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PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

June 19–July 16, 1938

The accompanying table summarizes the prevalence of eight important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the PUBLIC HEALTH REPORTS under the section "Prevalence of Disease." The table gives the number of cases of these diseases for the 4-week period ending July 16, the number reported for the corresponding period in 1937, and the median number for the years 1933–37.

DISEASES ABOVE MEDIAN PREVALENCE

Measles.—The number of reported cases of measles dropped more than 60 percent during the current 4-week period as compared with the preceding 4 weeks. In comparison with the corresponding period in preceding years the incidence was high; the number of cases (32,402) was about one and one-half times that for 1937 and about 35 percent in excess of the 1933–37 median. The incidence in the East South Central and Pacific regions was about normal; in the New England and West South Central regions it was comparatively low, but other regions reported an unusually high incidence.

Smallpox.—The number of cases of smallpox reported for the 4 weeks ending July 16 was 648, as compared with 479, 534, and 532 for the corresponding period in 1937, 1936, and 1935, respectively. The current incidence was about 35 percent in excess of that for this period in 1937, and also of the 1933–37 median, which is represented by the 1937 figure. The North Atlantic regions remained apparently free from the disease and the South Atlantic and Mountain regions reported about the normal seasonal incidence, but all other sections reported very appreciable increases over the seasonal expectancy.

Influenza.—The influenza incidence (1,384 cases) during the current period was slightly higher than during the corresponding period in 1937 and about 20 percent above the expected seasonal incidence. The increase seemed to be due mostly to a comparatively high incidence in the South Atlantic, West South Central, and Mountain regions; in all other regions the influenza situation was very favorable.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period June 19–July 16, the number for the corresponding period in 1937, and the median number of cases reported for the corresponding period 1933–37[1]

Division	Current period	1937	5-year median	Current period	1937	5-year median	Current period	1937	5-year median	Current period	1937	5-year median
	Diphtheria			Influenza [2]			Measles [3]			Meningococcus meningitis		
United States [1]	1,145	1,249	1,528	1,384	1,269	1,170	32,402	22,810	24,029	150	296	296
New England	17	59	62	12	5	5	2,013	1,661	2,772	2	14	7
Middle Atlantic	208	241	294	23	19	19	10,786	8,422	8,422	24	81	72
East North Central	235	317	317	108	124	177	10,566	7,655	7,655	25	33	51
West North Central	85	83	135	47	322	118	1,800	341	1,166	11	19	16
South Atlantic	210	160	180	339	220	237	3,073	1,688	1,694	35	53	53
East South Central	78	82	90	91	60	109	547	967	559	38	43	26
West South Central	141	155	178	598	399	246	546	859	859	10	29	18
Mountain	77	41	42	71	52	31	1,129	559	559	4	3	9
Pacific	94	111	118	95	68	133	1,942	658	2,076	1	21	21
	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
United States [1]	157	771	653	6,366	8,017	8,017	648	479	479	1,706	1,770	1,911
New England	9	15	15	758	652	652	0	0	0	20	14	24
Middle Atlantic	18	27	39	1,708	2,381	2,381	0	0	0	95	132	151
East North Central	20	56	26	1,962	2,932	3,037	137	98	86	137	139	192
West North Central	13	46	11	510	751	751	228	188	188	57	114	114
South Atlantic	27	58	25	341	288	330	4	1	7	486	479	516
East South Central	41	160	34	127	105	135	17	5	4	334	345	352
West South Central	13	356	16	262	241	163	52	16	22	438	446	446
Mountain	4	9	4	225	217	217	81	101	72	84	48	61
Pacific	12	44	44	473	450	584	129	70	70	55	53	56

[1] 48 States. Nevada is excluded and the District of Columbia is counted as a State in these reports.
[2] 44 States and New York City.
[3] 46 States. Mississippi and Georgia are not included.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—The favorable record of diphtheria continued during the current 4-week period. The cases numbered 1,145, which marks a new low for this season of the year. In the South Atlantic and Mountain regions the incidence was somewhat above the normal seasonal level, but in all other regions the incidence was relatively low.

Meningococcus meningitis.—The meningitis situation was very favorable in all sections of the country. For the 4 weeks ending July 16 there were 150 cases reported, as compared with 296, 362, and 392 for the corresponding period in the years 1937, 1936, and 1935,

respectively. The current incidence was only about 10 percent above the average for the years 1932–34, years in which the incidence of meningitis was unusually low.

Poliomyelitis.—The number of cases (157) of poliomyelitis was the lowest reported since 1929, when the cases for this period totaled 132. A rather sharp increase in this disease is normally expected at this season of the year, and at times this period has marked the beginning of very serious outbreaks, but for the country as a whole, as well as for several of the geographic regions, the current incidence is considerably below the seasonal expectancy. Of the total number of cases, 20 occurred in Alabama, 16 in New York, 13 in Mississippi, 11 in California, 9 in Illinois, and 8 each in Virginia and Louisiana, two thirds of the cases being reported from those 7 States; no more than 5 cases were reported from any other State.

Scarlet fever.—The number of reported cases of scarlet fever dropped from approximately 12,600 for the preceding 4-week period to 6,366 for the 4 weeks ending July 16. The incidence is the lowest recorded for this period in 8 years. Definite decreases from the 1933–37 median were reported from the Middle Atlantic and East North Central regions, where the disease has been unusually prevalent, and minor increases were reported from the New England and West South Central regions; in all other regions the incidence was about normal for this season of the year.

Typhoid fever.—The number of cases (1,706) of typhoid fever was slightly below that for the corresponding period in 1937 and about 10 percent below the 1933–37 median. The 84 cases reported from the Mountain region represent the highest incidence recorded for this section in recent years. In all other regions the incidence was relatively low. The West North Central region reported the lowest number of cases (57) for this period in the 10 years for which these data are available.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ending July 16, based on data received from the Bureau of the Census, was 10.3 per 1,000 inhabitants (annual basis). The rate for the corresponding period in each of the 2 preceding years was 11.0. The current rate is the lowest since 1933, when a rate of 9.9 was recorded for this period.

A CASE OF HUMAN INFECTION WITH B. PSEUDOTUBERCULOSIS RODENTIUM

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The organism *B. pseudotuberculosis rodentium* (Pfeiffer) has received considerable attention in the past, undoubtedly because of its very close resemblance to *Pasteurella pestis*. Both of these organisms produce similar pathology in laboratory animals and resemble each other both culturally and antigenically. Meyer and Batchelder (*1*), in 1926, pointed to the danger of confusing lesions of pseudotuberculosis in wild rats with those of plague. Reimann and Rose (*2*) suggested the possible relationship pathologically between tularaemia and the three cases of pseudotuberculosis rodentium infection in human beings recorded in the literature. Poppe (*3*) even suggested in 1927 that tularaemia might be a form of human pseudotuberculosis.

Malassez and Vignal (*4*), in 1883, reported the first work with the organism. They inoculated guinea pigs with a nodule from the arm of a child dead with tuberculous meningitis and described a modified form of tuberculosis in the pigs. This is the first report of the disease in guinea pigs. As to whether it was produced by transfer of the human material or occurred spontaneously in the guinea pigs is open to question. A similar condition was described in guinea pigs by Dor (*5*), in 1888, Charrin and Roger (*6*), in 1888, Nocard (*7*), in 1889, and by Zagari (*8*), in 1890. Priesz (*9*), in 1894, compared the organism described by Pfeiffer (*10*), in 1890, and that of Malassez and Vignal (*4*) and decided they were identical. He suggested the name *pseudotuberculosis rodentium*, though he called it a streptobacillus.

There are many reports in the European literature concerning pseudotuberculosis rodentium, but the isolation of the organism is rare in the United States. Meyer and Batchelder (*1*) reported cases in guinea pigs and also isolation of the organism from a wild rat. Bishop (*11*), in 1932, reported the study of an outbreak of pseudotuberculosis in guinea pigs.

According to the literature, a great variety of animals are susceptible to pseudotuberculosis. It has been described in the horse (Schlaffke (*12*), 1921), cow (Mazzini, 1897), pig (Scennikov, 1928), goat (Baumann, 1927), rabbit (Roemisch, 1921), hare (Lerche, 1927), cat (Leblois, 1920), guinea pig (Ramon, 1914), wild rat (Meyer and Batchelder (*1*), 1926), hen (Christensen, 1927), and the monkey (Christianson, 1918).

When the literature concerning the disease in man is reviewed, it is seen at once that many of the reported cases must be doubted

as having been caused by pseudotuberculosis rodentium, while still others do not give sufficient evidence upon which an opinion may be based. Schütze (*13*), in 1929, reviewed the literature and arrived at the opinion that there were four indubitable cases. One of these, however, is very inconclusive—that of Albrecht (*14*), in 1910, as he made the diagnosis by injecting pieces of resected gut into two guinea pigs and only one contracted the disease. It is possible that this one may have had pseudotuberculosis before being injected with the human material. The cases of (a) Lorey (*15*), in 1911, who isolated the organism from the blood during the life of a man whose symptoms suggested typhoid, and from his liver and spleen after death, (b) Saisawa (*16*), in 1913, who also succeeded in removing the organism from the blood during life in a man with high temperature and intestinal symptoms, and (c) Roman (*17*), in 1916, who isolated it from the liver and spleen of a case of pyrexia accompanied by abdominal pain, all seem to be quite authentic.

The most recent and most complete case encountered in the literature is one reported by Neugebauer (*18*), in 1933. It was a case of a 45-year old male with a mild bronchitis who developed some abdominal tenderness and a fever of 39° C. The patient died after about 20 days of an unexplained illness. At autopsy a purulent bronchitis was found, but the main pathology was found to be in the liver. Here were seen many necrotic foci, greyish white in color, and varying in sizes from ones of a millimeter or so in diameter up to several centimeters. The centers were not purulent. The liver specimens and histopathology were compared with those of the case reported by Roman, and were found to be similar. Typical *B. pseudotuberculosis rodentium* was isolated from the liver and spleen. Its identity was established both by its cultural characteristics and by the production of animal pathology.

The case which we desire to report will be the only case which we have found to be originally recorded in the English language. This is deemed to be worth while because of the characteristic lesions produced in human beings and in laboratory animals and because the disease may closely simulate the clinical picture of several well-known entities.

CASE HISTORY

H. L., male, age 29, single, an electrician, employed in servicing and repair of refrigerators, had never had any illness of any consequence. He denied venereal disease. There was no history of dysentery, although he had made several trips to South America on board ship some years ago. He lived with a brother, and ate most of his meals at home. There is no history of contact with any animals, except that in his work of repairing large refrigerators he may have had to handle some meat and possibly he handled some butchered rabbits. The family history is irrelevant.

On November 22, 1937, he was unusually talkative, but no other symptoms were noted. The following morning he had to quit work because of generalized aches and pains and a fever. He was treated at home for several days under a diagnosis of influenza; but, because he was unable to eat, and because of vomiting incident to the high fever, he was taken to Providence Hospital, Seattle, on November 29, 1937. Up to that time the physical findings had remained negative for any localization of infection. General supportive measures were carried out, and glucose was given intravenously. When the patient was seen in consultation on December 2, 1937, general physical examination was negative except for slight rigidity of the neck. He was rather stuporous and could not cooperate. A lumbar puncture was made with entirely negative findings.

The next day the patient had very slight icterus and there was slight but definite tenderness over the liver on heavy percussion and on pressure. An X-ray film of the chest was made, showing a high diaphragm on the right but no definite pulmonary pathology. There was slight cyanosis.

On December 5 the icterus was more definite and the liver tenderness was more pronounced. A diagnosis of liver abscess was made and exploratory laparotomy was performed by Dr. Raymond Zech. At operation the liver was found to be enlarged and was studded with multiple small, yellowish white, nonsuppurative areas 2 to 3 mm in diameter. A small portion was removed for microscopic examination, and nothing else was done.

December 6 the neck was definitely rigid. Icterus was increasing. The right diaphragm was up to the lower angle of the scapula. There was no definite consolidation in the lungs. Very few râles were heard.

On December 8, another lumbar puncture was made with entirely negative findings.

The patient rapidly grew weaker and died of toxemia and exhaustion on December 9, 1937, at 9:10 p. m. An autopsy was made a short time after death.

A summary of laboratory findings is as follows:

Urinalysis

	Nov. 29	Dec. 1 (6 a. m.)	Dec. 2 (24-hr.)	Dec. 4 (24-hr.)	Dec. 6 (a. m.)
Specific gravity	1.024	1.024	1.024	1.025	1.018
Reaction	5.0	7.5	7.0	4.5	4.0
Albumin (percent by volume)	1.75	2	1	1	2
Sugar	Neg.	Tr.	Neg.	Neg.	Neg.
Pus cells	occ.	occ.	1 per h.p.f.	6 per h. p. f.	occ.
Casts	Sm. granular, 25 per l. p. f.; coarse granular, 6 per l. p. f.				

Blood count

	Hemoglobin	Erythrocytes	Leucocytes	Polys.	Monos.
	Percent			*Percent*	*Percent*
Nov. 29	88	4,020,000	8,200		
Nov. 30			9,250	80	20
Do			9,250	80	20
Dec. 1			9,250	83	17
Dec. 2			9,150	78	22
Dec. 3			9,150	89	11
Dec. 5	[1] 104	5,030,000	12,100	89	11
Dec. 9	[2] 96	4,520,000	9,100	91	9

[1] 14.2 gm.
[2] 14 gm.

