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ISOLATION OF POLIOMYELITIS VIRUS FROM HUMAN SERUM BY DIRECT INOCULATION INTO A LABORATORY MOUSE

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The demonstration of the presence of poliomyelitis virus in human blood has been accomplished in only one instance (*1*). This successful isolation was obtained by Ward, Horstmann, and Melnick in a study in which 75 human blood pools comprising samples from 111 individuals were injected intracerebrally into rhesus monkeys. Attempts to infect mice, guinea pigs, and rabbits with the isolated strain were unsuccessful. Moreover, despite numerous endeavors, it has been only rarely possible to demonstrate the presence of poliomyelitis virus in the blood of experimentally infected monkeys (*2*, *3*, *4*, *5*, *6*). All of the latter isolations were accomplished by inoculation of the infected monkey blood into other susceptible monkeys.

The present report deals with the isolation of an agent, which bears most of the characteristics of the poliomyelitis virus, by means of direct inoculation of laboratory mice with serum obtained from a patient who had a clinical syndrome diagnosed as poliomyelitis.

CASE REPORT

The patient, W. W., a 29-year-old bank employee, was admitted to the medical service of The New York Hospital on September 29, 1946.

Chief complaint.—Band-like pain across the abdomen on anterior flexion of the neck, of 3 day's duration.

Present Illness.—Early in September 1946, 3 weeks before admission, the patient developed a sore throat and fever accompanied by cough, productive of moderate amounts of yellowish sputum. A physical examination by his physician was reported to have disclosed "congestion of the left lower lobe." The patient remained ill at home 4 days and then became essentially free of symptoms. He

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did note, however, that he tired more easily and seemed to be less alert during the week preceding admission. On September 26 (3 days before admission) he noted that when he leaned forward he experienced a sudden pain in band-like distribution across his abdomen just below the costal margin. The pain did not radiate elsewhere and was not present posteriorly. On straightening his neck the pain would immediately disappear. Simultaneously with the onset of this pain, the patient noted a frontal headache of 1 hour's duration. He was again examined by the same physician, who had seen him during his illness 3 weeks previously, and was told that no abnormal findings were present in the chest. Forty-eight hours after the onset of the band-like pain, the patient was awakened by a dull, severe, constant aching pain over both costovertebral areas, the lower back, buttocks and hamstring muscles. This pain was slightly relieved by change in position and finally disappeared within 2 hours after application of heat. Although not conscious of nausea, he vomited orange juice taken for breakfast. His temperature was 99.5° F. at this time During the ensuing 24 hours the pain in the lower back and thigh returned. He felt drowsy and lethargic. His temperature remained between 99.5° and 101° F. and his physician noted that his neck was very slightly stiff on anterior flexion. Neither the patient nor his physician noted weakness of any muscle group. Because of the possibility of poliomyelitis, he was admitted to the hospital.

Physical examination.—The patient, a well developed and well nourished man, did not appear to be ill. The temperature was 38.2° C.; pulse rate 100; respiratory rate 20; blood pressure 140 mm. Hg., systolic, and 70 mm. diastolic. There was no evidence of petechial or other type of eruption on skin or mucous membranes. The neck was not stiff when the patient was examined in the supine position. When the patient sat erect, however, attempts at anterior flexion of the head were limited by a slight degree of stiffness. Moreover, the patient consistently complained of the band-like pain across his abdomen whenever this maneuver was attempted. There was no impairment of motion of the neck from side to side, or on rotation. Examination of the cranial nerves disclosed no abnormalities. There was no loss of power of any of the extremities, or of the facial muscles. Sensation to painful stimuli, touch, temperature, position and discrimination of two points were normal throughout the entire body. The patient could not sit up in bed at an angle of 90 degrees without flexing his knees. Straight leg raising to 90 degrees was possible without production of pain. Further flexion of the legs on the trunk produced pain in the hamstring muscles which the patient thought was a normal sensation. He was unable to touch the floor while standing with knees extended, however, prior to the illness he could accomplish this maneuver without difficulty. His gait was normal and both the superficial and deep reflexes were present symmetrically and were not abnormal.

Laboratory examination.—On admission (September 29, 1946) cerebrospinal fluid was clear, was not under increased pressure and contained 90 cells per cmm., all of which were lymphocytes. The Pandy test was negative and the fluid was sterile on culture. A repeat examination on October 7, 1946, disclosed a clear fluid which contained 15 cells: 10 lymphocytes and 5 polymorphonuclear leucocytes. Blood Hb. 16.0 gms. RBC 5.4 million. WBC 9,000 per cmm., with 71 percent neutrophiles (nonsegemented forms 5 percent), 28 percent lymphocytes and 3 percent large mononuclears. Urine—clear, pH 6.5, specific gravity 1.004, albumin 0, sugar 0, microscopic examination of the sediment disclosed no abnormalities. Bacterial cultures obtained from the nasopharynx and stool disclosed only the usual flora.

Course of illness.—During the first 48 hours after admission the patient's temperature fell to normal where it remained thereafter. Abdominal pain on anterior head flexion, the minimal stiffness of the neck and the pain along the hamstring muscles slowly disappeared during the ensuing 12 days. At the time of discharge (October 10, 1946), 13 days after admission, the patient was free from symptoms save for very questionable weakness of the hamstring muscles. He was advised to live a semiambulatory regimen at home for 1 month before returning to work. During the month subsequent to discharge, the weakness disappeared entirely and the patient was completely free of symptoms.

Examination in March 1947, approximately 5 months after the acute illness, disclosed no abnormalities.

On the basis of the clinical findings and of the examinations of the cerebrospinal fluid, it was assumed that the patient had a virus infection with involvement of the central nervous system, presumably poliomyelitis. As the entire course of the illness was so benign, he received no therapy other than rest in bed.

EXPERIMENTAL

The specimens of serum and cerebrospinal fluid obtained from W. W. on the day of his admission to the hospital (September 29, 1946) were each inoculated intracerebrally into 12 dilute brown agouti (dba) mice, 12 albino Swiss mice, 2 hamsters, and 2 cotton rats. As none of the animals presented any signs of illness, a series of blind passages was initiated. At intervals ranging from between 6 and 27 days after the original inoculation, one animal from each group was sacrificed and bacteriologically sterile 10 percent suspension of the brain and spinal cord tissues was passed into another group of animals of the same species and strain. A total of three blind passages was made. One mouse (dba) developed paralysis of the hind legs on the twenty-second day after inoculation with the second mouse brain passage (also dba) of the serum of the patient. This animal was sacrificed and, after bacterial sterility of its brain and spinal cord tissues was verified, a 10 percent suspension of combined tissues was inoculated into 6 dba and 6 albino Swiss mice. All of these inoculated animals developed paralysis after an incubation period ranging from 9 to 26 days, and three of each group died during the same period. The remaining mice were sacrificed when moribund. The infection thus established has been carried through 22 consecutive mouse passages by continuous intracerebral transfers of brain and spinal cord tissues. This strain hereafter will be referred to as the W. W. strain of virus.

The incubation period of the infection in mice (time from inoculation until onset of signs of illness) varies from 3 to 44 days. Flaccid paralysis of one or more limbs is usually observed as an initial sign and the mice may remain paralyzed for several days before death occurs. In some instances the interval from the onset of paralysis to death of the animal ranged from 1 to 2 days. At the present time the great majority of mice inoculated with the $10^{-1.0}$ dilution of infected brain and spinal cord suspension succumb to the infection. The LD_{50} titers obtained in intracerebrally inoculated albino Swiss mice were: spinal cord $10^{-3.60}$, brain $10^{-1.50}$.

Pathogenicity for other rodents.—A 10 percent suspension of ninth mouse passage infected brain and spinal cord tissues was injected intracerebrally into 4 hamsters, 4 cotton rats and 4 guinea pigs. None of the inoculated guinea pigs presented any signs of illness during a 60-day observation period. Three of the inoculated hamsters developed paralysis 7, 12 and 39 days after inoculation, respectively. Suspensions of brain and spinal cord tissues of these animals were inoculated into another group of hamsters which, in turn, developed paralysis. Up to the present time, the virus has been carried through seven hamster passages.

Two of the four cotton rats, inoculated with the same mouse brain and spinal cord suspension which produced disease in the hamsters, developed paralysis 15 and 20 days after inoculation, respectively. Suspensions of infected brain and spinal cord tissues were injected into another group of cotton rats, one of which died on the eighth day after inoculation. The remaining animals (30 days after inoculation) have shown no signs of illness. In another experiment three cotton rats inoculated intracerebrally with the 13th mouse passage of infected brain and spinal cord suspension developed paralysis on the fifth day after inoculation. The second passage cotton rats became paralyzed on the sixth to seventh days after inoculation. Thus far the virus has been carried through seven cotton rat passages.

Pathogenicity of the virus for rhesus monkeys.—Monkey No. 1 was inoculated intracerebrally with 0.5 ml. of a 10 percent suspension of seventh mouse passage infected brain and spinal cord tissues. During the 60-day observation period the monkey showed no signs of illness, but its serum, drawn 30 days after inoculation, neutralized the W. W. strain in contrast to the pre-inoculation serum which exhibited no neutralizing power (see below). Monkey No. 2 was inoculated intracerebrally with 0.5 ml. of a 10 percent suspension of ninth mouse passage infected brain and spinal cord tissues. The monkey developed paralysis of the left wrist on the thirteenth and paralysis of the left forearm on the fourteenth day after inoculation. The monkey became progressively weaker and was sacrificed on the twentieth day at which time both legs were also paralyzed. Several blocks of spinal cord tissue were embedded in celloidin and paraffin. About 100 sections, stained according to Nissl's and Einarson's methods, were examined. Destruction of nerve cells, proliferation of glia, and perivascular infiltration were present in virtually every section of the spinal cord. The changes were uniformly characteristic of poliomyelitis.

