in two so as to form the separate plates, they are transported to the assembly room, where they are made into the finished battery.

This room occupies a large portion (55 by 78 feet) of a 1-story brick building, with a monitor roof, having its peak 28 feet above the floor level. In the northern portion of this room the battery assembling is conducted. This is done chiefly on three long workbenches and a roller conveyer.

On the first of these three workbenches there are 12 racks, each of which is so arranged as to hold groups of the necessary number of battery plates, either negative or positive, along with the necessary binding posts or connecting posts for each particular group of plates as the case may be. The "burner's bench," as this one is called, is equipped with gas connections and oxygen-gas blow torches connected with tanks of oxygen by means of rubber tubing.

The burner stands on one side of this workbench while directly opposite to him stand several men who are engaged in racking up the plates and placing the necessary binding posts in the racks therewith. The burner proceeds to melt down the lugs which compose the corner of the plates, and after this is done he adds a quantity of lead by melting it from a bar held in his left hand. This serves completely to unite the binding posts and all of the plates into a single group. The burner then proceeds to the next group of plates and so on until the rack is finished, after which he proceeds to the next rack and so on the length of the workbench.

If one watches this operation very closely he will observe that from time to time a puff of lead fumes arises from the surface of the metal while it is being melted, and that during this time the nose and mouth of the burner is usually fairly close to the surface of the molten metal and often in the direct path of the fumes arising from it. There are other sources of lead in this workroom. The handling of the lead plates in their present dry condition must give rise to a certain amount of lead dust. Occasionally, too, it happens that some of the plates are not properly assembled and, therefore, these must be cut apart. This operation is accomplished on a small power-driven circular saw and this, too, must give rise to a certain amount of



objectionable lead dust. As noted earlier, the process of burning is a fairly continuous operation, and it would therefore seem that the men who rack the plates and the burner are more or less continuously exposed to these fumes.

The other operations being conducted in this room do not require our detailed consideration. They merely consist in the placing of wooden separators between the plates forming each battery group, the placing of these groups of plates in the battery jars, and, lastly, the assembly of the battery jars in the battery box. The only other lead-burning operation which takes place is a rather limited one, in which the connectors between the binding posts are soldered. This operation consists in the placing of a connector on the binding posts, the tapping of the connector in place by means of a mallet, and the melting of a small amount of lead from a stick onto the junction between the post and the connector.

There is no artificial ventilation of any kind provided in this workroom.

Because of the fact that the burner moves from rack to rack, as previously described, it became necessary to employ a special technique in obtaining samples of the atmosphere which he breathes. In order to do this we provided the impinger sampling apparatus with a very long rubber tube connection between the sampling bottle and the suction pump. This enabled us to move the connecting flask alongside the head of the worker as he progressed along the bench from rack to rack. We believe that the samples which we have obtained in this manner are representative of the atmosphere breathed by the lead burner. Table 7 presents the necessary data concerning these samples and the analytical results obtained.

TABLE 7.—*Lead content of the air of the assembly room*

Sample No.	Position	Volume of sample		Lead found	Lead per cubic feet of air
		Cu. ft.	Mg.	Mg.	Mg.
11.....	Following burner.....	90	1.291		0.0143
13.....	do.....	67	.594		.0089
Average.....					.0116
12.....	General room air.....	90	.863		.0096

These observations indicate that the atmosphere breathed by the lead burner varied from 0.009 to 0.014 mg. per cubic foot of air and averaged 0.0116 mg. per cubic foot. This is an amount slightly in excess of that obtained by analysis of the general room air which, according to sample 12, is 0.0096 mg. of lead per cubic foot of air.

The sample of general room air was obtained on the second row of workbenches in this assembly room.

In all, we have had the opportunity of making clinical studies on 10 men who were engaged in this assembly room, either as burners or as assemblymen. In Table 8 is presented a summary of the occupational and medical findings of these 10 workers.

Reference to Table 8 shows seven of these workers to be suffering from chronic lead poisoning. One of the workers (No. 26) presents a questionable case; he has a rather low hemoglobin of 65 per cent and a low red-cell count, somewhat under 4,000,000. He also presents questionable evidence of a lead line and vague gastro intestinal symptoms. Two of the workers are negative; No. 33, who, while his total duration of employment as noted earlier is 1½ years, has been working only in the winter season of each year, and No. 27, whose duration of employment is 1.7 years and who presents no signs or symptoms of any interest.

Four of the seven positive cases are of workers who have been engaged for 1½ years or less. It would appear from this that the presence of approximately 0.0096 mg. of lead per cubic foot of air in the form which is present in this workroom is productive of lead poisoning in a shorter time than an approximately equal amount of lead in a different form such as is to be found in the casting room. We realize the shortcomings of our data; but it is our feeling that the evidence presented is perhaps suggestive, at least, that the form in which the lead is present in the atmosphere has a decidedly important bearing on the time factor in the production of lead poisoning.

The peculiarly acute and rapid effects brought about by the inhalation of lead tetraethyl are evidences, we feel, of the exhibition of the tendency of lead in certain highly dispersed forms to be more readily taken up by the blood stream. According to Doctor Hamilton<sup>10</sup> "lead burners have work which German and English authorities consider particularly dangerous." She adds: "As we shall see later, the most recent British reports show more lead poisoning among the lead burners than among any other workmen in the electric-accumulator factories, and British factory inspectors are advising the installation of air exhausts at the workbench to protect these men." Certain questions concerning the temperature of the burning torch arise which can not be discussed here. In the 1912 Report of the Inspector of Factories of Great Britain there is presented a table which shows that the rate of lead poisoning in the lead-burning operation was higher than any other observed in one English factory.

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<sup>10</sup>Loc. cit.

TABLE 8.—Occupational histories and clinical findings of assembly-room employees

No.	Occupation	Years of employment	Previous lead exposure	Previously poisoned	Lead line	Stippled cells	Per cent hemoglobin	Red blood cells, in millions	Other blood changes	Symptomatology			Chronic poisoning
										Gastrointestinal	Nervous	Neuromuscular	
26	Assemblyman	3.1	—	—	+(?)	—	65	4.4	—	—	—	—	(?)
27	do	1.7	—	—	—	—	—	—	—	—	—	—	—
28	do	1.5	—	—	—	7.0	60	4.1	++	—	—	—	—
29	do	.5	—	—	—	2.5	—	—	++	—	—	—	—
30	do	1.7	—	+	+	13.0	—	—	++	—	+	—	—
31	do	.6	—	—	+	107.0	—	—	++	—	—	—	—
32	do	1.2	+	+(?)	+	37.0	65	4.3	—	—	—	—	—
33	do	1.5	+	—	—	—	—	—	—	—	—	—	—
34	do	4.0	—	—	—	8.0	—	—	+	+	—	—	—
35	do	3.0	—	+	—	6.0	—	—	+	++	—	—	+

<sup>1</sup> 6 weeks as a mixer.

<sup>2</sup> Intermittent.

The protection of the worker in the assembly room calls for the use of exhaust ventilation. It appears to us to be the only remedy for conditions which our survey has revealed in this workroom. We recommend that for the existing burner's table and first assembly table (on which the separators are placed in the battery groups) there be substituted tables having slotted or grilled tops made of either metal or wood, and that these table tops be provided with exhaust ventilation so that all the dust which is produced above these two tables may be removed by the exhaust ventilation. The provision of such ventilation, we feel, will serve to remove the greater portion of the lead which is produced in the handling of the plates. There still remains the lead vapor, or oxide, which is produced by the burning operation in the assembling of the battery groups. For the removal of this, we would recommend the provision of an exhaust ventilation duct along the whole length of the table and at a level some slight distance above the racks. It is essential that this duct be placed so as not to interfere with the lead burner or the men racking the plates. The location of this duct is, therefore, a matter of some question and we hesitate to point out exactly where it should be. It seems to us, however, that a point approximately 5 inches above the rack and about 6 inches from the burner's working place would yield the best results. The duct need not be larger than 3 inches in diameter and should be so designed that the entering air would have a velocity of approximately 1,000 feet a minute.

#### MISCELLANEOUS AND CONTROL STUDIES

It remains for our discussion to deal with certain miscellaneous operations and control studies which we have made. We have, for the sake of presentation, divided these data into three groups.

(1) Those workers who are exposed to lead to an uncertain degree in their occupation and in a given workroom. This group is composed of 5 repairmen.

(2) Those employees whose occupation brings them into various rooms at different times, thus producing a highly uncertain exposure. This group is composed of 2 machinists, 3 millwrights, and 1 janitor.

(3) Those workmen whose occupation theoretically brings them into no contact with lead dust. This group is composed of 3 packers, 3 storeroom clerks, and 4 charging-room workers.

It is obvious that such a classification is not a very precise one; for, in a plant such as the one under consideration, where many of the workrooms are not partitioned off from each other, lead dust finds its way into workrooms where the particular operation under consideration is not the source of the dust.

In only one case, namely, that of the charging room, do we feel that the room is sufficiently removed from all others and sufficiently self-inclosed to yield interesting information from the point of view of the lead content of the air. The charging shop occupies a separate building, 108 feet long by 30 feet wide, with a peaked roof 24 feet above the floor level. The apparatus in this room consists of 18 charging benches, at the end of each of which is an electrical panel board for regulating the electric current to the batteries being charged. At one side of the room is a long bench, known as the filling bench, on which the batteries are placed during the time that they are being filled with a solution of acid. On the opposite side of the room is a finished battery conveyor by means of which the completed and charged batteries are conveyed to the shipping room.

The batteries are brought into the room on the filling bench and filled with acid by means of a hose from a tank outside of the building. The batteries are removed from the filling bench to the charging bench and are connected by means of leads to the charging circuit. They are tested from time to time until they finally arrive at the point where they have been sufficiently charged, when they are removed to the finished battery conveyor on the side of the room. From here they are delivered by gravity to the packing and shipping room.

No artificial ventilation is required or provided in this workroom, and there appears to be no real lead hazard. The only hazard which may exist is that due to the possibility of electric shocks, which is rather limited, and that due to the possibility of acid burns, which undoubtedly take place from time to time but which are of a minor character.

We have taken two samples of the atmosphere of this workroom, each of 90 cubic feet. Both samples were found to be negative for lead.

Table 9 presents the pertinent data with respect to the three groups of workmen previously described.

TABLE 9.—Occupational histories and clinical findings of miscellaneous and control groups of workers

No.	Occupation	Years of employment	Previous lead exposure	Previously poisoned	Lead line	Stippled cells	Per cent hemo-globin	Red blood cells, in millions	Other blood changes	Symptomatology			Chronic poisoning
										Gastro-intestinal	Nervous	Neuro-muscular	
37	Repairer.....	1.0	—	—	—	—	—	—	—	—	—	—	—
40	do.....	1.4	—	—	—	32	—	—	+	+	—	—	+
41	do.....	1.5	—	—	—	—	—	—	—	—	—	—	—
42	do.....	1.8	—	—	—	—	65	3.4	±	—	—	—	—
43	do.....	5	—	—	—	—	—	—	—	—	—	—	—
25	Janitor.....	1.3	—	—	—	—	—	—	+	—	—	—	—
38	Machinist.....	2.4	—	—	—	—	—	—	—	+	—	—	—
39	do.....	4.2	—	—	—	—	—	—	—	+	—	—	—
53	Millwright.....	1.7	—	1(?)	+	—	—	—	—	—	—	—	—
54	do.....	3.0	—	—	+	8	—	—	+	—	—	—	+
55	do.....	7.5	—	—	+	—	—	—	+	—	—	—	+
48	Packer.....	1.5	+	1	—	10	65	—	+	—	—	—	+
49	do.....	1.1	—	—	—	—	—	—	—	+	—	—	—
60	do.....	7.0	+	1	—	1	—	—	—	+	—	—	+
36	Store room clerk.....	2.0	—	—	—	—	60	4.1	+	+	—	—	±
51	do.....	6.6	—	—	—	—	—	—	+	+	—	—	—
52	do.....	7.6	—	—	—	—	—	—	+(?)	—	—	—	—
44	Charger.....	2.7	—	—	—	—	—	—	—	—	—	—	—
45	do.....	5.5	—	—	—	—	—	—	—	—	+	—	—
46	do.....	8.0	—	—	—	—	—	—	—	—	—	—	—
47	do.....	5	—	—	—	—	—	—	—	—	—	—	—

<sup>1</sup> Worked within range of dust of mixing room for 1 month, and developed lead poisoning.

