PUBLIC HEALTH REPORTS

VOL. 47 OCTOBER 14, 1932 NO. 42

AN EPIDEMIC OF MOTOR NEURITIS IN CINCINNATI, OHIO, DUE TO DRINKING ADULTERATED JAMAICA GINGER

HISTORY, SYMPTOMATOLOLOGY, AND CLINICAL REPORT

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On March 9, 1930, press dispatches reported an epidemic paralysis of the legs in Oklahoma City ascribed to drinking Jamaica-ginger extract, popularly known as "jake." On the following day there was admitted to the Cincinnati General Hospital a tabetic whose condition was complicated by a rapidly progressive foot and wrist drop, and within a few days patients began coming with self-made diagnoses. The daily admissions are shown in Figure 1 (see p. 2056) for the months of March and April. Up to and including May, 316 cases were admitted. Confiscation of the suspected shipment by the State prohibition department, passage of a city ordinance prohibiting the sale of Jamaica ginger except on a physician's prescription, and the widespread publicity given by the press are to be credited with the suppression of the epidemic.

ONSET

It is a matter of common knowledge that Jamaica-ginger extract had a wide use as a beverage in dry communities even before the passage of the Volstead Act, and has been nationally used since. It had proved harmless in our group of patients up until late in February, 1930. So many of our patients were habitual drinkers of "jake" that it was usually difficult to identify the adulterated dose, but many of them reported that they had noticed unusual effects from the contents of the bottles marketed in late February and early March. There was a difference in taste and smell, and frequently violent vomiting and intestinal cramps and severe diarrhea followed the drinking of the extract.

More could be learned from patients whose use of "jake" had been confined to isolated consumption during the period in which this shipment was marketed. Two intelligent young men (not included in this series, as they were treated privately) drank four bottles each about Christmas time, with no untoward results. On March 1 each had two more bottles from the same stock, while a companion brought

his from another source. The first two acquired the syndrome within three weeks. The third was unaffected.

SYMPTOMS

The syndrome presented was a bilateral wrist and foot drop, with atrophy and the common signs of a degenerative neuritis. Tenderness of the nerve trunks was conspicuously rare, though many patients complained for some days of severe nocturnal pains of lancinating character in the third or fourth week after the onset. Objective sensory loss was not found, and the cranial nerves were not affected. A few patients had involuntary clay-colored stools, but the small number could easily justify the assumption of coincidence. No single case of the Korsakow psychosis developed; but one real mental condition was seen, a mildly paranoid episode. Conduct disorders requiring disciplinary measures were not infrequent, but so many of the patients were of an extremely low social level that this frequency is scarcely to be ascribed to the poison which caused the neuritis.

PROGRESS

The uniformity of the anatomical progress was startling, but the degree of disability varied widely. The weakness began first in the feet, and within a few weeks appeared in the hands. The amount of motor incapacity was not infrequently so slight that the patients remained ambulatory; cases were treated in the out-patient department of the hospital, and many were handled by physicians in their offices. No estimate of this number can be made. Physicians in neighboring cities have also made personal reports of such cases. On the other hand, many who walked into the hospital later became bedfast, and in many the paralysis of the hands was so complete as to require feeding by nurses.

Recovery has been slow. The large number of cases and the mental level of many of the patients made a follow up impossible; only the discharge from hospital can be used as a criterion. As of February 6, 1932, 119 cases were still under the care of the Cincinnati General Hospital or affiliated institutions. Of this number, 34 were bedridden, 8 walked with crutches, 57 used canes, and 20 were able to walk unassisted. The hands of 72 were in good condition, while those of 32 were moderately impaired, and of 15 badly contractured.

Six deaths occurred in the Cincinnati General Hospital, but in every case some other pathological condition adequate to cause death was found. The death of but one patient who died in Cincinnati can be

ascribed purely to this intoxication. This case was under the care of Dr. A. R. Vonderahe, and was made the subject of a special report, which is appended.

PATHOLOGY

The report of Vonderahe shows that neither in the patients dying of intercurrent disease in the Cincinnati General Hospital nor in the patient whose death was due solely to intoxication with this poison was there evidence of an infectious disease of the nervous system, but there were changes typical of neuritis and anterior horn-cell degeneration.

ETIOLOGY

With the outbreak of the epidemic it became urgent to know whether we had on our hands a highly infectious disease, and so isolation was practiced. Epidemic poliomyelitis was ruled out by the age of the patients, the symmetry of the paralysis, and the absence of febrile reaction. A careful study by Dorst and associates gave consistently negative laboratory findings in 75 cases. The later post-mortem reports confirmed his conclusions.

The postulate that we had merely an unusual number of alcoholic neuritics was equally untenable. Table 1 shows the admission for alcoholic neuritis for the years 1901–1910 and 1915–1930, inclusive. A comparison of these with the admission of over 300 cases in two months succeeding March 9 furnishes convincing disproof of such an assumption. Further analysis below shows how large a number of patients, not excessive drinkers of any sort of alcoholic beverage, were poisoned by a very small amount of the extract. This was marketed in bottles containing about 14 drams; and as Jamaica-ginger extract comes under the pure food and drug act, it is guaranteed to contain about 92 per cent of alcohol. It is usually diluted with water or soda water for beverage purposes, though a few patients boasted of drinking it "straight." The most startling proof of toxicity is furnished by the case of a tailor who had never tasted "jake" until February, 1930, when he was advised to use some for abdominal cramps. He sent a colored porter for a bottle (14 drams); each drank approximately half, and both developed the characteristic syndrome. Such an amount of alcohol is obviously too small to produce alcoholic neuritis, and the same is true in the case of those who drank four or five bottles.

TABLE 1.—*Hospital admissions for alcoholic neuritis, 1901–1910, 1915–1930*

Year	Number of patients admitted	Year	Number of patients admitted
1901	1	1918	2
1902	0	1919	2
1903	6	1920	1
1904	4	1921	1
1905	3	1922	2
1906	2	1923	1
1907	3	1924	1
1908	6	1925	0
1909	4	1926	1
1910	8	1927	0
		1928	1
Total	37	1929	2
		1930	1
1915	3		
1916	7	Total	36
1917	11		

Figures for all years are not obtainable.

As we were unable to find a record of any toxic effect of properly prepared ginger extract in the literature, and as many of the habitual drinkers recognized a difference of taste and smell and unusual gastrointestinal effects, while some patients were paralyzed by extremely small amounts, it seems impossible to evade the conclusion that the syndrome was produced by some adulterant of the ginger extract. Of the 10 patients who denied its use, some no doubt were truthful, and their paralysis remains unexplained. Communications from Federal officers who investigated the cases in neighboring counties and States show that the trail of the "jake" peddler could practically be followed by his victims.

INVESTIGATION OF THE POISON

The clinical syndrome and the known insidious ways in which arsenic has found its way into foods and beverages demanded a search for this metal at the outset. However, none of the large number of analyses made on ginger extract bought in the open market or in the large quantity furnished us by the prohibition department, and known to have come from the contaminated shipment, gave any but such faint traces as can be found in most any substance submitted to the Gutzeit test. Kehoe and Thamann, of the University of Cincinnati, made repeated search for arsenic in the urine, and found only negligible traces. It must be remembered that Hamilton found arsenic in the urine for from 19 to 140 days after intoxication. Thamann took conservative doses of arsenic as Fowler's solution, and his urine gave immensely more severe reactions than did that of the patients examined. Foulger (personal communication to the dean of the medical school) sought evidence for arsenical intoxication in examination of the patient's hair. In short hair from males he demonstrated three times as much arsenic as in hair taken from a barber's floor for con-

trol. He also hoped to find in the long hair of women a record of the periods of arsenic ingestion. Taking the growth of hair at 1/40 to 1/60 of an inch per day, he cut hair from the heads of four women into inch lengths, which would therefore correspond to 8-day periods, if allowance is made for the diffusion rate into the hair. While he felt that peaks of arsenic content in the hair corresponded with periods of "jake" consumption, there are many facts which throw doubt on the value of the method. One of these women consistently denied ever using "jake," and there seems to be little doubt that this beverage is the source of the paralysis. In another woman whose variations of arsenic content corresponded with her original statements as to the time of consumption, a second interview showed her to be entirely vague about dates; in fact she only knew she had drunk the extract "since Christmas." Also a second clipping of hair taken immediately adjacent to one showing higher amounts of arsenic failed to give the faintest traces with the same test. The highest amount of all was found in the hair of a woman who is a total abstainer.

In the Manchester beer epidemic of 1901 it must be recalled that one-fourth to 1½ grains of arsenic as As_2O_3 were found per gallon (1). Comparing the infinitesimal amounts of arsenic found in these Jamaica-ginger extracts or the patient's hair or urine, it seems necessary to reject arsenic as the poison at fault. We are informed by officials of the Government laboratories that the population is consuming with impunity very appreciable amounts of arsenic in fruits and fruit juices as the result of the use of insecticides on the plants. The postulate that this epidemic was the result of arsenic acting on patients suffering from avitaminosis is unsupported by chemical experience and contradicted by the epidemiological study of this disease, which shows the patients as a whole to have consumed an average diet for persons in their stations in life. Poisoning from ricin, a constituent of crude castor oil, was suggested. The toxicology of ricin has been studied, and no such syndrome as produced here was found (2) (3) (4).

The great frequency of poisoning from denatured alcohol in recent years, coupled with the authorized technique of extracting Jamaica-ginger root, indicated a search for toxic denaturants. The Federal Government authorizes the use of grain alcohol containing 5 per cent methyl alcohol for the original extraction of the ground root. The loss of alcohol in this process is considerable, enough to warrant the use of this cheap tax-free alcohol. The extract is next evaporated *in vacuo*, and the solvent is recovered and reused. The residue is dissolved in (taxed) grain alcohol and marketed. Of the numerous denaturants authorized by the Government, many were chemicals whose toxicity is quite unknown even to experts, and it seemed not impossible that without any guilt of the manufacturers, alcohol so

denatured was inadvertently used in the process. A nonvolatile denaturant would remain in the residue in the approved technique, and so appear in the final product. Obviously it would be there if the same mistake were made with the final solvent.