Isolation of virus from blood of monkey.—Serum obtained from monkey No. 2 on the fourteenth day after inoculation, during the initial stage of paralysis, was inoculated intracerebrally into 12 dba mice and 6 albino Swiss mice. None of the inoculated mice showed any signs of illness but 1 albino Swiss mouse and 2 dba mice were sacrificed 13 and 14 days after inoculation and suspensions of the brain and spinal cord tissues were inoculated into 3 groups, each containing 6 albino Swiss mice. In two of these groups of mice, no symptoms were noted, but among 6 mice inoculated with one of the brain and spinal cord suspensions from the dba mice, one developed paralysis on the thirteenth and one on the sixteenth day after inoculation. These two

mice were sacrificed and the virus carried through nine mouse generations. The virus was identified by a neutralization test in which the human W. W. convalescent serum and the postinoculation serum of monkey No. 2 completely neutralized a 10 percent brain and spinal cord suspension.

IDENTITY OF THE VIRUS

Neutralization tests.—The patient W. W. was bled on October 25, 1946, 15 days after discharge from the hospital, and his serum submitted to neutralization test against the eighth mouse passage of the W. W. strain of virus. Unfortunately no more acute phase serum was available and a normal rabbit serum was used as control. Included in the test were lymphocytic choriomeningitis monkey antiserum, and a serum sample obtained from EH, an individual who had suffered the year before from a paralytic disease which resembled poliomyelitis. The technique described by Brown and Francis (7) was followed, except that the final serum-virus mixtures contained 10 percent concentration of infected brain and spinal cord tissues which had been incubated for 90 minutes at 37° C. and had been subsequently kept at room temperature for 30 minutes.

The results of the first test are summarized in table 1. The majority of the mice inoculated with mixtures of W. W. strain and normal rabbit serum, or with W. W. strain and lymphocytic choriomeningitis antiserum, succumbed to infection. In contrast, all mice inoculated with the mixture of W. W. strain and W. W. convalescent serum survived and partial, but significant, degree of protection was provided by the EH serum. The results of this first test indicate that the W. W. virus apparently is not related to lymphocytic choriomeningitis and that it was derived from the serum of W. W.

TABLE 1.—*Preliminary neutralization test with the W. W. strain of virus*

Serum	Number of mice dying at days postinoculation—								Mortality ratio of mice
	4	5	8	11	13	14	16	19	
W. W. convalescent	-----	-----	-----	-----	-----	-----	-----	-----	0/10
EH	1	1	-----	-----	-----	-----	-----	-----	2/10
Lymphocytic choriomeningitis	1	-----	-----	2	3	2	-----	1	9/10
Normal rabbit	-----	2	1	-----	1	1	1	1	7/10

In order to establish the relationship of the W. W. strain to a recognized type of poliomyelitis virus, convalescent serum of W. W. and normal monkey serum were mixed respectively with equal amounts

of serial tenfold dilutions of MEF1 poliomyelitis virus (*8*). The respective mixtures were incubated as indicated above and inoculated intracerebrally into mice. The LD_{50} titers were: virus plus W. W. serum $<10^{-1.00}$; virus plus normal monkey serum $10^{-3.60}$. These results seem to indicate that the W. W. serum neutralized not only the W. W. strain but also the recognized MEF1 poliomyelitis strain of virus.

As a further step toward identification, a neutralization test was performed with the ninth mouse passage of the W. W. strain, and with Lansing and MEF1 immune monkey sera. Included in the same test were pre-inoculation and post-inoculation samples of monkey No. 1 sera (see above). Three dilutions of the W. W. strain were mixed respectively with equal amounts of undiluted serum and after incubation each of the mixtures was inoculated into 8 to 10 mice. The results of the test are shown in table 2.

TABLE 2.—*Neutralization test with the W. W. strain of virus and homologous and heterologous poliomyelitis sera*

Serum	Mortality ratio of mice inoculated with mixtures of serum and dilutions of virus—		
	1:10	1:50	1:100
Lansing and SK [1]	2/10	1/10	0/10
MEF1	0/8	0/8	0/8
Monkey No. 1, preinoculation	7/10	5/10	10/10
Monkey No. 1, postinoculation	1/10	1/10	0/10
W. W. convalescent	0/10	------	------
Normal monkey pool	8/8	8/8	6/8

[1] Pooled sera of monkeys surviving infection with the Lansing strain of virus and subsequently hyperimmunized with the monkey "Yale"-SK strain of poliomyelitis.

It may be observed that both the Lansing-SK and MEF1 sera showed a definite neutralizing ability against the W. W. strain of virus. Furthermore, serum of monkey No. 1 obtained after inoculation with the W. W. virus showed a definite neutralizing power against the latter strain as compared to the pre-inoculation sample.

Finally, to determine whether a serological relationship existed between the W. W. and Lansing (*9*) viruses, the above sera were tested against a Lansing infected mouse brain suspension. In addition, a MV-PCMS-XI poliomyelitis immune monkey serum was tested against both Lansing and W. W. strains. Also included in the test were sera of three clinically diagnosed poliomyelitis patients (Ta, Fl, and He) obtained about one and one-half to three and one-half months after onset of illness. These sera were tested against both W. W. and Lansing viruses. The results of the test are summarized in table 3.

TABLE 3.—*Comparative neutralization test with the W. W. and Lansing strains of virus and human and monkey sera*

Sera		Mortality ratio of mice inoculated with mixtures of undiluted serum and dilutions of respective strains					
Species	Virus	1:10		1:50		1:100	
		W. W.	Lansing	W. W.	Lansing	W. W.	Lansing
Monkey	Lansing and SK [1]	2/10 [2]	1/8	1/10 [2]	3/8	0/10 [2]	0/8
	MEF1	0/8 [2]	0/8	0/8 [2]	0/8	0/8 [2]	0/8
	MV-PCMS-XI	5/9	7/8	3/10	7/8	4/10	6/8
	Normal	9/10	8/8	8/10	8/8	6/10	6/7
Human	Ta: Polio. paralyzed	2/8	1/9	1/8	-----	0/8	-----
	Fl: Polio. convalesc.	10/10	10/10	------	-----	------	-----
	He: Polio. convalesc.	10/10	9/9	------	-----	------	-----
	W. W.: W. W. convals.	0/10	0/10	------	-----	------	-----

[1] See legend, table 2.
[2] Data from table 2.

As may be seen the Lansing-SK and MEF1 sera showed definite neutralizing power against both Lansing and W. W. strains. It is of interest, however, that the MV-PCMS-XI immune serum exerted only a negligible neutralizing effect against both Lansing and W. W. strains. Among the human sera tested, the convalescent W. W. serum neutralized both the W. W. and Lansing strains. Of the Ta, Fl, and He sera, only Ta provided protection. A comparison of the clinical histories of these three patients indicated that Ta had a severe case of poliomyelitis and was still paralyzed at the time of bleeding, whereas the other two individuals had been bled during convalescence when most, if not all, signs of illness had subsided.

Cross-immunity studies.—In experiment A (table 4) a group of 21–28 day old albino Swiss mice was immunized against the W. W. strain by weekly intraperitoneal inoculations with 0.5 ml. of a 10 percent suspension of W. W. infected brain and spinal cord tissues. Each mouse received four injections. Ten days after the last inoculation the immunized mice, as well as an equal number of non-immunized control mice of the same age and strain, were inoculated intracerebrally with a 10 percent suspension of MEF1 infected mouse brain and spinal cord tissues. In experiment B (table 4) two groups of mice were immunized as above against the Lansing and MEF1 viruses, and then challenged with the homologous and W. W. strains respectively.

The results of the two experiments, summarized in table 4, indicate that mice immunized against the W. W. strain showed definite resistance to challenge with the MEF1 strain, and conversely mice immunized against the MEF1 strain were resistant to challenge with the W. W. strain. Furthermore, the degree of protection provided by

immunization with the Lansing strain was equally significant, regardless of whether the W. W. or homologous strains were employed for challenge.

TABLE 4.—*Cross-immunity tests with the W. W., MEF1 and Lansing strains*

Experiment	Mice immunized against—	Mortality ratio of mice challenged [1] by intracerebral route with the following strains		
		W. W.	MEF1	Lansing
A	W. W.	---	2/13	---
	Normal controls	---	13/13	---
B	Lansing	1/13	---	3/9
	MEF1	1/9	0/7	---
	Normal controls	15/15	6/6	11/11

[1] With a 10-percent suspension of infected mouse brains and spinal cords.

Attempts to isolate a virus from the colony of normal dba mice.—The possibility existed that the dba mice in which the W. W. strain was isolated had been harboring this virus prior to inoculation with the acute phase human and monkey sera. Accordingly uninoculated mice were studied for the presence of the W. W. virus. Two groups of dba mice were selected and from each group one or two mice were sacrificed. A 10 percent suspension of the brain and spinal cord tissues was then inoculated into another group of 6 dba mice. One or two of the inoculated mice were likewise sacrificed on the seventh to tenth days after inoculation. These passages were carried through four generations. The mice were observed for 60 days but none of the animals showed any signs of illness.

COMMENT AND SUMMARY

On the basis of the evidence which has been presented, it would seem that the W. W. virus isolated in the dba mice was present in the circulating blood of the patient and is a strain of poliomyelitis virus.

In support of the human origin of the virus are the following facts:

1. The human serum, from which the virus was apparently isolated by blind passage through dba mice, was obtained during an acute illness characterized by the clinical and cerebrospinal fluid findings customarily observed in poliomyelitis.

2. The patient's convalescent serum contained neutralizing antibodies against the W. W. virus, as well as against two recognized strains of poliomyelitis virus (MEF1 and Lansing).

3. By the use of the same technique employed with the acute phase human serum, it was possible to isolate an apparently identical virus from the blood of a monkey which, following the intracerebral inoculation with the W. W. virus, developed the characteristic clinical and histopathological evidences of acute poliomyelitis.

