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HEREDITARY TRANSMISSION OF TULARÆMIA INFECTION BY THE WOOD TICK, *DERMACENTOR ANDERSONI* STILES [1]

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In May 1924, Parker, Spencer, and Francis [2] submitted data demonstrating the stage to stage transmission of *Bacterium tularense* from larva to adult of the wood tick *Dermacentor andersoni* Stiles. New data, here presented, demonstrate the hereditary transmission of this bacterium from infected female ticks to their progeny.

The ticks used in these experiments were part of a lot of laboratory-reared adult ticks, all of them the progeny of one female and free from any demonstrable infection as shown by tests of the antecedent larvae and nymphs and the apparent absence of disease in the hosts upon which they were engorged. The strain of tularæmia employed was the same as that used in the previous studies [2] and had been carried in part in ticks and in part by guinea pig transfers since it was secured in May, 1923, from wild adult ticks. The experimental studies were made at the Rocky Mountain Spotted Fever and Tularæmia Laboratory of the United States Public Health Service at Hamilton, Mont.

Feeding and infection of parent females.—On May 22, 1924, two Belgian rabbits were each infested with 23 (some male and some female) ticks confined in a brass gauze capsule secured in place by adhesive plaster. The rabbits were immediately inoculated by the cutaneous method with spleen tissue of a guinea pig just dead of tularæmia. Both hosts were dead of typical tularæmia on May 27, and four engorged female ticks were recovered and put aside. The remaining ticks were at once transferred to two normal rabbits, from which they were removed on June 2, and 11 more engorged females were secured, making a total of 15. The latter two host rabbits died June 13 and 14, respectively, with lesions suggestive of subacute tularæmia. Each of the fed female ticks deposited fertile eggs.

Feeding of resultant larvae, nymphs, and adults.—The 15 lots of larvae were fed, each on a separate Belgian rabbit, at various dates between August 8 and September 15. Five lots failed to engorge;

[1] The experimental studies upon which this paper is based were made in cooperation with the Montana State Board of Entomology.

[2] Tularæmia. XI. Tularæmia Infection in Ticks of the species *Dermacentor andersoni* Stiles in the Bitterroot Valley, Montana. Pub. Health Rep., Vol. 39, No. 19, May 9, 1924, pp. 1057-1073.

10 were carried through to the nymphal stage. Ten nymphs of each of these 10 lots were placed on separate guinea pigs, one group being attached October 15, the others December 16; the nymphs of two of the latter lots did not feed. Only three lots of the remainder of the unfed nymphs survived the winter. These were engorged on Belgian rabbits beginning March 26, 1925, and the resultant adults were fed in lots of 30 each on 8 Belgian rabbits, beginning December 8, 1925.

Tests for infection in eggs, larvae, nymphs, and adults.—Each lot of eggs was tested for infection by injecting approximately 100 eggs, emulsified in salt solution, subcutaneously into a guinea pig. The interval between the beginning of egg deposition and the test varied from 3 to 37 days for the different lots. Infection in larvae and nymphs was tested in two ways—first by the effect on the host upon which they fed, and, second, by the subcutaneous injection of the fed or partly fed ticks into guinea pigs. The adults were tested only by permitting them to feed on susceptible animals.

Absence of positive evidence in host animals or in the guinea pigs into which test material was injected was not taken as final proof of the absence of infection. Had this been done only one lot of ticks would have shown positive evidence beyond the egg stage. If the spleen was at all enlarged, transfers from the initial test animal were made whenever possible, usually by the subcutaneous injection of spleen tissue emulsified in salt solution. Such transfers were essential since experience has shown that in some ticks infection is of such marked virulence that the test animal will die before lesions become typical, while in others it is so attenuated that chronic, subacute, or inapparent infections are produced. By repeated transfers these are usually changed into strains producing typical, acute, recognizable infections, although this procedure may fail completely even with material from a known infected source. Numerous desirable transfers were omitted because of enforced absences from the station. A final positive diagnosis was not made unless the necropsy findings were unmistakably typical of acute tularæmia infection, i. e., spleen enlarged, both spleen and liver studded with many small, grayish-white necrotic foci, and glands characteristically caseous.

RESULTS

Tularæmia infection was demonstrated, by animal tests, in the eggs, larvae, or nymphs of 8 of the 15 parent females. In two lines of descent it was recovered from the eggs only; in one line from eggs and larvae; in two lines from eggs and nymphs; in one line from larvae and nymphs, in two lines from the nymphs only; and in none was it recovered from the adults.

Of the seven lines of descent in which infection was not demonstrated, it is not known whether it was acquired by the parent females. Because of failure of the ticks to feed, only 2 of these 7 lots of progeny were tested beyond the egg stage and only 1 of them was tested as nymphs.

Five egg tests were positive, four of them resulting in typical lesions in the guinea pigs injected. For the fifth, one transfer was necessary. No positive tests were secured from lots of eggs tested more than ten days after egg deposition began.

Only two definitely positive tests were secured from larvae.

Nymphal infection was demonstrated in the nymphs from 5 females; 1 test being made 166 days (October 15, 1924) the others 208 days (December 16, 1924) after the beginning of parent female engorgment. The intervals following larval feeding were, respectively 65, 92, 98, 115, and 117 days.

The larvae and nymphs of one female caused typical lesions in their respective hosts, and death resulted. In all other positive larval and nymphal tests one or more transfers were necessary either from the host animal or from the guinea pig into which the partly fed or engorged ticks were inoculated

The eight groups of adult ticks fed December 8, 1925, caused no lesions typical of tularæmia in their respective hosts. The latter were killed and autopsied 15 days after tick attachment. Enlargement of the spleen was the only gross pathology observed. Owing to circumstances beyond our control, tissue transfers were not made, and fed ticks were not tested further by inoculation into guinea pigs as was done in earlier tests of the larvae and nymphs. These eight groups of adults represented three of the four lots of nymphs shown infective by tests made December 16, 1924. The adult tick tests were made 257 days after the beginning of nymphal feeding (March 26, 1925), 357 days after the positive nymphal tests of December 16, 1924, and 565 days after parent female attachment (May 22, 1924).

Recovery of pure cultures from progeny of parent females.—The use of a repeatedly proved strain of tularæmia, the fact that the parent females were free from any demonstrable infection before their infective feed, and the typically specific character of the necropsy findings were strong evidence of the identity of the infection recovered from the eggs, larvae, and nymphs. Complete confirmation, however, was supplied by the recovery, by Francis, of pure cultures of *Bacterium tularense* from the spleen tissue of one of the guinea pigs injected with eggs and from the spleen of another that was injected with engorged nymphs descended from a different female. The spleens from which these cultures were recovered had been placed in pure neutral glycerine, immediately following necropsy, and forwarded to the Hygienic Laboratory at Washington, D. C.

DISCUSSION

The results of the experiments recorded above demonstrate the hereditary transmission of *Bacterium tularense* in the intermediate host, *Dermacentor andersoni*. Eight of 15 female ticks engorged on infected hosts transmitted infection to their progeny. In two of the eight positive lines of descent, however, infection was recovered only from the eggs and was not demonstrated in the resultant larvae or nymphs.[3] In the other six (in only three of which was egg infection demonstrated) it was recovered from the larvae or nymphs or both. In no one line of descent was it recovered from all three of these stages. Four of the five lots of eggs that gave positive tests caused typical acute infections in the egg-injected guinea pigs. Only one lot of larvae and but one of nymphs, however, caused typical acute death of their hosts. Both were from the same parent. In all other positive tests, tissue transfers from the host animals or from the egg-injected or tick-injected guinea pigs (which were apparently well when killed and autopsied) were necessary in order that the presence or apparent absence of infection might be established.

The results, as stated, suggest: (1) That not all infected females transmit infection to their progeny; (2) that the virulence of infection so transmitted may vary in the progeny of different females, even though all were infected from a common source; and (3) that in some lines of descent the infection may die out. The same apparent tendencies have been noted in the much more extensive studies of the virus of Rocky Mountain spotted fever in the same tick species.

The fact that in each line of descent not all stages were shown infective and that many of the positive tests were apparent only after one or more transfers from the initial test guinea pigs indicates the inadvisability of placing undue reliance on apparently negative tests.

The apparent failure to recover infection from the three lots of adults shown to have been infective as nymphs does not necessarily indicate absence of the infectious agent; but even if the agent were absent, the finding would not detract from the significance of the positive results secured with eggs, larvae, and nymphs. The data justify the conclusion that hereditary transmission occurs, but do not warrant any deduction as to the percentage of infected females that thus transmit the bacterium or as to how far it will persist in the subsequent stages. These points doubtless are determined by the conditions attendant upon any particular test.

The hereditary transmission of *Bacterium tularense* by *Dermacentor andersoni* is of interest for two reasons: (1) Hereditary transmission

[3] Prior to recorded experiments infection in the eggs had been demonstrated in lots from several females which had been infected as larvae. The tests were not carried further.

of the bacterium assures a greater number of infected ticks in nature than if stage to stage transmission were confined within the limits of a single generation, and it therefore becomes significant as a definite aid in the natural maintenance of infection; (2) although the hereditary transmission of certain protozoa, of some species of rickettsiae and of symbiotic microorganisms by insects or arachnids is a recognized phenomenon, this paper is believed to be the first record of similar transmission of a known bacterium.

THE SUSCEPTIBILITY OF THE COYOTE (*Canis lestes*) TO TULARÆMIA [1]

By R. R. PARKER, Special Expert, United States Public Health Service, and EDWARD FRANCIS, Surgeon, United States Public Health Service

In June, 1925, Dr. J. H. Garberson, of Miles City, Mont., reported a case of tularæmia, apparently caused by the bite of a coyote (*Canis lestes*). The patient, D. S., was bitten on the hand, June 13, by one of five coyote pups which he was removing from their den, close to the Musselshell River, about 2 miles southeast of Melstone. Typical onset of tularæmia occurred June 15. A persistent ulcer at the site of the coyote bite and enlarged axillary glands characterized this illness. The patient gave no history of tick bite or of having handled rabbits or of any other probable source of infection other than the coyote bite. Blood serum collected from the patient June 27, 1925, and forwarded to the Hygienic Laboratory of the Public Health Service at Washington, D. C., was tested by Francis, and found to agglutinate *Bacterium tularense* in all dilutions from 1:10 to 1:640, thus confirming the diagnosis of tularæmia. Numerous rabbit bones were noted near the den, and rabbits seen in the vicinity were sluggish. One of the coyote pups died four days after capture, but the patient did not know whether or not this was the one that had bitten him. The remainder of the litter were killed two weeks later.

Because the evidence so strongly favored the coyote as the infecting agency, the question arose whether the bite was infective because of acute tularæmia infection in the coyote itself or because the latter had eaten an infected wild rabbit or other infectious material shortly before biting, the resulting transfer of infection having been purely mechanical. The desirability of testing the susceptibility of coyotes was thus suggested. This was made possible by the cooperation of Doctor Garberson, through whom three coyote pups were secured. The experiments were carried on at the Rocky Mountain Spotted Fever and Tularæmia Laboratory of the Public Health Service at Hamilton, Mont.

[1] The work upon which this paper is based was performed in cooperation with the Montana State Board of Entomology.

The tests were made by feeding the coyotes the flesh of Belgian rabbits and guinea pigs just dead of acute tularæmia. This method of giving infectious material was used because it simulated the most likely natural means by which coyotes might acquire the infection. Rodents, especially rabbits, are an important item in coyote diet, and tularæmia was known to be prevalent among rabbits in the locality in which this case occurred. All three coyotes died of acute infection, the specific nature of which was indicated by agglutination tests made before and after the first infective feed, by the occurrence of typical infection in guinea pigs inoculated with tissue obtained at autopsy, and by the recovery from these guinea pigs of pure cultures of *Bacterium tularense*. The agglutination and cultural tests were made by Francis, to whom the necessary sera and tissues were forwarded.

At the times the tests were begun the coyotes (A, B, and C) were approximately four and one-half, five, and six months old, respectively.

COYOTE A

Blood serum secured from coyote A on July 21 failed in all dilutions to agglutinate *Bacterium tularense*.

Between July 29 and August 21 this coyote was fed the spleens and livers of eight Belgian rabbits and five guinea pigs and the entire carcasses of two other guinea pigs. All of these animals had died of acute tularæmia.

Serum drawn August 6 did not agglutinate *Bacterium tularense*. On August 17 it agglutinated in all dilutions up to 1 : 40; on August 27, in all dilutions up to 1:80; and on September 4, in dilutions of 1:10 and 1 : 20.

Death occurred September 20, 53 days after the first infective feed. The carcass was packed in ice and autopsy was made September 21. Parts of the following tissues were emulsified in salt solution and guinea pigs were injected subcutaneously with each, respectively: Salivary glands, axillary glands, inguinal glands, spleen, and lungs. The guinea pig injected with salivary-gland emulsion died September 28; the pigs injected with axillary-gland and spleen emulsions died September 30. Necropsy findings in all three were typical of acute tularæmia. The remaining tests were negative. The infection in the guinea pig injected with salivary-gland emulsion remained typical throughout a series of seven guinea-pig transfers, from one of which a pure culture of *Bacterium tularense* was isolated.

COYOTE B

Serum drawn September 8 did not agglutinate *Bacterium tularense*.

Coyote B was fed the carcasses of four Belgian rabbits dead of typical tularæmia, the carcasses of two being fed on September 9, and of one each on September 11 and 14.

On September 18, nine days after the first infective feed, the serum of this coyote agglutinated *Bacterium tularense* in dilutions up to 1 : 320 and partially at 1 : 640; on September 28 agglutination was positive in all dilutions up to 1 : 320.

Coyote B died October 1, 22 days after this first infective feed. Autopsy was performed immediately after death, but no lesions typical of tularæmia were found. Parts of the following tissues were emulsified in salt solution and guinea pigs were inoculated subcutaneously, with each emulsion, respectively: Salivary glands, axillary glands, inguinal glands, spleen, and liver. The guinea pig inoculated with salivary-gland emulsion died typically on October 10. The lesions remained typical through a series of six guinea-pig transfers, from one of which a pure culture of *Bacterium tularense* was recovered. The other tests were negative.

COYOTE C

Serum of coyote C taken September 4 and again October 13 failed in all dilutions to agglutinate *Bacterium tularense*.

Coyote C was fed one Belgian rabbit on October 18 and two guinea pigs on October 20 and again on October 22, all having just died of typical tularæmia.

On October 23 the blood serum failed to agglutinate *Bacterium tularense* in all dilutions; on October 30 it agglutinated in dilutions of 1 : 10, 20, 40, and 80.

The coyote was killed when dying, October 31, 13 days after the first infective feed. No characteristic lesions of tularæmia were found at autopsy. The left prescapular gland showed a condition at one extremity suggestive of beginning abscess formation. Parts of the following tissues were emulsified in salt solution and two groups of guinea pigs were inoculated with each emulsion, one group subcutaneously, the other intraperitoneally: Salivary glands, prescapular glands, axillary and retroscapular glands, inguinal glands, spleen, and liver. Two guinea pigs were also inoculated with heart blood. Only the two inoculated with the emulsion of the prescapular glands showed evidence of infection. Both died typically, one November 5 the other November 8. The lesions remained characteristic in a series of four guinea-pig transfers, from one of which a pure culture of *Bacterium tularense* was secured.

SUMMARY AND DISCUSSION

Three young coyotes, 4 to 6 months of age, were infected with acute tularæmia by feeding them the tissues of Belgian rabbits and guinea pigs just dead of typical infections. Death occurred respectively, 53, 22, and 13 days after the first feed of infectious material. The course of infection in each case was attended by loss of appetite,

and by an increasing weakness and emaciation. At autopsy the tissues showed no gross pathological lesions such as are characteristic of acute tularæmia in laboratory animals. Diagnosis was made by the following findings: (*a*) The coyote sera did not show specific agglutinating properties for *Bacterium tularense* before the first feed of infectious material, but did give specific reactions beginning the second week thereafter; (*b*) typical acute tularæmia infection was produced in guinea pigs injected with emulsions of autopsy material from each coyote; and (*c*) pure cultures of *Bacterium tularense* were isolated from the tissues of guinea pigs in which the infection from the coyotes was propagated.

The recovery of typical tularæmia from the salivary glands of two of the experimental coyotes indicates the possibility of human infection by the bite of an infected coyote. Besides the case noted as evidently due to this cause, another case reported by Dr. C. T. Pigot, of Roundup, Mont., also in 1925, was apparently infected by the bite of a ground squirrel (*Citellus richardsoni*).

These data are of especial interest as indicating a hitherto unsuspected avenue for the transfer of tularæmia infection from its normal hosts to man; namely, by the bite of a wild rodent or carnivore.

The susceptibility of coyotes and the evident possibility of fatal infection suggest that tularæmia may be a factor, at least, in the diminution of the abundance of coyotes which is so frequently noted following the decimation of rabbit populations in the same localities.

