PUBLIC HEALTH REPORTS

VOL. 50

APRIL 19, 1935

No. 16

STUDIES ON THE ORIGIN OF A NEWLY DISCOVERED VIRUS WHICH CAUSES LYMPHOCYTIC CHORIOMENINGITIS IN EXPERIMENTAL ANIMALS

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In an earlier communication Armstrong and Lillie (1) described a previously unidentified virus which was encountered during the transmission, in monkeys, of infectious material from an individual who died at St. Louis during the 1933 encephalitis epidemic.

Two additional strains of virus similar clinically, pathologically, and immunologically in experimental animals to this earlier-described virus have since been isolated at the National Institute of Health. The second strain was encountered during attempts to transmit experimental infection from the brain of an individual (A. O.) who died in Maine of a peculiar clinical type of encephalitis.

The patient, A. O, white female, 46 years of age, married, had onset of illness on September 27, 1934, with severe headache and chills. The temperature was 104.6° F., and the patient was delirious.

On September 28, the temperature was 100.° F.; the patient was mentally upset, and her neck was stiff. Spinal fluid showed increased pressure, 200 cells, mainly lymphocytes. There was no increase in globulin or sugar.

On September 29, the spinal fluid was bloody, sterile to culture.

Two blood counts made during the illness gave 18,000 W. B. C. each.

Death occurred on September 30.

The brain frozen in dry ice was received at the National Institute of Health on October 2, 1934. Transfers from the interior of the brain gave a pure culture of staphylococci and in stained sections cocci distributed throughout the brain tissue were seen by Surg. R. D. Lillie. No negri bodies were found.

¹ This virus is distinct from that isolated by Muckenfuss, Armstrong, and McCordock (2), and by Webster and Fite (3), which has been shown to be the causative agent of the St. Louis type of encephalitis.

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Rabbits, guinea pigs, mice, and monkeys were inoculated intracerebrally with material from the deeper portions of the brain emulsified in saline. The majority of these inoculated animals died of purulent encephalitis. A monkey died on the eleventh day, and the pathological report by Surgeon Lillie stated that lymphocytic choriomeningitis was present. Material from the noninoculated cortex of the dead monkey when transferred to fresh monkeys resulted in symptoms suggestive of experimental lymphocytic choriomeningitis. The infection was conveyed to mice and the virus identified as similar to the original strain. There were, however, some qualitative differences; for example, this second strain appeared to be more virulent for monkeys but less so for mice than was the original strain.

The third strain was recovered from a monkey inoculated with the virus of poliomyelitis (monkey strain) of which it died. The animal, however, showed pathological lesions suggestive of choriomeningitis, and the virus was recovered through the inoculation of organ emulsions into fresh monkeys and thence to mice. The recovery of this third strain indicates that the virus was present among our experimental animals and throws doubt upon the human origin of the earlier strains, although we cannot be certain that the virus may not originally have been introduced among our stock animals through inoculations with human material.

As a further check upon the spontaneous occurrence of the disease among our stock monkeys, serum-virus protection tests through the intracerebral inoculation of white mice were carried out.

PROTECTION TESTS ON MONKEY SERA

Sera from 44 monkeys which had never been experimentally inoculated with this virus were tested and no protective antibodies were demonstrable in 39 of them. There were 5, however, whose sera possessed moderate to strong neutralizing properties. On the other hand, the sera of 13 animals which had recovered from a clinical attack following inoculation with the virus showed strong protective Thus it appears that immunity, presumably the result properties. of spontaneous infection, was present among our monkeys (5 of 44) bled during the first 3 months of 1935. This conclusion is further supported by the fact that among 51 monkeys inoculated for the first time with a strain of our virus, by various routes, there were 3 in which no febrile or recognizable response occurred. Serum from one of these animals was later tested and found to possess highly potent antibodies. The remaining 48 of the 51 inoculated monkeys reacted with fever and symptoms, and in many instances the virus was recovered from the blood or spinal fluid or the disease was verified pathologically.

Individual white mice, likewise, were not infrequently encountered which withstood intracerebral doses of virus, a fraction of which usually killed the majority of mice in from 6 to 8 days. Whether such resistance is the result of a natural variation in the mice or of a specific immunity following spontaneous infection with the virus is not clear; however, the evidence in the case of monkeys suggests the latter alternative.

PROTECTION TESTS ON HUMAN SERA

Since the virus readily, even spontaneously, infects monkeys, since 2 of our 3 strains may have originated in human sources, and since the experimental disease in monkeys, as previously pointed out, (1) has marked resemblances to the human ailment designated as "lymphocytic" or "aseptic" meningitis, the search for specific antibodies in human sera is of extreme interest. Sera from 166 persons were, therefore, submitted to the protection test against one or more of our 3 strains of virus (protocol I). The sera examined were from normal persons, from those recovered from the St. Louis type of encephalitis, poliomyelitis, and other types of central nervous involvement. Among these 166 sera there were 155 in which no protective antibodies could be demonstrated, while in 8 instances questionable protective properties were indicated. Three additional human sera were encountered which gave, on repeated tests, a high degree of protection, equal to that observed in the sera of our experimentally immunized monkeys, and which are therefore of special interest.

Sera (2 parts)	Dilution virus (1 part)	Day of death after inocu- lation	Number of mice that survived
Immune monkey 811	1:500 1:3,333	3	3 4
Nonimmune monkey 871	1:16,666 1:500 1:3,333	7, 8, 8, 8 8, 9, 10	4 0 1
Immune person, L. O. P	1:16,666 1:500 1:3,333	10, 12	2 4 4
Nonimmune person, E. W	1:16,666 1:500 1:3,333	11 8, 9, 9, 11 9, 9, 9	3 0 1
	1:16,666	3	3

Sample protocol I. Serum-virus protection test. Ex. 45

The serum from one of these cases (M. T.) also possessed antibodies against the Freeman strain of encephalitis virus (St. Louis type), she having suffered an attack of that disease during the Illinois outbreak of 1934.²

M. T., white female, age 20, single, seamstress; parents and two brothers living and well. Past history: Measles, chicken pox, and

² The writers are indebted to Dr. S. C. Crispin, of Danville, Ill., and to Dr. W. H. Tucker, assistant epidemiologist of the Illinois State Department of Health, for supplying us with the history of the case and the serum from it.

whooping cough as a child; tonsils removed 1924. No other serious illness prior to encephalitis, which began on August 29, 1934, with severe chills and headache, fever 103° F., pain in neck, nausea, vomiting, and constipation.