<sup>2</sup> Paster for 4 years.

<sup>3</sup> Paster for 3 months.

<sup>4</sup> Paster for 3 months. Acutely leaded at that time.

It will be observed that of the first group, one of the repairmen is at the present time suffering with chronic lead poisoning. His examination revealed the presence of 32 stippled cells, certain other blood changes, and the presence of tremors. In the second group of workmen it will be observed that there exist two possibly questionable chronic cases. And, finally, in the last group, composed of packers, storeroom clerks, and battery chargers, there was found to be one case of chronic lead poisoning. This case was that of a packer who was exposed to lead in a previous occupation and suffered from acute lead poisoning. He presented 10 stippled cells, a rather low hemoglobin (65 per cent) and certain other blood changes, as well as neuromuscular symptoms. His previous occupation was that of a paster for a duration of three months. All of the other workers in this group were negative, and the four battery chargers, in whose workroom we found no lead, were completely negative for lead poisoning.

#### SUMMARY AND CONCLUSIONS

The present study deals with the findings of sanitary surveys of the workrooms of a small plant devoted to the manufacture of storage batteries. The results of the analyses of 16 samples of the atmos-

pheric air for lead and of the clinical and laboratory examination of 56 workers who have been engaged in various tasks in the making of storage batteries are given. Suitable controls have been provided, both for the air examinations and for the clinical aspects of the problem.

The air analyses disclosed the fact that, in the charging room, used as a control, there existed no lead, whereas in the other work-rooms the quantity of lead found in the atmosphere was as follows:

TABLE 10.—*Summary of air analyses for lead*

Room	Average lead content per cubic foot of air
Charging room.....	Mg. 0.0000
Casting room.....	.0090
Pasting room.....	.0362
Mixing room.....	.0453
Assembly room (burning operation).....	.0116
Assembly room (general atmosphere).....	.0096

The examination of the workers in these various rooms indicated that in the charging room there existed no cases of chronic lead poisoning, whereas in the other rooms the total number of men examined and the number of cases of chronic lead poisoning were found to be as follows:

TABLE 11.—*Summary of clinical findings*

Room	Number of men examined	Number of men positive for chronic lead poisoning
Casting room.....	12	4
Pasting and mixing room.....	8	5
Converting room.....	4	4
Assembly room.....	10	7
Miscellaneous group.....	11	2
Control group.....	10	1

<sup>1</sup> Was a paster for 3 months and suffered from acute lead poisoning.

Our studies indicate that, whereas some of these cases of chronic lead poisoning are undoubtedly in some measure attributable to previous exposure in a different atmosphere, and some of the symptomatology and blood findings are due possibly to the existence of previous acute lead poisoning, the majority of these cases of chronic lead poisoning are due to the inhalation of the lead in the atmosphere as tabulated above.

For many years the most satisfactory evidences concerning the relation between the occurrence of poisoning and the quantity of

lead in the atmosphere have been those of Legge. His statistics correlated with the findings of Duckering on the quantity of lead in the atmosphere led him (Legge) and others to believe that "2 mg. per day is the lowest dose which when inhaled as dust or fumes may in the course of years set up lead poisoning."<sup>11</sup> It appears from our findings that workers exposed to an average amount of 0.009 mg. of lead per cubic foot of air in the casting room do not present evidences of chronic lead poisoning when the duration of exposure is one and one-half years or less; but when the duration of occupation is two and one-half years or more, evidences of chronic lead poisoning are present. We realize the rather limited amount of data which we present on this point, but we feel that our conclusions more nearly harmonize with those of Teleky, who, according to Oliver,<sup>12</sup> is of the opinion that the ingestion of a little more than 1 mg. of lead per day for several months causes plumbism. Based on Legge's figures for the total amount of air respired in a working day of eight hours, our air analyses in the casting shop indicate that the inhalation of 1.45 mg. of lead per day produces lead poisoning within two and one-half years.

Our studies clearly indicate that the exposure to lead in this plant constitutes a real hazard and that this hazard appears to be variable throughout the different workrooms. In all the workrooms there appears to be an unusual incidence of lead poisoning, with the exception of those rooms in which lead is not handled or is handled only to a very limited extent. Even those workers whose duties bring them into the various workrooms intermittently, such as machinists and millwrights, have suffered in the past from acute lead poisoning and at the present time, in one instance at least, from chronic lead poisoning.

One further interesting and important finding which our study disclosed is that in the assembly room where the amount of lead in the air was found to be 0.0096 mg. per cubic foot, an amount only slightly in excess of that found in the casting room (0.009 mg. per cubic foot), the amount of lead poisoning was considerably more than in the casting room. In the assembly room we considered 7 of the 10 men examined to be suffering from chronic lead poisoning, whereas in the casting room but 4 men of 12 examined were considered to be suffering from this disease. And, moreover, of these 4 men in the casting room who are now suffering from chronic lead poisoning, 2 suffered from acute lead poisoning at some previous time, due to working in some other workroom; whereas of the 7 assembly room workers who are at present suffering from chronic lead poisoning, there is only one case of possible acute lead poisoning attributable to exposure in some other workroom. In addition to this, our data

<sup>11</sup> Legge, T. M., and Goadby, K. W.: *Bleivergiftung und Bleiaufnahme*. Julius Springer, Berlin. 1921.

<sup>12</sup> Oliver, T.: *Lead Poisoning*. London and New York. 1914.

indicate that lead poisoning is brought about in the assembly room in a considerably shorter time than it is in the casting room. Of the 7 men considered positive, 5 have been employed  $1\frac{1}{2}$  years or less, whereas in the casting room 3 of the 4 men who are positive have been employed for periods of 8 years, 6 years, and 7 years, respectively. We realize that our evidence on this point is rather limited, but feel that it is indicative in a general way that the greater amount of poisoning and its more rapid course in the assembly room is to be attributed to the more finely divided state of the lead which is encountered by the workmen in this room. The lead in this case is probably in the form of lead vapor or very finely divided lead oxide, and this, we believe, is accountable for the higher incidence of poisoning in this shop than we have found in the casting shop.

Structural changes in the buildings and equipment and changes in the method of handling the material, as well as the installation of certain ventilation systems, have been suggested for the improvement of conditions in this plant in an effort to eliminate the lead poisoning hazard. We have recommended the alteration of the door connecting the casting and the pasting and mixing rooms in order to prevent the transport of dust from these latter rooms into the casting room. Certain recommendations have been suggested for the handling and replacing of trimmings in the melting pot; and the use of chimneys and hoods over the casting pots has been recommended, in an effort to prevent the possibility of the release of lead vapor in the atmosphere.

We have further recommended, in connection with the mixing and pasting operations, that these two be completely separated by constructing a partition across the mixing room portion of the present workroom. Structural changes have been suggested for the rearrangement of the bucket elevator. It has further been suggested that the present dry mixing machines be discarded and replaced by some completely inclosed type.

In connection with the pasting room, we urge the removal of all stored material from this workroom, that the workroom floor be completely scraped and covered with a fresh covering of smooth cement, and that all workbenches be covered with sheet metal and have a raised ledge about their perimeter so as to prevent the dropping of material from the bench to the floor. Each workbench should further be provided with a glazed stoneware crock of a satisfactory size to be used as a receptacle for the paste by the pasting operator. The floor of the workroom should be washed down at the close of each workday by means of a hose and metal scraper when none of the workmen are present.

It is urged that the circular saw used for the cutting of the grids into individual plates be properly inclosed and provided with a suitable exhaust fan.



In the assembly room we have made two major suggestions. The first of these is the provision of tables having slotted or grilled tops provided with exhaust ventilation so that all of the dust produced above these tables may be removed by the exhaust system. For the removal of the lead vapor in the burning operation we urge the provision of an exhaust ventilation duct along the whole length of the table and at a level slightly above the racks. This duct should be provided with a suction velocity of approximately 1,000 cubic feet a minute.

It is to be pointed out in closing that plant hygiene as well as personal hygiene is of the utmost importance in connection with industries handling lead in any form. It is by strict attention to these seemingly insignificant but actually very important details of hygiene that a lead hazard may be held in complete control in a plant such as that under consideration. Whenever sweeping is done in a lead plant the opportunity for the inhalation and ingestion of lead is increased and, therefore, sweeping should be carried on only when none of the regular workers are present. To protect the cleaners the floors of the workroom should be always cleaned by some wet method.

For a long time the plant under consideration was not equipped with clothing lockers nor shower baths for the workmen. During the course of our study a change room was constructed in the free story of the assembly room. Here clothing lockers and shower baths for the men were erected, as well as sinks for ordinary washing purposes. This was an excellent step in the promotion of personal hygiene. It remains however, to point out that in most modern plants two lockers are usually provided for the workers—one in which they may keep their street clothes and the second in which they may keep their working clothes. By this procedure the street clothes of the men do not become contaminated with lead.

The provision of shower baths is certainly a commendable step in the promotion of personal hygiene. It is necessary, however, that the men make use of these facilities, and every effort should be made by the management to see that the men take shower baths before leaving the plant at night. Further steps, too, should be taken to enlighten the employees concerning the care which they should exhibit in matters regarding personal cleanliness, and they should, moreover, be instructed in such matters of personal hygiene as will minimize the possibility of lead being taken into the body. It should be pointed out that the chewing of tobacco or smoking in the workroom furthers the opportunity for lead to be taken into the mouth, and that thorough washing of the hands and face prior to eating also serves to lessen the possibilities in this direction. The eating of lunch or food of any kind in any workroom of the plant should be

prohibited, and this rule should be enforced, even to the extent of dismissing employees who willfully continue to neglect it. Only by the setting aside of a room or eating place for this specific purpose is it possible to enforce such a rule, and we feel that this should be done at once.

And, lastly, it remains to point out that one method exists by which it is possible in practically all cases to eliminate acute lead poisoning among the workers. This is by the practice of periodic physical examinations, with particular emphasis on the study of the blood of the workers at such times. By keeping continuous records of the conditions of the workers and particularly of their blood studies the plant physician will be enabled to compare the changes taking place from time to time and thus be warned of a possible oncoming attack of acute disease. It will be possible then to forestall acute lead poisoning by transferring the worker to some room in which he will not be exposed or by a short lay-off from work.

