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RIBOFLAVIN DEFICIENCY IN MAN (ARIBOFLAVINOSIS)[1]

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Many of the early writers on pellagra (*1*) recognized that certain symptoms of the disease sometimes occurred without the skin lesions, and the term "pellagra sine pellagra" was introduced to designate these symptoms. In 1912 Stannus (*2*), in describing pellagra in Nyasaland, particularly noted lesions in the angles of the mouth which he called "angular stomatitis." Similar lesions with various other symptoms have been described by numerous other observers. In 1928 Jenner Wright (*3*) in Sierra Leone described lesions at the mucocutaneous junction associated with nervous system lesions which were cured by cod liver oil and yeast. Lesions which appear to be similar in many respects have been seen by Fitzgerald (*4*) (1932) in an Assam prison; Moore (*5*) (1934) in school children in Nigeria; Landor and Pallister (*6*) (1935) in the prisons of Singapore and Johore, and Aykroyd and Krishnan (*7*) (1936) in school children in South India.

As early as 1918 Goldberger, Wheeler, and Sydenstricker (*8*) suggested that two different dietary factors may be involved in pellagra, and in 1925 Goldberger and Tanner (*9*), in their experiments with casein, noted that the patients developed a dry, glazed vermilion border of the lips, erosions at the angles of the mouth, reddening of the lips, and seborrhea about the nose. They diagnosed these lesions as pellagra sine pellagra. They also saw in some a pasty, caseous accumulation in the nasolabial folds which cleared up when dried yeast was added to the diet.

In 1933 Wheeler (*10*) saw similar lesions in connection with the use of a haddock diet and discusses at some length the possibility of these symptoms being a different condition from pellagra as characterized by the typical dermatitis. He was unable to conclude definitely, however, whether the lesions were due to a marginal deficiency

[1] From the Division of Chemistry, National Institute of Health.

in the pellagra-preventive factor, or to a deficiency in some other vitamin.

In 1938 Sebrell (*11*) discussed the possibility of clinical pellagra being a multiple deficiency, and pointed out that riboflavin deficiency might occur in man simultaneously with, or independently of, pellagra. Therefore, in view of the uncertainty existing in regard to the etiology of the syndrome designated pellagra sine pellagra, a series of observations was made in order to determine whether it is due to a deficiency in nicotinic acid or some other vitamin.

A brief preliminary note on part of the observations has already been published (*12*).

OBSERVATIONS

A group of 18 adult white women in an institution were given a careful physical examination and found to be in good general condition except for mental disorders and physical defects of a nature which would not interfere with the observations. There were no interfering skin, lip, or buccal lesions.

The diet was then changed from the general, varied institution menu to the ration given in tables 1 and 2, which is a modification of that used by Goldberger and Tanner (*9*). The diet was prepared in a special diet kitchen under the direct, careful, and constant supervision of a trained dietitian,[4] who weighed each item in the diet. The entire group ate simultaneously at one table under the supervision of attendants and nurses. Any food left on the plates at the end of the meal was weighed and an approximation of the actual food intake at each meal was obtained.

TABLE 1.—*Casein diet* [1]

BREAKFAST AND SUPPER [2]

		Ounces	Grams	Protein	Fat	Carbohydrate
				Gm.	*Gm.*	*Gm.*
Mixture	Cornmeal	2.5	71.0	6.0	3.3	52.5
	Cowpeas	.16	4.54	1.0	.06	2.7
	Lard	.25	7.0	0	7.0	0
	Casein [3]	.81	23.0	19.7	.07	0
	Salt and pepper					
Brown gravy	Flour	.25	7.0	.8	.07	5.0
	Lard	.25	7.0	0	7.0	0
	Salt and pepper					
Cane sirup		1.5	42.35	0	0	30.0
Loaf bread		1.8	51.0	4.1	2.4	26.5
Coffee, little sugar						
Total		7.51	212.89	31.6	19.9	116.7
Calories (total 772) [2]				126.0	179.0	467.0

[1] Calculated from the Chemical Composition of American Food Materials. By W. O. Atwater and A. P. Bryant. U. S. Dept. of Agr. Bull. No. 28.

[2] In addition, 0.25 oz. (7 grams) of sirup, equaling 20 calories, was given for supper.

[3] Analysis of the casein yielded moisture 9.14 percent, nitrogen 13.69 percent, and ether extract 0.3 percent.

[4] We are very grateful to Mrs. Marie S. Echols, dietitian, U. S. Public Health Service, for her able assistance in carrying out this phase of the work.

TABLE 2.—*Casein diet* [1]

DINNER

		Ounces	Grams	Protein	Fat	Carbohydrate
				Gm.	*Gm.*	*Gm.*
Mixture	Cornmeal	2.5	71.0	6.0	3.3	52.5
	Cowpeas	.16	4.54	1.0	.06	2.7
	Lard	.25	7.0	0	7.0	0
	Casein	.81	23.0	19.7	.07	0
	Salt and pepper					
Brown gravy	Flour	.25	7.0	.8	.07	5.0
	Lard	.25	7.0	0	7.0	0
	Salt and pepper					
Cane sirup		1.5	42.35	0	0	30.0
Cornbread	Cornmeal	2.0	57.0	4.8	2.7	42.2
	Lard	.125	3.6	0	3.6	0
Cod liver oil		.5	14.0	0	14.0	0
Tomato juice [2]		4.0	112.0	.98	.08	4.0
Total				33.23	37.88	136.4
Calories (total 1,020)				134.0	340.0	546.0

[1] Calculated from the Chemical Composition of American Food Materials. By W. O. Atwater and A. P. Bryant. U. S. Dept. of Agr. Bull. No. 28.

[2] Analysis of the tomato juice yielded moisture 94.33 percent, ash 0.95 percent, nitrogen 0.14 percent, and ether extract 0.07 percent.

The ration was prepared as follows: White cornmeal, coarsely ground cowpeas, leached casein,[3] and calcium carbonate were weighed into the inner portion of a large double boiler with a little salt and sufficient water to cook satisfactorily. Nineteen portions were cooked in order to be able to remove 18 without loss. After cooking 1½ hours enough water was added to bring the mixture to the proper consistency. After deducting the weight of the container, the remainder was divided by 19, and 18 portions served. The one portion remaining in the container served as a check against errors in serving.

The gravy which was served on the cereal legume mixture was prepared by browning white flour, adding lard, and bringing to a convenient volume with water. Nineteen servings were prepared and 18 served.

The cornbread was prepared by mixing white cornmeal and lard with salt and water, and baking. After baking, the total weight was divided by 19, and 18 portions served.

The loaf bread was prepared in the institution bakery from 100 pounds of white flour, 6 pounds of lard, 5 pounds of sugar, and water. This yielded 152 loaves of 1 pound each. The freshly made bread was delivered to the diet kitchen each day and was sliced and weighed by the dietitian before each meal.

The sirup was a commercial cane sirup served by volume as drawn from the barrel.

The tomato juice was a commercially canned variety to which isrup of iodide of iron was added. This was served by volume, and

[3] Commercial casein leached for a week in daily changes of acidulated water. (After McCollum, Simmonds, Shipley, and Park: Bull. Johns Hopkins Hosp., 33: 298 (1922).)

the cod liver oil (U. S. P. XI) measured by volume individually into the same cup. Once each week beginning on the 13th week of the experiment 3.3 mg. of crystalline thiamin chloride and 30 mg. of crystalline ascorbic acid in solution were also added to each serving of tomato juice.

The coffee given at breakfast and supper contained a small amount of sugar, but no milk. The total daily food allowance is given in table 3. If the entire ration was consumed, a total of approximately 2,584 calories was taken daily, which was derived from 96.5 gm. of protein, 77.6 gm. of fat, and 374.8 gm. of carbohydrate. Thus, the protein supply is ample and of good quality, the carbohydrate-fat ratio is adequate, and the energy intake is sufficient for adult, non-working women.

TABLE 3.—*Daily food allowance*

Meal	Calories	Protein	Fat	Carbohydrate
		Gm.	*Gm.*	*Gm.*
Breakfast	772	31.6	19.9	116.7
Dinner	1,020	33.3	37.8	136.4
Supper	792	31.6	19.9	121.7
Total	2,584	96.5	77.6	374.8

The mineral and vitamin analysis of the ration is given in table 4. It is seen that the ration supplied 15 mg. of iron, 1.5 gm. of calcium, 1.2 gm. of phosphorus, 7.4 mg. of iodine, 9,324 International Units of vitamin A, 435 I. U. of B_1, 14.3 mg. of ascorbic acid (vitamin C), and 1,190 I. U. of vitamin D. The ration is somewhat low in nicotinic acid and contains very little riboflavin.

TABLE 4.—*Mineral and vitamin content of diet*

Food	Quantity	Iron	Calcium	Phosphorus	Iodine	Vitamins			
						A	B_1	C	D
	Gm.	*Gm.*	*Gm.*	*Gm.*	*Gm.*	*I. U.*	*I. U.*	*Mg.*	*I. U.*
Cornmeal	270.0	0.0024	0.048	0.513	------	------	175	------	------
Cowpeas	13.5	.0010	.013	.061	------	------	23	------	------
Casein	69.0	------	------	.593	------	------	------	------	------
Lard	47.5	------	------	------	------	------	------	------	------
Flour	56.5	.0005	.011	.051	------	------	21	------	------
Cane sirup	133.0	.0097	.280	.058	------	------	39	------	------
Calcium carbonate	3.0	------	1.200	------	------	------	------	------	------
Tomato juice	112.0	------	.006	.016	------	924	37	10	------
Cod liver oil	14.0	------	------	------	------	8,400	------	------	1,190
Iodide of iron	0.1	.0016	------	------	0.0074	------	------	------	------
Thiamin chloride [1]	------	------	------	------	------	------	140	------	------
Ascorbic acid [1]	------	------	------	------	------	------	------	4.3	------
Total	------	.0152	1.558	1.292	.0074	9,324	435	14.3	1,190

[1] Calculated as one-seventh of weekly dose.

On this ration 10 of the 18 women developed symptoms similar to those previously described as pellagra sine pellagra between the 94th and 130th days. There was maceration in each angle of the mouth, the lips were reddened along the line of closure, and the mucosa appeared thin, shiny, and denuded. We have called this lesion a cheilosis (morbid condition of the lips). The fissures in the angles of the mouth resemble the lesions described as perlèche. Smears taken from 2 cases showed what appeared to be gram-positive diplococci. On culture the organism was found to be a streptococcus. No Monilia were seen. In addition to the lip lesions there was also a scaly, greasy desquamation in the nasolabial folds, on the alae nasi, in the vestibule of the nose, and, in a few instances, on the ears and eyelids.

One of these 10 women had developed mild skin lesions of pellagra, beginning on the 36th day and progressing until a definite diagnosis was made on the 76th day. At this time treatment was started with a daily dose of 30 mg. of nicotinic acid. The skin lesions completely disappeared in 30 days. In spite of the continued administration of this amount of nicotinic acid daily, the cheilosis appeared 21 days after the skin lesions had completely healed and 127 days from the beginning of the experiment. On the 130th day the nicotinic acid was increased to 100 mg. daily. Forty-five days later the lesions were still present and increasing in severity. Treatment was then started with 0.025 mg. of riboflavin per kilogram of body weight daily, and the lesions completely disappeared in 6 days.

Five of the 10 women with the cheilosis were treated with a daily dose of 100 mg. of nicotinic acid for from 5 to 43 days without benefit. Four were given 1 mg. of synthetic riboflavin for 3 days and then all 5 were given 0.025 mg. per kilogram of body weight daily. The symptoms entirely disappeared in 4 in 10, 12, 13, and 24 days. The fifth woman, who was very obese and whose weight fluctuated around 210 to 215 pounds, showed slow improvement, and after 49 days the daily dose of riboflavin was increased to 0.05 mg. per kilogram of body weight. The lesions then improved more rapidly but failed to heal completely, and after 36 days the daily dose was increased to 0.075 mg. of riboflavin per kilogram of body weight. After 20 days the lesions had completely disappeared.

The remaining 4 women with the cheilosis did not receive any nicotinic acid. Treatment was started with daily doses of 1 mg. or 2 mg. of synthetic riboflavin for from 3 to 10 days, after which the daily dose was changed in all cases to 0.025 mg. per kilogram of body weight. The symptoms completely disappeared after 5, 6, 20, and 47 days of treatment.

In all 10 of the women with the cheilosis, treatment with riboflavin was discontinued as soon as the lesions had entirely disappeared. In all 10 the cheilosis recurred between the 177th and 293d days.

Treatment was again started with a daily dose of 0.025 mg. of synthetic riboflavin per kilogram of body weight and all symptoms disappeared in from 4 to 20 days. The results are summarized in table 5.