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

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III. PREVIOUS STUDIES OF CHRONIC BENZOL POISONING

Whereas in acute benzol poisoning, the history, the onset, and the course of symptoms lead readily to a diagnosis; in chronic benzol poisoning we find a wholly different picture. The onset is insidious, the early symptoms are generally overlooked, and it is not until the condition becomes relatively grave that it receives medical attention. In 1897, Santesson (45) reported his now famous cases of chronic benzene poisoning. In the tire department of a rubber factory in Upsala, nine young women using benzene cement were taken ill with rather odd symptoms of headache, dizziness, weakness, and hemorrhages of the mucous membranes. Purpuric spots on the skin, bleeding from the nose and gums, vomiting of blood, and bleeding from the vagina were among the more startling features. Various

degrees of anemia were present with the red cell count markedly depressed, in one case down to 600,000 per cubic millimeter, and with the hemoglobin reduced to 20 per cent. Of these cases, four proved fatal within one to four months from the beginning of exposure. Shortly after Santesson's report, Lenoir and Claude (46) reported a fatal case of benzol poisoning in a workman who had been employed for several years in a dyehouse where benzene was used. In due time, weakness and loss in weight resulted, then the appearance of bleeding from the nose and gums. Later purpuric spots appeared on the skin and the general condition progressively became more grave until the man suddenly died. On autopsy, hemorrhages in the gastric mucosa, in the intestines, and under the endocardium were discovered. Myocardial infarcts were also found. The recognition of the symptoms as a distinct entity due to the effects of benzene, led Oliver (47) to stress the benzene hazard, to outline the general symptomatology of the disease, and to recommend hygienic instructions as to its prevention in industry.

Selling (48), in 1910, was the first to report cases of chronic benzol poisoning in the United States. Of the three cases recorded, two terminated fatally. These cases occurred among the workers in the coating room of a tin-can plant in Baltimore, Md. Fourteen young girls were employed in this workroom. Their duties consisted in operating the coating machines, and in handling the tin can ends after a thin layer of rubber dissolved in benzol had been applied to the margin of the can end. In this process, the ends, after emerging from the machine, are still warm, and under such conditions the benzol continues to evaporate into the room air. The concentration of benzol in the air was apparently sufficient to produce fatal poisoning in this case. In Selling's report he states that 10 gallons of benzol were used each day, and that the room was provided with excellent natural ventilation, the windows being kept wide open. These facts are interesting in that they suggest the subtle influence of benzol, despite precautions that may be taken to obviate this hazard. The essential features of Selling's cases are as follows:

Case 1.—White female, age 14. Doctor Girdwood was treating her mother for pneumonia and his attention was called to the girl by her pallor and the presence of purpuric spots. She was feeling well, but shortly after had hemorrhages from the nose and mouth and was advised to enter the hospital. She dated the onset of her illness at about a month before admission, when she noticed blue spots on her arms and legs. Shortly after these had appeared, she suffered bleeding from the nose, gums, and throat. For the week prior to admission, she had been confined to bed because of weakness and dizziness. No joint pains or gastrointestinal symptoms were present. On examination the patient showed pallor, pale membranes, bluish macules 1–3 mm. scattered over arms, legs, and trunk, and bleeding from the gums. Ophthalmoscopic examination showed moderate neuro-retinal edema, with pale fundi and great numbers of small hemorrhages. The blood count on admission showed red cells 1,090,000 per c. mm. and white blood cells

1,280 per c. mm., with 28 per cent of hemoglobin (Sahli). The red cells were smaller than usual and pale and moderate anisocytosis (variation in size of cells) was present. Marked leucopenia (deficiency in white blood cells) with a predominance of mononuclear forms was found. The platelets were much reduced. The girl died eight days after entry. The autopsy revealed muscles of a deep red color; blood pale and watery. The heart muscle and, to a less extent, the liver, showed fatty degeneration. The anatomical diagnosis was purpura hemorrhagica (probably toxic); hemorrhages into the skin, viscera, and serous surfaces. Pallor of the organs.

Case 2.—White female, age 14 years. She complained of spots on body and pain in the side. The general history and physical signs were almost identical with those of Case 1. She had been working five months. The blood showed 2,100,000 red cells and 560 white cells per c.mm., with a preponderance of mononuclears, and hemoglobin 37 per cent. The case proved fatal. The anatomical diagnosis on autopsy was purpura hemorrhagica; hemorrhages in the skin, viscera, and serous surfaces; pallor of the organs; hyperplasia of the bone marrow; acute pleurisy.

Case 3.—Female, age 14, working for three months in the coating room. Presented symptoms of anorexia, abdominal pain, vomiting and headache, with fainting spells. Red cells, 4,900,000; W. B. C., 4,400 per c.mm.; Hb., 54 per cent. This patient recovered and was discharged after six days. An examination of the other workers in the coating room revealed four more who had purpuric spots, but they were entirely free from other symptoms.

Selling (48) stresses the fact that benzene is a powerful leucotoxin, destroying the white cells of the blood and attacking the entire hematopoietic or blood-forming system in general. In 1911 two similar cases (49) were reported of chronic benzene poisoning characterized by purpura hemorrhagica and anemia, and occurring in a rubber works. The same year Glaser (50) reported on an investigation into several cases of benzene-vapor poisoning occurring in a can factory. Fourteen girls were examined and all were found to be anemic. Four had to be removed to a hospital, where one died and one was critically ill. In these cases the presence of anilin and nitrobenzol complicated the picture and was held responsible for the sickness. In 1916 McClure (51) emphasized the value of treatment by repeated transfusions and reported a case occurring in the same can factory as that in which the cases reported by Selling occurred. A woman 31 years of age complained of bleeding from the nose and mouth, the appearance of black and blue spots on her body, and also weakness and anorexia. Extreme breathlessness, with anemia, developed later. The red cell count was 1,460,000 and the white cells 1,110 per c. mm.; hemoglobin, 25 per cent. Bleeding time was 14½ minutes. Thirteen successive transfusions were given, and although the first three or four did not produce any definite improvement, the persistent administration of blood led finally to her uneventful recovery and discharge. In his paper McClure also advocates splenectomy in some of the cases of persistent anemia. Numerous other cases of chronic benzene poisoning in can factories are on record. Hogan and

Shrader (52) report three cases of chronic poisoning occurring in young women and resulting in two fatalities. As in the previous cases, the workers were engaged in tending the machines in which rubber cement was sprayed around the margin of the can ends. The evaporation of the benzol in the poorly ventilated room was again the cause of the fatalities. The symptoms and signs presented were almost identical with Selling's cases. They are briefly presented below:

Case 1.—Young girl of 17 years. Had been working six weeks in the "dope room" inspecting the tops and bottoms of cans. On March 4 she felt weak and had no appetite. Later she developed headache and noticed blue spots on her arms and legs. On March 14 she experienced difficulty in swallowing food, had a sore mouth, and also spat up blood. Examination showed undernourishment, anemia, purpuric spots over arms and legs, stomatitis, and bleeding from the vagina. Marked anemia was present. R. B. C., 1,240,000; W. B. C., 600 per c. mm.; Hb. 39. Anisocytosis and poikolocytosis were present. She was transfused shortly after admission to the hospital, but grew weaker and died.

Case 2.—A woman of 25, admitted with a diagnosis of placenta previa. An attempt was made to deliver her and a macerated fetus was obtained. Her history is not clear. Blood examination showed R. B. C., 470,000 per c. mm.; W. B. C., 538 per c. mm.; Hb. 19 per cent.

Case 3.—Young girl of 15, employed in a can factory, working on can tops. Three days after starting work she had a nosebleed, and a week later hemorrhages from the mucous membranes, loss of appetite, and later persistent bleeding from the vagina. Nausea, vomiting, poor sleep, and extreme fatigue were also present. She was admitted to the hospital April 9. On April 12 her blood count showed 880,000 red cells and 950 white cells per c. mm., with a preponderance of mononuclear forms. Hb., 16 per cent. She was given three transfusions. Immediately favorable response resulted and six days later her blood count showed 2,184,000 red cells and 3,050 white cells per c. mm., with 44 per cent hemoglobin. The total mononuclear count had decreased from 60 per cent to 48 per cent, and the polynuclear elements had increased from 38 per cent to 50 per cent.

These cases emphasize the marked susceptibility of young girls to chronic benzene poisoning and also indicate the rapid onset of grave symptoms. Dr. Alice Hamilton (53) cites, from a personal communication, the case of a woman, aged 41, working in a can factory where cans to which covers had been cemented on with a benzol paste were being delivered. Despite downward suction ventilation and a fresh air supply from above, this patient developed headache, nausea, bleeding from the nose and gums, with a rash and purpura over the lower extremities. Her blood count showed 2,512,000 red cells and 2,000 white cells per c. mm. Hemoglobin, 35 per cent. The case proved fatal, although two blood transfusions were given. Legge (54), in 1918, reported the first known cases of chronic benzol poisoning occurring in Great Britain. Two men were employed in spreading rubber cement on balloon fabric. In September, 1917, the men were transferred to a new spreading room where 20 spreading machines had been installed, but of which only three were in use,

two of them being operated by the men whose cases are under discussion. The composition used was a mixture of rubber dissolved in pure benzol. After the lapse of approximately three months there appeared symptoms of malaise and anemia, with subcutaneous and submucous hemorrhages, bleeding from the nose, gums, and bowels. The first man stopped work on December 11, 1917, and died December 18, 1917, the second stopped work on December 13, 1917, and at the time of Legge's report he was still seriously ill in hospital. An autopsy on the man who died showed submucous hemorrhages in the intestinal tract and under the endothelium of the heart. The bone marrow exhibited the characteristic changes of an aplastic anemia. About six months later the remaining worker, who was employed on the third machine, became ill with typical symptoms of chronic benzene poisoning and died. The workroom in which these men had been employed was a partitioned area of a larger spreading room. Its dimensions were 12 feet long by 12 feet wide and 11 feet high. It had four windows on one side and two on the other. None of the workers in the main spreading room showed any signs of poisoning. Analysis of the air in the partitioned workroom showed benzol concentrations of 210 to 1,050 parts per million by volume, with an average of 550 parts. Doctor Dale, of the Medical Research Committee, advocated the substitution of xylol in place of benzol as the solvent, believing it to be considerably less toxic. Returning to America, we find Harrington (34), in 1917, reporting five cases of chronic poisoning, with three deaths, in the tire-building department of a rubber establishment. Preparatory to vulcanizing, the tires were wiped with a cloth which was moistened with benzol by means of a can so arranged as to deliver a specified amount of benzol on the cloth. This process of moistening the cloth occurred about eighteen times a day. The cases are briefly recorded below:

Case 1.—Fatal. Male 33 years old. Had been in the tire-building department for about a year when his gums started to bleed almost daily. Shortly after this, bluish spots appeared on his body and extremities. Nosebleed, extreme weakness, and dyspnea soon appeared and he entered the hospital about five weeks after the onset of symptoms. Examination revealed spongy, bleeding gums, pale membranes, slight tremor of head, ecchymotic spots on skin. Vertigo and visual disturbances were also evident. R. B. C., 2,288,000; W. B. C., 5,000; Hb., 60 per cent. A blood transfusion was given but the symptoms grew progressively worse; delirium and convulsions then appeared, followed shortly by death.

Case 2.—Fatal. Male, 40 years old. Had been working in tire-building department for 11 or 12 months. Several months before entering the hospital he became aware of increasing weakness, fatigue, and dyspnea. He began to lose weight, had frequent headaches, and was unable to work continuously. He developed uncontrollable nosebleed and finally had to give up work because of the epistaxis, weakness, and dizziness. Hospital examination revealed pallor, pale membranes, ulceration and a bleeding spot in the nose, spongy bleeding gums, and ecchymotic spots on the extremities. Palpitation and vertigo were

also present. R. B. C., 3,400,000; W. B. C., 10,000; Hb., 65 per cent. Bleeding of the nose was frequent; twitching of head and limbs developed with later paralysis; then delirium, Cheyne-Stokes breathing, and loss of sphincter control appeared, the patient finally lapsing into coma and death.

Case 3.—Fatal. Male, 30 years of age. Since 1915 had been working in the tire-building department. Late in 1915 he noticed red spots on the face and neck. In spring of 1916 he began to feel weak and had headaches and dizzy spells. He consulted a physician, whose report disclosed pallor, general weakness, loss in weight, dyspnea, palpitation, weak gait, headache, dizziness, tinnitis, low red and white blood cell counts. Two months later he had severe nosebleed with aggravation of all his past symptoms. Entered the hospital two weeks later because of uncontrollable nosebleed. Evidences of ecchymoses on legs were then present. R. B. C., 2,136,000; W. B. C., 2,000; Hb., 40 per cent; total mononuclears, 69 per cent; polynuclears, 29 per cent. Developed acute lobar pneumonia. Autopsy report: Acute lobar pneumonia, pulmonary edema, anemia, exhaustion consequent on poisoning by benzol.

In addition, Harrington reports several nonfatal cases, one of a tire builder who developed a papular eruption on arms, feet, ears, and neck. Blebs then formed, accompanied by fever and itching. This patient stated that 16 or 18 other workers among the 120 employed in that department suffered from similar lesions. These local symptoms disappeared on the substitution of naphtha for benzol. In the Bulletin of the New York State Industrial Commission (55) two fatal cases are reported in men who were employed in the coating of fabrikoid with a rubber mixture dissolved in benzol. The benzol fumes escaped from the hot rubber-coated fabric which readily dried as it emerged from the spreader. The symptoms were those typical of chronic benzol poisoning. The first case was that of a man who had been working for about nine months when he noticed that he was bleeding from the gums and had red spots on his legs. Shortly afterwards he had severe nosebleed, which became more frequent. He entered the hospital and died about two weeks later. The second man had been working only for about five months. He had not been feeling well and one night he had a severe nosebleed with bleeding from the gums. His temperature rose to 104°F. The symptoms continued until he died. During a subsequent controversy which arose, it was asserted that the company had been negligent in not attempting to remove the fumes, as even before the mishap some complaint had been made regarding the ventilation. It was brought out that the other men in the coating room were subject to relatively frequent nosebleeds, but that still others had been employed in the coating room for 15 years without suffering any bad effects. Tests were made of the room air in the vicinity of the coating machines, and the highest concentration found was less than 5 per cent of benzol.

Newton (56) reports some interesting cases of early poisoning in his study of industrial poisons during the war. He cites the cases of three chemists who were exposed to benzene vapor for periods approx-

imating two weeks. The first case, aged 30, had been intensively exposed for about two weeks and complained of headache, loss of appetite, lassitude, and loss of weight. Sudden abdominal pain, with nausea and vomiting, developed. Physical examination was negative. A leucopenia of 1,200 white cells per c. mm. was present. He states that the intoxication in this case was largely from inhalation. The condition, including the leucopenia, was entirely cleared up after a month's leave of absence. The second case was that of a chemist exposed for about two weeks, who showed a leucopenia of 1,250 white cells per c. mm. but complained of no symptoms. Continuing with his work, but with hygienic instructions, the white count rose to 3,200 per c. mm. within a month. The third case was that of a chemist, similarly exposed for about two weeks, who presented a leucopenia of 1,700, with a red cell count of 3,680,000 per c. mm. Allowed to continue with his work, but following out hygienic instructions, the blood count at the end of a month showed 3,200 white cells and 4,352,000 red cells per c. mm. The above cases are interesting in that they show the early effects of chronic benzol poisoning upon the cell content of the blood, a condition which may exist without the presence of other symptoms, and which suggests a practical and ready means of detecting incipient cases. The chief danger in chronic benzol poisoning arises from the fact that only after serious damage has occurred does the patient realize the gravity of his condition. The institution of periodic blood examination of all employees exposed to benzol fumes, irrespective of concentration or ventilation devices, would go far toward avoiding such hazards.

Flandin and Roberti (57) call attention to a fatal case of purpura in an automobile factory. A young woman had been employed for about two months in a small "hot room," using a solution of rubber dissolved in benzol. The room was poorly ventilated, and five other employees were working in it. They were free to take frequent rest periods outside in the fresh air. The patient in question, who was previously well, developed headache, dizziness, and pallor, fatigue, nausea and vomiting; then, later, ecchymoses appeared on the extremities and body, and also bleeding from the membranes. There resulted a progressive anemia, with fever. The blood showed R. B. C. 2,110,000; W. B. C. 1,600; Hb. 20 per cent; polynuclears 27 per cent; mononuclears 67 per cent. Death followed within three weeks of the onset of symptoms. Previous to this fatality three other cases of purpura had occurred among the employees of this "hot room," one of the cases proving fatal. Starr (58) stresses the importance of detecting the early symptoms of benzol poisoning, emphasizing the fact that the late advanced signs and symptoms usually imply the existence of considerable damage to the organs and tissues and the loss of much valuable time so far as a possible

cure is concerned. He calls attention to a rather unusual outbreak of chronic poisoning. Benzol cement, for economic reasons, is used to some extent in the millinery industry in place of sewing. That the hazard in such use is not negligible is shown by his report. In a millinery establishment a young girl employed as a hat maker presented symptoms of gastralgia, irritation of the upper respiratory passages, headache, and tachycardia upon which a diagnosis of benzol poisoning was based. On inquiry it was found that the girls were employed in brushing benzol cement on buckram hat forms on which they then pressed the cloth or other material to be attached. Natural ventilation in the workroom was poor and no means for artificial ventilation were provided. The benzol odor was quite distinct, even at some distance from the workers. For a while the windows had been closed because of the cool weather prevailing outside, and it was at this time that the chief difficulty arose. The company officials claimed that for the past four years the same brand of cement had been used and that no trouble had arisen. Twenty-two of the 27 girls who actually handled the cement presented definite symptoms of chronic benzol poisoning. Two out of four others who did not handle the cement, but who were exposed to the fumes in the room, also gave evidence of being affected. The chief symptoms elicited were "irritation of the mucous membrane of the upper respiratory tract, nausea, vomiting, burning sensation in the epigastrium, frequent urination, giddiness, slight air hunger, and weakness." Starr states that "These symptoms have a tendency to grow worse after the affected person leaves the atmosphere of the factory." Since the cement in this case was only one-third benzol and two-thirds carbon tetrachloride, it is probable that the latter substance contributed to the symptom complex.