Physical findings: September 1, 1934, neck rigid, abdominal reflexes absent, deep reflexes slightly exaggerated, drowsy but easily aroused. Spinal fluid clear, moderately increased pressure, 62 cells, 52 percent polys., 48 percent lymphocytes. Sugar, 82 mg. No organisms seen on smear. Highest temperature 103.6° F. on fifth day of illness. Blood count on September 1 showed 15,800 W. B. C., 81 percent polys., 15 percent small lymphocytes, and 4 percent large lymphocytes. Temperature normal on eighth day. Clinical diagnosis, encephalitis.

Blood drawn on October 24, 1934, gave strong protection against strains of our virus as well as against the Freeman strain of encephalitis virus. Sera from the father, mother, and one brother collected March 1, 1935, failed to show protective antibodies, while the patient's serum collected at the same time again gave strong protection against the choriomeningitis virus.

The second individual (L. O. P.) whose serum showed the presence of potent neutralizing antibodies was an attendant at the National Institute of Health who was engaged in various work and who occasionally handled infected monkeys. Four other persons who were more constantly exposed to infected monkeys, however, showed no demonstrable protective antibodies in their sera.

L. O. P., colored, male, 38, married, was not clear as to his child-hood infections. He was operated upon for appendicitis in 1919, but otherwise denies serious illness. He came to the laboratory in 1931, and his sickness record here reveals an occasional illness of a day or two, usually attributed to a headache. In January 1934 he was absent for 4 days with "grippe," and in October 1934 he had his tonsils removed. There was no history suggestive of central nervous involvement.

This case suggests that immunity may develop in the absence of recognizable central nervous system involvement, possibly the result of a subclinical infection. On the other hand, we have shown that, in exprimental animals, the virus is widely distributed throughout various organs, i. e., there is no marked neurotropism, and it is conceivable that immunity may result from systemic infection without involvement of the central nervous system.

The third serum to show the presence of potent protective antibodies against the choriomeningitis virus was from a patient (L. P.) with clinically typical lymphocytic aseptic meningitis, living in Virginia.

³ The writers are indebted to Dr. W. A. Bloedorn, of Washington, D. C., and to Lieut. Commander P. F. Dickens, Medical Corps, U. S. Navy, for supplying us with the clinical and laboratory findings and the serum from this case.

L. P., white male adult, seen by Dr. W. A. Bloedorn on April 2, 1934, temperature 101.2° F., coryza, nausea, and vomiting; photophobia and slight lethargy, neck stiff, Kernig positive. Spinal tap gave clear to hazy fluid under slightly increased pressure. Laboratory studies by Dr. P. F. Dickens revealed 1260 lymphocytes, 4 polys., and 20 red blood cells. Kahn, Wassermann, and gold chloride tests negative. Chlorides 710 milligrams per 100 cc. Culture negative. W. B. C., 11,000; 76 percent polys. Uneventful recovery.

Blood collected for serum-virus neutralization test on March 5, 1935 (11 months after attack) gave strong protection against strains of our virus.

SUMMARY

- 1. The isolation of three similar strains of a newly described virus is reported.
- 2. Spontaneous infection among our stock monkeys has been demonstrated by the isolation of the virus from a noninoculated monkey and by the demonstration of specific antibodies in the sera of 5 out of 44 such animals.
- 3. The possibility that the virus may affect man is suggested, since two of our recovered strains are possibly of human origin. The ready and even spontaneous infection of monkeys with the virus, together with the fact that human sera (3 from 166) possessing potent specific antibodies for the virus have been encountered, points in the same direction.
- 4. As previously noted (1), the disease in monkeys resembles the human ailment designated as lymphocytic or aseptic meningitis, and serum collected from a person 11 months following a clinical attack of this disease gave strong protection against strains of our experimental virus. The finding of immunity in the serum of an exposed individual giving no history suggesting this disease, however, indicates that immunity may develop in the absence of central nervous symptoms.

REFERENCES

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- (2) Muckenfuss, R. S., Armstrong, Chas., and McCordock, H. A.: Studies on the experimental transmission of encephalitis. Pub. Health Rep., 48 (1933): 1341-1343.
- (3) Webster, L. T., and Fite, G. L.: A virus encountered in the studies of material from cases of encephalitis in St. Louis and Kansas City epidemic of 1933. Science, 78 (1933): 663-665.

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INFECTIOUS ENCEPHALITIS

The United States Public Health Service has recently issued a publication 1 which comprises the reports of various investigators and presents the contribution of St. Louis to the knowledge of a comparatively new type of encephalitis. It is now generally recognized that the disease of 1933 was a distinct type of infectious encephalitis; and this report illustrates the fact that within a year or two of its recognition, an amount of information was obtained comparable to that achieved regarding poliomyelitis during several decades. This measure of success, it is believed, was due to the cooperative endeavor of workers from various official and research institutions concerned, including the health services of the city, universities, State, and Nation.

The formation of a metropolitan health council for current and prompt interchange of information regarding the epidemic was a notable feature of the handling of the situation. Epidemics generally know no sharp administrative boundaries, and this council therefore comprised the local health organizations of all neighboring Missouri and Illinois municipalities. Although the bulletin describes chiefly the historical, epidemiological, experimental, bacteriological, pathological, and clinical phases of the epidemic, a note is made of two important features which are not to be neglected: The toll of human suffering which such an epidemic causes, and the faithful care of the nurses and physicians who ministered to the sick.

The encephalitis epidemic in St. Louis in 1933 showed, in the mass, clinical differences from the better known type of encephalitis commonly called "lethargic encephalitis" or "sleeping sickness." In cases of the St. Louis encephalitis the onset was more abrupt and the fever was higher than in the disease prevalent since the World War; paralysis of the eye muscles was rare, and serious progressive after effects were notably lacking, recovery usually being prompt and complete, in contrast to the older disease. In St. Louis there was also more uniformly evidence of a mild meningeal disturbance. The classification of the different forms of encephalitis which come into question is given in the bulletin as follows:

I. Infectious encephalitis:

1. Type Λ , or Economo or lethargic type, chiefly sporadic.

2. Type B, chiefly epidemic.

(a) Japanese form.(b) St. Louis form.