TABLE 5.—*Summary of results of treatment*

Patient number	Days to first symptom	Nicotinic acid			Day began riboflavin	Day symptoms disappeared	Day discontinued riboflavin	Day symptoms recurred	Day resumed riboflavin	Day symptoms disappeared
		Day started	Daily dose	Day discontinued						
1	131				136	183	183	293	332	352
2	128	131	100	160	136	241	244	265	272	284
3	114	131	100	160	136	149	148	204	221	226
5	95				129	135	140	177	202	216
8	128	77 131 190	30 100 30	177	177	183	183	260	265	280
9	128	131	100	160	136	160	160	179	193	206
10	100				129	150	150	236	250	254
11	130	131	100	177	176	184	185	249	265	283
14	130	131	100	160	136	148	150	193	221	226
16	107				129	133	140	186	197	209

In the patient who required the large dosage of riboflavin in the first attack, symptoms cleared up with the 0.025 mg. per kilogram of body weight per day, but in spite of continuing this dosage, maceration occurred in each angle of the mouth beginning on the 293d day and progressed until a small, transverse fissure was seen at the left angle of the mouth on the 330th day. The dosage was increased to 0.05 mg. per kilogram at that time and, although improvement occurred, the lesions still had not entirely healed by the 362d day. The daily dose was then increased to 0.1 mg. per kilogram of body weight. On the 364th day the fissure at the angle of the mouth was healing and there was slight denudation of the lower lip. The observations were then discontinued.

The 8 of the 18 women who had not developed the cheilosis by the 139th day were separated into 2 groups of 4 each. One group began a daily preventive dose of 0.025 mg. of synthetic riboflavin per kilogram of body weight. No symptoms were observed in this group during the observation period of 365 days except for mild skin lesions of pellagra in 1, beginning on the 362d day. The other group of 4 continued the ration without the addition of riboflavin. Two of this group developed the cheilosis on the 191st and 293d day, and a third showed slight maceration in the angle of the mouth, with a lesion in the vestibule of the nose, on the 200th day. All 3 were given a daily dose of 0.025 mg. of synthetic riboflavin per kilogram of body weight. The lesions then disappeared after 8, 9, and 25 days. One of these on self-restricted food intake also developed hyperesthesia of the feet, and a daily dose of 6.6 mg. of thiamin was started on the 275th day.

The fourth woman did not show lesions of any kind during the 365 days of observation on the diet. The results are summarized in table 6.

TABLE 6.—*Preventive test*

Patient No.	Days to first symptom	Day began riboflavin	Day symptoms disappeared	Day discontinued riboflavin
4		139		
6		139		
7		139		
15		139		
12	293	307	316	317
13	200	221	229	229
17				
18	191	265	290	

A careful record of the daily food consumption was kept by weighing back the food left on each plate at each meal. These data are only an

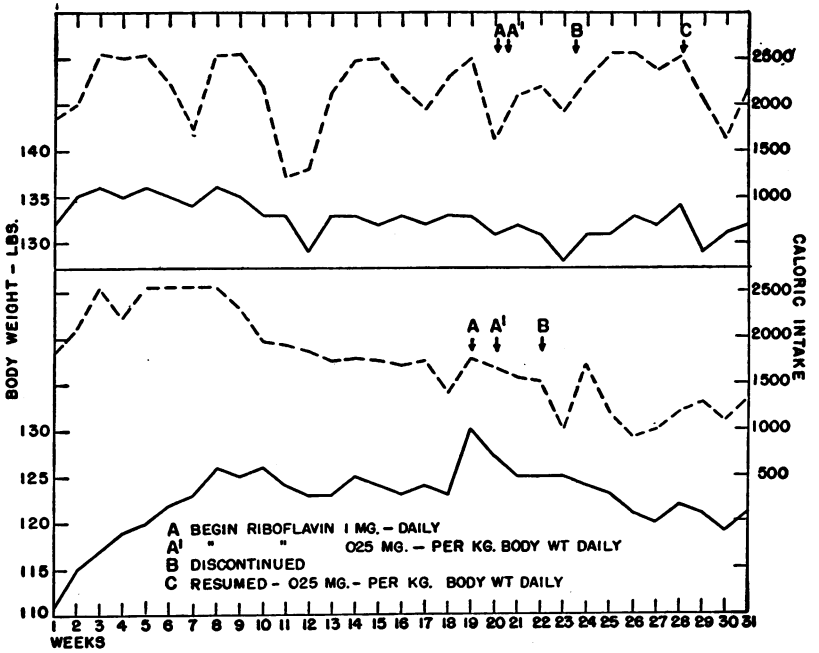

FIGURE 1.

approximation, owing to such factors as unavoidable waste and mixture of various items of the diet on the plate. The data are, therefore, averaged by the day for each week. The patients were weighed once weekly and the body weight correlated with the calculated food intake. Owing to lack of space only sample records of these data from two patients are shown in figure 1. It is seen that there is no consistent

increase in appetite or significant change in body weight during treatment with riboflavin.

Hemoglobin determinations, red blood cell and white blood cell counts were done at regular intervals. These data are presented in table 7. There does not appear to be any significant change during the period of observation.

DISCUSSION

These observations indicate that the lesions on the lips, the fissures in the angles of the mouth, and the seborrheic accumulations around the nose are manifestations of a deficiency in riboflavin.

Since the publication of our preliminary note on part of these observations, Vilter, Vilter and Spies (*13*) have reported increased vigor, improvement in sense of well-being, and improvement in the cutaneous lesions in 4 cases of pellagra following the administration of 50 mg. of riboflavin daily. Oden, Oden, and Sebrell (*14*) have found naturally occurring cases of riboflavin deficiency, without the skin lesions of pellagra, in Georgia.

It is to be noted that one of the women failed to respond to the daily dose of 0.025 mg. of riboflavin and that it was necessary to increase this dose to 0.075 mg. daily before rapid healing occurred. This observation, together with the fact that the lesions disappeared rather slowly in some of the other women, leads us to believe that this dosage is rather low and that considerably larger amounts should be used in the clinical treatment of the condition. Oden, Oden, and Sebrell (*14*) used 5 mg. daily with success in 3 cases, and Spies, Bean, and Ashe (*15*) have found from 5 to 50 mg. per day to be effective.

In addition to the lesions of riboflavin deficiency, other observers (*15*, *16*) have pointed out that the peripheral neuritis of beriberi occurs in some cases of pellagra. It, therefore, appears that we should revise our concept of clinical pellagra in that the condition may be a mixture of symptoms from three different deficiencies, namely, nicotinic acid, riboflavin, and thiamin chloride, and that any one may occur alone or in combination with any other. Therefore, in order to avoid further confusion, it is suggested that the diagnosis of pellagra should be confined to that syndrome which responds to nicotinic acid, namely, skin lesions, gastro-intestinal lesions, stomatitis, and mental disturbances, while the peripheral neuritis which responds to thiamin chloride should be diagnosed as beriberi, and the lesions described in this paper, which respond to riboflavin, require a new designation since their true nature has not been hitherto recognized. We have suggested the word "ariboflavinosis" for this purpose. Where the clinical condition is characterized by the simultaneous presence of more than one of these syndromes, a diagnosis of a multiple deficiency is indicated.

TABLE 7.—*Blood examinations*

Patient No.	Day of experiment																														
	0–2			23–25			65–67			106–108			147–149			189–191			217–219			259–261			291–293			333–335		
	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC	Hb	RBC	WBC
1	13.0	3.8	6.5	12.4	4.18	7.4	12.5	4.43	7.0	13.3	4.15	7.4	11.9	3.96	7.6	12.0	4.3	5.0	11.8	4.0	7.5	13.0	4.3	5.0	12.5	3.71	4.3	11.7	3.76	4.3
2	13.4	4.0	9.1	13.1	3.86	8.3	14.2	3.88	8.0	12.5	3.75	6.9	13.3	4.0	10.0	12.8	3.3	9.7	13.3	4.05	7.5	13.0	3.9	9.1	12.2	3.3	9.0	12.7	3.6	10.8
3	12.4	3.9	6.2	11.9	4.29	6.3	12.1	4.32	13.0	10.9	4.23	5.2	12.5	4.4	7.2	11.4	3.88	6.9	12.0	3.46	6.5	13.0	3.8	5.5	12.3	3.89	6.0	11.0	3.22	5.6
4	12.1	4.5	7.0	12.8	3.9	7.0	12.5	3.90	5.5	12.5	4.0	6.5	12.5	4.0	8.3	13.0	3.49	9.1	12.0	3.96	6.0	13.0	3.82	7.0	11.9	3.6	7.7	11.6	3.74	6.0
5	12.2	4.6	7.0	11.8	4.25	7.2	12.4	4.15	5.6	11.5	3.89	4.5	12.5	4.17	7.2	11.5	3.96	6.2	12.0	4.0	6.2	12.0	3.6	5.3	10.8	3.6	4.3	10.0	3.08	4.3
6	12.2	3.8	9.0	12.0	4.11	9.7	13.6	4.06	9.5	11.0	3.72	7.1	12.5	3.75	8.6	11.8	3.7	7.0	12.0	3.5	7.4	12.0	3.7	8.4	12.0	3.9	7.2	11.4	3.9	5.0
7	11.2	4.5	9.2	10.2	3.83	7.4	11.3	4.01	8.7	11.6	3.93	6.8	11.4	3.86	7.0	10.1	3.7	10.0	10.5	3.6	7.5	11.4	3.7	8.4	10.4	3.79	7.8	10.0	3.5	9.0
8	10.4	3.3	6.2	10.0	3.32	5.9	10.9	3.24	6.8	10.4	3.92	6.8	11.3	3.83	6.3	10.5	3.4	4.0	11.0	3.5	7.2	11.6	3.81	6.1	9.8	3.13	7.6	8.8	3.0	7.8
9	12.0	4.3	9.3	11.4	3.83	5.0	12.1	4.07	5.2	12.7	4.16	4.6	13.0	4.0	6.3	11.5	3.55	5.6	13.0	4.04	7.0	13.7	4.27	14.7	12.0	3.7	5.5	11.9	3.63	4.0
10	12.0	4.0	12.5	11.4	3.86	10.2	13.5	4.01	8.2	13.0	4.44	9.3	13.4	4.2	10.2	12.6	3.4	6.5	13.5	3.7	10.0	13.9	4.12	10.8	13.1	3.7	11.0	11.9	3.38	9.3
11	12.5	4.7	9.7	12.5	4.62	7.0	14.5	3.59	10.5	12.2	4.28	6.2	12.3	3.8	8.4	11.5	3.5	6.0	12.0	4.0	5.0	12.0	3.6	5.0	11.0	3.65	6.3	11.4	3.95	6.5
12	9.0	4.5	7.2	11.0	3.64	7.1	11.8	3.93	20.0	11.0	3.96	8.3	12.6	3.5	6.4	11.8	3.9	8.0	11.6	3.91	7.1	10.5	3.5	5.9	11.0	3.7	7.3	11.6	3.46	6.7
13	11.8	4.6	5.0	11.4	3.84	5.3	12.5	3.13	4.0	12.0	4.44	3.0	13.0	3.91	5.0	13.0	3.8	6.0	13.7	4.07	5.5	12.5	3.6	4.0	12.7	3.84	4.2	11.7	3.7	4.0
14	14.5	3.7	6.6	11.0	3.39	7.8	14.6	3.85	5.8	14.0	4.39	5.9	13.0	3.82	6.9	13.5	4.07	5.0	13.4	4.0	6.0	14.0	4.28	4.3	13.5	3.9	5.8	11.5	3.37	5.7
15	11.4	3.9	10.4	11.5	4.15	9.0	14.6	4.05	10.2	13.1	4.24	10.5	13.1	3.81	12.0	12.2	3.9	6.3	12.2	3.92	9.0	13.3	3.79	11.5	12.0	3.4	10.0	12.9	3.78	9.0
16	10.0	3.6	5.4	12.5	3.66	8.4	11.9	3.93	6.0	10.1	3.49	7.3	10.0	3.3	7.2	9.0	3.2	5.0	9.5	3.16	7.7	11.0	3.45	6.9	9.0	2.91	6.3	9.0	3.09	8.2
17	11.9	3.7	6.5	12.0	4.14	4.6	11.6	3.6	4.0	12.5	3.54	4.1	12.8	3.56	6.3	13.5	4.01	8.0	13.0	3.77	7.0	13.0	3.56	5.0	12.0	3.5	5.2	11.0	3.31	3.5
18	12.0	4.3	9.4	11.9	4.0	4.2	9.4	3.86	4.1	10.0	4.33	4.3	10.3	3.94	6.5	10.0	3.76	7.0	11.0	3.86	6.3	11.3	3.91	7.2	11.8	3.86	7.8	9.5	3.46	5.5

The above considerations make it important to emphasize especially the role of an adequate diet, and the use of foods naturally rich in nicotinic acid, thiamin, and riboflavin, in the treatment of these conditions.

The crystalline vitamin preparations are very valuable therapeutic agents and should be used after a correct diagnosis has been made, but their limitations in the presence of multiple deficiencies must also be recognized, and an adequate diet is also one of the essentials for the proper treatment of deficiency diseases. Fortunately, yeast and liver and most natural foodstuffs that are rich in one of these vitamins also contain the others to some extent. It is only when the treatment of a deficiency disease, such as pellagra, beriberi, or ariboflavinosis is undertaken with a crystalline vitamin preparation alone, without giving due attention to the diet, that the possibility of the simultaneous presence of symptoms from a deficiency in one of the other vitamins becomes of serious importance and the use of other vitamin preparations may be necessary to secure relief from all symptoms.

SUMMARY

Thirteen out of 18 women receiving a special diet low in riboflavin content developed a reddened, denuded lesion of the lips, maceration and fissuring in the angles of the mouth, and seborrheic accumulations at the nasolabial folds. These lesions disappeared following the daily administration of synthetic riboflavin; they reappeared following the discontinuance of the riboflavin, and again disappeared following riboflavin therapy. Six of these women were treated for varying lengths of time with nicotinic acid without benefit.