Of the authorized denaturants, brucine and emetine are known to produce neuritis (5), but neither our research nor any other known to us has demonstrated this poison at fault.

The poison at fault seems to have been established by Smith and Elvove, of the National Institute of Health, United States Public Health Service (6) (7) (8). They have consistently found tri-ortho-cresyl phosphate in confiscated samples of "jake," and experimental poisoning of dogs, cats, monkeys, and chickens produced a motor paralysis comparable to the human syndrome.

CLINICAL REPORT

EPIDEMIOLOGY

Age.—These patients were all adults, and they ranged from 21 to 79 years of age. The majority of them were in the middle adult period of life, 138, or 68.7 per cent, being between the ages of 41 and 60, as shown by the accompanying table.

Age	Number of patients
21–25	2
26–30	6
31–35	11
36–40	15
41–45	34
46–50	40
51–55	34
56–60	30
61–65	17
66–70	7
71	5
	201

Sex.—In this series 191 were male and 10 were female.

Color.—There were 198 white and 3 negro patients studied.

ETIOLOGY

It has been the accepted belief that this epidemic has resulted from some poison contained in Jamaica ginger that had been used for beverage purposes. Therefore an attempt was made to determine the incidence of "jake" drinking among these patients, and at the same time to list any other alcoholic beverage that might have been used. The most common drinks were Jamaica ginger, "moonshine" whisky and home-brew, although other things had been used by a few, such as "canned heat" and denatured alcohol.

Drink	Number admitted using drink	Drink	Number admitted using drink
Ginger, "moonshine," and home-brew	96	Ginger alone	31
Ginger and home-brew	22	Home-brew alone	
Ginger and "moonshine"	42	"Moonshine" alone	3
"Moonshine" and home-brew	5	Nothing	2

Tabulating these figures in another manner, we get the following results:

Total number of patients admitting drinking "jake," 191, or 95.2 per cent.

Total number of patients admitting drinking "moonshine," 146, or 72.6 per cent.

Total number of patients admitting drinking home-brew, 123, or 61.2 per cent.

These figures show that Jamaica ginger was the most common beverage used by this group of patients. Not only did over 95 per cent of them admit drinking it, as compared with 72 per cent that had used "moonshine" and 61 per cent home-brew, but there were 31 patients who stated they had had nothing but Jamaica ginger, as compared with 3 who were partial to "moonshine" exclusively, and none at all who stated that home-brew was their only drink. It should be stated here that the vast majority of these patients were habitual consumers of alcoholic drinks, and that this history of what they drank included only that used for a period of at least three months previous to the onset of their paralysis. It has been of interest to see whether there was any quantitative relationship between the amount of ginger consumed and the severity of the paralysis. If a constant direct relationship could be established, then this also might be added evidence of the fact that Jamaica ginger caused this epidemic. Therefore an attempt to do this was made in the following manner: After a few of these patients had been interviewed, it became evident that they themselves frequently distinguished between what they called "new jake" and "old jake" and they firmly believed that the so-called "new jake," which they recognized by either taste or appearance, was the cause of their condition. In the case of 95 patients we were able to get a history not only of drinking this so-called "new jake" but also of how much of it had been consumed. Then these cases were divided arbitrarily into those with severe paralysis and those with relatively mild paralysis. Listed finally according to amount consumed, we find the following percentages in severe paralysis.

Less than 1 bottle	9 out of 11 cases severe—81.8 per cent severe.
1 bottle	13 out of 21 cases severe—61.9 per cent severe.
2 bottles	11 out of 17 cases severe—64.7 per cent severe.
3 bottles	9 out of 12 cases severe—75 per cent severe.
4 bottles	5 out of 6 cases severe—82.5 per cent severe.

5 bottles	5 out of 5 cases severe—100 per cent severe.
6–10 bottles	10 out of 11 cases severe—91 per cent severe.
10–25 bottles	8 out of 9 cases severe—88.8 per cent severe.

These figures show a fairly constant relationship between the amount of "new jake" consumed and the severity of the paralysis. It is our belief, therefore, that this epidemic has been caused by the drinking of Jamaica ginger—first, because such a high percentage of the patients admitted drinking it; and, second, because a direct relationship can be shown to exist between the amount of "new ginger" taken and the severity of the paralysis.

Not only is it believed that Jamaica ginger was the cause of the epidemic, but it is also believed that it was due to the drinking of a new supply of the extract, which flooded the market at about the time the epidemic started. It is also believed that it was not due to the cumulative effect of continued use of this drug as a beverage. This belief is held for two reasons: First, the lack of relationship between the amount of all "jake" which each patient had taken and the severity of the paralysis; and, second, the comparatively short period of time in which all of these cases appeared.

We were able in 187 cases to determine roughly whether or not the extract had been used to any very great extent as a beverage. We then divided these cases into two groups—first, those who in their lifetime stated that they had taken not more than five bottles of it; and, second, those who had taken more than five bottles. Obviously the second group contained all those who had been using it for some time as a beverage, while the first consisted of those who had used it only very recently for the first time, or who had taken it for medicinal purposes. Then each of these was classified as to whether the paralysis was severe or mild according to the arbitrary standards used before. The following table presents the results:

Group of patients	Total number of patients	Severe	Mild	Per cent severe
Used less than 5 bottles	41	28	13	68.3
Used more than 5 bottles	146	105	41	71.9

Thus it is seen that there was very little difference between the two in the percentage of severe cases.

As will be mentioned again, a large number of these patients complained that the ginger which paralyzed them gave them more or less of a gastrointestinal upset shortly after drinking it. We were able to obtain such a history in 125 patients. Assuming that these patients were poisoned on the date of this upset, and including 13

others in whom we can establish with certainty from their history the date that they were poisoned, we found that a large percentage of them were afflicted within a very short period, as shown below:

Time period	Number of cases	
Feb. 1–7	0	
8–14	5	
15–21	4	
22–28	10	
Mar. 1–7	31	
8–14	37	123, or 89 per cent.
15–21	35	
22–28	10	
29–31	4	
Apr. 1–7	2	
8–15	0	

It is not reasonable to suppose that anything except a new supply of ginger would cause all these patients, who had been drinking ginger for such variable periods of time and in such greatly different amounts, to become paralyzed during about the same period of time and with a paralysis that did not depend in severity on the amount that they had taken previously.

SYMPTOMATOLOGY

The first symptoms of which these patients complained after drinking the "new" Jamaica ginger were those of gastroenteritis. They were not present in every patient. However, 125 out of 201, or 62.2 per cent, gave a history of such a condition. In addition, 5 others complained of the same symptoms, 4 of whom had been drinking "moonshine," and the other beer. Three of these five were among those who denied drinking any ginger, but one of them stated that he believed something (possibly ginger) had been added to the homemade drink, as it did not taste as it should. This gastrointestinal upset occurred within a few hours after drinking, and varied greatly in its severity. In some it consisted only in a feeling of nausea; in others there was only a diarrhea. In the majority, however, there was nausea, vomiting, abdominal cramps, and diarrhea. In a few the condition was severe enough to cause blood in the stool. As a rule these symptoms lasted only a day or so, and then after an interval the symptoms of neuritis appeared.

This interval, which we will designate as the paralysis interval, varied a great deal in length. The shortest was 1 day and the longest 42 days. In 142 cases in which it could be determined (consisting of the 125 who had the gastrointestinal upset, and 17 others in whom it was possible to establish the time when the ginger which

caused the paralysis had been taken), the paralysis interval was as shown in the following table:

Paralysis interval in days	Number of cases
1–7	70
8–14	39
15–21	18
22–28	9
29–35	3
36+	3

The severity of the paralysis bore a direct relationship to the paralysis interval, i. e., there were more cases of severe paralysis among those with a short interval than among those with a long one. This is demonstrated in the table presented below.

Paralysis interval in days	Number of cases	Severity of cases		Per cent severe
		Mild	Severe	
1–7	70	14	56	80
8–14	39	12	27	69.3
15–21	18	4	14	77.8
22–28	9	4	5	55.5
29–35	3	2	1	33.3
36+	3	2	1	33.3

The paralysis, which began in the lower extremities in every patient except 2, in whom the hands were first involved, was preceded by pain in the calf muscles of the legs. It was present in 168, or 83.6 per cent, and, as a rule, lasted only a few hours. It was described as an "aching pain" or a "tired feeling" or as "cramps." Some of the patients stated that they felt as if they had been walking too much.

The paralysis was typical of a multiple neuritis involving the motor fibers only. It began in the distal portion of the extremities and was always bilateral. The flexor and extensor muscles were equally involved. Often the first abnormality the patient recognized was that his "feet were flopping." This was due to the foot drop that soon was present in every case. These patients all developed a steppage gait. It soon spread up the extremity, so that within a few days the more severe cases were unable to walk at all.

The hands became involved as a rule some time after the onset of the paralysis. In 137 cases in which this was determined the interval was as follows:

Interval between onset of paralysis and involvement of hands, in days	Number of cases	
Same day	5	
1–5	27	120, or 86.1 per cent
6–10	47	
11–15	41	
16–20	7	
21–25	6	
26–30	8	
30+	1	
	137	

This was first noticed, as a rule, by a loss of the strength in the fingers and by inability to perform finer movements, such as buttoning their clothes and writing. The paralysis then spread up their arms, as it did their legs, and in a number of patients there finally developed complete paralysis of the hands. Not only the extremities suffered; in certain cases there was marked weakness of the abdominal and back muscles which prevented the patient from even sitting up in bed. In no case, however, was there any demonstrable paralysis of the diaphragm or intercostal muscles, and likewise, the cranial nerves did not become involved.

The following table shows the severe degree which the condition reached. The time which elapsed before it became most severe as a rule was between 2 and 3 weeks after the onset of the paralysis.