4. Absence of the W. W. virus in the normal dba mouse colony was indicated by the fact that no signs of illness were elicited in the normal dba mice tested by similar blind passage technique.

Unfortunately it was not possible to test the patient's pre-illness or acute phase sera for the presence of neutralizing antibodies against the W. W. virus.

The classification of the W. W. strain as a virus of poliomyelitis appears justified on the basis of the following facts:

1. Inoculation of the virus into two rhesus monkeys was followed by the development of neutralizing antibodies against the W. W. virus in one, and the characteristic and histopathological changes of acute poliomyelitis in the other.

2. Both the W. W. virus and the Lansing virus were neutralized to approximately the same degree by Lansing-Sk, MEF1 and convalescent serum from one patient with clinical poliomyelitis. In addition, the W. W. virus was also neutralized by the convalescent serum of another patient who had had clinical poliomyelitis. Neither the W. W. virus nor the Lansing virus were neutralized by convalescent sera from two other patients with clinical poliomyelitis.

3. Mice immunized with the W. W. virus were protected to a significant degree against subsequent challenge with MEF1 poliomyelitis virus. Conversely, mice immunized against MEF1 as well as against the Lansing virus, were similiarly protected against subsequent challenge with the W. W. virus.

Available literature contains no reports of successful isolation of poliomyelitis virus from the blood of humans or of monkeys by direct inoculation of laboratory mice. Moreover, in only one investigation (*10*) has the virus been transmitted directly from a human source to mice by the inoculation of material derived from feces. In other experiments poliomyelitis virus of human origin has been adapted to the mouse only after passage in the cotton rat (*8, 9, 11*) or in the monkey (*10*).

On the basis of the observations given in the present report, it would seem that the technique of blind passage through mice should be further explored as a possible method for the direct isolation of poliomyelitis virus from human sources.

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[3] The Lansing strain was obtained from Dr. Charles Armstrong after the W. W. strain was established in mice and its characteristics were recognized to be similar to poliomyelitis virus. The dba mice used in these studies were procured from Rockland Farms, New City, N. Y., and the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine. Inquiry revealed that in neither of these places was any work being conducted with poliomyelitis virus.

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THE EFFECT OF TOPICALLY APPLIED FLUORIDES ON DENTAL CARIES EXPERIENCE [1]

V. REPORT OF FINDINGS WITH TWO, FOUR AND SIX APPLICATIONS OF SODIUM FLUORIDE AND OF LEAD FLUORIDE

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Previous reports (*1*, *2*, *3*, *4*) in this series on the effect of topically applied fluorides on dental caries experience indicated:

(1) Approximately 40 percent reduction in dental caries incidence is effected when the initial application of a 2-percent sodium fluoride solution to the teeth is preceded by dental prophylaxis and subsequent applications are made at the rate of one or two per week to a minimum of 7 and a maximum of 15.

(2) The caries prophylactic effect of 15 applications is not greater than that obtained with 8 applications.

(3) Two, four and six applications of a 2-percent sodium fluoride solution effect 9, 20 and 21 percent reductions, respectively, when the initial application is not preceded by dental prophylaxis.

Jordan and his associates, using a 2-percent sodium fluoride solution and the same technique of application described here, reported 5, 10, and 21 percent reductions in caries incidence respectively following one, two and three applications (*5*). It is the purpose of this report to present the results of additional studies designed to determine the minimum number of applications of 2-percent sodium fluoride required to achieve maximum caries reduction, and to test the comparative effectiveness of lead fluoride. The results of laboratory investigations have suggested that lead fluoride might be more effective than sodium flouride as a topical agent for the prevention of dental caries (*6*, *7*).

Briefly, the results of the study indicate that four applications of a 2-percent solution of sodium fluoride, applied as described in the section on materials and methods, and preceded by dental prophylaxis, are the minimum number required to give maximum reduction in caries incidence. Two applications of sodium fluoride resulted in 21.7 percent less initial caries in treated teeth than in untreated teeth, four treatments reduced initial caries in treated teeth by 40.7 percent, and six resulted in a 41.0-percent reduction. No significant reduction was shown with two, four, or six applications of a saturated lead fluoride solution.

[1] From the Dental Section, States Relations Division, United States Public Health Service, Washington, D. C., in cooperation with the Division of Dental Hygiene, Ohio State Department of Health, and the Troy-Miami County Department of Health. Appreciation is expressed for the cooperation of Messers. T. E. Hook and Robert Duncan, superintendents of schools at Troy and West Milton, Ohio, respectively, and to Rev. A. J. Mentick, St. Patrick's School, Troy, Ohio.

MATERIALS AND METHODS

During a four-month period beginning in January 1946, six groups of Troy and West Milton, Ohio, school children received a series of topical fluoride applications to the teeth in half the mouth. Three of the groups received two, four, and six applications of a 2-percent sodium fluoride solution, respectively. The other three groups received two, four, and six applications of an 0.06-percent lead fluoride solution. Half the children in each group were treated on the right side of the mouth, the other half on the left side. A dental prophylaxis, consisting of necessary scaling and polishing, was administered to each child prior to the initial fluoride treatment. The teeth in the untreated mouth quadrants served as controls. A detailed dental examination was made by one of the authors (D. J. G.) for each of the 1,782 children in the 6 groups before the first topical application was administered.

The examinations were made with a No. 4 plane mouth mirror and a double end No. 5 explorer under artificial light, and with compressed air available for use at the discretion of the examiner. The treatment consisted of isolating the teeth of the treated side with cotton rolls, drying with compressed air, and wetting the crown surfaces with the fluoride solution. The solution was allowed to dry in air for approximately 4 minutes; then the cotton rolls were removed and the child dismissed. In general, the treatments were given twice a week over a 1-, 2- or 3-week period. All treatments were administered by dental hygienists under the supervision of a dentist.

One year after the series of fluoride applications were completed, the teeth of the children in the six treatment groups were reexamined. At the time of reexamination the examiner had no knowledge as to which teeth had been treated and which were untreated. Of the 1,782 children included in the initial study groups, 1,555 were available for reexamination. Analysis of the data on caries experience is confined to the erupted permanent teeth present in the mouth at the time of the initial examination.

The children ranged in age from 7 to 15 years, and included approximately 90 percent of the children enrolled in elementary and first year high school grades in the public and parochial schools of Troy and West Milton, Ohio. Less than 3 percent of the children were colored. They are distributed throughout the six studies reported, and are included in the analysis. The number of males and females in each age group is approximately equal. The age distribution of the children according to the number of sodium or lead fluoride treatments received, is shown in table 1. Age classification refers to age at the time of the initial examination and treatment.

TABLE 1.—*Age distribution of 1,555 Miami County, Ohio, school children available for reexamination at the end of a 1-year study period, according to the number of sodium fluoride or lead fluoride applications each received*

Fluoride solution and number of applications	All ages	Children by age at time of treatment								
		7	8	9	10	11	12	13	14	15
Sodium fluoride:										
2 applications	301	33	37	36	41	36	51	32	26	9
4 applications	247	19	20	34	34	36	28	28	28	20
6 applications	259	35	30	26	40	40	31	25	27	5
Total	807	87	87	96	115	112	110	85	81	34
Lead fluoride:										
2 applications	272	31	39	34	33	33	42	24	24	12
4 applications	214	15	19	27	36	32	21	26	28	10
6 applications	262	32	47	32	32	34	21	30	26	8
Total	748	78	105	93	101	99	84	80	78	30

FINDINGS

Topically applied sodium fluoride.—Table 2 presents the caries experience during the study year in untreated and sodium fluoride treated permanent teeth, by upper and lower mouth quadrants and by number of fluoride applications.

TABLE 2.—*Dental caries experience during the 1-year period ending April 1947, for permanent teeth in the* sodium fluoride-*treated and untreated mouth quadrants of the mouths of 807 Miami County, Ohio, school children*

Treatment groups by treated and untreated quadrants	Number of noncarious teeth, April 1946	New DF [1] teeth, April 1947	DF surfaces in new DF teeth	New DF surfaces in previously carious teeth	Total new DF surfaces
Upper					
2 applications:					
Treated quadrant	1,058	109	120	78	198
Untreated quadrant	1,082	144	152	72	224
4 applications:					
Treated quadrant	911	70	77	60	137
Untreated quadrant	917	135	148	61	209
6 applications:					
Treated quadrant	857	58	64	45	109
Untreated quadrant	846	89	93	57	150
Lower					
2 applications:					
Treated quadrant	1,174	89	99	86	185
Untreated quadrant	1.168	109	125	94	219
4 applications:					
Treated quadrant	985	70	84	49	133
Untreated quadrant	972	101	112	86	198
6 applications:					
Treated quadrant	967	34	42	47	89
Untreated quadrant	971	67	76	50	126

[1] DF—Carious (decayed or filled).

In the upper jaws of the children who received two applications of sodium fluoride, 109 fluoride-treated teeth became carious, while 144 untreated teeth showed new caries, a difference of 24.3 percent. In the upper mouth quadrants of the 4-treatment group, 70 treated teeth became carious as compared with 135 teeth not treated, a difference of 48.1 percent. In the group that received six treatments there were 58 newly carious teeth in the upper treated quadrants, and 89 in the untreated quadrants, a difference of 34.8 percent.

In the lower mouth quadrants of the children given 2 sodium fluoride applications, 89 newly carious teeth occurred on the treated side and 109 on the untreated side, a difference of 18.3 percent. In the 4-treatment group initial caries occurred in 70 treated lower teeth as compared with 101 untreated teeth, a difference of 30.7 percent. The group receiving 6 applications developed 34 carious teeth in the fluoride-treated lower quadrants and 67 in the untreated quadrants, a 49.3 percent difference.

Combining initial caries experience in upper and lower mouth quadrants, there is a difference between treated and untreated permanent teeth of 21.7 percent achieved by two applications of sodium fluoride, 40.7 percent by four applications and 41.0 percent by 6.