Four of the remaining 5 women began a daily preventive dose of synthetic riboflavin on the 139th day and showed no lesions of any kind during the 365 days of observation.

One woman did not receive any riboflavin therapy and showed no lesions at any time during the 365 days of observation.

CONCLUSIONS

Lesions on the lips and seborrheic accumulations on the face similar in appearance to the condition formerly described as pellagra sine pellagra occurred in women on a diet low in riboflavin and were alleviated and prevented by the administration of synthetic riboflavin, but were not benefited by nicotinic acid. The conclusion therefore seems warranted that these lesions are a manifestation of riboflavin deficiency.

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HUMAN SERUM AS A STABILIZER OF SCARLET FEVER STREPTOCOCCUS TOXIN DILUTED FOR THE DICK TEST [1]

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The Dick test when made with test toxin of the required potency is a reasonably reliable method for determining susceptibility to scarlet fever. Using a reaction of 10 × 10 mm. or greater as an indication of susceptibility, the author has observed an attack rate of 0.7 per 1,000 in grammar school children who reacted negatively to the test as compared with a rate of 6.8 in their untested classmates (it is estimated that this untested control group is 44.7 percent Dick positive). The tests in this study were made with freshly diluted test toxin, a portion of each lot being returned from the field for check testing. However, such rigid requirements cannot be laid down for the test toxin offered the medical profession by the biological laboratories where a dating period must be allowed. At the present time a dating period of not more than 6 months is permitted the diluted toxin. Obviously such toxin should show no significant deterioration within

[1] From the Division of Infectious Diseases, National Institute of Health.

that period, if kept under the conditions recommended on the label; deterioration, nevertheless, sometimes occurs. In view of this fact, some thought has been given to the possibility of finding a diluent which will insure greater stability to the test toxin.

As the result of some other work with the use of colloidal substances in scarlet fever toxin, a study has been made of the stabilizing effect of human serum on toxin diluted for the Dick test.[2] Human serum was selected because its presence would not introduce a foreign protein into the test solution.

EXPERIMENTAL METHODS

Preparation of the diluent.—The Scarlet Fever Committee recommends to its licensed laboratories the use of a 10 percent phosphate buffer of pH 7.0 in normal saline, to which 0.4 percent of phenol is added. Such a buffered diluent at different pH levels has been used in the present study, either alone or with varying amounts of sterile, human serum. Later the amount of serum used was fixed at a 1:500 dilution.

Types of glass containers.—These included pyrex glass bottles with and without glass stoppers, three types of flame-sealed glass ampules, and eight types of rubber stoppered ampules, of which six were clear glass and two amber. The object was to try various qualities of both glass and stopper.

pH concentration of the diluent.—A pH level of 7.0 heretofore had been considered essential for stability of the test toxin. In the present study it was thought advisable to include a wider range. Altogether five levels were studied, beginning with 6.6 and increasing at 0.2 intervals.

Temperature.—Observations were made at 37° C., room temperature, the varying temperatures encountered during transit by ordinary mail, and at 5° C.

EXPERIMENTAL RESULTS

It is to be expected that the presence of a 1:500 concentration of human serum in the diluted test toxin would not significantly alter the character of the human skin reaction to the toxin. Nevertheless, tests were made on a group of student nurses with freshly diluted toxin, with and without serum. The individual reactions are shown in table 1, from which it must be concluded that the serum has no significant influence on the size of the reaction. In some instances it seemed that the reaction from the serum-containing toxin was slightly more intense and possessed a more clearly defined border.

A series of toxin dilutions was next set up at pH 7.0 in glass stoppered pyrex bottles of liter size with varying amounts of human serum.

[2] Since this work was begun, Glenny and Stevens (Brit. Med. J., 1:709 (1937)) have reported on a similar study of diphtheria toxin for the Schick test.

These were stored at room temperature in a dim light. The toxin dilution in plain buffer soon began to show some deterioration (table 2), whereas the test toxins containing serum showed no loss of potency over a period of 651 days. Because of the comparable results obtained in human tests (table 1) with and without 1:500 serum and the stability shown in table 2 with this amount, it was accepted for use in the succeeding experiments.

TABLE 1.—*Protocol showing the Dick test reactions on human test subjects when tested simultaneously on the forearms with National Institute of Health standard control toxin freshly diluted with and without human serum*

Subject [1]	Standard control toxin diluted in plain or buffered saline [2]	Standard control toxin diluted in buffered saline + 1: 500 human serum [2]	Subject [1]	Standard control toxin diluted in plain or buffered saline [2]	Standard control toxin diluted in buffered saline + 1: 500 human serum [2]
EGB	22×27 P.	23×27 P.	FMP	20×25 FP.	19×23 P.
MJB	18×18 FP.	18×18 FP.	VKR	20×32 P.	19×27 P.
WMD	15×28 FP.	16×21 FP.	SKR	16×26 FP.	19×23 P.
CWH	16×19 FP.	14×14 FP.	DMR	16×21 VFP.	17×21 FP.
LEM	13×18 VFP.	13×18 VFP.	LSS	17×22 FP.	16×18 FP.
MEM	15×18 VFP.	13×18 VFP.	MV	20×26 FP.	20×28 FP.
RLM	12×15 VFP.	13×15 VFP.	MRW	15×17 VFP.	15×17 VFP.
LIKM	15×26 VFP.	20×25 FP.	RAW	15×16 FP.	15×16 FP.
NMM	21×23 P.	21×23 P.	E	19×26 FP.	20×23 FP.

[1] 28 other subjects were negative to both toxins.
[2] The letters following the measurements indicate the intensity of the reaction as very faint pink, faint pink, or pink.

TABLE 2.—*Stability of Dick test toxin diluted in buffered saline of pH 7.0, with and without human serum, contained in one-liter glass stoppered pyrex bottles, and stored at room temperature and in light*

Date of testing	No serum		1 STD of control toxin	Serum 1:500, trial toxin	1 STD of control toxin	Serum 1:1,000, trial toxin	1 STD of control toxin	Serum 1:2,500, trial toxin
	1 STD of control toxin	Trial toxin						
Apr. 27, 1937 [1]								
June 1, 1937	19×25+ [2]	15×15±	18×22+	16×26+	14×20+	15×18+	11×11+	14×14+
July 7, 1937	16×26+	14×24+	14×16+	15×19+	13×14+	16×16+	16×17+	20×20+
Aug. 10, 1937	16×25+	16×25+	15×18+	15×16+	16×17+	20×20+	13×20+	17×20+
Sept. 9, 1937	15×24+	15×20±	16×17+	15×20+	15×25+	14×24+	16×23+	17×25+
Dec. 29, 1937	15×20+	10×15±	16×18+	18×20+	18×18+	18×20+	16×18+	16×20+
Apr. 27, 1938	16×16+	Lost	14×20+	16×20+	18×20+	20×20+		
Feb. 13, 1939 [3]	15×25+	15×15+	14×18+	16×20+	15×20+	14×20+	17×19+	20×20+

[1] Dilutions prepared on this date.
[2] The symbols following the reaction measurements indicate the intensity of the reactions both in color and swelling. The symbols grade upward as ±, +, ++, and +++.
[3] Total elapsed time is 651 days.

The influence of varying the pH level is shown in table 3. In this experiment batches of toxin were buffered at pH 6.6, 6.8, 7.0, 7.2, and 7.4, respectively, with and without serum. The results at 6.6 are not shown in the table since they are the same as for the other levels. The respective dilutions were placed in 100-cc. cork stoppered pyrex bottles, stored at 37° C. for 7 months and then at 5° C. for the remainder of the 561 days of the experiment. The dilutions containing

no serum, irrespective of the pH level, showed a progressive deterioration of the skin reacting factor. At the same time no dilution containing 1:500 serum showed significant deterioration. A freshly diluted standard control was not used in this experiment until after a lapse of 591 days, when each serum-containing dilution was tested against the control with the following results:

	6.8	*7.0*	*7.2*	*7.4*
Reaction to the freshly diluted control toxin	16×20+	10×12+	15×20+	16×17+
Reaction to the serum-containing test toxin	15×20+	14×16+	16×19+	17×17+

TABLE 3.—*Stability of Dick test toxin diluted in buffered saline of different pH levels, with and without human serum, contained in cork stoppered pyrex bottles of 100 cc. capacity, stored at 37° C. for 7 months, and then at 5° C. for an additional period*

Date of testing	pH 6.8		pH 7.0		pH 7.2		pH 7.4	
	Serum 1:500	No serum	Serum 1:500	No serum	Serum 1:500	No serum	Serum 1:500	No serum
July 27, 1937 [1]								
Aug. 4, 1937	20×20++	16×19+	17×21++	15×19+	16×30++	15×25+	20×30++	18×30+
Aug. 10, 1937	18×20+	14×20±	20×21+	15×15±	14×25+	15×25±	18×20+	16×20±
Aug. 17, 1937	18×21+	15×16±	19×22+	10×12±	19×26+	14×20±	19×30+	15×18±
Aug. 25, 1937	19×24+	15×15±	17×22+	15×16±	17×30+	15×20+	19×26+	13×16±
Sept. 1, 1937	20×30++	20×20+	22×25+	18×20+	21×40++	17×30+	20×30++	15×20+
Sept. 9, 1937	20×24+	12×15±	21×21+	10×10±	17×30+	15×25±	16×25+	11×15±
Sept. 23, 1937	22×25++	16×20±	22×24++	14×15±	16×30++	13×25+	21×30++	15×20+
Nov. 1, 1937	18×20+	5 ×7±	15×15+	8 ×10±	15×21+	11×15±		
Nov. 26, 1937	22×28++	20×25+	20×22++	13×15+	19×25++	13×18+	19×24++	13×18+
Dec. 29, 1937	18×20++	12×18+	19×20++	14×15+	18×34++	14×15+	18×24++	15×16+
Apr. 27, 1938	22×25+	15×18±	15×24++	14×17±	20×20+	16×18±	16×30+	14×20±
Feb. 13, 1939 [2]	16×30+	Neg.	18×21+	Neg.	20×20+	Neg.	17×26+	6 ×8+

[1] Dilutions prepared on this date.
[2] Total elapsed time of the experiment is 561 days. A fifth dilution at pH 6.6 showed similar reactions.

Consideration was next given to the type of glass and the stopper used in the dispensing vials. As previously stated, the object was to include ampules of varying qualities of glass and stoppers. All of the filled ampules were placed in an inverted position so that the toxin solution would come in contact with the stopper and were stored at 37° C. for 181 days, and then at 5° C. for 332 days. Ampules were prepared for the full pH range, but because of the large number, sufficient test animals were not available to include all variations shown in table 4. Random sampling of ampules both as to type and pH level invariably gave similar results and it was therefore decided to report in detail only on the 7.0 and 7.4 pH level. These levels were selected because the former has heretofore been considered the most favorable to the diluted toxin, and the latter, the most detrimental. In this experiment again the data indicate that diluted toxin containing 1:500 human serum showed no deterioration in the skin-reacting factor, irrespective of the type of glass or stopper or the pH level. This is in contrast to the marked deterioration shown in the corresponding dilutions without serum.

TABLE 4.—*Stability of Dick test toxin when diluted in buffered saline at different pH levels, with and without human serum, contained in various types of ampules, stored at 37° C. for 180 days and then at 5° C. for an additional period*

Ampules of the following types were filled with test toxin, with and without 1:500 human serum on Sept. 20, 1937	Elapsed time 67 days			Elapsed time 122 days		
	Control toxin	Serum 1:500	No serum	Control toxin	Serum 1:500	No serum
Buffered at pH 7.0						
1 cc. nonsol. glass, flame sealed						
1 cc. nonsol. glass, red rubber stopper						
5 cc. nonsol. glass, pure gum stopper		18×18++	15×15+	17×19+	18×23+	Neg.
5 cc. nonsol. glass, red rubber stopper		19×20+	13×13±	16×16+	16×18+	10×15±
1 cc. typhoid vaccine vial, red rubber stopper.				16×28+	15×26+	10×11±
1 cc. nonsol. glass, flame sealed						
1 cc. poor quality glass, flame sealed [1]						
2 cc. poor quality amber, red rubber stopper.				16×20+	15×20+	4×6+
10 cc. N. I. H. serum vial		19×21++	19×20+	15×20+	17×30+	10×15±
5 cc. amber glass, black rubber stopper		17×17+	8×7±	14×18+	18×20+	11×15±
2 cc. rabies vaccine vial, red rubber stopper.				15×20+	15×23+	9×13±
Buffered at pH 7.4						
1 cc. nonsol. glass, flame sealed						
1 cc. nonsol. glass, red rubber stopper						
5 cc. nonsol. glass, pure gum stopper		20×29++	14×20+			
5 cc. nonsol. glass, red rubber stopper		15×21+	12×13±			
1 cc. typhoid vaccine vial, red rubber stopper.						
1 cc. nonsol. glass, flame sealed						
1 cc. poor quality glass, flame sealed [1]						
2 cc. poor quality amber, red rubber stopper.						
10 cc. N. I. H. serum vial, red rubber stopper.		20×35++	18×25+			
5 cc. amber glass, black rubber stopper		17×19+	5×6±			
2 cc. rabies vaccine vial, red rubber stopper.						