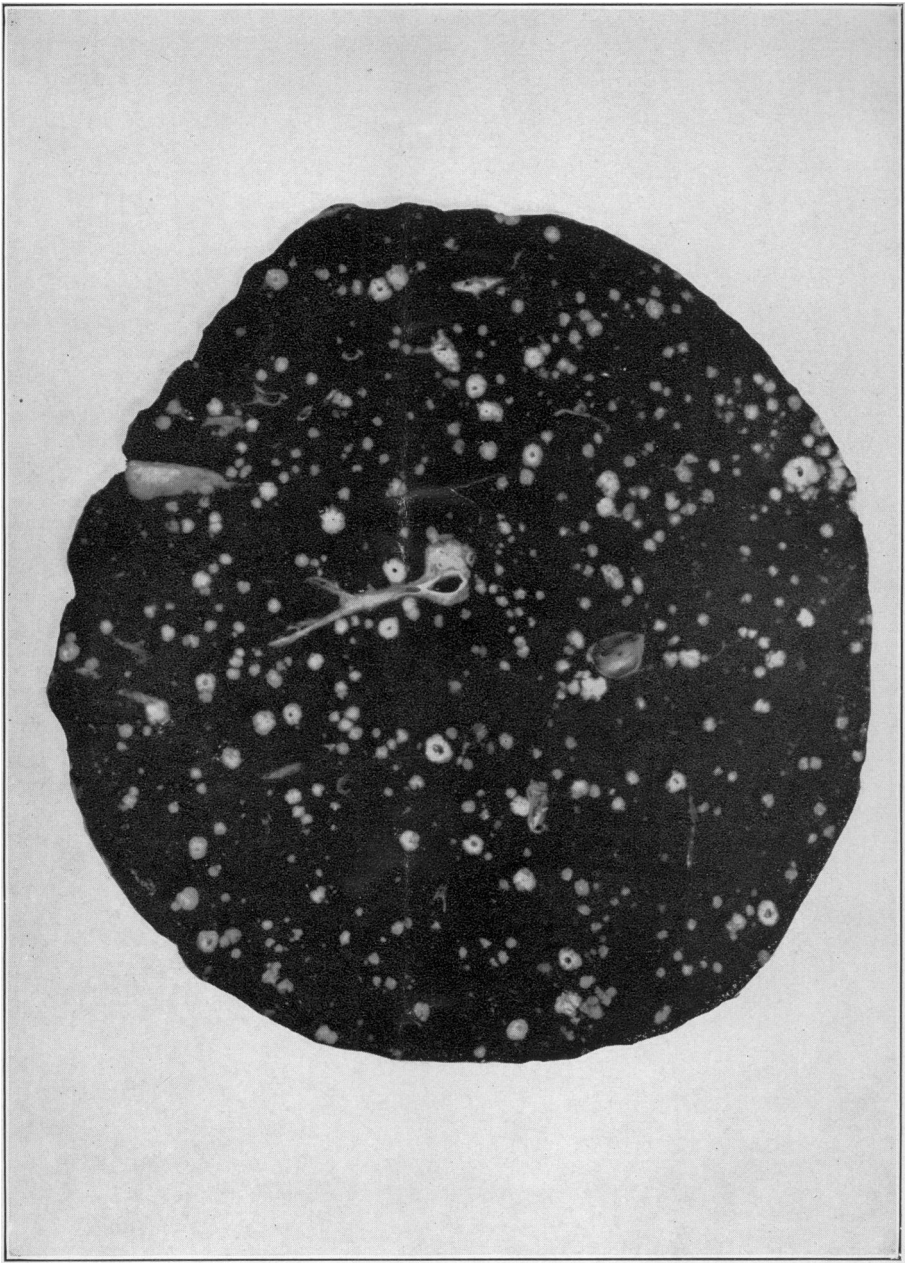

FIGURE 1.—Cross section of liver from human case.

Special tests

Date	Test	Results
Dec. 1	Stool for parasites and ova	None found.
Dec. 2	Typhoid	Negative.
	Para A and B } Agglutinations	Negative.
	Undulant fever	Negative.
Dec. 3	Blood culture (Dec. 2, 1937, 8 a. m.)	Negative (24 hrs.).
Dec. 4	Blood culture (Dec. 2, 1937, 8 a. m.)	Negative (48 hrs.).
	2nd Widal taken Dec. 2, 1937	Negative.
	Icterus index	24.5.
Dec. 6	Smear from liver	No tb. found.
Dec. 8	Blood culture (Dec. 2, 1937)	Gram-neg. bacilli.
Dec. 10	Blood culture (Dec. 8, 1937)	Negative (48 hrs.).
	Spinal fluid	Leucocytes, 0.
		Globulin, negative.
		Wassermann, negative. (Kolmer.)

X-ray

Dec. 3, Chest.—Shows a slight increase in density of the left costophrenic angle. The remainder of lung fields is clear. The right diaphragm is held unusually high, suggestive of subdiaphragmatic pathology.

Diagnosis.—Congestion left lung field. Probable subdiaphragmatic pathology.

The microscopic sections on the material removed for biopsy were described as follows:

"Section shows an intact capsule surrounding the liver lobules. In the central portion immediately beneath the capsule there is an accumulation of necrotic cells surrounded by connective tissue containing polymorphonuclear cells, eosinophiles, and round cells. The liver cells are heavily laden with blood pigment. Surrounding the hepatic vessel there is a dense cellular infiltration consisting of round cells, plasma cells, and an occasional polymorphonuclear cell. There is no evidence of giant cells."

POST MORTEM

The gross findings at autopsy were reported by Dr. Alfred L. Balle as follows:

"This is the unembalmed body of a fairly well developed white male 29 years of age. The skin in general shows a yellowish color from jaundice. The chest is symmetrical. There are no palpable glands in the cervical, axillary, or inguinal regions. There is a recent unhealed right rectus surgical incision. The extremities show no changes.

"The subcutaneous fat in the usual midline incision measures 2 cm in thickness. The peritoneum is moist, smooth, and glistening. The peritoneal cavity contains a large amount of straw-colored fluid. The bowels are of uniform caliber. The liver is 8 cm below the costal border in the right mid-clavicular line. The capsule of the liver is studded with numerous small, yellowish-gray necrotic areas measuring from 1 mm to 1 cm in diameter. The liver parenchyma is firm and reddish brown in color. The necrotic areas show no liquification, and they are uniformily distributed throughout the parenchyma. The gall bladder is large and distended with bile. There is a small number of the same necrotic areas in the head of the pancreas. There is a hyperplasia of some of the mesenteric nodes, with two of them showing calcification. The spleen is large, about three times normal size. The capsule is smooth and the cut surfaces show a diffuse lymphoid hyperplasia with a diminution of the Malpighian corpuscles. The cecum is mobile, and its mesentery is attached to the left side. There is also a fibrous band binding the ascending colon to the descending colon. When the large and small bowels are opened, there is no evidence of ulceration or infiltration of the bowel wall. The

kidneys show no changes except for cloudy swelling. The adrenal glands are unchanged. The prostate is not enlarged and is soft.

"When the chest is opened, the lungs fail to meet in the midline by a distance of 8 cm. Both lungs are well aerated and lie free in their pleural cavities. The right pleural cavity contains about 250 cc of bloody fluid. The cut surfaces of the lungs show no evidence of pulmonary consolidation. When the heart is opened, there are no valvular changes. The endocardium is smooth.

"Multiple necrotic areas of the liver.

"Marked enlargement of liver.

"Splenomegaly—lymphoid hyperplasia.

"Necrotic areas in head of pancreas.

"Sero-sanguinous fluid in right pleural cavity.

"Ascites.

"Jaundice.

"Recent unhealed surgical incision."

The material from some of the lesions in the liver was injected into a guinea pig and the animal died the following day with lesions in the liver resembling those exhibited by the patient.[1] The same organism which was recovered from the blood culture was also recovered from the liver lesion. A culture made from the liver lesion of the patient was agglutinated by the patient's own serum in a dilution of 1 to 320.

The graphic chart shows the type of temperature, pulse, and respiration reaction to this infection.

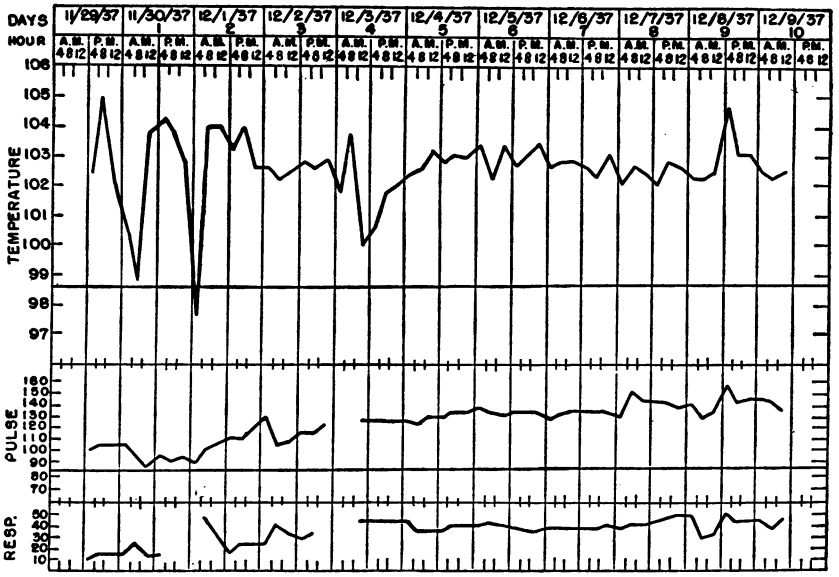

Chart showing type of temperature, pulse, and respiration in infection with *B. pseudotuberculosis rodentium*.

BACTERIOLOGY

The bacteriological work was done with a pure culture isolated from the liver of the patient after death. All experimental observations were made at the National Institute of Health, Washington,

[1] In the light of later work with pure cultures, this observation is open to question.

D. C. The culture was received on solid media and immediately plated on agar for purity. From isolated single colonies, broth and agar slants were inoculated.

On agar the colonies were small, discrete, very slightly raised, opalescent, and had smooth edges. Gram stains made on smears from agar revealed Gram-negative coccoid-like organisms. In broth the organism grew in flake-like clumps that settled to the bottom leaving very little turbidity. After several days, flakes could be seen on the sides of the tubes and eventually a heavy pellicle was formed. Gram stains made from the broth revealed a Gram-negative, short, thick bacillus growing singly and in short chains. There was a definite tendency to bipolar staining.

Early workers with the organism occasionally reported motility (Courmont (*19*); Nocard and Masselin (*7*); Kossel and Overbeck (*20*)). However, the organism has been generally considered nonmotile. Arkwright (*21*), in 1927, stated that it would appear that the organism was sufficiently motile at 20° C. to distinguish it from *P. pestis*. He further stated that the absence of motility was often a temporary feature of a culture and may be only the result of growth on special media or may be a nonmotile variant. "The presence or absence of motility under these different circumstances has been consistently correlated with definite changes in the agglutination phenomena." Bishop (*11*) could not demonstrate motility, even when grown at 20° C., in any of the six strains she isolated from guinea pigs. Motility could not be demonstrated in our culture when grown either in broth or the water of condensation of an agar slant either at room temperature or 37° C. The organisms, however, revealed marked Brownian movement.

Table 1 compares the cultural characteristics of our organism with those of cases reported in the literature for *B. pseudotuberculosis rodentium* as given in a review by Schütze (*13*). The only variation from the typical was the slight fermentation of sorbitol. This, however, has been noted by various observers previously. The results as given were recorded up to 7 days when incubated at 37° C. Late fermentation of certain of the carbohydrates necessitates a prolonged period of observation of at least 7 days.

Since Reimann and Rose (1931) (*2*) and Reimann (1932) (*22*), as well as Poppe (1927) (*3*), suggested the possible relationship between pseudotuberculosis rodentium and tularaemia, agglutinations were set up with cultures of our organism and diagnostic tularaemia antiserum. There was no agglutination in any dilution. Neither was there any agglutination of our organism by diagnostic *Brucella* antiserum.

TABLE 1.—*Comparison of cultural characteristics of organism in case here reported with those of cases in the review of the literature by Schütze*

	Pseudotuberculosis rodentium (Schütze review, compiled)	S 274 (our culture)		Pseudotuberculosis rodentium (Schütze review, compiled)	S 274 (our culture)
Motility	±	−	Rhamnose	+	+
Gram	−	−	Salicin	+	+
Indole	−	−	Trehalose	+	+
H_2S	−	−	Xylose	+	+
Gelatin	−	−	Amygdalin	−	−
Litmus milk	neut.—alk.	neut.—alk.	Erythritol	−	−
Arabinose	+	+	Inositol	−	−
Galactose	+	+	Inulin	−	−
Dulcite	−	−	Lactose	−	−
Dextrose	+	+	Raffinose	−	−
Levulose	+	+	Sacharose	−	−
Maltose	+	+	Sorbitol	−	Tr.
Mannitol	+	+	Nitrites	+	+
Glycerin	+	+			

Pasteurella pestis has been recognized by many observers to be very closely related to *B. pseudotuberculosis* both culturally and antigenically. Its close relationship is discussed by Schütze (*13*). This close relationship is emphasized and discussed at length by Wu Lien Teh, J. W. H. Chun, R. Pollitzer, and C. Y. Wu in their comprehensive monograph (*23*) (1936). They place the organism in the *Pasteurella* group. The ultimate in recording the similarity of *B. pseudotuberculosis rodentium* and *P. pestis* was reached by Bezsonova and coworkers (*24*), in 1936. These authors report five cases of spontaneous transmutation of *pestis* into *pseudotuberculosis* among a collection of 214 strains of plague.

The characteristic pleomorphism of plague on 3 percent salt agar was studied and compared with that of *pseudotuberculosis* on the same medium; it consisted of coccoid, longer rods, and some swollen forms, but it was not the characteristic pleomorphism of plague. In litmus milk, three known cultures of *P. pestis* produced a permanent slight acidity. One known culture of *pseudotuberculosis* and our test culture both failed to change litmus milk until after the fourth day, when a definite alkalinity was produced.

A phenol-killed saline suspension of a 24-hour culture (No. 274) was injected into two rabbits intravenously. The suspension was approximately of the density of (500 million cc) standardized typhoid vaccine. Injections were given as follows:

First day	0. 5 cc
Fourth day	. 5 cc
Eighth day	1. 0 cc

The animals were bled from the heart on the 15th day. The serum was then used for agglutination tests. The culture from the human strain (No. 274), and two strains of *pseudotuberculosis rodentium* in the National Institute of Health collection (received from MacConkey, Pasteur Institute, about 1908, No. 519 and No. 111) were used as

antigens. These three cultures were all difficult to emulsify, and suspensions in physiologic saline (0.85 percent) showed a marked tendency to settle spontaneously. It was found that by emulsifying a 24-hour agar culture grown at room temperature in 0.30 percent saline and shaking well with glass beads, a smooth, fairly stable, antigen could be prepared. These were then used in the agglutination tests. Saline of 0.30 percent concentration was used throughout to prevent spontaneous agglutination.

Agglutinations

Antiserum prepared in rabbits with culture No. 274 (human strain of this study)

Antigen	Dilutions of antiserum										
	1:10	1:20	1:40	1:80	1:160	1:320	1:640	1:1280	1:2560	1:5120	Control
Culture No. 274	4	4	4	4	4	4	4	3	2	1	0
Pseudotuberculosis rodentium 519	4	4	4	4	4	4	4	3	2	1	0
Pseudotuberculosis rodentium 111	4	4	4	4	4	4	3	2	1	±	0

A granular type of agglutination was seen in all tubes, especially when viewed through a hand lens.

When the antiserum was absorbed by either No. 519 or No. 111, there no longer was agglutination of No. 274 by the antiserum.