Table 3 shows the percentage less new caries experience in sodium fluoride-treated teeth than in untreated teeth in upper and lower jaws, and in both jaws, during the 1-year period.

TABLE 3.—*Percentage less new caries experience during the 1-year period ending April 1947 in the permanent teeth of* sodium fluoride-*treated mouth quadrants of a group of Miami County, Ohio, school children*

Number of applications	Upper jaw	Lower jaw	Both jaws
	Percentage less newly carious teeth		
2	24.3	18.3	21.7
4	48.1	30.7	40.7
6	34.8	49.3	41.0
	Percentage less newly carious surfaces in previously carious teeth		
2	−8.3	8.5	1.2
4	1.6	43.0	25.9
6	21.1	6.0	14.0

Data showing the occurrence of newly carious surfaces in previously carious teeth are also presented in tables 2 and 3. The number of additional surfaces becoming carious was less in the sodium fluoride-treated than in the untreated carious teeth in all three study groups. There were 1.2 percent less additional carious surfaces in previously carious teeth associated with 2 applications, 25.9 percent with 4 applications, and 14.0 percent with 6.

The difference in the case of the six-treatment group might be expected to be at least as large as that for the four-treatment group, based on the parallel experience of the two groups in numbers of newly carious teeth that developed during the study year. This irregular pattern of difference was noted in the previous study in this series in which prophylaxis was omitted from the treatment procedure (*4*). The reason for the inconsistency is not known.

Topically applied lead fluoride.—The caries experience in lead fluoride-treated and untreated permanent teeth for the 1-year study period by upper and lower mouth quadrants, and by groups of children who received two, four and six applications is presented in table 4.

TABLE 4.—*Dental caries experience during the 1-year period ending April 1947 for permanent teeth in the* lead fluoride-*treated and untreated mouth quadrants of the mouths of 748 Miami County, Ohio, school children*

Treatment groups by treated and untreated quadrants	Number of noncarious teeth, April 1946	New DF [1] teeth, April 1947	DF surfaces in new DF teeth	New DF surfaces in previously carious teeth	Total new DF surfaces
Upper					
2 applications:					
Treated quadrant	975	102	116	63	179
Untreated quadrant	972	110	120	60	180
4 applications:					
Treated quadrant	754	70	79	59	138
Untreated quadrant	785	70	75	71	146
6 applications:					
Treated quadrant	841	76	85	51	136
Untreated quadrant	828	67	73	59	132
Lower					
2 applications:					
Treated quadrant	1,075	90	100	80	180
Untreated quadrant	1,079	94	106	82	188
4 applications:					
Treated quadrant	868	55	62	63	125
Untreated quadrant	858	57	68	68	136
6 applications:					
Treated quadrant	959	42	49	58	107
Untreated quadrant	960	52	56	63	119

[1] DF—carious (decayed or filled).

Among permanent teeth in upper mouth quadrants, 102 treated teeth in children given 2 applications of lead fluoride as compared with 110 untreated teeth became carious during the study year, a difference of 7.3 percent. In the four-treatment group the same number of newly carious teeth developed in treated as in untreated mouth quadrants, while in the children given 6 lead fluoride applications 76 treated and only 67 untreated permanent teeth decayed, a difference of 13.4 percent in favor of untreated teeth.

In lower mouth quadrants there were 90 new carious treated teeth as compared with 94 untreated teeth among children receiving 2

topical lead fluoride applications, a 4.3 percent difference. In the group receiving 4 topical applications 55 permanent treated teeth became carious as compared with 57 untreated teeth, a difference of 3.5 percent. In the six-treatment group 42 treated and 52 untreated teeth developed initial decay, a difference of 19.2 percent.

Table 5 combines the initial caries experience for the teeth in upper and lower mouth quadrants and in both jaws. Two, four and six applications of lead fluoride resulted in 5.9, 1.6 and 0.8 percent less newly carious teeth, respectively.

TABLE 5.—*Percentage less new caries experience during the 1-year period ending April 1947 in the permanent teeth of* lead fluoride-*treated mouth quadrants of a group of Miami County, Ohio, school children*

Number of applications	Upper jaw	Lower jaw	Both jaws
	Percentage less newly carious teeth		
2	7.3	4.3	5.9
4	0.0	3.5	1.6
6	−13.4	19.2	0.8
	Percentage less new carious surfaces in previously carious teeth		
2	−5.0	2.4	−0.7
4	16.9	7.4	12.2
6	15.3	7.9	11.5

Data on the occurrence of newly carious surfaces in previously carious teeth are also presented in tables 4 and 5. The groups receiving two, four and six applications of lead fluoride showed, respectively, a 0.7-percent increase, and 12.2 and 11.5 percent reductions in the number of additional carious surfaces in teeth that were previously carious.

The findings of this study indicate that a 0.06-percent solution of lead flouride, topically applied as described, is not effective in inhibiting dental caries.

SUMMARY

A study of the incidence of dental caries in six groups of children in Miami County, Ohio, in which three of the groups received two, four and six topical applications of a 2-percent solution of sodium fluoride and three groups received two, four and six topical applications of a 0.06-percent lead fluoride solution to the teeth in half the mouth has been presented and analysed. The fluoride treatment series was preceded by a dental examination and prophylaxis and applications were administered at the rate of two per week. The follow-up ex-

aminations were made 1 year after completion of the fluoride treatments. The teeth in the untreated upper and lower mouth quadrants served as controls.

Analysis of the data for the first study year indicate:

(1) The incidence of initial caries in permanent teeth noncarious at the time of treatment with two, four and six topical applications of sodium fluoride, was 21.7, 40.7 and 41.0 percent less, respectively, in treated than in untreated teeth.

(2) The number of additional permanent tooth surfaces which became carious in previously carious teeth during the 1 year period was 1.2, 25.9 and 14.0 percent less in sodium fluoride treated carious teeth, given two, four and six applications, respectively, than in untreated teeth.

(3) The results of this investigation together with those of studies previously reported indicate that four topical applications of sodium fluoride preceded by dental prophylaxis affords maximum reduction in dental caries incidence (approximately 40 percent).

(4) Two, four and six topical applications of a 0.06-percent solution of lead fluoride are not associated with a significant reduction in the incidence of dental caries.

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RECOVERY OF ORNITHOSIS VIRUS FROM PIGEONS IN BALTIMORE, MARYLAND [1]

By DORLAND J. DAVIS, *Surgeon, United States Public Health Service,* and C. LEROY EWING, *Director, Bureau of Laboratories, Baltimore City Health Department*

The infection of pigeons (*Columba livia*) with a virus of the psittacosis-lymphogranuloma venereum group was first reported in South Africa by Coles (*1*) and in the United States by Pinkerton and Swank (*2*) in 1940. Later Meyer (*3, 4*) related the infection to human disease by isolating virus from a flock of racing pigeons and the same virus from the lung of a human who had been exposed to the birds and who developed a fatal pneumonitis. The name "ornithosis" has been proposed (*4*) for the disease contracted from birds other than psittacine birds in order to distinguish it from psittacosis.

Although nearly all reported cases of ornithosis in which the source of the virus could be traced have been contracted from privately owned pigeons, there remains the question of possible danger to humans from exposure to feral or wild pigeons present in or about public parks, squares, or buildings. In order to secure data on which to estimate this potential danger to the population of Baltimore, Md., an investigation of the infection among wild pigeons was undertaken in 1945.

Although some human infections with this virus may not have been recognized, only two cases of ornithosis were reported to the Baltimore City Health Department in the years 1944 and 1945. The first case was a 38-year-old white female who became ill on July 6, 1944, with evidence of a severe pneumonitis. Her serum fixed complement in the presence of psittacosis antigen.[2] The patient's son raised pigeons in the back yard of their home. Ornithosis virus was isolated from two of these pigeons at the National Institute of Health.

The second reported case, a 43-year-old colored male, had onset of disease on September 1, 1945, and exhibited the signs and symptoms of a moderately severe pneumonitis. His serum taken during the third week of illness fixed complement in a dilution of 1:256 in the presence of psittacosis antigen. The patient's son raised pigeons in the back yard of his home but was not sick himself, and his serum did not contain demonstrable antibodies against psittacosis virus. Pigeons from this loft could not be secured for examination.

The fixation of complement in the presence of psittacosis antigen by serum from an infected bird has been a simple and useful method of

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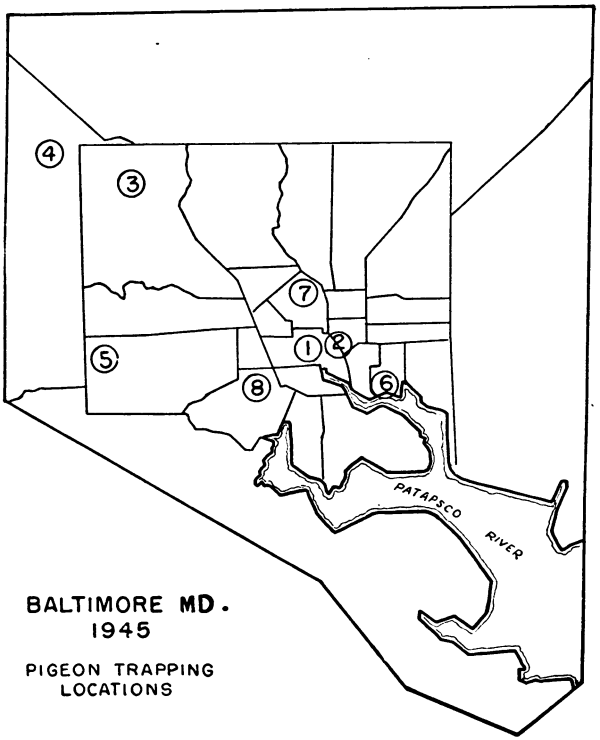
[1] From the Division of Infectious Diseases, National Institute of Health and the Baltimore City Health Department.