Ampules of the following types were filled with test toxin, with and without 1:500 human serum on Sept. 20, 1937	Elapsed time 152 days			Elapsed time 391 days		
	Control toxin	Serum 1:500	No serum	Control toxin	Serum 1:500	No serum
Buffered at pH 7.0						
1 cc. nonsol. glass, flame sealed				14×16+	15×18+	4×6+
1 cc. nonsol. glass, red rubber stopper				14×20+	14×18+	Neg.
5 cc. nonsol. glass, pure gum stopper	15×18+	17×19+	10×15±	15×18+	15×18+	5×7±
5 cc. nonsol. glass, red rubber stopper	15×22+	17×20+	13×15+	15×17+	14×15+	3×4+
1 cc. typhoid vaccine vial, red rubber stopper.	15×21+	20×20+	Neg.	15×16+	15×19+	2×4+
1 cc. nonsol. glass, flame sealed				15×20+	16×20+	13×14+
1 cc poor quality glass, flame sealed [1]						
2 cc. poor quality amber, red rubber stopper.	18×20+	16×19+	13×18±	15×16+	13×16+	Neg.
10 cc. N. I. H. serum vial	18×23+	20×23+	14×15+	15×17+	13×15+	Neg.
5 cc. amber glass, black rubber stopper	15×18+	15×15+	10×12±	15×18+	14×16+	Neg.
2 cc. rabies vaccine vial, red rubber stopper.	17×24+	16×18+	13×14±	14×15+	14×15+	Neg.
Buffered at pH 7.4						
1 cc. nonsol. glass, flame sealed				14×18+	16×23+	Neg.
1 cc. nonsol. glass, red rubber stopper				15×17+	15×16+	Neg.
5 cc. nonsol. glass, pure gum stopper				15×20+	17×19+	10×11±
5 cc. nonsol. glass, red rubber stopper				16×21+	18×24+	Neg.
1 cc. typhoid vaccine vial, red rubber stopper.				16×21+	17×18+	7×11±
1 cc. nonsol. glass, flame sealed				16×20+	16×18+	16×16±
1 cc. poor quality glass, flame sealed [1]						
2 cc. poor quality amber, red rubber stopper.				17×21+	14×22+	Neg.
10 cc. N. I. H. serum vial, red rubber stopper.				17×23+	14×18+	6×7±
5 cc. amber glass, black rubber stopper				16×25++	16×20+	Neg.
2 cc. rabies vaccine vial, red rubber stopper.				17×22++	18×19++	4×6±

[1] All ampules broken.

A fifth experiment was set up to test the influence of varying climatic conditions on the diluted toxin. One set of dilutions, ampuled in amber glass vials of poor quality, with red rubber stoppers, was sent by ordinary mail to Honolulu and returned. A second set, ampuled in recovered rabies vaccine vials with red rubber stoppers, received from a commercial laboratory, was sent by ordinary mail to Santiago, Chile, and returned. Each set was tested for potency immediately upon return, 34 and 66 days later, respectively, and then stored at 5° C. until the second retest was made. The results as shown in table 5 are in agreement with those obtained in the previous experiments. No deterioration in the skin reacting factor takes place in those dilutions containing a 1:500 dilution of human serum as contrasted to the almost complete deterioration in the control vials.

TABLE 5.—*Stability of Dick test toxin diluted in buffered saline at various pH levels, with and without human serum, contained in two types of ampules and sent through ordinary mail channels*

pH level of dilutions	To Honolulu and return[1] 2-cc. amber ordinary glass vials, red rubber stopper			To Santiago, Chile, and return[2] 2-cc. clear ordinary glass vials, red rubber stopper		
	1 STD of standard control toxin	Dilution containing 1:500 serum	Dilution containing no serum	1 STD of standard control toxin	Dilution containing 1:500 serum	Dilution containing no serum
	FIRST RETEST (ELAPSED TIME 34 DAYS)			FIRST RETEST (ELAPSED TIME 66 DAYS)		
6.6						
6.8		18×18+	6×8±		20×25+	Neg.
7.0		16×18+	5×6±		20×20+	Neg.
7.2		16×25+	6×10±		18×20+	Neg.
7.4		15×20+	10×13±		18×20+	Neg.
	SECOND RETEST (ELAPSED TIME 499 DAYS)			SECOND RETEST (ELAPSED TIME 499 DAYS)		
6.6	15×20+	15×17+	Neg.	16×21+	16×24+	Neg.
6.8	14×17+	14×14+	Neg.	15×18+	14×15+	8×9±.
7.0	18×24+	20×21+	10×10±.	18×25+	20×20+	10×15±.
7.2	16×19+	15×20+	Neg.	17×26+	16×20+	7×9+.
7.4	14×18+	15×18+	Neg.	16×20+	15×22+	11×13±.

[1] These ampules were filled and mailed to an address in Honolulu on Oct. 1, 1937. They were returned on the next mail boat and retested when received on Nov. 4, 1937.

[2] These ampules were filled and mailed to an address in Santiago, Chile, on Oct. 1, 1937, and returned upon receipt. They were received by the National Institute of Health on Dec. 6, 1937, and were retested at once. During the remaining interval, until the second retest on Feb. 15, 1939 (total elapsed time 499 days), all ampules were stored at 5° C.

It should be added that all of the tests recorded in each table, except table 1, were made on the ears of susceptible white rabbits. A control injection of serum diluted without toxin was made in each rabbit and in no instance did this cause a reaction. A series of samples was selected at random at the end of the experiments and titrated against NY-5 antitoxin. The skin reacting factor was completely neutralized, without exception. At the end of the 37° C. incubation

period the toxin in the pH 7.0 amber glass, black rubber stoppered ampule (table 4) with and without serum was tested against a freshly diluted standard control on 18 and 17 pupil nurses, respectively. The reactions were in agreement with those observed on the rabbits and reported in table 4.

CONCLUSIONS

Within the limits of the experiments set up in this study, the data indicate that the presence of 1:500 human serum, when added to the 10-percent phosphate buffer in normal saline commonly used in this country for the preparation of the Dick test toxin, effectively protects the skin reacting factor against deterioration for an interval of time considerably over the present allowable dating period, within a temperature range of 5° to 37° C., and a pH level of 6.6 to 7.4. There is no evidence that the presence of so small a quantity of human serum significantly influences the resulting skin reaction in the human or the rabbit.

CEREBRAL PATHOLOGY IN RODENTS IN ENDEMIC TYPHUS AND ROCKY MOUNTAIN SPOTTED FEVERS[1]

By R. D. Lillie, *Surgeon*, R. E. Dyer, *Senior Surgeon*, and N. H. Topping, *Assistant Surgeon, United States Public Health Service*

Focal brain lesions in man in European epidemic exanthematic typhus were described by von Prowazek (*1*), Fraenkel (*2*), Aschoff (*3*), Benda (*4*), Ceelen (*5, 6*), Grzywo-Dabrowsky (*7*), Wolbach, Todd, and Palfrey (*8*), Lupu and Petrescu (*9*), and others.

As the susceptibility of guinea pigs was already known (Nicolle et al., *10*), description of brain lesions in this animal followed at once, and has been repeated by several authors (*6, 7, 8, 11, 12, 13, 14*). Similar lesions in guinea pigs were reported in tabardillo by Mooser (*15*), in Manchurian typhus by Kodama and Takahashi (*16*), in endemic typhus of the eastern United States by Dyer, Ceder, Lillie, Rumreich, and Badger (*17*), and in Malayan shop typhus by Lewthwaite and Savoor (*18*).

In Rocky Mountain spotted fever quite similar lesions were observed in man by Pinkerton and Maxcy (*19*), Lillie (*20*), and Harris (*21*), in São Paulo typhus by Meyer in Gomes' report (*22*), and in Malayan rural typhus by Lewthwaite (*23*); the lesions in guinea pigs have been reported by Lillie (*20*) for eastern spotted fever, by Lewthwaite and Savoor (*24*) for Malayan rural typhus, and by Lillie and Dyer (*25*) for eastern and western spotted fevers.

[1] From the Divisions of Pathology and Infectious Diseases, National Institute of Health. A short paper based on data included in this article was presented at the Third International Congress for Microbiology, Sept. 4, 1939.

190322°—39——2

Numerous other animals have been shown to be susceptible to the virus of typhus fever, giving either febrile reactions or inapparent infections. Monkeys (*Macacus sinicus*) were used by Nicolle (*26*), (*M. rhesus* and *Cebus capuchinus*) Anderson and Goldberger (*27*), Nicoll, Krumwiede, Pratt, and Bullowa (*28*), *Macacus* in Malayan rural and urban typhus by Lewthwaite and Savoor (*18, 24, 29*), a chimpanzee (*Pan satyrus*) by Nicolle (*26*), and gibbons by Lewthwaite and Savoor (*18, 24, 29*). Cats (*Felis domesticus*) are reported as susceptible by Lépine and Lorando (*30*), and Le Chuiton, Berge, and Pennanéac'h (*31*), dogs by Nicolle and Conseil in 1912, as insusceptible (*32*), and by Combiesco and Angelesco as susceptible (*33*).

A febrile disease with petechial eruption and Weil Felix reaction is produced by inoculation of the pig (*Sus serofa*) (Violle, *34*).

The ass (*Equus asinus*) was first reported as insusceptible by Nicolle and Conseil (*32*), and later as susceptible by Nicolle and Conseil (*35*), and Blanc and Martin (*36*). Rabbits undergo an inapparent infection and exhibit the Weil Felix reaction (Nicolle and Blaizot, *37*). Nicolle had previously regarded this animal as insusceptible (*32*). Rats present an inapparent infection (Nicolle and Lebailly, *38*, Nicolle, *39, 40*, Otto and Winkler, *41*). Rats have been repeatedly found infected in nature, both *Rattus norvegicus* and *Rattus rattus rattus*, also *Mus* (*Rattus?*) *gentilis* (*42*) and *Rattus rattus alexandrinus* (*47*).

Other species found susceptible include mice (*Mus musculus*) (Nicolle, *39, 40*), gerbils or merions (*Meriones shawi*) (Nicolle, *39, 40*), (Atlas) squirrels (*Xerus* (*Atlantoxerus*) *getulus*) (Blanc, Noury, and Baltazard, *44*), and spermophiles or ground squirrels (*Citellus citellus*) (Lépine, *45*, Combiesco et al., *46*, Jelin and Grossman, *47* (Odessa ground squirrel)). Bruynoghe and Jadin (*43*) reported the susceptibility of the meadow mouse (*Arvicola arvalis*) and the dwarf mouse (*Mus minutus*); Ronse (*48*) of hedgehogs and garden mice (lérot: *Myoxus nitela*). Dyer (*49*) reported susceptibility of mice (*Mus musculus musculus*), woodchucks (*Marmota monax monax*), meadow mice (*Microtus pennsylvanicus pennsylvanicus*), and white-footed mice (*Peromyscus leucopus noveboracensis*); Brigham (*50*) of opossums (*Didelphys virginiana*) and (*51*) of cats (*Felis domestica*), old-field mice (*Peromyscus polionotus polionotus*), and cotton mice (*Peromyscus gossypinus gossypinus*) with apparent infections, and wood rats (*Neatoma floridana rubida*), cotton rats (*Sigmodon hispidus hispidus*), rice rats (*Oryzomys palustris palustris*), and flying squirrels (*Glaucomys volans saturatus*) with inapparent infections. The infection in nature of the old-field mouse was reported by the same author (*52*). Brigham (*53*) further recorded inapparent infections in gray and fox squirrels (*Sciurus carolinensis carolinensis* and *Sciurus niger niger*), 1 of 4 swamp rabbits (*Sylvilagus aquaticus aquaticus*), a chipmunk

(*Tamias striatus striatus*), and a skunk (*Mephitis elongata*). Abortion and death on the fifth day with recovery of virus were recorded for one cottontail rabbit (*Sylvilagus floridanus mallurus*). The gray fox (*Urocyon cinereoargenteus cinereoargenteus*) was recorded as insusceptible.

Among all these susceptible species histologic lesions in the brain have been reported, aside from the guinea pig, only in the Macedonian spermophile (*44*) or Odessa ground squirrel (*46*), and in the cat (*80*). In the latter, Lépine and Lorando noted a "minimal reaction," a meningeal reaction predominantly in septa and a slight subcortical infiltration. The reaction was mononuclear ("monocytaire"). No typhus nodes or vasculitis were observed. Lépine's Macedonian spermophiles showed cerebral congestion with perivascular infiltration. Jelin and Grossman described a perivascular lymphocyte and plasma cell infiltration and typhus "nodes" in the brain of the Odessa ground squirrel after 6 to 7 days of fever. Similar lesions appeared earlier in liver, lung, and spleen, and later in the heart.

Following Brigham's demonstration of the susceptibility and occasional natural infection of the old-field mouse (*Peromyscus polionotus polionotus*) (*51*, *52*), a series of these mice was inoculated with the Wilmington strain of guinea pig passage endemic typhus virus and killed when the simultaneously inoculated guinea pigs reached the eleventh day of fever, at which time the brain reaction was expected to be at its maximum in the guinea pigs (Lillie and Dyer, *25*). When brain lesions were found in 6 of 11 of these captured wild mice, it was determined to explore the brain reaction in other species of susceptible native rodents.

