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SIX ADDITIONAL CASES OF LABORATORY INFECTION OF TULARÆMIA IN MAN[1]

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Six cases of tularæmia have occurred among the personnel of the Rocky Mountain Spotted Fever and Tularæmia Laboratory of the United States Public Health Service located at Hamilton, Mont. The first two became ill July 4, 1924, the third October 2, 1924, and the fourth, fifth, and sixth on May 18, 19, and 20, 1925, respectively.

Only two members of the laboratory personnel actually engaged in performing or assisting at necropsy of guinea pigs and rabbits infected with tularæmia or engaged in holding these animals or in handling ticks infected with tularæmia have escaped infection.

The 6 cases at Hamilton, Mont., increase to 17 the total number of cases of tularæmia in laboratory workers. Of the earlier cases 2 occurred in the plague laboratory of the United States Public Health Service in San Francisco,[2] [3] 6 in the Hygienic Laboratory of the United States Public Health Service in Washington, D. C.,[4] and 3 in the Lister Institute of Preventive Medicine, London, England.[5]

In all 17 cases the infection gained entrance to the body without leaving the slightest evidence of a local lesion or without causing glandular enlargement of any consequence. The clinical picture was more like that of typhoid fever than of any other infection, thus characterizing this uniform series of 17 cases of tularæmia as being of the typhoid type of disease, as defined by Francis.[3]

CASE I

R. R. S., male, age 36, physician in charge of the Rocky Mountain Spotted Fever and Tularæmia Laboratory, Hamilton, Mont.

Onset.—July 4, 1924, patient became ill with a feeling of general malaise, weakness, and indefinite pains in the abdomen. The next two days were marked by weakness and constipation, and a temper-

[1] From the Rocky Mountain Spotted Fever and Tularæmia Laboratory of the United States Public Health Service, Hamilton, Mont.

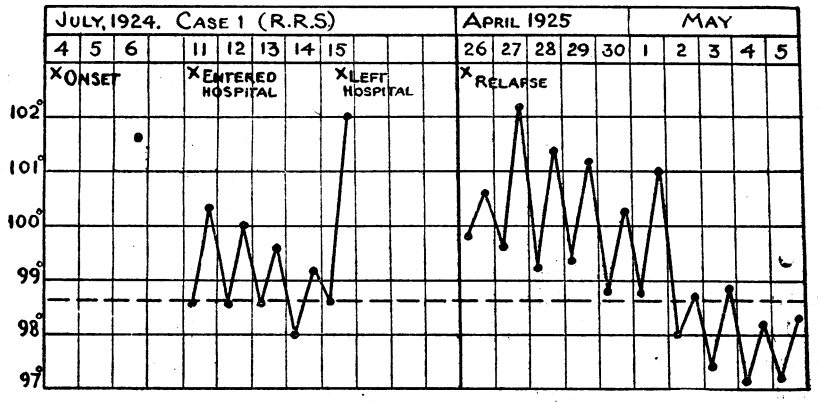
[2] McCoy, G. W., and Chapin, C. W.: *Bacterium tularense*, the cause of a plaguelike disease of rodents. Pub. Health Bull, 53, United States Public Health Service, January, 1912.

[3] Francis, Edward: Tularæmia, J. A. M. A., Apr. 25, 1925, vol. 84, p. 1247.

[4] Lake, G. C., and Francis, Edward: Six cases of tularæmia occurring in laboratory workers. Pub. Health Rep., vol. 37, pp. 392-413 (Feb. 24), 1922. Reprinted in Bull. 130, Hyg. Lab., United States Public Health Service, March, 1922.

[5] Ledingham, J. C. G., and Fraser, F. E.: Tularæmia in man from laboratory infection. Quart. J. Med., July, 1924, pp. 365-382.

ature of 101.5° was recorded on the evening of July 6. Headache and fever persisted during the next five days, causing hospitalization on July 11 and a suspicion of typhoid. This suspicion was fortified by a slight nosebleed, the appearance of the tongue, and the report (from a local laboratory) of a positive Widal on blood taken about July 10. The patient had received antityphoid inoculations in 1913 and 1918. Serum collected July 30 gave a negative Widal at the Hygienic Laboratory. The period July 11 to 15 was spent in bed at the hospital (see temperature chart), but on the afternoon of July 15 the patient left the hospital, with the result that in the evening his temperature rose to 102°. Rest in bed for one week longer was accompanied by a gradual return to normal termperature. During convalescence the patient tired more quickly than usual. Work was resumed July 28 and recovery was apparently complete,



Temperature chart, Case 1

as evidenced by the fact that the patient won a tennis tournament in October.

Agglutination.—The agglutination titers of the blood serum for *Bacterium tularense* at various times following the onset were: 20 days, 640; 28 days, 1,280; 7 months, 640; 9 months, 640; 1 year, 320. A relapse (April 26 to May 1, 1925) apparently caused no rise in the agglutinin titer which on April 11 was 640 and on June 19 was 320. The titer was 640 on November 3, 1925, and 320 on April 8, 1926.

Relapse.—April 26 to May 1, 1925, was spent in bed in the hospital with fever (see temperature chart) but with an absence of symptoms other than constipation and a general feeling of uneasiness in the abdominal region, especially at night. Weakness preceding, during, and after the relapse was a notable feature. Unusual strain was felt preceding the relapse, for a period of 10 days, during which time the patient traveled 3,000 miles by train, retiring late, rising early, not sleeping well, meeting people, making addresses, and driving by

automobile. For five months following the relapse, half-time work was performed on account of tiring readily.

On March 11, 1926, patient's appendix was removed and was found to have undergone chronic changes, with great thickening of the wall and almost complete obliteration of the lumen.

CASE 2

S. S. M., male, age 30, laboratory assistant in Rocky Mountain Spotted Fever and Tularæmia Laboratory at Hamilton, Mont.

Onset.—July 4, 1924, was taken ill suddenly with cramps in the abdomen. The following morning there was fever, headache, and sore throat, accompanied by considerable enlargement and inflammation of the tonsils. The patient remained in his room for the first two days but did not at any time confine himself strictly to bed, although he was weak and had a slight elevation of temperature in the afternoons. Throughout the illness he complained of pain in the left eyeball. There was constipation and abdominal pain and tenderness. Abdominal pain was the most uncomfortable symptom. The highest recorded temperature was 102.6°, but complete record was not kept. The temperature reached 101° on July 12, and 100.2° on July 13. The patient went to his home on July 13 and spent the next two weeks in complete rest. Symptoms gradually subsided and he returned to work July 28, performing full-time duty with difficulty. Return to normal health was rapid.

Agglutination.—The agglutination titer of the blood serum for *Bacterium tularense* on the twenty-fifth day following the onset was 1,280, and on the forty-sixth day it was 640.

CASE 3

R. R. P., male, age 36, entomologist, special expert in the Rocky Mountain Spotted Fever and Tularæmia Laboratory at Hamilton, Mont.

Onset.—October 2, 1924, became ill with headache, weakness, lumbar pains, chills, sweating, and a fever which the patient says he thought must have reached 104° F., although the temperature was not taken. The first week was marked by continuous aching pain in the lumbar region, soreness in upper and lower abdomen especially on movement, and shifting pains in the arms, legs, and feet; there was headache of the frontal and occipital regions, tenderness of the eyeballs, and neuralgic pains of the temples and top of the head. October 10 to 25 was the most uncomfortable period of the illness, due to pains which shifted from joint to joint, remaining in a joint about five days. Because immobilization of a joint brought complete relief from pain there were times when the patient scarcely moved. Marked consti-

pation and increasing weakness characterized the febrile period, which continued for 34 days.

Skin eruption.—The period October 10 to 25 was also marked by a skin eruption which appeared first behind the ears and, spreading, took the form of a band about 3 inches wide across the back of the neck. The eruption gradually extended to the face, sides of neck and forehead; on October 18, it was noted on the elbows; and it finally appeared on the backs of the hands, on wrists, and fingers, and, to a slight degree, on the forearms and arms. The eruption consisted of red, raised papules, mostly about one-fourth inch in diameter; a few were larger. The skin peeled on the areas of eruption and scaling reached its height about November 6.

Agglutination.—The agglutination titers of the blood serum for *Bacterium tularense* at various times following the onset of illness were: 6 days, 0; 14 days, 640; 21 days, 1,280; 7 months, 320; 9 months, 160; 15 months, 160. As in Case 1 a relapse (June 12 to 20, 1925) did not result in a rise of the agglutination titer, which on May 16 was 320 and on July 1 was 160.

Half time was spent at work November 22 to December 15; and after the latter date full-time work was done with difficulty owing to weakness and nervousness.

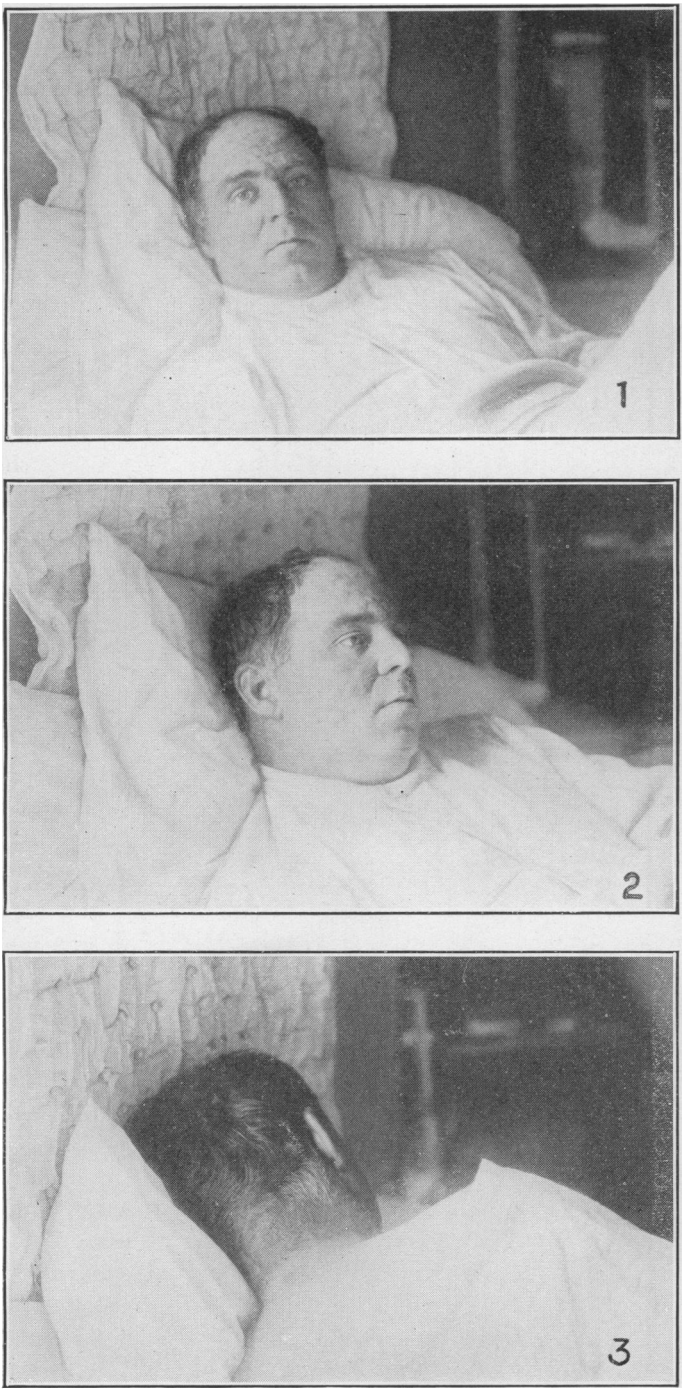
Relapse.—May 20 to 23, 1925, the patient experienced chilly sensations, general weakness, and slight headache, but no fever. Though there was increasing weakness he continued work until June 12, on which date (due probably to overexertion incident to a seven-day inspection and collecting trip in eastern Montana) a febrile attack began which lasted eight days and was accompanied by chilly sensations, headache, weakness, muscular pains, tenderness of the eyeballs, soreness of throat, and constipation; and for two days there was a scattered eruption of general distribution and exactly like that which accompanied the initial illness.

Temperature.—(See temperature chart.)

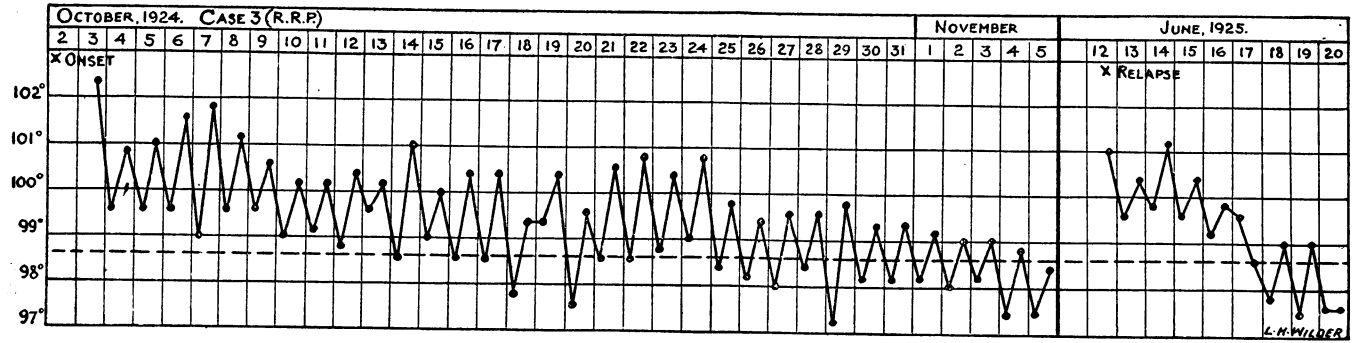
CASE 4.

E. W. M., male, age 33, laboratory assistant in the Rocky Mountain Spotted Fever and Tularæmia Laboratory at Hamilton, Mont.

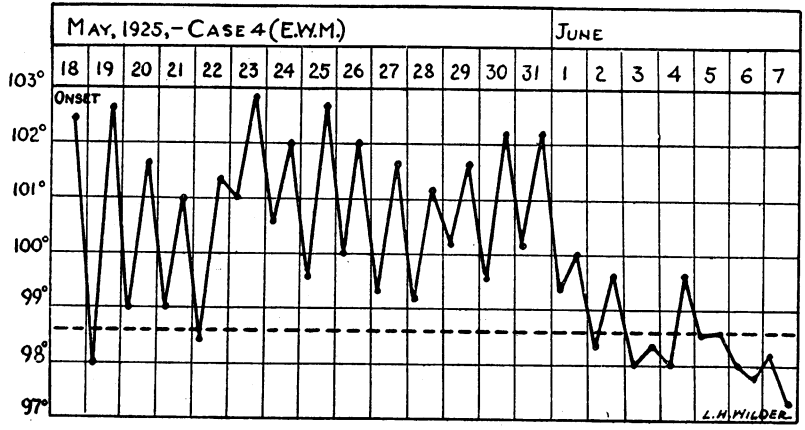
Onset.—May 18, 1925, at 9 a. m., became suddenly ill in the laboratory, with chill, headache, backache, and dizziness. Temperature was 100.4° F. at 11 a. m., when he was sent home. At 2 p. m. his condition was markedly aggravated. He had fogged vision and deafness and did not respond when addressed unless roused by shaking. He was immediately taken to the local hospital and had to be assisted. In the right anterior cervical region there was much tenderness and slight swelling. There was a slight involvement of the



Skin eruption in tularaemia, Case 3. Photographs taken October 23, 1924



Temperature chart, Case 3



Temperature chart, Case 4

right tonsil. In the evening severe backache and shooting pains in the muscles of the legs made the patient very uncomfortable.

May 19, in the morning, owing to a return to normal temperature and an almost complete remission of symptoms, the patient left the hospital and returned home. During the day there was headache, chilliness, cough, and sore throat, followed at night by a severe chill, fever, and sweating. Patient was up and about most of the day.

Owing to an almost normal temperature May 20 the patient was up and drove his car to the laboratory but did not perform any work. He complained of sore throat, coughed, and was chilly.

May 21: Throat very sore; had a chill; was up and about the house.

May 22: Throat and neck very sore; chilly; perspired freely at night.

From May 23 until temperature returned to normal the patient remained in bed.

Sore throat and soreness of the nasal passages were prominent symptoms throughout the illness. For several days the patient was unable to breathe through the nose; the passages were so tender that it could not be blown. The teeth were sore. Constipation was marked during the entire illness. Nosebleed was noted five times. Deafness was noted while confined to bed. Blurred vision was present from onset until several days after temperature became normal. Abdominal pain was not marked and was confined to left side.

An eruption, like that of Case 3, appeared May 28 on the back of the neck and forehead and extended to the front of the neck, hands, and right knee. A few spots appeared on the arms and forearms. Small pustules appeared on the back and arms.

The patient returned to work July 5 and performed full-time duty, although with difficulty. Afternoon and evening rises in temperature occurred at intervals for several weeks. On June 30 a note was made that the spleen was palpable. Work has been continuous to the time of this report, but effects of the illness were still apparent in November, 1925.

Agglutination.—The agglutination titers of the blood serum for *Bacterium tularense* on various days following the onset of illness were as follows: 5 days, 0; 11 days, 160; 18 days, 320; 25 days, 1,280; 71 days, 320; 87 days, 320.

Temperature.—(See temperature chart.)

CASE 5

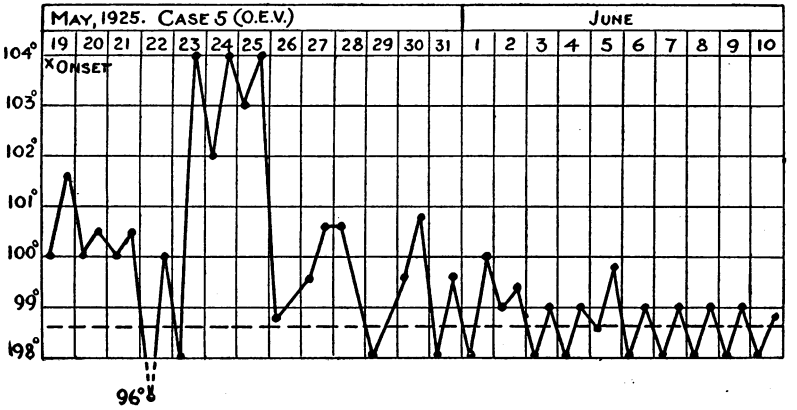
O. E. V., male, age 34, laboratory attendant in the Rocky Mountain Spotted Fever and Tularæmia Laboratory at Hamilton, Mont.

Onset.—May 19, 1925, the patient was feeling ill when he came to work and was noticeably sick at 11 a. m., but he continued to work until evening, when he took to bed. The onset was accompanied by

headache, pain in the eyes, pain in the back, abdominal pains, shifting pains in the muscles, chilly sensation, and fever. The patient remained in bed during his illness except that on the fourth day (May 22) there was an amelioration of symptoms and a remission of temperature to 96° in the morning, going to 100° in the afternoon, and the patient was up and about the house in the afternoon. May 23 the temperature in the morning was 98° but reached 104° in the evening and reached 104° on the two following days.

The throat was sore during the first few days, but there was no cough. During the febrile period the chief complaint was of pain in the left side of the chest and back. At times it was painful to take a full breath. Sweating occurred during the night and after sleeping. Coldness of the feet was continual.

The patient did not return to work in the laboratory; he began work elsewhere on July 1, but it was not until September that he felt normal.



Temperature chart, Case 5

Agglutination.—The agglutination titers of the blood serum for *Bacterium tularense* on various days following the onset were: 4 days, 0; 10 days, 10; 22 days, 160; 31 days, 160; 63 days, 160.

Temperature.—(See temperature chart.)