Valuable assistance was received in this study from Dr. J. Wilfrid Davis, Director, Bureau of Communicable Diseases and Dr. Wilmer H. Schulze, Director Sanitary Section, Baltimore City Health Department.

[2] Test performed by Dr. K. F. Meyer, George Williams Hooper Foundation, San Francisco, Calif.

estimating the number of infected pigeons in a given flock. However, virus has been isolated from birds whose serum was negative (4), possibly because the serum had been tested in the early stages of infection. Attempts at isolation of virus have frequently been unsuccessful in birds whose serum reacted positively, probably indicative of a past infection.



Although details of the mode of excretion of the virus from pigeons are not completely known, isolation of infective virus from birds would appear to yield more definite information concerning their infectivity than the reaction of serum. Actual isolation of the virus, therefore, was attempted from each of 100 wild adult pigeons trapped at various locations in the city of Baltimore.

The accompanying map shows the eight locations of capture of the 100 pigeons. The first pigeons were trapped on July 12, and the trappings continued until September 18, 1945. Wire cages operated by personnel of the Baltimore City Health Department proved successful in trapping them after some early difficulties.

In order to reduce the risk of handling any birds which might be infected, the birds were killed immediately by being placed in a paper bag with a wad of cotton soaked with chloroform. They were then

kept at approximately 4° C. until examined 1 to 7 days later. The laboratory studies were done in the National Institute of Health at Bethesda, Md., where special precautions had been established to minimize the risk to personnel.

As shown in table 1, a virus belonging to the psittacosis group was recovered from 15 of the 100 pigeons examined. Infected birds were found in four of eight separate locations in the city. This would suggest that the infection is ubiquitous throughout the city.

TABLE 1.—*Percentage of captured wild pigeons infected with virus of ornithosis from each capture location in Baltimore, Md.*

Capture location number	Address	Number of pigeons captured	Number of pigeons infected	Percentage infected
1	Court House	21	1	4.7
2	City Hall	[1] 26	4	15.4
3	Oakford and Granada Aves	20	8	40.0
4	3803 Ferndale Aves	1	0	0
5	22 and 27 Benkert St	9	0	0
6	Recreation Pier, Broadway	6	0	0
7	921 North Charles St	1	0	0
8	Bush and Hamburg Sts	16	2	12.5
Total		100	15	15.0

[1] Mouse passages uncompleted for 1 pigeon.

At autopsy, the gross appearance of the viscera of each pigeon was noted. The spleen and part of the liver from each bird were removed and ground in a mortar. Sterile saline was added to make a 5- to 10-percent suspension. Routinely, this material from each pigeon was inoculated intraperitoneally into three white mice of stock breed in 0.3-cc. amounts.

After 1 week, two mice from each lot were sacrificed, and 0.03 cc. of an approximately 5-percent saline suspension of their pooled spleens was injected intracerebrally into the second lot of four mice. The brains of any recently dead or dying mice of this transfer were removed aseptically. Impression smears were made from the cut surface and stained by Machiavello's technique. The characteristic clusters of elementary or Levinthal-Coles-Lillie bodies were readily identified, when found. After 1 week of observation, two mice from lots in which there was doubtful or no evidence of infection were sacrificed, and suspensions of their pooled brains passed intracerebrally to a third lot. The transfers were not carried further unless an infection was suspected but not demonstrated.

First-passage mice inoculated intraperitoneally rarely appeared sick, and although many were kept for 4 or 5 weeks, only one died. On autopsy, 7 or more days after inoculation, animals subsequently

found to be infected occasionally showed some peritoneal fluid and a friable spleen covered with a thin layer of exudate. Mice inoculated intracerebrally with infected spleen or brain nearly always showed manifest signs of illness in 48 hours and were dead in 3 to 6 days.

In some of the early attempts at isolation, the first intracerebral passage was done 2 or 3 weeks after the original intraperitoneal inoculation. Of the 15 isolations, 12 were made with the second lot of mice on the first intracerebral passage, approximately 10 to 12 days after the pigeon was examined. Two isolations were made on the second intracerebral passage, 17 to 20 days later, and one isolation was obtained in the third intracerebral transfer.

The spleens of all pigeons subsequently shown to be carriers of the virus were enlarged, but no other obvious abnormality was observed in any of the birds examined. A number of birds from which no virus was recovered had enlarged spleens.

Four virus strains were inoculated into the yolk sac of developing chick embryos. All grew readily, and three of these, after being established by several yolk-sac passages, killed mice intraperitoneally and intracerebrally with 50-percent endpoints (*5*) as shown in table 2.

TABLE 2.—*50-percent endpoint titrations in mice following intraperitoneal and intracerebral injections of yolk sacs infected with 3 strains of ornithosis virus*

Virus from pigeon No.—	LD_{50} 0.3-cc. amounts injected intraperitoneally	LD_{50} 0.03-cc. amounts injected intracerebrally
43	$>1\times10^{-1}$	6.8×10^{-3}
57	6.8×10^{-2}	1×10^{-4}
65	$>1\times10^{-1}$	1×10^{-4}

It is apparent that the virus is 10^5 to 10^7 times as lethal when injected into the brain of a mouse as when injected into the peritoneal cavity. Mouse-brain virus of the first intracerebral transfer from pigeon No. 43 killed mice intraperitoneally in 0.3-cc. amounts at LD_{50} 6×10^{-2} and intracerebrally in 0.03-cc. amounts at LD_{50} 1.3×10^{-5}. One strain of virus was recovered from the spleen of a mouse inoculated intraperitoneally 50 days previously, indicating a carrier state for at least this time period.

Antigen prepared from these three strains by ether extraction of heavily infected yolk sacs, after the method used for rickettsial antigens (*6*), fixed complement in the presence of human psittacosis serum, pigeon ornithosis serum, and pooled human lymphogranuloma venereum serum, but not in the presence of normal human serum.

SUMMARY

Active ornithosis virus identified by morphological and serological studies was recovered from the organs of 15 of 100 adult wild pigeons captured in Baltimore, Md. Infected birds were found within the city at four of eight different capture locations. In spite of the demonstrated prevalence of infection among wild pigeons, the paucity of recognized human ornithosis suggests that wild pigeons do not constitute a serious public health hazard to the population of Baltimore under conditions now prevailing in that city.

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DEATHS DURING WEEK ENDED SEPT. 13, 1947

[From the Weekly Mortality Index, issued by the National Office of Vital Statistics]

	Week ended Sept. 13, 1947	Corresponding week, 1946
Data for 93 large cities of the United States:		
Total deaths	8,264	8,607
Median for 3 prior years	8,238	
Total deaths, first 37 weeks of year	342,796	337,587
Deaths under 1 year of age	735	690
Median for 3 prior years	614	
Deaths under 1 year of age, first 37 weeks of year	27,596	23,677
Data from industrial insurance companies:		
Policies in force	67,164,412	67,284,664
Number of death claims	11,963	10,605
Death claims per 1,000 policies in force, annual rate	9.3	8.2
Death claims per 1,000 policies, first 37 weeks of year, annual rate	9.4	9.7

INCIDENCE 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

REPORTS FROM STATES FOR WEEK ENDED SEPT. 20, 1947

Summary

For the first time since June 14, a decline occurred in the reported weekly incidence of poliomyelitis. A total of 881 cases was reported, as compared with 959 last week, 1,425 for the corresponding week last year, and a 5-year (1942–46) median of 864. Slight increases occurred in the Middle Atlantic, West North Central, South Atlantic, and Mountain areas. The 17 States reporting currently 12 or more cases are as follows (last week's figures in parentheses): *Increases*—New York 158 (129), New Jersey 28 (22), Michigan 77 (70), Wisconsin 27 (17), Minnesota 29 (12), Nebraska 16 (7), North Carolina 27 (15), California 32 (19); *decreases*—Massachusetts 28 (43), Rhode Island 12 (16), Connecticut 15 (19), Pennsylvania 40 (65), Ohio 146 (213), Indiana 17 (33), Illinois 89 (106); West Virginia and Tennessee reported the same numbers as for last week—17 and 12, respectively. The total reported since March 15 (the approximate average date of seasonal low incidence) is 5,885, as compared with 16,739 for the same period last year and an average of 11,715 for the corresponding periods of the past 4 years.

One case of smallpox was reported in Mississippi during the current week, and 3 cases of anthrax were reported in Arkansas. Of the total of 21 cases of infectious encephalitis North Dakota reported 10, and of 125 cases of undulant fever Michigan reported 13, Illinois 10, Iowa 9, and California 8.

Cumulative figures since the respective average low seasonal incidence dates are below the corresponding 5-year medians for diphtheria, influenza, meningococcus meningitis, poliomyelitis, scarlet fever, smallpox, typhoid and paratyphoid fever, and typhus fever, while the figures since August 30 for measles, and since the first of the year for the dysenteries (combined), Rocky Mountain spotted fever, tularemia, and whooping cough are above the corresponding medians. The reported incidence of undulant fever to date is above that for the corresponding periods of the past 2 years.

Deaths registered during the current week in 93 large cities of the United States totaled 8,269, as compared with 8,264 last week, 8,246 and 8,205, respectively, for the corresponding weeks of 1946 and 1945, and a 3-year (1944–46) median of 8,205. Deaths under 1 year of age totaled 743, as compared with 736 last week and a 3-year median of 607. Deaths, all causes, to date totaled 351,065, as compared with 345,833 for the same period last year.