Typhuslike paravascular glia nodes and intracerebral vasculitis of mixed proliferative and perivascular exudative type were encountered in 6 of 11 old-field mice (*Peromyscus polionotus polionotus*), 5 of 7 white-footed mice (*Peromyscus leucopus noveboracensis*), both of 2 *Peromyscus eremicus eremicus*, all of 7 deer mice (*Peromyscus maniculatus gambelii*), none of 4 cotton mice (*Peromyscus gossypinus gossypinus*), both of 2 *Reithrodontomys* sp., all of 7 gray mice (*Mus musculus musculus*), all of 9 white mice (*Mus musculus musculus albinus*), and 7 of 12 white rats (*Rattus norvegicus albinus*).

The time of the killing of these mice for study of the brain reaction was based on the supposition that the incubation period and the evolution of lesions would be similar to the process in guinea pigs. To test this hypothesis further, a series of 32 white mice was inoculated with guinea pig testicular washings of the Wilmington strain and 4 were killed each day 8, 11, 12, 14, 16, 18, and 22 days later.

Five transverse sections of each of the 32 brains were made through the frontal area, the thalamic area, the midbrain, the pons and cerebellum, and the enlargement of the medulla. The total number of focal

lesions for each mouse were counted in these sections, and the totals averaged for each day. It was found that the maximum average counts were obtained on the fourteenth and sixteenth days.

TABLE 1.—*Variation of number of focal lesions in mouse brains according to the length of time after inoculation with endemic typhus*

Day killed	Individual counts	Aggregate	Average
8	1, 10, 4, 3	18	4.50
11	0, 47, 31, 1	79	19.75
12	10, 26, 16, 69	121	30.25
14	196, 24, 48, 11	279	69.75
16	12, 149, 24, 31	214	53.50
18	75, 4, 2, 37	115	28.75
20	1, 4, 1, 61	67	16.75
22	3, 3, 22, 5	33	8.25

This corresponds approximately to the estimate based on the reactions in guinea pigs.

On comparing the detailed pathology in white mice with that in guinea pigs (*25*), it appears that vascular lesions, and particularly proliferative vascular lesions, are relatively more frequent in *M. musculus*, and the paravascular glia nodes less frequent, that focal lesions are relatively less numerous in cerebral cortex and more numerous in midbrain and hindbrain, that the great preponderance of nodes in the cerebellar cortex in guinea pigs is replaced by a preponderance of vascular lesions in mice, that in place of a preponderance of vascular lesions in the corpora striata the type distribution of lesions in mice is about average. These findings are given in table 2. Among 93 sections of chorioid plexus shown, 13 presented slight lymphocyte infiltration; 80 were negative. Meninges usually showed slight lymphocyte infiltration. Capillary thrombosis was recorded once.

The 10 gray mice (*M. musculus musculus*) studied showed an even greater preponderance of vascular lesions (57.3 percent perivascular lymphocyte infiltration, 24.4 percent endothelial proliferative, and only 18.3 percent paravascular glia nodes), and greater shift in lesion distribution toward the hindbrain at the expense of the cerebral cortical lesions. Chorioid plexal infiltration was rare, meningeal lymphocyte infiltration slight, and capillary thrombosis absent.

The white rats (*Rattus norvegicus albinus*) were inoculated during July 1939, and killed on the fourteenth and sixteenth days. As expected from the behavior of the disease in guinea pigs during the summer, lesions were few or often absent. However, some typical paravascular glia cell nodules were seen, perivascular lymphocyte infiltration was present, and the most prominent intracerebral lesion was a concentric proliferation of fusiform adventitia cells of scattered small vessels, compressing or obliterating the lumen. Lesions tended to occur most often in midbrain and hindbrain. Chorioid plexal

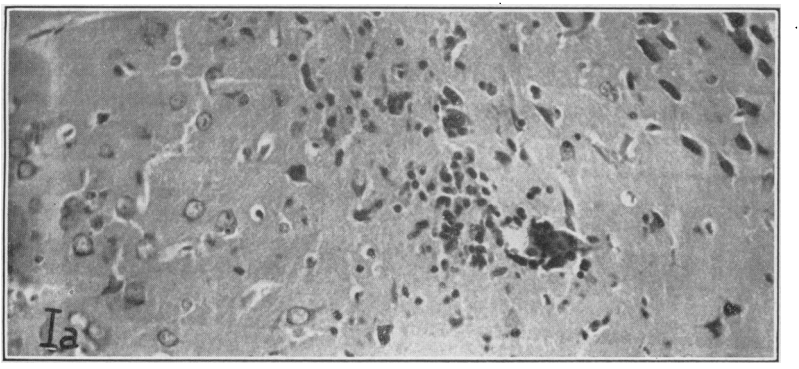

FIGURE 1a.—Endemic typhus, node and vascular lesions, temporal cortex, white mouse 14702. (× 435.)

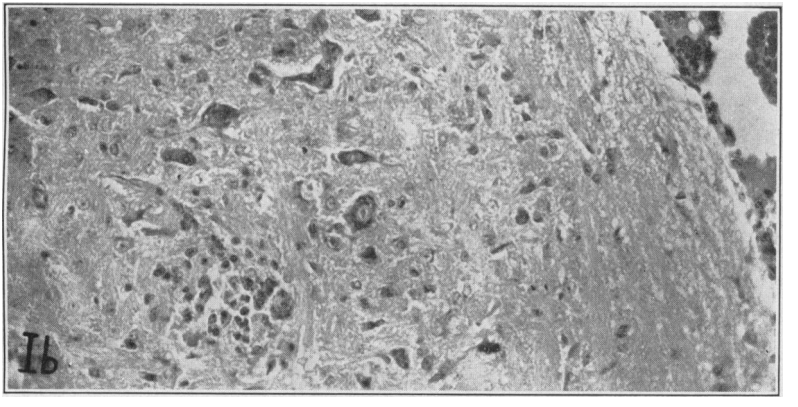

FIGURE 1b.—Endemic typhus, node, medulla, white mouse 14676. (× 435.)

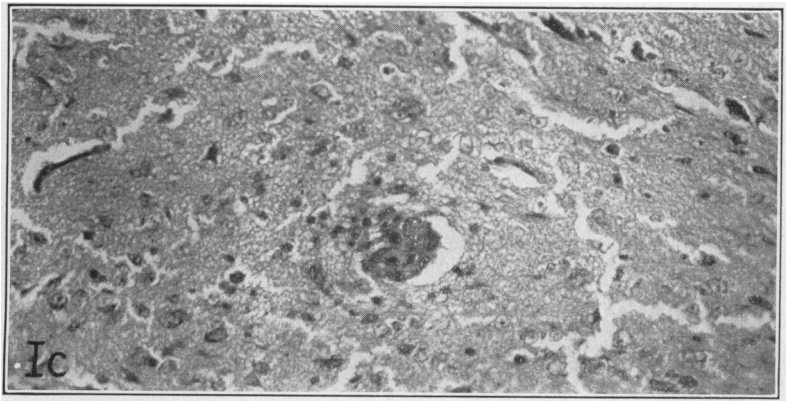

FIGURE 1c.—Endemic typhus, concentric vascular proliferation, thalamus, white mouse 14676. (× 435.)

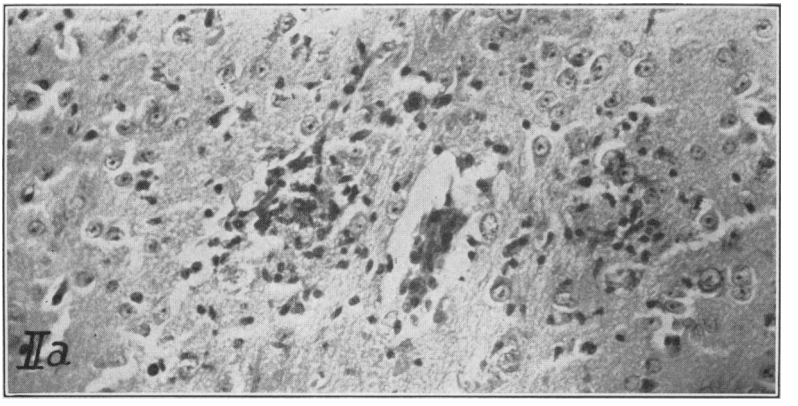

FIGURE 2a.—Spotted fever, node and vascular lesion, thalamus, white mouse 14326. (× 435.)

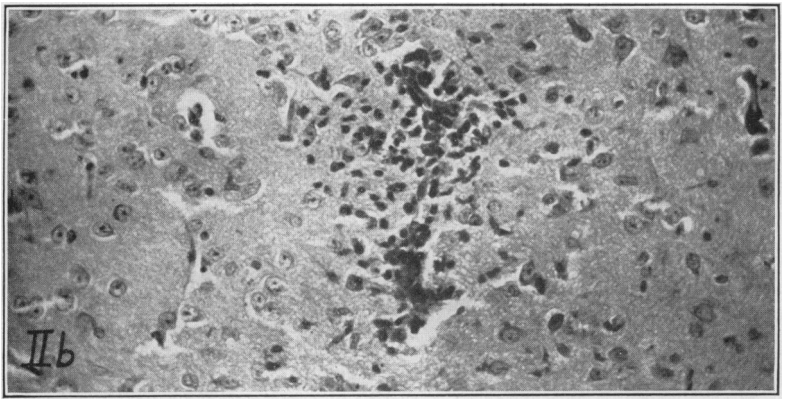

FIGURE 2b.—Spotted fever, node and vessel, hypothalamus, white mouse 14326. (× 435.)

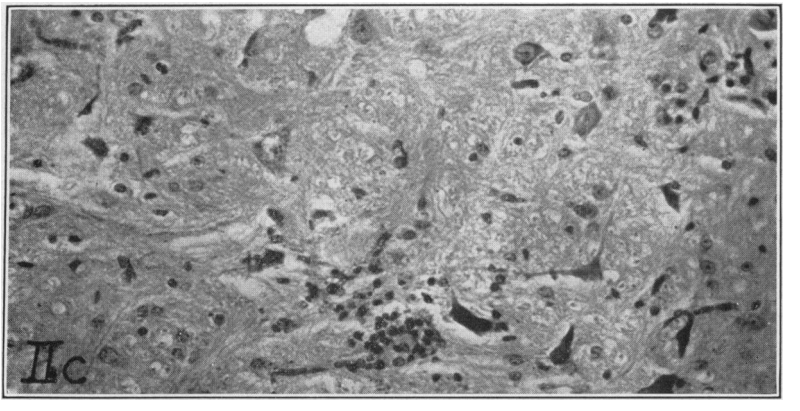

FIGURE 2c.—Spotted fever, concentric vascular proliferation, medulla, white mouse 14327. (× 435.)

infiltration was absent, meningeal reaction very slight, and capillary thrombosis was not observed. Rats killed on the sixteenth day showed more lesions than those killed on the fourteenth day.

TABLE 2.—*Topographic distribution and types of brain lesions of endemic typhus in guinea pigs and various species of mice*

Species and type of lesion	Aggregate number of focal lesions										
	Cerebral cortex				Cerebral nuclei		Midbrain and hindbrain				Total
	Frontal	Parietal	Temporal	Hippocampus	Corpora striata	Thalamus	Midbrain	Pons	Medulla	Cerebellum	
Guinea pigs (391), ref. (*25*):											
Perivascular lymphocyte	1,083	734	222	520	571	509	262	259	215	76	4,451
Proliferative vascular	319	184	48	106	66	141	115	56	78	66	1,179
Total vascular	1,402	918	270	626	637	650	377	315	293	142	5,630
Glia nodes	1,002	854	328	485	171	410	378	288	280	324	4,520
Total focal	2,404	1,772	598	1,111	808	1,060	755	603	573	466	10,150
White mice (30):											
Perivascular lymphocyte	99	39	23	35	36	41	37	39	22	76	447
Proliferative vascular	44	21	14	13	20	23	11	9	5	11	171
Total vascular	143	60	37	48	56	64	48	48	27	87	618
Glia nodes	79	29	18	8	27	21	22	25	14	11	254
Total focal	222	89	55	56	83	85	70	73	41	98	872
Peromyscus polionotus polionotus (5):											
Perivascular lymphocyte	10	2	1	16	4	1	6	6	0	0	46
Proliferative vascular	4	1	0	5	0	2	5	3	0	0	20
Total vascular	14	3	1	21	4	3	11	9	0	0	66
Glia nodes	17	6	4	13	2	3	8	9	0	4	66
Total focal	31	9	5	34	6	6	19	18	0	4	132
Peromyscus maniculatus gambelii (7):											
Perivascular lymphocyte	31	9	5	14	14	44	15	17	7	8	164
Proliferative vascular	16	9	1	8	4	11	5	1	5	2	62
Total vascular	47	18	6	22	18	55	20	18	12	10	226
Glia nodes	55	29	12	24	15	36	20	18	12	16	237
Total focal	102	47	18	46	33	91	40	36	24	26	463
Peromyscus leucopus noveboracensis (5):											
Perivascular lymphocyte	9	9	1	2	3	6	10	6	13	0	59
Proliferative vascular	16	7	5	4	2	7	8	7	7	3	66
Total vascular	25	16	6	6	5	13	18	13	20	3	125
Glia nodes	31	17	10	14	5	21	36	24	17	9	184
Total focal	56	33	16	20	10	34	54	37	37	12	309

TABLE 2.—*Topographic distribution and types of brain lesions of endemic typhus in guinea pigs and various species of mice*—Continued