3. Other types, possibly the Australian.

II. Post- or para-infectious encephalitis, chiefly seen following measles, smallpox, vaccinia, or chicken pox.

It is thus believed that the St. Louis disease was a new entity and led to an extensive epidemic of encephalitis for the first time on the

¹ Public Health Bulletin No. 214.

Western Hemisphere. A small outbreak, almost exactly similar, was reported by the Illinois State Department of Health in 1932 in the eastern part of Illinois and was restudied in connection with the St. Louis disease. Aside from this one prior incident, epidemics in Japan, particularly in 1924 and 1929, afford the the closest parallels to the St. Louis outbreak.

The dates of onset formed a sharp peak in the last week of August, extending their upward and downward slopes hardly more than a month on either side of this period. There were 575 cases in St. Louis city, with a population of 836,979, and 520 in St. Louis County, with a population of 244,850. The fatality rate was 22.5 percent in the city and 17.5 percent in the county. The incubation period had a possible range of from 4 to 21 days. No predisposition or immunity was detected as to sex, race, or economic status; but there was a very striking increase in the incidence of the disease with age, from 54 cases per 100,000 population under 10 years old, to 419 cases per 100,000 population 80 to 89 years old. No other known infectious disease shows such a regular progression from the lowest to the highest age groups. This peculiarity was also characteristic of the 1932 Illinois outbreak and the two large Japanese outbreaks. There was also a distinct tendency for the disease to be more fatal in the higher age groups, with a case fatality rate of 80 percent in those over 80 years old, and less than 10 percent in all under 50 years.

In addition to the St. Louis area there were 3 foci in 1933 to the east of St. Louis and 3 to the west, 2 of the former being in Illinois and the third in Louisville, Ky. Those to the west were in Columbia, Kansas City, and St. Joseph, Mo. In all places where the disease has appeared there was a notable freedom from multiple cases in the same family, or from other obvious contagion between cases. One striking feature of the epidemic was a progressive increase in the rate of incidence with distance from the older parts of the city—from 31 cases per 100,000 population in the river wards to 142 in the outlying western sections of the city, and 212 in the county.

Comparison of the relative numbers of patients using different water supplies and milk supplies readily eliminated these two factors from consideration as important vehicles for the spread of the infection. The possibility of an insect vector, particularly the mosquito, was, on the other hand, not so easily eliminated; but prolonged and repeated attempts to transmit the disease to susceptible animals by mosquitos were unavailing, and human experiments conducted at two prisons far outside the epidemic area were likewise negative.

The successful transmission of the disease to animals (monkeys and mice only out of all the different laboratory animals tried), with the consequent proof that this disease was due to a specific filterable

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virus different from the viruses causing other known disease, was the most striking positive result of the work accomplished. Attempts were made to transmit the disease to monkeys from the brains in 15 fatal human cases, and in 7 of these successful transmission was obtained, the first symptoms appearing from 8 to 15 days after the first inoculation. Three of these strains of virus were also established in mice; Dr. Webster, of the Rockefeller Institute of Medical Research, was the first to inoculate mice successfully with material sent him from St. Louis. It is of great significance that mouse experiments were successful not only by inoculating the virus in the brain, but also merely by dropping it into the nostril.

The blood serum of human patients convalescent from the disease had the power of neutralizing the virus. This neutralizing power is not found in serum from other types of encephalitis (showing that this is a new disease), though studies completed since those reported in this bulletin show neutralization in a small proportion of the serum obtained from other localities and in a slightly larger proportion of serum obtained from persons in St. Louis who had no known infection with the disease. In other words, the virus was probably spread through a considerably larger proportion of the population than merely the patients who suffered with obvious attacks of the disease. When the virus has become established in mice this species is much more readily infected than monkeys, susceptibility being practically 100 percent by the nose or (in dilutions up to 1:1,000,000) by injections into the cranium. The virus at ordinary temperatures outside the body rapidly loses its power to infect.

The study of the role which streptococci might play in the causation of the disease was important. Streptococci producing green colonies were obtained rather readily from the nose and throat of encephalitis patients, also from normal people, and such streptococci produced changes in the brain when introduced into the cranium of rabbits. At first sight these changes might be suggestive of the human disease, but consideration of the incubation period and the details of the symptoms and changes showed that they were really different from those found in human encephalitis and, further, the symptoms and changes caused by streptococci from encephalitis patients were similar to those caused by streptococci from normal people. Other studies with serum and the cultures also revealed no relation between these germs and the causation of the human disease.

The pathological studies were based on 63 autopsies which showed as the essential pathological process in the disease an acute nonpurulent inflammation of the central nervous system, characterized by intense congestion of the blood vessels with minute hemorrhages, inflammation both of the nervous system itself and the envel-

oping meningeal membranes with various types of mononuclear cells, and evidence of toxic degeneration in the nerve cells. The differences in the pathology of this disease from that of the old form of infectious encephalitis (Economo or Type A) are as follows:

- 1. The meninges show more intense infiltration with mononuclear cells than is usually found in the lethargic type.
- 2. The inflammatory foci are more widespread throughout the brain, often occurring in great numbers in the cerebral cortex, and are not restricted to the midbrain or basal ganglia.
- 3. Degenerative changes in the nerve cells are more frequent and neuronophagia is more marked.
- 4. The nerve cells in the nuclei of the cranial nerves, especially the oculomotor, rarely show degenerative changes.
 - 5. There is more extensive involvement of the spinal cord.

The milder cases of the St. Louis type, however, could not be certainly differentiated from the lethargic type in pathology. The description of the pathology of the Japanese cases coincides with the severe examples of the St. Louis type.