Telegraphic morbidity reports from State health officers for the week ended Sept. 20, 1947, and comparison with corresponding week of 1946 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none was reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended—		Median 1942–46	Week ended—		Median 1942–46	Week ended—		Median 1942–46	Week ended—		Median 1942–46
	Sept. 20, 1947	Sept. 21, 1946		Sept. 20, 1947	Sept. 21, 1946		Sept. 20, 1947	Sept. 21, 1946		Sept. 20, 1947	Sept. 21, 1946	
NEW ENGLAND												
Maine	2	1	1					2	3	0	3	3
New Hampshire										0	0	0
Vermont	0	4					1	5	2	0	0	0
Massachusetts	3	9	4				14	44	35	0	5	3
Rhode Island	1	2					2	2	1	1	0	0
Connecticut	1	1	1			1	19	7	6	0	0	0
MIDDLE ATLANTIC												
New York	11	18	10		[1] 2	[1] 2	46	75	30	4	5	13
New Jersey	0	0	1	2	6	1	23	10	10	2	3	5
Pennsylvania	3	9	7		[2] 1	[2] 1	30	42	32	1	5	5
EAST NORTH CENTRAL												
Ohio	10	12	6	3	2	2	20	34	19	0	2	3
Indiana	4	5	9	12	14	4	3	4	4	3	4	2
Illinois	1	6	6		1	1	31	14	19	2	6	6
Michigan [3]	4	2	3		1	1	23	22	22	3	3	3
Wisconsin	1	6	2		4	12	32	23	23	2	1	1
WEST NORTH CENTRAL												
Minnesota	9	4	4				40	2	5	3	0	2
Iowa	1	0	4				7	5	5	1	2	1
Missouri	1	5	4	1	2	2		2	3	1	1	3
North Dakota	0	0	2		5	1	8	6	1	0	0	0
South Dakota	3	3	3				1			0	0	0
Nebraska	1	1	2		5	3	1		1	0	0	0
Kansas	2	5	4			1	6	1	3	0	1	1
SOUTH ATLANTIC												
Delaware	0	0	0				2			1	0	0
Maryland [3]	10	11	7		2	1	3	6	6	0	1	2
District of Columbia	0	0	0				1	4	1	0	0	1
Virginia	2	9	12	204	161	74	17	15	4	0	3	2
West Virginia	2	5	6	4		1	19			1	1	2
North Carolina	21	11	37				1	8	4	1	1	1
South Carolina	11	3	16	185	14	113	44		5	1	0	0
Georgia	11	15	26	2		0		9	5	0	0	0
Florida	2	7	7	4	1	1	2	2	2	1	3	1
EAST SOUTH CENTRAL												
Kentucky	9	27	11			1	2	43	4	2	3	2
Tennessee	6	10	13	17	2	6	8	3	3	2	2	2
Alabama	7	18	18	12	31	15	2	5	4	0	0	1
Mississippi [3]	7	6	7	8			2			0	0	1
WEST SOUTH CENTRAL												
Arkansas	5	4	6	7	12	12		2	2	1	1	1
Louisiana	7	6	9	1		1	1		4	2	1	1
Oklahoma	1	3	5	18	5	17	3	2	2	0	0	1
Texas	20	25	36	239	454	442	33	49	30	7	4	4
MOUNTAIN												
Montana	0	0	4			1	9	10	10	0	0	0
Idaho	1	0	0	10	2		1	7	5	0	0	0
Wyoming	0	2	0			2	2		2	0	0	0
Colorado	9	6	4			8	2	4	4	0	1	1
New Mexico	0	1	1		3	1	4	2	1	1	0	0
Arizona		1	1	19	25	25	5	3	3	0	0	1
Utah [3]	0						4	2	5	0	1	0
Nevada		0	0	0						0	0	0
PACIFIC												
Washington	3	6	6			1	20	8	14	0	1	1
Oregon	0	2	2	7		2	9	17	17	0	0	0
California	12	24	17	3	2	12	38	38	49	2	5	11
Total	204	295	326	767	757	757	541	539	539	45	69	83
38 weeks	7,941	11,123	8,926	305,806	194,853	84,920	187,151	641,654	541,518	2,686	4,735	6,578
Seasonal low week [4]	(27th) July 5–11			(30th) July 26–Aug. 1			(35th) Aug. 30–Sept. 5			(37th) Sept. 13–19		
Total since low	1,644	2,495	2,439	4,293	4,656	4,656	1,649	1,569	1,566	45	69	83

[1] New York City only. [2] Philadelphia only.
[3] Period ended earlier than Saturday.
[4] Dates between which the approximate low week ends. The specific date will vary from year to year.

Telegraphic morbidity reports from State health officers for the week ended Sept. 20, 1947, and comparison with corresponding week of 1946 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended—		Median 1942–46	Week ended—		Median 1942–46	Week ended—		Median 1942–46	Week ended—		Median 1942–46
	Sept. 20, 1947	Sept. 21, 1946		Sept. 20, 1947	Sept. 21, 1946		Sept. 20, 1947	Sept. 21, 1946		Sept. 20, 1947 [5]	Sept. 21, 1946	
NEW ENGLAND												
Maine	2	0	2	5	23	12	0	0	0	1	0	0
New Hampshire	2	9	2	2	2	2	0	0	0	0	0	0
Vermont	2	2	4	0	1	1	0	0	0	0	0	0
Massachusetts	28	16	29	33	28	68	0	0	0	1	9	9
Rhode Island	12	14	1	1	2	4	0	0	0	0	0	0
Connecticut	15	1	11	0	8	8	0	0	0	0	0	1
MIDDLE ATLANTIC												
New York	158	90	90	42	75	82	0	0	0	6	7	7
New Jersey	28	9	17	14	15	19	0	0	0	1	3	2
Pennsylvania	40	14	14	33	48	72	0	0	0	5	5	7
EAST NORTH CENTRAL												
Ohio	146	46	37	37	60	79	0	0	0	2	3	8
Indiana	17	22	11	11	25	28	0	4	1	2	1	5
Illinois	89	210	93	22	34	61	0	0	0	3	3	5
Michigan [3]	77	69	28	27	43	44	0	0	0	6	2	3
Wisconsin	27	94	26	15	28	49	0	0	0	2	1	1
WEST NORTH CENTRAL												
Minnesota	29	128	23	10	13	26	0	0	0	1	0	1
Iowa	5	31	14	3	9	21	0	0	0	0	0	1
Missouri	9	72	10	4	3	22	0	0	0	3	1	4
North Dakota	2	30	2	5	1	4	0	0	0	0	1	0
South Dakota	0	9	1	1	1	1	0	0	0	0	1	0
Nebraska	16	33	10	16	22	10	0	0	0	0	11	0
Kansas	5	68	11	5	18	25	0	0	0	1	2	1
SOUTH ATLANTIC												
Delaware	6	4	2	1	2	1	0	0	0	0	0	0
Maryland [3]	9	2	3	3	5	14	0	0	0	0	1	3
District of Columbia	3	2	2	7	2	6	0	0	0	0	0	1
Virginia	10	6	6	6	32	32	0	0	0	5	4	7
West Virginia	17	2	3	17	27	42	0	0	0	4	1	2
North Carolina	27	2	3	8	17	48	0	0	0	3	2	2
South Carolina	4	0	2	1	0	6	0	0	0	4	0	4
Georgia	5	3	3	10	9	19	0	0	0	5	5	5
Florida	1	15	2	3	2	4	0	0	0	0	4	1
EAST SOUTH CENTRAL												
Kentucky	9	12	5	7	32	28	0	0	0	8	0	7
Tennessee	12	5	6	10	13	35	0	0	0	4	4	9
Alabama	2	18	3	3	9	18	0	0	0	2	0	3
Mississippi [3]	2	3	3	2	12	10	1	0	0	1	7	5
WEST SOUTH CENTRAL												
Arkansas	[6] 2	13	4	4	5	7	0	0	0	2	1	3
Louisiana	0	20	5	1	7	5	0	0	0	0	10	9
Oklahoma	1	15	15	5	8	8	0	0	0	1	1	5
Texas	4	33	33	18	22	22	0	1	0	9	10	16
MOUNTAIN												
Montana	1	13	7	5	2	5	0	0	0	1	0	0
Idaho	9	7	2	14	3	5	0	0	0	0	2	0
Wyoming	1	9	1	0	0	4	0	0	0	0	0	0
Colorado	4	49	11	2	5	7	0	1	0	3	0	5
New Mexico	1	14	2	7	4	3	0	0	0	1	2	4
Arizona	0	5	4	6	5	4	0	0	0	1	3	0
Utah [3]	0	24	22	3	4	4	0	0	0	0	0	0
Nevada	1	0	0	0	0	0	0	0	0	0	0	0
PACIFIC												
Washington	8	44	20	15	12	19	0	0	0	0	0	2
Oregon	1	14	12	4	8	14	0	0	0	1	2	1
California	32	124	54	34	59	79	0	0	0	15	5	4
Total	881	1,425	864	482	765	1,128	1	6	5	104	114	167
38 weeks	[6] 6,497	17,206	8,882	64,567	89,992	102,603	149	292	317	2,849	3,114	4,072
Seasonal low week [4]	(11th) Mar. 15–21			(32nd) Aug. 9–15			(35th) Aug. 30–Sept. 5			(11th) Mar. 15–21		
Total since low	[6] 5,885	16,739	8,485	2,464	3,697	4,799	2	13	10	2,364	2,639	3,256

[3] Period ended earlier than Saturday.
[4] Dates between which the approximate low week ends. The specific date will vary from year to year.
[5] Including paratyphoid fever reported separately as follows: Massachusetts 1 (salmonella infection); New York 1; Pennsylvania 1; Indiana 1; Minnesota 1; Virginia 2; Georgia 1; Texas 1; California 9.
[6] Correction: Arkansas, poliomyelitis week ended September 6, 6 cases (instead of 7). Deducted from cumulative totals.

Telegraphic morbidity reports from State health officers for the week ended Sept. 20, 1947, and comparison with corresponding week of 1946 and 5-year median—Con.