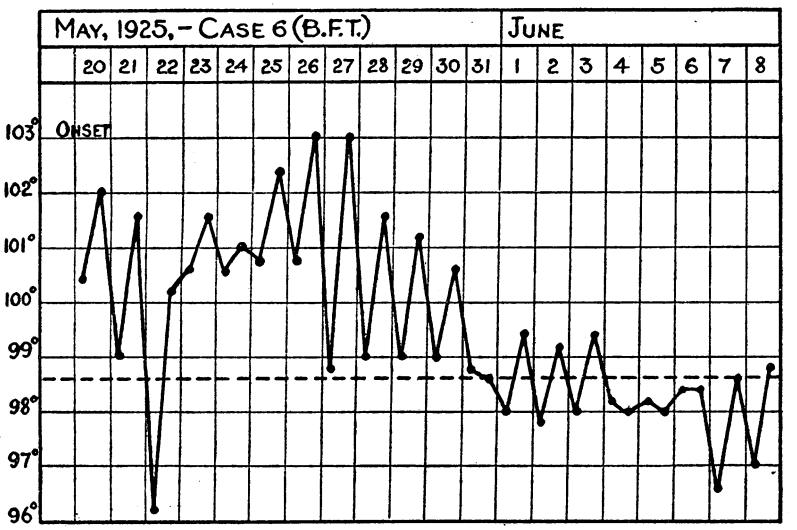
CASE 6

B. F. T., male, age 30, laboratory attendant in the Rocky Mountain Spotted Fever and Tularæmia Laboratory at Hamilton, Mont.

Onset.—May 20, 1925, although feeling weak and "stretchy," since the previous evening, went to work with a temperature of 100.4°, but went home at noon feeling very weak and complaining of stiff joints, sore muscles, stiff neck, and pain on taking a deep breath; had a chill, fever, and profuse sweating, his temperature reaching 102° in the evening.

His illness was marked by weakness, aching of the eyeballs, blurred vision, pains in the teeth, backache, soreness of the abdomen, "stretchy" feelings, chilliness, nosebleed twice on the sixth day, and coughing which began on the seventh day and continued throughout the eighth day. There was no sore throat such as characterized the illness of E. W. M.

The fall in temperature noted on the chart (May 22) was accompanied by a relaxation of symptoms to such an extent that the patient went to work on that date, sure that his illness was ended. He found, however, that work tired him so much that he returned home, went to bed, and remained until recovery. He resumed full time work July 1, and by August 15 was feeling normal.



Temperature chart, Case 6

Agglutination.—The agglutination titers of the blood serum for *Bacterium tularense* on various days following the onset were: 3 days, 0; 9 days, 80; 16 days, 1,280; 23 days, 320; and 42 days, 320.

Temperature.—(See temperature chart.)

SUMMARY OF SYMPTOMS

(1) *Employment.*—Our six cases comprised all but two of the total personnel of the laboratory actually engaged in performing or assisting at necropsies of guinea pigs and rabbits infected with tularæmia or engaged in holding infected animals or employed in handling ticks infected with tularæmia.

(2) *Onset.*—A sudden onset of rather high fever characterized the beginning of illness which, in most of the cases, occurred while the patients were at work.

(3) All six cases manifested the typhoid type of symptoms.

(4) *Remission of temperature.*—Cases 4, 5, and 6 illustrate the rule that, following the initial high temperature of one, two, or three days' duration there is a remission of temperature lasting for one, two, or three days, followed by a secondary rise to high temperature. This remission is accompanied by an amelioration of symptoms which reflects itself in the conduct of the patient and in the physician's opinion that the case is merely one of some ephemeral fever. Of our patients, for example, Case 4 left the hospital and was up and about for several days; Case 5 was up and dressed; and Case 6 attempted to return to work.

Cases 1 and 2 are without temperature records for the first few days, which probably finds its explanation in the same remittance of fever and temporary abeyance of symptoms. Case 3 showed a remission of temperature on the third day.

(5) *Absence of local lesions or glandular enlargement.*—There was no local ulcer or sore or evidence of tick bite, nor was there evident enlargement of any lymph glands.

(6) *Duration of fever.*—The febrile periods ended between the fourteenth and twenty-fourth days, except that in Case 3 it was 34 days.

(7) *Diagnosis.*—Agglutination of *Bacterium tularense* by the patient's blood serum was negative in tests made during the first week as follows: On the third day in Case 6; on the fourth day in Case 5; on the fifth day in Case 4; and on the sixth day in Case 3. All cases gave positive agglutination in the second week and in all subsequent tests.

(8) *Eruption.*—Case 3 had a very definite skin eruption which appeared on the eighth day and continued until the twenty-third day. The eruption consisted of red, raised papules and was followed by peeling and scaling. A similar but less extensive eruption occurred in Case 4.

(9) *Tonsillitis.*—Case 2 was at first tentatively diagnosed as tonsillitis. Case 4 had a very sore throat throughout his illness, with slight involvement of the right tonsil; the nasal passages were also involved.

(10) *Convalescence.*—Convalescence was marked by weakness and a tendency to tire readily on exertion. Return to normal weight was slow.

(11) *Relapses.*—Cases 1 and 3, after 10 months and 8 months, respectively, had relapses of fever lasting six and eight days, respectively. These relapses in each instance followed unusual excessive exertion on the part of the patients while on trips away from home.

CONTACT OF OUR CASES WITH TULARÆMIA

Continuous experimental studies on tularæmia have been in progress at the Hamilton laboratory since the early spring of 1923, following the finding of natural infection in ticks.

Cases 1 and 3 were on the station staff at the inception of these studies; onset of tularæmia occurred, respectively, on July 4 and October 2 of the following year, 1924. Both performed numerous autopsies in the routine way on guinea pigs and rabbits dead of tularæmia and transferred the infection by inoculation or vaccination to healthy animals. In addition, Case 3 handled many infected ticks.

Case 2 was employed beginning February 27, 1924. The onset of his illness was July 4, 1924. He performed autopsies and assisted in the transfer of infection from infected to healthy animals.

Case 4 was employed beginning May 24, 1924; onset of infection was May 18, 1925. He was continuously engaged in tick rearing operations and handled, during rearing, all ticks experimentally infected with tularæmia as well as the infected animals on which they fed. The use of rubber gloves for this work was impractical and contamination of the hands with animal urine and tick feces was unavoidable.

Cases 5 and 6 began work, respectively, on April 20 and March 15, 1925, and the onset of their illness was, respectively, May 19 and May 20, 1925. Except that Case 6 occasionally held guinea pigs and rabbits for vaccination, these attendants were sedulously kept from contact with tularæmia work.

All cases were exposed at various times to such danger of infection as may have attended the handling of numerous wild ticks that were brought in or sent to the laboratory for various purposes. Tularæmia infection has frequently been found in such ticks.

POSSIBLE SOURCES OF INFECTION

The following possible means of laboratory infection were recognized and guarded against: (1) Contamination of the skin; (2) the getting of infectious material into the eyes; (3) contamination of the mouth or the ingestion of infected material; and (4) tick bite. Although not recognized as a definite source of danger, bites of infected animals were carefully avoided.

Materials known to be sources of contamination were: Tissues of infected animals; nasal secretions and urine of rabbits, tissue of infected ticks (especially the body fluid); tick excreta.

PRECAUTIONS TAKEN

Throughout the tularæmia work every possible precaution has been taken to guard against human infection.

For the most part, rubber gloves were worn when making autopsies or transfers of infection; their use by assistants was invariable. Assistants holding animals wore heavy rubber or leather gloves. During the infesting of rabbits and guinea pigs with ticks, and their

subsequent removal, the use of gloves by the person handling the ticks was not feasible. This work was always performed by one of the writers when known infected ticks were used. On all occasions when tularæmia-infected animal tissue or ticks were handled, thorough and immediate cleansing and disinfecting of the hands was rigidly insisted upon, whether or not gloves had been used. All abrasions were treated with iodine. All ticks, whether of known infected history or not, were handled solely with forceps. Instruments were kept thoroughly disinfected and the boards used for autopsies were constantly kept in a barrel of cresol solution. Tables used for autopsies and for the infesting of animals with ticks and for the removal of ticks were cleansed and washed with disinfectants immediately following use. During the removal of ticks the table was frequently washed several times on account of the unavoidable scattering of tick excreta.

The writers were acutely conscious of the danger of infection and repeatedly impressed this danger on their assistants.

These laboratory infections, then, occurred in spite of unusual care and in face of the fact that each person was fully warned and acutely conscious of the risk.

Except Case 4, who reared ticks, assistants rarely performed any operation with tularæmia-infected animals or ticks except when assisting one of the writers.

MODE OF INFECTION

The portal of entry of *Bacterium tularense* in these cases is unknown, there being no evidence in any of them to indicate the primary seat of infection. There was no local ulcer or sore, nor was there evident enlargement of the lymph glands.

Cases 1, 2, and 3 became ill while performing routine procedure—autopsies and transfers of infection from infected to healthy animals. Tick excreta and tick tissue can be excluded as causal factors in Cases 1, 2, and 3, since none of them had handled ticks for some time prior to the infection. Tick bites are also excluded. In all three the source of infectious material was undoubtedly an infected animal; but there is no evidence of the means by which infection was transferred, nor of the point of entry.

The circumstances which attended the infection of Cases 4, 5, and 6, however, were quite different from those of the 14 cases of laboratory infection which had preceded them. None of them performed necropsy on infected animals. As previously stated, Cases 5 and 6 had been carefully kept from any direct contact with tularæmia work except that Case 6 occasionally held animals while they were vaccinated. While the duties of Case 4 frequently necessitated the manipulation of known infected ticks (experimentally infected in the

laboratory), he had handled none later than the middle of March, two months previous to the date of infection.

It is believed that the direct or indirect source of infection for Cases 4, 5, and 6 was a lot of several hundred ticks collected at our request from Owl Canyon, in the Bridger Mountains near Bozeman, Mont., by Prof. R. A. Cooley. These ticks, though subsequently found to be heavily infected with tularæmia, were supposed to be free from all infections, since no tick-borne disease had ever been reported within a radius of many miles of the point of collection. Supposed freedom from infection and a great abundance of ticks have made this canyon for many years a favorite place for the collecting of ticks for experimental purposes. For the very reason that no infection was suspected, these ticks were turned over to the men concerned (Cases 4, 5, and 6) for customary procedure for the engorging of females. This procedure is briefly described:

1. *Infestation of hosts.*—The ticks were received in pill boxes, which were placed on an island in a pan of water and the ticks were transferred by forceps, without handling, to small stoppered vials. From each vial they were transferred in a tangled mass to a shaved area on the belly of a guinea pig or rabbit and immediately covered by a brass gauze capsule which was secured to the animal by adhesive tape.

On May 5, one Belgian rabbit was infested, on May 6 two more were infested, and on May 8, seven guinea pigs. A total of 160 ticks were used, 12 to 23 being placed on an animal.

2. *Removal of ticks.*—The ticks were not removed until engorgement of the females was complete or unless a host animal died before this had been accomplished. In the latter case the ticks were transferred to a new host. Ten days is the usual period required for full engorgement. The removal was performed, while sitting, by two persons working across a narrow table. One held the animal on its back, grasping the front legs in one hand and the hind legs in the other, the animal being raised from and lowered to the table as necessary for the removal of the adhesive bindings. The second person first removed the tape and then carefully detached the ticks with forceps. The males were placed in a vial to be destroyed and the females in separate pill boxes for egg deposition. The ticks were not touched with the hands. The person holding the animal wore heavy rubber gloves. The person removing the bandages and ticks used the hands uncovered.

Partly fed ticks were transferred from dead guinea pigs to normal animals on May 10, 11, 13, 15, and 16. Engorged females were recovered from one Belgian rabbit on May 15 and another on May 16.

The original infestation of the host animals was performed by Cases 4 and 6. Following this and until May 16, all removals of ticks and transfers to new hosts were made by one of the writers, with the assistance of one of those two men. On May 16, however, the engorged females were removed from the second Belgian rabbit by Cases 4 and 5, the first and only time that Case 5 is known to

have come in contact with these ticks and animals, or with any source of infection. The rabbit was held by Case 5 and the adhesive strappings and ticks were removed by Case 4. The entire procedure was performed under the observation of one of the writers.

During the removal of the tape the rabbit began a loud and prolonged squealing which continued until the removal was completed and lasted fully one minute. Case 6, attracted by the squealing, came and leaned over the table and the rabbit to observe the cause of the disturbance. While squealing, the rabbit also passed urine, some of which was spattered on the face of Case 5, who was unable to release his hold of the rabbit. His face, however, was immediately washed with cotton soaked in cresol solution and the urine was similarly removed from the table. As soon as this rabbit could be released Case 5 thoroughly cleansed his face with soap and water.

During the period of the above-described operation, tularæmia infection in these ticks and animals was not suspected. All seven guinea pigs infested on May 8 died between the 10th and 16th, and the ticks were transferred as indicated above. The lesions in the dead guinea pigs were not diagnostic; however, they were sufficiently unusual to justify transfers from three of them being made by spleen emulsion to normal animals. Of the latter, one died of typical tularæmia on May 18, and further transfers from the other two caused death typically on May 30. All three Belgian rabbits also died with typical lesions of spleen and liver and also complete involvement of the lungs. The lungs of one of the guinea pigs were also affected.

It is likely that the putting of these ticks on their hosts was an operation devoid of risk, because the ticks were hibernated, unfed adults (hibernated ticks do not pass excreta until fed), and the animals were normal. Obvious danger, however, attended the transfer of the partially fed ticks from the dead guinea pigs and the removal of the engorged females.

So far as is known this is a complete record of the contact of Cases 4, 5, and 6 with tularæmia-infected ticks and animals for a period of nearly two weeks preceding onset. These cases, therefore, appear to center around the procedure attending the feeding of the Bozeman ticks. Case 4 was most often exposed, and Case 5 on May 16 only. As previously stated, except when the ticks were first placed on their hosts and on May 16, when the females were removed from one of the Belgian rabbits, one of the writers performed the more dangerous procedure of transferring and removing the ticks, Case 4 or Case 6 holding the animals.

The obvious sources of danger were the nasal secretions and urine of rabbits, the excreta of the ticks, and the possibility of contamination with the body fluid of an injured tick. While admitting these

possibilities they do not seem adequate to explain these cases. Case 5 was the only one to come in contact with rabbit urine. Contamination with nasal secretions seems improbable, except perhaps as noted below.

No tick is known to have been injured. Tick excreta was undoubtedly a highly potent source of danger, as this was present in abundance on each animal and always became more or less scattered when removing the tape and brass gauze capsule.

Another source of danger is to be mentioned in explanation of the possible method of infection in this special group of cases—i. e., inhalation or droplet infection. This explanation assumes that these men inhaled the infection from the air around the Belgian rabbit from which the engorged females were removed on May 16, the air having become laden with infection expelled by this rabbit during prolonged squealing as noted above. In support of this view the following points are noted: (1) The fact that all three cases became ill within a 48-hour period, May 18 to 20, is suggestive of an unsuspected means of transfer of infection; for it scarcely seems probable that the routine safeguards against recognized dangers could have failed so utterly for three men on three different occasions within such a short space of time. (2) Cases 4 and 5 were seated at a narrow table with the rabbit between them and were consequently in such close proximity to the rabbit that any danger of infection in the manner suggested was aggravated. (3) Case 6, attracted by the unusually long and loud squealing came and leaned directly over the rabbit. (4) This is the only known instance in which these three cases were simultaneously exposed to the danger of infection, and their onsets were sufficiently close to admit the likelihood of a common source of infection and time of exposure. (5) The periods of incubation are not opposed to the occurrence of infection on May 16, the date the rabbit was handled. (6) Case 4, owing to his removing the adhesive strappings, was closest to the rabbit during the squealing; he was the first to become sick and was also the most ill. Case 6 was in less close proximity than either Case 4 or Case 5, and for a shorter time; he was the last to become sick and was also the least ill. (7) The rabbit died May 24, which was seven days following tick removal on May 16; necropsy revealed a general and marked involvement of the lungs. (8) During the prolonged and forceful squealing, infectious material from lungs or nose may have been expelled into the adjacent air.

The suggestion that Cases 4, 5, and 6 may have contracted tularæmia by inhalation necessarily assumes that a rabbit which had sufficient pulmonary involvement when handled May 16 to give off droplet infection would live until May 23.

Acknowledgment.—We are indebted to Drs. H. D. Browning, Herbert Haywood, and G. A. Gordon of Hamilton, Mont., for clinical notes made while in attendance on these cases. The agglutination tests were made by Surg. Edward Francis at the Hygienic Laboratory, United States Public Health Service, Washington, D. C.

A CASE OF TULARÆMIA IN A LABORATORY WORKER

By Louis V. Dieter, Bacteriologist, United States Public Health Service

A case of tularæmia occurring in one of the personnel of the plague laboratory of the United States Public Health Service at Los Angeles, Calif., had its onset about January 4, 1926.

This is the eighteenth case reported of laboratory infection of tularaemia in man, the previous cases having been reported from laboratories located at San Francisco, Calif. (1) (2), Washington, D. C., (3) London, England, (4) and Hamilton, Mont. (5).

The patient had been working continuously since March 31, 1925, with strains of *Bacterium tularense* isolated by him from wild rats (6) received in the laboratory for routine examination for plague; he had performed numerous necropsies of rats and guinea pigs infected with these strains.

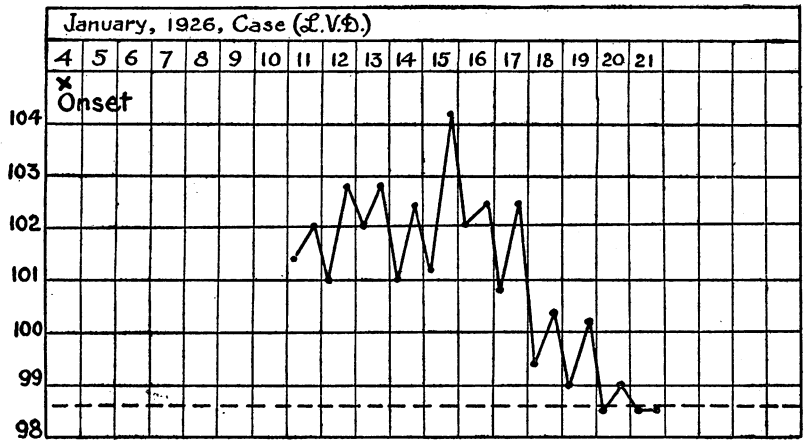
The patient (L. V. D.), male, age 40 years, bacteriologist in charge of the laboratory, became ill about January 4, 1926. Uncertainty exists as to the exact date of onset because the onset was not abrupt, but was manifested by a general feeling of malaise. About January 4 the patient first noticed a small papule on the back of the index finger of the left hand, near the middle of the proximal phalanx. This gradually enlarged during the next three or four days until it had the appearance of a small boil; when opened, pus escaped which showed the presence of *Staphylococcus aureus*. As tularæmia was not suspected, the pus was not injected into guinea pigs, so that it is uncertain whether or not the papule was the primary seat of the tularæmia infection.

The period January 4 to 9 was characterized by rise of temperature in the afternoons and evenings, chilly sensations, but no true chills, and a feeling of prostration and inability to obtain sufficient sleep. About January 9 the patient was forced to abandon work in the afternoons, and beginning January 12 he was forced to remain at home. From January 12 to 18 the symptoms suggested influenza and consisted of almost continuous headache, drowsiness, fever, chilliness during the day, extreme constipation throughout, and complete loss of appetite. There were occasional sharp pains in the right side of the chest and a continuous feeling of fullness and dull pain in the upper portion of the abdomen.

No glandular enlargements were noticeable at any time. Complete physical examination made by Surg. H. E. Trimble, United States Public Health Service, failed to disclose glandular enlargements or other physical abnormalities.

The symptoms gradually subsided until, by May 1, 1926, no ill effects were noticeable except an occasional feeling of weakness lasting for a few days.