Involvement of lower extremities:	
1. Those with inability to flex or extend ankles but with good power in thigh muscles	21
2. Those with weakness of thigh muscles but still able to walk	54
3. Those unable to walk but still able to extend knee against gravity	57
4. Those unable to move knees at all	69
Total	201
Involvement of upper extremities:	
1. Those with no demonstrable involvement of upper extremities	3
2. Those with weakness of fingers	88
3. Those with marked weakness of hands and arms but with ability to extend wrists against gravity	52
4. Those with complete paralysis of hands and with marked weakness of arm muscles	58
Total	201
Involvement of abdominal and back muscles	58

It is to be remembered, however, that the cases presented in this series are probably the most severe ones of the epidemic, as the series consists of those admitted to the wards of the hospital. Many with lesser degrees of paralysis were treated in the clinic, and probably a larger number remained at home.

Associated with this progressive, bilateral, symmetrical paralysis were other features. Nearly all of the patients showed some disturbance of the vasomotor control of their lower extremities (181 cases, or 90 per cent). This was shown by profuse sweating of the feet at times, or by marked cyanosis when the legs were allowed to hang over the edge of the bed, or by pronounced edema of the ankles. This edema was not cardiac in origin, as physical examination of the heart was negative in nearly every case. Skin manifestations were rare. A few showed a generalized erythematous blush which faded within a week or two. Desquamation of the palms and soles, varying from slight scaling to complete desquamation, was much more common, occurring in 85 cases, or 42.3 per cent. Three of the cases developed a sterile effusion into the knee joint, such as has been described as occurring in cases of peripheral neuritis. A peculiar finding was that the liver edge in a large proportion of these patients was easily palpable from 1 to 3 finger breadths below the costal margin. It was firm, smooth, and not tender. This may be due to an associated alcoholic cirrhosis, as many of these patients were heavy drinkers, or the relaxed abdominal wall may have made palpation more easy. It was present in 97 cases, or 43.3 per cent. Sphincter control was impaired in 16 cases, and in several of these there was complete loss. As a rule the bladder was more often affected than was the anal sphincter.

As stated before, the neuritis was limited entirely to the motor nerves. There were no areas of anesthesia present in any case. In 11 of 79 cases, or 13.9 per cent (where note was made of this condition), there was some hyperesthesia of the soles of the feet. The patients complained of a good deal of pain in the muscles of the legs. This was described as an "aching" or "cramping" pain, and was most severe during the night. In some there were spasmodic contractions of muscle groups, causing the legs to be jerked violently during sleep. Of the 201 cases, 138 complained of aching, and 106 of the violent spasmodic contractions. These pains usually began after the paralysis had been present for a week or so, and then, after disturbing their sleep for from 3 to 4 weeks, gradually left. In one patient they lasted over 8 weeks and were severe enough to require morphine for relief. Vibratory sense was intact in the few in which this was tried, and there was no impairment of proprioceptive sensation. Nerve-trunk tenderness was present in comparatively few, only 11 of 119, or 9.1 per cent, where note was made of this condition. In this same 119, 46, or 38.8 per cent, complained of pain on pressure over the calf muscles.

The reflexes in these patients showed nothing remarkable. They were absent if there was a paralysis of the muscles involved; otherwise they were normal. For example, the knee jerks were present if the

quadriceps was not paralyzed. Ankle clonus was uniformly absent, and there was no response to plantar stimulation in any case. The cranial reflexes were not involved.

General physical examination showed only those abnormalities that might be expected in a group of men of this class. There were a number of hernias, varicose veins, hemorrhoids, etc. On the whole it can be stated that this epidemic produced no demonstrable changes that could be discovered during a routine physical examination, especially of the heart and lungs. Examinations by Dr. Albert Brown showed the fundi to be normal.

The patients themselves had no complaints other than the paralysis and the accompanying pain. Their appetites remained good, and they apparently enjoyed good health for the most part. They accepted their condition philosophically on the whole, and remained in good spirits. However, six patients of this series died while in the hospital. One had a carcinoma of the stomach, another developed a pulmonary endarteritis with multiple lung infarcts, and a third died suddenly, probably from an attack of angina pectoris. Post-mortem examinations were performed in all these cases.

SIGNIFICANT LABORATORY WORK

The laboratory work done on these patients showed no deviation from the normal. Urine examinations were done in 124 cases. A trace of albumin was present in 5; 6 others showed a few hyaline casts. White blood counts on 106 showed that the count was between 5,000 and 10,000 in 85 per cent of the cases. Only 4 were higher than 13,000.

RESULTS OF LABORATORY EXAMINATIONS

White blood count	Number of cases
5,000	4
6,000	6
7,000	30
8,000	22
9,000	13
10,000	16
11,000	5
12,000	4
13,000	2
14,000	1
15,000	1
16,000	1
17,000	1
	106

A red blood count was done on 45 patients. In all but 3 the count was over 4,000,000.

Red blood count	Number of cases
5,000,000 plus	14
4,500,000–5,000,000 plus	16
4,000,000–4,500,000	12
3,500,000–4,000,000	3
	45

The percentage of hemoglobin was determined in 85 cases. In 74 it was 80 per cent or over.

Per cent hemoglobin	Number of cases
100	5
95	3
90	18
85	13
80	35
75	8
70	3
	85

In 90 patients a differential count was made. No abnormalities were noted except that there was some increase in the percentage of large mononuclear cells; in some cases these reached as high as 20 per cent.

Spinal punctures were done on 120 of the patients. The fluid was normal in every case except 2, in which there was a trace of globulin, and 5, in which there was a slight increase in the cell count. This does not include 12 cases in which both cells and globulin were present, and who also had a positive spinal fluid Wassermann. Blood Wassermanns were taken in 128 cases and were returned positive in 29 cases, an incidence of 22.7 per cent. Blood cultures were taken in 100 cases; all were negative. Blood chemistry was done in 30 cases, but no significant variations were found.

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PATHOLOGICAL REPORT[1]

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Although the number of cases of paralysis due to drinking Jamaica ginger has reached several thousands, there have been few reports on the pathological changes. This is due, in large part, to the fact that the disease, while severely disabling, is scarcely ever fatal.

Jeter (1) studied a case complicated by pronounced arteriosclerosis and senile changes. He found a perineural exudate composed largely of lymphocytes, a few polymorpholeucocytes, many red blood cells, and a slight amount of fibrin. Turley (2), studying apparently the same case, found degenerative changes in the spinal cord and medulla. Goodale and Humphreys (3) reported autopsy observations on three cases. These patients died from causes other than the sequelae of Jamaica ginger poisoning—vegetative endocarditis, multiple abscesses of both kidneys, myocardial degeneration, tubular nephritis, and generalized arteriosclerosis. Studying this material, they found myelin and axis cylinder degeneration of the radial, sciatic, external popliteal, anterior tibial, and posterior tibial nerves. They did not find degeneration of the anterior roots of the lumbar cord of the one case examined. They were unable to confirm the observations of Jeter that the perineurium was thickened.

The writer (4) reported observations of four cases of Jamaica-ginger paralysis which came to autopsy. One of these cases, a woman aged 59, presented no complications, either clinically or pathologically, of disease other than Jamaica-ginger paralysis. Her illness ran the usual course of foot drop and wrist drop, with later development of bulbar paralysis. In addition to these four cases, five others came to autopsy at the Cincinnati General Hospital, all, however, with complicating pathological factors of severity. The cases complicated by other factors served, however, to confirm the findings reported in the uncomplicated case. There was found a myelin sheath degeneration, involving scattered areas of the anterior tibial and radial nerves of the anterior roots of the lumbar and cervical areas of the spinal cord. The anterior horn cells in the cervical and lumbar areas showed central chromatolysis as the predominating type of cell change; in addition, other cells showed diffuse chromatolysis, marked swelling, eccentric nuclei, and a few cells showed marked shrinking. In all specimens there were varying amounts of amyloid bodies. There was little or no reactive cell change and none of the characteristic signs of inflammation. Serial sections through the medulla in the uncomplicated case (4) showed central chromatolysis and diffuse chromatolysis in the dorsal motor nucleus of the vagus,

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hypoglossal nucleus, and nucleus ambiguous. There were numerous degenerated myelin fibers in the root of the vagus nerve.

Similar pathological changes have recently been found by Smith and Lillie (5) in experimental animals poisoned with tri-ortho-cresyl phosphate.

CONCLUSION

The myelin sheath degeneration, in conjunction with the central chromatolysis, suggests that the toxic agent affects primarily the peripheral nerves and, subsequently, the central nervous system. In the fatal cases, death results from an ascending paralysis which finally involves the vital centers of the medulla. The pathological changes also serve to indicate that the toxin is not of infectious origin.

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EPIDEMIOLOGICAL REPORT

By T. J. LeBlanc, Sc. D., *Associate Professor of Preventive Medicine*, and W. E. Brown, M. D., *Associate Professor of Preventive Medicine, University of Cincinnati*

Of the total number of cases of paralysis of unknown origin that were admitted to the Cincinnati General Hospital, 117 cases have been investigated epidemiologically. The accompanying questionnaire indicates the character of the information that was secured. Each of the patients was interviewed and the blanks were filled out by the authors. In each case the questionnaire was supplemented by a line of questioning to the end that each blank might be complete and as accurate as possible. This consumed a considerable amount of time, and for that reason the investigation was stopped after data on 117 cases had been secured, when it became apparent that further questioning would only confirm what was already indicated.

QUESTIONNAIRE

UNKNOWN PARALYSIS

Name ---- Age ---- Sex ---- C ---- Marital state ----
Occupation ---- Home address ----
House—Owned ---- Rented ---- Roomer ---- Weekly income ----
Time of residence at above ---- Other places in past year ----
Time of onset ---- Time admitted ----

SYMPTOMS (initial)

RESPIRATORY Gastro-enteric C. N. S.

Lab. findings: Urine ---- Feces ---- Blood ----
Spinal fluid ---- Other ----

INTAKE

Meal previous to illness ---- Date ----

Cooked	Raw	Beverages
Meat, vegetables, etc.----	Vegetables, fruits---- [OVER]	Milk, wine, beer, etc.