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### COURT DECISION RELATING TO PUBLIC HEALTH

*City's sewage disposal plant not enjoined.*—(Texas Court of Civil Appeals; *City of Wylie et al. v. Stone*, 16 S. W. (2d) 862; decided April 10, 1929.) An action was brought against the city of Wylie and its mayor and aldermen to enjoin the operation of the city's sewage-disposal plant and to abate same, on the ground that its operation was a public and private nuisance. The jury's findings justified the conclusion that the plant as operated was a nuisance. The jury also found that the plant could have been located and operated in a practical manner on another site that would not have caused injuries to others similar to those inflicted on plaintiff; that plaintiff's land was depreciated in market value \$5,000; and that the city would suffer \$8,000 loss if an injunction were granted. The trial court abated the plant as a nuisance, enjoining the defendants from operating same at the location complained of. On appeal the judgment of the trial court was reversed by the court of civil appeals.

One of defendants' contentions was that, the legislature having authorized the governing authorities of the city to locate and establish the sewage plant, the nuisance, if any resulted from its operation, was legalized. The appellate court, however, held that the nuisance complained of did not belong to the category of those legalized, but, in a proper case, could be enjoined.

Another contention of defendants was that the trial court had erred in not directing a verdict in their favor because the evidence failed to show the existence of a nuisance, but this was overruled, for

the reason that the jury's findings justified the conclusion that the disposal plant as operated was a nuisance.

In passing on another point raised by the defendants the court of appeals decided that "the jury should have been required to find in substance whether or not, under all the facts and circumstances, there existed a reasonable necessity for the location of the disposal plant at the point selected."

It was also contended that the plaintiff was not entitled to injunctive relief as a matter of right, but should have been relegated to his action for damages. In discussing this phase of the matter the court said:

It does not follow that every nuisance will be enjoined; courts will consider the equities pro and con. Ordinarily, where there exists an adequate remedy at law, a nuisance will not be suppressed by injunction; especially is this true in cases of the nature of the one under consideration, where the interest and convenience of an entire community is involved as against a private or personal inconvenience or loss that may be measured in dollars and cents. \* \* \*

One of the major functions of a municipal government is to insure proper sanitation and preserve public health; to this end the law, by express provisions of the statute and by many implications therefrom, has committed to governing authorities of cities, such as Wylie, the duty of establishing and operating sewer systems. They are authorized to purchase necessary lands, either within or without the city limits (art. 1108, R. S. 1925), and, when necessary, may acquire lands by condemnation proceedings (art. 1109b, R. S. 1925). Therefore, wherever these authorities act the presumption will be indulged, until overcome by a preponderance of evidence, that they acted in good faith, with due regard to the rights and interest of all concerned, and found a reasonable necessity for making the particular location. Courts are not authorized to substitute theirs for the judgment of authorities whom the law has clothed with exclusive authority to act, and their discretion, when exercised, should not be disturbed except in a clear case of abuse, which we do not find was committed in this case.

## DEATHS DURING WEEK ENDED JUNE 29, 1929

*Summary of information received by telegraph from industrial insurance companies for the week ended June 29, 1929, and corresponding week of 1928. (From the Weekly Health Index, July 3, 1929, issued by the Bureau of the Census, Department of Commerce)*

	Week ended June 29, 1929	Corresponding week, 1928
Policies in force.....	74, 143, 542	71, 497, 850
Number of death claims.....	13, 474	13, 084
Death claims per 1,000 policies in force, annual rate.....	9. 5	9. 6

Deaths from all causes in certain large cities of the United States during the week ended June 29, 1929, infant mortality, annual death rate, and comparison with corresponding week of 1928. (From the Weekly Health Index, July 3, 1929, issued by the Bureau of the Census, Department of Commerce)

City	Week ended June 29, 1929		Annual death rate per 1,000, corresponding week, 1928	Deaths under 1 year		Infant mortality rate, week ended June 29, 1929 <sup>1</sup>
	Total deaths	Death rate <sup>1</sup>		Week ended June 29, 1929	Corresponding week, 1928	
Total (64 cities).....	6,236	11.0	11.3	569	689	49
Akron.....	37			5	7	52
Albany.....	40	17.4	12.6	3	0	59
Atlanta.....	72	14.8	16.2	12	13	125
White.....	37			7	6	
Colored.....	35	( <sup>2</sup> )	( <sup>2</sup> )	5	7	
Baltimore.....	182	11.5	11.3	15	18	48
White.....	138			11	10	44
Colored.....	44	( <sup>2</sup> )	( <sup>2</sup> )	4	8	63
Birmingham.....	57	13.4	16.5	2	10	18
White.....	24			1	3	15
Colored.....	33	( <sup>2</sup> )	( <sup>2</sup> )	1	7	23
Boston.....	182	11.9	13.6	13	31	36
Bridgeport.....	27			4	6	69
Buffalo.....	147	13.8	9.5	11	16	47
Cambridge.....	22	9.1	9.1	5	3	90
Camden.....	24	9.3	16.6	2	4	35
Canton.....	10	4.5	8.1	1	6	24
Chicago.....	616	10.2	10.0	64	42	57
Cincinnati.....	131			12	12	70
Cleveland.....	153	7.9	9.4	14	23	41
Columbus.....	77	13.5	14.9	6	5	56
Dallas.....	45	10.8	11.5	5	11	
White.....	37			5	7	
Colored.....	8	( <sup>2</sup> )	( <sup>2</sup> )	0	4	
Dayton.....	52	14.7	9.1	7	1	111
Denver.....	87	15.5	13.0	4	7	39
Des Moines.....	32	11.0	10.0	6	5	108
Detroit.....	317	12.0	8.9	45	30	72
Duluth.....	25	11.2	9.4	1	1	24
El Paso.....	38	16.9	12.9	5	9	
Erie.....	22			3	5	61
Fall River.....	17	6.6	10.1	2	4	38
Flint.....	35	12.3	5.6	3	4	36
Fort Worth.....	30	9.2	10.4	3	2	
White.....	25			2	1	
Colored.....	5	( <sup>2</sup> )	( <sup>2</sup> )	1	1	
Grand Rapids.....	22	7.0	8.6	1	6	15
Houston.....	84			8	11	
White.....	61			7	10	
Colored.....	23	( <sup>2</sup> )	( <sup>2</sup> )	1	1	
Indianapolis.....	90	12.3	10.7	4	6	32
White.....	74			4	5	37
Colored.....	16	( <sup>2</sup> )	( <sup>2</sup> )	0	1	0
Jersey City.....	63	10.1	13.0	5	11	39
Kansas City, Kans.....	27	11.9	10.2	0	2	0
White.....	16			0	1	0
Colored.....	11	( <sup>2</sup> )	( <sup>2</sup> )	0	1	0
Kansas City, Mo.....	96	12.8	12.3	11	8	93
Knoxville.....	19	9.4	7.4	3	4	66
White.....	15			3	2	73
Colored.....	4	( <sup>2</sup> )	( <sup>2</sup> )	0	2	0
Los Angeles.....	280			15	22	44
Louisville.....	86	13.7	12.7	7	5	57
White.....	69			6	3	56
Colored.....	17	( <sup>2</sup> )	( <sup>2</sup> )	1	2	63
Lowell.....	17			1	4	23
Lynn.....	15	7.4	8.9	1	0	27
Memphis.....	88	24.2	19.2	5	8	59
White.....	47			2	4	38
Colored.....	41	( <sup>2</sup> )	( <sup>2</sup> )	3	4	94

<sup>1</sup> Annual rate per 1,000 population.

<sup>2</sup> Deaths under 1 year per 1,000 births. Cities left blank are not in the registration area for births.

<sup>3</sup> Data for 72 cities.

<sup>4</sup> Deaths for week ended Friday.

<sup>5</sup> In the cities for which deaths are shown by color, the colored population in 1920 constituted the following percentages of the total population: Atlanta, 31; Baltimore, 15; Birmingham, 38; Dallas, 15; Fort Worth, 14; Houston, 25; Indianapolis, 11; Kansas City, Kans., 14; Knoxville, 15; Louisville, 17; Memphis, 38; Nashville, 30; New Orleans, 26; Richmond, 32; and Washington, D. C. 25.

Deaths from all causes in certain large cities of the United States during the week ended June 29, 1929, infant mortality, annual death rate, and comparison with corresponding week of 1928. (From the Weekly Health Index, July 3, 1929, issued by the Bureau of the Census, Department of Commerce)—Continued

City	Week ended June 29, 1929		Annual death rate per 1,000, corresponding week, 1928	Deaths under 1 year		Infant mortality rate, week ended June 29, 1929
	Total deaths	Death rate		Week ended June 29, 1929	Corresponding week, 1928	
Milwaukee.....	82	7.9	9.3	20	12	88
Minneapolis.....	73	8.4	8.3	3	4	19
Nashville.....	59	22.1	17.2	10	5	161
White.....	31			5	3	109
Colored.....	28	( <sup>9</sup> )	( <sup>9</sup> )	5	2	314
New Bedford.....	17			1	2	21
New Haven.....	37	10.3	9.4	3	3	46
New Orleans.....	136	16.6	16.8	15	11	74
White.....	66			6	1	42
Colored.....	70	( <sup>9</sup> )	( <sup>9</sup> )	9	10	151
New York.....	1,206	10.5	11.9	99	162	41
Bronx Borough.....	143	7.9	8.8	3	7	9
Brooklyn Borough.....	414	9.4	10.8	41	65	42
Manhattan Borough.....	453	14.4	16.8	38	67	46
Queens Borough.....	135	8.3	7.5	15	19	61
Richmond Borough.....	31	10.8	16.0	2	4	36
Newark, N. J.....	78	8.6	11.5	5	8	26
Oakland.....	53	10.1	10.3	4	1	44
Oklahoma City.....	33			5	3	100
White.....	42	9.9	9.6	3	3	35
Colored.....	26	9.4	9.0	4	0	71
Philadelphia.....	417	10.6	11.5	33	53	47
Pittsburgh.....	131	10.2	10.6	8	16	28
Portland, Oreg.....	69			5	2	57
Providence.....	43	7.9	12.1	10	14	88
Richmond.....	56	15.1	10.5	3	6	42
White.....	37			1	3	21
Colored.....	19	( <sup>9</sup> )	( <sup>9</sup> )	2	3	82
Rochester.....	59	9.4	9.7	9	1	76
St. Louis.....	192	11.8	12.8	14	21	47
St. Paul.....	40			1	5	10
Salt Lake City <sup>4</sup> .....	35	13.3	12.9	2	2	31
San Antonio.....	59	14.1	13.2	11	15	—
San Diego.....	39			2	1	38
San Francisco.....	135	12.1	10.8	6	10	38
Schenectady.....	15	8.4	11.2	4	6	127
Seattle.....	83	11.3	10.6	5	4	53
Somerville.....	9	4.6	8.7	0	0	0
Spokane.....	27	12.9	15.8	1	1	26
Springfield, Mass.....	29	10.1	9.1	1	2	17
Syracuse.....	59	13.1	12.3	2	1	24
Tacoma.....	33	15.6	11.8	1	1	26
Toledo.....	64	10.7	11.9	8	7	75
Trenton.....	27	10.2	15.0	3	6	54
Washington, D. C.....	101	9.6	12.4	6	10	35
White.....	57			2	4	17
Colored.....	44	( <sup>9</sup> )	( <sup>9</sup> )	4	6	76
Waterbury.....	9			0	3	0
Wilmington, Del.....	29	11.8	11.8	3	3	78
Worcester.....	40	10.6	10.8	3	5	38
Yonkers.....	13	5.6	12.5	1	1	23
Youngstown.....	31	9.3	9.9	3	1	43

<sup>4</sup> Deaths for week ended Friday.