Division and State	Whooping cough			Week ended September 20, 1947							
	Week ended—		Median 1942–46	Dysentery			Encephalitis, infectious	Rocky Mt. spotted fever	Tularemia	Typhus fever, endemic	Undulant fever
	Sept. 20, 1947	Sept. 21, 1946		Amebic	Bacillary	Unspecified					
NEW ENGLAND											
Maine	12		14								1
New Hampshire											
Vermont	14	7	17								6
Massachusetts	162	121	121		5						
Rhode Island	20	18	18								
Connecticut	98	39	34	1							3
MIDDLE ATLANTIC											
New York	233	146	266	6	1			1			3
New Jersey	225	164	151	1				1			
Pennsylvania	254	113	133								5
EAST NORTH CENTRAL											
Ohio	366	73	152			1		1	1		3
Indiana	30	22	20			1	1		1		
Illinois	172	145	145	7	9		3		1		10
Michigan [3]	271	189	189		1						13
Wisconsin	166	245	199						1		5
WEST NORTH CENTRAL											
Minnesota	108	8	36				1				5
Iowa	31	11	15								9
Missouri	20	14	14								2
North Dakota	6		6				10				
South Dakota	6	2	2								2
Nebraska	2	4	3				3				2
Kansas	29	20	20						1		5
SOUTH ATLANTIC											
Delaware	4	3	2								1
Maryland [3]	78	40	53					[7] 3			2
District of Columbia	19	7	7								
Virginia	89	25	25			78		2	1		2
West Virginia	7	18	6								5
North Carolina	50	36	50					3	4	3	2
South Carolina	85	3	37	2	10		1			2	3
Georgia	56	10	15	1	6	1		1	1	8	6
Florida	25	10	10							3	
EAST SOUTH CENTRAL											
Kentucky	35	35	40		2						
Tennessee	25	12	20			1		3	1		5
Alabama	50	5	14					3		3	5
Mississippi [3]	4			1					1	1	2
WEST SOUTH CENTRAL											
Arkansas	19	5	10	8					[7] 1		
Louisiana	6	8	8						1	5	1
Oklahoma	19	6	7	3	1			1	1		2
Texas	336	159	127	17	265	23				7	2
MOUNTAIN											
Montana	16	4	23								
Idaho	4	8	8				1				
Wyoming	4	6	6								
Colorado	54	13	32								
New Mexico	18	8	8		1						
Arizona	26	6	15	1		13					3
Utah [3]	16	3	20						5		2
Nevada		1									
PACIFIC											
Washington	47	21	17	2							
Oregon	9	7	8	1							
California	117	62	122	3	6		2				8
Total	3,443	1,862	2,217	54	307	118	22	19	21	32	125
Same week: 1946	1,862			37	199	138	17	3	13	110	116
Median, 1942–46	2,217			30	561	213	18	6	11 [4]	150	[8] 102
38 weeks: 1947	119,156			2,188	11,874	7,508	428 [4]	[7] 495	[7] 1,128	1,539	4,518
1946	74,147			1,748	12,714	5,070	480	515	697	2,588	3,754
Median, 1942–46	95,586			1,383	12,714	6,047	480	427	655	2,946 [8]	3,638

[3] Period ended earlier than Saturday.

[7] Delayed reports: Maryland, Rocky Mountain spotted fever, 3 cases; Arkansas, tularemia, 5 cases. These cases included in cumulative totals only.

[8] 2-year average, 1945–46.

Anthrax: California, 3 cases.

Territory of Hawaii, week ended September 20, 1947: Bacillary dysentery 8; measles 1; endemic typhus fever 1; whooping cough 28.

WEEKLY REPORTS FROM CITIES [1]

City reports for week ended Sept. 13, 1947

This table lists the reports from 87 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

Division, State, and City	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
NEW ENGLAND												
Maine:												
Portland	0	0	------	0	------	0	1	1	1	0	0	3
New Hampshire:												
Concord	0	0	------	0	------	0	1	0	0	0	0	------
Vermont:												
Barre	0	0	------	0	------	0	1	0	0	0	0	------
Massachusetts:												
Boston	3	0	------	0	11	0	9	32	7	0	0	45
Fall River	0	0	------	0	1	0	1	1	0	0	0	11
Springfield	0	0	------	0	1	0	0	1	0	0	0	8
Worcester	0	0	------	0	------	0	3	5	3	0	0	8
Rhode Island:												
Providence	1	0	------	0	------	1	2	5	2	0	1	19
Connecticut:												
Bridgeport	0	0	------	0	1	0	0	1	0	0	0	------
Hartford	0	0	------	0	1	0	0	3	0	0	0	11
New Haven	0	0	1	0	------	0	0	2	1	0	0	53
MIDDLE ATLANTIC												
New York:												
Buffalo	0	0	------	1	------	0	5	7	1	0	0	3
New York	7	1	------	0	15	3	59	36	14	0	4	93
Rochester	0	0	------	0	------	0	3	20	2	0	0	6
Syracuse	0	1	------	0	------	1	0	6	2	0	0	33
New Jersey:												
Camden	0	0	------	0	------	0	1	2	0	0	0	------
Newark	0	0	1	0	1	0	0	4	2	0	0	46
Trenton	0	0	------	0	------	0	0	0	1	0	0	9
Pennsylvania:												
Philadelphia	1	0	------	0	3	0	8	7	4	0	0	109
Pittsburgh	0	0	------	0	1	1	7	2	1	0	0	21
Reading	0	0	------	0	2	0	2	0	1	0	0	------
EAST NORTH CENTRAL												
Ohio:												
Cincinnati	0	0	------	0	------	0	5	25	3	0	0	3
Cleveland	0	0	1	0	3	0	4	38	7	0	1	121
Columbus	3	0	1	1	3	0	1	11	3	0	0	23
Indiana:												
Fort Wayne	0	0	------	0	------	0	0	1	0	0	1	7
Indianapolis	2	0	------	0	------	0	4	2	1	0	1	22
South Bend	0	0	------	0	------	0	0	0	0	0	0	------
Terre Haute	0	0	------	0	------	0	2	0	0	0	0	------
Illinois:												
Chicago	1	0	------	0	19	0	14	64	7	0	1	63
Michigan:												
Detroit	1	0	------	1	5	0	10	31	9	0	0	120
Flint	0	0	------	0	1	1	2	3	1	0	0	------
Grand Rapids	0	0	------	0	10	0	0	0	0	0	0	32
Wisconsin:												
Kenosha	0	0	------	0	------	0	0	0	0	0	0	9
Milwaukee	0	0	------	0	6	0	1	1	3	0	0	37
Racine	0	0	------	0	2	0	1	0	2	0	0	10
Superior	0	0	------	0	------	0	0	0	0	0	0	13
WEST NORTH CENTRAL												
Minnesota:												
Duluth	0	0	------	0	3	0	0	0	1	0	0	24
Minneapolis	4	0	------	0	8	0	4	2	3	0	0	27
St. Paul	0	0	------	0	------	0	5	2	0	0	0	------
Missouri:												
Kansas City	0	0	------	1	------	0	3	7	3	0	0	8
St. Joseph	0	1	------	0	------	0	0	1	0	0	0	------
St. Louis	0	0	------	0	3	0	5	3	1	0	0	21

[1] In some instances the figures include nonresident cases.

City reports for week ended Sept. 13, 1947—Continued

Division, State, and City	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
WEST NORTH CENTRAL—continued												
Nebraska:												
Omaha	0	1		0		0	0	3	1	0	0	1
Kansas:												
Topeka	0	0		0	3	0	0	0	1	0	0	13
Wichita	0	0		0	4	0	2	0	0	0	0	7
SOUTH ATLANTIC												
Delaware:												
Wilmington	0	0		0		0	1	7	1	0	0	
Maryland:												
Baltimore	3	0		0	1	0	3	1	2	0	0	81
Cumberland	1	0		0		0	0	1	0	0	0	2
Frederick	0	0		0		0	0	0	0	0	0	
District of Columbia:												
Washington	0	0		0		1	3	3	4	0	0	16
Virginia:												
Richmond	0	0		0		0	2	1	0	0	0	1
Roanoke	0	0		0		0	0	1	0	0	0	
West Virginia:												
Charleston	0	0		0		0	0	0	0	0	0	
Wheeling	0	0		0		0	0	0	0	0	0	
North Carolina:												
Raleigh	0	0		0		0	0	0	0	0	0	6
Wilmington	0	0		0		0	0	0	0	0	0	
Winston-Salem	0	0		0		0	3	1	0	0	0	5
South Carolina:												
Charleston	0	1		0	1	1	1	0	0	0	1	6
Georgia:												
Atlanta	0	0		0		0	0	1	0	0	3	1
Brunswick	0	0		0		0	0	0	0	0	0	
Savannah	0	0		0	1	0	1	0	1	0	0	9
Florida:												
Tampa	0	0		0		1	0	1	2	0	1	2
EAST SOUTH CENTRAL												
Tennessee:												
Memphis	0	0		0	1	1	6	2	1	0	1	5
Nashville	1	0		0		0	0	0	2	0	0	5
Alabama:												
Birmingham	1	0		0		0	0	0	0	0	0	
Mobile	0	0		1		0	1	0	0	0	0	
WEST SOUTH CENTRAL												
Arkansas:												
Little Rock	0	0		0		0	0	0	0	0	0	
Louisiana:												
New Orleans	0	1	3	0		0	6	1	2	0	0	3
Shreveport	0	0		0		0	1	0	0	0	0	
Oklahoma:												
Oklahoma City	0	0		0		0	1	0	1	0	0	4
Texas:												
Dallas	1	0	1	1		0	2	1	3	0	1	2
Galveston	0	0		0		0	0	0	0	0	0	
Houston	0	0		0	1	0	4	2	0	0	0	
San Antonio	1	0		0		0	0	1	1	0	1	
MOUNTAIN												
Montana:												
Billings	0	0		0		0	0	0	0	0	0	1
Great Falls	0	0		0		0	0	0	0	0	0	3
Helena	0	0		0		0	0	0	0	0	0	3
Missoula	0	0		0		0	0	0	0	0	0	
Idaho:												
Boise	0	0		0		0	0	9	0	0	0	
Colorado:												
Denver	4	0		0		0	1	3	4	0	0	23
Pueblo	0	0		0		0	1	0	0	0	0	14
Utah:												
Salt Lake City	0	0		0		0	1	0	2	0	0	3

City reports for week ended Sept. 13, 1947—Continued

Division, State, and City	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles cases	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
PACIFIC												
Washington:												
Seattle	0	0	------	0	------	0	3	3	1	0	0	4
Spokane	0	0	------	0	2	0	0	1	0	0	0	2
Tacoma	0	0	------	0	------	0	0	0	1	0	0	6
California:												
Los Angeles	1	0	------	0	4	0	2	7	4	0	1	13
San Francisco	0	0	------	0	9	1	1	2	1	0	0	5
Total	36	6	8	6	127	12	211	377	121	0	18	2,164
Corresponding week, 1946*	78	------	21	4	120	------	199	------	204	0	11	757
Average 1942-46*	56	------	29	[2] 6	[3] 136	------	[2] 213	------	252	0	26	768

*Exclusive of Oklahoma City.
[2] 3-year average, 1944-46.
[3] 5-year median, 1942-46.