Agglutination.—About 30 days after the onset, the patient tested his blood serum for agglutination of strains of *Bacterium tularense* which had been isolated from rats in his laboratory; agglutination occurred in dilution of 1:320, but not in higher dilution, and control serum reacted negatively. About 50 days after onset he again tested his serum for agglutination of *Bacterium tularense* with the result that agglutination occurred in dilution of 1:1280, but not in higher



Temperature chart of case of tularæmia (L. V. D.) (laboratory infection)

dilution. The latter test was controlled by serums taken from two men belonging to the station personnel who were connected with outside work and who had recovered from attacks of influenza; their serums did not agglutinate in dilutions higher than 1:10.

Five months after onset the patient's serum was tested at the Hygienic Laboratory, United States Public Health Service, Washington, D. C., and was found to agglutinate a strain of *Bacterium tularense* isolated from a rabbit obtained from the Washington, D. C., market; agglutination was prompt and complete in dilutions of 1:10, 20, 40, 80, 160, and 320, but not in higher dilutions.

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BENZOL POISONING AS AN INDUSTRIAL HAZARD

Review of Studies Conducted in Cooperation with the Subcommittee on Benzol of the Committee on Industrial Poisoning of the National Safety Council [1]

By LEONARD GREENBURG, Associate Sanitary Engineer, Office of Industrial Hygiene and Sanitation, United States Public Health Service

I. THE CHEMISTRY AND INDUSTRIAL USES OF BENZOL

Benzene (C_6H_6) commercially called benzol, is a colorless, limpid, highly refractive liquid having a somewhat pleasing characteristic odor. It boils at 80.2° C., yielding a very inflammable vapor, which on burning produces a smoky flame. Its specific gravity is 0.899 at 0° C. It is highly insoluble in water, more soluble in alcohol, and completely miscible with ether, acetic acid, and carbon disulphide. It is an excellent solvent for rubber, gums, fats, and resins of various kinds.

This interesting and highly important substance was discovered by Michael Faraday, in 1825, in the liquid produced by the compression of the illuminating gas obtained from the distillation of certain oils by the Portable Gas Co., of Paris, France. Benzol was, however, probably known to Shellenz some 40 years earlier. In 1833 benzol was prepared by Mitscherlich by the destructive distillation of benzoic acid with lime. He determined its composition, measured its vapor pressure, and named the substance "benzin" or "benzine." [2] Leibig suggested the modification of this term to benzol to indicate its oily origin. Marignac recovered benzol in 1842 from the distillation of phthalic acid with lime, and Berthelot, in 1866, by heating acetylene.

[1] Since 1922 the Committee on Benzol (C.-E. A. Winslow, chairman, L. Greenburg, vice chairman, W. S. Paine, secretary, H. Bradshaw, J. W. S. Brady, A. C. Fieldner, C. F. Horan, L. E. Weber, and J. M. Weiss) has been conducting an extensive study of benzol poisoning as a hazard in American industry. The results of the whole study will be published in a special bulletin for the National Safety Council by the National Bureau of Casualty and Surety Underwriters. The review of the literature and the field studies were made by Dr. J. W. S. Brady, Dr. J. J. Batchelor, Mr. J. R. Dexter, Dr. J. Newton Shirley, and the writer.

[2] The term "benzine," spelled with an "i," is now popularly applied to an entirely different substance (a mixture of C_6H_{14} and C_7H_{16}), a petroleum product.

The presence of benzol in coal tar was first demonstrated by Leigh in 1842 and was confirmed by Hoffman in 1845. At that time Hoffman suggested the use of the name benzene for this substance.

In 1865 Kekulé announced the structural formula of benzene, and on this as a basis laid a foundation for the theories of aromatic compounds (1).

The earliest research on the commercial production of benzol from coal tar was that of Mansfield in 1849. In 1869 Caro Clemen and Engelhorn obtained patents on a process of recovering benzol from illuminating gas.

Illuminating gas, however, does not yield much benzol, without lowering its illuminating power, and it was only when similar processes were applied to coke-oven gas that substantial results were obtained.

In 1884 Carves obtained a patent on a process of removing benzol from coke-oven gas by a washing method. The technical use of this method followed from the studies of von Hüssener and later of Brunck, who set up the first plant in operation in Germany for the recovery of benzol in 1887.

Since that time the recovery of benzol from coking-plant gases has steadily extended, and now far exceeds recovery from city gas works.

The commercial uses of benzol rarely require that this substance be chemically pure. In practice, three types of benzol are used in industry, in addition to various other substances to which the name benzol is applied, although without justification from a chemical standpoint. The usual commercial products are the following:

Pure benzol—a clear colorless liquid of a charcteristic odor. B. P., 80.2° C.

Ninety per cent benzol.—So called because in the distillation of coal tar 90 per cent distills over at a temperature less than 100° C. It is composed of 80–85 per cent benzol, 13–15 per cent toluol, 2–3 per cent xylol, and sometimes contains as impurities traces of olefins, paraffins, sulphuretted hydrogen, and other substances.

Fifty per cent benzol.—This substance contains 50 per cent of constituents which distill below 100° C. and 90 per cent of constituents which distill below 120° C. It is a highly mixed product, with only 40 to 50 per cent benzol, the remainder being higher homologues and impurities.

The other substances often called benzols are various toluols, xylols, and solvent naphthas.

Solvent naphtha.—This material is called solvent naphtha because it is used extensively (especially in England) for dissolving rubber. it is relatively free from benzol and consists largely of xylol, its homologues, and other unknown hydrocarbons (2).

In the production of benzol, the raw coal tar from the coke works is distilled in malleable iron distilling retorts using steam as the heating medium. Four fractions are usually recovered in this process: Light oil, 1–3 per cent; medium oil, 9–13 per cent; heavy oil, 7–11 per cent; and anthracene oil, 12–18 per cent. The remainder, comprising 52–60 per cent, is called retort pitch.

The light oil constitutes the basis for the manufacture of benzol. It is a liquid of sp. gr. 91–95; in color it is yellow to dark brown. Over 80 per cent of it consists of aromatic substance as follows: Naphthalene, 16 per cent; benzol and its homologues, 74 per cent; phenol, 4–15 per cent; bases, 1–3 per cent; sulphur bearing substances, about 0.1 per cent; Nitrile, 0.2–0.3 per cent; acetone, cumene, indine, 1.0–1.5 per cent; Olefins, 3.5 per cent; Paraffins, 0.5–1 per cent; other unsaturated compounds, 1–1.5 per cent (3).

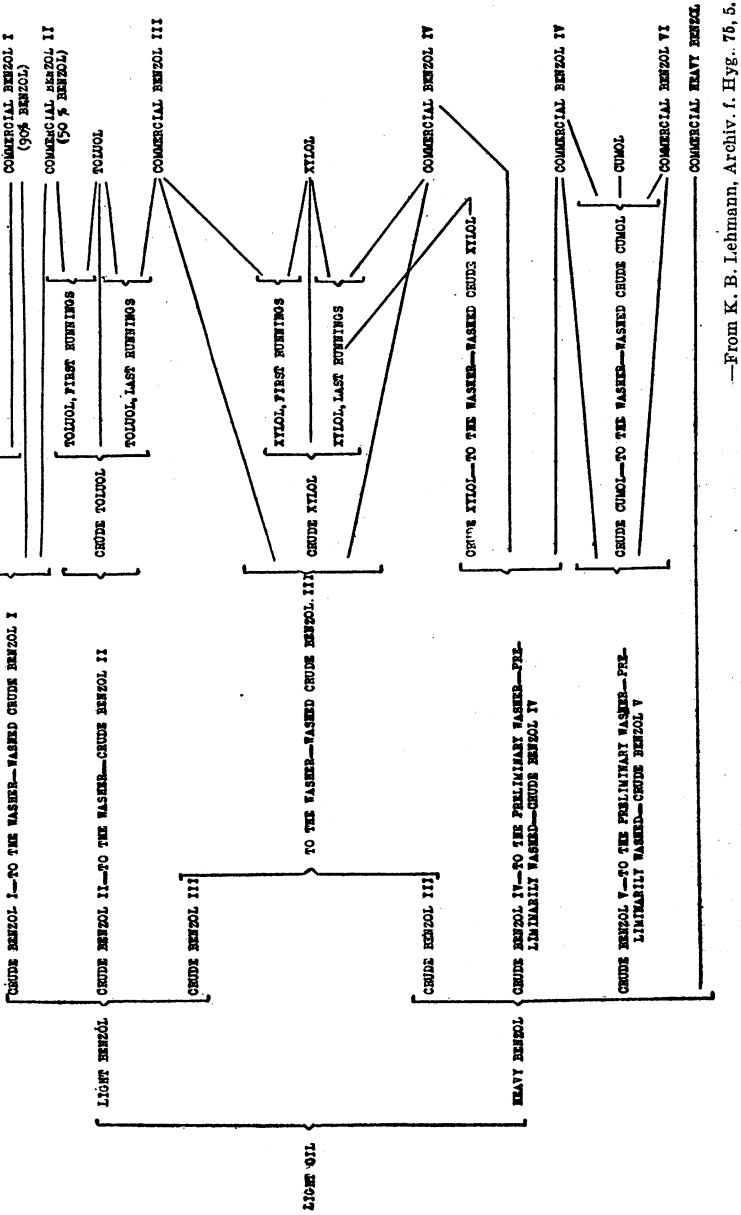
The method used for the recovery of benzol from coking-plant gases may be found in any complete handbook on industrial chemistry. The chapter by Dodge in Rogers' Industrial Chemistry has been freely drawn on in preparing the following brief summary:

Essentially this process consists in removing the benzol from the coke-oven gas by taking it up in a wash oil and distilling the benzol from the wash oil by a continuous process.

The gas is cooled either by direct contact with water or in tubular coolers to about 70° F. It is then passed through a series of scrubbers (usually three). In these scrubbers, which are usually tall steel tanks or towers filled with wooden grids to present a large surface of contact, the wash oil takes up the benzol vapors. After leaving the first gas scrubber the oil is passed through a heat exchanger, where it is heated by the vapors leaving the still to about 90° C., then through a second heat exchanger, where it is further heated by the debenzolized wash oil leaving the still to about 110° C.

It is then passed through the preheater and further heated by steam to 130° to 150° C. After this it passes to the still proper, which is of the continuous type, where the light oil is driven out by steam distillation, leaving the debenzolized oil, which leaves the base of the still, passes through the oil to oil-heat exchanger, the wash oil coolers, and is then ready to be pumped to the scrubbers for a second saturation with light oil from the gas.

The light oil vapor mixed with steam after passing through the vapor to oil-heat exchanger is condensed and flows to the light oil tank after being separated from the condensed steam or water in the decanter. The yield of light oil from coke-oven gas is from 2¼ to 4 gallons per ton of dry coal carbonized, varying with the volatile content of the coal and much influenced by the type of oven and the heats employed. The relation of the amounts of benzol and toluol



—From K. B. Lehmann, Archiv. f. Hyg., 75, 5.

produced are also influenced by the heats employed, low heats increasing the production of toluol and xylol.[3]

Prior to the World War the industrial uses of benzol grew steadily, but not sensationally. In Germany, according to Ullman (2), 100 kilograms of benzol cost 175–400 marks in 1882. By 1890 this price had been cut in half, and 4,000–5,000 tons a year were produced. By 1896 the price had dropped to 25–50 marks, and the production had risen to 7,000 tons. By 1901 the price was 20 marks and the production 28,000 tons, rising to 40,000 tons in 1904, and to 90,000 tons in 1908.

During the war the production of benzol was greatly stimulated by the demand for toluol along with which large quantities of benzol are commercially produced, a condition which has naturally led to the more widespread use of benzol as a starting point for the manufacture of various organic compounds, as, for example, in the anilin color industry and in the production of pharmaceuticals and chemicals for the photographic industries. In addition, benzol is used as a motor fuel by blending it with gasoline, and, above all, as an organic solvent in the rubber industry, in artificial leather manufacture and lacquer production, and in similar processes.

In considering the practical problem of the use of benzol in industry it is important to remember that two very distinct types of processes are involved. In such industries as (*a*) the distillation of coal and

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[3] Following are the specifications for coke oven light distillates as prepared by the Barrett Co., one of the large producers of coal-tar distillates in the United States (4):

SPECIFICATIONS FOR COMMERCIALLY PURE BENZOL, NOT NECESSARILY NONCORROSIVE

(To be commonly known as "Commercially pure benzol")

Color.—The visible color shall be not darker than a solution of 0.0030 gm. of potassium bichromate in 1 liter of water. Comparison to be made in Nessler tubes, 50 c. c. size.

Distillation.—The product shall distill from start to dry within 2° C., within which degrees shall be included the true boiling point of pure benzol (80.2° C.).

The method of distillation shall be in accordance with method E–4, Jour. Ind. Eng. Chem. 10, 1006 (1918).

Acid wash.—The wash shall be not darker than No. 4 Barrett colorimetric scale. The test for wash shall be made in accordance with directions for test E–6, Jour. Ind. Eng. Chem. 10, 1008 (1918).

Acidity.—Finished product shall contain no "free" acid.

Specific gravity.—Specific gravity at 15.5° C. shall lie between 0.875 to 0.886. Specific gravity shall be determined by the use of Westphal balance.

SPECIFICATIONS FOR COMMERCIAL 90 PER CENT BENZOL

(To be commonly known as "Commercial 90 per cent benzol")

Color.—The visible color shall be not darker than a solution of 0.0030 gm. of potassium bichromate in 1 liter of water. Comparison to be made in Nessler tubes, 50 c. c. size.

Distillation.—The product shall start distillation (initial or first drop) at not below 76.2° C.; 90 to 95 per cent shall distill at 100° C; all shall distill (end point) not above 120° C.

The method of distillation shall be in accordance with method E–4, Jour. Ind. Eng. Chem., 10, 1006 (1918).

Acid wash.—The wash shall be not darker than No. 6 Barrett colorimetric scale. The test for wash shall be made in accordance with directions for test E–6, Jour. Ind. Eng. Chem. 10, 1006 (1918).

Acidity.—Finished product shall contain no "free" acid.

Specific gravity.—Specific gravity at 15.5° C. shall lie between 0.875 and 0.887. Specific gravity shall be determined by the use of Westphal balance.

coal tar in the production of benzol, (*b*) the blending of motor fuels, and (*c*) the chemical industries, including oil extraction, dye and dye intermediates, manufacture of paints, varnishes, and stains, and of paints and varnish removers, benzol is used in large quantities; but the very nature of the industry demands that it be kept in a closed pipe-line system, any openings representing a loss of valuable vapors and making the system an inefficient one with a correspondingly large financial loss. In the class of plants mentioned, chronic benzol poisoning may be expected to play but a minor rôle, the hazard in acute cases being due to carelessness in the cleaning of tanks, breaks in the piping system, or similar accidents.

With regard to this first class of processes it seems certain that, with proper care in construction, maintenance, and operation, the use of benzol can be made sufficiently safe to warrant its employment. It

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(Footnote 3 Continued)

SPECIFICATIONS FOR COMMERCIALLY PURE TOLUOL

(To be commonly known as "Commercially pure toluol")

Color.—The visible color shall be not darker than a solution of 0.0030 gm. of potassium bichromate in 1 liter of water. Comparison to be made in Nessler tubes, 50 c. c. size.

Distillation.—The product shall distill from start to dry within 2° C., which degrees shall include the true boiling point of toluol (110.4° C.).

The method of distillation shall be in accordance with method E-4, Jour. Ind. Eng. Chem., **10**, 1006 (1918).

Acid wash.—The wash shall be not darker than No. 4 Barrett colorimetric scale. The test for wash shall be made in accordance with directions for test E-6, Jour. Ind. Eng. Chem., **10**, 1008 (1918).

Acidity.—Finished product shall contain no "free" acid.

Specific gravity.—Specific gravity at 15.5° C. shall lie between 0.864 and 0.874. Specific gravity shall be determined by the use of Westphal balance.

SPECIFICATIONS FOR COMMERCIAL XYLOL

(To be commonly known as "Commercial xylol." Formerly known as noncorrosive solvent naphtha (benzol 160°))

Color.—The visible color shall be not darker than a solution of 0.0030 gm. of potassium bichromate in 1 liter of water. Comparison to be made in Nessler tubes, 50 c. c. size.

Distillation.—The product shall distill not over 5 per cent by volume at 130° C.; 90 to 95 per cent shall distill at 160° C.; all shall distill (end point) at not above 180° C.

The method of distillation shall be in accordance with method E-4, Jour. Ind. Eng. Chem., **10**, 1006 (1918).

Acid wash.—The wash shall be not darker than No. 12 Barrett colorimetric scale. The test for wash shall be made in accordance with directions for test E-6, Jour. Ind. Eng. Chem., **10**, 1006 (1918).

Acidity.—Finished product shall contain no "free" acid.

Sulphur content.—The product shall be free from H_2S and SO_2 as qualitatively determined in test E-9, Jour. Ind. Eng. Chem., **10**, 1010 (1918).

Copper corrosion.—Commercial xylol shall meet Standard Method Copper Corrosion test dated June 21, 1921.

SPECIFICATIONS FOR SOLVENT NAPHTHA (NOT NECESSARILY NONCORROSIVE)

(To be commonly known as "Solvent naphtha")

Color.—The visible color shall be not darker than a solution of 0.0030 gm. of potassium bichromate in 1 liter of water. Comparison to be made in Nessler tubes, 50 c. c. size.

Distillation.—The product shall distill not over 5 per cent by volume at 130° C.; 90 to 95 per cent shall distill at 160° C.; all shall distill (end point) at not above 180° C.

The method of distillation shall be in accordance with method E-4, Jour. Ind. Eng. Chem., **10**, 1006 (1918).

Acid wash.—The wash shall be not darker than No. 12 Barrett colorimetric scale. The test for wash shall be made in accordance with directions for test E-6, Jour. Ind. Eng. Chem., **10**, 1010 (1918).

Acidity.—Finished product shall contain no "free" acid.

is true that fatal accidents have occurred and will no doubt continue to occur in such processes, just as such accidents occur from the use of steam boilers. The danger is, however, in both cases a controllable one, to be met by careful attention to safety provisions and not by the abandonment of the substance in question. To this phase of the subject we have therefore devoted but little special attention.

In a second group of processes represented by the use of benzol (*a*) in the rubber industry, (*b*) in artificial leather manufacture, (*c*) in sanitary can manufacture, (*d*) in dry cleaning, and (*e*) in connection with the handling of paints, varnishes, and stains, benzol is employed chiefly as a solvent or a vehicle, and as a part of the process it must be removed so as to leave the originally dissolved substances in place. The method of removal of the benzol is usually to permit it to evaporate. In most cases evaporation takes place while the compound is cold; in some, however, the compound may be warmed, a procedure which naturally removes the benzol with greater rapidity. The vapors arising when benzol is thus permitted to evaporate in the workroom atmosphere constitute the principal cause of chronic benzol poisoning. It is chiefly in plants of this type that we have carried on our studies.

In the rubber industry benzol finds two exceedingly important uses. In rubber tire building the metal core on which the tire is built is usually painted with rubber cement. This cement is often made in considerable quantities, for, during the course of the workday, large amounts of this substance are used. The rubber, after being compounded in the general compound room, is brought to the cement house, where it is cut into small pieces (about 4 pounds) and broken down in a pair of heated "break-down" rollers. It emerges in a rather porous condition and warm. It is then placed in a vertical tank containing benzol and provided with agitators and is stirred continuously until it is in solution. It would appear that this process might easily be conducted without hazard, but in making our studies we found one plant in which this was far from being the case.

In the further steps of tire building benzol is used in many plants. The layers of rubberized cord or cloth are carefully placed on the metal tire-building core, each layer being moistened with benzol prior to the application of the next layer of fabric. In addition to these two uses of benzol in the rubber industry there are, of course, many others. In fact, this substance has found very wide use due to its excellent solvent properties for rubber.