<sup>5</sup> In the cities for which deaths are shown by color, the colored population in 1920 constituted the following percentages of the total population: Atlanta, 31; Baltimore, 15; Birmingham, 39; Dallas, 15; Fort Worth, 14; Houston, 25; Indianapolis, 11; Kansas City, Kans., 14; Knoxville, 15; Louisville, 17; Memphis, 38; Nashville, 30; New Orleans, 26; Richmond, 32; and Washington, D. C., 25.

# PREVALENCE OF DISEASE

*No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring*

## UNITED STATES

### CURRENT WEEKLY STATE REPORTS

These reports are preliminary and the figures are subject to change when later returns are received by the State health officers

Reports for Weeks Ended June 29, 1929, and June 30, 1928

*Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended June 29, 1929, and June 30, 1928*

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928
<b>New England States:</b>								
Maine.....		1		5	84	24	1	0
New Hampshire.....					75	36	0	0
Vermont.....		1			1	56	0	0
Massachusetts.....	67	56		9	408	524	7	1
Rhode Island.....	5	4		1	26	194	0	0
Connecticut.....	29	13	1	1	49	247	1	1
<b>Middle Atlantic States:</b>								
New York.....	275	314	17	16	586	1,647	6	29
New Jersey.....	69	126		1	104	795	3	6
Pennsylvania.....	133	150			820	1,685	7	9
<b>East North Central States:</b>								
Ohio.....	55	84	4	50	878	995	7	7
Indiana.....	11	16		12	98	196	0	0
Illinois.....	155	130	8	93	1,114	185	12	8
Michigan.....	94	62		3	445	594	53	6
Wisconsin.....	18	8	5	75	761	41	7	0
<b>West North Central States:</b>								
Minnesota.....	11	19	3	2	127	39	2	1
Iowa.....	4	4			52	9	0	0
Missouri.....	38	22			38	235	8	3
North Dakota.....					64	7		0
South Dakota.....		1			8	9	1	0
Nebraska.....	7	3		7	46	34	1	1
Kansas.....	13	6	1		337	58	4	0
<b>South Atlantic States:</b>								
Delaware.....		1			8	35	0	0
Maryland <sup>1</sup> .....	24	16	5	8	15	135	0	1
District of Columbia.....	4	5		1	13	95	0	0
West Virginia.....	7	4	12	41	92	24	1	0
North Carolina.....	21	13			12	131	0	0
South Carolina.....	10	2	89	132		27	0	0
Georgia.....	9	2	9	19	21	24	1	0
Florida.....	5	3	4	16	12	21	0	0

<sup>1</sup> New York City only.

<sup>2</sup> Week ended Friday.

Cases of certain communicable diseases reported by telegraph by State health officers  
for weeks ended June 29, 1929, and June 30, 1928—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928
<b>East South Central States:</b>								
Kentucky.....	5	1			20	121	0	3
Tennessee.....	5	4	5	45	6	42	2	1
Alabama.....	17	7	8	60	33	106	0	1
Mississippi.....	7	3					0	0
<b>West South Central States:</b>								
Arkansas.....	8	2	11	10	5	61	3	1
Louisiana.....	8	16	9	8	34	20	2	0
Oklahoma <sup>1</sup> .....	1	7	3	25	16	70	2	0
Texas.....	17	6	8	35	68	104	2	0
<b>Mountain States:</b>								
Montana.....	1	2			15	4	2	0
Idaho.....					8		0	1
Wyoming.....		3		2	13		0	0
Colorado.....	4	4	1		13	9	2	0
New Mexico.....	2				11	16	1	0
Arizona.....	3	4				6	2	2
Utah <sup>1</sup> .....	3	2	4	9	8		2	1
<b>Pacific States:</b>								
Washington.....	15	3			81	48	3	0
Oregon.....	5	5	9	2	87	37	0	0
California.....	58	79	21	18	96	38	6	3
Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928
<b>New England States:</b>								
Maine.....	0	0	8	13	0	0	10	5
New Hampshire.....	0	0	8	13	0	0	0	0
Vermont.....	0	0	3	6	0	9	9	1
Massachusetts.....	2	2	106	166	0	0	3	21
Rhode Island.....	0	0	4	20	0	0	1	1
Connecticut.....	2	1	16	27	0	1	0	2
<b>Middle Atlantic States:</b>								
New York.....	3	7	150	214	1	7	17	11
New Jersey.....	0	2	49	146	0	0	8	5
Pennsylvania.....	0	1	190	221	0	1	31	9
<b>East North Central States:</b>								
Ohio.....	0	3	128	99	46	13	20	7
Indiana.....	0	1	47	34	65	35	3	3
Illinois.....	2	1	203	149	79	28	10	15
Michigan.....	0	0	165	138	67	24	5	6
Wisconsin.....	0	0	90	61	14	13	1	1
<b>West North Central States:</b>								
Minnesota.....	0	0	56	66	7	2	3	1
Iowa.....	0	0	21	22	22	23	2	0
Missouri.....	1	0	20	42	16	24	11	11
North Dakota.....	0	1	21	12	11	4	1	0
South Dakota.....	0	1	5	10	23	5	0	1
Nebraska.....	0	0	15	19	28	37	1	5
Kansas.....	0	1	53	37	44	78	8	5
<b>South Atlantic States:</b>								
Delaware.....	0	0		4	0	0	2	0
Maryland <sup>1</sup> .....	0	3	30	17	0	0	5	11
District of Columbia.....	0	0	5	21	0	0	1	0
West Virginia.....	0	0	9	13	17	8	8	10
North Carolina.....	5	2	13	15	0	12	36	18
South Carolina.....	1	2	4		1	0	59	59
Georgia.....	0	0	9	5	0	0	41	35
Florida.....	0	0	2	2	0	10	3	6

<sup>1</sup> Week ended Friday.<sup>2</sup> Figures for 1929 are exclusive of Oklahoma City and Tulsa.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended June 29, 1929, and June 30, 1928—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928	Week ended June 29, 1929	Week ended June 30, 1928
<b>East South Central States:</b>								
Kentucky.....	0	0	31	24	6	11	4	8
Tennessee.....	2	0	5	9	0	8	36	33
Alabama.....	3	1	7	2	0	20	46	38
Mississippi.....	0	0	5	2	1	3	41	21
<b>West South Central States:</b>								
Arkansas.....	0	0	9	5	5	11	17	13
Louisiana.....	0	1	10	5	0	8	19	39
Oklahoma <sup>1</sup> .....	0	1	20	14	25	51	13	27
Texas.....	1	0	24	31	15	31	11	13
<b>Mountain States:</b>								
Montana.....	0	0	2	4	6	9	5	3
Idaho.....	0	0	1	2	9	1	0	0
Wyoming.....	0	0	6	1	12	0	4	0
Colorado.....	0	0	7	31	0	3	5	1
New Mexico.....	0	0	2	3	2	0	2	1
Arizona.....	0	0	1	4	1	3	38	1
Utah <sup>1</sup> .....	0	0	13	2	12	11	0	2
<b>Pacific States:</b>								
Washington.....	0	1	15	8	35	35	4	1
Oregon.....	0	3	11	7	19	20	1	2
California.....	3	8	185	107	18	18	11	9

<sup>1</sup> Week ended Friday.

<sup>2</sup> Figures for 1929 are exclusive of Oklahoma City and Tulsa.

**SUMMARY OF MONTHLY REPORTS FROM STATES**

The following summary of monthly State reports is published weekly and covers only those States from which reports are received during the current week:

State	Menin- gococ- cus menin- gitis	Diph- theria	Influ- enza	Ma- laria	Meas- les	Pellag- ra	Pollo- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
<i>April, 1929</i>										
Hawaii Territory....	25	31	256	-----	95	-----	1	11	0	7
<i>May, 1929</i>										
California.....	108	268	164	10	651	4	15	2, 116	303	48
Colorado.....	14	30	1	-----	172	-----	0	98	75	5
Idaho.....	19	6	23	-----	103	-----	2	40	48	4
Indiana.....	4	50	21	-----	2, 375	-----	1	1, 080	322	21
Kansas.....	13	20	5	1	3, 005	-----	0	442	266	12
Mississippi.....	1	25	831	8, 252	1, 211	2, 494	2	22	7	95
Montana.....	10	5	1	-----	449	-----	1	66	40	3
Pennsylvania.....	61	636	-----	1	8, 531	-----	12	1, 685	0	92
South Dakota.....	4	11	-----	-----	209	-----	2	79	164	3
Virginia.....	13	52	512	59	851	46	0	119	2	48
Washington.....	34	33	59	-----	1, 080	-----	0	133	214	16
Wisconsin.....	25	120	121	-----	7, 756	-----	2	745	80	21



<i>April, 1929</i>		Cases	Mumps:	Cases
<b>Hawaii Territory:</b>			California.....	2905
Chicken pox.....	37		Colorado.....	168
Conjunctivitis, follicular.....	8		Idaho.....	18
Diphtheria.....	31		Indiana.....	17
Dysentery (amebic).....	2		Kansas.....	472
Hookworm disease.....	4		Mississippi.....	381
Impetigo contagiosa.....	1		Montana.....	40
Influenza.....	256		Pennsylvania.....	1419
Leprosy.....	11		South Dakota.....	39
Mumps.....	10		Washington.....	349
Tetanus.....	2		Wisconsin.....	489
Whooping cough.....	426		<b>Ophthalmia neonatorum:</b>	
			California.....	5
			Mississippi.....	10
			Pennsylvania.....	18
			Wisconsin.....	3
			<b>Paratyphoid fever:</b>	
			Washington.....	1
			<b>Puerperal septiceemia:</b>	
			Mississippi.....	30
			Pennsylvania.....	21
			Washington.....	2
			<b>Rabies in animals:</b>	
			California.....	74
			Mississippi.....	7
			<b>Rabies in man:</b>	
			Colorado.....	1
			<b>Rocky Mountain spotted or tick fever:</b>	
			California.....	4
			Colorado.....	5
			Idaho.....	11
			Montana.....	5
			Washington.....	3
			<b>Septic sore throat:</b>	
			Idaho.....	1
			Indiana.....	1
			Montana.....	1
			<b>Tetanus:</b>	
			California.....	6
			Pennsylvania.....	8
			Washington.....	1
			<b>Trachoma:</b>	
			California.....	13
			Colorado.....	1
			Kansas.....	1
			Mississippi.....	6
			South Dakota.....	2
			Wisconsin.....	1
			<b>Tularaemia:</b>	
			California.....	2
			Virginia.....	1
			<b>Typhus fever:</b>	
			Virginia.....	3
			<b>Undulant fever:</b>	
			California.....	4
			Colorado.....	1
			Kansas.....	1
			Montana.....	2
			<b>Vincent's angina:</b>	
			Kansas.....	1
			<b>Whooping cough:</b>	
			California.....	1,535
			Colorado.....	77
			Idaho.....	6
			Indiana.....	212
<i>May, 1929</i>				
<b>Actinomycosis:</b>				
California.....	1			
<b>Anthrax:</b>				
Pennsylvania.....	1			
<b>Chicken pox:</b>				
California.....	2,826			
Colorado.....	269			
Idaho.....	16			
Indiana.....	303			
Kansas.....	287			
Mississippi.....	588			
Montana.....	101			
Pennsylvania.....	2,171			
South Dakota.....	21			
Virginia.....	467			
Washington.....	539			
Wisconsin.....	1,463			
<b>Dengue:</b>				
Mississippi.....	7			
<b>Dysentery:</b>				
California (amebic).....	11			
California (bacillary).....	6			
Mississippi (amebic).....	222			
Mississippi (bacillary).....	2,152			
Pennsylvania.....	1			
<b>Dysentery and diarrhea:</b>				
Virginia.....	334			
<b>German measles:</b>				
California.....	103			
Colorado.....	28			
Kansas.....	176			
Montana.....	2			
Pennsylvania.....	566			
Washington.....	21			
Wisconsin.....	69			
<b>Granuloma coccidioidal:</b>				
California.....	5			
<b>Hookworm disease:</b>				
California.....	4			
Mississippi.....	420			
<b>Impetigo contagiosa:</b>				
Colorado.....	1			
<b>Leprosy:</b>				
California.....	2			
<b>Lethargic encephalitis:</b>				
California.....	4			
Kansas.....	2			
Pennsylvania.....	11			
Washington.....	7			
Wisconsin.....	3			

Whooping cough—Continued.	Cases	Whooping cough—Continued.	Cases
Kansas.....	255	South Dakota.....	13
Mississippi.....	1,687	Virginia.....	858
Montana.....	7	Washington.....	405
Pennsylvania.....	1,795	Wisconsin.....	966

**PLAGUE-INFECTED GROUND SQUIRRELS IN CALIFORNIA**

The director of public health of the State of California reports that on June 25, 1929, plague infection was proved in one ground squirrel from a ranch 7 miles south of Arroyo Grande, San Luis Obispo County. On June 27, plague infection was proved in 2 squirrels from a ranch 15 miles east of Santa Ynez, Santa Barbara County.