Reifschneider (59) also draws attention to the high percentage of workers giving evidence of early symptoms of benzol poisoning. Out of 60 workers employed in the coating room or "dope room," in a can factory, 48 per cent complained of headaches, 36 per cent of dizziness, 16 per cent of pains in the abdomen, 12 per cent of nosebleed. Out of 60, two had red cell counts under 2,000,000, nine had counts under 2,500,000, thirteen had counts between 2.5 and 3.5 million. Reifschneider directs attention to an apparent family characteristic of high susceptibility to benzol. In one family there were three cases, two of which were fatal, while the third had a secondary anemia and was removed before other symptoms developed.

Meda (60) calls attention to the frequency of chronic benzol poisoning in Milan. Here many women are employed in the manufacture of raincoats and other rubber goods in which rubber cement is used. He reports three fatal cases occurring in the winter of 1921 among young women who used rubber cement in this work. Four other similar

fatalities occurred in another factory, in addition to several other cases of poisoning which did not prove fatal. Four additional fatal cases occurred in 1922. In this connection Meda cites a case illustrating the particular danger of benzol poisoning in cases complicated by pregnancy. A young married woman had been engaged in raincoat manufacture for seven years without manifesting any untoward symptoms. Several months after becoming pregnant, however, definite signs of poisoning rapidly set in with increasing severity. Headache, dizziness, nausea, bleeding gums, and purpura appeared. Her delivery was normal, but the symptoms progressed, the red cell count dropping to 600,000 per c. mm., and the white cell count to 1,700 cells. The hemoglobin fell to 15 per cent. With careful treatment, however, this patient recovered. Pugliese (61), who studied these cases in Milan, found concentrations of 1,000 parts benzol per million parts of air, and states that serious poisoning may result from concentrations of 200 to 300 parts per million parts of air. In the Milan cases Meda calls attention to the apparent spontaneous outbreaks of chronic benzol poisoning which seem to appear more frequently in the winter months and to which young women are peculiarly susceptible. He believes that among other predisposing factors, chlorosis, tuberculosis, and pregnancy play a prominent part.

Brücken (62) relates two interesting cases occurring in a rubber factory where girls were employed in cementing rubber balls. One, a girl of 22, had been employed for three years. Headache, dizziness, and weakness developed, while later, blue spots readily appeared on the slightest injury. Frequent and excessive menstruation and an extreme secondary anemia were also present. His other case was that of a young woman, aged 23 years, who had also been employed for about three years. The usual symptoms of chronic poisoning appeared, headache, dizziness, palpitation, bleeding gums, frequent menstruation, blue spots on the arms and legs. An advanced anemia was also present. Both cases responded favorably to rest and medical treatment. Faure-Beaulieu and Levy-Bruhl (63) give some interesting neurological findings in the case of a female factory worker who developed malaise, faintness, nausea, palpitation, and general loss of strength. In time nervous symptoms appeared which indicated lesions of the posterior columns and of the pyramidal tracts. The main neurological signs were: Increased tendon reflexes, bilateral clonus, positive Babinski, impairment of deep sensitivity, pseudo-tabetic lesions with paresthesias (pain and temperature sense impairment), ataxia and paraplegia, and motor impairment, signs all indicating sclerosis of the dorsal columns and pyramidal tracts. Such lesions were probably due to the combined effects of the prolonged and persistent anemia, and to the possible

effect that the benzol itself might have exercised on the central nervous structure, inasmuch as it has been found that benzol finds special lodgment to excess in these tissues. The blood count in this case showed the usual severe secondary anemia and other findings associated with advanced chronic benzol poisoning.

Teleky and Weiner (64) give several interesting cases occurring in connection with the manufacture of rubber goods. Of 11 cases examined, only 2 were found to be free from any evidences indicating benzol poisoning. The symptoms presented were variable—headaches, nausea, eructations, vomiting, tendency to bleed from membranes, irritation of conjunctivæ, and menstrual irregularities being the most frequent. The usual secondary anemia with reduced platelets and inversion of the leucocyte-lymphocyte formula were present. The substitution of benzine for benzene for a period of eight weeks resulted in considerable alleviation of the symptoms, the definite improvement being marked both objectively and subjectively. They emphasize the value of the substitution of other solvents in place of benzol, together with careful medical supervision, believing that blood counts offer a ready means of detecting the early cases. Rohner, Baldridge, and Hansmann (65) made a careful study of a case of chronic benzol poison in an effort to correlate the morbid findings described in man and animals exposed to the action of this solvent, and to observe the effects of benzol in chronic myelogenous leukemia. The patient had been exposed to benzol fumes for about three months and presented the typical findings of chronic benzol poisoning. The red cells were reduced to 860,000 and the white cells to 1,400 per c. mm.; hemoglobin, 20 per cent. The platelets were reduced to 70,000 per c. mm. Autopsy showed numerous hemorrhages into skin membranes and meninges. A bronchopneumonia was present; also focal necrosis of the liver.

Schwenke (66), reporting typical cases of benzol poisoning, concludes that the fumes of crude benzene or the first runnings appear to be more toxic than the purified product.

In addition to chronic cases of poisoning by inhalation, local lesions have been reported from the prolonged handling of compounds containing benzol. Carozzi (67), in the results of his inquiry into the Italian printing trade, states that "among linotypists and pressmen we may occasionally observe a peculiar symptomatology, confirmed by the examination of the blood and caused by the continual use of benzol, benzine, mineral oil, and benzographol (pricking, tingling, numbness in the extremities; the number of blood corpuscles is greatly increased)." Vignolo Lutati (68) cites two cases of occupational dermatitis in the interdigital webs of fingers. The hands had been frequently wetted in solution of india rubber. Inquiry

showed that a number of other workmen had been obliged to give up work because of similar skin lesions. In Harrington's (34) study of 120 men employed in tire building, a tire builder stated that 16 to 18 men employed in his department had developed lesions similar to his own. The worker in question had been employed for two years, but during the last three months a papular eruption had appeared on his arms, feet, ears, and neck. These lesions disappeared when naphtha (presumably petroleum naphtha) was substituted for benzol. Milian (69) also records a case of erythema of the extremities in a worker who handled dyes dissolved in benzene. Simonin (13) noted irritation of the skin, with swelling and itching, in acute poisoning by swallowing. Benzene erythema and other cutaneous lesions are relatively frequent in workers handling compounds of benzene. Wolff (70) showed a passing disintegrating effect of benzol on the skin when there is prolonged contact.

In summarizing this review of the literature the following factors should be emphasized in regard to chronic benzol poisoning.

Chronic benzol poisoning occurs with greater frequency in cold weather or the winter months when natural ventilation is usually reduced to a minimum by closed windows and doors so that the concentration of the poison in the atmosphere reaches a maximum. Atmospheric conditions of temperature and humidity also play an important part. At times of high heat and high humidity, other things being equal, spontaneous and sporadic outbreaks are most likely to appear. Young girls are especially predisposed to benzol poisoning, as are also pregnant women. Conditions of general ill health, tuberculosis, chlorosis, and other conditions lowering the general vitality or interfering with free function of the eliminative organs have a strong predisposing effect. The early symptoms comprise headache, dizziness, malaise, loss of appetite, ready fatigue, shortness of breath, and burning of the eyes, throat, and respiratory-membranes. Later, nausea and vomiting, with epigastric burning and pain, may appear, and sensations of chilliness and general weakness, with bleeding from the mucous membranes of the nose, mouth, gastro-intestinal tract, and genito-urinary tract. Too frequent menstruation, with prolonged and uncontrollable bleeding from the vagina, is very common. About this time petechiae and purpuric hemorrhages may appear on the body and extremities. Burnet (71) regards purpura as a more serious prognostic sign than bleeding from the membranes. Other lesions of the skin may appear in the form of itching erythema, dry scaling, or vesicular papules. Later, complications affecting the central and peripheral nervous system may develop. Various forms of neuritis, with lesions affecting the dorsal column and the pyramidal tracts, may appear, giving rise to pares-

thesias, anasethesias, impaired locomotion, tremor, and trophic disturbances (63). The blood changes are typical.

A leucopenia develops in most cases after exposure and increases in severity, although in some cases this is not absolute. The leucopenia may be extreme, reaching almost a complete absence of white blood cells. A reduction in the red cell count then appears, but somewhat later than the leucopenia. The resultant anemia may become extreme, the red cell count being lowered to less than 500,000 per c. mm. A corresponding reduction in the hemoglobin occurs to as low as 15 per cent. A considerable reduction in the platelet count appears, especially in the later stages of poisoning. Blood smears present the picture of an aplastic anemia showing but slight changes in the appearance of the red cells. Some pallor and anisocytosis are common. Almost complete absence of regenerative forms with very few megaloblasts and normoblasts are noted, with scantiness of platelets. A marked diminution of the granular types of white blood cells is seen, with a relative increase in the mononuclear forms. The leucotoxic effect is first noticed and is more pronounced on the myelocytic than on the lymphatic elements, so that an inversion of the leucocyte formula results. This gives a relative increase in the lymphocytes and a lowering of the polymorphonuclear forms.

In rapidly developing cases, however, this inversion of the leucocyte formula may not appear, according to Fontana (72). A relative increase in the large mononuclear cells is common. Eosinophilia has also been noted in early poisoning, but this disappears rapidly on cessation of exposure (13), (73). The urine may show albumin, fat, casts, hemoglobin, and conjugated sulphobodies. On autopsy the typical findings consist of hemorrhages in the skin, mucous and serous surfaces, and the viscera, pallor of the organs, with muscles of a deep red color. Submucous hemorrhages are found along the gastrointestinal tract, in the pleura, pericardium, endocardium. The marrow of the long bones presents the typical picture of an aplastic anemia.

The treatment in chronic benzol poisoning consists of change of employment in the very early cases, preferably to open-air work, together with the observance of general hygienic measures. These early cases generally respond very promptly to treatment. In the severe cases of advanced poisoning in which hemorrhages and anemia have occurred, treatment consists of rest, fresh air, sunshine, iron-containing foods, and the general hygienic measures that are used in combating secondary anemias, tonics, hydrotherapy, ultraviolet ray, etc. In advanced anemia with purpura and hemorrhages, McClure emphasizes the value of frequent and repeated blood transfusions. It is well to bear in mind that the anemia and other signs frequently continue and progress for long periods after removal from

exposure, so that carefully adjusted treatment and prolonged convalescence are essential.

IV. THE EFFECT OF BENZOL UPON THE BLOOD CELLS AND ITS USE AS A THERAPEUTIC AGENT

The most characteristic pathological effect of benzol is, perhaps, its destructive influence upon the cells of the blood and the blood-forming organs. This effect is so important that it deserves detailed consideration. It seems also desirable to refer to the use of this property of benzol in the treatment of certain diseases characterized by an excess of white blood cells, even at the cost of a slight digression from the subject of industrial benzol poisoning, so that the literature of all phases of the subject may be presented in one place.

The typical result of exposure to benzol is a decrease in white blood cells, and this is often followed by a similar reduction in red cells. The first reaction may, however, be an increase in red cells, and, with very minute doses, there may even be an initital increase in the white cell. These early increases are undoubtedly due to slight destruction of cells in the circulating blood overbalanced by an increased production of new cells by the cell-forming or hematopoietic organs (the spleen, lymph nodes, and bone marrow). Prolonged dosage or temporary high dosage is always accompanied, however, by damage to these hematopoietic organs themselves and by marked reduction in blood cell content.

The temporary stimulating influence of slight amounts of benzol is illustrated by the work of Rabe and Hirshland (74), who found that the administration of minute doses (first to thirtieth decimal dilutions) to men and women produced an increase in white cells (with a decrease in red cells, which seems somewhat anomalous) and an increase in the amount of urine. So, too, Langlois and Desbouis (75) found that exposure to small quantities of benzene fumes in low concentration led to the production of a leucocytosis. There likewise resulted a hyperglobulia, the increase in the red blood cells amounting to as much as 33 per cent above normal. There was, however, no coincident increase in the hemoglobin. Langlois and Desbouis found that within 10 days after exposure the blood returned to its normal state. They also pointed out that these increases in red and white cells were due to an intense hematopoiesis and not to loss of the blood plasma. Similar findings have been noted by other observers (48), (76), (77), (78).

The normal destructive influence of benzol in higher concentration is illustrated by the work of Pappenheim (79), who, by injecting benzol subcutaneously, obtained a leucopenia with a relative decrease in the polymorphonuclear cells, while the lymphocyte count remained more

or less unchanged. The leucopenia occurred in the peripheral blood stream, but in the splanchnic vessels he observed an increase in the leucocyte count. This leucotoxic effect of benzol was made the subject of considerable investigation by Selling (48) (76), who reported three cases of chronic benzol poisoning with two deaths. In these cases marked leucopenia and anemia were observed and the autopsy findings consisted primarily of hemorrhages in the skin, mucosa, and mesentery. Prolonged studies of the effect of benzol upon the hematopoietic system in animals threw important light upon the degenerating and regenerating influences affecting the entire hematopoietic system. From these animal experiments Selling emphasized the leucotoxic action of benzol, which not only destroyed the white cells in circulation but extended its destructive influence to the entire hematopoietic system, the bone marrow, lymph nodes, and spleen, and also led to fatty degeneration of the other organs. The leucopenia he found to affect the polymorphonuclear cells to a considerably greater extent than the lymphocytes, and, similarly, the myelocytic structures more than the lymphadenoid. Little direct effect was noted in the circulating red cells, however. In addition to the anemia, pallor and some anisocytosis were present, but no poikilocytosis. Autopsy revealed processes amounting to almost complete aplasia in the bone marrow, this aplasia affecting all the cell types. In the spleen a destruction of the parenchynal elements leading to an aplasia was noted, the aplasia affecting the cells of both the malpighian corpuscles and the pulp strands. Fontana (72), in his experimental work, noted a diminution in the volume of the spleen in rabbits poisoned with benzol. Similar destruction was noted in the follicles of the lymph glands. Of the other organs the liver and kidney showed varying grades of fatty degeneration, and hemorrhages were present in the serous and mucous surfaces. Selling noted that if the benzol injections were discontinued in time, active regeneration occurred in the organs and islands of blood-forming cells developed in the marrow. In the spleen active myeloid-metaplasia occurred and islands of myelocytic and erythroblastic tissue developed in addition to the usual splenic structures. Similar findings have been noted by other workers (72) (78) (104).

Weiskotten (78) and his colleagues call attention to a marked initial fall in the absolute number of both amphophiles and small mononuclears. This preliminary fall is followed by a primary rise in the leucocyte count, which he shows to be due mainly to an increase in the amphophiles. This is the phenomena observed by Rabe and Hirshland and Langlois and Desbouis. This primary rise is followed by a secondary drop in the white cell count, which again is due mainly to a drop in the amphophilic cells, the small mononuclear cells playing

but a secondary rôle in either the increase or decrease. The amphophilic curve follows closely that of the total white cell curve, and toward the end of the experiment, when benzol has been discontinued, the total white count assumes a new level which is generally somewhat lower than the original normal level. This lowered new level is again due in part to the failure of the small mononuclears to rise to the point existing before the beginning of injections, whereas the polynuclear cells do approximate the original level (80). In benzene inhalation experiments similar results were obtained, but no diphasic leucopenia was observed (103). In their study of the red cell reduction, Weiskotten and Steensland (80) found that the compensatory metaplasia in the spleen was of slight significance and that this organ played an insignificant rôle in either the destruction or regeneration of the red or the white cells following benzol poisoning. A relative eosinophilia has been noted in acute and early chronic poisoning, but this rapidly disappears on removal from exposure (13) (73). Duke (81), experimenting with rabbits, obtained an initial rise in the platelet count up to 1,780,000, but this was followed by a rapid fall to a value as low as 61,000. Hurwitz and Drinker (82) found that the platelet count might remain high with a low white cell count, and in the light of their bone marrow findings they suggested that this indicated either very rapid regeneration of the megacaryocytes (the origin of platelets) or a greater resistance of the megacaryocyte to the action of benzol. Weiskotten and his colleagues (83) noted a similar temporary increase in the platelets or thrombocytes, but this increase was in all cases followed by a gradual fall.