In St. Louis the spinal fluid showed, as a rule, increased pressure and increased protein content, with a cell count somewhat elevated, 40 to 80 per cubic millimeter being the commonest range. These were chiefly lymphocytes. The spinal fluid sugar was usually below 70 mm per 100 cc. A striking difference from the older form of encephalitis with its frequent distressing sequels was the rapid and complete recovery in the vast majority of cases. With few exceptions the patients who survived the disease and had no complications were entirely well at the end of the arbitrarily fixed isolation period of 3 weeks. Practically all of the few patients who showed residual symptoms at that time had by 3 months from the onset given such remarkable evidence of improvement as to encourage the hope and belief that there was a good chance of ultimate complete recovery.

One unusual section of the report deals with public information and the reaction of the public. At no time during the period of the epidemic was there the slightest evidence of a psychological panic, and at no time did the people of the metropolitan area lose confidence in the capability and diligence of their health leaders or in the value of the scientific procedures which were being openly and frankly discussed. The readiness with which permission was granted for autopsies was an index and product of this popular interest and confidence and a most useful aid in solving the problems of the disease.

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DEATHS FROM EXCESSIVE HEAT IN KANSAS, 1934 1

By EARLE G. BROWN, M. D., Secretary, Kansas State Board of Health

Excessive heat was reported as the cause of 291 deaths in Kansas in 1934. This total is the highest ever recorded for this cause in the State since death records have been kept. The number of deaths from excessive heat was exceeded only by deaths from automobile accidents and accidental falls in that group charged to external violence.

Deaths from excessive heat reported in Kansas for the period of 23 years are as follows:

1934	291	1926	21	1918	29
1933	30	1925	23	1917	27
1932	27	1924	11	1916	36
1931	75	1923	16	1915	3
1930	65	1922	13	1914	46
		1921			
1928	25	1920	11	1912	18
		1919			

Heat prostrations were reported in five of the months, May to September, inclusive. From the death certificates and the use of the supplemental report form, the day of occurrence of the heat prostration or heat stroke was secured in 288 of the 291 fatal cases. The first reported case occurred on May 7, and the last on September 1. Seven fatal heat strokes were charged to June, 159 to July, and 118 to August. The highest number for any one day, 26, was reported on July 20, and the second highest, 20, on August 10.

Certain data pertaining to daily maximum and minimum temperatures and the day of the heat strokes for June, July, and August are shown in figure 1. The maximum and minimum temperatures are the average of 24 stations located in various sections of the State, and as recorded in the Kansas Section of Climatological Data for June to September, inclusive. According to S. D. Flora, meteorologist, Topeka, these averages may be considered as the State average. Both maximum and minimum temperatures follow a similar curve.

Referring to figure 1, it will be noted that on July 10 the maximum temperature rose to 106° F., dropped 2 degrees the following day, increased to 106° on July 12, and then equaled or exceeded this temperature for a period of 9 successive days. Twelve fatal heat strokes were recorded on July 17, 15 on July 19, 26 on July 20, and 18 on July 21. A second high peak was reached on August 9. In both months the high number of fatal heat strokes occurred following a

¹ For further information regarding excessive mortality in the drought-heat area during the summer of 1934, the reader is referred to the article "Maximum Temperatures and Increased Death Rates in the Drought Area", by Selwyn D. Collins and Mary Gover, published in the Public Health Reports for Aug. 31, 1934, pp. 1015–1018. (Reprint no. 1645.)—Ed.

number of days of exceptionally high temperatures. A record of the humidity rate for the State as a whole is not available, but the relative humidities undoubtedly were abnormally low.

Classifying the heat deaths into three groups, 249 were placed in the home group, 15 were the result of heat strokes in public places, and 27 were classed as industrial.

In the home group, 16 deaths occurred in children under 5 years, 14 of which were in babies under 1 year. One hundred and eighty-three deaths, or 73 percent, were in persons 65 years or over.

In the industrial group, 15 were reported as having originated in agriculture—5 in wheat fields, 4 in cornfields, 2 in hayfields, 2 in

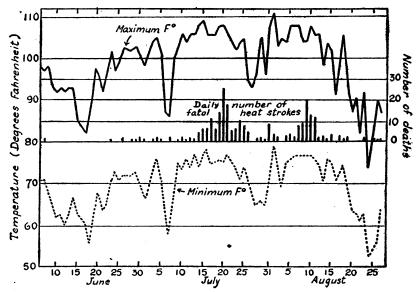


FIGURE 1.—Daily maximum and minimum temperatures and daily number of fatal heat strokes reported in Kansas for June, July, and August, 1934.

pastures or fields while herding cattle, 1 while digging a well, and 1 while working on a silo.

Data pertaining to the classification of heat deaths by age groups and place of attack are presented in table 1.

IABLE	1.—Deains	jrom	excessive	neui,	υy	uye	ana	piace	v)	SITUNE	,
 					A	ze					

	Age										
Place	All ages	0-4	5–9	10-14	15-24	25-29	30-39	40-49	50-59	60-64	65 and over
Total	291	16	2	3	3	3	11	20	29	21	183
Home Public place ¹ Industry ²	249 15 27	16 0 0	2 0 0	2 1 0	0 1 2	2 0 1	6 2 3	14 1 5	17 3 9	17 2 2	173 5 5

¹ Street or sidewalk, 6: in automobile on highway, 4; club, 1; park, 2; other places, 2.

Agriculture, 15; manufacture, 3; transportation and other public utility, 3; construction, 2; trade. 1; other industries, 3.

The average age at death of those persons having a heat stroke in a public place was 53.8 years. The average age of those persons who suffered heat strokes while engaged in industry was 50.7 years. Excluding the 14 deaths of infants under 1 year, the average age at death of the remaining 235 persons placed in the home group was 71.6 years.

Excessive heat was a less serious cause of death in the western half of the State than in the eastern half. In the western half of the State 19 deaths were reported as follows: Ellis County and Hays City, 3; Smith and Pratt Counties, 2 each; and one death each in Osborne, Russell, Barton, Stafford, Barber, Norton, Trego, Gove, Ford, Clark, Finney, and Hamilton Counties.

One hundred and thirty-six persons suffered heat strokes in cities of more than 2,500 population, constituting 46.7 percent of the heat deaths. This total is compared with an approximate total of 30 percent of the State population living in such cities.