Beverages not with meals ---- Available sample ---- Where----
Homemade beer ---- Amount ---- Age ----
Maker ---- Method ---- Ingredients ----
Source of ingredients ----
Wine ---- Amount ---- Age ---- Kind ----
Ingredients ---- Source of ---- Containers ----
Whisky ---- Amount ---- How made ----
Where obtained ---- Kind of still ----
Jamaica ginger ---- Amount ---- Brand ----
Where obtained ---- Mixed with what----
OTHER BEVERAGES (cider, etc.)----
Dates of consumption of each kind of beverage ----
Social activities with dates (1) (2), etc----
Number (with sexes) at parties ----
Individuals affected (names and addresses) ----
Individuals NOT affected (names and addresses)----

ADDITIONAL NOTES

ONSET

The first recorded onset occurred on February 25. The onset of the disease seems sharp and clearly defined, and there was no difficulty in obtaining from the patient a reasonably accurate testimony on this point. The sudden weakness or loss of control of the feet is so dramatic that its development makes an indelible impression on the patient's mind, usually in connection with some activity that was

interrupted by the onset. After the first case, cases followed rapidly (with the exception of the last three days in February, a point of little significance that would probably disappear on questioning more patients) until March 28, when the last date of onset was recorded. Figure 1 shows the distribution of cases on a time basis with reference to the dates of onset. From this it is seen that there is little or no evidence for an original case with increasing numbers of secondary cases following after a lag period, with the epidemic gaining momentum relatively slowly as a result of such an interrupted accumulative process; nor is there any evidence of scattered cases following each other at long-time intervals. This epidemic began sharply, accumulated cases rapidly, and ended sharply. Such a course suggests that

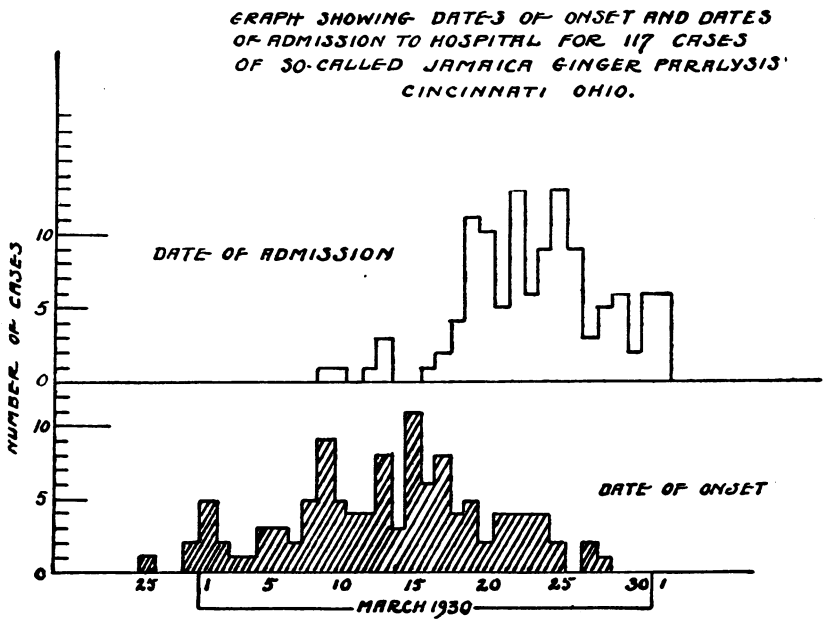

FIGURE 1

the causative agent appeared suddenly, was of sufficient potency and had such a distribution that it afflicted a large number of persons in a relatively short time, and then suddenly disappeared from the field of action. In short, this epidemic of paralysis may be said to be explosive in nature.

ADMISSIONS

The first admission was reported on March 9; and beginning March 16, cases were admitted in rapidly increasing numbers. The distribution of admissions is not as significant as that of onsets, because admission is so much a function of the patient and his attitude toward hospitalization, his income, and fortuitous circumstances, such as being picked up by the police, falling on the street, etc. In addition,

there is the factor of availability of hospital space. The sharp peaks in the distribution of admissions probably represent the opening of new wards or some other change that made additional space available.

RESIDENCE, SOCIAL STATUS, AND OCCUPATION OF PATIENTS

Practically all the patients lived within a 1-mile radius of Government Square. With some exceptions they lived in the cheaper class of rooming houses or hotels. The exceptions to this rule are important, however, since they indicate that the paralysis was not confined to a single social class. Eighteen were laborers and fourteen were cooks.

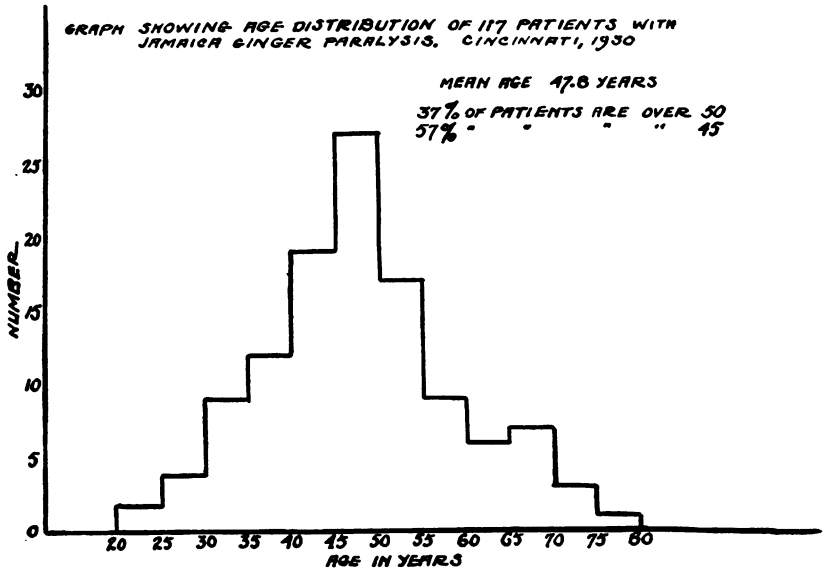

FIGURE 2

The occupations of the remainder were variously given as peddler, ragpicker, salesman, watchman, barber, packer, foreman, stockyard man, carpenter, truck driver, painter, porter, junkman, clerk, welder, cigar maker, shoemaker, collector, tailor, handbill passer, printer, pool-room worker, and some other occupations. With some exceptions, the patients represent a social status lower than would be indicated by the occupations. The exceptions, as in the case of "residence," are important. A construction foreman, with an income of $85 a week, might compare favorably in economic status with a college professor. The graphs representing income and age (Figs. 2 and 3) clearly show that the patients we are dealing with are both low in earning power and fairly advanced in years.

AGE, SEX, AND COLOR

The ages range from 21 to 79 years. The mean age is 47.8 years. To indicate the massing of the older ages, 37 per cent are over 50 years of age and 57 per cent are over 45 years of age. It is significant that there are no cases of school age or pre-school age. Out of 117 cases, 11 were females and 1 was colored. Both of these findings are epidemiologically significant and have a great bearing on the question of infection. No known infectious disease shows any such sex and color incidence.

INCOME

As will be noted from the income graph, the distribution is decidedly skew, being massed at the end representing the lowest incomes. It

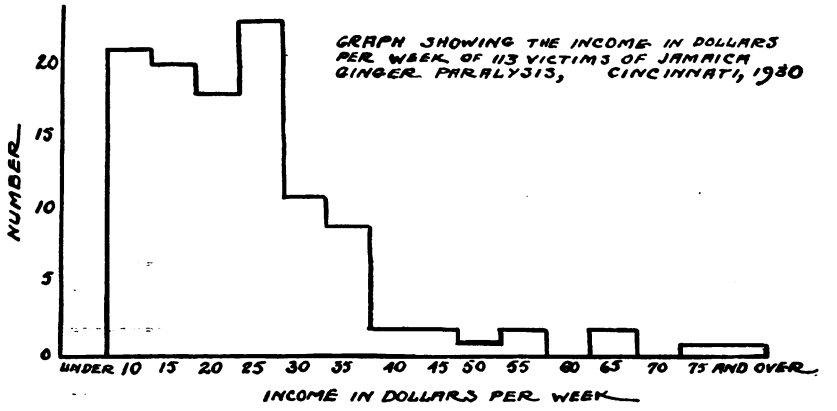

FIGURE 3

must be remembered that the incomes listed were those received by the patients when at work. The majority of these patients had not been working for from three to six months prior to the onset, and so their real income is much lower than is indicated.

DIET

In general, the diet of these patients may be said to be poor, although again there were exceptions. Some individuals, especially those in the income groups of $40 a week and over and who maintained household establishments, enjoyed diets adequate in every respect. The poorest diets were found among those persons who lived in a single room and did their own cooking. No information of real significance was gained from careful questioning on diet. No one factor was common to all the cases, not even coffee, since some of the patients drank milk or buttermilk instead of tea or coffee. Uncooked vegetables were common, but several individuals had not

eaten uncooked vegetables for months. In short, findings with reference to food intake were entirely negative.

HISTORY OF CONTACT

A striking fact was brought out during the questioning on contact, namely, that a fair proportion of the patients lived and led lonesome lives. They worked alone, lived alone, had no friends and no social contacts. Indeed it would be difficult to imagine anyone having less contact with the people about them than some of these patients. Contrasted with this group were those who were married and who maintained homes, and those who were accustomed to enjoy their leisure moments in the company of kindred spirits. Thus, excepting those instances where paralysis occurred in husband and wife, no common factor appeared in the social history.

BEVERAGE HISTORY

The first significant fact appeared when the findings on beverage intake were assembled. Of the 117 patients studied, the following beverage histories were elicited:

Home-brew, "moonshine," and Jamaica ginger	44
"Moonshine" and Jamaica ginger	27
Home-brew and Jamaica ginger	12
Jamaica ginger only	17
Home-brew and "moonshine"	4
Home-brew	3
Home-brew, "moonshine," Jamaica ginger, and others	3
Home-brew, Jamaica ginger, and others	1
"Moonshine," Jamaica ginger, and others	1
Jamaica ginger and others	2
"Moonshine"	2
No Jamaica ginger	9
No beverages (tabetic)	1

In the above table, "others" refers to such things as "canned heat," denatured alcohol ("derail," "third rail"), cologne, perfumes, toilet waters, flavoring extracts, etc.