Study of the bone-marrow sections showed that as active necrosis developed in the bone marrow, the thrombocyte curve fell; but as regeneration of the marrow set in, active thrombocytosis appeared, with an abrupt rise in the thrombocyte count. Sections of bone marrow at the beginning of this thrombocytosis showed large numbers of megacaryocytes in the regenerating marrow, this seeming to indicate that thrombocytes originate in this tissue. Similar decreases in the platelet count have been noted by other observers (48) (72). When the reduction in the platelet count becomes extreme, purpura hemorrhagica, with hemorrhages from the membranes, may take place. Associated with the decreased platelet count, Hurwitz and Drinker (82) have noted a reduction in the circulating prothrombin, but only in one case could they lower the platelets sufficiently to reproduce symptoms of hemorrhagic disease. Duke (81) noted prolonged bleeding time in benzol poisoning, but this occurred only when the platelets fell to a dangerously low level. Forbes and Hompe (84), exposing cats to an atmosphere of benzol vapor sufficient to keep them unconscious, found no constant change in the coagulation time or in the prothrombin content. No hemorrhages were found after five

hours' exposure and no evidence of hemolysis was present, and the urine was never dark or cloudy. It would seem that the changes in the bleeding and coagulation time are not so likely to appear in acute poisoning as in chronic poisoning, a fact which probably explains the lack in uniformity of results obtained by Forbes and Hompe. McClure (51) observed greatly prolonged bleeding time in his cases, as have also most other observers in either animal experiments or in clinical observation.

The unfavorable influence of benzol upon the blood cells has led to further important research into its effect upon the general serological and immunological aspects of the blood. Winternitz and Hirschfelder (85), working on experimental pneumonia in rabbits, found that the resistance of these animals was strikingly reduced after they had been given benzol injections sufficient to produce a leucopenia. The animals were rendered leucopenic by benzol administration, and intratracheal insufflations of virulent pneumococci in graded doses were then administered. It was found that the resistance of the animals so treated was markedly reduced, so much so that they all died within 13 to 27 hours after injection, while in the control animals not treated with benzol the average interval preceding death was 61 hours. It was also found that the cells of the exudate in the lungs were characteristic and proportional to the type and the number of cells found in the bone marrow at the time of death. Besides the usual cell elements, the exudate included such rare forms as megacaryocytes and granulocytes corresponding to the types found in the marrow. Kline and Winternitz (86) further emphasize the lowering of resistance that accompanies such leucopenia. Animals suffering from leucopenia caused by benzol readily succumb, while animals treated with injections of toluol (homologue of benzene which produces a leucocytosis) not only have normal resistance but exhibit an increase in resistance proportional in some degree to the leucocytosis produced. They note, moreover, that the blood count is not always a safe index of bone marrow activity. Weiskotten and Steensland (87), in studying the effect of benzol upon rabbits, noted that evidences of active acute infection frequently appeared which were not present prior to the injections. They suggest that benzol administration by lowering the resistance may stir up latent or quiescent infection. In three out of the four animals developing spontaneous infections after benzol treatment, there was absence of the typical bone marrow aplasia, and they conclude that the white cell count is not an absolutely safe guide in the therapeutic administration of benzol. Similar observations had been made by other observers.

Weiskotten, Schwartz, and Steensland (88) have noted that the interval between the first injection of benzol and the onset of the deuterophase (secondary decline in the number of white blood cells)

varied between 8 and 13 days. This corresponds closely with the period required for sensitization after injection of antigens. By subjecting rabbits to successive sets of injections the investigators found that there occurred no changes in the deuterophase, suggesting that it was attributable to an antigen-antibody reaction. Schiff (89) demonstrated, however, that small doses of benzol increased the anaphylactic reaction of guinea pigs sensitized to sheep serum, while large doses decrease their sensitiveness.

Hektoen (90), using defibrinated sheep blood as antigen found a marked reduction in the formation of percipitins and hemolysins and in phagocytic power in rabbits which had been treated with benzol. In some animals he found almost an entire suppression of these immune bodies. Together with these findings there appeared the usual picture of leucopenia and injury to the hematopoietic apparatus. Rusk (91) likewise found a similar depression in the production of hemolysins and precipitins when animals had been treated with benzol. Simonds and Jones (92) using washed dog blood corpuscles, as antigen, made a study of the hemolytic titer in rabbits previously treated with subcutaneous injections of benzol. They found that the titer of the serum in the benzol-treated animals ranged from one-sixteenth to one-quarter that of control animals. Using killed cultures of *B. typhosus* as antigen they found a marked decrease in the production of agglutinins, but to a lesser extent than in the hemolysins. Using typhoid bacilli, they found a markedly reduced power to produce opsonins, but this was noted to a still lesser extent than either the reduction in the agglutinins or hemolysins. White and Gammon (93) noted that rabbits which had been treated with benzol inhalation were considerably less resistant to tuberculous infection than animals not so treated, and Camp and Baumgartner (94), in their study of the inflammatory reactions in rabbits treated with benzol, found that little resistance was offered to the entrance and growth of bacteria in such animals.

It appears from the foregoing that, apart from variation and individual susceptibility and the effect of atmospheric conditions, such as heat, etc., all of which may modify the benzol hazard in any particular case, there may also result in benzol poisoning a depression in the fundamental defense mechanisms of the blood which markedly lowers resistance to other disease processes, all of which considerations contribute still more to our realization of the seriousness of the benzol hazard.

Suprisingly little has been done in regard to the ultimate fate of benzol in the body. It would appear from the work already reported, however, that a considerable portion of the absorbed benzol is excreted in an unchanged condition through the lungs. Juvalta (95), on feeding phthalic acid, found that the benzene ring was broken down

in the animal body. Jaffe (96), in experiments upon dogs and rabbits, noted that, following the prolonged administration of benzene, very small quantities of an N-free acid were present in the urine. The formula was determined to be $C_6H_6O_4$, an oxidation product of benzol, and was found to be identical with muconic acid. This represented, however, but a minute fraction of the total benzene administered. Fuchs and Soos (97) confirmed the work of Jaffe. Sohn (98) found that benzene influenced the oxidative or katabolic processes in the organism, as was shown by an increase in neutral sulphur and changes in the amount of urea and NH_3. Albuminuria also appeared. Brewer and Weiskotten (99) made daily determinations of the phenol content of the urine of rabbits prior to and subsequent to daily injections of benzol, the determinations extending throughout the entire cycle of the leucocyte curve. They found that the phenol content of the urine increased during the prophase, or initial drop in the white cell count, but that no increase in the phenol content occurred during the deutrophase, or secondary drop in the count. They conclude from this that the secondary drop in the leucocyte curve is not the result of slow or of delayed absorption of the benzol, as was suggested by Selling (76). Underhill and Harris (100), studying the influence of benzol upon metabolism found a marked increase in creatine output within 48 hours after injection of the substance. This was shortly followed by a sharp fall in the creatine output, but a secondary rise in the excretion appeared still later. There was a less marked increase in the output of total nitrogen, and the total nitrogen elimination curve followed closely that of the elimination of creatine although the total nitrogen was not increased to so great an extent as the creatine. They conclude that benzol exerts a marked catabolic influence on the body structures as a whole.

V. THE EXTENT OF THE BENZOL HAZARD IN INDUSTRY IN THE UNITED STATES

Passing from the review of the existing literature of benzol poisoning first of all will be considered the evidence obtained in regard to the seriousness of the problem as it exists in industry in the United States at the present day.

In order to throw light upon this general question, and also to obtain information as to the localities where more intensive studies could profitably be undertaken, a preliminary list was prepared of 324 industrial establishments which, from the nature of their products, might be expected to use benzol. Through the courtesy of Mr. W. S. Paine, of the Aetna Life Insurance Co., the following questionnaire was forwarded to all of the 324 industrial establishments in July, 1923.

PRELIMINARY QUESTIONNAIRE FOR BENZOL STUDY

1. Is benzol used or produced in any of the processes in your plant?
   If so, would you be willing to give some information as to the extent to which it is used and the amounts employed?
2. How many of your employees are in processes where benzol is present?
   (*a*) Fumes.
   (*b*) Liquid.
3. How many are directly exposed to benzol?
   (*a*) Fumes.
   (*b*) Liquid.
4. Have you experienced definite cases of poisoning?
5. Have you considered benzol poisoning as a cause of illness, absenteeism, or labor turnover among your employees on account of any special type of sickness, for example—
   (*a*) Anemia, purpura, hemorrhages, gastro-intestinal or nervous disturbances, neurasthenia, disturbance of menstrual function in women workers, or
   (*b*) Sudden cases of collapse, shock, cyanosis or heart failure.
6. What precautions are you taking to eliminate hazard from benzol poisoning?
   (*a*) Fumes.
   (*b*) Liquid.

Name of firm: ----------
Address ----------
Please forward to:

One hundred and forty replies were received in answer to this questionnaire. Fifty-six of those who answered replied that they did not use benzol in any form, leaving 84 firms who did make use of this substance. Some idea as to the amount of benzol used by the firms replying to the questionnaire may be gained from the following figures: Thirty-six replies gave no definite information in regard to this point; in 19 cases the amount of benzol used was insignificant; in 6 cases it was between 5 and 25 gallons per week; in 4 cases between 26 and 99 gallons; in 10 cases between 100 and 500 gallons; in 3 cases between 501 and 999 gallons; in 6 cases over 1,000 gallons—reaching the very high figures of 2,200, 3,000, 7,500, 8,000, 10,000, and 30,000 gallons per week, respectively. The six largest users included three rubber companies (8, 23, and 25) and three large chemical concerns (13, 24, and 19), one of the latter being principally concerned with the supply of products for the making of can seals. It is interesting to note that out of 15 companies reporting cases of benzol poisoning, only 2 were among those using less than 100 gallons of benzol per week; 4 were among those using between 100 and 1,000 gallons; 3 were among the 6 firms using over 1,000 gallons; while 6 were among the 36 firms giving no information as to the amount of benzol used.

A more helpful analysis from our standpoint deals with the number of employees exposed to possible toxic effects of the benzol. We have this information reported for 67 firms, 73 industries having

failed to state the number of employees exposed in processes where benzol constitutes a hazard. The general results are indicated in the table below, with comparative figures showing the number of firms in each class which reported having had cases of benzol poisoning.

TABLE 1

	Number of employees exposed to possible benzol poisoning						
	0–4	5–9	10–24	25–49	50–99	100 and over	No information
Number of establishments in each class	30	14	10	7	2	4	73
Number of establishments reporting cases of poisoning	3	1	3	3	1	4	----

Out of 23 establishments with 10 or more employees exposed to benzol poisoning, 8 were rubber factories, 5 were chemical works, 4 were paint and varnish makers, 3 were gas plants making benzol as a by-product, and 3 were plants of other types.

It will be noted that out of 44 establishments with less than 10 employees exposed to benzol poisoning only 4 reported having experienced cases of poisoning. Of 17 firms with from 10 to 49 employees exposed, 6 had experienced cases of poisoning, while of the 6 firms with more than 50 persons exposed to benzol all but one had experienced industrial poisoning by benzol. It is of course possible that facilities for the diagnosis of benzol poisoning may influence data such as Table 1 presents, and that full-time physicians in large industries may naturally be expected to diagnose cases of industrial poisoning more frequently than persons who are not familiar with the symptoms of such conditions.

Table 2 presents the more important data with regard to 33 individual industries whose reports seemed of special interest for one reason or another. The table includes (*a*) all plants reporting cases of benzol poisoning; (*b*) all plants reporting the exposure of 10 or more employees to the possibility of benzol poisoning and (*c*) all plants reporting the use of 500 or more gallons of benzol per week. The plants are arranged in order according to the number of employees exposed to benzol and are identified by arbitrary key numbers, since the information obtained in this study was in many cases of a confidential nature.

TABLE 2.—*Data with regard to establishments where benzol hazard may exist*

Key Number	Product	Gallons of benzol used per week	Number employees exposed	Poisoning cases	General precautionary measures used
8	Rubber goods	2,200	1,080	Yes	Ventilation.
21	do		150	Yes	Do.
9	Chemicals	525	100	Yes	Closed containers; ventilation.
15	do		100	Yes	Closed apparatus; ventilation; helmets; partial elimination of benzol.
5	Rubber goods	100	50	Yes	Ventilation; medical examination.
26	Celluloid, leather, cloth.		50	Yes	Local ventilation; medical examination.
27	Japanned goods	60	35	No	Ventilation.
6	Gas by-products	500	32	Yes	
25	Rubber goods	7,500		No	Closed containers; ventilation.
23	do	8,000	30	Yes	Do.
28	do		25	No	Limiting use of benzol.
29	Chemicals		25	No	
17	do		25	Yes	Closed containers; ventilation.
30	Rubber goods	25	25	No	
31	Gas by-products		20	No	Closed containers.
38	Paints and varnishes.		20	No	Ventilation; closed containers.
32	Cans		18	No	Local ventilation.
11	Paints, varnishes		15	Yes	Respirators.
33	Gas by-products		13	No	Closed containers; ventilation.
20	Rubber goods	25	12	Yes	Local ventilation; rubber gloves.
22	Vegetable oils		12	Yes	Medical examination; education.
34	Chemicals		10	No	Closed containers; ventilation.
35	Paints, varnish	250	10	No	Education.
10	Chemicals		10	No	Closed containers; care in cleaning tanks.
19	Can solvents	10,000	8	Yes	Ventilation.
24	Chemicals	30,000	7	No	
36	do	750	4	No	Closed containers; ventilation.
37	Benzol used in cutting asphalt.	700	4	No	Closed containers; low temperatures.
13	Chemicals	3,000	4	No	Closed containers.
12	Paints, varnish	150	3	Yes	Change of employment.
7	Rubber goods	5	3	Yes	Ventilation; limitation of use of benzol.

The result of this preliminary study was to indicate that the industrial firms using benzol were generally alive to the dangers involved and had in most cases taken definite precautions for the protection of their employees. Nevertheless out of 23 establishments in which 10 or more persons were exposed to benzol, 11, or nearly one-half, reported cases of poisoning; while of 6 plants in which 50 or more employees were exposed, all but 1 reported poisoning.

During the second year of the investigation many additional reports of benzol poisoning were obtained in those plants which were studied more intensively, and the net results of the questionnaire and the field study are presented in Table 3.

TABLE 3.—*Deaths and illness due to the use of benzol in industry, 1922-1924*

Plant No.	Accidental exposure to heavy concentrations		Habitual exposure to lower concentrations		Plant No.	Accidental exposure to heavy concentrations		Habitual exposure to lower concentrations	
	Deaths	Illnesses	Deaths	Illnesses		Deaths	Illnesses	Deaths	Illnesses
5			4		48	2			1
6		1			50				1
7				1	59				2
8				44	61				2
9	1				62				
11				1	67	1		2	7
12				Several.	76				1
15			3	Several.	78			2	3
16				1	83				1
17		1		1	93				7
20				2	95				2
23				2					
27				2	Total	4	2	11	81

In the period going back for several years prior to 1925 the necessarily very incomplete data of this survey thus revealed 15 fatalities and 83 cases of sickness due to benzol, a record of considerable significance. It is generally accepted that reports to the various State health departments understate the real situation with regard to industrial disease, and yet in the State of Ohio 29 compensable cases of benzol poisoning occurred during the five-year period ending June 30, 1925 (101) During the last year of our work no attempt was made at a further canvass of new cases; but at lest seven fatalities resulting from benzol poisoning were brought to the attention of the committee as occurring during the first nine months of 1925.

The subsequent portions of this report deal with the committee's studies of chronic benzol poisoning in some detail. It may, however, be well briefly to describe here some of the acute cases of poisoning, which have come directly or indirectly to the attention of the committee during the progress of the work.

Plant No. 6. A worker operating a benzol still was overcome by fumes when the distilling process started too rapidly and the rectifying chamber ran over.

Plant 67. An employee working in a trench to repair a leak in a pipe containing benzol was overcome by the vapors. He recovered in a few minutes and returned to work. He collapsed a second time and died without regaining consciousness.

Plant 48. In this plant, benzol was used in a chemical process. The process depended on volatilizing the benzol, and leaks have frequently occurred. The release of vapors due to these leaks resulted in the death of two workmen who were attempting to shut off the system. Recently a leak permitted such a great quantity of fumes to escape that they traveled around a fire wall, became ignited, and burned up the plant. Apparently the exhaust ventilating ducts which were of fairly large size did not check the fire hazard or save the workmen from poisoning in this case.

Benzol was used as the solvent in the grease-extraction process at a garbage-reduction plant. The tank containing the benzol was supported on several masonry supporting-walls which, with the tank bottom, formed a closed pocket. This tank was found to be leaking and the workman who went under the tank into this inclosure was killed while looking for the leak.

PUBLIC HEALTH ENGINEERING ABSTRACTS

Cities Notify Public of Water Purity. Regulations Promulgated by the State Department of Health of Minnesota. Anon. *Water Works Engineering*, vol. 79, No. 6, March 15, 1926, pp. 323–324. (Abstract by H. B. Foote.)

In this article the regulations governing the posting of signs notifying the public of the approval of the public water supply are set forth. Fees required for the inspection and analysis of the water followed by the approval of the sign posting are given. These fees are annual and are set at the following figures:

Population of municipality	Fee for each separate water-supply investigation
Under 1,000	$15.00
1,000 to 5,000	20.00
5,000 to 20,000	25.00
20,000 to 100,000	35.00
Over 100,000	50.00

Any extra investigations over the annual one must be paid for at the same rate as set forth.

Two official signs are given; each is 18 by 24 inches and should be of metal. The signs must be removed if for any reason the municipality fails to comply with the regulations and any recommendations set forth by the State board of health. It is optional with the city whether it shall come under this ruling or not, but no city shall post signs unless it has complied with all requirements.