From these serological studies, our culture No. 274 was considered to be identical with these two known strains of *pseudotuberculosis rodentium*. Previously in this report, reference was made to the lack of agglutination of our culture with tularaemia and abortus antisera. In addition to these, agglutination tests were made with *S. suipestifer*, *S. enteritidis*, *S. aerytrycke*, *E. typhi*, *S. dysenteriae*, *S. paradysenteriae* (Flexner), and *S. paradysenteriae* (Sonne) antisera, but in no instance was there agglutination. All agglutination tests were macroscopic, test-tube method, incubated at 37° C. for 2 hours, placed in the refrigerator over night; and the final reading was made in the morning.

The results of animal inoculation were interesting in differentiating *pseudotuberculosis* from *pestis*. Guinea pigs were first selected for tests of pathogenicity. A saline suspension of a 24-hour agar culture of living organisms was roughly standardized so that its density was about that of typhoid vaccine. A subcutaneous dose of 0.25 cc was inoculated into the left groin, and a similar amount was given intraperitoneally to a second pig.

The pig inoculated intraperitoneally was alive on the third day, when he was killed. Fibrinous deposits were seen on the surface of the liver and spleen. These two organs were studded with very small white spots. Broth cultures were made from the heart blood, peritoneal fluid, and urine, and a saline emulsion was made of the liver

and spleen. The latter was also inoculated subcutaneously into one guinea pig and intraperitoneally into another.

In 24 hours there was a good growth in all of the broth cultures. The same organism was present in the cultures that had been inoculated into the animals, as shown by identical cultural characteristics listed in table 1, for the organisms recovered from the heart blood and from the liver and spleen emulsion.

The guinea pig inoculated intraperitoneally with an emulsion of the liver and spleen from the pig discussed above died on the 7th day. At autopsy, the liver and spleen were again covered with a deposit of fibrin and both organs were studded with greyish white spots. Again the heart blood and liver and spleen emulsion yielded pure cultures of the original organism. This was considered to be fairly conclusive evidence that the organism isolated from the human case was capable of producing the pathology. In this animal, there were small greyish white lesions in the lungs.

The pig inoculated subcutaneously with an emulsion of liver and spleen had bilateral lymphadenitis in the inguinal regions. It was killed on the 7th day, and it also had lesions of the liver, spleen, and lungs. Dr. George W. McCoy, of the Public Health Service, was present at the autopsy and stated that the gross pathology closely resembled that of plague or tularaemia in that particular guinea pig.

The pig inoculated subcutaneously with 0.25 cc of a broth culture was finally killed on the 24th day. A large local abscess filled with caseous material was present at the site of inoculation. The regional glands were enlarged and had caseous centers. The liver had many greyish white areas, larger than those seen previously. The spleen also had many characteristic lesions.

In summarizing the guinea pig inoculations, the following facts are brought out:

(1) 0.5 cc of saline emulsion of the organism inoculated intraperitoneally is fatal in 7 to 10 days, producing characteristic pathology in the liver and spleen.

(2) 0.5 cc of saline emulsion of the organism inoculated subcutaneously is fatal in from 15 to 45 days. It produces a local slough, regional lymphadenitis, lesions in liver, spleen, and sometimes lungs.

(3) Pure cultures can be recovered from heart blood, regional lymph glands, liver, spleen, and occasionally urine.

White rats have been used to differentiate plague from pseudotuberculosis. Plague is pathogenic for these animals, producing characteristic lesions, while pseudotuberculosis is relatively harmless. Our test culture was inoculated into four white rats, two by the intraperitoneal route and two by subcutaneous injection. One of each of these groups was killed after 3 weeks. The lymph glands were not enlarged and there were no lesions of the spleen, liver, or lungs in

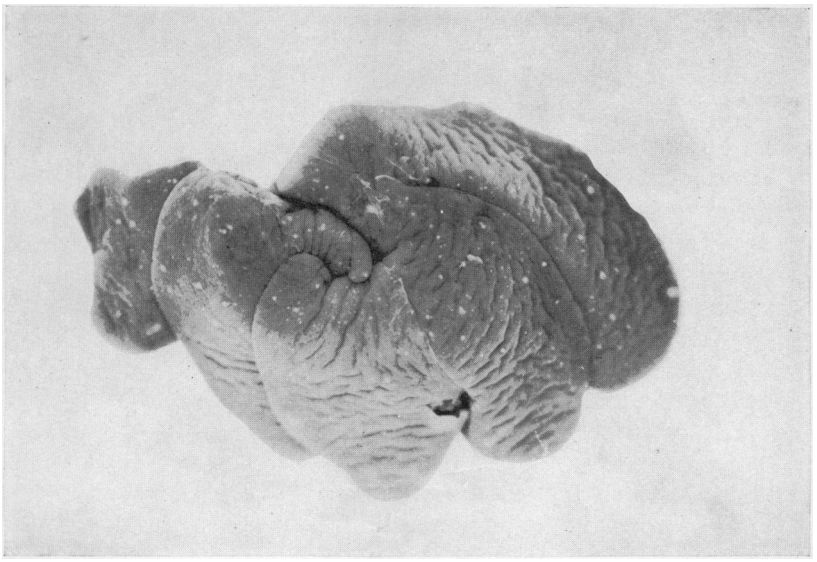

FIGURE 2.—Liver of guinea pig inoculated subcutaneously with 0.5 cc of a saline emulsion (× 1½, reduced approx. ⅓).

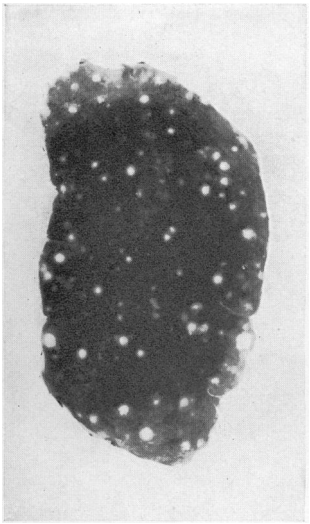

FIGURE 3.—Spleen from same guinea pig (× 1½).

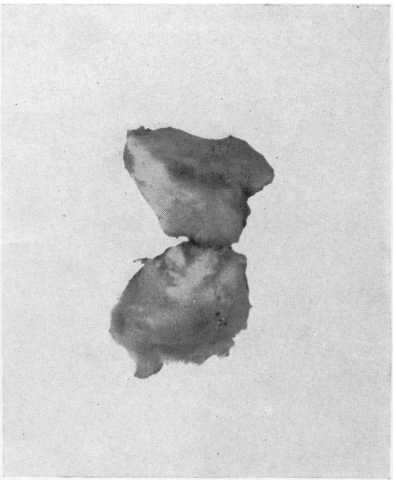

FIGURE 4.—Caseous inguinal lymph node from same guinea pig (× 1½).

either animal. The second two were killed after 6 weeks and again no lesions were found, nor were we able to culture the organism from the heart blood, lymph glands, liver, or spleen.

In rabbits, the organisms produced as typical a picture as in guinea pigs. A washed saline suspension of living organisms was inoculated intravenously in two rabbits; one received 0.5 cc and was dead on the 4th day, and one received 0.25 cc and died on the 5th day. Very small greyish white lesions were seen in the spleen; no gross lesions were visible in the liver or lungs. Two rabbits were inoculated with a living saline suspension subcutaneously. A necrotic area was found at the site of inoculation on the abdomen. There was marked lymphadenopathy of the axillary and inguinal glands. At autopsy, typical greyish white nodules were seen on the liver and spleen. These were firm, with some softening of the centers. The lymph glands were caseous. Pure cultures of *pseudotuberculosis rodentium* were recovered from the heart blood, lymph glands, liver, and spleen.

Histopathological studies on the experimental animals were made by the division of pathology at the National Institute of Health. Several of the individual protocols are presented in the following:

PSEUDOTUBERCULOSIS

G. P. Autopsy No. 12895. March 9, 1938

(Killed February 2, 1938)

Liver.—There is much partially karyorrhectic fibrinopurulent exudate on the capsule, with numerous large clumps of Gram-negative bacilli. The liver substance shows numerous foci of necrosis scattered throughout the lobules and particularly beneath the capsule. Part of these focal necroses are composed of coagulated necrotic liver cells, which may or may not retain pale basophilic nuclei. Some of these coagulated foci contain interstitial hemorrhage. Usually the contained Kupffer cells are swollen and lightly basophilic. Part of the foci are of mixed type and the remainder of the foci are composed of granular oxyphil debris, with quite numerous intact and fragmenting polymorphonuclear leucocytes. Some such foci may contain hemorrhage; others not. Often, but sometimes not, there are large clumps of Gram-negative bacilli within these foci, and sometimes, but more often not, there is some marginal proliferation of large foamy epithelioid cells. The bacilli are short and thick, with rounded ends. More often they are only slightly basophilic and rather poorly staining. Sometimes deeply stained organisms are seen. Usually the granules of the polymorphonuclear leucocytes are dissolved and are not stainable. A few of the necrotic liver cells show early calcification. Moderate numbers of mitoses are seen in Kupffer cells diffusely and in focal necroses. Kupffer cells are often swollen, and there is some coarse fatty vacuolation of the liver cells diffusely.

Spleen.—Shows similar capsular exudate; the splenic follicles are usually small but hyperplastic. The lymph clefts in them are dilated, and contain free nuclear debris and fragmenting cells. The follicle reticulum cells are somewhat swollen and may be phagocytic for nuclear debris, and a few foci of necrosis are seen within follicles. The pulp is congested, contains numerous polymorphonuclear leucocytes, few normoblasts, and shows a little peritrabecular infiltration by small and

large lymphocytes. There are quite numerous small hyalin thrombi, and there is irregular focal reticuloendothelial swelling and vacuolation in spleen pulp. There are numerous focal necroses containing granular oxyphil debris, polymorphonuclear leucocytes, fragmenting leucocytes, red corpuscles and large clumps of bacilli similar to those seen in the liver. More often than not, there is proliferation of foamy reticulum cells or epithelioid cells, often marginally.

Lymph node.—A small lymph node shows dilated sinuses, irregular sinus reticuloendothelial proliferation of quite marked grade with focal purulent abscesses. The lymph node follicles are quite hyperplastic.

PSEUDOTUBERCULOSIS

G. P. 10. Autopsy No. 13010. March 23, 1938

Spleen.—Follicles of moderate size, moderately hyperplastic, and showing follicle reticulum cell swelling. Pulp shows marked sinus reticuloendothelial hyperplasia, dilated sinuses containing a moderate amount of blood and moderate numbers of polymorphonuclear leucocytes. There is quite marked peritrabecular and interstitial infiltration by lymphocytes, large lymphocytes, and quite numerous plasma cells.

Liver.—Quite dense periportal infiltration by lymphocytes and plasma cells, focal interstitial lymphocyte infiltration, occasional periportal granuloma of large fusiform and polygonal epithelioid cells.

Kidney.—Normal.

Lymph node.—Shows a large central caseopurulent abscess bordered by palisading fusiform or irregularly disposed stellate and polygonal epithelioid cells.

Subcutaneous inoculation lesion.—There is central caseopurulent abscess encapsulated by fibroblasts and fibrosis, with some polymorphonuclear infiltration adjacent to the abscess cavity. This is surrounded by fibrosing atrophic skeletal muscle, and there are a few calcified muscle fibers.

Lung.—One lobe shows emphysema with scattered peribronchial nodules of fragmenting purulent pneumonic exudate with interstitial fibroblast proliferation and columnar epithelium lining some of the antra.

A second lobe is extensively consolidated, showing a nodular purulent pneumonia with interstitial fibroblast proliferation and lymphocyte infiltration, alveolar epithelioid macrophage exudation, focal abscessing, and diffuse edema and pus in the bronchi.

In a third lobe similar process is seen. There is more proliferation of columnar epithelium into the antra and there is a large area of coagulated necrotic lung.

PSEUDOTUBERCULOSIS

Rabbit 6. Autopsy No. 13034. March 23, 1938

Diagnosis.—Subacute pseudotuberculosis.

Lung.—One lobe is extensively consolidated; there are a few intra-alveolar masses of fibrin or caseous exudate invaded largely by fibroblasts and epithelioid cells. Many discrete and coherent epithelicid cells within alveoli grading into solid epithelioid masses. Much interstitial fibroblast and epithelioid proliferation, with focal lymphocyte infiltration grading into large granulomata, some over 1 mm in diameter; these may present focal karyorrhectic necrosis. Small arteries and veins show focal intimal fibroblast and epithelioid proliferation grading into solidly obliterating masses of polygonal epithelioid cells with the caps including clumps of polymorphonuclear leucocytes or nuclear debris. There are few caseous thrombi; occasional megakaryocytes are seen in capillaries.

In the second lobe there are scattered nodules of intra-alveolar and interstitial formation of epithelioid granulomata. There is much endangeitis as described above grading into obliteration with polymorphonuclears and focal karyorrhexis and polygonal epithelioid cells.

Omentum.—Shows few foci of slight mesothelial proliferation or slight interstitial fibroblast proliferation.

Pancreas.—Normal.

Pancreatic lymph node.—Shows much macrophage exudation into the sinuses, marked sinus reticuloendothelial hyperplasia grading into solid masses of polygonal epithelioid cells, which may in turn exhibit central karyorrhexis or even caseation.

Adrenals.—Show irregular interstitial lymphocyte infiltration in the medulla and reticular zone, and a few caseous thrombi in capillaries in the reticular zone.

Spleen.—Very markedly enlarged, only three or four small lymphoid follicles are recognizable, the pulp shows confluent, karyorrhectic caseous nodular necrosis, apparently of macrophages and reticuloendothelial cells with interstitial hemorrhage and hemorrhagic pulp in the interstices between the nodules; little surviving spleen pulp which is congested.

Liver.—Shows very numerous focal caseous karyorrhectic or fibrinous necroses, part showing marginal proliferation of fusiform epithelioid cells and fibroblasts, occasionally grading into nodules of solid polygonal or loose fusiform epithelioid cells forming granulomata which occasionally contain giant cells or are infiltrated by lymphocytes. There are no necrotic liver cells remaining recognizable.

Heart.—There is one calcified muscle fiber seen in the left ventricle, one focus of fibroblastic replacement of muscle in the right ventricle.

Kidney.—Finely granular convoluted tubules containing a little albuminous exudate and one small focus in the cortex of replacement by fat cells.

Pseudotuberculosis

Rabbit 5. Autopsy No. 13023. March 23, 1936

Spleen.—Much enlarged, follicles are of moderate size and hypoplastic, pulp is engorged with hemolyzed blood filling the dilated sinuses, there are many foci of caseocoagulative necrosis without marginal reaction, composed probably of necrotic monocytes. These foci vary in size from a few cells to about 0.2 mm in diameter.