Counties reporting more than 10 deaths (city totals included in county) from heat prostration include the following:

Douglas	12	Lyon	10
Lawrence	7	Emporia	4
Franklin	11	Miami	17
Ottawa	6	Shawnee	14
Labette	12	Topeka	9
Parsons	9	Wyandotte	43
Leavenworth	10	Kansas City	
Leavenworth city	6	<u>-</u>	

DEATHS DURING WEEK ENDED MAR. 30, 1935

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Mar. 30, 1935	Corresponding week, 1934
Data from 86 large cities of the United States: Total deaths. Deaths per 1,000 population, annual basis. Deaths under 1 year of age. Deaths under 1 year of age per 1,000 estimated live births. Deaths per 1,000 population, annual basis, first 13 weeks of year. Data from industrial insurance companies: Policies in force. Number of death claims. Death claims per 1,000 policies in force, annual rate. Death claims per 1,000 policies, first 13 weeks of year, annual rate.	8, 611 12. 0 571 52 12. 8 67, 659, 314 13, 584 10. 5 10. 9	8, 855 12. 3 659 61 12. 7 67, 693, 698 14, 075 10. 8 11. 1

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

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

Reports for Weeks Ended Apr. 6, 1935, and Apr. 7, 1934

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Apr. 6, 1935, and Apr. 7, 1934

	Diph	theria	Influ	Influenza		Measles		gococcus ngitis
Division and State	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut Middle Atlantic States:	9 2	1 1 15	19	1	92 1 7 520 242 1, 191	14 188 70 2, 622 16 23	0 0 0 1 1 1	0 0 0 2 0
New York New Jersey Pennsylvania	29	61 18 67	1 7 16	1 26 15	2, 983 1, 562 6, 227	1, 058 702 6, 371	27 1 4	3 1 4
East North Central States:	13	32 11 28 11 4	16 41 21 13 36	26 15 18 3 84	1, 520 370 2, 947 3, 887 1, 729	1, 621 894 1, 911 148 1, 429	13 9 23 1 4	1 3 11 1 5
West North Central States: Minnesota Iowa Missouri North Dakota South Dakota Nebraska Kansas	10 23 8 2 4	4 6 45 3 4 5	6 56 3 2	9 87 1 1	1, 056 1, 889 649 24 32 392 1, 725	316 258 839 106 350 244 345	1 5 8 0 1 5 2	1 0 4 0 1 1
South Atlantic States: Delaware Maryland 2 District of Columbia Virginia West Virginia North Carolina South Carolina Georgia 4 Florida	1 3 18 14 15 10 4 2	2 8 6 21 14 19 12 4	17 5 120 8 233	11 1 51 58 500	22 61 72 938 440 342 49	146 1, 689 375 2, 035 47 3, 201 639 780 444	0 7 10 5 1 6 1	1 0 0 4 1 0 0 0

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Apr. 6, 1935, and Apr. 7, 1934—Continued

	• ′	•	•	•	•			
	Dipl	theria	Infi	uenza	Me	asles		gococcus ingitis
Division and State	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934
East South Central States: Kentucky Tennessee 3 Alabama 3 Mississippi West South Central States:	4 6	8 7 11 8	36 78 144	32 73 56	738 82 441	668 878 977	1 2 3 3	4 2 0 1
Arkansas Louisiana Oklahoma 4 Texas 3 Mountain States:	14	5 19 5 78	19 16 124 614	34 22 80 445	88 138 198 163	249 401 439 1, 492	0 1 5 0	0 0 2 2
Montana ⁵	5 6	1 3 5	218 4 14	402 1	601 33 174 381 38	46 62 210 374 138	2 0 0 1 2	1 0 1 0 1 0
Arizona Utah ² Pacific States: Washington Oregon ⁵ California ³	6	1 1	21 2 81	27 4 3 40	63 6 262 210	23 440 153 103	1 2	1 0
California 3 Total	508	630	2, 073	2, 176	1, 313 35, 976	828 36, 362	174	63
First 14 weeks of year	9, 953	11, 784	93, 384	37, 375	352, 180	344, 599	1, 826	781
Division and State	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut Middle Atlantic States: New York	0 0 0 0 0	0000	13 7 12 261 6 130	15 11 7 234 2 77 835	00000	0000	5 0 2 0 0	31 1 0 1 0 2
New Jersey Pennsylvania Pentsylvania East North Central States: Ohio Indiana Illinois Michigan Wisconsin	1 0 0 1 1 0	1 2 5 1 0 1 0	171 757 877 204 1, 197 247 462	239 999 820 190 532 699 189	0 0 0 1 0 26	0 0 1 1 5 0 28	0 9 3 0 5 5 2	• 1 2 8 5 3 2
West North Central States: Minnesota Iowa Missouri North Dakota South Dakota Nebraska Kansas outh Atlantic States:	1 0 0 0 1 1 0	0 0 0 0 0 0	225 50 60 74 10 42 57	64 69 117 45 6 38 74	4 3 2 0 2 23 23	8 2 5 0 0 2 2	0 1 3 0 0 0	1 0 2 0 1 0 4
Outh Atlantic States: Delaware	0 0 1 0 0 8 0	0 0 0 1 0 0 0 2	20 126 113 38 64 29 5 7	9 81 7 21 87 27 10 7	0 0 2 0 1 0 0	0 0 0 1 0 0	0 2 0 2 4 11 1 2 4	2 6 0 3 3 0 5 7

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Apr. 6, 1935, and Apr. 7, 1934—Continued

	Polion	Poliomyelitis		t fever	Smallpox		Typho	id fever
Division and State	Week ended Apr. 6, 1935	Week ended Apr. 7, 1934						
East South Central States:								
Kentucky	0	0	37	57	1 0	1	1	2
Kentucky	0	0	18	44	ĺÒ	0	7	Ī
Alabama 3	0	0	111	10	10	Ŏ	7	i
Mississippi West South Central States:	0	2	3	3	0	6	2	ã
West South Central States:			I	f .			1	1
Arkansas	0	0	3	5	1	1	0	
Louisiana		0	7	25	1	1	12	11
Oklahoma 4	0	0	13	47	0	4	2	ī
Texas 3	3	2	60	100	105	73	20	•
Mountain States:								
Montana 5	0	0	7	9	3	0	0	1
Idaho 3	0	0	11	2	0	1	0	. 0
Wyoming 4	0	0	17	9	11	1	1	0
Colorado	0	0	277	33	16	5	0	2
New Mexico		0	16	13	3	1	3	. 1
Arizona		3	32	25	1	0	0	0
Utah ?	0	0	92	7	0	0	.1	0
Pacific States:	j							
Washington	0	0	57	66	15	9	2	3
Oregon 5	0	0	76	20	3	9	3.	Q
California 3	5	6	240	141	3	2	3	7
Total	21	30	7, 515	6, 128	261	169	130	153
First 14 weeks of year	356	286	99, 950	84, 797	2, 749	2, 057	1,796	2, 086

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week.