Chlorination of Water and Sewage. Earle B. Phelps. *Journal Boston Society of Civil Engineers*, vol. 13, No. 4, April, 1926, pp. 150–167. (Abstract by E. C. Sullivan.)

This paper discusses chlorination in the light of the developments of the past 16 years. Inquiry is made into the principle and chemical basis of disinfection, the legitimate and proper rôle of chlorination in water supply practice, and the accomplishment and possibly some of the shortcomings of the process itself. It is estimated that not less than 30,000,000 people in the United States, living in some 3,000 communities, are to-day supplied with a chlorinated domestic water.

When calcium hypochlorite was first applied to drinking water, through fear that the addition of the chemical substance would lead to public disapproval the solution of hypochlorite was referred to as "potential oxygen" in an attempt to disguise the true nature of the treatment, based upon the theory that chlorine acted solely as an oxidizing agent. Later investigations have shown the oxidation theory to be untenable and that in comparison with other oxidizing agents the relative germicidal power of chlorine is much greater than

its relative oxidizing power. The view is now fairly established that the germicidal action of chlorine is a specific toxic action similar in its general effects to the action of phenol, copper sulphate, and other nonoxidizing germicides. It is pointed out that chlorine is in no sense a suitable reagent for oxidation of organic matter in general.

At the time when chlorination was first introduced it was felt that there was grave danger that this new process might be too readily accepted as a substitute for more thorough measures of water and sewage treatment. For once in the history of sanitation there was an actual danger that a sanitary achievement of real merit might be too readily accepted, for the cheapness and simplicity of the process threatened to bring about a condition of unsound practice which might discredit the process itself.

During the past 16 years the process of chlorination has become almost universal in water-works practice. It has not only been very generally adopted in the case of slightly polluted waters, but has also been used as an adjunct to filtration processes of all sorts. This development has permitted the use of higher loading factors upon filters, and has also, in many cases, provided a wide margin of safety between a badly polluted water and the consumer. There is considerable discussion at the present time of the propriety of the use of chlorination alone in the treatment of moderately polluted water supplies. Chlorination is an ideal method that provides a wide margin of safety at a low cost when superimposed as an independent process upon the usual treatment by filtration. However, in most cases where filtration is followed by chlorination somewhat greater reliance is placed upon the latter. Thus, chlorination becomes part of the major process and the margin of safety is narrowed. It is a matter of judgment just how far the "margin of safety" purpose of chlorination may safely be encroached upon in order to economize in capital and operating expenditures. It is not the matter of filtered or unfiltered waters that determines the limits of chlorination, but rather the quality of the water with which chlorination must deal. Exactly the same conditions prevail in the chlorination of raw water or waters not otherwise treated.

The writer also discusses the general reliability of chlorination processes as measured by the possibility and effects of mechanical failure; the reliability of control methods for regulating the dosage; the germicidal efficiency as affected by variable water conditions; and other similar matters. The desirability of duplicate apparatus is pointed out. The control of the dosage by the orthotolidine test is discussed. In each type and condition of water there is a certain relation between chlorine dosage, bacterial efficiency, and residual chlorine after a given lapse of time. When the test is properly standardized for a given water it becomes a valuable and almost indispensable guide

to proper dosage. The significance of "aftergrowths" is likewise touched upon. They should be looked upon with suspicion until their status in each case has been more fully determined.

The results of chlorination are discussed to considerable extent, data for a number of cities being cited. The author mentions that the gross treatment of statistical data in comparative typhoid fever rates before and after chlorination has often lead to erroneous conclusions. Suggestions are made for the proper handling of such data.

Tastes and odors are discussed briefly. For phenol wastes there seems to be no remedy except to keep the offending wastes out of the river. Mention is made of a slightly higher rate of dosage to overcome taste and odor troubles resulting from the chlorination of a water supply rich in organic matter.

The chlorination of swimming pools and also of sewage is discussed. In the usual sewage treatment processes bacterial removal is but incidental to the primary purpose of these oxidation processes. Chlorination possesses great merit for efficiency and economy in producing a reasonably disinfected effluent. Its use will be more frequently employed in the future as its merits become better understood and as need for better protection of inland and coastal waters becomes more manifest. The specific action of chlorine as a toxic agent in distinction from its action as an oxidizing agent is a general principle of chlorination, best illustrated in sewage practice.

Typhoid Epidemic from Deep Well, Polluted by Surface Seepage. R. O. Heater. *Water Works Engineering*, vol. 79, No. 5, March 1, 1926, p. 272. (Abstract by F. C. Dugan.)

In drilling a well a small stream of water was encountered at about 155 feet, or about one-half the total depth, but it was not cased off. Ten years afterwards, due to a typhoid outbreak, it was discovered that water from an abandoned rock quarry was evidently seeping into this small stream. It was cased off and the water supply has not shown any contamination since.

Pure Water—No Typhoid. Anon. *Ohio Health News*, Ohio Department of Health, vol. 2, No. 5, March 1, 1926, pp. 3–4. (Abstract by Isador W. Mendelsohn.)

East Liverpool, Ohio, procures its water supply from the Ohio River at a point only 44 miles below Pittsburgh. The effect of the installation of a modern water-purification plant upon the typhoid incidence in the city is shown in the accompanying table, the factor of improvement in quality of milk being also responsible for the favorable data.

Data on typhoid fever at East Liverpool

Year	Population	Deaths from all causes, children under 5 years		Deaths from typhoid fever, all persons	
		Total	Rate [1]	Total	Rate [1]
1915	20,900	92	341	16	75.8
1916	21,000	125	600	15	71.4
1917	21,100	124	683	15	71.1
1918 [2]	21,200	115	540	18	84.9
1919 [3]	21,300	93	437	7	32.9
1920	21,411	96	451	10	45.7
1921	21,700	97	447	12	55.3
1922 [4]	22,000	84	382	4	18.2
1923	22,300	87	390	1	4.5
1924	22,600	49	217	1	4.4
1925	22,900	----	----	0	0

[1] Rate per 100,000.
[2] Water-purification plant first placed in operation in May, 1918.
[3] Plant placed in charge of consulting analyst on part-time basis in May, 1919.
[4] Plant placed in charge of present filtration superintendent on full-time basis in September, 1922.

Determining Chemical Dosage for River Water. K. C. Armstrong, assistant chemist, Metropolitan Utilities District of Omaha, Nebr., *Water Works Engineering*, vol. 79, No. 5, March 1, 1926, pp. 267–268 and 292. (Abstract by F. C. Dugan.)

Variations in pumpage and changes in turbidity constitute a problem in determining the proper chemical dosage for the sedimentation system of the Omaha water supply. A rather unique apparatus for determining the amounts of coagulant necessary is described. The article shows what can be accomplished in the reduction of chemicals by proper laboratory control.

DEATHS DURING WEEK ENDED JUNE 26, 1926

Summary of information received by telegraph from industrial insurance companies for week ended June 26, 1926, and corresponding week of 1925. (From the Weekly Health Index, July 1, 1926, issued by the Bureau of the Census, Department of Commerce)

	Week ended June 26, 1926	Corresponding week, 1925
Policies in force	64,836,039	60,370,192
Number of death claims	12,056	11,123
Death claims per 1,000 policies in force, annual rate	9.7	9.6

Deaths from all causes in certain large cities of the United States during the week ended June 26, 1926, infant mortality, annual death rate, and comparison with corresponding week of 1925. (From the Weekly Health Index, July 1, 1926, issued by the Bureau of the Census, Department of Commerce)

City	Week ended June 26, 1926		Annual death rate per 1,000 corresponding week, 1925	Deaths under 1 year		Infant mortality rate, week ended June 26, 1926 [2]
	Total deaths	Death rate [1]		Week ended June 26, 1926	Corresponding week, 1925	
Total (64 cities)	6,196	11.3	10.7	[3] 711	712	[3] 57
Akron	36			3	4	32
Albany [4]	23	10.1	11.1	1	2	21
Atlanta	73			8	12	
White	31			3		
Colored	42	([5])		5		
Baltimore [4]	225	14.5	10.9	16	25	47
White	168			12		43
Colored	57	([5])		4		65
Birmingham	84	20.8	19.8	11	12	
White	37			4		
Colored	47	([5])		7		
Boston	177	11.7	10.6	26	29	73
Bridgeport	31			5	1	85
Buffalo	140	13.4	13.6	10	9	42
Cambridge	34	14.5	9.2	2	4	33
Camden	27	10.7	13.4	2	4	34
Canton	18	8.5	5.4	4	0	89
Chicago [4]	529	9.0	8.8	56	66	50
Cincinnati	105	13.3	11.8	8	11	50
Cleveland	200	10.9	8.7	28	18	73
Columbus	54	9.9	12.1	5	8	46
Dallas	44	11.5	12.9	5	6	
White	36			5		
Colored	8	([5])		0		
Denver	54	9.9	14.5	4	4	
Des Moines	25	8.9	11.8	2	4	33
Detroit	269	10.9	8.7	37	27	60
Duluth	18	8.3	10.9	2	0	47
El Paso	37	17.7	18.4	13	10	
Erie	22			3	7	57
Fall River [4]	28	11.1	8.5	5	4	73
Flint	17	6.5	6.8	1	5	17
Fort Worth	30	9.8	11.6	5	7	
White	27			5		
Colored	3	([5])		0		
Grand Rapids	30	10.0	11.2	2	5	29
Houston	57			7	7	
White	31			6		
Colored	26	([5])		1		
Indianapolis	64	9.1	10.5	5	6	37
White	51			3		25
Colored	13			2		110
Jersey City	69	11.3	9.1	9	10	64
Kansas City, Kans	25	11.1	10.8	0	2	0
White	18			0		0
Colored	7	([5])		0		0
Kansas City, Mo	79	11.0	11.9	7	10	
Los Angeles	198			17	28	47
Louisville	78	13.1	12.6	14	2	121
White	56			7		70
Colored	22	([5])		7		439
Lowell	25			0	4	0
Lynn	23	11.5	6.1	3	1	75
Memphis	85	25.0	23.0	13	19	
White	45			5		
Colored	40	([5])		8		
Milwaukee	91	9.2	9.4	11	7	51
Minneapolis	78	9.4	8.9	6	10	33
Nashville [4]	52	19.8	14.9	6	5	
White	32			2		
Colored	20	([5])		4		

[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 62 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, Fort 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 26, 1926, infant mortality, annual death rate, and comparison with corresponding week of 1925. (From the Weekly Health Index, July 1, 1926, issued by the Bureau of the Census, Department of Commerce)—Continued

City	Week ended June 26, 1926		Annual death rate per 1,000 corresponding week, 1925	Deaths under 1 year		Infant mortality rate, week ended June 26, 1926
	Total deaths	Death rate		Week ended June 26, 1926	Corresponding week, 1925	
New Bedford	24			3	6	52
New Haven	34	9.7	9.9	4	4	55
New Orleans	136	16.9	18.4	16	18	
White	81			9		
Colored	55	(5)		7		
New York	1,227	10.8	10.0	145	139	59
Bronx Borough	188	10.9	8.3	15	17	50
Brooklyn Borough	380	8.8	8.8	46	40	47
Manhattan Borough	507	14.1	12.1	68	68	75
Queens Borough	114	7.8	6.6	14	12	63
Richmond Borough	38	13.9	15.1	2	2	35
Newark, N. J	74	8.4	8.9	15	6	72
Norfolk	38	11.4	11.4	3	6	56
White	15			0		0
Colored	23	(5)		3		149
Oakland	45	9.0	11.9	9	3	104
Oklahoma City	22			2	5	
Omaha	42	10.2	7.6	2	3	21
Paterson	33	12.0	10.7	2	7	35
Philadelphia	453	11.8	10.7	42	45	56
Pittsburgh	135	11.1	11.1	18	20	60
Portland, Oreg	56			5	8	51
Providence	67	12.7	10.1	7	6	58
Richmond	59	16.3	14.0	9	12	113
White	39			4		78
Colored	20	(5)		5		175
Rochester	74	12.0	10.0	10	7	80
St. Louis	174	10.9	10.8	15	10	
St. Paul	54	11.4	11.9	5	5	44
Salt Lake City	25	9.8	11.9	2	3	28
San Antonio	51	13.0	17.4	15	23	
San Diego	23	10.9	16.2	2	4	42
San Francisco	123	11.3	13.7	8	5	48
Schenectady	18	10.1	10.1	3	2	87
Seattle	77			4	2	37
Somerville	14	7.3	11.6	1	2	26
Springfield, Mass	31	11.1	10.3	5	6	72
Syracuse	45	12.8	10.3	5	4	63
Tacoma	18	8.9	8.0	2	1	47
Toledo	68	12.1	8.7	9	4	87
Trenton	29	11.3	18.2	4	4	67
Utica	39	19.7	11.8	4	3	88
Washington, D. C	134	13.2	10.5	11	15	63
White	84			5		41
Colored	50	(5)		6		109
Waterbury	22			5	4	107
Wilmington, Del	22	9.3	8.5	2	4	47
Worcester	52	14.0	14.5	12	5	138
Yonkers	22	9.9	7.3	1	0	22
Youngstown	25	7.9	12.7	4	4	51

For footnotes 4 and 5, see p. 1436.

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 July 3, 1926

ALABAMA

	Cases
Cerebrospinal meningitis	1
Chicken pox	9
Diphtheria	5
Influenza	7
Malaria	38
Measles	65
Mumps	14
Pellagra	40
Pneumonia	13
Poliomyelitis	1
Scarlet fever	7
Smallpox	46
Tetanus	1
Tuberculosis	42
Typhoid fever	48
Whooping cough	41

ARIZONA

German measles	4
Scarlet fever	1
Tuberculosis	1
Typhoid fever	1

ARKANSAS

Chicken pox	4
Diphtheria	2
Influenza	23
Malaria	96
Measles	23
Mumps	13
Paratyphoid fever	1
Pellagra	38
Scarlet fever	3
Trachoma	2
Tuberculosis	18
Typhoid fever	27
Whooping cough	50

CALIFORNIA

	Cases
Cerebrospinal meningitis:	
Los Angeles	1
Los Angeles County	1
Pasadena	1
Stockton	1
Chicken pox	108
Diphtheria	101
Influenza	5
Lethargic encephalitis:	
Glendale	1
San Francisco	1
Measles	275
Mumps	73
Poliomyelitis:	
Los Angeles	1
Sacramento County	1
San Francisco	1
Scarlet fever	70
Smallpox	6
Tuberculosis	189
Typhoid fever	14
Whooping cough	71

COLORADO

Chicken pox	36
Diphtheria	6
German measles	2
Influenza	6
Malaria	1
Measles	62
Mumps	1
Pneumonia	1
Scarlet fever	14
Smallpox	7
Tuberculosis	67
Typhoid fever	11
Whooping cough	42

CONNECTICUT

	Cases
Cerebrospinal meningitis	1
Chicken pox	35
Diphtheria	7
German measles	12
Influenza	1
Malaria	1
Measles	153
Mumps	5
Pneumonia (broncho)	22
Pneumonia (lobar)	16
Scarlet fever	46
Tuberculosis (all forms)	26
Typhoid fever	4
Whooping cough	17

DELAWARE

Diphtheria	2
Malaria	2
Measles	11
Tuberculosis	5
Typhoid fever	4
Whooping cough	2

FLORIDA

Chicken pox	8
Diphtheria	8
German measles	1
Malaria	7
Measles	13
Mumps	8
Pneumonia	2
Scarlet fever	4
Smallpox	21
Tetanus	1
Tuberculosis	14
Typhoid fever	23
Whooping cough	12

GEORGIA

Chicken pox	4
Conjunctivitis (infectious)	1
Diphtheria	4
Dysentery	9
Influenza	1
Malaria	26
Measles	75
Mumps	3
Pellagra	1
Pneumonia	4
Scarlet fever	4
Septic sore throat	1
Smallpox	27
Tuberculosis	28
Typhoid fever	19
Typhus fever	3
Whooping cough	5

IDAHO

Chicken pox	12
Measles	4
Mumps	2
Scarlet fever	3
Smallpox	4
Whooping cough	5

ILLINOIS

	Cases
Cerebrospinal meningitis:	
Bond County	1
Bureau County	1
Saline County	1
Chicken pox	178
Diphtheria	43
Influenza	40
Lethargic encephalitis:	
Cook County	1
Du Page County	1
Measles	679
Mumps	45
Pneumonia	147
Scarlet fever	142
Smallpox	14
Tuberculosis	244
Typhoid fever	17
Whooping cough	173

INDIANA

Chicken pox	36
Diphtheria	16
Influenza	7
Measles	230
Pneumonia	6
Scarlet fever	49
Smallpox	46
Trachoma	1
Tuberculosis	22
Typhoid fever	3
Whooping cough	75

IOWA

Cerebrospinal meningitis	1
Chicken pox	10
Diphtheria	13
German measles	4
Measles	44
Mumps	3
Pneumonia	1
Scarlet fever	16
Smallpox	12
Tuberculosis	12
Typhoid fever	2
Whooping cough	13