Species and type of lesion	Percentages of focal lesions										
	Cerebral cortex				Cerebral nuclei		Midbrain and hindbrain				
	Frontal	Parietal	Temporal	Hippocampus	Corpora striata	Thalamus	Midbrain	Pons	Medulla	Cerebellum	Total
Guinea pigs (391), ref. (*85*):											
Perivascular lymphocyte	10.7	7.2	2.2	5.1	5.6	5.0	2.6	2.6	2.1	0.8	43.8
Proliferative vascular	3.1	1.8	0.5	1.0	0.7	1.4	1.1	0.5	0.8	0.6	11.6
Total vascular	13.8	9.0	2.7	6.1	6.3	6.4	3.7	3.1	2.9	1.4	55.4
Glia nodes	9.9	8.4	3.2	4.8	1.7	4.0	3.7	2.8	2.8	3.3	44.6
Total focal	23.7	17.5	5.9	10.9	8.0	10.4	7.4	5.9	5.6	4.7	100.0
White mice (30):											
Perivascular lymphocyte	11.4	4.5	2.6	4.0	4.1	4.7	4.2	4.5	2.5	8.5	51.2
Proliferative vascular	5.0	2.4	1.6	1.5	2.3	2.6	1.3	1.0	0.6	1.3	19.6
Total vascular	16.4	6.9	4.2	5.5	6.4	7.3	5.5	5.5	3.1	9.8	70.9
Glia nodes	9.1	3.3	2.1	0.9	3.1	2.4	2.5	2.9	1.6	1.3	29.1
Total focal	25.5	10.2	6.3	6.4	9.5	9.7	8.0	8.4	4.7	11.2	100.0
Peromyscus polionotus polionotus (5):											
Perivascular lymphocyte	7.5	1.5	0.8	12.1	3.1	0.8	4.5	4.5	0	0	34.8
Proliferative vascular	3.1	0.8	0.0	3.8	0	1.5	3.8	2.3	0	0	15.2
Total vascular	10.6	2.3	0.8	15.9	3.1	2.3	8.3	6.8	0	0	50.0
Glia nodes	12.9	4.5	3.1	9.8	1.5	2.3	6.1	6.8	0	3.1	50.0
Total focal	23.5	6.8	3.8	25.7	4.5	4.5	14.4	13.7	0	3.1	100.0
Peromyscus maniculatus gambelii (7):											
Perivascular lymphocyte	6.7	1.9	1.1	3.0	3.0	9.5	3.2	3.7	1.5	1.7	35.4
Proliferative vascular	3.5	1.9	0.2	1.7	0.9	2.4	1.1	0.2	1.1	0.4	13.4
Total vascular	10.2	3.8	1.3	4.7	3.9	11.9	4.3	3.9	2.6	2.1	48.8
Glia nodes	11.9	6.3	2.6	5.2	3.2	7.8	4.3	3.9	2.6	3.4	51.2
Total focal	22.0	10.1	3.9	9.9	7.1	19.7	8.6	7.8	5.2	5.6	100.0
Peromyscus leucopus noveboracensis (5):											
Perivascular lymphocyte	2.9	2.9	0.3	0.6	1.0	1.9	3.2	1.9	4.2	0	19.1
Proliferative vascular	5.2	2.3	1.6	1.3	0.6	2.3	2.6	2.3	2.3	0.9	21.4
Total vascular	8.1	5.2	1.9	1.9	1.6	4.2	5.8	4.2	6.5	0.9	40.5
Glia nodes	10.0	5.5	3.2	4.5	1.6	6.8	11.7	7.8	5.5	2.9	59.5
Total focal	18.1	10.7	5.2	6.5	3.2	11.0	17.5	12.0	12.0	3.9	100.0

In old-field mice (*P. polionotus polionotus*), the type distribution of lesions is more like that in guinea pigs, the nodal type of lesion being somewhat more frequent and the perivascular lymphocyte infiltration less frequent. More of the vascular lesions are of the endothelial swelling and proliferation type. Capillary thrombosis was noted twice. The proportion of lesions occurring in the cerebral cortex is about the same as in the guinea pig, that in the cerebral nuclei less, and that in midbrain and hindbrain more, particularly in midbrain and pons. As in the guinea pig, vascular lesions predominate in the corpora striata and "nodes" in the cerebellum. Focal lymphocyte infiltration of chorioid plexus was not infrequent, and meningeal infiltration was regularly present.

In *P. maniculatus gambelii*, the frequency of the types of lesions is essentially similar to that in old-field mice, while the topographic

distribution is more like that in *M. musculus*, with about the same proportion of cortical lesions, more in basal nuclei and fewer in the hindbrain. Capillary thrombi were recorded 6 times. Meningeal lymphocyte infiltration was regularly present, and few foci of lymphocyte infiltration were seen in chorioid plexus.

In the white-footed mice (*P. leucopus noveboracensis*), the greatest concentration of lesions in midbrain and hindbrain is seen, and capillary endothelial swelling and proliferation are more frequent than vessel sheath lymphocyte infiltration. In this species the paravascular nodes comprise the highest proportion of the total focal lesions seen. Capillary thrombi were not recorded. Meningeal lymphocyte infiltration was usually present, chorioid plexal occasionally.

In the 2 *Peromyscus eremicus eremicus*, the brain reactions were scanty. The usual types of lesions, nodes, and vessels with sheath lymphocyte infiltration were present, and focal lymphocyte infiltration was noted in meninges and chorioid plexus.

In lesion types, the 2 *Reithrodontomys* sp. seemed similar to the *P. leucopus noveboracensis* in showing more "nodes" than vascular lesions and more endothelial swelling and proliferation than sheath lymphocyte infiltration. There was also some tendency to increased frequency in lesions in the hindbrain. Capillary thrombi were not seen, plexal infiltration was not noted, and meningeal lymphocyte infiltration was present.

Guinea pigs and rhesus monkeys were shown to be susceptible to Rocky Mountain spotted fever (Ricketts, *54*, King, *55*). Later Ricketts (*56*) found gophers, or ground squirrels, and horses susceptible. Rabbits had been inoculated by Wilson and Chowning in 1904, and their susceptibility was confirmed by Ricketts and Gomez (*58*) and Gomez (*59*). Ricketts (*60*) further reported susceptibility of ground hogs, rock squirrels, chipmunks, and mountain rats. McClintic (*61*) reported as susceptible the Columbian ground squirrel (*Citellus columbianus*), and 1 of 4 badgers (*Taxidea taxus*), and noted the infectivity of the blood of inoculated weasels (*Putorius arizonensis*) 5 but not 10 days after infection. Later (*62*) he reported the woodchuck (*Marmota flaviventris*) and the rock squirrel (*Callospermophilus lateralis cinerascens*) as susceptible. Coyotes and cats were resistant.

Rucker (*63*) listed the susceptible animals known in 1912, adding the wood rat (*Neotoma cinerea*) which had been suggested by Ricketts (*60*), and the cottontail rabbit (*Sylvilagus nuttalli*), and further classing the chipmunks as yellow bellied and white bellied (*Eutamias luteiventris* and *E. quadrivittatus umbrinus*). Fricks (*64*) noted that white rats (*Rattus norvegicus albinus*) were highly susceptible but white mice were resistant. Badger (*65*) recovered spotted fever virus from inoculated dogs (*Canis familiaris*) and a lamb (*Ovis aries*). Pups had fever, the lamb and adult dogs had none. Jellison (*66*) recovered

spotted fever virus up to the tenth day from meadow mice (*Microtus pennsylvanicus modestus*) and once from a dwarf mouse (*Microtus nanus*). He obtained similar results from deer mice (*Peromyscus maniculatus artemisiae*) but was unable to recover virus from inoculated gray mice (*Mus musculus*).

Travassos (*67*) reported 3 passages of São Paulo typhus virus through rats, and noted that Monteiro (*68*) had recovered this virus from wild and white rats after one passage.

In spite of the accumulation of reports of insusceptibility of *Mus musculus* to spotted fever (*64, 66*), we again tried this species but did not recover the virus in guinea pigs. Brain reactions were produced in some mice and not in others. In one lot of 7 mice inoculated with guinea pig passage virus and killed 16 days later, 2 showed no lesions, 1 showed a few, there were moderate reactions in 3, and in 1 the reaction was graded as ++. In a second lot of 5 mice, 2 showed no lesions, 2 slight reactions, and 1 a ++ reaction. In 2 mice inoculated with first passage mouse brain, marked reactions were obtained, but virus was not recovered in simultaneously inoculated guinea pigs. Brains of mice inoculated with mouse brain passage virus occasionally showed slight to moderate reactions up to the fifth mouse passage, but none thereafter. A total of 45 mouse brains was studied in this series.

Of the focal lesions 38.3 percent occurred in cerebral cortex, 20.2 percent in thalamus and basal ganglia, and 41.5 percent in midbrain and hindbrain. In typhus fever in white mice the corresponding figures are 48.4, 19.2, and 32.3 percent, showing the same type of distribution difference reported previously in guinea pigs.

As regards type, half (50.6 percent) of the lesions were perivascular lymphocyte infiltration, 12.8 percent endothelial and adventitial vascular proliferation, and 36.6 percent "nodes."

The nodes are fairly compact and well limited nodules of loosely packed, interstitially placed, almost naked, darkly staining, rounded and elongated, leptochromatic glia nuclei. Some nodules are composed of larger, apparently foamy, and more closely packed cells. The nodes are often evidently paravascular in location. Chorioid plexus was observed in 138 locations and in 13 showed slight, focal, or moderate lymphocyte infiltration. Meninges generally showed slight diffuse or focal lymphocyte infiltration.

To explore further the production of brain reactions to Bitterroot strains of spotted fever, a series of laboratory reared wild rodents was inoculated in July 1939, and killed usually 12 days later. Results are shown in table 3.

TABLE 3.—*Brain reactions in wild rodents inoculated with Bitterroot spotted fever virus and killed 12 days later*

Species	Number of mice	Number of lesions counted in 5 cross sections
White mouse (*Mus musculus albinus*)	6	0, 0, 3, 0, 1, 0.
White rat (*Rattus norvegicus albinus*)	6	0, 0, 2, 0, 1, 1.
Cotton rat (*Sigmodon hispidus hispidus*)	5	0, 0, 0, 2, 0.
Desert mouse (*Peromyscus eremicus eremicus*)	5	0, 0, 1, 0, 0.
Cotton mouse (*Peromyscus gossypinus gossypinus*)	6	0, 0, 0, 0, 1, 12.
Deer mouse (*Peromyscus maniculatus gambelii*)	6	0, 0, 0, 0, 0, 0.
Old-field mouse (*Peromyscus polionotus polionotus*)	5	0, 0, 0, 0, 0.
White-footed mouse (*Peromyscus leucopus noveboracensis*)	5	0, 0, 0, 1, 0.
Meadow mouse (*Microtus pennsylvanicus pennsylvanicus*)	5	6, 3, 5, 5, 3.

Reactions were negligible or absent except in one cotton mouse and in the meadow mice. Since these inoculations were made in July and it has been shown that endemic typhus gives its minimum brain reactions in guinea pigs during the hot months (*69*), it would seem advisable to repeat this experiment later in the year.

It is interesting to observe that the brain lesions in *Microtus* were predominantly in the midbrain and hindbrain (16 lesions, 4 in cerebral cortex, 2 in thalamus and basal ganglia). Of the 22 lesions, 2 were perivascular lymphocyte infiltration, 8 were vascular endothelial and adventitial swelling and proliferation, and 12 were "nodes." Meningeal lymphocyte infiltration and vascular proliferative reactions were scanty, and chorioid plexal involvement was absent.

CONCLUSION

Characteristic typhus nodes and vascular lesions are produced in the brain in endemic typhus fever in the old-field mouse (*Peromyscus polionotus polionotus*), the white-footed mouse (*P. leucopus noveboracensis*), in the desert mouse (*P. eremicus eremicus*), in the deer mouse (*P. maniculatus gambelii*), in *Reithrodontomys* sp., in gray and white mice (*Mus musculus*), and in the white rat (*Rattus norvegicus albinus*). No lesions were seen in inoculated cotton mice (*Peromyscus gossypinus gossypinus*). The lesions in these rodents are comparable to those seen in guinea pigs, but vary in proportion of lesion types and in distribution from species to species.

In Rocky Mountain spotted fever similar lesions are irregularly and inconstantly, but rather frequently, produced in white mice, and scanty reactions are seen in meadow mice (*Microtus pennsylvanicus pennsylvanicus*) and in an occasional cotton mouse (*Peromyscus gossypinus gossypinus*).

The increase in relative frequency of midbrain and hindbrain lesions noted in guinea pigs in spotted fever as compared with typhus is noted also in white mice. The greatest number of brain lesions in

endemic typhus in white mice are seen 14 to 16 days after inoculation, which corresponds closely to the time of maximum cerebral reaction in guinea pigs.