SUMMARY OF BEVERAGE INTAKE

Jamaica ginger, either with or without others	107, or 91.5 per cent.
No Jamaica ginger	10, or 8.5 per cent.
Jamaica ginger only	17, or 14.5 per cent.

Place of obtaining beverages.—All drinks, and especially the Jamaica ginger, were obtained within a 1-mile radius of Government Square. The ginger was obtained from places grouped in "nests," one on Court Street between Vine and Walnut, one on lower Central Avenue, one

at Longworth and Plum Streets, and one at the foot of Broadway. Specific addresses of places where Jamaica ginger was purchased are on file in the college of medicine of the university.

DISCUSSION

The first question that had to be settled from an administrative standpoint was, Is this disease contagious? Upon the answer depended the arrangement of patients and the nursing. The answer appeared to be in the negative, because of—

1. No significant history of contact;
2. No convincing evidence of secondary cases;
3. Well-defined and limited age distribution;
4. Unusual sex incidence;
5. Unusual color incidence;
6. Clinical findings;
7. Laboratory findings.

The above evidence is by no means conclusive and does not rigorously exclude the possibility of some entirely new contagious disease which would exhibit such epidemiological characteristics, and of course such possibilities must ever be kept in mind. However, no matter how intriguing the prospect, the slight possibility of some exotic disease must be sacrificed for the time being to the urgent necessity for an administrative decision. When a hospital is operating almost at full capacity and a new disease appears at the rate of 5 to 10 cases per day until more than 250 cases are admitted, there is no time to marshal complete evidence, weigh fine points, nor to enter into academic discussions. In this particular case it is admitted that the decision was somewhat of a gamble, but the odds seemed to favor the noncontagious viewpoint and subsequent developments served to confirm that stand.

We will turn now briefly to a consideration of the patients and their habits. In the tabulation on beverage history it is seen that nine persons denied having used Jamaica ginger. As time passed, these patients sooner or later changed their testimony. A wife denied the fact, while her husband in another ward gave a history in which he glibly named the brand which his wife was accustomed to drink. One man who denied drinking any beverage was shown to be tabetic and moved to another ward. After three months there remained only one patient who still denied drinking Jamaica ginger, and there is reason to believe that he was not telling the truth. He felt slightly superior to the other patients and in his denial there seemed to be an element of desire to set himself apart from what he considered a low social order. In other words, the final findings in 117 cases of paralysis were that all but one person drank Jamaica ginger, and this one was suspected of being unreliable.

In connection with brands it appears that in this epidemic the brand is of no significance, since we had cases traced almost to every brand. We also had testimony to the effect that empty bottles were refilled by dealers, and it is suspected that a single bulk shipment might have been distributed to several bottling houses, later to appear under several different labels.

With reference to the time required for the causative agent to act, it is impossible to give an accurate figure. Most of the patients had been drinking Jamaica ginger for some time, many of them for a period of 10 years—a significant fact, since it shows that the Jamaica ginger per se was not responsible. The vast majority of patients continued to drink up to the time of onset, some even to the time of admission. This obscures the time required for the effects to make themselves manifest. In those cases in which Jamaica ginger was taken at one specific time and none afterwards, the time between ingestion and the onset of paralysis is highly variable. If we are to believe the testimony of these patients, the time element varied between 13 hours, the shortest, and 6 weeks, the longest. The best estimate we can offer is to say that 7 to 14 days elapsed between the drinking of the Jamaica ginger and the beginning of paralysis.

With reference to general symptoms, nothing of significance could be allocated to the respiratory system. Almost all patients had had colds or sore throats some time previous to the onset of paralysis, but none felt that these disturbances were increased either in frequency or intensity. There were some exceptions in those who had no colds or sore throats all winter. Concerning gastrointestinal symptoms, 43 patients, or 38 per cent, suffered some disturbance between the drinking of Jamaica ginger and the onset of paralysis. In many it was diarrhea with cramps, in others diarrhea without cramps, while still others had cramps only. The striking fact is the relatively low incidence of symptoms to be followed by such a grave disturbance of the nervous system.

The difference in sex incidence needs no explanation. Color differences are probably purely expressions of economic differences. Negroes tend to drink "moonshine" at $1 to $2 a gallon in preference to Jamaica ginger at 25 to 35 cents for a 2-ounce bottle. The one colored patient in our group had been given the Jamaica ginger by a white man, a tailor, who was not accustomed to drinking. He was suffering with cramps and was advised to take Jamaica ginger for relief. The negro porter was sent out to procure one bottle. The tailor took half of the contents of the bottle and disliked it so much that he gave the remaining half to the negro. Both are in the hospital, helplessly paralyzed.

It should be kept in mind that the patients here dealt with represent to some degree a selected sample, simply because they came or were

brought to the General Hospital. This selection would act to place social status and economic levels lower than if such a factor had not operated. However, many persons were treated by private physicians in homes or private hospitals, and indeed a few cases were known to have occurred among the so-called "élite." Therefore, this paralysis did not confine itself exclusively to any lower fraction of the social structure, but rather to those persons who had ingested a certain kind of Jamaica ginger, and the effects followed regardless of the person's color, creed, or social condition. From information gleaned in interviewing patients it is probably a conservative estimate to say that the total number of cases of paralysis in Cincinnati approximated 1,000.

From an administrative standpoint the epidemic was not handled as deftly as a student of the phenomenon might have wished. As soon as cases began to appear the newspapers came out with large headlines hinting at charges of manslaughter, so that almost immediately it became impossible to obtain authentic samples of suspected materials. But, finally, a most interesting piece of evidence, cases of paralysis ceased abruptly when the sale of Jamaica ginger was prohibited by ordinance.

SUMMARY

1. An epidemic of an unusual type of paralysis occurred in Cincinnati, beginning approximately February 25 and extending approximately to March 28, 1930.

2. A sample group of 117 cases was studied from an epidemiological standpoint.

3. No history of contact could be elicited to which any real significance could be attached.

4. No common factor appeared in food intake.

5. One hundred and six of the 117 patients were males, and only 1 was colored.

6. Ages ranged from 21 to 79, with a mean age of 47.8 years.

7. Upon first questioning, all but 9 patients admitted drinking Jamaica ginger. As time passed, 8 of these changed their testimony and the 1 remaining is believed to be untruthful.

8. The time between the ingesting of the Jamaica ginger and the onset of paralysis varied between 13 hours and 6 weeks. Seven to fourteen days is a fair approximation of this factor, but only an approximation.

9. The majority of patients had been regular drinkers of Jamaica ginger, some over a period of 10 years, and so Jamaica ginger per se was not the causative agent.

10. The epidemic ceased with the prohibition of the sale of Jamaica ginger.

CONCLUSIONS

This particular epidemic of an unusual form of paralysis was probably caused by the ingestion of Jamaica ginger, containing some element, unknown at that time, not ordinarily found in Jamaica ginger. This element made its appearance in the ginger probably sometime during January or February, 1930. This date, however, is only an approximation.

COURT DECISION RELATING TO PUBLIC HEALTH

Statute concerning sanitary wrapping of bread held violative of constitutional requirement regarding a law's title and subject matter.—(Minnesota Supreme Court; Egekvist Bakeries, Inc., *v.* Benson, Atty. Gen., et al., 243 N. W. 853; decided July 15, 1932.) In 1927 the legislature enacted chapter 351, entitled "An act regulating the weight of bread." In 1931 this law was amended by chapter 322, entitled "An act to amend sections 2 and 3, chapter 351, General Laws 1927, relating to the weight and sanitary wrapping of bread." In a suit brought to enjoin the enforcement of the said 1931 law, the validity of the law was questioned on the ground that it violated the requirement of the State constitution that no law should embrace more than one subject, which should be expressed in its title.

The supreme court, referring to a former decision in which the holding had been that an amendatory act, entitled as such and nothing more, must remain not only within the title but also germane to the actual subject matter of the amended act, held that the act involved in the instant case offended both limitations. The original 1927 law, according to the court, was well within its title, as it regulated the weight of bread sold or exposed for sale and nothing else, but respecting the 1931 law the court had this to say:

* * * In requiring wrapping it goes beyond both title and scope of the original act. Neither made reference to wrapping of bread. Both were concerned only with regulating weight. It is a misleading misnomer to call a later act concerning wholly the wrapping of bread amendatory of the former law concerning weight only. It is distinctly new legislation on a new subject.

The law of 1931 is not helped by the concluding phrase in the title, "relating to the weight and sanitary wrapping of bread." When that phrase is reached, the act is already limited to amending old law. The qualifying words characterize old law to be changed rather than a new subject of new law. * * *

DEATHS DURING WEEK ENDED SEPTEMBER 24, 1932

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

	Week ended Sept. 24, 1932	Corresponding week, 1931
Data from 85 large cities of the United States:		
Total deaths	6,750	6,751
Deaths per 1,000 population, annual basis	9.6	9.8
Deaths under 1 year of age	573	679
Deaths under 1 year of age per 1,000 estimated live births [1]	47	53
Deaths under 1,000 population, annual basis, first 38 weeks of year	11.2	12.1
Data from industrial insurance companies:		
Policies in force	70,528,828	74,796,694
Number of death claims	10,919	13,063
Death claims per 1,000 policies in force, annual rate	8.1	9.1
Death claims per 1,000 policies, first 38 weeks of year, annual rate	9.7	9.9

[1] 1932, 81 cities; 1931, 77 cities.