Kidney.—Convoluted tubules are dilated, finely granular and contain hyalin and granular oxyphil exudate. There is one focus of interstitial fatty infiltration in the cortex.

Liver.—Very numerous fibrinokaryorrhectic thrombi grading into fibrinokaryorrhectic caseous necroses with or without coagulated liver cell cords and with or without slight marginal Kupffer cell proliferation.

Lung.—Slight to rather marked congestion, marked diffuse serous exudation, focal hemorrhage, few small foci in which septa are coagulated and necrotic, and one fibrinocaseous thrombus in a vein.

SUMMARY

A human case of infection with *B. pseudotuberculosis rodentium* is presented. This is the first original report of its kind we have found in the English or American literature, and apparently the fifth authentic report in the medical literature. Observations on the bacteriology, pathogenicity, serology, and pathology are presented and discussed in the text.

CONCLUSIONS

1. *B. pseudotuberculosis rodentium* (Pfeiffer) is capable of infecting man, resulting in a clinical disease of fever, vague abdominal tenderness, and in this case jaundice. At autopsy numerous greyish white nodules were seen throughout the liver.

2. Our culture *B. pseudotuberculosis rodentium* is pathogenic for guinea pigs and rabbits but not for the four white rats inoculated with a similar dosage and in a similar manner.

3. Characteristic lesions in the liver, spleen, and lymph nodes are produced by the organism in guinea pigs and rabbits.

4. The organism may be differentiated from *Pasteurella pestis* by cultural characteristics and animal inoculation.

5. A case of human infection with *B. pseudotuberculosis rodentium* is described for the first time in the United States.

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STUDIES ON DENTAL CARIES

V. FAMILIAL RESEMBLANCE IN THE CARIES EXPERIENCE OF SIBLINGS[1]

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INTRODUCTION

Similarities within families with respect to relative immunity or susceptibility to dental caries have been noted by several investigators. In a study of 325 children and their parents, Day and Sedwick (*1*) report that the parents of children with extensive caries showed an average loss of two permanent teeth more than the parents of children with low levels of caries. Bunting (*2*) states that "inherited or inherent individual characteristics, in a small percentage of cases, are more important determinant factors in caries susceptibility than ordinary dietary considerations." Kappes (*3*) has reported that, among the parents of 50 children with "good teeth," 2 parents had "poor teeth" and 9 had "good teeth," while among the parents of 50 children with "poor teeth," 13 had "poor teeth", and 2 had "good teeth." Detlefsen (*4*) has observed that the extent of caries in the first permanent molars appears to have a small but appreciable genetic background. In a discussion of the problem of clinical control of dental caries, Kugelmass (*5*) points out that "hereditary predisposition to caries susceptibility or immunity appears evident in some children."

Detailed analyses of the constitution of the caries problem in large population groups would appear to warrant further study of the relation between familial factors and relative susceptibility to caries. Accordingly, an investigation of this relationship was developed as one of a series of studies (*6*, *7*, *8*, *9*) on dental caries in grade school children. The present paper is concerned largely with a description of the dental status of the siblings of one group of children designated as "caries immunes," and of the siblings of another group designated as "caries susceptibles." Analysis of the data furnishes quantitative evidence which indicates that significantly less caries is found in the deciduous and in the permanent teeth of the siblings of the immune group than in the siblings of the caries susceptible group.

[1] From Child Hygiene Investigations, Division of Public Health Methods, National Institute of Health, United States Public Health Service.

The preceding papers of this series are as follows:

I. Dental status and dental needs of elementary school children. By Henry Klein, C. E. Palmer, and J. W. Knutson. Pub. Health Rep., 53: 751-765 (May 13, 1938).

II. The use of the normal probability curve for expressing the age distribution of eruption of the permanent teeth. By Henry Klein, C. E. Palmer, and M. Kramer. Growth, 1: 385-394 (1937).

III. A method of determining post-eruptive tooth age. By C. E. Palmer, Henry Klein, and M. Kramer. (In press.) Growth.

IV. Tooth mortality in elementary school children. By John W. Knutson, and Henry Klein. Pub. Health Rep., 53: 1021-1032 (June 24, 1938).

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MATERIAL AND METHODS

Basic data [2] for the present study were obtained from dental examinations of 4,416 elementary school children who comprised 94 percent of the entire enrolled grade school population of a small urban community, Hagerstown, Md. Records of these examinations contained observations on the number of deciduous and permanent teeth present in the mouth and detailed descriptions of the location and specific surface involvement of the teeth by carious lesions. Carious lesions were those so designated by trained dental officers on the basis of a careful clinical examination with mirror and sharp pointed pig-tail explorer. Pits and fissures in which the explorer caught were itemized separately. For the permanent teeth, caries experience was measured by means of a count of decayed, missing, or filled (DMF)[3] teeth and tooth surfaces. In counting carious tooth surfaces, remaining roots and missing (extracted) permanent teeth were considered equal to five carious surfaces. For the deciduous teeth, an equivalent measure of the total caries experience was not possible, since definite information which would show whether or not a missing deciduous tooth had ever been carious was not available from the data collected. For the deciduous teeth, therefore, caries experience was expressed by means of a count of teeth and tooth surfaces which, at the time of the examination, were actually carious or were filled.

Definitions and the selection of immune and susceptible children.— For purposes of the present analysis, certain children from among the 4,416 examined were selected as "caries immunes" and "caries susceptibles." Immune children were defined as those who at ages 10 through 15 showed *in the permanent teeth* no objective evidence of caries experience (no DMF teeth). Caries susceptible children were defined as those who at age 10 had 6 or more; at age 11, 7 or more; at age 12, 8 or more; at age 13, 9 or more; and at ages 14 and 15, 10 or more DMF teeth.

These criteria are clearly arbitrary, and it is recognized that entirely homogeneous groups were not selected thereby. An important disturbing factor probably arises from the fact that different levels of relative immunity to caries are represented in the immune group. For example, a child 15 years of age who has no carious permanent teeth may possess a higher level of immunity than a child of 10 who, though not having DMF teeth at this age, may develop the disease by the age of 15. Similar disturbing factors may be present in the definition of susceptibles, although it seems probable that these are partially obviated by the graduated scale of defining susceptibility in terms of the severity of the disease.

[2] A full description of the manner of collecting these data and a general analysis of the findings are given in reference *6*.

[3] For a full discussion of the DMF concept, see reference *10*.

In connection with a discussion of the comparability of the immune and susceptible children, it is necessary at this point to mention the unexpected finding of significant differences in the populations of teeth in the two groups of children. In brief, this consisted of the finding of a greater average number of erupted permanent teeth in the susceptibles than in the immunes and, as a direct corollary, a smaller average number of deciduous teeth present in the mouths of the susceptibles than in the mouths of the immunes. The full significance of this observation is not immediately apparent and requires additional study. In explanation of the observation, however, it may be mentioned that the method of designating the immunes and the susceptibles probably accounts, in part, for the differences, since children selected as susceptibles were required to have a specified minimum number of DMF teeth, and those selected as immunes no DMF teeth.

By these criteria, children with larger numbers of erupted permanent teeth may tend to fall into the susceptible group; those with smaller numbers of erupted teeth may tend to fall into the immune group. A specific mechanism which might bring about this differential selection may be illustrated as follows: If the teeth of a particular child tend to erupt at an early age, these teeth will have been exposed to the environment of the mouth longer, at the same chronological age, than will be those of a child whose teeth tend to erupt at a relatively late age. Since, as will be shown in a subsequent paper in this series of studies, attack by dental caries is related to the length of time the teeth are exposed in the mouth, children whose teeth erupt early may be expected to show, at the same chronological age, more caries than children whose teeth erupt late. The selection of susceptible children (those having a specified minimum number of DMF permanent teeth) may tend to pick out individuals who are "early eruptors";[4] immune children, on the other hand, may tend to be "late eruptors." The presence of relatively more early eruptors in the susceptible group and relatively more late eruptors in the immune group may be the factor which accounts for the finding of more erupted permanent teeth in the former than in the later group of children. As just indicated, however, the full significance of this finding is not clear at the present time. Nevertheless, its possible implications must be borne in mind in studies on the relative immunity and susceptibility of children to dental caries.[5]

[4] "Early eruptors" may be expected to have, at comparable ages, more permanent teeth erupted than "late eruptors."

[5] In order to compare the caries experience of two groups of children of the same chronological age, one group having significantly more permanent teeth erupted than the other, it would appear to be necessary to take account of the fact that the teeth of one group have been exposed to the environment of the mouth for a longer period of time than the teeth of the other group. One method of taking account of this factor involves the expression of caries experience in terms of "posteruptive tooth age." An application of this method will be found in a later section of this paper.

Of the grade school pupils examined, 357 children were designated as caries immune and 270 as caries susceptible by means of the criteria previously described. Since nearly all of the children of elementary school age in the community were examined, dental records for essentially all of the brothers and sisters (of school age) of these immune and susceptible children were available for study. Some of the caries immunes and caries susceptibles were, however, themselves siblings. Actually, 2 immune siblings were observed in each of 27 families and 2 susceptible siblings were observed in each of 13 families; 3 immunes were found in each of 2 families and 3 susceptibles in 3 families; 4 immunes were observed in 1 family. In each of two families, one immune and one susceptible was found.

Definitions and selection of siblings of immune and susceptible children.—In order to select children who may be considered as representative of *siblings of immunes* and *siblings of susceptibles*, it is necessary to define, specifically, at this point, the method of designating siblings. This definition may be subdivided to apply to two major groups of families, namely, (*a*) those families in which only one immune or susceptible child was observed, and (*b*) those in which there were observed more than one immune or more than one susceptible child. In the first group of families, all children examined, except the immune or susceptible child, were designated as siblings. In the second group, the older or eldest immune or susceptible child was eliminated and the other children, regardless of their immunity or susceptibility, were designated as siblings. For the two families in which both an immune and a susceptible were found, the immune child was counted among the siblings of the susceptible and the susceptible child was counted among the siblings of the immunes.

Index cases and siblings —By means of the procedures and criteria discussed in the preceding sections, two special classes of children were obtained: First, a group of 184 immune and a group of 117 susceptible *propositi or index cases;* second, a special class of *siblings* composed of a group of 306 brothers and sisters of immune index cases and a group of 182 brothers and sisters of susceptible index cases. Among the 306 siblings of immunes, there were included, by the method used for designating siblings, 34 children who meet the criteria for susceptibles and 1 who meets the criteria for the immunes. Among the 182 siblings of susceptibles, 19 children meet the criteria of susceptibles and 1 child meets the criteria of immunes.

Since each index case represents a family containing one or more immune or susceptible children, it may be noted that 182 "caries immune families" and 117 "caries susceptible families" constitute the number of different family groups from which the children are drawn. The age and sex distributions of the 301 index cases through which

these families were selected, as well as the distributions for their 488 siblings, are given in table 1.

TABLE 1.—*Age and sex distributions of 301 caries immune (I) and caries susceptible (S) index cases and those of their 488 siblings. (Data derived from dental examinations of 4,416 grade school children, Hagerstown, Md.)*

Sex	Caries group	Age (years last birthday)										
		6	7	8	9	10	11	12	13	14	15	All ages
		Index cases										
Boys	S	------	------	------	------	3	8	13	8	8	7	47
	I	------	------	------	------	26	22	16	26	7	1	98
Girls	S	------	------	------	------	14	7	14	17	12	6	70
	I	------	------	------	------	30	26	19	6	3	2	86
Both sexes	S	------	------	------	------	17	15	27	25	20	13	117
	I	------	------	------	------	56	48	35	32	10	3	184
		Siblings										
Boys	S	3	11	15	10	17	15	14	8	2	2	97
	I	16	17	25	19	21	16	20	15	11	1	161
Girls	S	8	5	8	14	15	6	9	13	4	3	85
	I	7	13	28	24	17	8	18	14	15	1	145
Both sexes	S	11	16	23	24	32	21	23	21	6	5	182
	I	23	30	53	43	38	24	38	29	26	2	306

FINDINGS

Caries immunes and caries susceptibles within the same family.—Preliminary to the presentation of findings on the dental status of the siblings of the immune and susceptible children, information of some significance regarding one aspect of the familial characteristic of caries can be derived from a study of the distribution of caries immunes and caries susceptibles within families. Thus, the fact may be derived from the material presented above that 2 or more immunes were found in 30 families, 2 or more susceptibles were found in 16 families, and in only 2 families were both an immune and a susceptible observed. No detailed test will be presented here of the statistical significance of this distribution of occurrence of more than one immune, or susceptible, child in the same family; but it is apparent that certain families are marked by caries immunity while other families are characterized by caries susceptibility.[6] Such an impression may be derived from the simple fact that 46 out of 48 families each contain 2 or more children who show similarity in respect to caries immunity or susceptibility while in only 2 families does this direct similarity fail to hold. This preliminary finding strongly suggests the existence of familial resemblances in caries immunity or caries susceptibility.

[6] The meaning of the terms "caries immunity" and "caries susceptibility," as used in this report, is restricted to the meaning supplied in the definitions and discussion given in the section of this paper headed "Material and methods."

Methodological considerations in the comparison of caries experience in the permanent teeth of the siblings.—On the basis of the data just presented, siblings of immune children might be expected to show significantly lower levels of caries experience than siblings of caries susceptible children. Before proceeding with the presentation of detailed data in this connection, however, it becomes necessary to discuss the fact that the siblings of the susceptible index cases have a larger average number of erupted permanent teeth and a smaller average number of deciduous teeth than the siblings of immune children. This difference between the two groups of siblings parallels the previously mentioned difference between the susceptible and immune children themselves. Data revealing this finding are shown in table 2, which gives tabulations of the average numbers of permanent and deciduous teeth in the siblings and their index cases. The differences between the two groups of index cases are readily apparent. For the sibling groups, if comparisons between the separate age-sex groups are restricted to those in which the averages are based on more than five children, it becomes evident that siblings of immunes definitely have fewer permanent teeth at comparable ages than do the siblings of the susceptibles.