Dysentery, amebic.—Cases: New Haven 1; New York 7; St. Louis 1; Charleston, S. C., 2; Houston 1.
Dysentery, bacillary.—Cases: Providence 1; New York 1; Charleston, S. C., 2; New Orleans 1.
Dysentery, unspecified.—Cases: San Antonio 3.
Rocky Mountain spotted fever.—Cases: New York 1; Columbus 1.
Tularemia.—Cases: Cleveland 1; Nashville 1.
Typhus fever, endemic.—Cases: Tampa 1; Memphis 1; New Orleans 5; Dallas 1; Galveston 1.

Rates (annual basis) per 100,000 population, by geographic groups, for the 87 cities in the preceding table (latest available estimated population, 34,373,100)

	Diphtheria case rates	Encephalitis, infectious, case rates	Influenza		Measles case rates	Meningitis, meningococcus, case rates	Pneumonia death rates	Poliomyelitis case rates	Scarlet fever case rates	Smallpox case rates	Typhoid and paratyphoid fever case rates	Whooping cough case rates
			Case rates	Death rates								
New England	10.5	0.0	2.6	0.0	39	2.6	47.1	133.3	37.	0.0	2.6	413
Middle Atlantic	3.7	0.9	0.5	0.5	10	2.3	39.3	38.9	13	0.0	1.9	148
East North Central	4.3	0.0	1.2	1.2	30	0.6	27.0	107.9	22	0.0	2.5	282
West North Central	8.0	4.0	0.0	2.0	42	0.0	38.2	36.2	20	0.0	0.0	203
South Atlantic	6.6	1.7	0.0	0.0	5	5.0	23.2	28.1	17	0.0	8.3	213
East South Central	11.8	0.0	0.0	5.9	6	5.9	41.3	11.8	18	0.0	5.9	59
West South Central	5.1	2.5	10.2	2.5	3	0.0	38.1	10.2	18	0.0	5.1	23
Mountain	31.8	0.0	0.0	0.0	0	0.0	23.8	95.3	48	0.0	0.0	373
Pacific	1.6	0.0	0.0	0.0	25	1.6	9.8	21.3	11	0.0	1.6	49
Total	5.5	0.9	1.2	0.9	19	1.8	32.1	57.3	18	0.0	2.7	192

PLAGUE INFECTION IN CLEAR CREEK, CUSTER, LA PLATA AND PARK COUNTIES, COLORADO, AND KITTITAS COUNTY, WASH.

Plague infection has been reported proved in ectoparasites and tissue from rodents collected in Colorado and Washington, as follows:

COLORADO

Clear Creek County.—Proved positive on August 15, a pool of 95 fleas from 25 ground squirrels, *Citellus richardsonii elegans*, taken on August 7, 15 miles east of Georgetown via Highways Nos. 6 and 40.

Custer County.—Proved positive on July 21, lungs and nodes from 1 prairie dog, *Cynomys* sp., taken at the Custer County Airfield, east edge of Silvercliff.

La Plata County.—Proved positive on July 18, a pool of 74 fleas from 2 marmots, *Marmota* sp., taken 15–20 miles north of Durango on U. S. Highway No. 550; and proved positive on July 19, a pool of 46 fleas from 4 ground squirrels, *Citellus variegatus*, taken in same locality.

Park County.—Proved positive on August 5, a pool of 8 fleas and 1 tick from 19 ground squirrels, *Citellus lateralis*, taken 5 miles west of Como; proved positive on August 11, a pool of 150 fleas from 60 ground squirrels, *C. elegans*, taken 5 miles north and 5 miles west of Fairplay; and proved positive on August 12, a pool of 130 fleas from 16 prairie dogs, *Cynomys* sp., taken July 30 at a locality 10 miles east and 5 miles south of Jefferson.

WASHINGTON

Kittitas County.—Proved positive on August 25, a pool of 230 fleas from 60 field mice, *Peromyscus* sp., taken August 14 at a locality 6 miles southeast of Kittitas.

TERRITORIES AND POSSESSIONS

Puerto Rico

Notifiable diseases—4 weeks ended August 30, 1947.—During the 4 weeks ended August 30, 1947, cases of certain notifiable diseases were reported in Puerto Rico as follows:

Disease	Cases	Disease	Cases
Chickenpox	6	Syphilis	164
Diphtheria	33	Tetanus	9
Dysentery, unspecified	2	Tuberculosis (all forms)	805
Gonorrhea	182	Typhoid fever	8
Influenza	73	Typhus fever (murine)	5
Malaria	264	Whooping cough	130
Measles	1		

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended August 30, 1947.—During the week ended August 30, 1947, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox		1		6	29	3	15	5	22	81
Diphtheria		1		9	3	1	2	3	1	20
Dysentery:										
Amebic					1	2				3
Bacillary				9		1				10
Unspecified					1					1
Encephalitis, infectious						16	15			31
German measles					13			2	12	27
Influenza		41			2	18				61
Measles			1	48	39	4	13	7	14	126
Meningitis										
Meningococcus		1		1	1		1			4
Mumps		1		7	54	6	4	6	24	102
Poliomyelitis		2	1	7	75	105	28	7	25	250
Scarlet fever		2	3	19	18	1	1	1	2	47
Tuberculosis (all forms)		23	4	127	16	94	12	6		282
Typhoid and paratyphoid fever				16				1	2	19
Undulant fever				4					1	5
Venereal diseases:										
Gonorrhea	1	14	12	84	87	49	26	66	(1)	339
Syphilis		17	10	94	37	10	11	11	(1)	190
Other forms		2							(1)	2
Whooping cough				31	64	49	3	18	27	192

[1] Report for the current period for British Columbia not received.

FINLAND

Notifiable diseases—July 1947.—During the month of July 1947, cases of certain notifiable diseases were reported in Finland as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	13	Paratyphoid fever	350
Diphtheria	380	Poliomyelitis	30
Dysentery, unspecified	4	Scarlet fever	142
Gonorrhea	1,449	Syphilis	307
Malaria	9	Typhoid fever	90

GREAT BRITAIN

England and Wales—Poliomyelitis.—During the week ended September 6, 1947, 708 cases of poliomyelitis were reported in England and Wales, bringing the total to date to 4,984 cases.

JAPAN

Notifiable diseases—4 weeks ended August 30, 1947, and accumulated totals for the year to date.—For the 4 weeks ended August 30, 1947, and for the year to date, certain notifiable diseases were reported in Japan as follows:

Disease	4 weeks ended Aug. 30, 1947		Total reported for the year to date	
	Cases	Deaths	Cases	Deaths
Diphtheria	1,055	71	20,148	1,750
Dysentery, unspecified	13,915	2,312	27,250	4,781
Encephalitis, Japanese "B"	[1] 66	[1] 25	[1] 73	[1] 29
Gonorrhea	17,692	----	140,816	----
Influenza	102	----	[2] 2,378	----
Malaria	1,789	2	9,045	17
Measles	10,499	----	[2] 454,308	----
Meningitis, epidemic	183	93	2,850	916
Paratyphoid fever	823	44	3,151	168
Pneumonia	5,921	----	[2] 91,088	----
Scarlet fever	160	6	1,857	44
Smallpox	1	0	376	38
Syphilis	11,297	----	95,155	----
Tuberculosis	34,686	----	179,722	----
Typhoid fever	3,137.	359	11,675	1,375
Typhus fever	18	1	994	81
Whooping cough	16,596	----	[2] 107,639	----

[1] Includes suspected cases.
[2] For the period Mar. 30 to Aug. 30, 1947.

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—Except in cases of unusual incidence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during recent months. All reports of yellow fever are published currently.

A table showing the accumulated figures for these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

Cholera

China—Shanghai.—For the week ended September 13, 1947, 8 cases of cholera were reported in Shanghai, China.

Indochina (French)—Tonkin State.—For the period August 21–31, 1947, 60 cases of cholera with 4 deaths were reported in Tonkin State, French Indochina.

Siam (Thailand).—For the week ended August 9, 1947, 71 cases of cholera with 41 deaths were reported in Siam (Thailand).

Plague

Siam (Thailand).—For the week ended August 9, 1947, 2 cases of plague were reported in Siam (Thailand).

Typhus Fever

Colombia.—For the month of August 1947, 287 cases of typhus fever with 5 deaths were reported in Colombia.

Yellow Fever

Colombia.—Yellow fever has been reported in Colombia as follows: Municipality of Muzo, Boyaca Department, July 20 to August 7, 1947, 2 cases, 2 deaths; Municipality of Cucuta, North Santander Department, July 20 to August 7, 1947, 1 case, 1 death.

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