In the manufacture of so-called sanitary tin cans solder is not used to fix the top and bottom of the can to the cylindrical body portion, but these are crimped together instead, and it is necessary to provide some means of rendering the can absolutely air-tight. This is most

successfully accomplished by means of a rubber cement. The can ends are punched from sheet metal by means of an automatic punch press and then raised to the feeding end of a coating machine. In this machine the can end is held on a revolving vertical spindle, and as it revolves a very fine stream of benzol rubber cement is forced under air pressure on the can end. After the disk has made one complete revolution and a thin band of the cement is in place, the disk is automatically ejected and slides down a small incline to an oven, in which the benzol is largely but by no means completely evaporated. The can ends emerge from this oven in a warm condition and with benzol evaporating from the ring of cement about the periphery. It is at this point that the inspectors, usually young women, are exposed to the benzol fumes while inspecting, cleaning, and packing the can ends.

In the artificial leather industry, benzol is used in immense quantities. In producing artificial leather, cloth is fed through a machine at one point of which is a knife blade stretching across the full width of the cloth. Here the compound with which the cloth is to be coated is applied. This compound is a viscous liquid consisting of nitrocellulose and castor oil which is dissolved in ethylacetate or other suitable solvent. Benzol, in proportion of approximately 60 per cent, may be added as a diluent and derives much of its value from the fact that it is miscible with the other solvents and also with the nitrocellulose. The required color is given to the compound by the addition of suitable pigments, and the whole batch is thoroughly mixed until a homogeneous fluid results. This batch, when ready for delivery to the coating machine, is evenly spread over the cloth as it passes under the knife edge. The cloth next passes to an inclosed heated chamber in which the volatile substances are driven off, the colored nitrocellulose remaining behind on the cloth. The cloth emerges from the chamber in a warm condition and, although rather dry, still has a certain amount of solvent evaporating from its surface. It is this solvent which is still evaporating from the surface of the cloth and the solvent vapor escaping from the hot chamber which create a poison hazard. There is also a certain amount of evaporation which takes place from the surface of the pool of compound directly at the knife edge where it is being applied to the cloth, and this further increases the benzol content of the workroom air.

In the process of dry cleaning, the clothing and other articles to be cleaned are placed in horizontal tanks containing benzol, in which, by the continuous agitation of the tank, the grime and grease are dissolved. After this treatment the clothing is placed in centrifugal extractors by means of which the excess of benzol is removed. The articles are then taken from the extractor and, as a rule, are placed

in a warm-air cabinet in which the last traces of the benzol are volatilized. During this complete procedure from the time the washing tank is opened in order to permit of the addition of the articles to be cleaned to the time when the clothes are benzol free there exists much opportunity for the dissemination of benzol fumes in the workroom air, with the attendant dangers associated with these vapors.

Lastly, in the use of paints and penetrating stains, wood dyes, benzol finds very widespread use. This substance has deep penetrating power and for this reason is of great value for such purposes. When the paints are spread on the surface to be coated or treated, the vehicle evaporates, leaving the pigments behind. In the case of paint and varnish removers, the compound is permitted to remain on the surface to be cleaned for some time preparatory to the process of scraping the surface. The hazard involved in this procedure must be small as compared with that in the use of benzol containing paints. It has been shown that the volatility of benzol is markedly affected by the other substances present in paint removers. Of 8.7 grams of benzol exposed in an open Petri dish, 98 per cent was evaporated in 100 minutes; whereas of 8.9 grams of a mixture composed of 55 per cent benzol, 45 per cent acetone, and 3 per cent paraffin only 0.4 per cent was evaporated in 300 minutes at the same temperature.[4]

II. ACUTE BENZOL POISONING

As in the case of so many toxic substances used in industry, instances of benzol poisoning fall into two rather distinct groups—acute and chronic poisoning, respectively. As a rule the cases of acute poisoning are obvious and readily diagnosed from a consideration of the history, symptoms, and circumstances surrounding the individual case. Some cases of acute benzol poisoning are due to the specific toxic effects of benzol, while others are undoubtedly due to benzol asphyxiation caused by the greater density of benzol vapor as compared with air, with the consequent exclusion of the air. Cases of asphyxiation are most likely to occur when a worker enters an inclosed space such as a tank which formerly contained benzol. Cases of chronic poisoning, however, are generally more obscure, the history of exposure is not so definite, and the symptom-complex and signs of poisoning as a rule are not so evident.

Acute benzol poisoning has been generally encountered in industrial practice, although case reports of benzol taken with suicidal intent or poisoning occurring in its nonindustrial application are also relatively frequent. The early literature with reference to benzol as an industrial hazard is not unnaturally based on continental experience and later to a lesser extent on that obtained in England.

[4] Personal communication from Weiss and Downs.

It was not until the outbreak of the World War that benzol poisoning to an alarming extent manifested itself in the United States as a result of the wholesale production of benzene and its derivatives as by-products of the benzol manufactured for war purposes. Attention was first drawn to this problem in America by Alice Hamilton in her reports on industrial poisons encountered in the manufacture of explosives in this country (5). Prior to the war, however, Selling and others had reported on the toxic effects of benzol manifested in workers engaged in certain industries.

In 1862 there appeared in the Lancet (6) under the title of "A new industrial poison" a case report of a death due to the inhalation of vapor and the ingestion of a fluid containing "benzole." The patient became sick and drowsy, somewhat cyanotic, and developed a feeble pulse, the symptoms being those of gastro-intestinal irritation. On autospy no trace of the toxic substance was found in the stomach. Congestion of the brain, lungs, and liver was present, and congestive patches were present in the gastric mucosa. The coroner decided that death was due to prussic acid, inasmuch as the "benzole" was found to contain some of this substance. Benzol as such, however, appears to have played a part in this fatality.

Two very early cases of benzol poisoning are recorded in St. George's Hospital Reports for 1877–78 (7). In each of these instances a small amount of benzoline (benzol) was swallowed by mistake. Emetics were used apparently with success, although no mention is made of the outcome. In 1889 Averill (8) reported a case in which 3 to 4 drams of benzol in castor oil was taken by mistake with toxic effects. The patient recovered after receiving medical attention.

In 1910, at the International Congress of Industrial Hygiene, Rambousek (9) presented a report of 22 cases of acute benzol poisoning, 18 of which proved fatal. Most of the acute cases appear to have occurred in or around coal-tar stills and benzol tanks. He cites a case taken from the Factory Inspector's Report of Germany, for the year 1902 (10), of a worker who was overcome while painting the interior of an iron reservoir with a benzol-asphalt paint. In 1903 several other acute cases were recorded. While cleaning an extracting apparatus, a workman was fatally overcome by benzol fumes (11). Another worker who forgot to open the cold-water valve of a condenser was overcome, with fatal results, by the uncondensed vapors which were dispersed in the general room atmosphere (12). Another interesting case is recorded in the German Factory Reports (11) in which a worker engaged in the manufacture of antipyrene inhaled the concentrated benzol vapor with fatal results.

Several cases of poisoning by swallowing benzene have been recorded. Simonin (13) reports such a case of accidental poisoning in which the patient survived, but developed an odd type of skin

condition consisting of swelling and edema. Schmitz (14) records a fatal case of poisoning by injestion, the autopsy findings of which will be discussed later in this report. Hetzer (15) reports an unusual case in which a man who had swallowed benzene was given prompt medical treatment and survived, but developed an intense toxic gastritis and later pyloric stenosis. Nick (16) cites the case of a workman who swallowed 80 grams of commercial benzene and developed the usual acute symptoms. Five cubic centimeters of a 10 per cent lecithin emulsion (Merck) was given by intravenous injection, about two hours after poisoning resulted, and definite improvement was noted within an hour. Thirty-six hours later all outward signs of poisoning had disappeared.

In the Prussian Factory Inspector's Report for the year 1904 (1) two fatal cases are described in workmen engaged in cleaning out a tar still. Death was due to fumes presumably of benzol, although this was not definitely established. In 1905 (17) three cases of poisoning, one of them fatal, occurred in a rubber factory where a benzol rubber composition was being mechanically spread on fabric. The fabric, after being coated, passed over a long heated plate to facilitate drying, a procedure which gave rise to the production of large quantities of benzol vapor in the room air. The same year, 1905 (18), a worker was overcome by benzol fumes while cleaning out an empty tank which had contained crude benzol. A short while previously another similar case had occurred in the same factory. In another plant (19) two other cases of unconsciousness occurred, the workmen being overcome on entering a tank containing heavy oil used for cleaning purposes. In the Report of the Union of Chemical Industry for the year 1905 (20) a case is cited in which a night workman neglected to open a valve which allowed benzol distillate to flow into a collecting tank, and 8,000 liters of benzol overflowed. The workman was found dead in front of the building. Lewin (21) reports an interesting case in which an extracting vessel was being cleaned. The vessel had contained naphthaline and benzine, but had been empty for 22 hours. Furthermore, it had been washed out three times with cold water and twice with steam, then again allowed to stand all night filled with cold water. The workman who entered the tank was equipped with a pipe supplying compressed air. Despite these precautions he was overcome by the fumes of benzol which had dropped into the tank from a leaking overhead pipe. Several men who attempted to effect a rescue were overcome and one of the rescuers died within ten minutes after exposure. The original workman was finally saved by an engineer equipped with a breathing helmet. Dr. Alice Hamilton (22) reports a similar case which occurred in England. A benzene tank car had been emptied, washed with water, steamed, filled with water for 20 hours, then

washed out twice, again boiled for 12 hours, and finally left for 10 days with the manhole open. Despite these precautions the workman who entered the tank collapsed, and although he was removed in time, one of his rescuers succumbed. These cases have led to the belief that muscular exertion tends to increase the susceptibility to poisoning and to decrease correspondingly the prospects for recovery in acute cases. That such a belief is tenable is illustrated by the many cases in which a passive victim who has been rendered unconscious by the benzol vapor recovers despite his relatively prolonged exposure, while an active rescuer succumbs with a fatal termination despite his relatively brief exposure to the same concentration of vapor. Lehmann stresses individual susceptibility as the important factor in such cases. That marked variation in individual susceptibility does exist is amply demonstrated by clinical and experimental observation, findings with which we are in accord. It is possible and perhaps even likely, however, that in some cases the degree of activity of the subject plays a part in determining the outcome of any particular exposure.

Yet another case may be cited of a worker who was rendered unconscious while inspecting a benzene still. Two fellow workmen who attempted to rescue him were also overcome, one of them dying. All three had been negligent in failing to observe the rules requiring that the still be blown out with steam and that a watch mate be on guard outside for any emergency (23).

Two fatal cases are reported (24) from a factory where thick tar was being distilled under pressure and where the air pumps got out of order. The steam and the tar constituents were cooled and led into a drain. Near this drain was a ventilating shaft, and the two workmen were killed by the vapors rising through this vent duct. In 1908 two other fatal cases (25) occurred in the cleaning of a railway tank car. The tank had been washed, but the workman, who entered with the approval of the foreman, succumbed and his mate on guard at the manhole had apparently gone in to his assistance as he also was found within the tank.

In 1909 (26) a very interesting case of benzol asphyxiation occurred in a coke-oven plant. Benzol was being collected in two large iron receivers. In order to control the valves it was necessary for the worker to enter a roofed-over pit in which the valves were located. During the filling period, the tank manholes were kept open. The only ventilation was that provided by two shafts which also served as entrances to the pit. A worker entered the pit to manipulate the valves and some time later he was found huddled up on the top of the receiver holding the valve by one hand. Later he fell to the bottom of the pit. Three fellow workmen who went to his rescue

were affected by the fumes and were forced to retire. A fourth worker was lowered with a rope, but with immediately fatal results. Finally, with the aid of a smoke helmet, a rescuer brought up the corpse of the first victim. Rambousek (9) suggests that possibly other volatile substances contributed to the poisoning in this case. In his summary he also describes the similar case of a foreman who failed to regulate a condensation system so that benzol fumes were permitted to escape, to which he succumbed as did also a rescuer, the foreman's case terminating fatally. Yet another such case is given in the Annual Report of the British Chief Inspector of Factories (27) in which a sudden change in pressure caused a more rapid distillation of benzol vapor than the condenser could deal with, so that the workman was overcome by the excess uncondensed fumes. Although he was resuscitated, he again succumbed when he returned to his work two nights later, the incident proving fatal.

Rambousek cites another fatal poisoning which occurred when a worker in an anilin factory inhaled the vapor from benzol which escaped from leaky valves. Beisele (28) records the case of a man who died while painting the inside of a tank with tar dissolved in benzol, the usual acute symptoms being followed by death within five minutes of exposure. Buchmann, in 1911 (29), reported an acute case of poisoning produced by benzol escaping from a piece of defective chemical apparatus. In the German Factory Inspector's Reports (30) for the year 1912, three cases of poisoning are described, two of these terminating fatally. One case was that of a workman painting the interior of a barrel with benzene containing paint; another that of a workman in a cleaning establishment who entered the washing machine in which there still remained a small quantity of benzol; and one in which the cold water had not been turned on for the condensation of benzol vapor, with the resultant escape of benzol fumes and the death of the workman. Heffter (31) reports a case of an assistant manager who, while opening a stopped pipe, succeeded in starting the benzene flow but was unable to stop it. Benzene splashed over his clothes, but he nevertheless continued working while two helpers who were with him felt sick and were obliged to leave. The foreman finally fainted and died, although he received three hours' treatment with oxygen. Albaugh (32) cites a possible case of fatal poisoning in a young man employed as a spray painter in a varnishing department. Robinson (33) cites three cases of death from benzol fumes where employees persistently entered tanks regardless of rules requiring the removal of fumes and without protecting themselves with life lines and other protective devices provided by the employers. He reports one of the cases in detail. A workman entered a tank which had but a slight odor of benzol, grew dizzy, crawled out and sat down on the ground

outside. In a few moments, however, he became unconscious and never recovered.

The demand for toluene for the manufacture of explosives during the World War led to the establishment of many plants for its production. Along with this enormous production of toluol, benzol was produced in very large quantities, and because of this wholesale increase in the handling of benzol, cases of acute poisoning suddenly increased. By 1916 Alice Hamilton (5) had already collected and reported 14 acute cases, with seven fatalities, in this country. At that time she placed benzene poisoning third on the list of industrial poisons. Of the 7 fatalities, 5 occurred among workmen in or around stills, the other 2 deaths occurring in the sulphonation of benzene during the synthetic production of phenol. Doctor Hamilton's first two cases were of steam fitters who were repairing pipes inside a benzene still. The still, before the repairs were begun, had been emptied and washed and was believed to be free from any dangerous concentration of benzol vapor. Shortly after entering the still, one of the men became unduly hilarious and excited, singing and shouting. Attempts were made to get him out through the narrow manhole, but he was so irrational that it took 10 minutes to rescue him. At first his fellow workman within the tank helped in the rescue, but later he also succumbed to the fumes and was rendered unconscious. It took about 20 minutes to get this second workman out of the still, at the end of which time he was dead. In another establishment two workmen entered a still which was supposed to be free from benzol but apparently was not, as they soon succumbed and lapsed into coma. Vigorous treatment was administered, but the one who had been in the still for the shorter period did not survive. Yet another case was that of a workman who attempted to stop a leak in a still but was overcome by benzol fumes, with fatal results. Two fellow workers who went to his aid were also overcome, but survived. In the sulphonating room of a factory the sulphonated benzene flowed into a "liming vat" and apparently had carried some benzene over with it which volatilized because of the heat. A workman was found dead beside the liming vat, and benzol fumes were shown to be escaping from the liming vat, the sulphonating kettle, and the supply pump in the same room.

Harrington (34) reported the cases of two steamfitters, repairing pipes in a benzene still which was provided with a small manhole. No mention is made of the preliminary cleansing of the still, but a fresh air supply was introduced through a 2-inch pipe under 60 pounds pressure. Within 45 minutes the older workman, aged 35, "acted crazy," becoming wildly excited and later unconscious. In the attempt to get him out through the manhole, which took 20 minutes, the younger man succumbed and was found dead.

Dworetzky (35) described the interesting history of a peculiar epidemic in a large Russian rubber factory. On March 25, 1914, many workers came down with headache and dizziness, nausea and vomiting, some falling to the floor unconscious. Several of them had to be sent home or to the hospital. Next day the workers who had returned were put to work in another room where nothing had occurred the previous day. Strange to relate the same group of symptoms again appeared, with a total of 102 persons affected. By the fourth day over 230 persons were involved; and after nine days of trial, the factory (employing 17,000 persons) was forced to close. The workers ascribed the symptoms to the use of a new rubber solvent. At first they said a colored odoriferous solvent was used which caused no trouble. When the solvent was changed, however, to a clear and less odorous one, the outbreak was precipitated. The management pointed out that this was unlikely, for they substituted only the very highest grade of Baku naptha products. The medical authorities (Bechterew) decided that the outbreak was one of hysteria or of benzene or benzol poisoning, together with hysteria. At the same time, in the tobacco factory of "Bogdanow" 18 workers (16 females and 2 males) came down with very similar symptoms, and in the tobacco firm of "Schaposchinkow" 20 workers fell unconscious to the floor. In the tobacco factory of Laferme eight cases appeared one day, and on the following day 89 additional cases, only one man in the entire factory remaining free from symptoms. Consideration was given at the time to the possibility of wholesale nicotine poisoning; but strangely enough seven cases appeared in a chocolate factory, then others in a garment factory, in a machine shop, and in other widely separated places of business. Fear was entertained by the populace that the poisoning (?) was due to a political plot. So intense was the excitement that the police suppressed all discussion of the cases and the factories closed their doors until the situation quieted down, so that the ultimate cause of the trouble was not ascertained. Dr. Alice Hamilton, while in Moscow in 1924, made inquiries concerning this outbreak and states that, after all the excitement had passed, investigation established the fact that the toxic substance responsible was benzene. It would seem that this "massenvergiftung" was, for the greater part, in the nature of hysteria, precipitated by a few actual cases of benzene poisoning which occurred in the rubber-making plant.

Adamkiewicz (36) reports a case of accidental acute poisoning in which a workman decended into a pump pit. After three hours he was carried out unconscious, but survived. A strong odor of benzol was present on the breath. Binder (37) cites a fatal case of a man employed in the making of benzol cement. He was overcome by fumes and later died. Cronin (38) reports five cases of posioning,

one being fatal. The fatal case was that of a workman who descended into a trench surrounding a large benzol tank, in order to tighten a nut at the site of a leak. He grew pale, dizzy, and weak and was forced to rest. On reattempting to work he collapsed and died shortly after. Other workers in the same trench complained only of slight dizziness after two hours' exposure. Later another workman shoveling earth back into the above mentioned trench became dizzy and collapsed and, on recovery, developed a sense of constriction in the chest, with tremor of extremities, and during the next four weeks had attacks of cardiac distress. Cronin cites three other cases of a spreader, a cement mixer, and a worker in a tank car, respectively, all of whom presented symptoms and signs of headache, dizziness, weakness, etc., which were relieved by going into the fresh air.

Cases of benzol poisoning occurring from the use of paints and lacquers containing this substance are of special interest. From this industry Rambousek (9) cites two interesting cases. In the first instance unconsciousness of a workman resulted from painting a retort with an anticorrosive paint. The accident was attributed to benzol fumes released by the paint in drying. The patient was rescued, given medical attention, and survived. In the second case, a rust-preventing paint with which a workman was painting the interior of a boiler, was the source of the benzol vapors. The paint contained considerable crude benzol, and the worker was overcome while working within a confined space. He was quickly resuscitated, but the effects incapacitated him for eight days. Rambousek calls attention to Schaefer's investigation (39) into the question of paints containing benzol. At the time (1909), Schaefer emphasized the hazard from benzol in paints, but considered the crude products, boiling between 130°–170° C., as relatively harmless. In 1905–6 many of the cases which occurred were attributable to the fumes of hydrocarbons other than benzol, and a case is cited in which a painter was engaged in painting the double bottom of a ship with "black varnish oil." Exposed to the concentrated fumes, he developed severe inflammation of the respiratory system, from which he eventually died. The black varnish oil was a mixture of coal-tar pitch in light coal-tar oil, the oil distilling at 170° C., and amounting to 31–33 per cent of the mixture. Schaefer cites other cases occurring in 1908–9. A workman was painting the inside of a boiler with a patented paint and was rendered unconscious. Three men going to his rescue were also rendered unconscious. He cites another fatal case due to the use of a patent color which contained 30 to 40 per cent benzol and which was being used within an inclosed space (a chain well). This case proved fatal despite the fact that the worker was allowed out in the open air at frequent intervals.