TABLE 2.—*Average numbers of erupted permanent teeth and of deciduous teeth present in the mouths of (a) 306 immune (I) and susceptible (S) index cases and (b) of their 488 siblings. (Data derived from dental examinations of 4,416 grade school children, Hagerstown, Md.)*

Item tabulated	Sex and caries designation	Age (years last birthday)									
		6	7	8	9	10	11	12	13	14	15
		(a) Index cases									
Average number erupted permanent teeth per child.	Boys (S)	------	------	------	------	(1)	22.4	26.2	26.9	27.5	27.4
	Boys (I)	------	------	------	------	15.4	19.3	20.7	26.6	26.1	(1)
	Girls (S)	------	------	------	------	19.5	24.4	25.6	27.2	27.2	27.5
	Girls (I)	------	------	------	------	16.6	21.4	24.2	26.7	(1)	(1)
Average number of deciduous teeth present per child.	Boys (S)	------	------	------	------	(1)	1.9	.1	.3	.1	.3
	Boys (I)	------	------	------	------	7.7	4.3	3.4	.3	.3	(1)
	Girls (S)	------	------	------	------	3.4	.3	.5	0	.1	0
	Girls (I)	------	------	------	------	6.0	3.1	1.4	.2	(1)	(1)
		(b) Siblings									
Average number erupted permanent teeth per child.	Boys (S)	(1)	7.3	10.7	13.7	15.6	21.1	22.8	26.3	(1)	(1)
	Boys (I)	4.7	6.7	10.2	12.3	14.4	19.2	23.3	25.2	27.6	(1)
	Girls (S)	6.6	10.6	11.8	16.5	18.0	21.8	25.9	26.5	(1)	(1)
	Girls (I)	4.9	8.7	11.3	13.1	16.5	22.6	23.8	25.1	27.5	(1)
Average number of deciduous teeth present per child.	Boys (S)	(1)	15.5	11.3	8.4	6.7	2.6	1.3	.1	(1)	(1)
	Boys (I)	17.4	14.8	12.4	9.7	7.9	3.9	1.6	1.2	.1	(1)
	Girls (S)	16.0	10.0	11.1	4.3	3.9	1.8	.6	.2	(1)	(1)
	Girls (I)	15.6	13.0	11.3	9.5	6.2	2.3	1.7	1.1	0	(1)

[1] Values based on less than 5 cases are omitted.

Further analysis of these data, although not reproduced here, leads to the significant conclusion that siblings of immunes have, during the period of eruption of the permanent dentition, an average of approximately one erupted permanent tooth less than is found in siblings of the susceptibles of comparable age. The finding of lesser numbers of deciduous teeth in susceptible siblings and greater numbers in the siblings of immunes may be explained, in part, by these differences in eruption of the permanent teeth.[7]

In the light of these findings, the question may well be raised as to what analytical procedures are required for making a precise comparison of the caries experience of the two sibling groups. For example, the question may be raised that, since larger numbers of teeth are present in the siblings of susceptibles, more teeth would be available for attack by caries and, because of this fact, more teeth might be found carious. Moreover, a larger number of erupted permanent teeth implies earlier eruption and, therefore, a longer period of mouth exposure during which attack by caries might take place.

This discussion leads directly to an attempt to express the caries experience in terms of the length of time the teeth are exposed to the risk of attack by caries. In a recent paper (*8*), a method for estimating the time of exposure of teeth in the mouth, *accumulated posteruptive tooth age*, is described. The following simple example will serve, for present purposes, to define the term "accumulated posteruptive tooth age": A child of exactly 7 years of age has five permanent teeth erupted. The two lower first molars erupted at 6 years of age, the two upper first molars at 6½ years, and the lower right central incisor erupted at 6 years, 9 months of age. The accumulated posteruptive tooth age for this child is, therefore, 3¼ years; 2 years for the lower molars, 1 year for the upper molars and one-fourth year for the incisor. This determination of length of time the teeth are exposed in the mouth is the sum of actually observed durations of exposure of the separate teeth. On the other hand, it is possible to estimate the average value of accumulated post-eruptive tooth age from quantitative data on the eruption of the teeth. Details of the mathematical derivation of these values are given in reference (*8*), in which is included a nomogram from which it is possible to obtain estimated average values of post-eruptive tooth ages for given average numbers of erupted permanent teeth.

[7] The finding of these differences in numbers of permanent and deciduous teeth in the sibling groups, which parallel similar differences between the two groups of index cases strongly suggests the existence of familial resemblances in eruption patterns.

Caries experience in the permanent teeth of the siblings.—Table 3 gives data from which it is considered possible to make an accurate and precise comparison of dental caries experience in the permanent teeth of the *siblings* of the immune and those of the susceptible index cases. The first section of the table shows the number of decayed, missing, and filled (DMF) permanent teeth per child; the second section shows the number of DMF tooth surfaces per child, and the third section gives the average accumulated post-eruptive tooth ages per child for each age-sex group of the siblings. Inspection of the upper two divisions of the table indicates that the siblings of the susceptibles generally have of the order of twice as many teeth and tooth surfaces attacked by caries as the siblings of the immunes.

TABLE 3.—*Number of decayed, missing, or filled (DMF) permanent teeth and tooth surfaces per child, and accumulated post-eruptive tooth age per child, of siblings of immunes (I) and siblings of susceptibles (S). (Data derived from dental examinations of 4,416 grade school children, Hagerstown, Md.)*

Item tabulated	Sex and caries designation	Age (years last birthday)									
		6	7	8	9	10	11	12	13	14	15
Number of DMF permanent teeth per child.	Boys (S)	[1]	1.0	1.9	3.0	3.0	3.9	6.0	5.0	[1]	[1]
	Boys (I)	0	.6	.6	1.0	1.4	1.6	2.7	3.0	2.6	[1]
	Girls (S)	.9	1.6	1.0	2.9	3.1	4.2	4.3	8.2	[1]	[1]
	Girls (I)	.3	.4	.9	1.0	1.5	1.6	2.3	2.9	4.5	[1]
Number of DMF permanent tooth surfaces per child.	Boys (S)	[1]	2.1	2.5	5.5	6.7	7.0	13.3	13.6	[1]	[1]
	Boys (I)	0	.7	.8	1.4	2.9	3.4	4.5	7.6	4.0	[1]
	Girls (S)	1.6	1.6	1.3	7.4	5.7	8.0	8.8	15.6	[1]	[1]
	Girls (I)	.6	.4	1.2	1.8	2.7	1.9	4.7	5.5	9.7	[1]
Accumulated post-eruptive tooth age in years per child [2].	Boys (S)	[1]	6.5	15.7	30.0	39.5	66.7	77.0	107.0	[1]	[1]
	Boys (I)	3.3	5.5	14.0	23.0	33.5	57.0	80.0	94.0	------	[1]
	Girls (S)	6.0	15.0	19.5	40.7	47.5	67.0	104.0	113.5	[1]	[1]
	Girls (I)	3.7	9.5	17.3	25.5	40.5	72.5	82.0	95.5	------	[1]

[1] Values based on less than 5 cases are omitted.

[2] The values of accumulated post-eruptive tooth age given here were derived from a nomogram (fig. 3 in reference 8) relating average number of erupted permanent teeth to tooth age. The average numbers of erupted permanent teeth for the different age-sex groups of siblings are given in table 2 of this paper. Since the relationship between the number of erupted teeth and post-eruptive tooth age cannot be considered as accurately determined when the number of erupted teeth approaches 28, the table does not give tooth age values for average numbers of erupted teeth above 27.

Although several different methods may be adopted for showing, in the two groups of siblings, the relation of post-eruptive tooth age to attack by caries, the purposes of the present discussion are best served by the graphic presentation of data as presented in figure 1. Despite some irregular variation of the individual curves, the heavy solid lines, representing moving averages[8] for the combined sexes, show very clearly a striking difference between the siblings of the susceptible and those of the immunes. When post-eruptive tooth age reaches an average of 10 years per child, the siblings of the immunes have slightly less than one carious surface per child, while

[8] Each point of the moving average represents the mean of six values, three for boys and three for girls. These six values were obtained by reading the ordinates from the irregular line curves for successive 10-year units of accumulated post-eruptive tooth age.

siblings of the susceptibles have nearly two carious surfaces per child; when tooth age reaches 50 years, siblings of immunes show less than three DMF surfaces, while siblings of the susceptibles show more than seven DMF surfaces. Ratios for the two sibling groups of the number of tooth surfaces attacked by caries for different comparable levels of tooth age indicate, further, that from two and one-fourth to

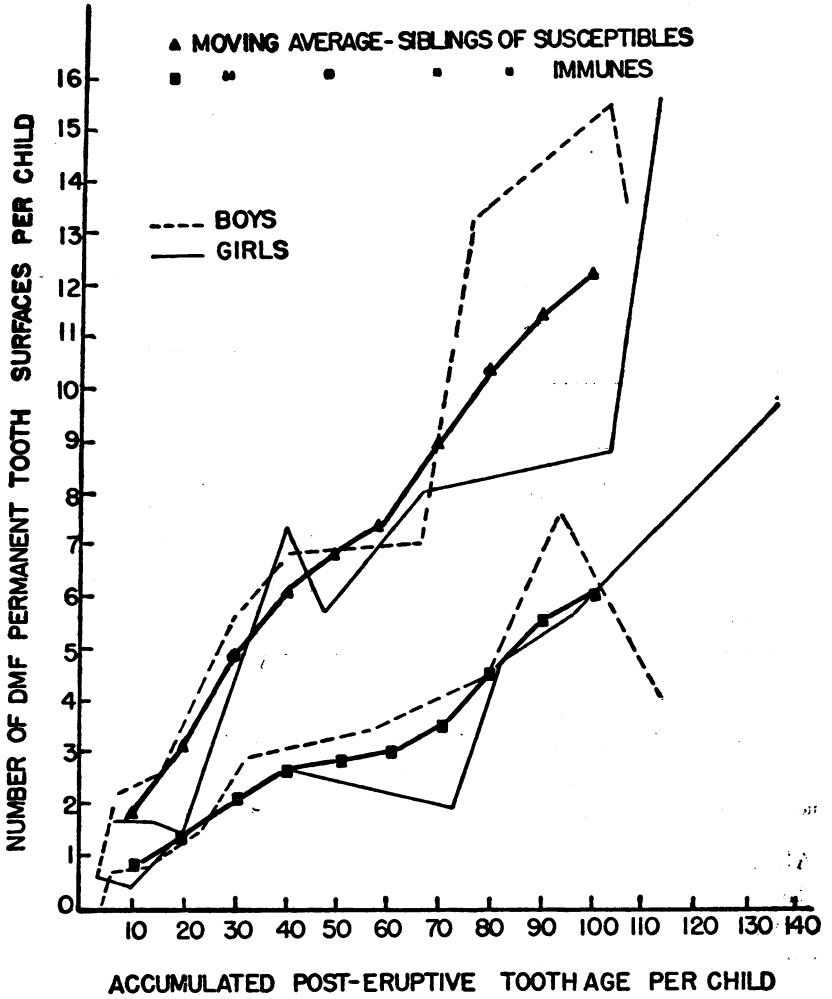

FIGURE 1.—The relation between accumulated post-eruptive tooth age and the number of decayed, missing, or filled (DMF) permanent tooth surfaces.

two and one-half times as much caries is present in the permanent teeth of siblings of the susceptibles as is found in siblings of the immunes.

Caries in the deciduous teeth of the siblings.—For reasons fully discussed in a previous publication (*6*) and mentioned earlier in this paper, caries experience cannot be completely reconstructed for the

deciduous teeth from survey data such as are available for the present study. An *approximation* of the level of caries in deciduous teeth may be obtained, however, by expressing the number of teeth or tooth surfaces observed to be carious, and (or) filled, as a ratio (times 100) of the total number of deciduous teeth present in the mouth.[9] Data arranged in this form are given in table 4 for the two sibling groups, and figure 2 illustrates graphically the relation between chronological age and the number of carious deciduous tooth surfaces per 100 deciduous teeth present in the mouth. In general, this material shows that caries in the deciduous teeth of siblings of susceptibles is approximately twice as extensive as in the siblings of the immunes.

TABLE 4.—*Data showing caries experience in the deciduous teeth of 488 siblings of immune (I) and susceptible (S) index cases. (Data derived from dental examinations of 4,416 grade school children, Hagerstown, Md.)*

Item tabulated	Sex and caries designation	Age (years last birthday)									
		6	7	8	9	10	11	12	13	14	15
Number of deciduous teeth present.	Boys (S)	[1]	170	170	84	114	39	18	1	[1]	[1]
	Boys (I)	278	251	311	185	166	62	32	18	1	[1]
	Girls (S)	128	50	89	60	58	11	5	3	[1]	[1]
	Girls (I)	109	169	317	229	105	18	31	16	0	[1]
Number of carious [2] teeth per 100 teeth present.	Boys (S)	[1]	43.5	61.2	57.1	51.8	64.1	55.6	100.0	------	------
	Boys (I)	11.2	29.1	36.0	37.8	34.3	41.9	43.8	33.3	100.0	------
	Girls (S)	55.5	76.0	48.3	63.3	55.2	90.9	60.0	100.0	------	------
	Girls (I)	30.3	18.9	34.7	32.8	35.2	16.7	48.4	37.5	------	------
Number of carious [2] tooth surfaces per 100 teeth present.	Boys (S)	[1]	89.4	154.1	122.6	128.1	182.1	144.4	500.0	------	------
	Boys (I)	20.1	57.8	69.5	91.4	66.9	72.6	84.4	61.1	200.0	------
	Girls (S)	126.6	238.0	100.0	133.3	124.1	154.6	100.0	200.0	------	------
	Girls (I)	45.0	36.7	70.7	65.9	69.5	22.2	119.4	100.0	------	------

[1] Values based on less than 5 cases are omitted.
[2] Decayed or filled.

Obviously this finding, and its interpretation, is not entirely conclusive, since the two sibling groups are markedly different in numbers of deciduous teeth present in the mouth, and, very probably, the length of time the deciduous teeth have been present in the mouth may be different for the two contrasted groups of children. From one point of view it is possible that the finding in the siblings of susceptibles of approximately double the amount of caries found in those of the immunes may constitute an *understatement* of the actual differences between the two groups. Thus it may be recognized that the finding of fewer deciduous teeth present in the mouths of the siblings of the susceptibles may represent, in part, the premature loss of these teeth because of severe caries. On the whole, therefore, it seems reasonable to conclude that the analysis of the data indicates a definite difference between the two groups of siblings in attack of the deciduous

[9] Such an arrangement of the data tends also to equalize differences, such as are present in the two sibling groups under investigation here, in the number of deciduous teeth present in the mouth.

teeth by caries and that this difference is of the order of two to one, in siblings of susceptibles as compared with siblings of immunes.