KANSAS

Cerebrospinal meningitis:	
Kansas City	1
Norwich	1
Chicken pox	20
Diphtheria	14
German measles	2
Influenza	2
Malaria	1
Measles	98
Mumps	8
Pneumonia	24
Poliomyelitis:	
Cheney	1
Scarlet fever	15
Smallpox	3
Trachoma	1
Tuberculosis	78
Typhoid fever	11
Whooping cough	155

LOUISIANA

	Cases
Diphtheria	11
Lethargic encephalitis	3
Malaria	38
Pellagra	11
Pneumonia	8
Scarlet fever	7
Smallpox	10
Tuberculosis	33
Typhoid fever	22
Whooping cough	14

MAINE

Cerebrospinal meningitis	2
Chicken pox	1
German measles	14
Lethargic encephalitis	1
Measles	120
Mumps	10
Pellagra	1
Pneumonia	5
Scarlet fever	17
Tuberculosis	13
Typhoid fever	2
Whooping cough	34

MARYLAND [1]

Chicken pox	43
Diphtheria	17
Dysentery	4
German measles	2
Influenza	12
Lethargic encephalitis	1
Measles	123
Mumps	37
Paratyphoid fever	2
Pellagra	1
Pneumonia (all forms)	29
Scarlet fever	30
Tetanus	1
Tuberculosis	75
Typhoid fever	15
Whooping cough	69

MASSACHUSETTS

Cerebrospinal meningitis	3
Chicken pox	101
Conjunctivitis (suppurative)	10
Diphtheria	47
German measles	89
Influenza	2
Lethargic encephalitis	3
Measles	417
Mumps	97
Ophthalmia neonatorum	25
Pneumonia (lobar)	56
Poliomyelitis	1
Scarlet fever	241
Septic sore throat	1
Tetanus	1
Trichinosis	1
Tuberculosis (all forms)	166
Typhoid fever	3
Whooping cough	126

MICHIGAN

	Cases
Diphtheria	83
Measles	429
Pneumonia	34
Scarlet fever	186
Smallpox	5
Tuberculosis	285
Typhoid fever	3
Whooping cough	116

MINNESOTA

Cerebrospinal meningitis	1
Chicken pox	80
Diphtheria	32
Influenza	2
Measles	169
Pneumonia	2
Scarlet fever	115
Smallpox	1
Tuberculosis	73
Typhoid fever	5
Whooping cough	33

MISSISSIPPI

Diphtheria	10
Scarlet fever	2
Smallpox	1
Typhoid fever	37

MISSOURI

(Exclusive of Kansas City and St. Louis)

Chicken pox	7
Diphtheria	3
Influenza	1
Measles	53
Mumps	1
Rabies in animals	3
Scarlet fever	12
Smallpox	1
Trachoma	4
Tuberculosis	6
Typhoid fever	6
Whooping cough	14

MONTANA

Cerebrospinal meningitis	1
Chicken pox	7
Diphtheria	5
German measles	2
Measles	44
Mumps	3
Poliomyelitis	1
Rocky Mountain spotted fever—Rock Springs	1
Scarlet fever	26
Smallpox	7
Tuberculosis	2
Typhoid fever	2
Whooping cough	1

NEBRASKA

Chicken pox	11
Diphtheria	3
Measles	33
Mumps	1
Scarlet fever	22
Smallpox	30
Tuberculosis	5
Whooping cough	36

[1] Week ended Friday.

NEW JERSEY

	Cases
Chicken pox	109
Diphtheria	83
Influenza	2
Measles	389
Pneumonia	48
Scarlet fever	146
Smallpox	3
Trachoma	1
Typhoid fever	15
Whooping cough	73

NEW MEXICO

Chicken pox	4
Diphtheria	3
Malaria	1
Measles	6
Mumps	1
Rabies in animals	1
Scarlet fever	1
Tuberculosis	18
Typhoid fever	6
Whooping cough	13

NEW YORK

(Exclusive of New York City)

Cerebrospinal meningitis	1
Chicken pox	168
Diphtheria	69
German measles	222
Influenza	4
Malaria	4
Measles	1,597
Mumps	106
Ophthalmia neonatorum	1
Pneumonia	152
Poliomyelitis	6
Scarlet fever	119
Septic sore throat	1
Smallpox	6
Tetanus	1
Typhoid fever	8
Vincent's angina	10
Whooping cough	279

NORTH CAROLINA

Chicken pox	57
Diphtheria	16
German measles	53
Measles	350
Poliomyelitis	5
Scarlet fever	9
Smallpox	39
Typhoid fever	39
Whooping cough	355

OKLAHOMA

(Exclusive of Oklahoma City and Tulsa)

Cerebrospinal meningitis—Pittsburg	1
Chicken pox	6
Diphtheria	1
Influenza	10
Malaria	62
Measles	35
Mumps	5

OKLAHOMA—continued

	Cases
Pellagra	19
Pneumonia	7
Poliomyelitis—Atoka	1
Scarlet fever	8
Smallpox	8
Typhoid fever	58
Whooping cough	20

OREGON

Chicken pox	24
Diphtheria	17
Influenza	6
Measles	64
Mumps	10
Pneumonia	3
Rocky Mountain spotted fever	1
Scarlet fever	19
Septic sore throat	1
Smallpox:	
Lane County	16
Portland	24
Scattering	14
Tuberculosis	16
Typhoid fever	6
Whooping cough	29

PENNSYLVANIA

Cerebrospinal meningitis—Lebanon	1
Chicken pox	275
Diphtheria	128
German measles	36
Measles	1,567
Mumps	59
Ophthalmia neonatorum	1
Pneumonia	20
Poliomyelitis—Morgan Township	1
Rabies	1
Scarlet fever	341
Smallpox	6
Tuberculosis	91
Typhoid fever	22
Whooping cough	429

SOUTH DAKOTA

Chicken pox	6
Diphtheria	7
Measles	19
Mumps	5
Pneumonia	1
Scarlet fever	35
Smallpox	2
Tuberculosis	2
Typhoid fever	2
Whooping cough	20

TENNESSEE

Chicken pox	5
Diphtheria	3
Dysentery	4
Influenza	5
Malaria	42
Measles	114
Mumps	3
Ophthalmia neonatorum	1
Pellagra	11
Pneumonia	8

TENNESSEE—continued

	Cases
Scarlet fever	9
Smallpox:	
Hancock County	8
Scattering	13
Trachoma	2
Tuberculosis	51
Typhoid fever	59
Whooping cough	48

TEXAS

Chicken pox	20
Dengue	4
Diphtheria	8
Dysentery	3
Influenza	7
Measles	5
Mumps	29
Paratyphoid fever	1
Pellagra	6
Pneumonia	4
Poliomyelitis	5
Scarlet fever	7
Smallpox	38
Trachoma	1
Tuberculosis	28
Typhoid fever	15
Whooping cough	96

UTAH

Chicken pox	18
Diphtheria	7
German measles	1
Measles	22
Mumps	11
Pneumonia	5
Scarlet fever	1
Smallpox	3
Tuberculosis	1
Whooping cough	124

VERMONT

Chicken pox	7
Measles	24
Mumps	23
Poliomyelitis	1
Scarlet fever	1
Whooping cough	12

VIRGINIA

Poliomyelitis—Wythe County	1
Smallpox—Scott County	1

WASHINGTON

Cerebrospinal meningitis—Seattle	1
Chicken pox	38
Diphtheria	5
German measles	10

WASHINGTON—continued

	Cases
Measles	94
Mumps	21
Scarlet fever	24
Smallpox	25
Tuberculosis	29
Typhoid fever	4
Whooping cough	18

WEST VIRGINIA

Chicken pox	5
Diphtheria	2
Influenza	1
Measles	299
Pellagra	1
Scarlet fever	17
Smallpox	4
Tuberculosis	20
Typhoid fever	2
Whooping cough	33

WISCONSIN

Milwaukee;	
Chicken pox	48
Diphtheria	15
German measles	4
Lethargic encephalitis	1
Measles	170
Mumps	4
Pneumonia	10
Scarlet fever	12
Tuberculosis	10
Whooping cough	43
Scattering;	
Cerebrospinal meningitis	1
Chicken pox	47
Diphtheria	17
German measles	43
Influenza	8
Measles	858
Mumps	62
Pneumonia	13
Scarlet fever	54
Smallpox	2
Tuberculosis	23
Whooping cough	97

WYOMING

Cerebrospinal meningitis—Sheridan County	1
Chicken pox	3
Diphtheria	1
German measles	3
Measles	5
Rocky Mountain spotted fever—Natrona County	1
Scarlet fever	5
Smallpox	1
Typhoid fever	1
Whooping cough	5

Report for week ended June 26, 1926

NORTH DAKOTA

	Cases
Chicken pox	1
German measles	8
Measles	11
Mumps	5

NORTH DAKOTA—continued

	Cases
Scarlet fever	31
Smallpox	1
Whooping cough	9

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
March, 1926										
Pennsylvania	7	716	----	5	14,943	----	3	2,464	4	167
May, 1926										
Alabama	4	56	185	126	1,767	106	5	51	186	46
Delaware	0	10	----	1	194	----	0	37	0	0
Idaho	6	24	0	0	60	0	1	68	48	8
Kansas	0	37	66	1	2,527	1	1	214	77	10
Massachusetts	8	205	74	0	3,187	0	4	956	0	30
New York	24	940	514	12	13,794	----	7	1,933	9	83
Oregon	14	73	99	----	407	----	0	238	107	16
Rhode Island	0	30	0	----	474	----	0	36	0	2
South Carolina	0	79	1,627	830	237	534	4	62	80	99
South Dakota	0	19	6	----	283	----	2	417	11	0
Virginia	4	69	1,315	125	3,328	38	3	206	69	35
Washington	27	85	28	----	292	----	0	289	143	24

GENERAL CURRENT SUMMARY AND WEEKLY REPORTS FROM CITIES

Diphtheria.—For the week ended June 19, 1926, 36 States reported 871 cases of diphtheria. For the week ended June 20, 1925, the same States reported 972 cases of this disease. One hundred cities, situated in all parts of the country and having an aggregate population of more than 30,300,000, reported 656 cases of diphtheria for the week ended June 19, 1926. Last year for the corresponding week they reported 651 cases. The estimated expectancy for these cities was 784 cases. The estimated expectancy is based on the experience of the last nine years, excluding epidemics.

Measles.—Thirty-three States reported 10,314 cases of measles for the week ended June 19, 1926, and 4,183 cases of this disease for the week ended June 20, 1925. One hundred cities reported 4,257 cases of measles for the week this year and 2,390 cases last year.

Poliomyelitis.—The health officers of 36 States reported 19 cases of poliomyelitis for the week ended June 19, 1926. The same States reported 44 cases for the week ended June 20, 1925.

Scarlet fever.—Scarlet fever was reported for the week as follows: Thirty-six States—this year, 2,434 cases; last year, 1,639 cases; 100 cities—this year, 1,356 cases; last year, 910 cases; estimated expectancy, 672 cases.

Smallpox.—For the week ended June 19, 1926, 36 States reported 346 cases of smallpox. Last year for the corresponding week they reported 603 cases. One hundred cities reported smallpox for the week as follows: 1926, 65 cases; 1925, 193 cases; estimated expectancy, 92 cases. No deaths from smallpox were reported by these cities for the week this year.

Typhoid fever.—Three hundred and thirty-six cases of typhoid fever were reported for the week ended June 19, 1926, by 35 States. For the corresponding week of 1925 the same States reported 597 cases of this disease. One hundred cities reported 65 cases of typhoid fever for the week this year and 123 cases for the corresponding week last year. The estimated expectancy for these cities was 90 cases.

Influenza and pneumonia.—Deaths from influenza and pneumonia were reported for the week by 95 cities, with a population of more than 29,700,000, as follows: 1926, 538 deaths; 1925, 472.

City reports for week ended June 19, 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	0	1	0	0	0	21	0	8
New Hampshire:									
Concord	22,546	0	0	0	0	0	0	0	2
Manchester	83,097	0	1	0	0	0	18	0	1
Nashua	29,723	0	0	2	0	0	1	0	0
Vermont:									
Barre	10,008	0	0	0	0	0	0	0	0
Burlington	24,089	2	0	0	0	0	13	0	0
Massachusetts:									
Boston	779,620	32	48	24	0	1	93	72	9
Fall River	123,993	1	3	1	1	1	1	1	3
Springfield	142,065	5	2	0	0	0	7	0	1
Worcester	190,757	8	3	5	0	0	1	0	1
Rhode Island:									
Pawtucket	69,760	1	1	0	0	0	1	0	0
Providence	267,918	0	6	1	0	1	25	0	4
Connecticut:									
Bridgeport	(1)	7	4	0	1	1	4	0	3
Hartford	160,197	2	4	2	0	0	10	0	3
New Haven	178,927	6	2	0	0	0	46	2	3
MIDDLE ATLANTIC									
New York:									
Buffalo	538,016	0	10	4	0	0	17	8	14
New York	5,873,356	152	239	142	30	11	269	71	114
Rochester	316,786	4	6	12	0	3	35	1	5
Syracuse	182,003	9	5	1	0	0	336	14	4
New Jersey:									
Camden	128,642	0	4	3	0	0	15	1	1
Newark	452,513	37	12	7	0	0	65	17	6
Trenton	132,020	9	3	1	0	0	21	2	4
Pennsylvania:									
Philadelphia	1,979,364	79	58	73	----	3	181	8	27
Pittsburgh	631,563	42	18	4	----	2	208	5	15
Reading	112,707	11	3	3	----	0	28	0	0

[1] No estimate made.

City reports for week ended June 19, 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									
Ohio:									
Cincinnati	409,333	6	7	10	0	0	163	9	7
Cleveland	936,485	44	19	34	1	0	21	3	14
Columbus	279,836	9	2	13	0	1	37	0	3
Toledo	287,380	44	5	4	0	2	179	0	3
Indiana:									
Fort Wayne	97,846	4	1	1	0	0	41	0	2
Indianapolis	358,819	8	3	0	0	0	17	2	8
South Bend	80,091	3	1	0	0	0	41	0	0
Terre Haute	71,071	1	1	0	0	0	6	0	1
Illinois:									
Chicago	2,995,289	174	78	52	5	1	286	13	38
Peoria	81,564	1	1	0	0	0	6	1	1
Springfield	63,923	2	0	0	0	0	7	0	1
Michigan:									
Detroit	1,245,824	87	35	74	0	3	46	5	18
Flint	130,316	4	2	0	0	0	94	0	1
Grand Rapids	153,698	4	2	1	0	0	69	0	2
Wisconsin:									
Kenosha	50,891	9	1	0	0	0	117	0	2
Madison	46,385	16	1	0	0	0	31	0	2
Milwaukee	509,192	107	11	6	0	0	303	25	9
Racine	67,707	5	0	0	0	0	221	5	2
Superior	39,671	0	0	1	0	0	2	0	1
WEST NORTH CENTRAL									
Minnesota:									
Duluth	110,502	2	1	0	0	0	53	1	5
Minneapolis	425,435	71	12	25	0	0	22	0	10
St. Paul	246,001	18	13	7	0	0	242	2	7
Iowa:									
Davenport	52,469	0	1	0	0	----	3	0	----
Des Moines	141,441	0	1	2	0	----	0	0	----
Sioux City	76,411	1	1	0	0	----	0	0	----
Waterloo	36,771	1	0	1	0	----	45	0	----
Missouri:									
Kansas City	367,481	2	4	2	2	2	13	0	6
St. Joseph	78,342	0	1	0	0	0	4	0	0
St. Louis	821,543	7	31	46	0	0	210	4	----
North Dakota:									
Fargo	26,403	2	1	0	0	0	0	1	0
Grand Forks	14,811	----	0	----	----	----	----	----	----
South Dakota:									
Aberdeen	15,036	0	1	0	0	----	1	1	----
Sioux Falls	30,127	0	0	0	0	0	6	0	1
Nebraska:									
Omaha	211,768	10	2	2	0	0	30	1	6
Kansas:									
Topeka	55,411	8	1	0	0	0	4	0	0
Wichita	88,367	0	1	1	0	0	4	0	1
SOUTH ATLANTIC									
Delaware:									
Wilmington	122,049	1	1	3	0	0	4	0	3
Maryland:									
Baltimore	796,296	53	15	10	3	0	20	77	19
Cumberland	33,741	0	0	0	0	0	1	0	0
Frederick	12,035	0	0	0	0	0	0	0	0
District of Columbia:									
Washington	497,906	16	6	8	0	0	101	0	11
Virginia:									
Lynchburg	30,395	----	1	----	----	----	----	----	----
Norfolk	(1)	5	0	1	2	0	10	0	7
Richmond	186,403	3	1	5	0	1	120	4	3
Roanoke	58,208	1	1	0	0	0	10	0	0
West Virginia:									
Charleston	49,019	1	0	2	1	0	9	0	0
Huntington	63,485	0	0	0	0	0	0	0	2
Wheeling	56,208	6	0	0	0	0	47	0	0
North Carolina:									
Raleigh	30,371	4	0	0	0	0	1	0	0
Wilmington	37,061	3	1	0	0	0	1	0	0
Winston-Salem	69,031	4	0	3	0	0	44	3	0

[1] No estimate made.