So far as the pathology of acute benzol poisoning is concerned, probably the earliest autopsy record is that of Sury-Bienz (40) in 1888. The findings in this case consisted of bright red spots on the body; the blood was fluid and dark red. Minute hemorrhages were found in pleural and intestinal mucosa. The lungs showed venous congestion, and a reddened condition of the lining of the air passages, which contained blood and mucous. In an acute case caused by inhalation of benzol, Beinhauer (41) found the heart-blood fluid, with engorgement of the abdominal vessels and hemorrhages into the gastric mucosa. Bloodstained mucus was found in the respiratory passages.

Buchmann (29) describes the autopsy findings in the acute case of a man killed by benzol escaping from a break in a chemical apparatus. Red spots were found on the skin, congestion of the internal organs was present, and minute hemorrhages in the pancreas were observed, though no striking findings were present on microscopical examination, and chemical tests of the organs were found to be negative for benzol. In her article on industrial poisons, Dr. Alice Hamilton (5) reports seven fatal cases of benzol poisoning. Two of these cases came to autopsy at the hands of Dr. H. S. Martland. The main findings as summarized by Doctor Hamilton follow:

Case 1.—Cyanosis (blueness) of the mucous membranes and finger tips; cyanosis of liver, spleen, and kidneys; dilation of the right heart, which was filled with dark fluid blood; pleural ecchymoses (small purple spots, due to hemorrhages under the surface of the tissue) and small areas of acute interstitial emphysema in the lungs.

Case 2.—Cyanosis of mouth, lips, and of finger tips; small amount of frothy fluid escaping from the mouth; cyanosis of the brain, heart, liver, and kidneys; petechial hemorrahages (small ecchymoses) in pleura and pericardium; reddened and irritated bronchi. On section of the lungs a decided odor of benzene was given off. There was an abnormal quantity of phenol in the urine, but not benzene.

In general, the symptoms and signs of acute poisoning by inhalation are faintness, dizziness, excitation, pallor and later flushing, weakness, headache, breathlessness, apprehension of death, tightness in the chest, visual disturbances, tremor, weakness in extremities, rapid pulse, cyanosis, unconsciousness or narcosis, collapse, tremor and convulsions, coma, acute mania or delirium preceding sudden death at times, or in other cases death occurring several hours to several days subsequent to exposure. In extreme concentrations, asphyxiation with death results from respiratory paralysis. When taken by mouth, the usual local signs and symptoms of an acute toxic gastritis are seen in addition to the general systemic manifestations.

In treatment of acute benzol poisoning the patient should, of course, be removed from the danger area and placed in a comfortable reclining

position. If carbon dioxide-oxygen, and respirator are at hand, this means of respiratory stimulation should be at once applied. If only oxygen tank and respirator be available, they may be used; and if no such apparatus be available, artificial respiration by the prone pressure or Schaeffer method should be applied until the patient has been breathing in a satisfactory manner for some minutes. The patient should be carefully watched, and at the first sign of respiratory failure or slowing of respiration the artificial respiration should be resumed. At all times it is important to keep the body warm by applying heat to the body and extremities. Cardiac and respiratory stimulants, such as caffeine, may be given.

In cases where benzol has been taken by mouth, the usual gastric lavage may be resorted to, followed by demulcents or bland drinks. Lecithin emulsion may be administered intravenously, as advocated by Nick, in doses of 5 c.c. of a 10 per cent solution.

Prolonged convalescence is essential in order to avoid complications.

Generally, cases of acute poisoning from inhalation are either rapidly fatal or respond favorably to treatment with more or less complete recovery within a short period. Late manifestations may appear, however. Cronin (38) reports spells of cardiac distress for four weeks after recovery. This was true also in Lewin's case, which presented cardiac distress, dizzy spells, and general depression. In the Report of the Chief Inspector of Factories and Workshops of Great Britain for 1918 (42) a case is reported of acute posioning due to too rapid distillation of benzol. The worker was overcome, but was revived and apparently passed through the experience sucessfully. On the second night of his return to work, however, for no apparent reason he lapsed into unconsciousness from which he never recovered. Inflammation of the respiratory tract and pleurisy may set in (43) (39) (9). Pyloric stenosis has been reported after poisoning by swallowing benzene (15). Albuminuria with casts and albumen may occur; also bloody stools (15). It is well to note that in many of the acute cases of poisoning reported, other volatile substances in addition to benzol have been present and probably have contributed to the seriousness of the picture presented. Schaeffer, in 1905–06, called attention to poisoning by the inhalation of other hydrocarbons. Hydrogen sulphide gas is frequently encountered in tar distilleries (9), and more rarely carbonic oxide (44). In many of the quick-drying paints, other solvents present in addition to the benzol probably play a part in the production of acute symptoms, and such symptoms are frequently attributed to benzene. That symptoms of acute benzol poisoning should be rather frequent is not surprising in the light of the findings given by Lehmann, who found that concentrations of 0.015 grams per liter of air (4,700 parts per

million) will produce confusion within half an hour, while concentrations from 0.02 to 0.03 grams per liter (6,160 to 9,190 parts per million) produce definite symptoms of poisoning within a few hours. That the benzol vapor in such concentrations is rapidly taken up is indicated by the fact that 80 per cent of the vapor is absorbed within a short period on exposure to concentrations of 4,700 parts per million. From these experiments, however, no definite ratio or proportion could be established between the absolute amount of benzol in the air and the amount absorbed. The signs and symptoms of acute poisoning will therefore depend not only on the concentration present but also upon the susceptibility of the individual and, to a lesser extent, perhaps, upon the activity in which he is engaged.

PUBLIC HEALTH ENGINEERING ABSTRACTS

Typhoid Versus Tourists.—Anon. *Ohio Health News*, vol. 2, No. 9, May 1, 1926, pp. 1–2. (Abstracted by Isador W. Mendelsohn.)

This article discusses various precautions to be observed by the camper to protect himself against disease, particularly the matter of safe water. After two summers' work there are 108 wells in Ohio now bearing the Seal of Safety of the Ohio State Department of Health.

The Engineering Aspects of Oyster Pollution.—R. E. Tarbett, Sanitary Engineer, United States Public Health Service, Washington, D. C., *American Journal of Public Health*, vol. 16, No. 1, January, 1926, pp. 5–12. (Abstracted by R. E. Tarbett.)

The question of oyster pollution is considered in two parts—first, conditions existing in growing areas; and, second, pollution due to insanitary handling, shucking, etc. Patrol over the sanitary conditions of growing and bedding areas appears to be the most important factor in the production of safe oysters. The protection of such areas, however, presents a difficult problem because of the many and varying influences involved.

The relation of the *B. coli* content of the shell liquor to the overlying water is discussed, with the conclusion that, with the data available, and without considering other data, the determination of the safety of oysters by the *B. coli* content of the shell liquor is misleading.

Oysters, being found only in tidal estuaries, are subject to possible polluting influences originating in fresh water streams discharging into those estuaries as well as to polluting influences in the tidal estuaries. Studies of pollution, therefore, involve studies of the streams above tidal influence, and of the tidal estuary or bay itself. These studies involve the estimates of amount of pollution entering the streams, together with the approximate dilution taking place under different conditions of stream flow and probable time interval

elapsing before entry of pollution to the area of tidal influence; they also give information on the amount of pollution discharged to tidal water, together with the action of tides, currents, bottom topography, and the like. Lags in tidal currents, vertical stratification, and barrier action of bars interfere greatly with estimates of time intervals and of movements of waters. In addition to the physical considerations, *B. coli* content of the water and of the shellfish should be studied.

Movements of water, effects of dilution, and conditions of pollution may best be studied by the establishment of *B. coli* contours under different conditions of tides and winds.

The handling, shucking, and packing of oysters appears somewhat analogous to the handling of milk, and should be controlled in the same manner in which the handling of milk is controlled.

Chlorination, in respect to oyster purification, should be considered only as an additional safeguard against accidental pollution and not as a substitute for sanitary control over growing areas.

Effect of Storage and Changing Sea Water on Contaminated Oysters.—Charles Krumwiede, William H. Park, Georgia Cooper, Marie Grund, Charles H. Tyler, and Carolyn Rosenstein, Bureau of Laboratories, Department of Health, New York City. The *American Journal of Public Health*, vol. 16, No. 3, March, 1926, pp. 263–268. (Abstracted by R. E. Tarbett.)

The experiments covered in this article are a continuation of the experiments on the effect of chlorination on oysters contaminated with typhoid bacilli, reported in the February issue of the *American Journal of Public Health.*

The experiments reported on in this article cover the observations of the effect of dry storage and the effect of changing sea water. The oysters in the experiment were obtained from Great South Bay, Long Island, and the water used was obtained from the same bay. Two lots of oysters were contaminated by suspending them in wire crates in tanks containing 8 gallons of contaminated sea water—Lot A, water contaminated by adding a suspension of feces containing *B. typhosus,* Lot B, water contaminated with freshly isolated cultures of *B. typhosus.* The oysters were allowed to drink actively over one night, and were then stored under temperature conditions approximating that of the colder months.

The materials used for examinations were as follows: Washings from the shell, using 10 c. c. of broth per oyster; the shell liquor; and the body of the oyster emulsified in 5 c. c. of broth. Examinations were made at intervals of 1, 5, 7, 9, 10, 14, 21, 28, 35, 41, and 49 days. All the oysters were dead at the end of 41 days. While the number of *B. typhosus* organisms that could be isolated was greatly reduced, positive results were, however, obtained in the shell liquor

and the body emulsion on the forty-ninth day. The shell-washing examination was negative at the end of two weeks in Lot A and five weeks in Lot B. A parallel series of observations on the presence of *B. coli* showed that the rate of reduction was somewhat parallel with that of *B. typhosus*. Experiments were made to show whether or not a dead oyster was a favorable culture medium for the typhoid bacillus. In oysters contaminated with pure cultures and held at room temperature there was slow multiplication, approximating 15 times, during the first 7 days, with a rapid rise in multiplication to approximately 1,000 times after 10 days. In ice-box temperature there was a moderate increase to the seventh day, followed by a decrease, with approximately the original number on the tenth day. Oysters contaminated with stool suspensions held at room temperature were so overgrown with other organisms as to prevent isolation by the direct plating method, but enrichment methods showed *B. typhosus* on the tenth day. At ice-box temperature this organism was also isolated on the tenth day. Reference is made to results of similar experiments carried on by others.

Fifty oysters were contaminated with typhoid feces and placed in 4 gallons of fresh sea water. This experiment was carried on during a period of 24 days, during which time the water was changed 18 times. The temperature of the water was kept between 60° and 70° F. The results indicated that three successive changes of water will give a reduction of about 99 per cent in typhoid bacilli. However, at the end of the twenty-fourth day, at which time the oysters were dead, it was possible to isolate the organism from the liquor-body emulsion and its shell.

The length of time that *B. typhosus* will survive in oysters during winter months in natural waters was also investigated. Oysters were obtained from beds in a condemned area and the experiment was carried on in the same area. These oysters were contaminated with a relatively light dose of typhoid feces and submerged in wire crates. The temperature conditions during the experiment were such as to indicate that the oysters were relatively inactive. The results show that the organism was isolated from the liquor up to the tenth day; from the bodies up to the thirty-first day; and from the gills up to the fifty-first day.

It the experiment Endo medium and brilliant green agar were employed, with brilliant green broth used for enrichment. The authors conclude that the only safe oyster is one which has been protected from any contamination with fecal pathogens for at least some months prior to harvesting.

A HEALTH STUDY OF TEN THOUSAND MALE INDUSTRIAL WORKERS

In the near future a bulletin[1] will be issued by the Section of Industrial Hygiene and Sanitation of the United States Public Health Service, entitled "A Health Study of 10,000 Male Industrial Workers."

This study represents the analysis of the physical condition of one of the largest groups of industrial workers yet studied, and includes workers in 10 industries.

Although the original study was made entirely from the viewpoint of industrial hygiene, the publication contains a large amount of basic material of especial value to the physiologist, relating to the blood pressure and pulse rate, vital capacity, measurements of the body and other physiological or physical facts ascertained in routine physical examination.

Of special interest to the physician is the contribution to medical knowledge made by this study regarding the distribution of defects and diseases at different ages. The graphs prepared to bring out these observations suggest the trend of certain diseases throughout the life of the industrial worker, and are also more or less applicable to the general male population of adult ages. This information, which represents a new point of view from which to judge causes of sickness and death, may be regarded as unique in the literature of vital statistics.

DEATHS DURING WEEK ENDED JUNE 19, 1926

Summary of information received by telegraph from industrial insurance companies for week ended June 19, 1926, and corresponding week of 1925. (From the Weekly Health Index, June 24, 1926, issued by the Bureau of the Census, Department of Commerce)

	Week ended June 19, 1926	Corresponding week 1925
Policies in force	64, 473, 873	60, 266, 214
Number of death claims	12, 127	12, 057
Death claims per 1,000 policies in force, annual rate	9. 8	10. 4

[1] Public Health Bulletin, No. 162.

Deaths from all causes in certain large cities of the United States during the week ended June 19, 1926, infant mortality, annual death rate, and comparison with corresponding week of 1925. (From the Weekly Health Index, June 24, 1926, issued by the Bureau of the Census, Department of Commerce)

City	Week ended June 19, 1926		Annual death rate per 1,000 corresponding week, 1925	Deaths under 1 year		Infant mortality rate, week ended June 19, 1926 [2]
	Total deaths	Death rate [1]		Week ended June 19, 1926	Corresponding week, 1925	
Total (66 cities)	6,589	11.9	11.4	767	743	[3] 60
Akron	31			2	5	21
Albany [4]	33	14.5	8.0	2	2	42
Atlanta	83			14	7	
White	52			9		
Colored	31	([5])		5		
Baltimore [4]	192	12.4	12.6	24	16	70
White	150			16		57
Colored	42	([5])		8		130
Birmingham	59	14.6	13.4	8	12	
White	26			3		
Colored	33	([5])		5		
Boston	176	11.7	12.0	21	22	59
Bridgeport	23			2	1	34
Buffalo	134	12.8	14.0	23	19	96
Cambridge	20	8.5	7.8	5	2	83
Camden	18	7.2	12.2	3	5	51
Canton	17	8.1	7.4	3	0	67
Chicago [4]	683	11.7	9.8	81	63	72
Cincinnati	128	16.2	13.4	13	8	81
Cleveland	197	10.7	8.5	19	19	49
Columbus	69	12.6	11.0	5	4	46
Dallas	48	12.5	18.3	8	9	
White	36			5		
Colored	12	([5])		3		
Dayton	47	13.8	11.2	2	1	31
Denver	50	9.1	13.9	6	7	
Des Moines	35	12.5	8.8	2	1	33
Detroit	306	12.4	11.1	52	37	84
Duluth	32	14.8	6.6	5	1	117
El Paso	26	12.4	11.9	6	8	
Erie	29			4	2	76
Fall River [4]	32	12.7	8.5	4	2	58
Flint	18	6.9	5.2	2	4	33
Fort Worth	17	5.6	9.9	5	15	
White	12			4		
Colored	5	([5])		1		
Grand Rapids	27	9.0	12.2	5	2	72
Houston	56			6	10	
White	41			5		
Colored	15	([5])		1		
Indianapolis	108	15.3	13.1	14	8	102
White	87			12		101
Colored	21			2		110
Jersey City	49	8.0	9.4	5	7	35
Kansas City, Kans	30	13.4	10.3	2	3	35
White	18			1		21
Colored	12	([5])		1		131
Kansas City, Mo	85	11.8	11.2	10	15	
Los Angeles	182			17	23	47
Louisville	72	12.1	13.1	3	7	26
White	53			3		30
Colored	19	([5])		0		0
Lowell	26			0	1	0
Lynn	29	14.5	13.2	3	2	75
Memphis	64	18.9	24.5	8	12	
White	32			7		
Colored	32	([5])		1		
Milwaukee	118	11.9	11.4	10	9	46
Minneapolis	101	12.1	11.8	15	11	83

[1] Annual rate per 1,000 population.
[2] Deaths under 1 year per 1,000 births. Cities left blank are not in the registration area for births.
[3] Data for 64 cities.
[4] Deaths for week ended Friday, June 18, 1926.
[5] 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, Forth Worth 14, Houston 25, Kansas City, Kans., 14, Louisville 17, Memphis 38, Nashville 30, New Orleans 26, Norfolk 38, Richmond 32, and Washington, D. C., 25.

Deaths from all causes in certain large cities of the United States during the week ended June 19, 1926, infant mortality, annual death rate, and comparison with corresponding week of 1925. (From the Weekly Health Index, June 24, 1926, issued by the Bureau of the Census, Department of Commerce)—Continued

City	Week ended June 19, 1926		Annual death rate per 1,000 corresponding week, 1925	Deaths under 1 year		Infant mortality rate, week ended June 19, 1926 [1]
	Total deaths	Death rate [1]		Week ended June 19, 1926	Corresponding week, 1925	
Nashville [4]	40	15.2	13.4	8	4	
White	21			6		
Colored	19	(5)		2		
New Bedford	18			4	4	70
New Haven	32	9.2	9.6	7	3	96
New Orleans	139	17.3	19.6	18	33	
White	80			8		
Colored	59	(5)		10		
New York	1,278	11.2	11.0	143	149	58
Bronx borough	166	9.6	8.3	10	16	33
Brooklyn borough	405	9.4	9.8	53	55	54
Manhattan borough	557	15.5	14.2	59	58	65
Queens borough	113	7.7	7.3	16	14	73
Richmond borough	37	13.5	19.2	5	6	88
Newark, N. J	87	9.9	10.6	12	12	57
Norfolk	39	11.7	9.2	6	4	112
White	15			1		30
Colored	24	(5)		5		249
Oakland	50	10.0	8.8	3	4	35
Oklahoma City	25			2	2	
Omaha	42	10.2	9.6	3	6	31
Paterson	28	10.2	9.6	3	7	52
Philadelphia	432	11.2	11.5	35	52	46
Pittsburgh	162	13.3	12.2	17	11	56
Portland, Oreg	74			2	11	20
Providence	62	11.8	12.5	7	3	58
Richmond	53	14.6	13.7	8	4	101
White	27			5		98
Colored	26	(5)		3		105
Rochester	62	10.1	8.4	3	3	24
St. Louis	185	11.6	12.5	12	25	
St. Paul	51	10.7	10.2	5	4	44
Salt Lake City [4]	33	12.9	10.4	2	5	28
San Antonio	67	17.0	20.3	22	22	
San Diego	34	16.1	13.8	2	3	42
San Francisco	163	15.0	11.3	9	6	54
Schenectady	15	8.4	9.0	1	1	29
Seattle	59			1	6	9
Somerville	17	8.9	8.4	1	1	26
Spokane	18	8.6	12.4	2	2	47
Springfield, Mass	27	9.7	9.9	4	2	58
Syracuse	36	10.2	11.7	6	6	76
Tacoma	16	7.9	12.0	3	3	70
Toledo	71	12.6	9.8	9	2	87
Trenton	29	11.3	16.6	5	3	84
Utica	25	12.7	14.4	4	1	88
Washington, D. C	143	14.1	14.8	19	15	108
White	87			11		91
Colored	56	(5)		8		146
Waterbury	28			4	2	86
Wilmington, Del	24	10.1	8.5	3	1	70
Worcester	55	14.9	10.7	3	7	35
Yonkers	24	10.8	9.2	3	2	67
Youngstown	30	9.5	7.2	5	4	64

For footnotes 1, 2, 4, and 5, see p. 1379.