SUMMARY AND CONCLUSIONS

This paper contains the preliminary results of a study on familial characteristics of dental caries. The basic data were derived from records of dental examinations of essentially all of the elementary school children in an urban community (Hagerstown, Md.), which has a population of approximately 30,000 persons The major steps in the analysis are as follows: From the dental records of 4,416 white

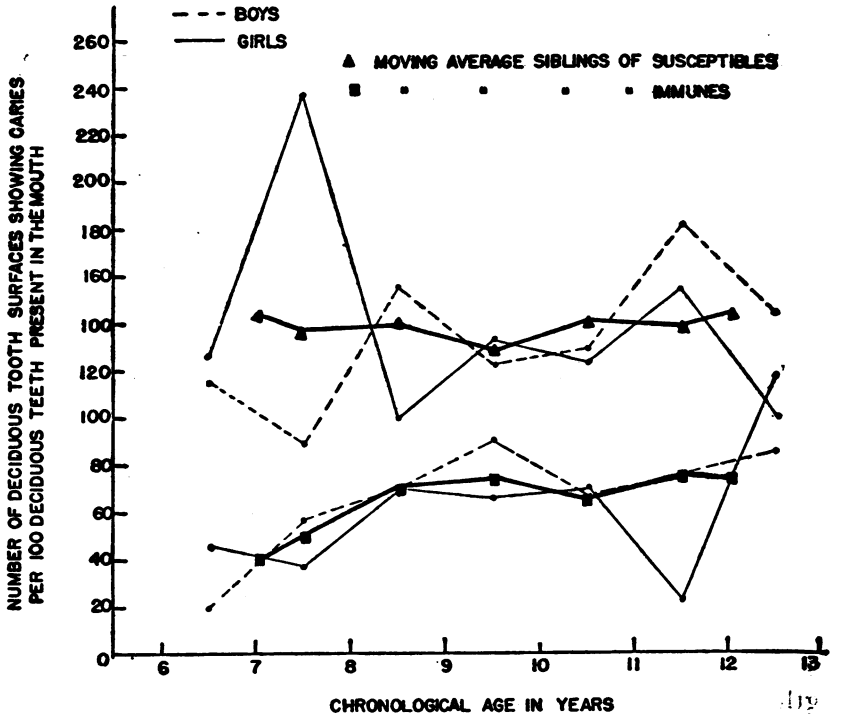

FIGURE 2.—The relation between chronological age and number of carious or filled deciduous tooth surfaces per 100 deciduous teeth present in the mouth. (The heavy solid lines represent 3-point moving averages, except that the points at the two ends of the age range represent 2-point averages.)

children, 2 defined groups were selected—one, those being relatively immune to caries, the other, those showing relatively high susceptibility to caries. Records for the brothers and sisters, of grade school age, of the "immunes" and "susceptibles" were then assembled and analyzed to show the level of caries in the two contrasted groups of siblings. The results of the analysis indicate that siblings of susceptibles have somewhat over twice as much caries in both the permanent and deciduous teeth as do siblings of the immunes. Since the material available for study constitutes a relatively large sample of children, it

is possible to conclude that the existence of familial resemblances in caries experience of siblings is definitely established. In this paper, and at the present time, no specific explanation is offered for the observed familial differences.

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UNSUCCESSFUL TREATMENT OF MALARIA WITH SULFONAMIDE COMPOUNDS[1]

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The publication of several favorable reports on the treatment of malaria with "prontosil" stimulated the trial of an accepted brand of sulfanilamide in this disease at the United States Marine Hospital in San Francisco. A translation of an article by Dr. Diaz de Leon, published in the Public Health Reports (*1*) indicated Dr. de Leon's complete satisfaction with "rubiazol" (the original "prontosil") in the treatment of 15 cases of tertian malaria. Van der Wielen (*2*) had previously reported the value of "prontosil" as a curative agent in two patients with (inoculated, therapeutic) quartan malaria. Hill and Goodwin (*3*), in a more extensive study, found complete success with "prontosil" in the treatment of 93 cases of estivo-autumnal and 7 cases of tertian malaria.

[1] Abstract of an article, by the same authors, entitled "The Unreliability of Sulfanilamide Compounds in the Treatment of Malaria," which appeared in the Hospital News for July 15, 1938.—Ed.

It was felt that if uniformly good results were obtainable with "prontosil," sulfanilamide orally should produce an equally favorable response. This opinion was based on the experimental work (*4*, *5*, *6*, *7*) which had demonstrated that sulfanilamide is the active principle of "prontosil." This belief has been accepted by the Council of Pharmacy and Chemistry (*8*). We were therefore surprised when the administration of sulfanilamide failed to cure three cases of malarial fever. Our complete disagreement with the above suggested that "prontosil" might be efficacious in this disease where sulfanilamide had failed. This led us to the treatment of a case of therapeutic quartan malaria with "prontosil" according to the method used by Hill and Goodwin; but this drug likewise failed in the therapy of malaria.

It must be noted here that all of our patients were hospitalized during their entire illness, which afforded the benefits of carefully controlled clinical and laboratory supervision. This aided greatly in the detection of the nonspecificity of the sulfonamide compounds in malaria.

SUMMARY OF CASE REPORTS

Case No. 1.—It was observed that sulfanilamide aborted the symptoms of one case of tertian malaria and caused the apparent disappearance of *Plasmodium vivax* from the peripheral circulation. This patient received sulfanilamide medication for 12 days before being discharged from the hospital as recovered. He weighed 118 pounds and was given 15 grains of sulfanilamide 4 times a day for the first 6 days and thereafter 10 grains 3 times a day until his discharge. He experienced a relapse, confirmed by the presence of numerous *P. vivax* in the blood smears, 12 days after leaving the hospital.

In the following cases of estivo-autumnal and tertian malaria respectively, it was evident that the plasmodia were surviving intensive sulfanilamide therapy for too long a period for the safety of the patient or the mental comfort of his physicians.

Case No. 2.—The patient in this case had estivo-autumnal malaria. He weighed 150 pounds and was given 15 grains of sulfanilamide 5 times a day for 3 days. The fever declined gradually with daily peaks at 40° C., 38.4° C., and 38° C., but *P. falciparum* were still plentiful in the blood smears at the end of that period. Quinine was then substituted for the sulfanilamide. It may be argued that the sulfanilamide therapy was not given a fair trial in this case. The patient had a malignant type of malaria, his general condition was poor, and his urinalysis showed albumin, casts, leucocytes, and red blood corpuscles. To persist longer with sulfanilamide and withhold quinine seemed dangerous.

Case No. 3.—In this second case of tertian malaria, it was felt that sulfanilamide was given a fair chance to prove its worth. This patient, who weighed 140 pounds, was given 15 grains of sulfanilamide every 4 hours for 4 days. He had his expected paroxysm of chills and fever on the evening of the first day of treatment and a recurrence of chills on the third day, with but little rise of temperature. *Plasmodium vivax* was still present in the blood smears at the end of 4 days, although he had received a total of 315 grains of sulfanilamide by that time. The change to quinine was promptly effective.

Case No. 4.—The "prontosil" treatment was tried in a patient undergoing quartan malarial therapy for cerebrospinal syphilis. When it was decided to

check his bouts of fever, 10 cc of "prontosil," 2.5 percent solution, were injected intramuscularly every 12 hours, as recommended by Hill and Goodwin. The first injection was administered during the height of the eleventh febrile attack. After he had received 50 cc of "prontosil" solution, *P. malariae* were still present in the blood smears in almost unchanged numbers, except that there appeared to be an unduly large number of gametocytes. It was then evident that the patient would have another paroxysm. In order to give the "prontosil" therapy every possible advantage, an extra 20 cc dose was injected intramuscularly in anticipation of this attack. It was felt that this would allow an adequate concentration of the drug in the blood stream at the time of liberation of the merozoites. The expected paroxysm of chills and fever occurred in unabated severity about an hour after the injection of this "prontosil." Since *P. malariae* could still be easily demonstrated in daily blood smears during the next 3 days, the "prontosil" was abandoned as useless. After quinine therapy was instituted, there were no further paroxysms of fever, but because of the gametocytes, a course of plasmochin was also given.

We believe the publication of our experience to be warranted as a check to the enthusiasm expressed in previous papers on the subject. It is realized that four cases constitute but a small group; but it is to be noted that consistent negative therapeutic results in only a few cases that are carefully controlled are sufficient to bring into question the reliability of favorable reports of a much larger series of cases observed under less controlled conditions. While it appears that sulfonamide compounds have a deterrent effect on the life cycle of the malarial plasmodium, their action does not seem sufficient for clinical cure in most cases. The effects on gametocytes are unknown, and the results may be confined to the production of a latency. This might prove dangerous not only to the patient's future health but to the public welfare of his community as well.

It is concluded that sulfanilamide and "prontosil" possess no practical chemotherapeutic value in malaria, and that they cannot be regarded as satisfactory or safe substitutes for quinine.

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DEATHS DURING WEEK ENDED JULY 16, 1938

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

	Week ended July 16, 1938	Corresponding week, 1937
Data from 88 large cities of the United States:		
Total deaths	7,753	[1] 8,933
Average for 3 prior years	7,952	
Total deaths, first 28 weeks of year	236,944	258,089
Deaths under 1 year of age	502	[1] 584
Average for 3 prior years	541	
Deaths under 1 year of age, first 28 weeks of year	14,865	16,161
Data from industrial insurance companies:		
Policies in force	69,130,363	70,073,939
Number of death claims	11,499	12,283
Death claims per 1,000 policies in force, annual rate	8.7	9.1
Death claims per 1,000 policies, first 28 weeks of year, annual rate	9.6	10.5

[1] Data for 86 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.

In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (------) represent no report, with the implication that cases or deaths may have occurred but were not reported to the State health officer.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 23, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median
NEW ENG.												
Maine	24	4	1	1	30	5	------	------	73	12	14	14
New Hampshire	0	0	0	0	------	------	1	------	41	4	3	3
Vermont	0	0	1	0	------	------	------	------	313	23	7	7
Massachusetts	1.2	1	9	9	------	------	------	------	197	167	85	126
Rhode Island	0	0	1	1	------	------	------	------	31	4	19	16
Connecticut	6	2	10	7	3	1	1	1	42	14	17	41
MID. ATL.												
New York	4	10	21	21	[1] 1.4	[1] 2	[1] 6	[1] 3	271	673	325	367
New Jersey [2]	10	8	3	10	2	2	------	------	78	65	183	183
Pennsylvania	7	13	23	28	------	------	------	------	141	275	787	553
E. NO. CEN.												
Ohio	6	8	6	13	------	------	4	3	45	58	127	173
Indiana [2]	15	10	2	8	------	------	8	10	24	16	56	24
Illinois [2]	12	18	28	17	5	7	6	8	38	58	167	167
Michigan [3]	11	10	14	11	------	------	------	------	355	329	115	77
Wisconsin	4	2	5	4	23	13	1	14	684	384	44	72
W. NO. CEN.												
Minnesota	16	8	1	5	2	1	------	------	155	79	8	25
Iowa [2]	2	1	7	6	------	------	1	------	84	41	7	7
Missouri	7	5	5	16	1.3	1	22	11	10	8	42	35
North Dakota	15	2	0	0	------	------	5	------	207	28	------	13
South Dakota	15	2	0	1	------	------	------	------	------	------	1	1
Nebraska	0	0	1	4	------	------	------	------	46	12	7	7
Kansas	6	2	4	4	8	3	3	2	48	17	8	8
SO. ATL.												
Delaware	0	0	0	0	------	------	------	------	100	5	2	2
Maryland [2 3 4]	16	5	5	5	19	6	1	2	78	25	13	33
Dist. of Col. [2]	50	6	8	3	------	------	------	1	58	7	20	12
Virginia [2]	15	8	8	8	------	------	------	------	114	59	54	37
West Virginia	6	2	2	9	25	9	12	4	31	11	35	17
North Carolina [2 4]	25	17	15	11	------	------	------	------	172	115	75	75
South Carolina [4]	25	9	0	3	189	68	38	44	36	13	19	19
Georgia [4]	30	18	7	7	------	------	------	------	------	------	------	------
Florida [4]	16	5	2	6	------	------	1	------	44	14	------	7

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 23, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median
E. SO. CEN.												
Kentucky	12	7	2	3	5	3	3	------	95	53	58	50
Tennessee [3]	9	5	5	5	25	14	1	8	59	33	99	47
Alabama [4]	20	11	8	13	34	19	9	4	9	5	6	17
Mississippi [3]	39	15	8	8	------	------	------	------	------	------	------	------
W. SO. CEN.												
Arkansas	15	6	3	3	31	12	3	2	56	22	6	4
Louisiana	59	24	7	11	15	6	6	8	10	4	2	5
Oklahoma	6	3	5	4	49	24	5	6	18	9	17	7
Texas [4]	19	23	28	28	63	74	39	39	17	20	104	104
MOUNTAIN												
Montana	0	0	1	1	------	------	------	------	319	33	4	4
Idaho	11	1	0	0	21	2	6	------	95	9	12	3
Wyoming	22	1	0	0	------	------	------	------	22	1	5	6
Colorado [3]	19	4	2	3	------	------	------	------	117	24	20	20
New Mexico	0	0	2	1	------	------	------	1	99	8	100	9
Arizona	38	3	1	1	139	11	10	------	481	38	7	7
Utah [2 3]	0	0	0	0	------	------	1	------	553	55	43	23
PACIFIC												
Washington	3	1	0	0	------	------	------	------	35	11	24	36
Oregon	15	3	2	2	36	7	13	10	41	8	7	14
California [3]	15	18	18	29	19	23	4	12	235	277	47	177
Total	12	301	281	342	16	313	210	238	128	3,126	2,801	2,801
29 weeks	18	13,097	12,525	16,615	77	44,716	273,534	140,981	1,067	754,176	235,831	337,055

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median
NEW ENG.												
Maine	0	0	0	0	0	0	4	0	30	5	5	5
New Hampshire	0	0	0	0	0	0	1	0	0	0	1	1
Vermont	0	0	0	0	0	0	0	0	41	3	------	3
Massachusetts	0	0	2	2	1.2	1	10	1	72	61	38	54
Rhode Island	0	0	0	0	0	0	0	0	69	9	8	7
Connecticut	3	1	0	0	3	1	0	1	36	12	7	7
MID. ATL.												
New York	1.6	4	5	4	0.8	2	8	11	34	84	66	136
New Jersey [3]	1.2	1	0	0	0	0	1	1	12	10	24	38
Pennsylvania	2	4	2	2	0.5	1	0	1	39	77	114	126
E. NO. CEN.												
Ohio	0	0	6	2	0	0	20	4	30	39	34	74
Indiana [3]	1.5	1	0	1	1.5	1	7	1	21	14	29	22
Illinois [3]	1.3	2	0	8	0.7	1	11	4	58	87	102	102
Michigan [3]	0	0	1	1	6	6	4	0	87	81	130	86
Wisconsin	0	0	2	0	0	0	2	0	77	43	56	63