**GENERAL CURRENT SUMMARY AND WEEKLY REPORTS FROM CITIES**

The 97 cities reporting cases used in the following table are situated in all parts of the country and have an estimated aggregate population of more than 31,490,000. The estimated population of the 90 cities reporting deaths is more than 29,920,000. The estimated expectancy is based on the experience of the last nine years, excluding epidemics.

*Weeks ended June 22, 1929, and June 23, 1928*

	1929	1928	Estimated expectancy
<i>Cases reported</i>			
Diphtheria:			
46 States.....	1,247	1,240	
97 cities.....	677	705	718
Measles:			
45 States.....	7,485	10,772	
97 cities.....	2,567	3,938	
Meningococcus meningitis:			
45 States.....	199	67	
97 cities.....	87	36	
Poliomyelitis:			
46 States.....	22	33	
Scarlet fever:			
46 States.....	2,377	2,113	
97 cities.....	899	856	705
Smallpox:			
44 States.....	635	497	
97 cities.....	55	44	53
Typhoid fever:			
46 States.....	391	397	
97 cities.....	50	39	69
<i>Deaths reported</i>			
Influenza and pneumonia:			
90 cities.....	501	525	
Smallpox:			
90 cities.....	0	0	

## City reports for week ended June 22, 1929

The "estimated expectancy" given for diphtheria, poliomyelitis, scarlet fever, smallpox, and typhoid fever is the result of an attempt to ascertain from previous occurrence the number of cases of the disease under consideration that may be expected to occur during a certain week in the absence of epidemics. It is based on reports to the Public Health Service during the past nine years. It is in most instances the median number of cases reported in the corresponding weeks of the preceding years. When the reports include several epidemics, or when for other reasons the median is unsatisfactory, the epidemic periods are excluded and the estimated expectancy is the mean number of cases reported for the week during non-epidemic years.

If the reports have not been received for the full nine years, data are used for as many years as possible, but no year earlier than 1920 is included. In obtaining the estimated expectancy, the figures are smoothed when necessary to avoid abrupt deviation from the usual trend. For some of the diseases given in the table the available data were not sufficient to make it practicable to compute the estimated expectancy.

Division, State, and city	Population, July 1, 1923, estimated	Chick- en pox, cases re- ported	Diphtheria		Influenza		Meas- les, cases re- ported	Mumps, cases re- ported	Pneu- monia, deaths re- ported
			Cases, esti- mated expect- ancy	Cases re- ported	Cases re- ported	Deaths re- ported			
<b>NEW ENGLAND</b>									
<b>Maine:</b>									
Portland	73,600	1	1	0		0	26	0	1
<b>New Hampshire:</b>									
Concord	( <sup>1</sup> )	0	0	0		0	15	0	1
Manchester	85,700	0	0	0		0	0	0	0
Nashua	( <sup>1</sup> )	0	0	0		0	0	0	0
<b>Vermont:</b>									
Barre	( <sup>1</sup> )	0	0	0		0	0	0	0
<b>Massachusetts:</b>									
Boston	799,200	67	39	17		0	26	38	11
Fall River	134,300	2	3	1		0	3	0	3
Springfield	149,800	20	2	2		0	1	1	0
Worcester	197,600	6	2	1		0	58	1	2
<b>Rhode Island:</b>									
Pawtucket	73,100		0						
Providence	286,300	0	4	1		0	23	1	1
<b>Connecticut:</b>									
Bridgeport	( <sup>1</sup> )	4	4	4		0	9	0	0
Hartford	172,300	3	3	5	1	1	5	7	3
New Haven	187,900	29	0	1		0	7	1	2
<b>MIDDLE ATLANTIC</b>									
<b>New York:</b>									
Buffalo	555,800	31	10	14		0	29	2	9
New York	6,017,500	180	223	191	1	1	72	266	116
Rochester	328,200	4	9	2		0	33	5	5
Syracuse	199,300	20	5	0		0	0	15	1
<b>New Jersey:</b>									
Camden	135,400	5	6	2		0	1	0	0
Newark	473,600	71	10	24		0	1	42	3
Trenton	139,000	0	2	0		0	21	0	2
<b>Pennsylvania:</b>									
Philadelphia	2,064,200	87	56	20	4	3	34	14	31
Pittsburgh	673,800	52	14	4		2	64	8	17
Reading	115,400	11	2	2		0	0	0	1
<b>EAST NORTH CENTRAL</b>									
<b>Ohio:</b>									
Cincinnati	413,700	11	5	4		3	1	2	10
Cleveland	1,010,300	89	23	11	2	2	169	3	10
Columbus	299,000	9	2	3		0	44	1	3
Toledo	313,200	29	4	0		0	129	12	5
<b>Indiana:</b>									
Fort Wayne	105,500	8	2	1		0	15	0	6
Indianapolis	382,150	14	3	0		1	60	2	8
South Bend	86,150	4	1	0		0	0	0	1
Terre Haute	73,550	2	0	0		0	7	0	0
<b>Illinois:</b>									
Chicago	3,157,400	87	65	153	6	3	714	18	53
Springfield	67,200	2	0	0		0	15	0	0

<sup>1</sup>No estimate of population made.

City reports for week ended June 22, 1929—Continued

Division, State, and city	Population, July 1, 1928, estimated	Chick-en pox, cases re-ported	Diphtheria		Influenza		Meas-les, cases re-ported	Mumps, cases re-ported	Pneu-monia, deaths re-ported
			Cases, esti-mated expec-tancy	Cases re-ported	Cases re-ported	Deaths re-ported			
<b>EAST NORTH CENTRAL—continued</b>									
Michigan:									
Detroit.....	1,378,900	65	38	76	-----	2	198	36	14
Flint.....	148,800	14	2	4	-----	0	6	0	3
Grand Rapids.....	164,200	6	1	1	-----	2	17	1	5
Wisconsin:									
Kenosha.....	56,500	7	0	0	1	0	40	4	0
Milwaukee.....	544,200	96	11	5	-----	0	256	19	5
Racine.....	74,400	14	1	0	-----	0	0	1	1
Superior.....	( <sup>1</sup> )	2	1	0	-----	0	11	1	0
<b>WEST NORTH CENTRAL</b>									
Minnesota:									
Duluth.....	114,800	6	1	0	-----	0	9	4	1
Minneapolis.....	455,900	66	12	5	-----	0	27	5	3
St. Paul.....	( <sup>1</sup> )	13	8	0	-----	2	40	11	4
Iowa:									
Davenport.....	( <sup>1</sup> )	0	1	0	-----	-----	1	0	-----
Des Moines.....	151,900	1	1	0	-----	-----	6	0	-----
Sioux City.....	80,000	3	1	0	-----	-----	1	2	-----
Waterloo.....	37,100	3	0	0	-----	-----	3	8	-----
Missouri:									
Kansas City.....	391,000	3	3	4	-----	0	3	0	4
St. Joseph.....	78,500	0	1	0	-----	0	15	0	0
St. Louis.....	848,100	13	26	32	-----	-----	9	8	-----
North Dakota:									
Fargo.....	( <sup>1</sup> )	5	0	0	-----	0	4	0	1
Grand Forks.....	( <sup>1</sup> )	0	0	0	-----	-----	8	0	-----
South Dakota:									
Aberdeen.....	( <sup>1</sup> )	1	0	0	-----	-----	1	2	-----
Sioux Falls.....	( <sup>1</sup> )	0	0	0	-----	-----	1	6	-----
Nebraska:									
Lincoln.....	71,100	6	0	0	-----	0	4	1	0
Omaha.....	222,800	3	2	2	-----	0	52	0	1
Kansas:									
Topeka.....	62,800	9	1	2	-----	0	17	0	0
Wichita.....	99,300	3	0	0	-----	0	82	5	2
<b>SOUTH ATLANTIC</b>									
Delaware:									
Wilmington.....	128,500	2	1	0	-----	0	6	0	1
Maryland:									
Baltimore.....	830,400	41	15	13	3	0	7	91	22
Cumberland.....	( <sup>1</sup> )	0	0	0	1	0	0	0	0
Frederick.....	( <sup>1</sup> )	0	0	0	-----	0	0	0	0
District of Columbia:									
Washington.....	552,000	3	6	10	1	0	13	0	6
Virginia:									
Lynchburg.....	38,600	7	0	0	-----	0	1	28	0
Richmond.....	194,400	5	1	5	1	0	1	1	0
Roanoke.....	64,600	6	0	0	-----	0	3	0	1
West Virginia:									
Charleston.....	55,200	3	1	0	-----	0	8	0	0
Wheeling.....	( <sup>1</sup> )	1	1	0	-----	0	16	0	2
North Carolina:									
Raleigh.....	( <sup>1</sup> )	6	1	0	-----	0	1	0	0
Wilmington.....	39,100	10	0	0	-----	0	0	0	0
Winston-Salem.....	80,000	0	1	0	-----	0	2	0	2
South Carolina:									
Charleston.....	75,900	0	0	0	1	0	0	0	2
Columbia.....	50,600	4	0	0	-----	0	1	5	2
Georgia:									
Atlanta.....	255,100	0	1	1	8	1	9	0	6
Brunswick.....	( <sup>1</sup> )	0	0	0	-----	0	0	0	0
Savannah.....	99,900	0	0	5	-----	0	0	0	1
Florida:									
Miami.....	156,700	0	1	1	-----	0	10	2	0
Tampa.....	113,400	1	1	0	-----	0	2	0	0

<sup>1</sup> No estimate of population made.