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DEATHS DURING WEEK ENDED NOVEMBER 11, 1939

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

	Week ended Nov. 11, 1939	Corresponding week, 1938
Data from 88 large cities of the United States:		
Total deaths	7,704	7,362
Average for 3 prior years	[1] 7,874	
Total deaths, first 45 weeks of year	370,242	363,975
Deaths under 1 year of age	432	420
Average for 3 prior years	[1] 496	
Deaths under 1 year of age, first 45 weeks of year	22,339	23,564
Data from industrial insurance companies:		
Policies in force	66,569,616	68,295,010
Number of death claims	9,407	7,752
Death claims per 1,000 policies in force, annual rate	7.4	5.9
Death claims per 1,000 policies, first 45 weeks of year, annual rate	9.9	9.2

[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 November 18, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median
NEW ENG.												
Maine	12	2	6	2	6	1	3	1	115	19	46	28
New Hampshire	0	0	0	0	----	----	----	----	41	4	0	3
Vermont	0	0	0	1	----	----	----	----	550	41	2	3
Massachusetts	11	9	5	8	----	----	----	----	323	275	177	82
Rhode Island	0	0	0	1	----	----	----	----	443	58	0	2
Connecticut	0	0	4	3	----	----	3	4	134	45	62	52
MID. ATL.												
New York [2]	8	19	24	25	[1] 8	[1] 11	[1] 11	[1] 11	60	149	315	315
New Jersey	32	27	13	17	19	16	10	9	20	17	18	41
Pennsylvania	35	69	54	54	----	----	----	----	20	39	66	69
E. NO. CEN.												
Ohio	37	48	46	57	26	34	----	32	21	27	15	63
Indiana	31	21	13	39	1	1	3	23	40	27	18	18
Illinois	26	39	46	46	7	10	27	22	18	28	32	32
Michigan [3]	6	6	29	29	----	----	----	1	169	160	54	46
Wisconsin	0	0	2	3	49	28	33	33	62	35	98	56
W. NO. CEN.												
Minnesota	0	0	7	7	----	----	2	1	169	87	156	45
Iowa	6	3	24	13	----	----	3	3	34	17	50	5
Missouri	19	15	29	15	----	----	4	41	12	9	7	31
North Dakota	7	1	9	5	37	5	4	4	15	2	389	11
South Dakota	8	1	3	3	23	3	2	----	38	5	43	4
Nebraska	8	2	6	6	----	----	1	----	8	2	1	3
Kansas	11	4	13	26	11	4	8	5	190	68	11	11
SO. ATL.												
Delaware	20	1	1	1	----	----	----	----	0	0	2	2
Maryland [3]	22	7	14	21	22	7	7	5	6	2	56	28
Dist. of Col	16	2	11	11	----	----	2	1	8	1	2	1
Virginia	127	68	85	72	167	89	118	----	21	11	37	37
West Virginia	40	15	12	35	35	13	10	21	5	2	17	23
North Carolina [4]	171	117	117	80	7	5	7	7	150	103	194	94
South Carolina [4]	66	24	16	15	1,306	478	284	284	14	5	4	6
Georgia [4]	48	29	14	41	196	118	31	----	15	9	13	0
Florida [4]	24	8	12	15	9	3	3	3	12	4	32	6

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended November 18, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median
E. SO. CEN.												
Kentucky	28	16	34	34	111	64	21	15	7	4	12	12
Tennessee [4]	60	34	22	50	67	38	38	38	16	9	6	6
Alabama [4]	95	54	31	44	326	185	55	55	7	4	12	6
Mississippi [3]	46	18	14	14	------	------	------	------	------	------	------	------
W. SO. CEN.												
Arkansas	57	23	29	16	134	54	69	13	7	3	15	1
Louisiana [4]	22	9	18	25	24	10	3	4	2	1	50	8
Oklahoma	30	15	31	25	68	34	57	42	10	5	19	4
Texas [4]	38	46	84	61	205	247	220	127	12	14	5	14
MOUNTAIN												
Montana	9	1	1	1	1,236	132	------	3	206	22	113	23
Idaho	0	0	0	0	------	------	------	3	10	1	55	7
Wyoming	22	1	0	0	------	------	------	------	196	9	4	4
Colorado	24	5	16	9	63	13	22	------	221	46	11	11
New Mexico	12	1	6	6	12	1	------	------	86	7	3	18
Arizona	86	7	4	4	773	63	116	41	37	3	2	2
Utah [3]	0	0	3	2	50	5	6	------	864	87	7	13
PACIFIC												
Washington	3	1	8	1	------	------	2	------	811	263	15	30
Oregon	10	2	3	2	89	18	11	27	94	19	8	16
California [4]	26	32	34	51	17	21	33	34	133	162	449	50
Total	32	802	953	1,064	81	1,711	1,229	970	77	1,910	2,703	2,703
46 weeks	18	20,388	25,447	25,447	165	160,713	56,018	110,893	316	359,527	775,362	687,530

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median
NEW ENG.												
Maine	0	0	0	0	0	0	0	0	48	8	4	20
New Hampshire	0	0	0	0	0	0	0	0	0	0	7	7
Vermont	0	0	0	0	0	0	1	0	0	0	4	7
Massachusetts	0	0	0	2	2.4	2	0	2	96	82	72	125
Rhode Island	0	0	1	0	0	0	0	0	23	3	5	12
Connecticut	0	0	0	0	0	0	0	0	104	35	42	38
MID. ATL.												
New York [3]	0	0	3	5	7	18	2	7	94	236	249	288
New Jersey	0	0	0	1	6	5	2	2	123	103	85	85
Pennsylvania	2	4	6	2	8	15	4	3	207	408	312	340
E. NO. CEN.												
Ohio	0	0	0	4	5	7	0	0	250	325	249	270
Indiana	0	0	1	1	1.5	1	0	1	248	167	150	161
Illinois	2.6	4	1	4	3	5	1	3	197	300	287	355
Michigan [3]	0	0	0	2	6	6	1	5	303	287	423	252
Wisconsin	0	0	0	0	9	5	1	1	206	117	123	203
W. NO. CEN.												
Minnesota	0	0	0	1	8	4	0	2	196	101	84	121
Iowa	0	0	0	1	24	12	0	2	105	52	70	70
Missouri	1.3	1	0	1	0	0	2	2	87	68	114	114
North Dakota	0	0	0	0	0	0	0	0	256	35	24	43
South Dakota	0	0	0	0	8	1	0	0	150	20	34	34
Nebraksa	0	0	0	0	11	3	0	1	65	17	25	33
Kansas	0	0	0	0	0	0	0	1	254	91	135	118

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended November 18, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median
SO. ATL.												
Delaware	0	0	0	0	0	0	1	0	177	9	9	7
Maryland [3]	0	0	0	1	6	2	0	1	139	45	40	80
Dist. of Col.	0	0	0	3	24	3	0	0	49	6	10	12
Virginia	4	2	3	3	0	0	2	1	101	54	60	60
West Virginia	0	0	0	1	19	7	0	0	218	81	86	102
North Carolina [4]	2.9	2	1	2	4	3	1	1	206	141	72	72
South Carolina [4]	0	0	1	1	2.7	1	2	0	85	31	13	12
Georgia [4]	0	0	1	0	0	0	1	1	70	42	38	32
Florida [4]	3	1	1	1	0	0	1	1	48	16	22	2
E. SO. CEN.												
Kentucky	1.7	1	6	5	16	9	1	2	149	86	96	69
Tennessee [4]	0	0	2	3	1.8	1	1	1	125	71	91	91
Alabama [4]	4	2	5	4	5	3	0	1	95	54	26	27
Mississippi [3]	0	0	0	0	5	2	1	1	43	17	11	13
W. SO. CEN.												
Arkansas	2.5	1	1	0	5	2	1	1	52	21	29	19
Louisiana [4]	2.4	1	0	1	0	0	0	2	34	14	26	20
Oklahoma	0	0	0	0	2	1	1	1	46	23	46	23
Texas [4]	0.8	1	0	1	5	6	1	2	42	51	97	66
MOUNTAIN												
Montana	0	0	1	0	0	0	0	0	318	34	26	32
Idaho	20	2	0	0	20	2	1	0	61	6	13	21
Wyoming	0	0	0	0	0	0	0	0	109	5	5	15
Colorado	0	0	0	0	0	0	0	4	207	43	28	42
New Mexico	12	1	0	0	37	3	0	0	136	11	20	23
Arizona	0	0	0	0	0	0	0	0	110	9	5	17
Utah [3]	0	0	0	0	60	6	0	0	149	15	12	31
PACIFIC												
Washington	3	1	0	0	3	1	0	1	111	36	43	43
Oregon	5	1	1	1	5	1	0	3	80	16	42	39
California [4]	2.5	3	1	1	21	26	1	12	147	179	209	209
Total	1.1	28	36	63	6	163	30	91	142	3,571	3,673	4,276
46 weeks	1.5	1,758	2,589	4,930	6	[5] 6,802	1,596	6,962	121	140,137	164,148	195,700

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases
NEW ENG.											
Maine	0	0	0	0	12	2	1	1	217	36	19
New Hampshire	0	0	0	0	0	0	0	0	41	4	0
Vermont	0	0	0	0	0	0	0	1	791	59	0
Massachusetts	0	0	0	0	1	1	1	1	158	134	134
Rhode Island	0	0	0	0	0	0	2	1	191	25	41
Connecticut	0	0	0	0	6	2	4	2	214	72	65
MID. ATL.											
New York [3]	0	0	0	0	2	5	8	11	141	352	659
New Jersey	0	0	0	0	2	2	1	5	188	158	394
Pennsylvania	0	0	0	0	5	10	31	23	160	315	438

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended November 18, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough		
	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases	1934–38, median	Nov. 18, 1939, rate	Nov. 18, 1939, cases	Nov. 19, 1938, cases
E. NO. CEN.											
Ohio	2	2	1	1	8	10	12	11	188	245	152
Indiana	1	1	10	2	4	3	1	1	64	43	14
Illinois	1	1	2	2	5	8	16	15	131	200	501
Michigan [3]	11	10	14	1	2	2	8	8	118	112	295
Wisconsin	5	3	2	2	0	0	1	1	239	136	435
W. NO. CEN.											
Minnesota	25	13	4	4	0	0	0	1	81	42	37
Iowa	32	16	3	3	4	2	3	3	12	6	29
Missouri	3	2	41	4	15	12	2	5	13	10	22
North Dakota	0	0	12	10	0	0	3	2	95	13	8
South Dakota	15	2	0	2	0	0	1	1	0	0	5
Nebraska	0	0	0	0	8	2	1	1	19	5	18
Kansas	3	1	3	2	8	3	1	5	48	17	20
SO. ATL.											
Delaware	0	0	0	0	39	2	0	2	354	18	6
Maryland [3]	0	0	0	0	12	4	5	8	148	48	34
Dist. of Col	0	0	0	0	16	2	1	1	89	11	9
Virginia	0	0	0	0	15	8	7	7	43	23	44
West Virginia	0	0	0	0	19	7	6	7	30	11	36
North Carolina [4]	1	1	0	0	1	1	10	4	114	78	272
South Carolina [4]	0	0	0	0	38	14	3	3	16	6	35
Georgia [4]	2	1	0	0	17	10	13	6	23	14	15
Florida [4]	0	0	0	0	9	3	6	3	33	11	17
E. SO. CEN.											
Kentucky	0	0	10	0	9	5	12	14	101	58	26
Tennessee [4]	0	0	1	1	2	1	5	11	115	65	23
Alabama [4]	2	1	0	0	4	2	3	6	25	14	44
Mississippi [3]	0	0	0	0	8	3	1	4	----	----	----
W. SO. CEN.											
Arkansas	0	0	1	0	25	10	3	4	37	15	15
Louisiana [4]	0	0	0	1	17	7	18	11	12	5	8
Oklahoma	4	2	4	1	6	3	13	11	0	0	7
Texas [4]	0	0	0	1	12	14	32	32	46	55	77
MOUNTAIN											
Montana	0	0	2	8	9	1	6	4	19	2	36
Idaho	10	1	0	0	20	2	8	2	0	0	2
Wyoming	22	1	0	2	0	0	0	0	22	1	1
Colorado	5	1	1	3	10	2	1	0	111	23	43
New Mexico	0	0	0	0	12	1	5	9	395	32	9
Arizona	0	0	1	0	25	2	6	1	61	5	2
Utah [3]	0	0	1	0	10	1	1	0	616	62	25
PACIFIC											
Washington	3	1	1	10	9	3	5	3	83	27	63
Oregon	0	0	8	0	30	6	3	3	109	22	1
California [4]	1	1	2	1	15	18	9	9	92	112	108
Total	2	61	124	124	8	196	279	279	109	2,702	4,244
46 weeks	8	9,062	13,395	6,579	10	11,922	13,404	14,121	138	157,405	187,136

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended Nov. 18, 1939, New York, 1 case.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended Nov. 18, 1939, 64 cases as follows: North Carolina, 1; South Carolina, 6; Georgia, 30; Florida, 2; Tennessee, 3; Alabama, 10; Louisiana, 2; Texas, 8; California, 2.
[5] Diagnosis was changed on 1 case reported as poliomyelitis in Pennsylvania during the week ended October 14, Public Health Reports of October 27, p. 1939.