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 23, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median
W. NO. CEN.												
Minnesota	0	0	0	0	0	0	1	0	57	29	21	27
Iowa [2]	0	0	1	1	2	1	3	0	27	13	31	19
Missouri	0	0	1	1	0	0	16	0	14	11	38	21
North Dakota	0	0	0	0	0	0	0	0	66	9	1	5
South Dakota	0	0	0	0	15	2	0	0	23	3	11	4
Nebraska	0	0	1	0	4	1	4	0	15	4	2	10
Kansas	6	2	0	1	0	0	4	1	59	21	17	17
SO. ATL.												
Delaware	0	0	0	0	0	0	0	0	20	1	1	------
Maryland [2 3 4]	6	2	2	2	0	0	1	0	9	3	8	13
Dist. of Col. [2]	0	0	0	1	0	0	0	0	8	1	3	3
Virginia [2]	1.9	1	6	2	4	2	1	1	21	11	7	17
West Virginia	6	2	1	1	0	0	1	1	31	11	11	11
North Carolina [2 4]	1.5	1	4	3	0	0	9	2	13	9	8	11
South Carolina [4]	6	2	1	0	0	0	1	0	6	2	10	2
Georgia [4]	1.7	1	1	1	3	2	3	1	17	10	5	4
Florida [4]	0	0	0	0	3	1	0	0	28	9	1	1
E. SO. CEN.												
Kentucky	5	3	2	2	1.8	1	18	2	23	13	14	12
Tennessee [2]	1.8	1	2	2	4	2	10	9	16	9	12	11
Alabama [4]	5	3	2	2	1.8	1	1	1	14	8	2	9
Mississippi [3]	0	0	0	0	8	3	13	1	15	6	7	7
W. SO. CEN.												
Arkansas	0	0	0	0	0	0	48	1	5	2	7	1
Louisiana	7	3	1	1	7	3	7	1	12	5	3	4
Oklahoma	0	0	1	1	0	0	53	0	25	12	18	6
Texas [4]	1.7	2	0	0	1.7	2	31	1	19	23	37	31
MOUNTAIN												
Montana	0	0	1	0	0	0	1	1	39	4	5	4
Idaho	11	1	1	1	0	0	0	0	32	3	8	2
Wyoming	0	0	0	0	0	0	1	0	22	1	3	5
Colorado [2]	15	3	1	1	0	0	3	0	112	23	5	9
New Mexico	0	0	0	0	12	1	2	0	62	5	9	6
Arizona	0	0	0	0	0	0	2	1	38	3	1	3
Utah [2 3]	0	0	0	0	0	0	0	0	90	9	7	7
PACIFIC												
Washington	9	3	0	0	0	0	0	1	50	16	8	11
Oregon	0	0	1	0	0	0	1	1	61	12	10	10
California	1.7	2	2	2	7	8	21	21	62	73	57	73
Total	1.8	45	50	50	1.7	43	324	227	36	899	1,002	1,131
29 weeks	2.8	2,008	3,921	3,860	0.9	668	1,670	1,599	185	133,844	161,216	161,216

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough	
	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases
NEW ENGLAND										
Maine	0	0	0	0	12	2	0	2	189	31
New Hampshire	0	0	0	0	10	1	1	1	------	------
Vermont	0	0	0	0	0	0	0	0	272	20
Massachusetts	0	0	0	0	2	2	6	6	105	89
Rhode Island	0	0	0	0	8	1	0	0	46	6
Connecticut	0	0	0	0	3	1	3	2	219	73

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 23, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough	
	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases	July 24, 1937, cases	1933–1937 median	July 23, 1938, rate	July 23, 1938, cases
MIDDLE ATLANTIC										
New York	0	0	0	0	5	12	9	13	251	624
New Jersey [1]	0	0	0	0	16	13	5	5	396	330
Pennsylvania	0	0	0	0	9	17	16	16	168	327
EAST NORTH CENTRAL										
Ohio	0	0	1	1	5	6	18	18	72	93
Indiana [2]	15	10	7	0	17	11	4	9	12	8
Illinois [2]	7	10	10	2	17	25	22	22	330	499
Michigan [3]	1.1	1	7	0	2	2	2	4	528	489
Wisconsin	5	3	2	6	0	0	1	2	597	335
WEST NORTH CENTRAL										
Minnesota	18	9	10	0	0	0	1	1	149	76
Iowa [2]	27	13	52	6	14	7	3	1	31	15
Missouri	14	11	0	0	27	21	21	21	43	33
North Dakota	30	4	6	0	0	0	5	1	214	29
South Dakota	8	1	1	1	0	0	0	1	158	21
Nebraska	4	1	0	2	0	0	0	0	80	21
Kansas	11	4	14	1	25	9	30	13	322	115
SOUTH ATLANTIC										
Delaware	0	0	0	0	0	0	1	2	120	6
Maryland [2] [4]	0	0	0	0	22	7	16	16	124	40
Dist. of Col. [3]	0	0	0	0	25	3	2	1	108	13
Virginia [2]	0	0	0	0	35	18	28	38	146	76
West Virginia	0	0	1	0	42	15	8	16	67	24
North Carolina [2] [4]	0	0	1	1	42	28	25	25	499	334
South Carolina [4]	0	0	0	0	64	23	11	26	289	104
Georgia [4]	0	0	0	0	88	52	43	43	88	52
Florida [4]	0	0	0	0	16	5	6	3	59	19
EAST SOUTH CENTRAL										
Kentucky	12	7	0	0	73	41	47	41	82	46
Tennessee [2]	1.8	1	0	0	70	39	53	53	87	48
Alabama [4]	0	0	0	0	27	15	12	16	88	49
Mississippi [3]	0	0	1	0	31	12	23	17	-------	-------
WEST SOUTH CENTRAL										
Arkansas	5	2	0	0	66	26	50	25	38	15
Louisiana	0	0	0	0	42	17	33	31	105	43
Oklahoma	0	0	0	0	49	24	30	30	39	19
Texas [4]	3	3	0	1	53	63	85	61	132	157
MOUNTAIN										
Montana	39	4	2	2	0	0	1	2	522	54
Idaho	85	8	17	2	11	1	0	0	63	6
Wyoming	22	1	0	0	0	0	1	0	155	7
Colorado [2]	5	1	1	0	49	10	1	3	234	48
New Mexico	12	1	0	0	37	3	2	8	161	13
Arizona	13	1	0	0	25	2	4	4	-------	-------
Utah [2] [3]	0	0	0	0	70	7	0	1	502	50
PACIFIC										
Washington	31	10	0	5	6	2	2	3	230	73
Oregon	10	2	2	2	0	0	4	3	142	28
California	13	15	1	1	5	6	12	8	203	240
Total	5	123	136	78	22	549	647	672	197	4,798
29 weeks	17	12,393	7,693	5,166	8	5,818	5,486	6,295	178	125,842

[1] New York City only.

[2] Rocky Mountain spotted fever, week ended July 23, 1938, 25 cases, as follows: New Jersey, 1; Indiana, 1; Illinois, 2; Iowa, 1; Maryland, 3; District of Columbia, 4; Virginia, 7; North Carolina, 2; Tennessee, 1; Colorado, 1; Utah, 2.

[3] Period ended earlier than Saturday.

[4] Typhus fever, week ended July 23, 1938, 47 cases, as follows: Maryland, 2; North Carolina, 2; South Carolina, 1; Georgia, 26; Florida, 3; Alabama, 7; Texas, 6.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Meningitis, meningococcus	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
June 1938										
Alabama	16	22	36	564	458	124	13	23	11	40
Georgia	0	17	33	195	467	119	10	30	2	155
Illinois	9	126	48	14	2,585	----	9	997	56	29
Maine	0	3	4	----	357	----	1	53	0	7
Mississippi	1	13	801	5,194	694	864	11	6	13	36
New Mexico	0	14	6	2	176	1	0	40	17	17
New York	30	92	----	14	13,583	----	9	1,798	0	28
Ohio	8	55	44	3	2,763	----	5	509	12	30
Rhode Island	2	3	----	----	37	----	0	102	0	1
Tennessee	10	13	44	148	324	27	4	61	2	70

June 1938

	Cases
Chickenpox:	
Alabama	59
Georgia	62
Illinois	1,289
Maine	217
Mississippi	285
New Mexico	34
New York	2,758
Ohio	851
Rhode Island	53
Tennessee	63
Conjunctivitis (acute infectious):	
Georgia	1
Dengue:	
Alabama	2
Georgia	4
Mississippi	10
Diarrhea and enteritis:	
Ohio (under 2 years)	254
Dysentery:	
Alabama (amoebic)	2
Georgia (amoebic)	10
Georgia (bacillary)	109
Illinois (amoebic)	11
Illinois (bacillary)	12
Illinois (amoebic carriers)	13
Mississippi (amoebic)	180
Mississippi (bacillary)	2,525
New Mexico (amoebic)	1
New Mexico (bacillary)	2
New Mexico (unspecified)	4
New York (amoebic)	3
New York (bacillary)	104
Ohio (bacillary)	4
Rhode Island (bacillary)	1
Tennessee (amoebic)	4
Tennessee (bacillary)	222
Encephalitis, epidemic or lethargic:	
Alabama	4
Illinois	8
New York	14
Ohio	3
German measles:	
Alabama	7
Illinois	48
Maine	14
New Mexico	3
New York	213
Ohio	31

	Cases
German measles—Con.	
Rhode Island	7
Tennessee	2
Hookworm disease:	
Georgia	617
Mississippi	564
Impetigo contagiosa:	
Illinois	12
Tennessee	2
Lead poisoning:	
Ohio	7
Mumps:	
Alabama	50
Georgia	82
Illinois	690
Maine	125
Mississippi	150
New Mexico	16
Ohio	642
Rhode Island	9
Tennessee	53
Ophthalmia neonatorum:	
Alabama	3
Illinois	1
New York	[1] 8
Ohio	73
Tennessee	2
Paratyphoid fever:	
Illinois	2
New Mexico	2
New York	9
Ohio	2
Tennessee	2
Puerperal septicemia:	
Mississippi	20
New Mexico	4
Ohio	4
Rabies (in man):	
Mississippi	1
Rabies (in animals):	
Alabama	68
Illinois	46
Maine	1
Mississippi	18
New Mexico	1
New York	[1] 3
Rhode Island	8
Rocky Mountain Spotted fever:	
Ohio	1
Septic sore throat:	
Georgia	16
Illinois	7
Maine	1

	Cases
Septic sore throat—Con.	
New Mexico	5
New York	94
Ohio	87
Rhode Island	1
Tennessee	2
Tetanus:	
Alabama	6
Georgia	2
Illinois	6
Maine	1
New York	6
Tennessee	3
Trachoma:	
Mississippi	6
New Mexico	2
Tennessee	2
Trichinosis:	
Georgia	1
New York	7
Tularaemia:	
Georgia	6
Tennessee	2
Typhus fever:	
Alabama	23
Georgia	61
New York	2
Undulant fever:	
Alabama	5
Georgia	11
Illinois	17
Maine	4
Mississippi	4
New Mexico	2
New York	37
Ohio	2
Rhode Island	1
Tennessee	1
Vincent's infection:	
Illinois	2
Maine	4
New York	[1] 77
Tennessee	3
Whooping cough:	
Alabama	242
Georgia	263
Illinois	1,051
Maine	147
Mississippi	1,151
New Mexico	74
New York	2,283
Ohio	676
Rhode Island	102
Tennessee	247

[1] Exclusive of New York City.

PLAGUE INFECTION IN FLEAS FROM CHIPMUNKS IN SAN BERNARDINO COUNTY, CALIFORNIA

Under date of July 22, 1938, Dr. W. M. Dickie, Director of Public Health of California, reported that plague infection had been proved in a pool of 15 fleas from 13 chipmunks, shot at the Osito Girls Camp, 3 miles southwest of Pine Knot, San Bernardino County, California.

PLAGUE INFECTION IN FLEAS FROM PRAIRIE DOGS AND IN FLEAS AND TISSUE FROM GROUND SQUIRRELS IN WYOMING

Under date of July 19, 1938, Senior Surgeon C. R. Eskey reported that plague infection had been demonstrated in pools of fleas and in tissue from rodents in Wyoming as follows:

A pool of 18 fleas collected from 8 prairie dogs (*Cynomys leucurus*) shot June 27, 1938, 8 miles northwest of Evanston, Uinta County, Wyoming.

In animal tissue and in pools of fleas from *Citellus armatus*, all within 2 to 8 miles of Cokeville, Lincoln County, Wyoming, as follows:

Tissue from 1 *Citellus armatus* found dead July 6, 1938; a pool of 37 fleas from 15 *C. armatus* shot July 6; in a pool of 158 fleas from 47 *C. armatus* shot July 7; in tissue from 1 *C. armatus* shot July 8; in pooled tissue from 3 *C. armatus* shot July 8; in tissue from 1 *C. armatus* found dead July 8; in tissue from 1 *C. armatus* shot July 8; in a pool of 18 fleas from 1 *C. armatus* shot July 8; in a pool of 3 fleas from 1 *C. armatus* found dead July 8; in a pool of 290 fleas from 113 *C. armatus* shot July 8; in a pool of 9 fleas from 1 *C. armatus* picked up sick July 9; in a pool of 197 fleas from 75 *C. armatus* shot July 9; and in a pool of 98 fleas from 142 *C. armatus* shot July 11.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 16, 1938

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	119	33	15	1,452	330	500	7	382	70	1,324	-------
Current week [1]	94	32	16	1,074	273	344	10	323	36	1,869	-------
Maine:											
Portland	0	------	0	1	2	0	0	0	0	0	18
New Hampshire:											
Concord	0	------	0	0	0	0	0	0	0	0	13
Manchester	0	------	0	0	0	0	0	0	0	0	11
Nashua	0	------	0	0	0	0	0	0	0	0	5
Vermont:											
Barre	0	------	0	0	1	0	0	0	0	0	4
Burlington	0	------	0	0	0	0	0	0	0	4	11
Rutland	0	------	0	0	0	0	0	0	0	0	3
Massachusetts:											
Boston	2	------	0	74	9	29	0	5	0	20	184
Fall River	1	------	0	0	5	2	0	0	0	0	42
Springfield	0	------	0	22	0	0	0	2	0	7	28
Worcester	0	------	0	1	4	1	0	0	0	7	41
Rhode Island:											
Pawtucket	0	------	0	0	0	0	0	0	0	2	14
Providence	0	------	0	0	1	2	0	1	0	11	61

[1] Figures for Tacoma, Wash., estimated; report not received.