City reports for week ended June 19, 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	0	1	0	7	0	1	0	1
Columbia	41,225	6	0	0	0	0	0	0	0
Greenville	27,311	1	0	1	0	1	0	0	0
Georgia:									
Atlanta	(1)	3	1	1	1	0	42	0	9
Brunswick	16,809	0	0	0	0	0	2	0	0
Savannah	93,134	0	1	1	1	0	0	1	1
Florida:									
Miami	69,754	0	-------	0	0	0	10	2	6
Tampa	94,743	1	1	0	0	0	2	0	5
EAST SOUTH CENTRAL									
Kentucky:									
Covington	58,309	1	1	0	0	0	3	0	1
Louisville	305,935	1	3	0	1	0	9	0	4
Tennessee:									
Memphis	174,533	2	1	1	0	1	49	3	4
Nashville	136,220	2	0	0	0	1	5	0	2
Alabama:									
Birmingham	205,670	2	0	2	1	1	63	1	8
Mobile	65,955	0	0	0	0	0	0	0	0
Montgomery	46,481	0	0	0	0	0	5	2	0
WEST SOUTH CENTRAL									
Arkansas:									
Fort Smith	31,643	0	1	0	0	-------	0	0	-------
Little Rock	74,216	1	0	1	0	0	13	0	0
Louisiana:									
New Orleans	414,493	0	5	3	2	4	2	0	7
Shreveport	57,857	0	0	1	0	0	1	0	0
Oklahoma:									
Oklahoma City	(1)	0	1	0	4	0	0	0	1
Texas:									
Dallas	194,450	11	2	2	0	1	1	1	0
Galveston	48,375	0	0	0	0	0	0	0	1
Houston	164,954	0	1	3	0	0	0	0	2
San Antonio	198,069	1	1	0	0	0	1	0	5
MOUNTAIN									
Montana:									
Billings	17,971	0	0	0	0	0	0	0	0
Great Falls	29,883	3	1	5	0	0	23	0	0
Helena	12,037	0	0	0	0	0	0	0	0
Missoula	12,668	0	0	1	0	0	0	1	1
Idaho:									
Boise	23,042	1	0	0	0	0	1	2	0
Colorado:									
Denver	280,911	28	10	6	0	-------	12	2	6
Pueblo	43,787	10	2	0	0	0	32	0	2
New Mexico:									
Albuquerque	21,000	0	1	1	0	0	1	1	0
Arizona:									
Phoenix	38,669	0	0	1	0	0	1	0	0
Utah:									
Salt Lake City	130,948	6	3	4	0	0	9	3	2
Nevada:									
Reno	12,665	0	0	0	0	0	0	0	0
PACIFIC									
Washington:									
Seattle	(1)	18	4	0	0	-------	28	27	-------
Spokane	108,897	28	2	2	0	-------	44	0	-------
Tacoma	104,455	-------	2	-------	-------	0	-------	-------	1
Oregon:									
Portland	282,383	7	4	6	0	1	36	5	7
California:									
Los Angeles	(1)	31	35	20	3	1	11	12	9
Sacramento	72,260	1	2	5	0	0	2	2	5
San Francisco	557,530	13	18	6	0	0	126	6	6

[1]No estimate made.

City reports for week ended June 19, 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	1	0	0	0	1	1	1	0	1	30
New Hampshire:											
Concord	0	2	0	0	0	2	0	0	0	0	7
Manchester	0	2	0	0	0	1	0	0	0	0	15
Nashua	0	1	0	0	0	0	0	0	0	0	7
Vermont:											
Barre	0	0	0	0	0	1	0	0	0	0	1
Burlington	0	0	0	0	0	0	0	0	0	1	8
Massachusetts:											
Boston	34	51	0	0	0	8	2	5	0	46	176
Fall River	2	0	0	0	0	3	2	0	0	1	32
Springfield	4	8	0	0	0	0	0	0	0	2	26
Worcester	5	10	0	0	0	5	0	0	0	2	55
Rhode Island:											
Pawtucket	1	0	0	0	0	0	0	0	0	0	11
Providence	4	1	0	0	0	1	1	1	0	3	62
Connecticut:											
Bridgeport	5	8	0	0	0	4	0	0	0	3	23
Hartford	3	0	0	0	0	2	0	0	0	2	33
New Haven	2	5	0	0	0	1	0	1	0	11	32
MIDDLE ATLANTIC											
New York:											
Buffalo	17	3	0	0	0	11	0	0	0	25	129
New York	130	233	0	0	0	[1] 132	14	15	1	66	1,278
Rochester	10	13	0	0	0	0	1	0	0	9	59
Syracuse	6	1	0	0	0	1	0	0	0	27	36
New Jersey:											
Camden	2	7	0	0	0	0	1	0	0	1	18
Newark	13	39	0	0	0	7	0	1	1	19	80
Trenton	1	5	0	0	0	3	0	1	0	0	29
Pennsylvania:											
Philadelphia	54	101	0	0	0	38	5	2	0	55	432
Pittsburgh	19	35	0	0	0	9	2	0	0	111	162
Reading	1	8	0	0	0	0	0	0	0	5	30
EAST NORTH CENTRAL											
Ohio:											
Cincinnati	6	13	2	3	0	12	1	1	0	11	128
Cleveland	15	61	2	0	0	10	2	0	0	66	197
Columbus	4	20	2	1	0	5	1	0	0	16	69
Toledo	9	10	1	0	0	4	1	1	0	42	67
Indiana:											
Fort Wayne	1	8	2	1	0	1	1	0	0	0	27
Indianapolis	7	12	6	9	0	10	1	0	0	27	107
South Bend	2	2	1	0	0	0	0	0	0	2	14
Terre Haute	2	4	1	0	0	0	0	0	0	2	20
Illinois:											
Chicago	72	110	2	1	0	52	3	2	1	50	683
Peoria	2	0	1	0	0	0	0	0	0	10	13
Springfield	1	4	1	0	0	1	0	1	0	9	13
Michigan:											
Detroit	49	112	5	0	0	25	3	0	0	56	306
Flint	3	16	1	0	0	2	1	0	0	6	18
Grand Rapids	3	13	0	0	0	2	0	0	0	10	27
Wisconsin:											
Kenosha	0	3	1	0	0	1	0	0	0	5	7
Madison	1	2	0	0	0	2	0	1	0	2	
Milwaukee	18	15	5	0	0	9	0	0	0	44	118
Racine	2	5	1	0	0	1	0	1	0	8	16
Superior	2	2	2	0	0	1	0	0	0	0	8

[1] Pulmonary tuberculosis only.

City reports for week ended June 19, 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											
Minnesota:											
Duluth	3	14	3	1	0	0	0	0	0	3	32
Minneapolis	20	61	8	0	0	2	1	0	0	2	101
St. Paul	13	38	3	0	0	3	0	0	0	26	54
Iowa:											
Davenport	0	0	3	0			0	0		0	
Des Moines	3	3	3	0			0	0			
Sioux City	1	9	2	3			0	0		3	
Waterloo	2	0	0	0			0	0		8	
Missouri:											
Kansas City	4	9	3	2	0	5	0	0	0	3	85
St. Joseph	0	0	0	1	0	1	0	0	0	0	25
St. Louis	18	63	2	0	0	13	3	4	0	48	185
North Dakota:											
Fargo	0	1	0	0	0	0	0	0	0	0	
Grand Forks	0		1				0				
South Dakota:											
Aberdeen	1	5	0	0			0	0		12	
Sioux Falls	0	1	0	0	0	0	0	0	0	7	8
Nebraska:											
Omaha	3	39	4	9	0	2	0	0	0	2	42
Kansas:											
Topeka	1	2	1	0	0	0	0	0	0	0	7
Wichita	1	4	4	0	0	0	0	1	0	6	22
SOUTH ATLANTIC											
Delaware:											
Wilmington	3	1	0	0	0	3	0	0	0	2	24
Maryland:											
Baltimore	16	36	0	0	0	14	3	1	0	56	192
Cumberland	0	1	0	0	0	0	0	0	0	0	5
Frederick	0	0	0	0	0	0	0	0	0	0	3
District of Col.:											
Washington	11	16	0	2	0	15	2	0	0	39	143
Virginia:											
Lynchburg	0		0				0				
Norfolk	1	1	0	0	0	1	0	0	0	23	
Richmond	1	5	0	1	0	4	1	0	0	0	50
Roanoke	1	1	0	1	0	0	1	0	0	0	14
West Virginia:											
Charleston	1	0	0	0	0	0	1	0	0	3	6
Huntington	0	0	1	0	0	0	0	0	0	0	15
Wheeling	2	2	0	0	0	0	0	0	0	1	16
North Carolina:											
Raleigh	0	0	0	3	0	0	0	0	0	16	13
Wilmington	0	0	0	0	0	1	0	1	0	13	11
Winston-Salem	0	1	1	0	0	1	1	0	0	2	15
South Carolina:											
Charleston	0	0	0	1	0	4	1	3	0	1	26
Columbia	0	0	0	0	0	0	1	3	0	0	
Greenville	0	0	0	0	0	0	1	1	0	4	10
Georgia:											
Atlanta	3	2	5	3	0	6	2	4	0	4	83
Brunswick	0	0	0	0	0	0	0	0	0	1	5
Savannah	1	0	0	1	0	2	2	0	0	0	30
Florida:											
Miami		1		0	0	0		0	0	7	37
Tampa	0	3	0	4	0	0	1	0	0	0	33
EAST SOUTH CENTRAL											
Kentucky:											
Covington	0	0	0	0	0	0	0	0	0	0	19
Louisville	3	5	0	0	0	7	1	2	0	2	72
Tennessee:											
Memphis	2	2	1	0	0	7	2	1	0	3	64
Nashville	1	0	1	1	0	3	2	0	0	2	38
Alabama:											
Birmingham	1	1	3	1	0	4	3	1	0	18	59
Mobile	0	0	1	0	0	2	1	0	0	0	24
Montgomery	0	1	0	0	0	0	1	0	0	2	

City reports for week ended June 19, 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	0	0	0	0			1	0		6	
Little Rock	0	7	0	1	0	3	1	0	0	0	
Louisiana:											
New Orleans	2	3	1	0	0	12	4	4	2	14	139
Shreveport	0	0	0	1	0	4	1	1	0	0	29
Oklahoma:											
Oklahoma City	1	0	4	0	0	0	1	2	0	0	25
Texas:											
Dallas	1	2	1	4	0	1	2	0	0	18	48
Galveston	0	0	0	0	0	1	0	0	0	0	17
Houston	0	1	1	0	0	3	1	0	0	0	58
San Antonio	0	3	0	0	0	5	1	2	2	0	67
MOUNTAIN											
Montana:											
Billings	1	1	0	0	0	0	0	0	0	0	6
Great Falls	2	1	1	0	0	0	1	0	0	0	5
Helena	0	0	0	0	0	0	0	0	0	0	2
Missoula	0	0	0	0	0	0	0	0	0	0	10
Idaho:											
Boise	1	0	0	2	0	0	0	0	0	0	4
Colorado:											
Denver	7	9	0	0	0	7	0	0	0	19	50
Pueblo	1	0	0	0	0	1	0	0	0	0	8
New Mexico:											
Albuquerque	0	0	0	1	0	3	0	0	0	9	15
Arizona:											
Phoenix	1	0	0	0	0	6	1	0	0	0	16
Utah:											
Salt Lake City	2	3	1	1	0	6	1	0	0	55	33
Nevada:											
Reno	0	0	0	0	0	0	0	0	0	0	2
PACIFIC											
Washington:											
Seattle	9	16	3	1			1	0		12	
Spokane	4	16	3	1			0	0		10	
Tacoma	2		2		0	1	0		0		16
Oregon:											
Portland	6	22	7	9	0	3	0	1	0	1	74
California:											
Los Angeles	16	38	3	3	0	16	2	1	0	10	182
Sacramento	0	1	1	2	0	1	0	1	0	1	28
San Francisco	11	6	2	0	0	11	1	1	1	4	169

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									
Connecticut:									
Hartford	1	1	0	0	0	0	0	0	0
MIDDLE ATLANTIC									
New York:									
New York [1]	1	1	5	0	0	0	0	1	0
Rochester	0	0	1	0	0	0	0	0	0
New Jersey:									
Camden	0	0	0	1	0	0	1	0	0
Newark	0	0	1	0	0	0	0	1	0

[1] Typhus fever, 1 case in New York City.

City reports for week ended June 19, 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
EAST NORTH CENTRAL									
Ohio:									
Columbus	0	0	0	1	0	0	0	0	0
Illinois:									
Chicag	1	1	0	0	0	1	1	1	0
Michigan:									
Detroit	2	0	0	0	0	1	0	0	0
Wisconsin:									
Milwaukee	1	1	0	0	0	0	0	0	0
WEST NORTH CENTRAL									
Minnesota:									
Minneapolis	1	0	0	0	0	0	0	0	0
St. Paul	0	0	1	0	0	0	0	0	0
SOUTH ATLANTIC									
Maryland:									
Baltimore	0	0	2	1	0	0	0	0	0
District of Columbia:									
Washington	0	0	0	0	1	1	0	0	0
Virginia:									
Norfolk	1	0	0	0	0	0	0	0	0
West Virginia:									
Huntington	0	0	0	0	0	0	0	0	1
North Carolina:									
Raleigh	0	1	0	0	0	0	0	0	0
South Carolina:									
Charleston [1]	0	0	0	0	1	1	0	0	0
Georgia:									
Atlanta	0	0	0	0	2	0	0	0	0
Savannah	0	0	0	0	0	3	0	0	0
Florida:									
Tampa	1	1	0	0	0	0	0	0	0
EAST SOUTH CENTRAL									
Alabama:									
Birmingham	0	0	1	0	1	1	0	0	0
Mobile	0	0	0	0	0	2	0	0	0
WEST SOUTH CENTRAL									
Arkansas:									
Little Rock	0	0	0	0	0	3	0	0	0
Louisiana:									
New Orleans	0	0	0	0	1	1	0	0	0
Shreveport	0	0	0	0	0	2	0	0	0
Texas:									
Dallas	0	0	0	0	0	2	0	1	1
Houston	0	0	0	1	0	0	0	0	0
MOUNTAIN									
Montana:									
Great Falls	1	0	0	0	0	0	0	0	0
Missoula	2	1	0	0	0	0	0	0	0
New Mexico:									
Albuquerque	0	0	0	0	1	0	0	0	0
PACIFIC									
Washington:									
Spokane	2	0	0	0	0	0	0	0	0
Oregon:									
Portland	1	2	0	0	0	0	0	0	0
California:									
Los Angeles	0	0	0	0	0	1	0	0	0
San Francisco	0	0	0	0	0	1	1	0	0

[1] Dengue, 2 cases at Charleston, S. C.

The following table gives the rates per 100,000 population for 103 cities for the five-week period ended June 19, 1926, compared with those for a like period ended June 20, 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 16 to June 19, 1926—Annual rates per 100,000 population—Compared with rates for the corresponding period of 1925 [1]

DIPHTHERIA CASE RATES

	Week ended									
	May 23, 1925	May 22, 1926	May 30, 1925	May 29, 1926	June 6, 1925	June 5, 1926	June 13, 1925	June 12, 1926	June 20, 1925	June 19, 1926
103 cities	148	[2] 117	[3] 144	[2] 122	[4] 152	[2] 117	116	[5] 138	114	[6] 113
New England	122	78	110	80	125	78	91	[7] 69	93	78
Middle Atlantic	202	138	210	145	243	134	155	155	166	124
East North Central	101	117	100	108	92	119	89	[8] 147	86	131
West North Central	243	[2] 145	187	[2] 163	183	[2] 207	141	[9] 289	129	[2] 167
South Atlantic	83	71	[3] 72	96	[4] 88	47	54	60	48	[10] 66
East South Central	37	36	11	42	11	16	11	26	5	16
West South Central	40	47	62	65	40	56	66	47	70	43
Mountain	129	127	139	127	74	109	176	127	185	146
Pacific	157	164	160	159	138	132	157	159	108	[11] 94

MEASLES CASE RATES

103 cities	579	[2] 1,434	[3] 569	[2] 1,283	[4] 594	[2] 1,014	558	[8] 864	416	[6] 372
New England	1,014	1,075	836	1,064	841	728	860	[7] 662	611	494
Middle Atlantic	615	1,133	701	956	771	751	724	797	542	585
East North Central	888	1,372	839	1,252	825	1,103	779	[8] 988	547	943
West North Central	233	[2] 3,437	137	[2] 3,061	111	[2] 2,209	131	[9] 1,552	84	[2] 1,260
South Atlantic	309	1,659	[3] 242	1,542	[4] 393	1,213	280	1,103	330	[10] 788
East South Central	310	2,999	200	2,376	121	1,660	194	1,396	105	695
West South Central	22	142	13	112	22	86	13	125	18	77
Mountain	176	1,384	240	1,302	37	1,247	92	919	74	701
Pacific	124	693	157	803	157	696	83	593	80	[11] 602

SCARLET FEVER CASE RATES

103 cities	297	[2] 309	[3] 267	[2] 274	[4] 256	[2] 231	170	[5] 256	159	[6] 233
New England	338	288	204	258	256	248	173	[7] 256	137	203
Middle Atlantic	264	256	270	212	262	209	155	195	144	221
East North Central	388	341	321	339	293	247	198	[8] 332	202	340
West North Central	539	[2] 721	514	[2] 695	466	[2] 416	315	[9] 673	317	[2] 480
South Atlantic	138	195	[3] 115	160	[4] 125	190	58	160	58	[10] 131
East South Central	226	176	168	171	116	195	147	78	147	47
West South Central	44	172	62	116	84	163	44	86	35	69
Mountain	314	173	398	100	324	218	268	118	139	127
Pacific	155	294	133	181	144	170	155	237	110	[11] 220

[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] Grand Forks, N. Dak., not included.
[3] Charleston, W. Va., not included.
[4] Wilmington, N. C., not included.
[5] Barre, Vt., Madison, Wis., St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[6] Grand Forks, N. Dak., Lynchburg, Va., and Tacoma, Wash., not included.
[7] Barre, Vt., not included.
[8] Madison, Wis., not included.
[9] St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[10] Lynchburg, Va., not included.
[11] Tacoma, Wash., not included.