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 Week Ended June 26, 1926

ALABAMA

	Cases
Cerebrospinal meningitis	1
Chicken pox	10
Diphtheria	5
Influenza	4
Lethargic encephalitis	1
Malaria	49
Measles	167
Mumps	10
Ophthalmia neonatorum	1
Pellagra	24
Pneumonia	25
Poliomyelitis	2
Rabies	1
Scarlet fever	6
Smallpox	18
Tetanus	6
Tuberculosis	348
Typhoid fever	33
Whooping cough	97

ARIZONA

Chicken pox	1
Diphtheria	2
German measles	5
Influenza	1
Measles	2
Mumps	2
Paratyphoid fever	2
Pneumonia	1
Scarlet fever	5
Trachoma	1
Tuberculosis	11
Typhoid fever	2
Whooping cough	7

ARKANSAS

	Cases
Chicken pox	8
Diphtheria	3
Hookworm disease	2
Influenza	4
Malaria	39
Measles	23
Mumps	2
Pellagra	12
Scarlet fever	8
Smallpox	1
Trachoma	2
Tuberculosis	7
Typhoid fever	26
Whooping cough	15

CALIFORNIA

Cerebrospinal meningitis:	
Long Beach	1
Los Angeles	2
Chicken pox	137
Diphtheria	100
Influenza	1
Measles	353
Mumps	133
Poliomyelitis:	
Alameda County	1
Long Beach	1
Maywood	1
Santa Paula	1
Scarlet fever	126
Smallpox	12
Tuberculosis	175
Typhoid fever	24
Whooping cough	43

COLORADO

	Cases
Chicken pox	28
Diphtheria	13
Influenza	1
Malaria	1
Measles	32
Mumps	2
Pneumonia	5
Scarlet fever	8
Smallpox	4
Tuberculosis	20
Typhoid fever	2
Whooping cough	21

CONNECTICUT

Cerebrospinal meningitis	1
Chicken pox	39
Diphtheria	13
German measles	24
Lethargic encephalitis	1
Measles	244
Mumps	5
Pneumonia (broncho)	24
Pneumonia (lobar)	26
Scarlet fever	49
Smallpox	1
Tetanus	1
Tuberculosis (all forms)	44
Typhoid fever	1
Whooping cough	33

DELAWARE

Malaria	2
Measles	22
Pneumonia	2
Scarlet fever	5
Tuberculosis	1
Typhoid fever	3

FLORIDA

Chicken pox	4
Diphtheria	8
German measles	1
Malaria	11
Measles	17
Mumps	9
Pneumonia	3
Poliomyelitis	1
Scarlet fever	4
Smallpox	13
Tetanus	1
Tuberculosis	8
Typhoid fever	9
Whooping cough	29

GEORGIA

Cerebrospinal meningitis	1
Chicken pox	7
Dengue	2
Diphtheria	1
Dysentery	40
Hookworm disease	1
Influenza	32
Malaria	41
Measles	72
Mumps	15
Pellagra	10

GEORGIA—continued

Pneumonia	11
Scarlet fever	1
Septic sore throat	4
Smallpox	22
Tuberculosis	25
Typhoid fever	47
Whooping cough	8

IDAHO

Cerebrospinal meningitis—Kellogg	1
Chicken pox	22
Diphtheria	3
Measles	4
Scarlet fever	1
Smallpox	1
Trachoma—Idaho Falls	1
Typhoid fever	1
Whooping cough	20

ILLINOIS

Cerebrospinal meningitis:	
Cook County	1
De Kalb County	1
La Salle County	1
Chicken pox	291
Diphtheria	92
Influenza	137
Lethargic encephalitis:	
Jo Daviess County	1
Kane County	1
St. Clair County	1
Shelby County	1
Measles	980
Mumps	66
Pneumonia	443
Poliomyelitis:	
Grundy County	1
Hardin County	1
Scarlet fever	191
Smallpox	12
Tuberculosis	533
Typhoid fever	16
Whooping cough	166

INDIANA

Chicken pox	56
Diphtheria	13
Influenza	12
Measles	467
Pneumonia	6
Scarlet fever	57
Smallpox	62
Tuberculosis	40
Typhoid fever	12
Whooping cough	80

IOWA

Cerebrospinal meningitis	2
Chicken pox	10
Diphtheria	12
German measles	1
Measles	35
Mumps	2
Scarlet fever	19
Smallpox	40
Tuberculosis	13
Whooping cough	41

KANSAS

	Cases
Cerebrospinal meningitis—New Albany	1
Chicken pox	21
Diphtheria	4
Dysentery	2
German measles	3
Influenza	8
Malaria	1
Measles	137
Mumps	6
Pneumonia	24
Poliomyelitis—Pratt	1
Scarlet fever	22
Smallpox	9
Tetanus	2
Tuberculosis	37
Typhoid fever	8
Whooping cough	99

LOUISIANA

Diphtheria	6
Influenza	24
Malaria	13
Paratyphoid fever	4
Pellagra	13
Pneumonia	39
Poliomyelitis	1
Scarlet fever	5
Smallpox	5
Tuberculosis	20
Typhoid fever	37
Whooping cough	9

MAINE

Chicken pox	5
Diphtheria	3
German measles	8
Lethargic encephalitis	1
Measles	176
Mumps	9
Pneumonia	8
Poliomyelitis	1
Scarlet fever	12
Tuberculosis	7
Typhoid fever	1
Whooping cough	14

MARYLAND [1]

Cerebrospinal meningitis	1
Chicken pox	47
Diphtheria	17
Dysentery	1
German measles	3
Influenza	3
Malaria	1
Measles	154
Mumps	67
Paratyphoid fever	2
Pneumonia (broncho)	15
Pneumonia (lobar)	20
Scarlet fever	79
Septic sore throat	4
Tuberculosis	122
Typhoid fever	9
Typhus fever	1
Vincent's angina	1
Whooping cough	50

[1] Week ended Friday.

MASSACHUSETTS

	Cases
Cerebrospinal meningitis	4
Chicken pox	167
Conjunctivitis (suppurative)	11
Diphtheria	67
German measles	132
Influenza	8
Lethargic encephalitis	3
Measles	580
Mumps	105
Ophthalmia neonatorum	5
Pneumonia (lobar)	60
Scarlet fever	191
Septic sore throat	1
Tetanus	1
Trachoma	1
Trichinosis	2
Tuberculosis (pulmonary)	138
Tuberculosis (other forms)	23
Typhoid fever	6
Whooping cough	172

MICHIGAN

Diphtheria	136
Measles	769
Pneumonia	29
Scarlet fever	244
Smallpox	13
Tuberculosis	51
Typhoid fever	7
Whooping cough	94

MINNESOTA

Cerebrospinal meningitis	1
Chicken pox	94
Diphtheria	50
Influenza	3
Lethargic encephalitis	1
Measles	361
Pneumonia	1
Scarlet fever	146
Smallpox	5
Tuberculosis	52
Typhoid fever	9
Whooping cough	28

MISSISSIPPI

Diphtheria	1
Scarlet fever	2
Smallpox	2
Typhoid fever	21

MISSOURI

(Exclusive of Kansas City)

Chicken pox	12
Diphtheria	53
Measles	200
Mumps	5
Pneumonia	1
Scarlet fever	73
Smallpox	8
Trachoma	4
Tuberculosis	37
Typhoid fever	16
Whooping cough	68

MONTANA

	Cases
Chicken pox	4
Diphtheria	11
Measles	21
Mumps	1
Rocky Mountain spotted fever	1
Scarlet fever	14
Smallpox	4
Whooping cough	1

NEBRASKA

Cerebrospinal meningitis	1
Chicken pox	13
Diphtheria	2
German measles	2
Measles	33
Mumps	1
Pneumonia	1
Scarlet fever	23
Smallpox	11
Tetanus	1
Tuberculosis	2
Whooping cough	12

NEW JERSEY

Cerebrospinal meningitis	3
Chicken pox	146
Diphtheria	69
Influenza	2
Measles	527
Pneumonia	54
Poliomyelitis	1
Scarlet fever	172
Trachoma	1
Typhoid fever	10
Whooping cough	86

NEW MEXICO

Chicken pox	3
Diphtheria	3
Measles	10
Pellagra	2
Scarlet fever	1
Smallpox	1
Tuberculosis	13
Typhoid fever	2
Whooping cough	23

NEW YORK
(Exclusive of New York City)

Cerebrospinal meningitis	1
Chicken pox	183
Diphtheria	68
German measles	258
Influenza	2
Lethargic encephalitis	2
Malaria	9
Measles	1,747
Mumps	103
Ophthalmia neonatorum	2
Pneumonia	159
Poliomyelitis	3
Scarlet fever	115
Smallpox	5
Tetanus	5
Typhoid fever	11
Vincent's angina	7
Whooping cough	338

NORTH CAROLINA

	Cases
Chicken pox	53
Diphtheria	18
German measles	54
Measles	256
Poliomyelitis	2
Scarlet fever	20
Septic sore throat	1
Smallpox	24
Typhoid fever	31
Whooping cough	313

OKLAHOMA

(Exclusive of Oklahoma City and Tulsa)

Cerebrospinal meningitis—Ottawa County	1
Chicken pox	2
Diphtheria	3
Influenza	28
Malaria	25
Measles	29
Mumps	2
Pellagra	14
Pneumonia	5
Scarlet fever	10
Smallpox	3
Typhoid fever	29
Whooping cough	71

OREGON

Anthrax	1
Cerebrospinal meningitis	2
Chicken pox	22
Diphtheria	17
Influenza	7
Measles	65
Mumps	17
Pneumonia	[1]3
Scarlet fever	26
Septic sore throat	1
Smallpox:	
Lane County	12
Scattering	11
Tuberculosis	10
Typhoid fever	6
Whooping cough	23

PENNSYLVANIA

Cerebrospinal meningitis:	
Cambridge Springs	1
Pittsburgh	1
Chicken pox	252
Diphtheria	129
German measles	66
Impetigo contagiosa	1
Malaria	1
Measles	1,840
Mumps	53
Pneumonia	18
Scabies	9
Scarlet fever	319
Trachoma	2
Tuberculosis	133
Typhoid fever	18
Whooping cough	351

[1] Deaths.

RHODE ISLAND

	Cases
Diphtheria	1
German measles	5
Influenza	1
Measles	42
Mumps	1
Ophthalmia neonatorum	2
Scarlet fever	2
Septic sore throat	1
Tuberculosis	14
Typhoid fever	1
Whooping cough	5

SOUTH DAKOTA

Cerebrospinal meningitis	1
Chicken pox	4
Diphtheria	1
Measles	16
Mumps	2
Scarlet fever	29
Smallpox	1
Typhoid fever	3
Whooping cough	2

TENNESSEE

Chicken pox	10
Diphtheria	4
Dysentery	3
Influenza	4
Malaria	29
Measles	148
Mumps	2
Ophthalmia neonatorum	1
Pellagra	25
Pneumonia	6
Rabies	1
Scarlet fever	6
Smallpox:	
Memphis	13
Scattering	5
Tuberculosis	46
Typhoid fever	23
Whooping cough	44

TEXAS

Anthrax	10
Cerebrospinal meningitis	1
Chicken pox	33
Dengue	4
Diphtheria	16
Dysentery	4
Influenza	38
Measles	16
Mumps	71
Paratyphoid fever	4
Pellagra	5
Pneumonia	10
Poliomyelitis	1
Scarlet fever	27
Smallpox	30
Trachoma	3
Tuberculosis	31
Typhoid fever	29
Whooping cough	102

UTAH

	Cases
Chicken pox	9
Diphtheria	6
German measles	8
Measles	19
Mumps	6
Pneumonia	1
Scarlet fever	6
Smallpox	1
Tuberculosis	1
Typhoid fever	1
Whooping cough	77

VERMONT

Chicken pox	28
Measles	56
Mumps	11
Scarlet fever	1
Typhoid fever	2
Whooping cough	29

WASHINGTON

Cerebrospinal meningitis:	
Spokane	5
Thurston County	1
Chicken pox	57
Diphtheria	13
German measles	12
Measles	107
Mumps	24
Poliomyelitis—Tacoma	1
Scarlet fever	32
Smallpox	13
Tuberculosis	8
Typhoid fever	6
Whooping cough	23

WEST VIRGINIA

Chicken pox	22
Diphtheria	12
Influenza	1
Measles	636
Scarlet fever	20
Smallpox	11
Tuberculosis	21
Typhoid fever	11
Whooping cough	22

WISCONSIN

Milwaukee:	
Chicken pox	88
Diphtheria	17
German measles	4
Influenza	1
Measles	209
Mumps	39
Pneumonia	10
Scarlet fever	16
Tuberculosis	13
Typhoid fever	1
Whooping cough	67

WISCONSIN—continued

Scattering:	Cases
Cerebrospinal meningitis	1
Chicken pox	72
Diphtheria	27
German measles	88
Influenza	13
Measles	881
Mumps	58
Pneumonia	9
Poliomyelitis	1
Scarlet fever	38
Tuberculosis	28
Typhoid fever	3
Whooping cough	58

WYOMING

	Cases
Chicken pox	7
Diphtheria	3
German measles	1
Measles	8
Mumps	3
Pneumonia	1
Rocky Mountain spotted fever:	
Fremont County	1
Johnson County	2
Natrona County	1
Scarlet fever	10
Whooping cough	12

Report for Week Ended June 19, 1926

NORTH DAKOTA

	Cases
Chicken pox	3
Diphtheria	2
German measles	10
Measles	21
Mumps	1
Scarlet fever	23
Smallpox	4
Tuberculosis	2

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	Cerebrospinal meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
May, 1926										
Colorado	0	71	7	----	342	----	0	133	7	3
Georgia	3	60	191	130	581	51	1	36	138	52
Illinois	13	300	233	9	5,095	1	4	1,397	137	34
Maine	3	9	374	0	1,403	0	1	95	0	13
Maryland	1	69	54	2	1,869	0	0	244	0	24
Michigan	0	349	23	0	6,441	----	4	1,288	45	23
Minnesota	2	208	16	0	3,526	----	2	1,313	52	9
Mississippi	2	40	1,294	5,334	1,693	1,178	1	31	77	109
Missouri	2	311	40	9	7,227	1	0	1,122	47	64
Montana	3	5	33	3	447	----	0	138	24	4
North Carolina	1	65	----	----	1,705	----	0	87	190	21
Ohio	6	313	164	2	8,824	0	0	1,363	201	39
Oklahoma [1]	1	35	540	168	599	92	1	173	114	58
West Virginia	2	57	103	----	3,394	----	0	165	17	57
Wyoming	0	6	8	----	26	----	0	120	6	0

[1] Exclusive of Oklahoma City and Tulsa.

RECIPROCAL NOTIFICATIONS

Notifications regarding communicable diseases sent during the month of May, 1926, to other State health departments by departments of health of certain States

Referred by—	Cerebrospinal meningitis	Diphtheria	German measles	Measles	Scarlet fever	Smallpox	Tuberculosis	Typhoid fever
Connecticut	1	----	----	----	1	----	----	----
Illinois	----	----	----	1	----	----	11	----
Minnesota	----	----	----	----	----	----	36	----
New York	----	2	1	----	1	3	----	1

PLAGUE-ERADICATIVE MEASURES IN LOS ANGELES, CALIF.

The following items were taken from the reports of plague-eradicative measures from Los Angeles, Calif.:

Week ended June 19, 1926:

Number of rats trapped	442
Number of rats found to be plague infected	0
Number of squirrels examined	1,165
Number of squirrels found to be plague infected	0
Number of mice trapped	289
Number of mice found to be plague infected	0

Date of discovery of last plague-infected rodent, Nov. 6, 1925.
Date of last human case, Jan. 15, 1925.

GENERAL CURRENT SUMMARY AND WEEKLY REPORTS FROM CITIES

Diphtheria.—For the week ended June 12, 1926, 36 States reported 1,083 cases of diphtheria. For the week ended June 13, 1925, the same States reported 1,028 cases of this disease. Ninety-seven cities situated in all parts of the country and having an aggregate population of nearly 29,750,000, reported 785 cases of diphtheria for the week ended June 12, 1926. Last year for the corresponding week they reported 645 cases. The estimated expectancy for these cities was 788 cases. The estimated expectancy is based on the experience of the last nine years, excluding epidemics.

Measles.—Thirty-four States reported 12,985 cases of measles for the week ended June 12, 1926, and 5,603 cases of this disease for the week ended June 13, 1925. Ninety-seven cities reported 4,932 cases of measles for the week this year, and 3,190 cases last year.

Poliomyelitis.—The health officers of 37 States reported 19 cases of poliomyelitis for the week ended June 12, 1926. The same States reported 46 cases for the week ended June 13, 1925.

Scarlet fever.—Scarlet fever was reported for the week as follows: Thirty-six States—this year, 2,677 cases; last year, 1,819 cases; 97 cities—this year, 1,460 cases; last year, 934 cases; estimated expectancy, 759 cases.

Smallpox.—For the week ended June 12, 1926, 37 States reported 499 cases of smallpox. Last year for the corresponding week they reported 662 cases. Ninety-seven cities reported smallpox for the week as follows: 1926, 95 cases; 1925, 204 cases; estimated expectancy 112 cases. Two deaths from smallpox were reported by these cities for the week this year—at Omaha, Nebr.

Typhoid fever.—Two hundred and seventy-seven cases of typhoid fever were reported for the week ended June 12, 1926, by 36 States. For the corresponding week of 1925, the same States reported 543 cases of this disease. Ninety-seven cities reported 70 cases of typhoid fever for the week this year and 149 cases for the correspond-

ing week last year. The estimated expectancy for these cities was 87 cases.

Influenza and pneumonia.—Deaths from influenza and pneumonia were reported for the week by 91 cities, with a population of more than 29,000,000, as follows: 1926, 585 deaths; 1925, 578.

City reports for week ended June 12, 1926

The "estimated expectancy" given for diphtheria, poliomyelitis, scarlet fever, smallpox, and typhoid fever is the result of an attempt to ascertain from previous occurrence how many cases of the disease under consideration 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 week 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 nonepidemic years.

If reports have not been received for the full nine years, data are used for as many years as possible, but no year earlier than 1917 is included. In obtaining the estimated expectancy the figures are smoothed when necessary to avoid abrupt deviations 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, 1925, estimated	Chicken pox, cases reported	Diphtheria		Influenza		Measles, cases reported	Mumps, cases reported	Pneumonia, deaths reported
			Cases, estimated expectancy	Cases reported	Cases reported	Deaths reported			
NEW ENGLAND									
Maine:									
Portland	75,333	1	1	0	0	0	54	1	2
New Hampshire:									
Concord	22,546	0	0	0	0	0	0	0	0
Manchester	83,097	0	1	0	0	0	27	0	0
Vermont:									
Barre	10,008		0						
Massachusetts:									
Boston	779,620	24	49	13	3	0	93	49	21
Fall River	128,993	1	3	2	0	0	10	2	0
Springfield	142,065	5	2	0	2	2	5	6	1
Worcester	190,757	4	4	2	0	0	0	5	4
Rhode Island:									
Pawtucket	69,760	3	1	0	0	0	5	0	2
Providence	267,918	0	7	4	0	1	42	0	2
Connecticut:									
Bridgeport	(1)	5	5	8	0	1	13	0	0
Hartford	160,197	2	5	0	0	1	7	0	5
New Haven	178,927	10	2	0	0	0	50	1	6
MIDDLE ATLANTIC									
New York:									
Buffalo	538,016	20	10	5	0	0	33	1	18
New York	5,873,356	173	246	194	21	10	419	69	127
Rochester	316,786	5	6	22	0	1	44	3	10
Syracuse	182,003	12	5	0	0	0	341	16	1
New Jersey:									
Camden	128,642	4	4	5	0	0	28	1	0
Newark	452,513	29	13	5	0	0	80	20	9
Trenton	132,020	3	3	0	0	0	27	0	2
Pennsylvania:									
Philadelphia	1,979,364	48	59	74		6	221	11	31
Pittsburgh	631,563	29	18	6		1	187	3	22
Reading	112,707	4	2	1		1	40	0	0
EAST NORTH CENTRAL									
Ohio:									
Cincinnati	409,333	6	7	8	0	2	162	10	1
Cleveland	936,485	53	18	29	0	2	30	3	12
Columbus	279,836	7	2	27	0	0	63	1	4
Toledo	287,380	41	5	0	0	2	287	0	2

[1] No estimate made.