## City reports for week ended June 22, 1929—Continued

Division, State, and city	Population, July 1, 1928, estimated	Chick- en pox, cases re- ported	Diphtheria		Influenza		Mes- sles, cases re- ported	Mumps, cases re- ported	Pneu- monia, deaths re- ported
			Cases, esti- mated expect- ancy	Cases re- ported	Cases re- ported	Deaths re- ported			
<b>EAST SOUTH CENTRAL</b>									
<b>Kentucky:</b>									
Covington.....	59,000	0	0	0	0	0	0	0	3
<b>Tennessee:</b>									
Memphis.....	190,200	8	0	1	1	0	1	1	3
Nashville.....	139,600	8	0	1	0	4	0	0	2
<b>Alabama:</b>									
Birmingham.....	222,400	4	1	1	4	1	1	4	7
Mobile.....	69,600	0	0	1	0	0	0	0	1
Montgomery.....	63,100	0	0	1	1	1	0	0	—
<b>WEST SOUTH CENTRAL</b>									
<b>Arkansas:</b>									
Fort Smith.....	( <sup>1</sup> )	0	0	0	0	0	0	2	—
Little Rock.....	79,200	2	0	0	0	0	0	0	1
<b>Louisiana:</b>									
New Orleans.....	429,400	0	4	7	1	3	7	0	10
Shreveport.....	81,300	2	0	0	0	0	2	2	1
<b>Oklahoma:</b>									
Tulsa.....	170,500	3	0	2	—	—	11	0	—
<b>Texas:</b>									
Dallas.....	217,800	1	3	6	1	1	38	0	1
Fort Worth.....	170,600	4	1	1	—	0	3	1	1
Galveston.....	50,600	0	0	0	0	0	0	0	0
Houston.....	( <sup>1</sup> )	0	2	2	—	0	0	0	7
San Antonio.....	218,100	0	2	2	—	0	1	0	1
<b>MOUNTAIN</b>									
<b>Montana:</b>									
Billings.....	( <sup>1</sup> )	4	0	0	—	0	2	1	1
Great Falls.....	( <sup>1</sup> )	4	0	0	—	0	6	2	0
Helena.....	( <sup>1</sup> )	0	1	0	—	0	0	0	0
Missoula.....	( <sup>1</sup> )	0	0	0	—	0	0	0	0
<b>Idaho:</b>									
Boise.....	( <sup>1</sup> )	0	0	0	—	0	4	0	2
<b>Colorado:</b>									
Denver.....	294,200	14	9	3	—	0	9	11	4
Pueblo.....	44,200	2	1	0	—	0	2	0	0
<b>New Mexico:</b>									
Albuquerque.....	( <sup>1</sup> )	6	0	0	—	0	1	0	1
<b>Utah:</b>									
Salt Lake City.....	138,000	14	3	0	—	0	2	65	0
<b>Nevada:</b>									
Reno.....	( <sup>1</sup> )	0	0	0	—	0	0	0	2
<b>PACIFIC</b>									
<b>Washington:</b>									
Seattle.....	383,200	14	3	1	—	—	13	13	—
Spokane.....	109,100	12	2	0	—	—	59	0	—
Tacoma.....	110,500	6	2	1	—	0	0	0	3
<b>Oregon:</b>									
Salem.....	( <sup>1</sup> )	2	0	3	—	0	3	2	0
<b>California:</b>									
Los Angeles.....	( <sup>1</sup> )	65	37	21	13	2	33	31	19
Sacramento.....	75,700	2	2	0	1	0	23	1	6
San Francisco.....	585,300	22	12	1	—	0	18	34	5

<sup>1</sup> No estimate of population made.

City reports for week ended June 22, 1929—Continued

Division, State, and city	Scarlet fever		Smallpox			Tuberculosis, deaths reported	Typhoid fever			Whooping cough, cases reported	Deaths, all causes
	Cases, estimated expectancy	Cases reported	Cases, estimated expectancy	Cases reported	Deaths reported		Cases, estimated expectancy	Cases reported	Deaths reported		
<b>NEW ENGLAND</b>											
<b>Maine:</b>											
Portland.....	1	3	0	0	0	1	0	0	0	1	18
<b>New Hampshire:</b>											
Concord.....	0	2	0	0	0	1	0	0	0	0	8
Manchester.....	0	0	0	0	0	0	0	0	0	0	15
Nashua.....	0	0	0	0	0	1	0	0	0	0	9
<b>Vermont:</b>											
Barre.....	0	0	0	0	0	0	0	0	0	0	1
<b>Massachusetts:</b>											
Boston.....	43	47	0	0	0	12	2	1	0	31	201
Fall River.....	3	1	0	0	0	5	1	0	0	5	21
Springfield.....	4	4	0	0	0	2	0	0	0	4	30
Worcester.....	7	6	0	0	0	0	0	0	0	16	45
<b>Rhode Island:</b>											
Pawtucket.....	1		0								
Providence.....	5	2	0	0	0	2	0	0	0	14	52
<b>Connecticut:</b>											
Bridgeport.....	5	2	0	0	0	0	0	0	0	0	37
Hartford.....	3	0	0	0	0	2	0	0	0	6	43
New Haven.....	2	3	0	0	0	1	1	1	0	2	38
<b>MIDDLE ATLANTIC</b>											
<b>New York:</b>											
Buffalo.....	16	24	0	0	0	6	1	0	0	0	101
New York.....	145	97	0	0	0	95	13	3	0	71	1,390
Rochester.....	8	4	0	0	0	1	1	0	0	17	64
Syracuse.....	4	4	0	0	0	1	0	0	0	21	38
<b>New Jersey:</b>											
Camden.....	4	2	0	0	0	0	0	1	0	1	28
Newark.....	16	10	0	0	0	4	0	0	1	74	95
Trenton.....	2	2	0	0	0	4	1	0	0	0	35
<b>Pennsylvania:</b>											
Philadelphia.....	54	36	0	0	0	31	3	0	0	70	455
Pittsburgh.....	19	25	0	0	0	12	0	1	1	40	196
Reading.....	1	4	0	0	0	0	0	0	0	1	32
<b>EAST NORTH CENTRAL</b>											
<b>Ohio:</b>											
Cincinnati.....	7	22	1	3	0	9	0	3	0	13	123
Cleveland.....	21	28	0	0	0	20	2	0	0	46	190
Columbus.....	4	3	1	1	0	7	1	1	0	33	69
Toledo.....	8	11	0	0	0	6	0	2	1	65	83
<b>Indiana:</b>											
Fort Wayne.....	1	0	1	1	0	1	0	0	0	1	17
Indianapolis.....	5	27	7	1	0	11	0	0	1	8	106
South Bend.....	1	2	1	0	0	1	0	0	0	2	17
Terre Haute.....	1	3	0	0	0	1	0	0	0	1	15
<b>Illinois:</b>											
Chicago.....	77	179	2	3	0	53	3	0	0	71	731
Springfield.....	1	4	0	2	0	2	0	0	0	1	18
<b>Michigan:</b>											
Detroit.....	51	99	2	7	0	33	2	1	1	94	291
Flint.....	4	22	1	8	0	0	0	0	0	1	26
Grand Rapids.....	5	3	0	0	0	1	0	0	0	16	42
<b>Wisconsin:</b>											
Kenosha.....	0	0	0	0	0	0	0	0	0	5	6
Milwaukee.....	16	15	0	2	0	5	1	1	0	94	87
Racine.....	3	1	0	0	0	1	0	0	0	0	14
Superior.....	2	0	1	0	0	1	0	0	0	12	9
<b>WEST NORTH CENTRAL</b>											
<b>Minnesota:</b>											
Duluth.....	5	3	1	0	0	0	0	0	0	0	27
Minneapolis.....	21	4	1	0	0	3	1	1	0	1	90
St. Paul.....	12	6	1	0	0	5	0	0	0	30	69
<b>Iowa:</b>											
Davenport.....	0	0	0	0	0	0	0	0	0	3	-----
Des Moines.....	2	7	2	0	0	0	0	0	0	1	-----
Sioux City.....	1	0	1	1	0	0	1	0	0	7	-----
Waterloo.....	1	1	1	1	0	0	0	0	0	4	-----

## City reports for week ended June 22, 1929—Continued

Division, State, and city	Scarlet fever		Smallpox			Tuber- cul- osis, deaths re- ported	Typhoid fever			Whoop- ing cough, cases re- ported	Deaths, all causes
	Cases, esti- mated expect- ancy	Cases re- ported	Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported		Cases, esti- mated expect- ancy	Cases re- ported	Deaths re- ported		
<b>WEST NORTH CENTRAL—CON.</b>											
<b>Missouri:</b>											
Kansas City.....	4	9	1	0	0	4	1	1	0	19	86
St. Joseph.....	0	0	2	0	0	2	0	0	0	0	27
St. Louis.....	14	8	2	0	0	8	2	7	1	75	203
<b>North Dakota:</b>											
Fargo.....	1	3	0	0	0	0	0	0	0	5	7
Grand Forks.....	1	0	0	0	0	0	0	0	0	0	0
<b>South Dakota:</b>											
Aberdeen.....	0	1	0	0	0	0	0	0	0	4	0
Sioux Falls.....	0	0	0	6	0	0	0	0	0	0	6
<b>Nebraska:</b>											
Lincoln.....	1	7	0	0	0	0	0	0	0	12	0
Omaha.....	2	1	0	1	0	1	0	0	0	4	51
<b>Kansas:</b>											
Topoka.....	1	2	0	0	0	1	0	0	0	15	21
Wichita.....	1	3	0	0	0	0	0	0	0	15	28
<b>SOUTH ATLANTIC</b>											
<b>Delaware:</b>											
Wilmington.....	2	0	0	0	0	1	0	0	0	3	24
<b>Maryland:</b>											
Baltimore.....	14	27	0	0	0	11	3	2	0	105	227
Cumberland.....	0	0	0	0	0	0	0	0	0	1	8
Frederick.....	0	0	0	0	0	0	0	0	0	0	2
<b>District of Colum- bia:</b>											
Washington.....	11	7	0	0	0	11	2	0	0	16	133
<b>Virginia:</b>											
Lynchburg.....	1	0	0	0	0	0	1	0	0	12	10
Richmond.....	1	2	0	0	0	2	1	0	0	4	51
Roanoke.....	0	0	0	0	0	1	0	0	0	4	14
<b>West Virginia:</b>											
Charleston.....	0	0	0	2	0	1	1	0	0	2	6
Wheeling.....	2	0	0	1	0	2	0	0	0	5	15
<b>North Carolina:</b>											
Raleigh.....	0	0	0	0	0	1	0	0	0	7	19
Wilmington.....	0	0	0	0	0	1	0	0	0	2	11
Winston-Salem.....	0	0	0	0	0	1	0	0	0	47	14
<b>South Carolina:</b>											
Charleston.....	0	0	1	0	0	1	1	0	0	2	23
Columbia.....	0	0	0	0	0	2	1	3	0	17	18
<b>Georgia:</b>											
Atlanta.....	3	2	2	0	0	5	2	0	0	24	106
Brunswick.....	0	0	0	0	0	0	0	0	0	0	6
Savannah.....	0	0	0	0	0	2	1	1	0	0	33
<b>Florida:</b>											
Miami.....	0	2	0	0	0	1	0	0	0	4	21
Tampa.....	0	1	0	0	0	0	1	0	0	0	16
<b>EAST SOUTH CENTRAL</b>											
<b>Kentucky:</b>											
Covington.....	0	2	0	0	0	1	0	0	0	0	15
<b>Tennessee:</b>											
Memphis.....	2	4	1	0	0	9	3	4	0	13	67
Nashville.....	1	1	1	0	0	1	2	2	0	3	59
<b>Alabama:</b>											
Birmingham.....	1	2	3	0	0	3	3	1	3	18	68
Mobile.....	0	0	0	0	0	0	2	1	0	0	15
Montgomery.....	0	4	0	0	0	0	0	0	0	0	0
<b>WEST SOUTH CENTRAL</b>											
<b>Arkansas:</b>											
Fort Smith.....	0	0	0	0	0	0	1	0	0	2	0
Little Rock.....	0	1	0	0	0	3	0	1	0	4	0
<b>Louisiana:</b>											
New Orleans.....	2	7	0	0	0	15	3	1	0	7	166
Shreveport.....	0	1	1	0	0	2	0	0	1	5	34