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 October 1, 1932, and October 3, 1931

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended October 1, 1932, and October 3, 1931

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931
New England States:								
Maine	3	8	2	----	1	31	0	0
New Hampshire	1	3	----	----	----	2	0	0
Vermont	1	----	----	----	1	9	0	0
Massachusetts	18	36	2	5	53	22	3	1
Rhode Island	5	8	----	----	1	4	0	2
Connecticut	5	2	----	3	2	2	0	0
Middle Atlantic States:								
New York	41	53	[1] 13	[1] 10	90	41	4	5
New Jersey	9	22	4	5	41	1	1	2
Pennsylvania	94	83	----	----	64	84	7	7
East North Central States:								
Ohio	75	116	4	2	37	22	1	0
Indiana	75	20	8	6	3	3	8	2
Illinois	83	70	7	1	14	15	3	4
Michigan	22	17	1	1	41	17	3	8
Wisconsin	14	8	16	12	49	16	1	1
West North Central States:								
Minnesota	11	21	3	----	22	4	2	2
Iowa	6	10	----	----	1	3	1	0
Missouri	67	49	----	----	----	----	3	1
North Dakota	2	5	----	----	10	----	0	1
South Dakota	1	13	----	----	2	8	0	0
Nebraska	21	14	----	1	5	2	0	0
Kansas	17	19	1	----	2	2	0	0
South Atlantic States:								
Delaware	2	3	----	3	----	----	0	0
Maryland [2] [3]	10	40	3	2	2	1	0	0
District of Columbia	3	11	----	----	2	----	0	0
Virginia	64	----	----	----	18	----	0	----
West Virginia	67	58	6	13	19	23	0	0
North Carolina	75	130	24	9	24	8	1	0
South Carolina	17	32	190	188	7	5	0	0
Georgia [3]	48	61	32	9	11	6	1	0
Florida [3]	11	16	1	----	----	17	0	0
East South Central States:								
Kentucky	74	144	----	----	47	----	0	1
Tennessee	65	103	15	13	----	2	2	1
Alabama [3]	94	116	10	----	2	8	1	1
Mississippi	35	146	----	----	----	----	2	0
West South Central States:								
Arkansas	37	47	18	----	2	1	0	0
Louisiana	24	32	10	----	5	----	1	1
Oklahoma [4]	68	111	13	1	----	1	0	0
Texas [3]	120	28	43	3	3	1	0	1

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended October 1, 1932, and October 3, 1931—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Oct. 1, 1932	Week ended Oct 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931
Mountain States:								
Montana	1	2	16	----	45	17	0	0
Idaho	5	6	----	----	----	----	0	0
Wyoming	1	----	----	----	1	----	1	0
Colorado	7	7	----	----	5	4	2	0
New Mexico	8	8	----	----	2	1	0	1
Arizona [5]	2	3	----	3	2	2	0	0
Utah [2]	----	2	----	6	1	1	1	1
Pacific States:								
Washington	8	5	3	----	6	7	0	2
Oregon	2	----	35	18	14	4	0	0
California	47	43	146	15	25	54	3	4
Total	1,466	1,726	626	329	682	451	52	49

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931
New England States:								
Maine	1	8	11	16	0	0	7	8
New Hampshire	0	22	4	10	0	0	0	0
Vermont	0	9	3	10	0	3	0	0
Massachusetts	2	112	133	103	0	0	8	4
Rhode Island	0	4	14	5	0	0	1	4
Connecticut	1	64	18	11	0	0	8	4
Middle Atlantic States:								
New York	16	275	161	104	0	0	26	39
New Jersey	22	52	56	44	0	0	11	20
Pennsylvania	110	50	238	167	0	0	71	93
East North Central States:								
Ohio	3	11	227	196	4	4	90	59
Indiana	1	6	88	35	0	7	21	18
Illinois	8	51	167	80	0	5	37	29
Michigan	8	112	113	69	0	1	22	16
Wisconsin	2	47	28	21	1	1	8	4
West North Central States:								
Minnesota	9	56	29	44	0	1	4	2
Iowa	2	13	33	14	7	11	41	3
Missouri	0	5	59	38	0	0	18	16
North Dakota	2	3	9	4	0	5	13	4
South Dakota	0	0	6	7	0	1	1	1
Nebraska	0	1	16	8	0	2	0	1
Kansas	2	0	61	35	0	0	11	14
South Atlantic States:								
Delaware	0	1	3	1	0	0	2	2
Maryland [2 3]	0	6	34	33	0	0	20	33
District of Columbia	2	4	8	6	0	0	1	0
Virginia	2	2	58	----	0	----	29	----
West Virginia	4	11	57	38	3	2	53	81
North Carolina	0	4	70	88	0	0	7	29
South Carolina	0	2	8	6	0	0	12	36
Georgia [3]	0	0	29	17	0	1	37	27
Florida [3]	0	3	2	4	0	0	1	3
East South Central States:								
Kentucky	1	1	71	62	0	0	51	102
Tennessee	0	2	66	39	3	34	36	82
Alabama [3]	1	0	57	30	0	2	24	30
Mississippi	1	0	7	26	0	4	12	31
West South Central States:								
Arkansas	0	1	11	20	0	2	12	13
Louisiana	2	0	6	16	0	1	17	59
Oklahoma [4]	1	1	19	29	0	4	40	58
Texas [3]	3	1	51	14	6	0	29	53

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended October 1, 1932, and October 3, 1931—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931	Week ended Oct. 1, 1932	Week ended Oct. 3, 1931
Mountain States:								
Montana	1	4	9	4	3	0	5	4
Idaho	0	0	2	13	0	7	2	11
Wyoming	1	1	6	0	2	0	3	1
Colorado	0	0	54	14	2	0	8	9
New Mexico	0	1	8	1	0	0	19	13
Arizona [4]	0	0	14	4	0	0	7	8
Utah [2]	0	0	2	3	0	0	1	0
Pacific States:								
Washington	3	5	17	28	5	0	6	4
Oregon	1	0	8	11	0	3	1	3
California	5	4	81	79	11	4	17	18
Total	217	955	2,232	1,607	47	105	850	1,049

[1] New York City only.
[2] Week ended Friday.
[3] Typhus fever, week ended Oct. 1, 1932, 21 cases: 1 case in Maryland, 5 cases in Georgia, 2 cases in Florida, 4 cases in Alabama, and 9 cases in Texas.
[4] Figures for 1932 are exclusive of Oklahoma City and Tulsa.
[5] Rocky Mountain spotted fever, week ended Oct. 1, 1932, 1 case in Arizona.

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	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
August, 1932										
Arkansas	-------	52	15	350	11	164	5	27	2	106
California	10	169	551	3	167	3	34	220	29	71
Kansas	5	47	4	-------	60	-------	9	72	7	67
Texas	1	184	50	1,003	-------	-------	3	77	-------	148
Virginia	2	105	-------	40	65	32	7	110	0	182

August, 1932	Cases
Chicken pox:	
Arkansas	9
California	228
Kansas	7
Virginia	21
Dysentery:	
California (amebic)	7
California (bacillary)	24
Dysentery and diarrhea:	
Virginia	559
Food poisoning:	
California	90
German measles:	
California	21
Kansas	8
Granuloma, coccidioidal:	
California	2
Hookworm disease:	
Arkansas	3
California	1
Impetigo contagiosa:	
Kansas	2
Jaundice, epidemic:	
California	2
Leprosy:	
California	2
Lethargic encephalitis:	
California	4
Kansas	1
Mumps:	
Arkansas	4
California	242
Kansas	27
Ophthalmia neonatorum:	
California	2
Paratyphoid fever:	
California	5
Kansas	6
Texas	13
Virginia	12
Psittacosis:	
California	2
Rabies in animals:	
California	32
Rabies in man:	
Kansas	1
Rocky Mountain spotted fever:	
Virginia	2
Septic sore throat:	
California	9
Kansas	4
Virginia	9
Tetanus:	
California	11
Kansas	4
Virginia	1
Trachoma:	
California	7
Kansas	3
Virginia	1
Trichinosis:	
California	34
Tularæmia:	
California	2
Virginia	2
Typhus fever:	
California	1
Virginia	3
Undulant fever:	
California	9
Kansas	6
Virginia	4
Vincent's angina:	
Kansas	8
Whooping cough:	
Arkansas	56
California	1,297
Kansas	248
Virginia	356

WEEKLY REPORTS FROM CITIES

City reports for week ended September 24, 1932

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

If the reports have not been received for the full nine years, data are used for as many years as possible but no year earlier than 1923 is included. In obtaining the estimated expectancy the figures are smoothed when necessary to avoid abrupt deviation from the usual trend. For some of the diseases given in the table the available data were not sufficient to make it practicable to compute the estimated expectancy.

Division, State, and city	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	1	0	0		0	0	0	0
New Hampshire:								
Concord	0	0	0		0	0	0	0
Nashua	0	0	0		0	0	0	0
Vermont:								
Barre	0	0	0		0	0	0	0
Burlington	0	0	0		0	0	0	0
Massachusetts:								
Boston	3	14	9		0	2	9	11
Fall River	0	2	1		0	0	1	0
Springfield	0	2	1		0	0	0	0
Worcester	0	3	1		0	1	0	3
Rhode Island:								
Pawtucket	0	0	0		0	0	0	0
Providence	0	3	1	1	0	0	2	0
Connecticut:								
Bridgeport	0	3	0		0	0	0	1
Hartford	4	1	1		0	0	0	2
New Haven	0	1	0		0	0	3	0
MIDDLE ATLANTIC								
New York:								
Buffalo	1	7	11		0	1	1	7
New York	15	69	36	7	3	23	29	72
Rochester	1	2	0		0	1	1	4
Syracuse	2	1	0		0	0	0	0
New Jersey:								
Camden	0	2	6		0	1	0	0
Newark	1	8	5	5	0	4	11	3
Trenton	1	1	0		0	0	0	5
Pennsylvania:								
Philadelphia	3	18	5		2	0	2	11
Pittsburgh	1	10	5	1	0	2	1	9
Reading	0	1	0		0	0	0	0
EAST NORTH CENTRAL								
Ohio:								
Cincinnati	0	5	1		0	0	0	1
Cleveland	10	16	2		0	1	1	5
Columbus	1	3	5		0	23	0	2
Toledo	0	3	2		0	2	0	0
Indiana:								
Fort Wayne	1	1	3		0	0	0	1
Indianapolis	3	5	0		0	0	7	6
South Bend	0	1	0		0	0	0	2
Terre Haute	0	0	1		0	0	0	1
Illinois:								
Chicago	22	51	9	1	1	1	8	16
Springfield	0	0	4		0	0	0	0