City reports for week ended June 12, 1926—Continued

Division, State, and city	Population July 1, 1925, estimated	Chicken pox, cases reported	Diphtheria		Influenza		Measles, cases reported	Mumps, cases reported	Pneumonia, deaths reported
			Cases, estimated expectancy	Cases reported	Cases reported	Deaths reported			
EAST NORTH CENTRAL—continued									
Indiana:									
Fort Wayne	97,846	9	1	0	0	0	60	0	3
Indianapolis	358,819	0	4	1	0	0	12	0	8
South Bend	80,091	2	1	1	0	0	59	0	2
Terre Haute	71,071	1	1	0	0	0	4	0	1
Illinois:									
Chicago	2,995,239	154	82	74	2	4	291	23	47
Peoria	81,564	0	1	0	0	0	0	1	0
Springfield	63,923	0	1	0	1	1	5	2	0
Michigan:									
Detroit	1,245,824	95	36	59	2	3	50	12	28
Flint	130,316	13	3	3	0	0	103	1	4
Grand Rapids	153,698	7	2	0	0	0	72	0	0
Wisconsin:									
Kenosha	50,891	8	1	0	0	0	64	0	1
Madison	46,385		0						
Milwaukee	509,192	79	11	11	3	3	282	28	16
Racine	67,707	1	0	1	0	0	242	13	1
Superior	39,671	0	0	0	0	0	6	0	0
WEST NORTH CENTRAL									
Minnesota:									
Duluth	110,502	8	1	0	0	0	91	0	0
Minneapolis	425,435	67	13	38	0	0	61	3	7
St. Paul	246,001		14						
Iowa:									
Davenport	52,469	1	1	1	0		5	0	
Sioux City	76,411	2	0	1	0		0	0	
Waterloo	36,771	2	0	1	0		31	1	
Missouri:									
Kansas City	367,481		5						
St. Joseph	78,342	2	0	0	0	0	8	0	2
St. Louis	821,543	7	36	62	1	1	328	4	
North Dakota:									
Fargo	26,403		1						
Grand Forks	14,811		0						
South Dakota:									
Aberdeen	15,036	0	0	0	0		6	0	
Sioux Falls	30,127	0	0	0	0	0	7	0	0
Nebraska:									
Lincoln	60,941	3	1	0	0	0	0	3	2
Omaha	211,768	10	2	5	0	0	45	1	8
Kansas:									
Topeka	55,411	19	1	1	0	0	10	0	0
Wichita	88,367	2	1	1	0	0	5	0	0
SOUTH ATLANTIC									
Delaware:									
Wilmington	122,049	0	1	3	0	0	1	0	3
Maryland:									
Baltimore	796,206	75	16	8	2	0	36	88	17
Cumberland	33,741	0	0	1	0	0	6	0	1
Frederick	12,035	0	0	0	0	0	0	2	1
District of Columbia:									
Washington	497,906	26	7	6	1	0	136	0	7
Virginia:									
Lynchburg	30,395	3	0	1	0	0	34	2	0
Norfolk	(1)	15	0	0	0	0	40	1	1
Richmond	186,403	4	1	1	0	1	118	5	3
Roanoke	58,208	2	1	0	0	0	14	0	2
West Virginia:									
Charleston	49,019	1	0	1	0	0	41	0	0
Huntington	63,485	0	0	1	0	0	0	0	1
Wheeling	56,208	5	0	0	0	0	81	0	1
North Carolina:									
Raleigh	30,371	9	0	2	0	0	1	0	0
Wilmington	37,061	2	0	0	0	1	3	1	2
Winston-Salem	69,031	3	0	8	0	0	16	0	0

(1) No estimate made.

City reports for week ended June 12, 1926—Continued

Division, State, and city	Population July 1, 1925, estimated	Chicken pox, cases reported	Diphtheria		Influenza		Measles, cases reported	Mumps, cases reported	Pneumonia, deaths reported
			Cases, estimated expectancy	Cases reported	Cases reported	Deaths reported			
SOUTH ATLANTIC—con.									
South Carolina:									
Charleston	73,125	1	0	1	2	0	2	0	2
Columbia	41,225	7	0	0	0	0	1	2	0
Greenville	27,311	0	0	0	0	0	0	1	0
Georgia:									
Atlanta	(1)	8	1	0	2	1	45	1	7
Brunswick	16,809	1	0	0	0	0	7	0	0
Savannah	93,134	0	0	0	0	0	4	0	1
Florida:									
Miami	69,754	2		4	1	0	14	1	3
St. Petersburg	26,847		0			0			0
Tampa	94,743	1	1	0	0	0	1	0	3
EAST SOUTH CENTRAL									
Kentucky:									
Covington	58,309	0	0	0	0	0	6	0	0
Louisville	305,935	3	3	1	2	1	16	0	9
Tennessee:									
Memphis	174,533	6	1	3	0	1	127	0	8
Nashville	136,220	0	0	0	0	4	9	0	2
Alabama:									
Birmingham	205,670	7	1	1	0	1	107	5	5
Mobile	65,955	0	1	0	0	0	0	0	0
Montgomery	46,481	0	0	0	0	0	4	2	0
WEST SOUTH CENTRAL									
Arkansas:									
Fort Smith	31,643	0	1	0	0		0	0	
Little Rock	74,216	1	0	0	0	0	25	1	0
Louisiana:									
New Orleans	414,493	1	5	4	6	4	0	0	9
Shreveport	57,857	0	0	0	0	0	0	0	0
Oklahoma:									
Oklahoma City	(1)	0	0	3	4	0	4	0	2
Texas:									
Dallas	194,450	11	2	3	0	0	2	0	5
Galveston	48,375	0	0	0	0	0	0	0	0
Houston	164,954	0	1	2	0	0	0	0	1
San Antonio	198,069	0	1	2	0	0	2	0	5
MOUNTAIN									
Montana:									
Billings	17,971	2	0	0	0	0	8	0	0
Great Falls	29,883	3	1	0	0	0	34	0	1
Helena	12,037	0	0	0	0	0	0	0	1
Missoula	12,668	0	0	0	0	0	4	1	0
Idaho:									
Boise	23,042	0	0	1	0	0	3	0	0
Colorado:									
Denver	280,911	33	9	5		1	26	0	2
Pueblo	43,787	6	2	1	0	0	18	0	3
New Mexico:									
Albuquerque	21,000	4	1	0	0	0	2	0	0
Arizona:									
Phoenix	38,669	1	1	1	0	0	0	0	5
Utah:									
Salt Lake City	130,948	14	3	7	0	0	8	8	2
Nevada:									
Reno	12,665	0	0	0	0	0	0	0	0
PACIFIC									
Washington:									
Seattle	(1)	29	4	7	0		54	2	
Spokane	108,897	32	2	3	0		15	0	
Tacoma	104,455	4	1	4	0	0	4	0	2
Oregon:									
Portland	282,383	17	4	11	0	0	54	2	5
California:									
Los Angeles	(1)	46	34	30	5	0	3	10	12
Sacramento	72,260	4	2	3	0	0	1	7	1
San Francisco	557,530	34	18	12	1	0	143	10	4

[1] No estimate made.

City reports for week ended June 12, 1926—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		
NEW ENGLAND											
Maine:											
Portland	1	2	0	0	0	1	1	0	0	2	25
New Hampshire:											
Concord	0	2	0	0	0	2	0	0	0	0	7
Manchester	1	6	0	0	0	1	0	0	0	0	26
Vermont:											
Barre	1		0				0				
Massachusetts:											
Boston	38	53	0	0	0	20	2	3	0	65	198
Fall River	2	1	0	0	0	4	1	1	0	2	37
Springfield	5	8	0	0	0	4	1	0	0	0	30
Worcester	6	12	0	0	0	4	0	1	0	2	45
Rhode Island:											
Pawtucket	1	2	0	0	0	1	0	0	0	3	16
Providence	6	2	0	0	0	2	0	0	0	3	54
Connecticut:											
Bridgeport	6	19	0	0	0	1	0	0	0	1	25
Hartford	3	0	0	0	0	1	0	0	0	3	
New Haven	2	7	0	0	0	2	0	2	0	2	38
MIDDLE ATLANTIC											
New York:											
Buffalo	17	7	0	0	0	12	1	3	0	30	156
New York	154	213	0	1	0	[1] 105	12	6	2	66	1,330
Rochester	12	6	0	0	0	8	0	2	1	9	97
Syracuse	8	3	0	0	0	2	0	0	0	24	52
New Jersey:											
Camden	3	6	0	0	0	0	0	0	0	5	24
Newark	15	29	0	0	0	8	1	0	0	37	97
Trenton	2	11	0	0	0	2	1	0	0	0	24
Pennsylvania:											
Philadelphia	61	76	1	0	0	46	5	1	0	40	9 436
Pittsburgh	22	35	1	0	0	12	1	0	0	71	172
Reading	1	6	0	0	0	1	0	0	0	6	21
EAST NORTH CENTRAL											
Ohio:											
Cincinnati	8	9	2	4	0	13	1	2	0	12	120
Cleveland	17	113	2	0	0	15	1	0	0	100	188
Columbus	6	26	2	1	0	5	0	0	0	17	71
Toledo	10	18	1	0	0	10	1	1	0	51	65
Indiana:											
Fort Wayne	2	10	3	1	0	0	0	1	0	6	30
Indianapolis	8	11	8	8	0	6	1	1	0	0	104
South Bend	3	8	1	0	0	1	0	0	0	2	11
Terre Haute	2	5	1	0	0	0	0	0	0	1	20
Illinois:											
Chicago	85	97	2	4	0	68	3	0	0	41	615
Peoria	2	1	0	0	0	1	0	0	0	9	14
Springfield	1	7	1	0	0	0	0	0	0	8	13
Michigan:											
Detroit	57	149	4	0	0	27	3	2	0	86	329
Flint	4	21	1	0	0	3	0	0	0	8	28
Grand Rapids	4	11	0	0	0	0	1	0	0	6	18
Wisconsin:											
Kenosha	1	3	1	0	0	0	0	0	0	6	11
Madison	1		1				0				
Milwaukee	19	14	6	0	0	6	0	0	0	61	127
Racine	3	0	1	0	0	0	0	0	0	15	16
Superior	1	4	2	0	0	0	1	0	0	0	5
WEST NORTH CENTRAL											
Minnesota:											
Duluth	3	18	3	0	0	2	0	0	0	3	23
Minneapolis	23	74	9	0	0	5	1	0	0	4	111
St. Paul	17		4				0				

[1] Pulmonary tuberculosis only.

City reports for week ended June 12, 1926—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		
WEST NORTH CENTRAL—continued											
Iowa:											
Davenport	0	3	4	0	----	----	0	0	----	1	----
Sioux City	1	11	2	2	----	----	0	0	----	2	----
Waterloo	2	0	0	0	----	----	0	0	----	4	----
Missouri:											
Kansas City	5	----	3	----	----	----	1	----	----	----	----
St. Joseph	1	2	0	0	0	0	0	0	0	1	36
St. Louis	22	73	3	6	0	14	3	1	1	52	193
North Dakota:											
Fargo	1	----	0	----	----	----	0	----	----	----	----
Grand Forks	1	----	0	----	----	----	0	----	----	----	----
South Dakota:											
Aberdeen	1	10	0	0	----	----	0	0	----	21	----
Sioux Falls	1	1	0	0	0	0	0	0	0	8	2
Nebraska:											
Lincoln	1	3	0	4	0	0	1	0	0	10	19
Omaha	3	70	4	5	2	0	0	0	1	1	53
Kansas:											
Topeka	1	5	1	0	0	0	0	0	0	7	22
Wichita	1	0	3	0	0	0	1	1	0	12	17
SOUTH ATLANTIC											
Delaware:											
Wilmington	4	2	0	0	0	1	0	0	0	2	29
Maryland:											
Baltimore	18	29	1	0	0	14	3	0	0	49	195
Cumberland	0	0	0	0	0	1	0	0	0	0	15
Frederick	0	0	0	0	0	0	0	0	0	0	5
District of Columbia:											
Washington	12	19	1	1	0	9	2	1	0	37	148
Virginia:											
Lynchburg	0	2	1	0	0	1	0	0	0	8	10
Norfolk	1	11	0	1	0	3	1	0	0	24	----
Richmond	2	16	0	1	0	3	1	2	0	4	44
Roanoke	0	0	0	1	0	1	0	0	0	0	21
West Virginia:											
Charleston	1	0	0	0	0	2	0	0	0	7	14
Huntington	1	0	0	0	0	0	0	0	0	0	14
Wheeling	2	1	0	0	0	0	1	0	0	3	12
North Carolina:											
Raleigh	0	0	1	0	0	0	1	0	0	8	10
Wilmington	0	0	0	1	0	0	0	0	0	5	10
Winston-Salem	0	2	2	0	0	2	1	0	0	2	15
South Carolina:											
Charleston	0	1	0	1	0	2	1	0	0	5	25
Columbia	0	0	1	0	0	0	1	3	0	0	----
Greenville	0	1	0	0	0	0	1	0	0	1	10
Georgia:											
Atlanta	4	0	6	0	0	5	2	8	1	13	75
Brunswick	0	0	0	0	0	0	1	0	0	0	7
Savannah	0	0	0	3	0	3	1	0	0	0	33
Florida:											
Miami	----	0	----	0	0	0	----	2	0	7	36
St. Petersburg	0	----	0	----	0	1	0	----	0	----	17
Tampa	0	1	0	11	0	0	1	0	1	0	29
EAST SOUTH CENTRAL											
Kentucky:											
Covington	0	2	0	0	0	1	1	0	0	0	11
Louisville	3	9	1	0	0	4	1	1	0	3	73
Tennessee:											
Memphis	2	3	1	0	0	8	2	7	0	3	----
Nashville	1	0	1	1	0	6	2	1	1	1	54
Alabama:											
Birmingham	1	1	5	5	0	3	2	0	0	29	65
Mobile	0	0	1	0	0	1	0	1	0	1	21
Montgomery	0	0	1	4	0	0	1	1	0	0	18

City reports for week ended June 12, 1926—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		
WEST SOUTH CENTRAL											
Arkansas:											
Fort Smith	1	1	0	0	-------	-------	0	0	-------	2	-------
Little Rock	1	7	1	0	0	3	1	0	0	0	-------
Louisiana:											
New Orleans	3	10	2	0	0	15	3	5	0	3	130
Shreveport	0	0	1	1	0	6	1	1	1	1	29
Oklahoma:											
Oklahoma City	1	0	5	0	0	4	0	1	0	0	25
Texas:											
Dallas	2	2	2	1	0	8	2	3	0	11	69
Galveston	0	0	0	6	0	1	1	0	0	0	11
Houston	1	0	1	0	0	6	2	0	2	0	51
San Antonio	0	0	0	0	0	9	1	3	0	0	79
MOUNTAIN											
Montana:											
Billings	1	2	1	0	0	0	0	0	0	0	4
Great Falls	2	2	2	0	0	0	1	0	0	4	14
Helena	0	0	0	0	0	0	0	0	0	0	5
Missoula	0	1	0	0	0	0	0	0	0	0	2
Idaho:											
Boise	0	0	1	5	0	0	0	0	0	0	3
Colorado:											
Denver	8	8	1	0	0	8	0	0	0	25	68
Pueblo	1	0	0	0	0	0	0	0	0	0	16
New Mexico:											
Albuquerque	0	1	0	1	0	4	0	1	0	3	16
Arizona:											
Phoenix	0	1	0	0	0	7	0	0	0	0	27
Utah:											
Salt Lake City	2	0	1	0	0	2	1	1	0	62	32
Nevada:											
Reno	0	0	0	0	0	0	0	0	0	0	2
PACIFIC											
Washington:											
Seattle	9	12	3	0	-------	-------	1	1	-------	8	-------
Spokane	3	19	3	0	-------	-------	0	0	-------	9	-------
Tacoma	2	3	2	10	0	0	1	0	0	2	25
Oregon:											
Portland	6	34	6	13	0	1	1	0	0	3	57
California:											
Los Angeles	16	37	4	9	0	32	2	2	1	4	214
Sacramento	1	0	0	1	0	2	0	0	0	0	17
San Francisco	12	17	1	0	0	11	1	2	0	7	141

Division, State, and city	Cerebrospinal meningitis		Lethargic encephalitis		Pellagra		Poliomyelitis (infantile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, estimated expectancy	Cases	Deaths
NEW ENGLAND									
Massachusetts:									
Boston	1	0	1	0	0	0	0	0	0
Worcester	0	0	0	0	0	0	0	1	0
MIDDLE ATLANTIC									
New York:									
New York	0	1	12	4	0	0	1	1	1
Rochester	0	0	1	0	0	0	0	0	0

City reports for week ended June 12, 1926—Continued

Division, State, and city	Cerebrospinal meningitis		Lethargic encephalitis		Pellagra		Poliomyelitis (infantile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, estimated expectancy	Cases	Deaths
MIDDLE ATLANTIC—continued									
New Jersey:									
Newark	0	0	1	0	0	0	0	0	0
Pennsylvania:									
Pittsburgh	0	1	0	0	0	0	1	0	0
EAST NORTH CENTRAL									
Ohio:									
Columbus	0	0	0	0	0	1	0	0	0
Indiana:									
Indianapolis	0	1	0	0	0	0	0	0	0
Illinois:									
Chicago	1	1	3	1	0	0	0	0	0
Michigan:									
Detroit	0	1	0	1	0	0	0	0	0
WEST NORTH CENTRAL									
Nebraska:									
Omaha	0	0	1	1	0	0	0	0	0
SOUTH ATLANTIC									
Maryland:									
Baltimore	1	0	0	0	0	0	1	0	0
South Carolina:									
Charleston	0	0	0	0	0	1	0	0	0
Florida:									
Miami	0	0	0	0	1	0	----	0	0
EAST SOUTH CENTRAL									
Alabama:									
Birmingham	0	0	1	0	2	0	1	0	0
WEST SOUTH CENTRAL									
Arkansas:									
Little Rock	1	0	0	0	0	1	0	0	0
Louisiana:									
Shreveport	0	0	0	0	0	1	0	0	0
Texas:									
Dallas	0	0	0	0	2	2	0	0	0
Houston	0	1	0	0	0	1	0	0	0
MOUNTAIN									
Montana: [1]									
Missoula	1	0	0	0	0	0	0	0	0
PACIFIC									
Washington:									
Spokane	3	0	0	0	0	0	0	0	0
Oregon:									
Portland	1	0	0	1	0	0	0	0	0
California:									
Los Angeles	1	0	0	0	0	0	0	0	0
Sacramento	0	0	0	0	2	0	0	0	0

[1] Rocky Mountain spotted fever, 1 case at Helena, Mont.