State	Menin- gococ- cus menin- gitis	Diph- theria	Influ- enza	Malaria	Measles	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
February 1935 Arizona Mississippi	3 7	13 24	1, 058 14, 824	1, 514	207 436	1 158	0	146 59	0	2 10
Nevada March 1935	3	3	34	1, 314	1	100	ő	16	ő	1
Arkansas	7 4 19 10	12 19 3 94 17	313 72 9 282 13	68	393 4, 137 31 2, 764 2, 173	23	0 1 0 1 1	23 399 95 1, 041 175	5 0 0 4 112	4 1 7 2

February 1935		February 1935		February 1935	
Chicken pox: Arizona	Cases 132 600 45	Hookworm disease: Mississippi	Cases 224 86 609	Undulant fever: Mississippi Whooping cough: Arizona Mississippi	157
Mississippi Dysentery: Arizona (amoebic) Mississippi (amoebic) Mississippi (bacillary) German measles: Arizona	5 45 221 47	Puerperal septicemia: Mississippi. Rabies in animals: Mississippi Trachoma: Arizona Mississippi.	28 18 35 10	Nevada	165 564

New York City only.
 Week ended earlier than Saturday.
 Typhus fever, week ended Apr. 6, 1935, 11 cases, as follows: Georgia, 5; Tennessee, 1; Alabama, 1; Texas,
 California, 1.
 Exclusive of Oklahoma City and Tulsa.
 Rocky Mountain spotted fever, week ended Apr. 6, 1935, 7 cases, as follows: Montana, 2; Idaho, 2; Wyoming, 1; Oregon, 2.

March 1935-Con.		March 1935-Con.		March 1935—Con.	
Chicken pox—Con. Delaware. Indiana. Nebraska. Conjunctivitis: Connecticut. Epidemic encephalitis: Connecticut. Indiana. German measles: Connecticut. Mumps: Arkansas. Connecticut. Delaware.	721 174 4	Mumps—Continued. Indiana	373 3 2 4 70	Tetanus: Connecticut. Delaware. Trichinosis: Connecticut. Undulant fever: Connecticut Delaware. Indiana. Whooping cough: Arkansas. Connecticut. Delaware. Indiana. Nebraska.	2 2 1 1 4

WEEKLY REPORTS FROM CITIES

City reports for week ended Mar. 30, 1935

[This table summarizes the reports received regularly from a selected list of 121 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference]

											
State and city	Diph- theria	Infl	luenza	Mea- sles	Pneu- monia	Scar- let	Small-	Tuber- culosis	Ty- phoid	Whoop-	Deaths,
	cases	Cases Deaths		cases	deaths	fever cases	cases	deaths	fever cases	cases	causes
Maine: Portland	0		0	0	4	3	0	0	1	7	21
New Hampshire: Concord Nashua	. 0		. 0	0	2	3	0	1	0	0	17
Vermont: Barre	0		0	0	0	0	0	1	0	0	4
Burlington Massachusetts: Boston	1 5		0	64 37	30	55	. 0	0 12	0	0 23	5 238
Fall River Springfield Worcester	2 0 1		0 0 0	24 160 5	0 3 4	2 19 16	0 0 0	2 0 3	0	16 14 9	34 40
Rhode Island: Pawtucket Providence	0		0	0 106	0 2	1 8	0	0 2	0	0	17 75
Connecticut: Bridgeport Hartford	0	1	1 0	8 51	3 6	14 10	0	1	0	0 18	32 35
New Haven	Ŏ	1	1	641	5	Ō	Ō	Ŏ	Ō	1	49
New York: Buffalo New York	0 25	1 18	2	176 1, 368	11 151	64 789	0	7 92	0	20 273	129 1, 55&
Rochester Syracuse New Jersey:	1 0		0	361 309	6	19 5	0	0	8	32 25	58 47
Camden Newark Trenton	1 0 0	2 8 0	1 0 1	308 16	4 6	8 18 7	0	0 10 1	0	0 73 2	34 100 32
Pennsylvania: Philadelphia	6	6 7	3	32 705	44 24	99 33	0	30	1	98 21	497 172
Pittsburgh Reading Scranton	1 0 0	ó	7 1 0	43 154	4 0	6	0	1 0	0	2 6	30
Ohio: Cincinnati	6		1	o	9	35	o	4	0	5	111
Cleveland Columbus Toledo	9 2 1	38 2 2	2 2 1	445 173 111	34 9 9	55 28 21	0 0 0	9 5 3	0	54 1 12	215 102 76
Indiana: Fort Wayne	1		0	12 94	4 15	3 18	0	1 4	0	0	22 119
Indianapolis South Bend Terre Haute	0		0	1 0	3 0	4	ô	1 0	0	ő	15 25
Illinois: Chicago Springfield	9	9	3 0	1, 519 29	61	683 16	0	40 0	0	103 4	709 25
Michigan: DetroitFlint	5 3	7	3 1	2, 184 189	36 5	122 19	0	19	0	83 5	307 23 30
Grand Rapids	ŏl		î	104	ŏΙ	13	ŏl	Ŏ	ŏl	7	30