Summary of weekly reports from cities, May 16 to June 19, 1926—Annual rates per 100,000 population—Compared with rates for the corresponding period of 1925—Continued

SMALLPOX CASE RATES

	Week ended									
	May 23, 1925	May 22, 1926	May 30, 1925	May 29, 1926	June 6, 1925	June 5, 1926	June 13, 1925	June 12, 1926	June 20, 1925	June 19, 1926
103 cities	58	[2] 18	[3] 47	[2] 19	[4] 45	[2] 15	36	[5] 17	35	[6] 11
New England	0	0	0	0	0	0	0	[7] 0	0	0
Middle Atlantic	2	0	2	1	4	0	2	0	1	0
East North Central	66	18	54	13	61	9	40	[8] 12	42	10
West North Central	66	[2] 28	68	[2] 44	92	[2] 40	50	[9] 34	58	[2] 32
South Atlantic	61	24	[3] 10	28	[4] 37	34	21	38	29	[10] 30
East South Central	404	62	389	62	105	83	273	52	184	10
West South Central	123	95	53	99	31	43	4	34	18	26
Mountain	28	18	55	36	37	27	28	46	18	27
Pacific	177	51	160	32	182	24	141	54	146	[11] 20

TYPHOID FEVER CASE RATES

103 cities	18	[2] 11	[3] 15	[2] 10	[4] 24	[2] 9	27	[5] 12	21	[6] 11
New England	24	9	17	7	29	0	24	[7] 17	19	19
Middle Atlantic	19	7	9	5	26	9	17	6	14	9
East North Central	5	5	7	9	9	5	9	[8] 4	6	4
West North Central	4	[2] 8	10	[2] 4	8	[2] 8	24	[9] 5	12	[2] 10
South Atlantic	36	32	[3] 39	26	[4] 39	32	61	26	46	[10] 25
East South Central	68	10	47	31	37	10	110	57	74	21
West South Central	62	26	62	13	84	9	110	52	123	30
Mountain	18	9	9	0	74	9	46	9	37	0
Pacific	6	19	8	11	8	8	14	13	6	[11] 9

INFLUENZA DEATH RATES

96 cities	14	15	[3] 12	12	[4] 10	8	7	[12] 10	6	[10] 7
New England	5	12	7	9	2	2	5	[7] 12	2	9
Middle Atlantic	11	16	9	11	11	6	6	9	4	9
East North Central	11	18	13	11	10	8	6	[8] 10	7	3
West North Central	17	8	17	13	4	8	8	[13] 3	6	4
South Atlantic	6	11	[3] 12	11	[4] 6	8	4	6	6	[10] 4
East South Central	79	36	37	26	47	36	16	36	32	16
West South Central	19	24	29	9	5	14	19	19	10	24
Mountain	18	0	0	9	28	18	9	9	0	0
Pacific	22	4	7	11	11	4	4	0	4	4

PNEUMONIA DEATH RATES

96 cities	123	141	[3] 119	[3] 120	[6] 123	[4] 105	99	[12] 95	78	[10] 87
New England	110	144	110	123	69	116	113	[7] 102	60	87
Middle Atlantic	143	173	145	145	167	130	130	109	93	95
East North Central	116	133	111	106	107	98	79	[8] 87	76	74
West North Central	76	94	57	83	55	50	57	[13] 48	32	75
South Atlantic	125	148	[3] 147	[3] 111	[4] 138	[3] 80	115	96	75	[10] 112
East South Central	126	171	158	171	116	125	58	125	95	99
West South Central	73	90	73	109	63	99	82	94	87	71
Mountain	166	82	74	91	92	146	102	82	139	100
Pacific	120	53	73	64	116	67	44	67	58	75

[2] Grand Forks, N. Dak., not included.
[3] Charleston, W. Va., not included.
[4] Wilmington, N. C., not included.
[5] Barre, Vt., Madison, Wis., St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[6] Grand Forks, N. Dak., Lynchburg, Va., and Tacoma, Wash., not included.
[7] Barre, Vt., not included.
[8] Madison, Wis., not included.
[9] St. Paul, Minn., Kansas City, Mo., Fargo, N. Dak., and Grand Forks, N. Dak., not included.
[10] Lynchburg, Va., not included.
[11] Tacoma, Wash., not included.
[12] Barre, Vt., Madison, Wis., St. Paul, Minn., Kansas City, Mo., and Fargo, N. Dak., not included.
[13] St. Paul, Minn., Kansas City, Mo., and Fargo, N. Dak., 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

THE FAR EAST

Report for week ended June 5, 1926.—The following report for the week ended June 5, 1926, was transmitted by the far eastern bureau of the health section of the League of Nations' Secretariat, located at Singapore, to the headquarters at Geneva.

Maritime towns	Plague		Cholera		Smallpox	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Iraq: Basrah	0	0	0	0	6	5
Ceylon: Colombo	1	1	0	0	0	0
British India:						
Bombay	-------	4	-------	0	30	17
Madras	-------	0	-------	1	0	0
Karachi	-------	3	-------	0	5	2
Negapatam	-------	0	-------	2	1	1
Vizagapatam	-------	0	-------	0	1	0
Siam: Bangkok	0	0	146	60	4	5
French Indo-China:						
Saigon and Cholon	4	2	29	22	0	0
Haiphong	0	0	58	51	0	0
Hongkong	0	0	0	0	2	1
China:						
Shanghai	0	0	1	0	0	0
Amoy	11	-------	0	0	1	0
Japan: Simonoseki	0	0	0	0	1	0
Kwantung: Dairen	0	0	0	0	6	2

Telegraphic reports from the following maritime towns indicated that no case of plague, cholera, or smallpox was reported during the week:

ASIA

British India.—Chittagong, Cochin, Tuticorin.

Federated Malay States.—Port Swettenham.

Straits Settlements.—Penang, Singapore.

Dutch East Indies.—Batavia, Surabaya, Samarang, Cheribon, Belawan Deli, Palembang, Sabang, Makassar, Menado, Banjermasin, Balik-Papan, Tarakan, Pontianak, Padang.

Sarawak.—Kuching.

British North Borneo.—Sandakan.

Portuguese Timor.—Dilly.

Philippine Islands.—Manila, Iloilo, Jolo, Cebu, Zamboanga.

French Indo-China.—Turane.

Formosa.—Keelung.

Japan.—Nagasaki, Yokohama, Osaka, Moji, Kobe, Niigata, Tsuruga, Hakodate.

Korea.—Chemulpo, Fusan.
Kwantung.—Port Arthur.
Manchuria.—Antung, Mukden, Changchun, Harbin.
U. S. S. R.—Vladivostok.

AUSTRALASIA AND OCEANIA

Australia.—Adelaide, Melbourne, Sydney, Brisbane, Rockhampton, Townsville, Port Darwin, Broome, Fremantle, Carnarvon, Thursday Island.
New Guinea.—Port Moresby.
New Zealand.—Auckland, Wellington, Christchurch, Invercargill, Dunedin.
New Caledonia.—Noumea.
Hawaii.—Honolulu.

AFRICA

Egypt.—Alexandria, Port Said, Suez.
Anglo-Egyptian Sudan.—Port Sudan.
Eritrea.—Massaua.
French Somaliland.—Jibuti.
British Somaliland.—Berbera.
Italian Somaliland.—Magadiscio.
Kenya.—Mombasa.
Tanganyika.—Dar-es-Salaam.
Seychelles.—Victoria.
Mauritius.—Port Louis.
Madagascar.—Tamatave, Majunga.
Zanzibar.—Zanzibar.
Portuguese East Africa.—Mozambique, Beira, Lorenço Marques.
Union of South Africa.—Durban, East London, Port Elizabeth, Cape Town.

Reports had not been received in time for distribution from:

British India.—Rangoon, Calcutta.

CHINA

Smallpox—Typhus fever—Chungking district—Under date of May 1, 1926, smallpox and typhus fever were reported among the military at Wanhsein, in the consular district of Chungking, China. A death from typhus fever was reported at Ichang, in a member of the foreign population.

GREAT BRITAIN (SCOTLAND)

Prevalence of certain infectious diseases—Glasgow—April 25–May 29, 1926.—During the five-week period ended May 29, 1926, 515 cases of measles with 34 deaths were reported at Glasgow,[1] Scotland; diphtheria including membranous croup and scarlet fever were reported prevalent with 215 cases and 373 cases, respectively; lethargic encephalitis acute, with 11 cases; tuberculosis (pulmonary), 220 cases; other forms of tuberculosis, 146 cases. Population, estimated, 1,034,500.

[1] Public Health Reports, Jan. 22, 1926, p. 154; Apr. 2, 1926, p. 639; May 7, 1926, p. 910; June 18, 1926, p. 1258.

GREECE

Plague—Patras—May 27, 1926.—Two cases of plague, one of which terminated fatally, were reported at Patras, Greece, May 27, 1926. The cases occurred in the same family and in a quarter of the city not far from the customhouse and the port.

INDIA

Plague-infected rats—Rangoon—January-April, 1926.—During the four-month period, January to April, 1926, inclusive, 57 plague-infected rats were reported found at Rangoon, India. During the same period, 140 cases of plague with 129 deaths were reported at Rangoon, of which 12 cases were stated to have been imported.

LATVIA

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

April, 1926

Disease	Cases	Disease	Cases
Diphtheria	68	Rabies	2
Dysentery	4	Scarlet fever	306
Lethargic encephalitis	1	Smallpox	3
Malaria	1	Tetanus	2
Measles	184	Tuberculosis	61
Mumps	40	Typhoid fever	47
Paratyphoid fever	4	Whooping cough	56
Puerperal fever	7		

Population estimated, 1,850,000.

MADAGASCAR

Plague—April 1–15, 1926.—During the period April 1 to 15, 1926, 42 cases of plague with 39 deaths were reported in the Island of Madagascar. The types of the disease were: Bubonic, with 14 cases; pneumonic, 18 cases; septicemic, 10 cases. For distribution of occurrence, according to locality, see page 1458.

MALTA

Communicable diseases—May 1–31, 1926.—During May, 1926, communicable diseases were reported in the Island of Malta as follows: Bronchopneumonia, 7 cases; chicken pox, 45; diphtheria, 7; erysipelas, 6; influenza, 4; lethargic encephalitis, 3; Malta (undulant) fever, 41; measles, 96; pneumonia, 20; scarlet fever, 1; trachoma, 80; tuberculosis, 20; typhoid fever, 20; whooping cough, 12. Population, civil, estimated, 223,088.

MEXICO

Gastroenteritis—Chihuahua.—During the period May 25 to June 13, 1926, gastroenteritis was reported present at Chihuahua, Mexico. The water supply of the city was stated to be unfiltered.

Smallpox—San Antonio de Arenales.—During the period January to June, 1926, smallpox was reported present, with a number of cases, in a community settlement at San Antonio de Arenales, situated about 100 miles distant from Chihuahua.

TUNISIA

Plague—Kairouan and vicinity.—Under date of June 9, 1926, 12 cases of plague with 8 deaths were reported in Tunisia. Of these, three cases occurred in the city of Kairouan and nine cases in a territory 30 miles south of Kairouan. It was stated that a sanitary cordon had been established and that an intensive campaign of anti-plague inoculation was being carried out.

UNION OF SOUTH AFRICA

Plague—Cape Province—Orange Free State—May 9–15, 1926.—During the week ended May 15, 1926, plague was reported in the Union of South Africa as follows: *Cape Province*—Three European cases, of which two ended fatally, occurring at a farm in the Middelburg District; *Orange Free State*—one fatal case, native, at Protestpan, 15 miles distant from Bultfontein.

Typhus fever—April, 1926.—During the month of April, 1926, 85 cases of typhus fever, with 14 deaths, occurring in the colored population, and 2 cases in the European population, were reported in the Union of South Africa. For distribution of occurrence, according to province, see page 1459.

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 9, 1926 [1]

CHOLERA

Place	Date	Cases	Deaths	Remarks
India				Apr. 25–May 1, 1926: Cases, 2,880 deaths, 1,943.
Calcutta	May 9–22	112	103	
Madras	May 16–22	1		
Rangoon	May 9–15	5	1	
Siam:				
Bangkok	do	327	173	

[1] From medical officers of the Public Health Service, American consuls, and other sources.

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

Reports Received During Week Ended July 9, 1926—Continued

PLAGUE

Place	Date	Cases	Deaths	Remarks
Greece:				
Patras	May 27	2	1	In same family; locality vicinity of port.
India				Apr. 25–May 1, 1926: Cases, 7,671; deaths, 1,764.
Bombay	May 9–15	4	3	
Karachi	May 23–29	1	1	
Madras Presidency	Apr. 25–May 1	16	13	
Rangoon	May 9–15	4	3	Plague-infected rats; January–April, 1926: 57.
Madagascar				Apr. 1–15, 1926: Cases, 42; deaths, 39.
Moramanga Province	Apr. 1–15	2	2	Septicemic.
Tananarive Province—				
Tananarive Town	do	3	3	Pneumonic and septicemic.
Other localities	do	37	34	Bubonic, pneumonic, septicemic.
Tunisia:				
Kairouan	June 9	3		In territory 30 miles south of Kairouan, 9 cases.
Union of South Africa:				
Cape Province—				
Middelburg District	May 9–15	3	2	Occurring on farm. In Europeans.
Orange Free State—				
Protestpan	do	1	1	In native. Locality 15 miles from Bultfontein.

SMALLPOX

Brazil:				
Rio de Janeiro	May 16–22	31	13	
China:				
Dairen	Apr. 26–May 9	31	6	
Harbin	May 14–27	14		
Shanghai	May 16–22	5	19	Cases, foreign; deaths, foreign and Chinese, in International Settlement and French Concession.
Wanshein	May 1			Locality, Chungking consular district. Present among troops.
Egypt:				
Alexandria	May 15–21	5		
Great Britain:				
England—				
Newcastle-on-Tyne	June 6–12	1		
India				Apr. 25–May 1, 1926: Cases, 6,675; deaths, 1,719.
Bombay	May 9–15	32	12	
Calcutta	May 9–22	32	22	
Karachi	May 16–29	24	8	
Madras	May 16–29	5	3	
Rangoon	May 9–15	2	1	
Iraq:				
Basra	May 9–22	11	9	
Java:				
East Java and Madoera	Apr. 18–May 1	9		
Latvia	Apr. 1–30	3		
Mexico:				
Mexico City	May 30–June 5	1		Including municipalities in Federal District.
San Antonio de Arenales	Jan. 1–June 30			Present in community settlement 100 miles distant from Chihuahua.
Poland				Apr. 11–May 1, 1926: Cases, 7.
Portugal:				
Lisbon	Apr. 26–May 23		3	
Oporto	May 31–June 5	1		
Siam:				
Bangkok	May 9–15	1	1	
Straits Settlements:				
Singapore	Apr. 25–May 1	1		

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

Reports Received During Week Ended July 9, 1926—Continued

TYPHUS FEVER

Place	Date	Cases	Deaths	Remarks
China:				
Ichang			1	Reported May 1, 1926. Occurring among troops.
Wanhsien				Present among troops, May 1, 1926. Locality in Chungking consular district.
Mexico:				
Mexico City	May 31-June 5	11		Including municipalities in Federal District.
Poland				Apr. 5-May 1, 1926: Cases, 382; deaths, 30.
Union of South Africa				April, 1926: Cases, 85, deaths, 14 (colored); European, 2 cases: Total, 87 cases, 14 deaths.
Cape Province				Apr. 1-30, 1926: Cases, 71; deaths, 11. Native.
Do	May 9-15			Outbreaks.
Grahamstown	do	1		Sporadic.
Natal				Apr. 1-30, 1926: Cases, 4. Native.
Orange Free State				Apr. 1-30, 1926: Cases, 7. Native.
Transvaal				Apr. 1-30, 1926: Cases, 3; deaths, 3. Native.

Reports Received from June 26 to 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.
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	

[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 from June 26 to July 2, 1926—Continued

SMALLPOX

Place	Date	Cases	Deaths	Remarks
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 30-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.
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		

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

Reports Received from June 26 to July 2, 1926—Continued

TYPHUS FEVER—Continued

Place	Date	Cases	Deaths	Remarks
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	
Poland				Mar. 28–Apr. 10, 1926: Cases, 191; deaths, 18.

YELLOW FEVER

Brazil:				
Bahia	May 9–22	3	2	