## City reports for week ended June 22, 1929—Continued

Division, State, and city	Meningococcus meningitis		Lethargic encephalitis		Pellagra		Poliomyelitis (infantile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, estimated expectancy	Cases	Deaths
<b>EAST NORTH CENTRAL</b>									
Ohio:									
Cincinnati.....	1	0	0	0	0	0	0	0	0
Cleveland.....	1	0	0	0	0	0	1	0	0
Toledo.....	2	2	1	1	0	0	0	0	0
Indiana:									
Indianapolis.....	1	0	0	0	0	0	0	0	0
Illinois:									
Chicago.....	3	3	0	0	0	0	0	0	0
Michigan:									
Detroit.....	25	10	1	1	0	0	1	0	0
Flint.....	4	3	0	0	0	0	0	0	0
Grand Rapids.....	1	0	0	0	0	0	0	0	0
Wisconsin:									
Milwaukee.....	3	1	0	0	0	0	0	0	0
Racine.....	2	1	0	0	0	0	0	0	0
<b>WEST NORTH CENTRAL</b>									
Minnesota:									
Minneapolis.....	1	0	0	0	0	0	0	0	0
St. Paul.....	1	0	1	1	0	0	0	0	0
Missouri:									
Kansas City.....	2	2	0	0	0	0	0	0	0
St. Louis.....	5	2	0	0	0	0	0	1	0
<b>SOUTH ATLANTIC</b>									
Maryland:									
Baltimore.....	2	1	0	0	0	1	0	0	0
South Carolina:									
Charleston <sup>1</sup> .....	0	0	0	0	3	2	0	0	0
Columbia.....	0	2	0	0	0	0	0	0	0
Georgia:									
Atlanta.....	0	2	0	0	0	0	0	0	0
Savannah <sup>2</sup> .....	0	0	0	0	1	1	0	0	0
Florida:									
Miami.....	0	0	0	0	1	0	0	0	0
<b>EAST SOUTH CENTRAL</b>									
Tennessee:									
Memphis.....	0	1	0	0	0	0	0	0	0
Alabama:									
Birmingham.....	0	0	0	0	1	0	0	0	0
Mobile.....	0	0	0	0	1	0	0	1	0
Montgomery.....	0	0	0	0	1	0	0	0	0
<b>WEST SOUTH CENTRAL</b>									
Louisiana:									
New Orleans.....	1	0	0	0	12	3	0	0	0
Shreveport.....	0	0	0	0	0	1	0	0	0
Texas:									
Dallas.....	1	0	0	0	1	1	0	0	0
Fort Worth.....	0	0	0	0	0	2	0	0	0
Houston.....	0	0	0	0	0	0	0	1	0
San Antonio.....	0	0	0	0	0	0	0	1	0
<b>MOUNTAIN</b>									
Montana:									
Missoula.....	2	0	0	0	0	0	0	0	0
Colorado:									
Pueblo.....	1	0	0	0	0	0	0	0	0
Utah:									
Salt Lake City.....	2	2	0	0	0	0	0	0	0
<b>PACIFIC</b>									
Washington:									
Seattle.....	2	0	0	0	0	0	0	0	0
California:									
Los Angeles.....	3	1	0	0	0	0	1	1	0
San Francisco.....	2	2	1	1	0	0	1	0	0

<sup>1</sup> Dengue: 1 case at Charleston, S. C.<sup>2</sup> Typhus fever: 1 case at Savannah, Ga.

The following table gives the rates per 100,000 population for 98 cities for the 5-week period ended June 22, 1929, compared with those for a like period ended June 23, 1928. The population figures used in computing the rates are approximate estimates, authoritative figures for many of the cities not being available. The 98 cities reporting cases have estimated aggregate populations of more than 31,000,000. The 91 cities reporting deaths have nearly 30,000,000 estimated population. The number of cities included in each group and the estimated aggregate populations are shown in a separate table below.

*Summary of weekly reports from cities, May 19 to June 22, 1929—Annual rates per 100,000 population, compared with rates for the corresponding period of 1928<sup>1</sup>*

DIPHTHERIA CASE RATES

	Week ended—									
	May 25, 1929	May 26, 1928	June 1, 1929	June 2, 1928	June 8, 1929	June 9, 1928	June 15, 1929	June 16, 1928	June 22, 1929	June 23, 1928
98 cities.....	136	131	<sup>2</sup> 125	124	110	136	<sup>3</sup> 107	146	<sup>4</sup> 112	119
New England.....	109	64	<sup>4</sup> 91	99	72	97	79	115	<sup>4</sup> 75	78
Middle Atlantic.....	188	213	168	178	148	221	131	242	125	185
East North Central.....	165	102	155	105	123	108	145	123	164	118
West North Central.....	100	73	110	84	96	53	<sup>6</sup> 65	68	87	63
South Atlantic.....	49	117	41	101	54	107	<sup>5</sup> 64	67	64	61
East South Central.....	14	42	7	63	20	28	41	28	34	14
West South Central.....	47	28	<sup>5</sup> 59	57	91	61	87	53	67	53
Mountain.....	61	71	<sup>6</sup> 38	71	61	35	<sup>7</sup> 36	44	26	35
Pacific.....	62	92	60	107	57	115	35	110	60	72

MEASLES CASE RATES

98 cities.....	906	1,309	<sup>2</sup> 663	1,218	737	1,026	<sup>3</sup> 485	866	<sup>4</sup> 425	663
New England.....	556	1,290	<sup>4</sup> 369	1,129	606	952	339	996	<sup>4</sup> 404	934
Middle Atlantic.....	194	2,192	183	2,170	169	1,771	143	1,403	123	1,106
East North Central.....	2,283	772	1,595	660	1,825	687	1,151	677	1,009	423
West North Central.....	1,440	943	1,032	755	1,059	597	581	534	504	342
South Atlantic.....	242	1,320	298	1,112	238	892	<sup>5</sup> 241	606	129	513
East South Central.....	27	743	54	596	41	435	41	442	41	512
West South Central.....	447	263	245	178	415	61	217	113	190	45
Mountain.....	318	893	<sup>6</sup> 254	992	192	735	<sup>7</sup> 267	682	218	337
Pacific.....	546	304	412	217	422	174	397	110	364	143

SCARLET FEVER CASE RATES

98 cities.....	269	233	<sup>2</sup> 271	209	209	193	<sup>3</sup> 189	167	<sup>4</sup> 149	144
New England.....	283	306	<sup>4</sup> 276	248	192	290	206	223	<sup>4</sup> 164	170
Middle Atlantic.....	196	268	196	201	135	191	129	162	100	146
East North Central.....	448	254	446	227	321	237	321	220	260	181
West North Central.....	208	207	179	233	165	164	110	155	77	139
South Atlantic.....	159	176	274	191	300	157	<sup>5</sup> 135	109	73	98
East South Central.....	136	84	122	365	95	49	75	77	88	49
West South Central.....	122	207	166	146	79	93	111	45	91	45
Mountain.....	113	18	<sup>6</sup> 103	71	78	106	<sup>7</sup> 71	71	96	27
Pacific.....	347	130	254	148	279	156	259	156	217	161

<sup>1</sup> The figures given in this table are rates per 100,000 population, annual basis, and not the number of cases reported. Populations used are estimated as of July 1, 1929 and 1928, respectively.

<sup>2</sup> Pawtucket, R. I., and Pueblo, Colo., not included.

<sup>3</sup> Raleigh, N. C., and Reno, Nev., not included.

<sup>4</sup> Pawtucket, R. I., not included.

<sup>5</sup> Raleigh, N. C., not included.

<sup>6</sup> Pueblo, Colo., not included.

<sup>7</sup> Reno, Nev., not included.

Summary of weekly reports from cities, May 19 to June 22, 1929—Annual rates per 100,000 population, compared with rates for the corresponding period of 1928—Continued

## SMALLPOX CASE RATES

	Week ended—									
	May 25, 1929	May 26, 1928	June 1, 1929	June 2, 1928	June 8, 1929	June 9, 1928	June 15, 1929	June 16, 1928	June 22, 1929	June 23, 1928
98 cities.....	14	17	19	12	8	11	16	10	9	7
New England.....	7	9	0	0	0	0	0	0	0	0
Middle Atlantic.....	0	0	0	0	0	0	0	0	0	0
East North Central.....	20	16	15	10	17	9	28	11	18	8
West North Central.....	15	27	15	29	12	22	12	23	6	23
South Atlantic.....	4	29	0	10	2	31	4	13	6	4
East South Central.....	27	63	7	56	14	35	54	56	0	28
West South Central.....	16	24	20	24	8	24	43	20	4	24
Mountain.....	35	133	56	53	52	71	45	44	61	9
Pacific.....	77	38	27	49	15	13	47	18	32	15

## TYPHOID FEVER CASE RATES

	8	8	7	12	8	9	9	7	8	7
98 cities.....	8	8	7	12	8	9	9	7	8	7
New England.....	7	11	2	57	7	2	11	2	5	9
Middle Atlantic.....	5	6	3	1	5	10	3	2	2	1
East North Central.....	3	5	3	3	3	7	4	3	4	2
West North Central.....	8	4	17	4	8	4	17	4	19	4
South Atlantic.....	15	6	19	17	17	11	9	17	13	13
East South Central.....	75	14	34	91	27	14	34	42	54	49
West South Central.....	12	12	20	32	28	32	20	36	36	28
Mountain.....	17	0	0	0	0	9	9	9	9	0
Pacific.....	10	36	2	18	12	10	20	20	5	15

## INFLUENZA DEATH RATES

	10	26	7	21	7	18	6	12	6	6
91 cities.....	10	26	7	21	7	18	6	12	6	6
New England.....	7	18	7	16	2	14	7	14	2	5
Middle Atlantic.....	8	21	4	24	5	19	4	11	3	9
East North Central.....	8	33	9	21	6	17	8	14	8	6
West North Central.....	15	18	3	21	3	21	9	6	6	0
South Atlantic.....	6	11	6	10	7	10	2	8	6	8
East South Central.....	44	130	0	38	22	77	7	31	15	0
West South Central.....	28	33	12	25	16	33	12	17	16	4
Mountain.....	9	53	19	44	35	0	0	9	0	0
Pacific.....	7	7	16	7	16	7	7	7	7	3

## PNEUMONIA DEATH RATES

	116	181	106	147	91	130	87	115	82	87
91 cities.....	116	181	106	147	91	130	87	115	82	87
New England.....	122	253	108	172	66	168	86	136	56	90
Middle Atlantic.....	129	212	113	183	105	148	98	132	89	110
East North Central.....	118	174	101	129	96	115	82	111	76	59
West North Central.....	123	126	120	89	81	95	54	129	48	64
South Atlantic.....	94	119	112	136	67	132	89	80	84	94
East South Central.....	104	253	111	153	59	161	104	115	118	46
West South Central.....	69	146	69	129	93	108	65	75	85	87
Mountain.....	139	124	122	106	61	89	116	53	78	115
Pacific.....	85	91	66	71	72	81	62	88	108	84

<sup>1</sup> Pawtucket, R. I., and Pueblo, Colo., not included.

<sup>2</sup> Raleigh, N. C., and Reno, Nev., not included.