City reports for week ended Mar. 30, 1935—Continued

Diph- Influenza Mea- Pneu- Scar- Small- Tuber- phoid inc								Whoop	Deaths,		
State and city	Diph- theria cases			Mea- sles cases	monia deaths	let fever	pox	culosis	phoid fever	ing cough	all causes
****	Cases	Cases	Deatils		doatils	cases			cases	cases	Causes
Wisconsin: Kenosha	0		0	157	0	6	2	1	0	6	7
Milwaukee Racine	1 0	1	1 0	219 31	6	163 22	0	2 0	0	40 5	107 10
Superior	ŏ		ŏ	138	2	70	ŏ	ŏ	ŏ	ĭ	6
Minnesota:	l										ļ
Duluth Minneapolis	4		0	724	1	119	0	3	0	15	94
St. Paul Iowa:	0		0	26	9	48	1	3	0	7	58
Davenport	0			. 0		3	0		0	0	
Des Moines Sioux City	0 2		0	414 5	0	1	0	0	1	0 5	36
Waterloo	ī			2		3	Ŏ		Ō	Ö	
Missouri: Kansas City	7		0	189	9	16	0	7	0	1	100
St. Joseph St. Louis	0 16		0	8 19	11 25	$\frac{2}{12}$	0	0 15	0	1 7	47 237
North Dakota:	į		1				_			-	
Fargo Grand Forks	0		0	0	1	6	0	0	0	0 1	3
South Dakota:							0		0	0	
Aberdeen Nebraska:	0			11		1					
Omaha Kansas:	2		1	58	6	6	0	2	0	1	45
Topeka											
Wichita	0	1	1	767	4	1	1	2	0	2	37
Delaware: Wilmington	0		0	13	11	6	0	0	0	o	33
Maryland: Baltimore	1	4	1	31	36	75	0	18	1	18	247
Cumberland	ó		ô	9	8	3	0	0	0	0	6
Frederick District of Colum-	0		0	0	1	0	0	0	0	0	4
bia:									ا	ا	
Washington Virginia:	14	4	3	52	20	118	0	21	0	3	174
Lynchburg Norfolk	1 0		0	34 87	8	0	0	0	0	6 7	15 39
Richmond	1		2	153	1	4	0	1	Ō	0	45
Roanoke	2		0	53	2	0	0	2	0	4	17
Charleston Huntington	0	4	1	20 7	2	0	0	2	0	3	36
Wheeling	ŏ		0	87	1	8	ŏ	1	ŏ	7	24
North Carolina: Raleigh	0		اه	2	2	1	ol	o	0	2	9
Wilmington	Ō		0	0	0	0	Q	0	0	2 14	11
Winston-Salem South Carolina:	0	3	0	2	3	5	٥١	1	0		16
Charleston Columbia	0	10	2 0	5	8 2	0	0	2	0	1 0	33 16
Greenville	ĭ		ŏ	ŏį	3	2	ŏ	δj	ŏ	ŏ	19
Georgia: Atlanta	1	9	2	2	5	5	0	7	o	3	74
Brunswick	0		0	0	1	0	0	0	0	0	4 38
Savannah Florida:	0		0	0	3	3	0	1	1	0	
Miami Tampa	0		8	0 50	4 0	0 2	0	1	0	0	28 24
Kentucky:	-		1		1	-		-	_	_	
Ashland	0	2	0	18	0	1	0	0	0	0	.0
Lexington Louisville	1 3	3	0	410	1 16	1 24	0	1 3	1	20	18 76
Tennessee:			- 1	1	1	- 1		2		7	
Memphis Nashville	4		1	0 8	12 5	4 2	0	3	0	9	67 50
Alabama: Birmingham	1	7	1	25	6	4	0	4	1	2	62
Mobile	0		2	1	1	1	0	3	0	0	27
Montgomery	0	1	0	26	0	0	0	0	0	6	
Arkansas: Little Rock	0		0	82	1	o	٥	0	اه	6	2
Louisiana:			į.				l	1		o	
New Orleans Shreveport	12 0	6	2 0	26 3	13 9	2	0	14	2	i!	137 68
	•		•		•		•				

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City reports for week ended Mar. 30, 1935-Continued

G4-4 1-14	Diph-	Inf	Influenza		Paeu-	Scar- let	Small-	Tuber-	Ty- phoid	Whooping	Deaths,
State and city	theria cases	Cases	Deaths	8163 C2368	monia deaths	fever cases	pox cases	culosis deaths	fever cases	cases	causes
Oklahoma:											
Oklahoma City Texas:	1	10	1	20	13	2	0	1	0	0	40
Dallas	8	1	1	0	10	3	0	4	0	0	63
Fort Worth	0		1	1	9	5	0	2	0	1	40
Galveston	0		0	2	1	1	9	1 3	0	0	13
Houston	11		2	1	5	0	9	3	0	0	64 58
San Antonio	2		3	2	4	2	0	7	1	0	98
Montana:					1			1 1			
	0		0	8	0	2	0	lol	0	0	4
Billings Great Falls	ŏ		ŏ	ŏ	ŏ	ō	ŏ	Ŏ	ŏ	4	8
Helena	Ō		Ō	39	i	Ō	Ŏ	Ŏ	ŏ l	1	8
Missoula	0		0	25	2	0	0	0	0	0	4
Idaho:									- 1		
Boise	0		0	11	0	3	0	1	0	0	4
Colorado: Denver		49						_			
Pueblo	6	49	1	255 165	4 2	181 14	0	5 1	0	2i	89 12
New Mexico:	0		- 1	100	- 4	12	٠ ٧	- 1	U	١٥	12
Albuquerque	0	2	0	3	2	2	0	1	0	6	8
Utah:	۰	- 1	۱		-	- 1	١	- 1	١	١	Ū
Salt Lake City	0		0	7	1	95	ol	3	o l	83	36
Nevada:			- 1		_	1	- 1	-	1		
Reno	0		0	0	0	0	0	0	0	0	4
Washington:	- 1		- 1		1	1	1		i		
Soattle	0	:	2	107	8	5	2	3	2	7	93
Spokane	ŏ	i	ōl	139	2	6	٥١	ĭ	õl	i۱	35
Tacoma	ō l		ŏl	3	2	ŏ	ŏ	ĩ l	ŏl	ŏ	23
Oregon:	!	- 1	1	1		- 1	- 1	- 1	1	1	
Portland	0	2	0	124	7	9	0	3	0	0	68
Salem	0	4 .		0		3	0		0	0	
California:							1				
Los Angeles Sacramento	28	30	2	59	20	60	2	12	0	13	843
San Francisco	6	6	0 2	44 11	0 11	25	0	5 8	2	20	25 184
ran Francisco	0 1	0 1	4 '	41 !	41 '	20 1	<u> </u>	0 1	A 1	20 1	104