City reports for week ended September 24, 1932—Continued

Division, State, and city	Chicken pox, cases reported	Diphtheria		Influenza		Measles, cases reported	Mumps, cases reported	Pneumonia, deaths reported
		Cases, estimated expectancy	Cases reported	Cases reported	Deaths reported			
EAST NORTH CENTRAL—continued								
Michigan:								
Detroit	7	29	10		0	9	7	9
Flint	0	2	0		0	0	0	1
Grand Rapids	1	1	0		0	0	1	1
Wisconsin:								
Kenosha	3	0	0		0	1	0	0
Madison	0	0	0			2	0	
Milwaukee	8	5	2		0	1	3	4
Racine	0	0	0		0	0	0	0
Superior	1	0	0		0	0	0	0
WEST NORTH CENTRAL								
Minnesota:								
Duluth	0	0	0		0	0	0	1
Minneapolis	3	13	2		0	0	6	6
St. Paul	6	4	0		0	0	2	4
Iowa:								
Des Moines	0	1	6			0	0	
Sioux City	0	1	1			1	1	
Waterloo	0	1	0			1	1	
Missouri:								
Kansas City	0	2	1		0	0	2	3
St. Joseph	0	0	0		0	0	0	2
St. Louis	3	18	16			0	0	2
North Dakota:								
Fargo	0	0	0		0	0	0	0
Grand Forks	0	0	0			2	0	
South Dakota:								
Aberdeen	0	0	0		0	1	0	0
Nebraska:								
Omaha	0	6	6		0	1	0	6
Kansas:								
Topeka	1	0	0		0	3	4	1
Wichita	0	1	0		0	0	0	0
SOUTH ATLANTIC								
Delaware:								
Wilmington	1	0	1		0	0	0	3
Maryland:								
Baltimore	4	11	5	1	2	1	14	11
Cumberland	0	0	0		0	0	0	1
Frederick	0	0	0		0	0	0	0
District of Columbia:								
Washington	1	9	2	1	1	1	0	8
Virginia:								
Lynchburg	0	3	0		0	0	0	1
Norfolk	0	1	1		0	1	0	3
Richmond	0	11	0		0	0	0	4
Roanoke	0	3	1		0	0	0	1
West Virginia:								
Charleston	0	0	1		0	0	0	0
Huntington	0		3		0	0	0	0
Wheeling	0	0	0		0	1	0	2
North Carolina:								
Raleigh	0	2	0		0	0	0	0
Wilmington	0	1	3		0	0	0	0
Winston-Salem	0	4	0		0	6	0	2
South Carolina:								
Charleston	0	0	1	6	0	0	0	0
Columbia	0	0	1		0	1	0	3
Greenville	0	1	0		0	0	0	0
Georgia:								
Atlanta	0	4	7	7	0	0	0	2
Brunswick	0	0	1		0	0	0	0
Savannah	0	0	5	2	0	1	0	0
Florida:								
Miami	1	1	2		0	0	0	0
Tampa	0	1	4		1	0	0	2

City reports for week ended September 24, 1932—Continued

Division, State, and city	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 SOUTH CENTRAL								
Kentucky:								
Covington		0						
Lexington	1		0		0	0	0	0
Louisville	4		7		0	0	4	6
Tennessee:								
Memphis	0	3	4		0	0	0	2
Nashville	0	2	0		0	0	0	1
Alabama:								
Birmingham	0	4	4	1	0	0	0	3
Mobile	0	0	3		0	0	0	0
Montgomery	0	3	3			0	3	
WEST SOUTH CENTRAL								
Arkansas:								
Fort Smith	0	1	0			0	0	
Little Rock	0	1	2		0	0	0	3
Louisiana:								
New Orleans	0	7	9		0	0	0	5
Shreveport	0	1	0		0	2	0	2
Oklahoma:								
Muskogee	0		5		0	0	1	0
Oklahoma City	0	2	4		1	0	0	1
Tulsa	0	2	2	0		0	0	
Texas:								
Dallas	1	6	30	1	1	1	0	1
Fort Worth	0	1	5		0	2	0	1
Galveston	0	0	2		0	0	0	2
Houston	0	6	4		0	0	0	4
San Antonio	0	2	9		0	0	0	1
MOUNTAIN								
Montana:								
Billings	0	0	0		0	0	0	0
Great Falls	0	0	0		0	1	0	0
Helena	1	0	0		0	0	0	0
Missoula	0	0	0		0	0	0	0
Idaho:								
Boise	0	0	0		0	0	0	1
Colorado:								
Denver	0	6	3		0	1	4	5
Pueblo	3	1	0		0	0	0	0
New Mexico:								
Albuquerque	0	0	0		0	3	0	0
Arizona:								
Phoenix	0	0	0		0	0	0	1
Utah:								
Salt Lake City	1	2	0		0	2	5	1
Nevada:								
Reno	0	0	0		0	0	0	0
PACIFIC								
Washington:								
Seattle	1	2	0			1	0	
Spokane	2	1	0			1	0	
Tacoma	0	2	0		0	0	0	1
Oregon:								
Portland	3	4	1	1	0	1	1	3
Salem	0	0	0	3	0	1	0	0
California:								
Los Angeles	11	18	16	86	1	3	9	6
Sacramento	1	1	0		0	1	0	0
San Francisco	28	6	2	3	0	3	4	7

City reports for week ended September 24, 1932—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	0	2	0	0	0	0	1	0	0	17	24
New Hampshire:											
Concord	0	0	0	0	0	0	0	0	0	0	6
Nashua	0	0	0	0	0	0	0	0	0	0	----
Vermont:											
Barre	0	0	0	0	0	0	0	0	0	0	2
Burlington	0	0	0	0	0	0	0	0	0	0	6
Massachusetts:											
Boston	20	28	0	0	0	6	2	1	0	10	189
Fall River	2	6	0	0	0	2	1	0	0	2	24
Springfield	1	0	0	0	0	0	1	1	0	6	26
Worcester	6	7	0	0	0	0	0	1	0	1	33
Rhode Island:											
Pawtucket	1	0	0	0	0	0	0	0	0	0	16
Providence	2	5	0	0	0	1	1	0	0	11	57
Connecticut:											
Bridgeport	1	5	0	0	0	0	0	0	0	2	15
Hartford	1	3	0	0	0	0	1	0	0	2	29
New Haven	1	1	0	0	0	0	1	0	0	3	30
MIDDLE ATLANTIC											
New York:											
Buffalo	7	10	0	0	0	3	1	2	1	1	109
New York	28	31	0	0	0	66	33	23	2	110	1,183
Rochester	2	6	0	0	0	1	1	0	0	7	50
Syracuse	2	5	0	0	0	0	0	0	0	16	34
New Jersey:											
Camden	1	2	0	0	0	0	1	0	0	1	18
Newark	4	5	0	0	0	12	1	0	0	17	87
Trenton	1	2	0	0	0	0	1	1	0	4	34
Pennsylvania:											
Philadelphia	23	23	0	0	0	29	8	12	0	24	386
Pittsburgh	12	15	0	0	0	8	2	1	0	20	130
Reading	0	1	0	0	0	1	0	0	0	6	21
EAST NORTH CENTRAL											
Ohio:											
Cincinnati	8	12	0	0	0	11	1	5	1	9	114
Cleveland	12	20	0	0	0	4	3	2	0	25	123
Columbus	4	19	0	0	0	1	1	1	1	0	73
Toledo	5	9	0	0	0	2	1	0	0	7	53
Indiana:											
Fort Wayne	1	0	0	0	0	1	0	1	0	0	14
Indianapolis	4	8	1	0	0	3	3	2	0	4	91
South Bend	2	1	0	0	0	2	0	0	0	2	15
Terre Haute	0	1	0	0	0	1	1	1	0	0	20
Illinois:											
Chicago	37	54	0	0	0	34	6	4	2	37	595
Springfield	0	1	0	0	0	0	1	0	0	0	21
Michigan:											
Detroit	27	30	1	0	0	16	4	4	0	108	196
Flint	5	3	0	0	0	0	1	1	0	0	12
Grand Rapids	4	1	0	0	0	0	0	0	0	17	16
Wisconsin:											
Kenosha	1	2	0	0	0	0	0	0	0	2	6
Madison	1	1	0	0	----	----	0	1	----	2	----
Milwaukee	8	1	0	0	0	5	0	1	0	40	98
Racine	2	0	0	0	0	0	0	0	0	3	12
Superior	1	0	0	0	0	0	0	0	0	0	5

City reports for week ended September 24, 1932—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	4	1	0	0	0	0	0	0	0	0	19
Minneapolis	13	5	0	0	0	1	1	4	0	15	85
St. Paul	8	4	0	0	0	1	1	1	0	13	55
Iowa:											
Des Moines	2	4	0	0			0	0		0	33
Sioux City	0	0	0	0			0	0		0	
Waterloo	1	0	0	0			0	1		0	
Missouri:											
Kansas City	4	7	0	0	0	4	1	0	0	1	82
St. Joseph	1	0	0	0	0	1	0	0	0	0	24
St. Louis	10	11	0	0	0	18	6	7	1	4	183
North Dakota:											
Fargo	0	0	0	0	0	0	0	0	0	0	5
Grand Forks	0	0	0	0			0	0		0	
South Dakota:											
Aberdeen	0	0	0	0			0	0		0	
Nebraska:											
Omaha	2	10	1	0	0	2	0	0	0	0	45
Kansas:											
Topeka	1	0	0	0	0	0	0	0	0	0	21
Wichita	2	2	0	0	0	0	0	0	0	0	
SOUTH ATLANTIC											
Delaware:											
Wilmington	1	2	0	0	0	0	1	0	0	0	14
Maryland:											
Baltimore	6	11	0	0	0	8	7	0	0	26	173
Cumberland	0	0	0	0	0	2	0	2	0	2	6
Frederick	0	1	0	0	0	0	0	0	0	0	7
District of Col.:											
Washington	7	5	0	0	0	10	4	2	0	6	117
Virginia:											
Lynchburg	0	2	0	0	0	1	1	0	1	15	13
Norfolk	1	1	0	0	0	0	1	0	0	2	29
Richmond	4	3	0	0	0	1	1	0	1	0	45
Roanoke	1	3	0	0	0	1	0	0	0	0	18
West Virginia:											
Charleston	2	1	0	0	0	0	1	0	0	0	21
Huntington		3		0	0	0		1	0	0	
Wheeling	1	0	0	0	0	0	0	0	0	1	16
North Carolina:											
Raleigh	1	1	0	0	0	1	0	0	0	2	5
Wilmington	1	1	0	0	0	0	0	1	1	0	10
Winston-Salem	3	2	0	0	0	2	0	0	0	5	16
South Carolina:											
Charleston	0	2	0	0	0	3	3	0	0	0	20
Columbia	0	0	0	0	0	1	0	0	0	0	25
Greenville	0	0		0	0	0	0	0	0	0	
Georgia:											
Atlanta	5	11	0	0	0	4	4	1	2	2	67
Brunswick	0	0	0	0	0	0	0	1	1	0	2
Savannah	0	0	0	0	0	6	0	5	0	0	20
Florida:											
Miami	0	0	0	0	0	0	0	0	0	0	17
Tampa	0	0	0	0	0	1	0	0	0	0	19
EAST SOUTH CENTRAL											
Kentucky:											
Covington	0		0				0				
Lexington		4		0	0			0	0	0	17
Louisville		5		0	0	1		2	0	7	84
Tennessee:											
Memphis	3	6	0	0	0	3	5	4	1	1	70
Nashville	2	0	0	0	0	1	4	1	1	1	29
Alabama:											
Birmingham	4	6	0	0	0	2	3	4	0	0	57
Mobile	1	0	0	0	0	1	0	0	0	0	21
Montgomery	0	1	0	0			0	0		0	