The following table gives the rates per 100,000 population for 103 cities for the five-week period ending June 12, 1926, compared with those for a like period ended June 13, 1925. The population figures used in computing the rates are approximate estimates as of July 1, 1925 and 1926, respectively, authoritative figures for many of the cities not being available. The 103 cities reporting cases had an

estimated aggregate population of nearly 30,000,000 in 1925 and nearly 30,500,000 in 1926. The 96 cities reporting deaths had more than 29,250,000 estimated population in 1925 and more than 29,750,000 in 1926. 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 9 to June 12, 1926—Annual rates per 100,000 population—Compared with rates for the corresponding period of 1925 [1]

DIPHTHERIA CASE RATES

	Week ended									
	May 16, 1925	May 15, 1926	May 23, 1925	May 22, 1926	May 30, 1925	May 29, 1926	June 6, 1925	June 5, 1926	June 13, 1925	June 12, 1926
103 cities	[2] 158	[3] 121	148	[3] 117	[4] 144	[3] 122	[5] 152	[6] 118	116	[7] 138
New England	149	87	122	78	110	80	125	[8] 79	91	[9] 69
Middle Atlantic	237	135	202	138	210	145	243	134	155	155
East North Central	[10] 102	96	101	117	100	108	92	[11] 120	89	[11] 147
West North Central	205	[3] 199	243	[3] 145	187	[3] 163	183	[3] 207	141	[12] 289
South Atlantic	81	77	83	71	[4] 72	96	[5] 88	[13] 51	54	60
East South Central	32	52	37	36	11	42	11	[14] 17	11	26
West South Central	53	82	40	47	62	65	40	56	66	47
Mountain	148	182	129	127	139	127	74	109	176	127
Pacific	[15] 132	175	157	164	160	159	138	132	157	159

MEASLES CASE RATES

103 cities	[2] 599	[3] 1, 565	579	[3] 1, 434	[4] 569	[3] 1, 283	[5] 549	[6] 1, 001	558	[7] 864
New England	1, 145	1, 198	1, 014	1, 075	836	1, 064	841	[8] 736	860	[9] 662
Middle Atlantic	765	1, 198	615	1, 133	701	956	771	751	724	707
East North Central	[10] 795	1, 371	888	1, 372	839	1, 252	825	[11] 1, 042	779	[11] 988
West North Central	76	[3] 4, 134	233	[3] 3, 437	137	[3] 3, 061	111	[3] 2, 209	131	[12] 1, 552
South Atlantic	311	1, 933	309	1, 659	[4] 242	1, 542	[5] 393	[13] 1, 244	280	1, 103
East South Central	152	3, 461	310	2, 999	200	2, 376	121	[14] 1, 702	194	1, 396
West South Central	13	155	22	142	13	112	22	86	13	125
Mountain	55	1, 393	176	1, 384	240	1, 302	37	1, 247	92	919
Pacific	[15] 170	679	124	693	157	803	157	696	83	593

SCARLET FEVER CASE RATES

103 cities	[2] 338	[3] 326	297	[3] 309	[4] 267	[3] 274	[5] 256	[6] 229	170	[7] 256
New England	345	312	338	288	204	258	256	[8] 251	173	[9] 256
Middle Atlantic	330	249	264	256	270	212	262	209	155	195
East North Central	[10] 368	356	388	341	321	339	293	[11] 246	198	[11] 332
West North Central	705	[3] 870	539	[3] 721	514	[3] 695	466	[3] 416	315	[12] 673
South Atlantic	156	222	138	195	[4] 115	160	[5] 125	[13] 175	58	160
East South Central	299	202	226	176	168	171	116	[14] 94	147	78
West South Central	70	155	44	172	62	116	84	163	44	86
Mountain	342	246	314	173	398	100	324	218	268	118
Pacific	[15] 187	259	155	294	133	181	144	170	155	237

[1] 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, 1925 and 1926, respectively.
[2] Superior, Wis., and Tacoma, Wash., not included.
[3] Grand Forks, N. Dak., not included.
[4] Charleston, W. Va., not included.
[5] Wilmington, N. C., not included.
[6] Concord, N. H., Madison, Wis., Grand Forks, N. Dak., Norfolk, Va., Wilmington, N. C., and Covington, Ky., not included.
[7] Barre, Vt., Madison, Wis., St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[8] Concord, N. H., not included.
[9] Barre, Vt., not included.
[10] Superior, Wis., not included.
[11] Madison, Wis., not included.
[12] St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[13] Norfolk, Va., and Wilmington, N. C., not included.
[14] Covington, Ky., not included.
[15] Tacoma, Wash., not included.

Summary of weekly reports from cities, May 9 to June 12, 1926—Annual rates per 100,000 population—Compared with rates for the corresponding period of 1925 — Continued

SMALLPOX CASE RATES

	Week ended									
	May 16, 1925	May 15, 1926	May 23, 1925	May 22, 1926	May 30, 1925	May 29, 1926	June 6, 1925	June 5, 1926	June 13, 1925	June 12, 1926
103 cities	[2] 44	[3] 26	58	[3] 18	[4] 47	[3] 19	[5] 45	[6] 15	36	[7] 17
New England	0	0	0	0	0	0	0	[8] 0	0	[9] 0
Middle Atlantic	7	0	2	0	2	1	4	0	2	0
East North Central	[10] 53	20	66	18	54	13	61	[11] 9	40	[11] 12
West North Central	76	[3] 36	66	[3] 28	68	44	92	[3] 40	50	[12] 34
South Atlantic	35	39	61	24	[4] 10	28	[5] 37	[13] 34	21	38
East South Central	173	119	404	62	389	62	105	[14] 88	273	52
West South Central	35	116	123	95	53	99	31	43	4	34
Mountain	28	55	28	18	55	36	37	27	28	46
Pacific	[15] 181	67	177	51	160	32	182	24	141	54

TYPHOID FEVER CASE RATES

103 cities	[2] 13	[3] 8	18	[3] 11	[4] 15	[3] 10	[5] 24	[6] 9	27	[7] 12
New England	12	0	24	9	17	7	29	[8] 0	24	[9] 17
Middle Atlantic	10	10	19	7	9	5	26	9	17	6
East North Central	[10] 6	5	5	5	7	9	9	[11] 5	9	[11] 4
West North Central	0	[3] 2	4	[3] 8	10	[3] 4	8	[3] 8	24	[12] 5
South Atlantic	25	4	36	32	[4] 39	26	[5] 39	[13] 34	61	26
East South Central	58	0	68	10	47	31	37	[14] 11	110	57
West South Central	75	43	62	26	62	13	84	9	110	52
Mountain	0	9	18	9	9	0	74	9	46	9
Pacific	[15] 3	8	6	19	8	11	8	8	14	13

INFLUENZA DEATH RATES

96 cities	[15] 14	16	14	15	[4] 12	12	[5] 10	[16] 8	7	[17] 10
New England	7	5	5	12	7	9	2	[8] 2	5	[9] 12
Middle Atlantic	12	17	11	16	9	11	11	6	6	9
East North Central	10	18	11	18	13	11	10	[11] 8	6	[11] 10
West North Central	11	6	17	8	17	13	4	8	8	[19] 3
South Atlantic	10	17	6	11	[4] 12	11	[5] 6	[13] 8	4	6
East South Central	74	31	79	36	37	26	47	[14] 39	16	36
West South Central	19	28	19	24	29	9	5	14	19	19
Mountain	55	18	18	0	0	9	28	18	9	9
Pacific	[15] 12	4	22	4	7	11	11	4	4	0

[2] Superior, Wis., and Tacoma, Wash., not included.
[3] Grand Forks, N. Dak., not included.
[4] Charleston, W. Va., not included.
[5] Wilmington, N. C., not included.
[6] Concord, N. H., Madison, Wis., Grand Forks, N. Dak., Norfolk, Va., Wilmington, N. C., and Covington, Ky., not included.
[7] Barre, Vt., Madison, Wis., St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[8] Concord, N. H., not included.
[9] Barre, Vt., not included.
[10] Superior, Wis., not included.
[11] Madison, Wis., not included.
[12] St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[13] Norfolk, Va., and Wilmington, N. C., not included.
[14] Covington, Ky., not included.
[15] Tacoma, Wash., not included.
[16] Concord, N. H., Madison, Wis., Norfolk, Va., Wilmington, N. C., and Covington, Ky., not included.
[17] Barre, Vt., Madison, Wis., St. Paul, Minn., Kansas City, Mo., and Fargo, N. Dak., not included.
[19] St. Paul, Minn., Kansas City, Mo., and Fargo, N. Dak., not included.

Summary of weekly reports from cities, May 9 to June 12, 1926—Annual rates per 100,000 population—Compared with rates for the corresponding period of 1925 — Continued

PNEUMONIA DEATH RATES

	Week ended									
	May 16, 1925	May 15, 1926	May 23, 1925	May 22, 1926	May 30, 1925	May 29, 1926	June 6, 1925	June 5, 1926	June 13, 1925	June 12, 1926
96 cities	[15] 123	150	123	141	[4] 119	[4] 120	[5] 123	[18] 106	99	[17] 95
New England	129	165	110	144	110	123	69	[8] 117	113	[9] 102
Middle Atlantic	143	165	143	173	145	145	167	130	130	109
East North Central	118	147	116	133	111	106	107	[11] 99	79	[11] 87
West North Central	55	81	76	94	57	83	55	50	57	[19] 48
South Atlantic	129	182	125	148	[4] 147	[4] 111	[5] 138	[20] 83	115	96
East South Central	152	182	126	171	158	171	116	[14] 132	58	125
West South Central	106	137	73	90	73	109	63	99	82	94
Mountain	157	91	166	82	74	91	92	146	102	82
Pacific	[15] 75	92	120	53	73	64	116	67	44	67

[4] Charleston, W. Va., not included.
[5] Wilmington, N. C., not included.
[8] Concord, N. H., not included.
[9] Barre, Vt., not included.
[11] Madison, Wis., not included.
[14] Covington, Ky., not included.
[15] Tacoma, Wash., not included.
[17] Barre, Vt., Madison, Wis., St. Paul, Minn., Kansas City, Mo., and Fargo, N. Dak., not included.
[18] Concord, N. H., Madison, Wis., Norfolk, Va., Charleston, W. Va., Wilmington, N. C., and Covington, Ky., not included.
[19] St. Paul, Minn., Kansas City, Mo., and Fargo, N. Dak., not included.
[20] Norfolk, Va., Charleston, W. Va., and Wilmington, N. C., not included.

Number of cities included in summary of weekly reports, and aggregate population of cities in each group, approximated as of July 1, 1925 and 1926, 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	
			1925	1926	1925	1926
Total	103	96	29, 944, 996	30, 473, 129	29, 251, 658	29, 764, 201
New England	12	12	2, 176, 124	2, 206, 124	2, 176, 124	2, 206, 124
Middle Atlantic	10	10	10, 346, 970	10, 476, 970	10, 346, 970	10, 476, 970
East North Central	16	16	7, 481, 656	7, 655, 436	7, 481, 656	7, 655, 436
West North Central	14	11	2, 594, 962	2, 634, 662	2, 461, 380	2, 499, 036
South Atlantic	21	21	2, 716, 070	2, 776, 070	2, 716, 070	2, 776, 070
East South Central	7	7	993, 103	1, 004, 953	993, 103	1, 004, 953
West South Central	8	6	1, 184, 057	1, 212, 057	1, 078, 198	1, 103, 695
Mountain	9	9	563, 912	572, 773	563, 912	572, 773
Pacific	6	4	1, 888, 142	1, 934, 084	1, 434, 245	1, 469, 144

FOREIGN AND INSULAR

BRAZIL

Yellow fever—Bahia—May 9–22, 1926.—During the two weeks ended May 22, 1926, three cases of yellow fever with two deaths were reported at Bahia, Brazil.

CANADA

Communicable diseases—May 30–June 12, 1926.—The Canadian Ministry of Health reports certain communicable diseases in seven Provinces of Canada for the two weeks from May 30 to June 12, 1926, as follows:

	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	Total
Cerebrospinal fever			2	1				3
Influenza	37				2			39
Poliomyelitis				2				2
Smallpox				24	12	7	3	46
Typhoid fever	1	2	16	15	2	2	4	42

Vital statistics—Quebec—February and March, 1926.—Births and deaths in the Province of Quebec for the months of February and March, 1926, have been reported as follows:

	February	March
Estimated population	2,570,000	2,570,000
Births	6,167	7,526
Birth rate per 1,000 population	28.79	35.14
Deaths (all causes)	2,813	3,559
Death rate per 1,000 population	13.13	16.61
Deaths under 1 year	887	1,113
Infant-mortality rate	143.8	147.9
Deaths from—		
Cancer	115	130
Cerebrospinal meningitis	19	11
Diabetes	9	21
Diphtheria	24	26
Heart diseases	323	431
Influenza	135	198
Measles	19	34
Poliomyelitis (infantile paralysis)	1	0
Scarlet fever	9	14
Syphilis	5	7
Tuberculosis (pulmonary)	191	236
Tuberculosis (other forms)		93
Typhoid fever	15	20
Whooping cough	31	38

CHINA

Plague—Amoy—May 1, 1926.—Under date of May 1, 1926, plague was reported prevalent in the city of Amoy, China.

Smallpox—South Manchuria Railway—May 16–22, 1926.—During the week ended May 22, 1926, 18 cases of smallpox were reported at 10 localities on the line of the South Manchuria Railway.

EGYPT

Plague—May 21–27, 1926—Summary.—During the week ended May 27, 1926, 4 cases of plague were reported in Egypt, the urban occurrence being 2 cases at Suez. From January 1 to May 27, 1926, a total of 43 cases was reported in Egypt, as compared with 53 cases occurring during the corresponding period of the preceding year.

Later occurrence.—Later reports show the occurrence of plague in Egypt as follows: Suez, May 28 to 30, 4 cases with 3 deaths (bubonic and pneumonic); Provinces of Beni-Suef, May 28 to June 3, 5 cases with 2 deaths; and Gharbieh, June 2, 1 case with 1 death (bubonic).

GREECE

Plague—Zante—May 17, 1926.—A press report has been received from Patras, Greece, under date of May 23, 1926, showing the occurrence of a case of plague in the island of Zante, six hours distant from Patras, May 17, 1926.

IRELAND (IRISH FREE STATE)

Typhus fever—Cork County.—A case of typhus fever was reported in the urban district, Cork County, Irish Free State, June 5, 1926.

MALTA

Communicable diseases—April, 1926.—During the month of April, 1926, communicable diseases were reported in the island of Malta as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Broncho-pneumonia	5		Pneumonia	7	
Chicken pox	64		Poliomyelitis	1	
Diphtheria	7	1	Scarlet fever	3	
Erysipelas	6		Trachoma	34	
Influenza	12		Tuberculosis	23	17
Lethargic encephalitis	1		Typhoid fever	15	2
Malta (undulant) fever	48		Whooping cough	20	
Measles	129	1			

Population, estimated, civil, December 31, 1925: 255,242.

MEXICO

Malaria—Tampico—June 1-10, 1926.—During the 10 days ended June 10, 1926, 8 cases of malaria with 3 deaths were reported at Tampico, Mexico.

PERU

Gastroenteritis—Lima—March, 1926.—During the month of March, 1926, 75 deaths from gastroenteritis were reported at Lima, Peru. Population, estimated, 200,000.

SPAIN

Mortality—Madrid—April, 1926.—During the month of April, 1926, 1,333 deaths were reported at Madrid, Spain, as compared with 1,628 deaths in January, 1,248 in February, and 1,584 in March, 1926. Population, estimated, 766,552.

Mortality in children.—Of the 1,333 deaths reported in April, 1926, at Madrid, 177 occurred in children under one year of age and 262 in the period one year to four years.

Principal causes of death.—The principal causes of death noted were heart disease with 98 deaths; tuberculosis (pulmonary), 147; pneumonia, 25; bronchitis, 109; other diseases of the respiratory organs, 203. There were 65 deaths from scarlet fever, including 4 deaths from scarlatina and 5 deaths from typhoid fever.

VIRGIN ISLANDS

Communicable diseases—May, 1926.—During the month of May, 1926, communicable diseases were reported in the Virgin Islands of the United States as follows:

Island and disease	Cases	Remarks
St. Thomas and St. John:		
Chancroid	3	
Gonorrhea	7	
Syphilis	3	
Tetanus	1	
Tuberculosis	1	Chronic pulmonary.
St. Croix:		
Chancroid	4	
Filariasis	3	Bancrofti.
Gonorrhea	1	
Leprosy	1	

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

The reports contained in the following tables must not be considered as complete or final as regards either the lists of countries included or the figures for the particular countries for which reports are given.

Reports Received During Week ended July 2, 1926 [1]

CHOLERA

Place	Date	Cases	Deaths	Remarks
India:				
Calcutta	Apr. 4–May 8	308	276	
Indo-China:				
Saigon	May 2–8	20	18	
Siam:				
Bangkok	do	255	143	

PLAGUE

China:				
Amoy	Apr. 18–May 1, 1926.			Prevalent.
Nanking	May 9–22			Do.
Egypt				May 21–27, 1926: Cases, 4. Jan. 1–May 27, 1926: Cases, 43. Corresponding period, 1925: Cases, 53.
City—				
Suez	May 21–27	2		
Do	May 28–30	4	3	Bubonic, 1 case, 2 deaths; 1 case, 1 death, pneumonic.
Province—				
Beni-suef	May 28–June 3	5	2	Bubonic and septicemic.
Gharbieh	June 2	1	1	Bubonic.
Greece:				
Athens	Apr. 1–30	7	2	Including Piraeus.
Do	May 1–31	9	2	Do.
Zante	May 17	1		
India:				
Bombay	May 2–8	1	1	
Iraq:				
Bagdad	Apr. 18–May 15	83	56	
Java:				
Batavia	Apr. 24–May 7	21	21	
Cheribon	Apr. 11–24	3	3	

SMALLPOX

Algeria:				
Algiers	May 21–31	4		
Brazil:				
Para	May 16–29	6	7	
Rio de Janeiro	May 2–15	45	11	
Santos	Mar. 1–7		1	
Canada				May 30–June 12, 1926: Cases, 46.
Alberta	May 29–June 12	3		
Manitoba	do	12		
Winnipeg	June 6–12	5	1	
Ontario				May 30–June 12, 1926: Cases, 24.
Kingston	May 23–29	3		
North Bay	May 2–22	5		
Saskatchewan				May 30–June 12, 1926: Cases, 7.
China:				
Chungking	May 2–15			Present.
Foochow	May 9–22			Do.
Hongkong	May 2–15	4	3	
Manchuria—				
An-shan	May 16–22	1		South Manchuria Ry.
Antung	do	2		Do.
Changchun	do	2		Do.
Fushun	do	3		Do.
Kai-yuan	do	1		Do.
Liao-yang	do	2		Do.
Mukden	do	1		Do.
Penhsihu	do	2		Do.
Teshihchiao	do	1		Do.
Wa-feng-tien	do	3		Do.

[1] From medical officers of the Public Health Service, American consuls, and other sources. For reports received from Dec. 26, 1925, to June 25, 1926, see Public Health Reports for June 25, 1926. The tables of epidemic diseases are terminated semiannually and new tables begun.

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER—Continued

Reports Received During Week Ended July 2, 1926—Continued

SMALLPOX—Continued

Place	Date	Cases	Deaths	Remarks
China—Continued				
Nanking	May 8–22			Present.
Shanghai	May 2–15	2	5	Cases, foreign. Deaths, population of international concession, foreign and native.
Swatow	May 9–15			Sporadic.
Great Britain:				
England—				
Bradford	May 23–29	1		
India:				
Bombay	May 2–8	24	12	
Calcutta	Apr. 4–May 8	133	128	
Iraq:				
Bagdad	May 9–15	1		
Basra	Apr. 18–May 8	9	4	
Japan:				
Nagoya	May 16–22		1	
Taiwan Island	May 11–20	24		
Yokohama	May 2–8	2		
Java:				
East Java and Madoera	Apr. 11–17	4		
Malang	Apr. 4–10	6	1	Interior.
Mexico:				
Guadalajara	June 8–14		2	
Mexico City	May 16–22	2		Including municipalities in Federal District.
Tampico	June 1–10		2	Varioloid.
Torreon	May 1–31		10	
Poland				Apr. 4–10, 1926. Cases, 7.
Portugal:				
Oporto	May 23–29	3		
Siam:				
Bangkok	May 2–8	1	4	
Union of South Africa:				
Transvaal—				
Johannesburg	May 9–15	1		

TYPHUS FEVER

Algeria:				
Algiers	May 21–31	2	1	
Chile:				
Antofagasta	May 23–29	3		
Ireland (Irish Free State):				
Cork	June 5	1		
Mexico:				
Mexico City	May 16–22	9		Including municipalities in Federal District.
Palestine				March, 1926: Cases, 6. Exclusive of Bedouin tribes and the British military forces.
Peru:				
Arequipa	Jan. 1–31		2	Out of date.
Poland				Mar. 28–Apr. 10, 1926: Cases, 191; deaths, 18.

YELLOW FEVER

Brazil:				
Bahia	May 9–22	3	2	