City reports for week ended July 16, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Connecticut:											
Bridgeport	0		0	0	1	0	0	0	0	1	25
Hartford	0		0	1	1	1	0	2	0	1	55
New Haven	0	1	1	3	4	1	0	0	1	7	33
New York:											
Buffalo	1		1	4	3	3	0	6	1	48	118
New York	23	3	3	310	50	41	0	54	5	303	1,269
Rochester	0		0	17	2	4	0	0	0	9	69
Syracuse	0		0	41	3	4	0	0	0	15	45
New Jersey:											
Camden	0		0	0	3	0	0	0	0	8	36
Newark	0		0	4	2	2	0	3	0	43	89
Trenton	0		0	0	1	1	0	3	0	7	29
Pennsylvania:											
Philadelphia	1	2	1	57	9	19	0	21	1	70	427
Pittsburgh	2		0	2	17	11	0	7	1	32	166
Reading	0		0	1	0	0	0	1	0	7	28
Scranton	0			1		0	0		0	0	
Ohio:											
Cincinnati	5		0	1	4	1	0	6	0	15	120
Cleveland	0		0	32	7	14	0	11	0	95	171
Columbus	0		0	3	0	2	0	3	0	1	76
Toledo	1		0	5	2	2	0	1	0	34	62
Indiana:											
Anderson	0		0	0	0	1	0	0	0	6	10
Fort Wayne	0		0	1	1	0	0	2	0	0	23
Indianapolis	0		0	4	5	5	0	6	1	6	102
South Bend	0		0	22	0	4	1	0	0	0	14
Terre Haute	0		0	0	0	3	1	0	0	0	21
Illinois:											
Alton	0		0	0	0	0	0	0	0	3	7
Chicago	7	3	2	27	22	41	0	37	0	296	650
Elgin	0		0	0	0	0	0	0	0	0	7
Moline	0		0	1	0	0	0	0	0	3	11
Springfield	0		0	0	0	0	4	0	1	6	31
Michigan:											
Detroit	5		1	16	7	28	0	8	0	226	216
Flint	0		0	17	3	5	0	0	0	13	45
Grand Rapids	0		0	39	2	7	0	0	0	1	29
Wisconsin:											
Kenosha	0		0	5	0	3	0	0	0	16	8
Madison	1		0	32	3	0	0	0	0	2	22
Milwaukee	0		0	14	4	13	0	7	0	125	94
Racine	0		0	2	0	2	0	0	0	16	15
Superior	0		0	1	0	1	0	0	0	5	12
Minnesota:											
Duluth	0		0	39	0	5	0	0	0	26	14
Minneapolis	0		0	27	0	3	1	2	0	9	83
St. Paul	1		0	12	2	2	0	1	0	23	48
Iowa:											
Davenport	0			0		0	0		0	0	
Des Moines	0		0	3	0	9	0	0	0	0	32
Sioux City	0			17		0	0		0	6	
Waterloo	0			1		1	0		0	2	
Missouri:											
Kansas City	1		0	0	4	3	0	3	0	2	107
St. Joseph	1		0	0	4	0	0	0	0	0	36
St. Louis	3		0	1	1	4	0	15	1	6	218
North Dakota:											
Fargo	0		0	3	0	1	0	0	0	1	5
Grand Forks	0			1		0	0		0	0	
Minot	0		0	4	0	0	0	0	0	0	3
South Dakota:											
Aberdeen	0			0		0	0		0	6	
Sioux Falls	0		0	0	0	0	0	0	0	0	12
Nebraska:											
Lincoln	0			8		2	0		0	11	
Omaha	0		0	5	6	0	0	2	0	1	70
Kansas:											
Lawrence	0		0	0	0	0	0	0	0	4	6
Topeka	0		0	1	0	0	0	0	0	24	16
Wichita	0		0	2	1	0	0	1	2	6	28

City reports for week ended July 16, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Delaware:											
Wilmington	1		0	0	2	0	0	3	0	5	25
Maryland:											
Baltimore	1	1	1	6	6	5	0	12	2	36	226
Cumberland	0		0	6	0	0	0	0	1	0	12
Frederick	0		0	0	0	0	0	0	0	0	7
Dist. of Col.:											
Washington	12		0	10	6	3	0	7	3	12	138
Virginia:											
Lynchburg	1		0	0	0	0	0	1	0	4	12
Norfolk	0		0	0	2	2	0	1	0	0	26
Richmond	1		1	38	1	2	0	2	0	3	58
Roanoke	0		0	0	0	0	0	0	0	0	13
West Virginia:											
Charleston	0		0	0	2	0	0	1	0	0	35
Huntington	0			0		0	0		0	0	
Wheeling	0		0	4	0	1	0	0	0	6	19
North Carolina:											
Gastonia	0			0		0	0		0	1	
Raleigh	0		0	0	1	0	0	3	0	13	21
Wilmington	0		0	0	1	0	0	0	0	10	13
Winston-Salem	1		0	35	1	0	0	3	0	4	17
South Carolina:											
Charleston	0	2		1	4	0	0	0	0	0	23
Florence	0		0	0	1	0	0	0	0	4	9
Greenville	0		0	7	4	0	0	0	0	1	37
Georgia:											
Atlanta	1	1	0	0	2	1	0	8	1	8	75
Brunswick	0		0	0	0	0	0	0	0	0	3
Savannah	0		0	0	0	0	0	3	0	6	26
Florida:											
Miami	2		0	0	0	0	0	1	0	2	27
Tampa	1		0	7	0	0	0	0	1	0	20
Kentucky:											
Ashland	0			0		0	0		0	0	
Covington	0	1	0	0	0	0	0	2	0	1	16
Lexington	0		0	0	2	0	0	1	0	7	3
Louisville	0		0	1	5	6	1	4	1	12	91
Tennessee:											
Knoxville	1		0	3	0	0	0	0	1	2	18
Memphis	0		0	0	6	0	0	3	2	18	109
Nashville	0	2	2	0	2	0	0	3	0	0	68
Alabama:											
Birmingham	1	1	0	3	2	0	0	3	3	0	63
Mobile	0		0	0	0	2	0	2	0	0	21
Montgomery	1			0		1	0		0	0	
Arkansas:											
Fort Smith	0			7		0	0		0	3	
Little Rock	0		0	0	3	0	0	1	0	0	8
Louisiana:											
Lake Charles	0		0	0	0	0	0	0	0	2	7
New Orleans	3	6	2	4	12	2	0	11	1	64	139
Shreveport	1		0	0	2	0	0	6	3	0	52
Oklahoma:											
Muskogee	0			4		0	0		1	0	
Oklahoma City	0		0	0	5	3	0	0	1	2	38
Tulsa	0			13		1	2		0	9	
Texas:											
Dallas	1		0	0	2	0	0	5	2	5	71
Fort Worth	0		0	0	4	3	2	1	0	3	34
Galveston	1		0	0	0	2	0	0	0	0	14
Houston	2		0	0	2	5	0	2	0	0	77
San Antonio	0	1	1	0	4	1	0	6	2	0	75
Montana:											
Billings	0		0	0	1	1	0	0	0	11	5
Great Falls	0		0	0	0	1	0	1	0	5	13
Helena	0		0	0	0	1	0	0	0	2	5
Missoula	0		0	0	0	0	1	0	0	0	13
Idaho:											
Boise	0		0	0	1	0	1	0	0	0	8

City reports for week ended July 16, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Colorado:											
Colorado Springs	0	------	0	0	0	1	0	2	0	8	9
Denver	3	------	0	5	3	11	0	1	1	20	82
Pueblo	1	------	0	9	1	2	0	2	0	5	9
New Mexico:											
Albuquerque	0	------	0	0	1	0	0	2	0	2	13
Utah:											
Salt Lake City	0	------	0	64	2	1	0	2	0	23	38
Washington:											
Seattle	0	------	0	2	1	3	0	0	0	23	59
Spokane	0	------	0	3	1	1	0	0	0	7	31
Tacoma	-------	------	-------	-------	-------	-------	-------	------	-------	-------	-------
Oregon:											
Portland	1	------	0	4	2	4	0	0	0	0	71
Salem	0	1	-------	0	-------	0	0	-------	0	1	-------
California:											
Los Angeles	9	8	0	33	14	23	1	15	0	27	326
Sacramento	0	------	0	21	1	0	0	1	0	11	22
San Francisco	0	1	0	9	0	5	0	9	0	23	133

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Maine:				Missouri:			
Portland	0	0	1	Kansas City	0	0	1
Rhode Island:				District of Columbia:			
Providence	0	0	1	Washington, D. C.	0	0	1
New York:				Virginia:			
Buffalo	1	0	0	Lynchburg	0	0	1
New York	1	1	1	Norfolk	0	0	1
Pennsylvania:				Tennessee:			
Pittsburgh	1	0	0	Memphis	0	1	0
Ohio:				Louisiana:			
Cleveland	1	0	0	New Orleans	1	1	1
Illinois:				Texas:			
Chicago	0	1	1	San Antonio	0	1	0
Springfield	1	0	0				
Michigan:							
Detroit	0	0	1				
Grand Rapids	0	0	1				

Encephalitis, epidemic or lethargic.—Cases: Dallas, 1; San Antonio, 1.
Pellagra.—Cases: Philadelphia, 1; Baltimore, 1; Lynchburg, 1; Charleston, S. C., 2; Atlanta, 5; Memphis, 1; Birmingham, 5; Montgomery, 1; Fort Smith, 3; San Antonio, 2.
Rabies in man.—Deaths: Detroit, 1.
Typhus fever.—Cases: Wilmington, N. C., 1; Atlanta, 1; Miami, 1; Mobile, 2; Lake Charles, 1; Galveston, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended July 2, 1938.—During the 2 weeks ended July 2, 1938, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia[1]	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis			1	1	6					8
Chickenpox		14	6	110	558	248	144	16	138	1,234
Diphtheria		9	2	57	9	6	3			86
Dysentery									6	6
Erysipelas				9	3	3	1		3	19
Influenza		11			23	1			11	46
Lethargic encephalitis					3			1		4
Measles		28	1	146	1,264	2	3	9	5	1,458
Mumps		30	200		76	44		28	3	381
Pneumonia		3			26				10	39
Poliomyelitis				1	3				2	6
Scarlet fever		19	11	113	111	28	59	58	38	437
Smallpox						10	11			21
Trachoma							4		14	18
Tuberculosis	4	30	23	103	72	3	8	1	28	272
Typhoid fever		1		42	6	5	3	1	1	59
Undulant fever				1	8		1		2	12
Whooping cough		9	13	164	277	58	2	6	75	604

[1] For 2 weeks ended July 6, 1938.

CUBA

Habana—Communicable diseases—4 weeks ended July 2, 1938.—During the 4 weeks ended July 2, 1938, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria	14		Tuberculosis	10	3
Malaria	[1] 14	1	Typhoid fever	[1] 31	4
Scarlet fever	1				

[1] Includes imported cases.

Provinces—Notifiable diseases—4 weeks ended June 25, 1938.—During the 4 weeks ended June 25, 1938, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer	2	1	1	5	----	1	10
Chickenpox	1	2	3	4	2	6	18
Diphtheria	----	19	2	7	----	11	39
Dysentery (bacillary)	----	----	----	11	----	----	11
Hookworm disease	2	76	----	----	----	----	78
Leprosy	----	----	----	1	----	----	1
Malaria	42	14	1	49	15	73	194
Measles	1	13	2	4	----	3	23
Poliomyelitis	1	----	1	----	2	1	5
Scarlet fever	----	1	----	----	----	1	2
Tuberculosis	28	52	57	34	27	42	240
Typhoid fever	11	52	26	74	24	85	272
Yaws	----	----	----	----	----	73	73

CZECHOSLOVAKIA

Communicable diseases—April 1938.—During the month of April 1938, certain communicable diseases were reported in Czechoslovakia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Cerebrospinal meningitis	63	23	Poliomyelitis	5	1
Chickenpox	220	----	Puerperal fever	32	15
Diphtheria	2,097	108	Scarlet fever	1,753	25
Dysentery	7	1	Trachoma	65	----
Influenza	136	----	Tularaemia	2	----
Lethargic encephalitis	2	2	Typhoid fever	324	30
Malaria	357	----	Typhus fever	11	1
Paratyphoid fever	21	2			

IRISH FREE STATE

Vital statistics—Quarter ended March 31, 1938.—The following vital statistics for the Irish Free State for the quarter ended March 31, 1938, are taken from the Quarterly Return of Marriages, Births, and Deaths, issued by the Registrar General and are provisional:

	Number	Rate per 1,000 population		Number	Rate per 1,000 population
Marriages	3,817	5.2	Deaths from:		
Births	14,056	19.2	Influenza	217	0.3
Total deaths	11,442	15.7	Measles	45	----
Deaths under 1 year of age	----	[1] 81	Puerperal sepsis	11	[1] 8
Deaths from:			Scarlet fever	30	----
Cancer	859	1.2	Tuberculosis (all forms)	860	1.2
Diarrhea and enteritis (under 2 years)	120	----	Typhoid fever	17	----
Diphtheria	96	----	Whooping cough	43	----

[1] Per 1,000 births.

JAMAICA

Communicable diseases—4 weeks ended July 9, 1938.—During the 4 weeks ended July 9, 1938, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox	3	29	Leprosy		2
Diphtheria	1	3	Scarlet fever	1	1
Dysentery	17	13	Tuberculosis	41	83
Erysipelas		1	Typhoid fever	9	42

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

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for July 29, 1938, pages 1322–1335. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China.—Cholera has been reported in China as follows: Week ended July 16, 1938, Canton, 2 cases; Hong Kong, 60 cases; Macao, 121 cases; Shanghai, 254 cases; Swatow, 56 cases. Week ended July 23, 1938, Tientsin, 2 cases.

Indochina (French).—During the week ended July 16, 1938, cholera was reported in French Indochina as follows: Annam Province, 137 cases; Tonkin Province, 63 cases; Hanoi, 12 cases.

Plague

Bolivia.—During the period June 6–12, 1938, plague was reported in Bolivia as follows: Santa Cruz Department, 1 case; Tarija Department, 44 cases.

United States.—Reports of plague-infected fleas in San Bernardino County, Calif., and of plague-infected fleas and squirrels in Uinta and Lincoln Counties, Wyo., appear on page 1373 of this issue of PUBLIC HEALTH REPORTS.

Typhus Fever

Colombia—Barranquilla.—During the week ended July 2, 1938, 1 death from typhus fever was reported in Barranquilla, Colombia.

Yellow Fever

Brazil.—Yellow fever has been reported in Brazil as follows: Amazonas State, May 18, 1938, 1 death; Minas Geraes State, June 20, 1938, 1 death.

Colombia—Cundinamarca Department.—Yellow fever has been reported in Cundinamarca Department, Colombia, as follows: May 15, 1938, 2 deaths; June 2, 1 death; June 4, 1 death; June 19, 1 death.

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