State and city		gococcus ngitis	Polio- mye- litis	State and city	Menin meni	Polio- mye- litis	
	Cases	Cases Deaths			Cases	Deaths	Cases
Massachusetts: Worcester	0			Kansas: Wichita			
Rhode Island	_	0	1		2	0	0
Providence		1	0	Baltimore	4	0	0
New York: Buffalo				District of Columbia:			_
New York	1 19	0 11	0 1	Washington Virginia:	13	4	0
		**	•	Lynchburg	1		0
New Jersey: Camden	0	0	1	Noriolk	1	1	Ŏ
Newark Pennsylvania:	1	0	0	West Virginia: Wheeling			
Philadelphia	2	2	1	North Carelina:	0	1	v
Pittsburgh	ĩ	ĩ	ō	Winston-Salem	1 1	2	1
Ohio:				Vantuokw		_ [_
Cincinnati Cleveland		4]	0	Louisville	3	2	0
Columbus	1	- 11	0	Tennessee: Nashville	2	1	
Toledo	3	2	ŏ	Alabama:	- 1	- 1	v
Indiana:				Birmingham	1	1	0
Indianapolis Illinois:	2	0	0	Arkansas: Little R ck	!	- 1	_
Chicago	13	2	0	Oklahoma:	1	1	0
ChicagoSpringfield	5	ã l	ŏ	Oklahoma City	1	1	0
Michigan: Detroit	1			Texas:	- 1	- 1	•
Wisconsin:	2	0	0	Fort Worth	0	0	1
Milwaukee	1	1 1	0	Los Angeles	0	ol	4
Iowa:	- 1	- 1	- 1	San Francisco	ŏl	ŏl	i
Sioux City	1	0	0		1	- 1	-
Missouri:	!	2		1	- 1		
Kansas City St. Joseph	1 1	2 2	0	1		i	
St. Louis	6	î l	ŏ∥	1	1	1	

Epidermic encephadilis.—Cases: Springfield, Mass., 1; New York, 1; Indianapolis, 2; St. Louis, 1 Birmingham, 2.

Pellagra.—Cases: Boston, 1; Winston-Salem, 1; Atlanta, 2; New Orleans, 1.

Typhus feber.—Cases: Atlanta, 2.

Dengue.—Cases: Miami, 1.

Rabies in man.—Deaths: Boston, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended March 23, 1935.— During the 2 weeks ended March 23, 1935, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada, as follows:

Disease	Prince Edward Island	Nova Scotia	New Bruns- wick	Quebec	Onta- rio	Mani- toba	Sas- katch- ewan	Al- berta	British Colum- bia	Total
Cerebrospinal men- ingitis. Chicken pox. Diphtheria.		1 11 3	16 1	281 10 6	1 450 12	107 9	58 2	5	3 119 1	5 1, 047 38 6
Erysipelas Influenza Lethargic encephali- tis		242	2	9 20	6 160 1	7 8	1	3	639	29 1,071 1
Measles Mumps Pneumonia Pollomyelitis		317 12 7	69	1, 210 3	5, 401 482 39 2	493 121	238 1 3	32 14	96 46 26	7, 856 6 76 75 5
Scarlet fever		30	10	285	274	42	38 4	16	50 2	745 6
Tuberculosis Typhoid fever	8	3	11	111 45	82	23 1	25 	6	39 3	308 49
Undulant fever Wheeping cough		3	2	249	274	63	101	4	105	801

ITALY

Communicable diseases—4 weeks ended October 14, 1934.—During the 4 weeks ended October 14, 1934, certain communicable diseases were reported in Italy, as follows:

•	Sept. 17-23		Sept. 24-30		Oct. 1-7		Oct. 8-14	
Disease	Cases	Com- munes affect- ed	Cases	Com- munes affect- ed	Cases	Com- munes affect- ed	Cases	Com- munes affect- ed
Anthrax Cerebrospinal meaingiiis Chicken pox Diphtheria and croup Dysentery Lethargic encephalitis Measles Poliomyelitis Scarlet fever Typhoid fever	35 7 82 427 21 2 330 17 324 1,056	29 7 58 225 13 2 131 17 150 529	45 3 51 574 39 1 476 18 351 1,140	30 3 41 295 20 1 160 17 166 592	25 8 56 629 35 469 15 388 1,036	29 7 39 340 17 156 12 175 510	11 4 57 625 40 488 17 360 929	11 4 37 331 20 137 16 174 501

JAMAICA

Communicable diseases—4 weeks ended March 23, 1935.—During the 4 weeks ended March 23, 1935, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kings- ton	Other localities	Disease	Kings-	Other localities
Chicken pox	11 1 8 1	28 1 9 1 3	Poliomyelitis Puerperal fever Tuberculosis Typhoid fever	53 10	2 3 107 41

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

(NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for Mar. 29, 1935, pp. 454-467. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued Apr. 26, 1935, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

Plague

Hawaii Territory—Hawaii Island—Hamakua District—Paauhau;—On March 18, 1935, one case of plague which proved fatal on March 25, 1935, was reported at Paauhau, Hamakua District, Island of Hawaii, Hawaii Territory. On March 26, 1935, two plague-infected rats were reported at Paauhau Landing, Hamakua District, Island of Hawaii, Hawaii Territory.

Morocco—Region of Saffi.—On March 30, 1935, 9 cases of plague with 5 deaths were reported in Ahmar Tribe, Region of Saffi, Morocco.

Typhus Fever

Egypt—Suez.—During the week ended March 30, 1935, one case of typhus fever was reported at Suez, Egypt.

Yellow Fever

Africa.—A report dated February 4, 1935, in regard to yellow fever in West Africa, states that the disease was present in Gambia, Nigeria, Ivory Coast, Gold Coast, and Sierra Leone. The Bathurst area, in Gambia, was said to be the most heavily affected region. No case had been reported in Liberia.

Sierra Leone—Freetown.—On March 21, 1935, one case of yellow fever was reported at Freetown, Sierra Leone.