<sup>3</sup> Pawtucket, R. I., not included.

<sup>4</sup> Raleigh, N. C., not included.

<sup>5</sup> Pueblo, Colo., not included.

<sup>6</sup> Reno, Nev., not included.

*Number of cities included in summary of weekly reports and aggregate population of cities of each group, approximated as of July 1, 1929 and 1928, respectively*

Group of cities	Number of cities reporting cases	Number of cities reporting deaths	Aggregate population of cities reporting cases		Aggregate population of cities reporting deaths	
			1929	1928	1929	1928
Total.....	98	91	31,568,400	31,052,700	29,995,100	29,498,600
New England.....	12	12	2,305,100	2,273,900	2,305,100	2,273,900
Middle Atlantic.....	10	10	10,809,700	10,702,200	10,809,700	10,702,200
East North Central.....	16	16	8,181,900	8,001,300	8,181,900	8,001,300
West North Central.....	12	9	2,712,100	2,673,300	1,736,900	1,708,100
South Atlantic.....	19	19	2,783,200	2,732,900	2,783,200	2,732,900
East South Central.....	6	5	767,900	745,500	704,200	682,400
West South Central.....	8	7	1,319,100	1,289,900	1,285,000	1,256,400
Mountain.....	9	9	598,800	590,200	598,800	590,200
Pacific.....	6	4	2,090,600	2,043,500	1,590,300	1,551,200

## FOREIGN AND INSULAR

### CANADA

*Provinces—Communicable diseases—Week ended June 22, 1929.*—During the week ended June 22, 1929, the Department of Pensions and National Health reported cases of certain communicable diseases from eight Provinces of Canada, as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Alberta	British Columbia	Total
Cerebrospinal meningitis.....					1	1	1	1	4
Influenza.....				2	1				3
Lethargic encephalitis.....								1	1
Smallpox.....				2	21		1	10	34
Typhoid fever.....			1	9	8	2	11		31

*Quebec Province—Communicable diseases—Week ended June 22, 1929.*—The Bureau of Health of the Province of Quebec reports cases of certain communicable diseases for the week ended June 22, 1929, as follows:

Disease	Cases	Disease	Cases
Chicken pox.....	17	Scarlet fever.....	90
Diphtheria.....	44	Smallpox.....	2
German measles.....	18	Tuberculosis.....	45
Influenza.....	2	Typhoid fever.....	9
Measles.....	55	Whooping cough.....	23
Mumps.....	17		

### CUBA

*Provinces—Communicable diseases—March 3–April 13, 1929.*—During the six weeks from March 3 to April 13, 1929, cases of certain communicable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer.....			5	1	4	1	11
Cerebrospinal meningitis.....		1			1		2
Chicken pox.....		37	10	19	16	35	117
Diphtheria.....		31	2	10	6		49
Malaria.....		23		1	12	177	213
Measles.....	25	564	8	25	14		636
Paratyphoid fever.....		1	2	3	1	7	14
Scarlet fever.....	4	13					17
Tetanus (infantile).....						1	1
Typhoid fever.....	16	50	13	29	10	37	155

**CZECHOSLOVAKIA**

*Communicable diseases—April, 1929.*—During the month of April, 1929, communicable diseases were reported in Czechoslovakia, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax.....	2	—	Puerperal fever.....	52	20
Cerebrospinal meningitis.....	28	5	Scarlet fever.....	1,181	23
Diphtheria.....	869	61	Trachoma.....	251	—
Dysentery.....	11	1	Typhoid fever.....	341	31
Malaria.....	4	—	Typhus fever.....	25	—
Paratyphoid fever.....	9	—			

**PORTO RICO**

*San Juan—Communicable diseases—Five weeks ended June 15, 1929.*—During the five weeks ended June 15, 1929, cases of certain communicable diseases were reported in San Juan, P. R., as follows:

Disease	Cases	Disease	Cases
Diphtheria.....	2	Syphilis.....	16
Filariasis.....	1	Tetanus.....	8
Malaria.....	5	Tuberculosis.....	51
Measles.....	24	Typhoid fever.....	6
Mumps.....	1	Whooping cough.....	4
Puerperal fever.....	1		

**STRAITS SETTLEMENTS**

*Vital statistics (comparative)—Year 1928.*—The Straits Settlements annual report on the registration of births and deaths for the year 1928 contains the following statistics:

	1928	1927		1928	1927
Population, estimated.....	1,095,635	1,059,968	Death rate per 1,000 population.....	28.76	33.55
Births.....	39,479	37,233	Infant mortality rate per 1,000 births.....	185.69	214.06
Birth rate per 1,000 population.....	36.03	35.13			
Deaths.....	31,516	35,561			

Deaths from certain diseases were reported during the year 1928 as follows:

Disease	Deaths	Disease	Deaths
Beriberi.....	1,146	Influenza.....	204
Bronchitis.....	502	Malaria.....	5,796
Cerebrospinal fever.....	14	Nephritis.....	824
Cholera.....	23	Plague.....	4
Cirrhosis of the liver.....	128	Pneumonia.....	2,679
Convulsions (infantile).....	5,040	Smallpox.....	2
Diarrhea and enteritis.....	1,230	Syphilis.....	204
Diphtheria.....	21	Tuberculosis, pulmonary.....	2,727
Dysentery.....	902	Typhoid fever.....	174
Heart disease.....	501		

## TRINIDAD

*Port of Spain—Vital statistics (comparative)—May, 1929.*—The following statistics for the month of May, 1929, with figures for May of the years 1925 to 1928, are taken from a report issued by the Public Health Department of Port of Spain, Trinidad:

*Month of May*

	1925	1926	1927	1928	1929
Number of births.....	152	164	135	153	131
Birth rate per 1,000 population.....	27.98	29.92	24.45	27.55	23.23
Number of deaths.....	116	139	121	145	127
Death rate per 1,000 population.....	21.35	25.36	21.91	26.11	23.53
Deaths under 1 year.....	24	29	22	20	14
Infant mortality rate per 1,000 births.....	157.89	177.44	162.96	130.72	106.87

## VIRGIN ISLANDS

*Communicable diseases—May, 1929.*—During the month of May, 1929, cases of certain communicable diseases were reported in the Virgin Islands as follows:

St. Thomas and St. John:	Cases	St. Thomas and St. John—Continued.	Cases
Chancroid.....	1	Tuberculosis.....	4
Dysentery.....	1	Uncinariasis.....	16
Gonorrhoea.....	2	St. Croix:	
Sprue.....	1	Chicken pox.....	1
Syphilis.....	6	Syphilis.....	3
Tetanus.....	2		





Indo-China (see also table below):

Place	Decem-ber, 1928	Janu-ary, 1929	February, 1929			March, 1929			April, 1929			May, 1929					
			1-10	11-20	21-28	1-10	11-20	21-31	1-10	11-20	21-30	1-10	11-20	21-30			
															1-10	11-20	21-30
Pnompenh.....	O	6	2	42	13	3	8	3	3	1	1	6	3	6	2	2	2
Saigon.....	D	1	1	36	10	1	5	2	2	1	1	6	1	4	4	1	1
Slam.....	D	224	195	165	397	2	1	3	3	4	4	17	15	20	19	43	73
Anthoang.....	D	160	138	117	269	3	85	114	86	245	310	196	9	9	113	121	67
Ayudhaya.....	D	16	4	2	1	2	2	3	3	24	2	2	14	14	14	14	14
Bangkok.....	D	22	50	61	35	15	1	3	3	13	36	36	9	20	9	7	8
Charoengso.....	D	6	43	36	21	10	13	31	31	20	34	22	17	4	4	4	4
Dhannapuri.....	D	16	9	1	1	2	2	1	1	15	1	1	1	1	1	1	1
Lobpur.....	D	10	3	3	1	1	1	1	1	6	1	1	1	1	1	1	1
Nagara Pathom.....	D	74	1	1	1	1	1	1	1	35	13	13	10	10	10	10	10
Nondpur.....	D	4	2	2	2	2	2	2	2	20	10	10	10	10	10	10	10
Pradhumdham.....	D	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Singhapuri.....	D	17	6	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Smud Prakar.....	D	30	10	1	1	1	1	1	1	1	1	1	1	1	1	1	1
On vessel:																	
S. S. Augby, at Saigon-Cholon.....	O																
S. S. Ekma, at Penang from Singapore.....	D																
S. S. Edinpara, at Madras.....	O																
S. S. Media, at Colombo from Calcutta.....	O																
S. S. Saka Meru, at Calcutta.....	O																
S. S. Tilawa, at Penang from Singapore.....	O																
S. S. Elephanta, at Penang from Calcutta.....	O																

Place	Decem-ber, 1928	Janu-ary, 1929	February, 1929			March, 1929			April, 1929			May, 1929					
			1-10	11-20	21-28	1-10	11-20	21-31	1-10	11-20	21-30	1-10	11-20	21-30			
															1-10	11-20	21-30
Indo-China (French) (see also table above):																	
Annam.....	C	79	29	40													
Cambodia.....	C	25	107	115													
Cochin-China.....	C	697	107	115													
Tonkin.....	C																











C	Hong Kong.....	319	307	189	92	21	16	11	5	8	10	5	6	2	2	2	2
D	Manchuria—	152	266	246	137	39	36	15	7	6	8	4	7	2	2	2	2
C	Changshun.....		3	2		1											
C	Fushun.....		6														
C	Harbin.....		6			1	2	1									
C	Kwantung—																
D	Dairen.....		2	9	1	6	2	2	1	8	4	4	6	2	2	2	2
D	Port Arthur.....		2	3	3	3	1	1		1	3	4	2	2	2	2	2
D	Mukden.....		1			1											
D	Shanghai—																
C	Foreigners only.....	23	23	11	4	1	2	2		2	2		2				2
D	Including natives.....	64	60	41	25	7	2	6	2	8	6	2	2				1
C	Swatow.....		33	9	33	22	32	20	19	23	14	13	12				
C	Tientsin.....		6	2	8	2	1	2	1	8	4						
C	Tsingtao.....		3	P	P	P	P	P	P	P	P	P	P				
C	Yunnanfu.....			7	P	P											
C	Colombia; Cartagena.....																
C	Curaçao (altrinthy).....																
C	Dominican Republic.....																
C	Dutch East Indies.....																
C	Balkpapan.....																
C	Belawan Dell.....																
C	Borneo.....		28	1	5	7		3			1	2	1				1
C	Samarinda.....		6	6	2												
C	Sumatra.....		30	28													
C	Celebes—Makassar.....		4	47	2	3	1										
D	Celebes.....		3	4	3	6	9	10	14	16	11	9	7	5	4		
D	Celebes—Makassar.....				2	5	4	7	9	9	10	8	6	4	2		
C	Sumatra—																
D	Baros.....	96	106	36	16												
D	Medan.....	30	26	5	2												
D	Palembang.....	9	11	9	7	1			1	1							
D	Palembang.....	5	5	6	3												
D	Palembang.....			9	9												
C	Ecuador (see table below).																
C	Egypt.....																
C	Gharbieh.....			1													
C	Port Said.....																
C	Suez.....																
C	France (see table below).																
C	Great Britain:																
C	England and Wales.....	733	890	1,063	1,156	339	374	354	356	277	362	268	242	272	191		
C	Birmingham.....						1					1					
C	Bradford.....						1					2					
C	Bristol.....		1	2	3												
C	Cardiff.....		4		1												
C	Castleford.....		48	85	56	5	14	5	7	5	5	5	4	2			