City reports for week ended September 24, 1932—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	----	0	----
Little Rock	0	1	0	0	0	4	1	2	0	0	7
Louisiana:											
New Orleans	3	1	0	0	0	8	4	0	0	1	130
Shreveport	1	0	0	0	0	2	0	1	0	1	25
Oklahoma:											
Muskogee	1	0	----	0	0	0	----	1	0	0	----
Oklahoma City	2	2	0	0	0	2	3	0	0	0	38
Tulsa	3	0	0	0	----	----	2	0	----	0	3
Texas:											
Dallas	3	9	0	0	0	1	2	2	1	3	50
Fort Worth	2	3	0	1	0	1	1	0	0	0	29
Galveston	1	0	0	0	0	2	0	1	0	0	16
Houston	1	5	0	1	0	3	0	0	1	0	71
San Antonio	0	1	0	0	0	10	1	0	0	0	62
MOUNTAIN											
Montana:											
Billings	1	2	0	0	0	0	0	0	0	0	7
Great Falls	1	1	0	0	0	0	0	1	0	0	6
Helena	0	0	0	0	0	0	0	0	0	2	6
Missoula	1	0	0	0	0	0	0	1	0	0	4
Idaho:											
Boise	0	0	0	3	0	0	0	0	0	0	5
Colorado:											
Denver	5	12	0	0	0	7	1	3	0	5	77
Pueblo	0	0	0	0	0	1	0	1	0	3	9
New Mexico:											
Albuquerque	0	1	0	0	0	2	1	1	0	0	8
Arizona:											
Phoenix	0	1	----	0	0	4	0	0	0	0	----
Utah:											
Salt Lake City	2	1	0	0	0	3	1	2	0	2	32
Nevada:											
Reno	0	0	0	0	0	0	0	0	0	0	3
PACIFIC											
Washington:											
Seattle	7	5	0	1	----	----	0	3	----	4	----
Spokane	2	1	1	0	----	----	0	0	----	4	----
Tacoma	1	4	1	0	0	0	0	0	0	0	17
Oregon:											
Portland	4	2	2	0	0	0	1	1	0	0	53
Salem	0	0	0	0	0	0	----	0	0	0	----
California:											
Los Angeles	11	11	0	0	0	17	2	0	0	51	267
Sacramento	1	0	1	0	0	2	1	0	0	0	16
San Francisco	6	7	0	0	0	6	2	0	0	33	154

City reports for week ended September 24, 1932—Continued

Division, State, and city	Meningococcus meningitis		Lethargic encephalitis		Pellagra		Poliomyelitis (infantile paralysis)		
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases, estimated expectancy	Cases	Deaths
NEW ENGLAND									
Massachusetts:									
Boston	1	0	0	0	0	0	5	0	1
Springfield	0	0	0	0	0	0	1	1	0
MIDDLE ATLANTIC									
New York:									
New York	7	3	0	0	0	0	18	9	1
New Jersey:									
Camden	0	0	0	0	0	0	0	2	0
Newark	0	0	0	0	0	0	2	1	0
Trenton	0	0	0	0	0	0	0	1	0
Pennsylvania:									
Philadelphia	1	0	0	0	0	0	2	65	8
Pittsburgh	0	0	0	0	0	0	1	2	0
Reading	0	0	0	0	0	0	0	1	0
EAST NORTH CENTRAL									
Indiana:									
Indianapolis	1	0	0	0	0	0	0	0	0
Illinois:									
Chicago	0	0	2	1	0	0	5	4	2
Michigan:									
Detroit	1	0	1	0	0	0	4	7	0
Flint	0	0	1	1	0	0	0	0	0
Wisconsin:									
Milwaukee	1	0	0	0	0	0	1	1	0
Racine	1	1	0	0	0	0	0	0	0
WEST NORTH CENTRAL									
Minnesota:									
Minneapolis	2	0	0	0	0	0	1	0	0
Iowa:									
Des Moines	0	0	0	0	0	0	1	2	0
Sioux City	0	0	0	0	0	0	0	1	0
Missouri:									
St. Louis	2	1	0	0	0	0	1	0	0
SOUTH ATLANTIC									
District of Columbia:									
Washington	0	0	0	0	0	0	1	2	0
Virginia:									
Norfolk	0	0	0	0	0	0	0	1	0
Richmond	0	0	0	0	0	0	0	2	0
South Carolina:									
Columbia	0	1	0	0	0	0	0	0	0
Georgia:									
Atlanta	0	0	0	0	1	1	0	0	0
Savannah [1]	0	0	0	0	0	1	0	0	0
EAST SOUTH CENTRAL									
Kentucky:									
Louisville	0	0	0	0	0	0	-------	1	0
Alabama:									
Birmingham	0	1	0	0	1	0	0	0	0
Montgomery	0	0	0	0	1	0	0	0	0
WEST SOUTH CENTRAL									
Louisiana:									
New Orleans	0	0	0	0	0	0	0	1	0
Texas:									
Houston	0	0	0	0	0	1	0	0	0
MOUNTAIN									
New Mexico:									
Albuquerque	0	0	0	0	1	1	1	0	0
PACIFIC									
Washington:									
Seattle	1	0	0	0	0	0	0	1	0
Spokane	0	0	0	0	0	0	0	1	0
California:									
Los Angeles	0	1	0	0	0	0	3	0	0
San Francisco	1	0	0	1	1	1	1	2	0

[1] Typhus fever: 3 cases at Savannah, Ga.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended September 17, 1932.—The Department of Pensions and National Health of Canada reports cases of certain communicable diseases for the week ended September 17, 1932, as follows:

Disease	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				1	3				4
Chicken pox			7	10	11	12	4	8	52
Diphtheria		1	16	51	13	2			83
Dysentery				5					5
Erysipelas					1	6			7
Influenza									1
Lethargic encephalitis				1			1		2
Measles			14	34	24	2	13	6	93
Mumps	3			50	6		1	13	73
Paratyphoid fever				5			1		6
Pneumonia				2			1		3
Poliomyelitis		1	98	17	2		1		119
Scarlet fever		8	30	24	10	4	4	4	84
Trachoma				1		6		1	8
Tuberculosis	1	3	48	66	39	64	5	16	242
Typhoid fever		1	44	11	5	4	1	2	68
Undulant fever				1				1	2
Whooping cough	3		70	106	37	8	2	5	231

No report was received from Prince Edward Island.

ITALY

Communicable diseases—Four weeks ended May 29, 1932.—During the four weeks ended May 29, 1932, cases of certain communicable diseases were reported in Italy as follows:

Disease	May 2-8		May 9-15		May 16-22		May 23-29	
	Cases	Communes affected	Cases	Communes affected	Cases	Communes affected	Cases	Communes affected
Anthrax	10	9	21	20	21	20	17	14
Cerebrospinal meningitis	24	23	21	14	16	13	12	11
Chicken pox	295	111	358	123	285	114	268	126
Diphtheria and croup	376	212	338	219	326	171	380	212
Dysentery	10	10	4	4	3	2	5	4
Lethargic encephalitis	2	2	2	2	5	5	3	3
Measles	2,698	337	2,146	340	2,486	339	2,321	343
Poliomyelitis	10	10	10	7	23	16	10	9
Scarlet fever	326	103	385	128	358	113	328	129
Typhoid fever	193	121	197	125	184	123	202	123

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

(NOTE.—A table giving current information of the world prevalence of the quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for September 30, 1932, pp. 1992-2005. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued October 28, 1932, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

Cholera

China.—Amoy, week ended September 17, 1932, 31 cases, 10 deaths; Canton, week ended September 24, 5 cases, 1 death; Hankow, week ended September 10, 29 cases, 4 deaths; Hong Kong, week ended September 24, 1 case, 1 death; Nanking, week ended September 17, 13 cases, 1 death; Shanghai, week ended September 17, 47 cases, 6 deaths; Tsingtao, week ended September 3, 5 cases, 1 death.

Cholera appeared in Chefoo, China, in June, 1932, but the second case did not occur until August. The peak was reached about August 15, with about 100 cases in the port. Latest reports indicated that the disease was declining rapidly.

Philippine Islands.—A case of cholera occurred in the city of Manila, P. I., October 4, 1932. During the week ended October 1 cases of cholera were reported in the Provinces of Samar, Iloilo, and Cebu.

Plague

Hawaii Territory.—A plague-infected rat was reported September 28, 1932, at Makawao, island of Maui, Territory of Hawaii.

Yellow Fever

Brazil.—Five cases of yellow fever were reported in the State of Pernambuco, Brazil, from June 28 to July 21, 1932.

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