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	Diphtheria	Influenza	Malaria	Measles	Meningitis, meningococcus	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid and paratyphoid fever
October 1939										
Alabama	146	129	1,036	16	10	25	4	179	0	20
Arizona	13	207	2	8	1	1	9	22	0	9
Colorado	44	36	1	57	2	----	40	102	20	23
Idaho	0	1	----	17	1	----	8	27	1	8
Kentucky	81	11	9	36	4	1	50	231	0	49
Maryland	38	35	1	20	1	4	10	160	0	30
Minnesota	17	9	----	28	1	----	115	164	5	2
Nebraska	3	----	----	14	1	----	6	49	1	1
New Mexico	8	4	1	6	0	1	43	35	0	30
New York	50	----	11	351	3	----	235	481	0	74
Pennsylvania	97	----	1	117	16	----	120	794	0	58
South Dakota	9	13	----	100	0	----	11	87	1	1
Tennessee	135	38	133	26	3	10	2	221	0	38
Vermont	1	----	----	65	0	----	15	22	0	0

October 1939

	Cases
Botulism:	
New York	1
Chickenpox:	
Alabama	12
Arizona	19
Colorado	90
Idaho	45
Kentucky	112
Maryland	93
Minnesota	236
Nebraska	12
New Mexico	26
New York	721
Pennsylvania	1,216
South Dakota	28
Tennessee	39
Vermont	141
Diarrhea:	
Maryland	32
New Mexico	3
Dysentery:	
Alabama (amoebic)	3
Arizona	97
Colorado (bacillary)	3
Kentucky (amoebic)	1
Kentucky (bacillary)	17
Maryland (bacillary)	31
Maryland (unspecified)	14
Minnesota (amoebic)	3
Minnesota (bacillary)	1
New Mexico (amoebic)	3
New Mexico (bacillary)	14
New York (amoebic)	6
New York (bacillary)	92
Pennsylvania (bacillary)	5
Tennessee (amoebic)	1
Tennessee (bacillary)	13
Encephalitis, epidemic or lethargic:	
Alabama	4
Arizona	11
Colorado	7
Maryland	1
Minnesota	1
New Mexico	2
New York	10
Pennsylvania	2
Tennessee	1
German measles:	
Alabama	1
Arizona	1

October 1939—Continued

	Cases
German measles—Con.	
Idaho	3
Kentucky	1
New Mexico	1
New York	49
Pennsylvania	32
Tennessee	3
Hookworm disease:	
Kentucky	8
Tennessee	2
Impetigo contagiosa:	
Maryland	34
Tennessee	15
Leprosy:	
Minnesota	1
New Mexico	1
Mumps:	
Alabama	27
Arizona	26
Colorado	29
Idaho	2
Kentucky	27
Maryland	13
Nebraska	11
New Mexico	13
Pennsylvania	325
South Dakota	8
Tennessee	19
Vermont	14
Ophthalmia neonatorum:	
Maryland	1
New York [1]	8
Pennsylvania	4
Tennessee	4
Puerperal septicemia:	
New Mexico	1
Tennessee	3
Rabies in animals:	
Alabama	11
New Mexico	1
New York [1]	19
Rocky Mountain spotted fever:	
Maryland	1
Septic sore throat:	
Colorado	4
Idaho	1
Kentucky	45
Maryland	12
Minnesota	14
Nebraska	3
New Mexico	13

October 1939—Continued

	Cases
Septic sore throat—Con.	
New York	52
Tennessee	13
Tetanus:	
Alabama	5
Kentucky	3
New York	7
Trachoma:	
Arizona	62
Trichinosis:	
New York	8
Tularaemia:	
Maryland	1
Minnesota	3
New Mexico	2
Tennessee	1
Typhus fever:	
Alabama	44
New York	2
Tennessee	36
Undulant fever:	
Alabama	4
Arizona	2
Colorado	1
Idaho	1
Kentucky	2
Maryland	3
Minnesota	6
New Mexico	2
New York	18
Pennsylvania	5
Tennessee	5
Vermont	1
Vincent's infection:	
Kentucky	7
Maryland	12
New York [1]	30
South Dakota	1
Tennessee	8
Whooping cough:	
Alabama	116
Arizona	42
Colorado	37
Idaho	8
Kentucky	187
Maryland	173
Minnesota	252
Nebraska	14
New Mexico	78
New York	1,122
Pennsylvania	1,116
South Dakota	13
Tennessee	142
Vermont	138

[1] Exclusive of New York City.

WEEKLY REPORTS FROM CITIES

City reports for week ended November 11, 1939

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	239	110	33	507	503	1,036	6	333	38	1,013	----
Current week[1]	125	84	33	283	326	681	0	272	51	771	----
Maine:											
Portland	0	----	0	2	1	0	0	1	0	11	26
New Hampshire:											
Concord	0	----	0	0	0	0	0	0	0	0	3
Manchester	0	----	0	0	0	0	0	0	0	0	14
Nashua	0	----	0	0	0	0	0	0	0	0	8
Vermont:											
Barre	0	----	0	0	0	0	0	0	0	0	2
Burlington	0	----	0	0	0	0	0	0	0	3	11
Rutland	0	----	0	0	0	0	0	0	0	0	11
Massachusetts:											
Boston	1	----	0	8	6	19	0	4	0	40	174
Fall River	1	----	0	0	0	0	0	0	0	12	30
Springfield	0	----	0	1	1	0	0	0	0	8	29
Worcester	1	----	0	2	2	0	0	1	0	10	44
Rhode Island:											
Pawtucket	0	----	0	0	0	1	0	0	0	0	14
Providence	0	1	1	33	1	5	0	3	1	20	53
Connecticut:											
Bridgeport	0	----	0	1	0	1	0	0	0	1	21
Hartford	0	----	0	1	0	1	0	1	0	34	39
New Haven	0	----	0	0	1	2	0	0	0	2	33
New York:											
Buffalo	0	----	1	5	7	12	0	4	0	0	116
New York	16	7	1	7	49	45	0	53	10	107	1,356
Rochester	0	1	0	0	6	2	0	1	0	12	74
Syracuse	0	----	0	0	2	1	0	0	0	2	35
New Jersey:											
Camden	0	----	0	0	1	4	0	1	0	2	23
Newark	1	1	0	2	3	8	0	1	0	22	95
Trenton	0	1	0	0	3	3	0	1	0	2	39
Pennsylvania:											
Philadelphia	1	4	2	2	19	33	0	21	3	54	430
Pittsburgh	4	2	2	1	10	32	0	7	0	8	168
Reading	2	----	0	0	0	1	0	1	0	2	30
Scranton	1	----	----	1	----	1	0	----	0	0	----
Ohio:											
Cincinnati	10	----	0	1	7	10	0	4	0	7	128
Cleveland	0	13	0	4	9	33	0	8	1	43	157
Columbus	10	2	2	1	5	2	0	2	0	0	84
Toledo	1	1	0	6	3	10	0	4	1	7	68
Indiana:											
Anderson	0	----	0	0	0	3	0	0	0	2	8
Fort Wayne	1	----	0	0	1	3	0	0	0	3	30
Indianapolis	3	----	3	6	6	19	0	2	0	15	100
South Bend	0	----	0	0	0	0	0	0	0	1	17
Terre Haute	0	----	0	0	1	2	0	0	0	0	17
Illinois:											
Alton	0	----	0	0	0	1	0	0	0	0	8
Chicago	7	6	2	9	35	87	0	27	0	59	669
Elgin	0	----	0	0	1	2	0	0	0	1	16
Moline	0	----	0	0	0	4	0	0	0	0	5
Springfield	----	----	----	----	----	----	----	----	----	----	----
Michigan:											
Detroit	4	----	0	3	7	72	0	10	0	27	210
Flint	0	----	0	0	2	5	0	0	0	8	31
Grand Rapids	0	----	0	2	0	11	0	0	0	0	27
Wisconsin:											
Kenosha	0	----	0	0	0	3	0	0	0	2	3
Milwaukee	0	----	0	2	1	25	0	4	0	23	89
Racine	0	----	0	0	0	1	0	0	0	0	20
Superior	0	----	0	0	0	3	0	0	0	0	14
Minnesota:											
Duluth	0	----	0	6	1	1	0	0	0	0	19
Minneapolis	1	----	1	6	7	22	0	2	0	10	107
St. Paul	0	----	0	0	3	15	0	2	0	34	58

[1] Figures for Springfield, Ill., and Fargo estimated; reports not received.

City reports for week ended November 11, 1939—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								
Iowa:											
Cedar Rapids	0			2		1	0		0	2	
Davenport	2			1		7	0		0	0	
Des Moines	0		0	0	0	13	3	0	0	0	21
Sioux City	0			0		6	0		0	0	
Waterloo	0			0		5	0		0	0	
Missouri:											
Kansas City	0		1	3	3	10	0	4	1	0	89
St. Joseph	0		0	0	0	1	0	0	0	0	21
St. Louis	0		0	2	7	16	0	3	1	6	186
North Dakota:											
Fargo											
Grand Forks	0			2		1	0		0	0	
Minot	0		0	0	0	1	0	0	0	0	8
South Dakota:											
Aberdeen	0			0		5	0		0	0	
Sioux Falls	0		0	0	0	5	0	0	0	0	6
Nebraska:											
Lincoln	1			0		1	0		0	0	
Omaha	0		0	0	2	1	0	2	0	0	54
Kansas:											
Lawrence	0		0	0	0	0	0	0	0	0	1
Topeka	0		0	1	0	10	0	0	0	0	5
Wichita	0	1	0	11	2	1	0	0	0	1	28
Delaware:											
Wilmington	0		0	0	4	3	0	1	0	9	25
Maryland:											
Baltimore	3	4	0	2	11	1	0	9	1	35	221
Cumberland	0	1	1	0	0	4	0	0	0	0	9
Frederick	0		0	0	0	1	0	0	0	0	3
Dist. of Col.:											
Washington	5	2	2	0	9	14	0	12	4	7	166
Virginia:											
Lynchburg	3		0	0	1	2	0	1	0	3	14
Norfolk	0	1	0	0	1	1	0	1	1	1	24
Richmond	1		0	0	6	7	0	2	2	1	55
Roanoke	0		0	1	0	1	0	0	0	0	16
West Virginia:											
Charleston	0	1	0	0	5	2	0	0	1	0	25
Huntington	2			0		0	0		0	0	
Wheeling	0	1	0	1	0	5	0	0	2	0	10
North Carolina:											
Gastonia	0			0		1	0		1	0	
Raleigh	3		0	1	0	5	0	1	0	0	6
Wilmington	1		0	0	0	0	0	1	0	0	16
Winston-Salem	8		0	1	0	5	0	0	0	1	5
South Carolina:											
Charleston	2	12	0	0	0	2	0	2	2	0	22
Florence	5	13	0	2	1	3	0	0	0	2	8
Greenville	2		0	0	0	0	0	0	0	0	7
Georgia:											
Atlanta	4	7	1	1	6	6	0	5	0	0	69
Brunswick	0		0	0	1	0	0	0	0	4	7
Savannah	1		0	0	1	1	0	1	0	2	29
Florida:											
Miami	0		0	0	1	2	0	3	0	0	31
Tampa	1	1	1	0	1	1	0	0	1	3	24
Kentucky:											
Ashland	1		0	0	0	0	0	0	0	0	4
Covington	1		0	1	1	2	0	1	0	1	16
Lexington	0		0	0	0	1	0	0	0	0	16
Louisville	1	1	0	0	6	4	0	0	0	43	71
Tennessee:											
Knoxville	0		0	0	1	10	0	0	0	0	27
Memphis	0		0	0	2	9	0	3	0	13	62
Nashville	0		1	0	5	5	0	3	0	1	
Alabama:											
Birmingham	0	4	2	0	3	2	0	4	0	0	62
Mobile	1		0	3	1	5	0	1	0	0	22
Arkansas:											
Fort Smith	0	2		0		2	0		0	0	
Little Rock	0		0	0	2	1	0	0	0	0	
Louisiana:											
Lake Charles	0		0	0	1	0	0	0	1	0	10
New Orleans	0	3	4	0	13	6	0	9	0	18	123
Shreveport	0		0	0	5	0	0	3	0	0	36

City reports for week ended November 11, 1939—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								
Oklahoma:											
Oklahoma City	0	2	0	0	1	1	0	1	0	0	34
Tulsa	1			0		1	0		0	1	
Texas:											
Dallas	5	1	1	0	5	4	0	4	0	1	74
Forth Worth	0		0	0	1	3	0	0	0	2	36
Galveston	2		0	0	1	1	0	1	0	0	16
Houston	4		0	0	2	3	0	3	0	0	74
San Antonio	0		1	4	2	1	0	7	1	0	54
Montana:											
Billings	0	2	0	0	0	2	0	0	0	0	5
Great Falls	0		0	1	0	3	0	0	0	1	4
Helena	0		0	0	0	0	0	0	0	0	7
Missoula	0		0	0	0	0	0	0	0	0	9
Idaho:											
Boise	0		0	0	0	0	0	0	0	0	4
Colorado:											
Colorado Springs	0		0	0	2	1	0	4	0	0	13
Denver	6		0	3	2	4	0	2	0	5	74
Pueblo	0		0	0	1	0	0	0	0	0	8
New Mexico:											
Albuquerque	0		0	1	1	0	0	1	0	0	12
Utah:											
Salt Lake City	0		0	16	2	7	0	1	0	49	34
Washington:											
Seattle	1		0	12	2	3	0	6	0	1	83
Spokane	0		0	1	0	8	0	0	0	0	17
Tacoma	1		0	102	0	4	0	0	0	0	24
Oregon:											
Portland	0		0	3	4	7	0	2	0	4	69
Salem	0			1		0	0		0	0	
California:											
Los Angeles	7	4	3	8	8	25	0	10	20	11	311
Sacramento	2	1	0	1	2	2	0	2	0	0	34
San Francisco	1	1	0	3	10	4	0	8	0	6	141

State and city	Meningococcus meningitis		Poliomyelitis cases	State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Massachusetts:				Iowa:			
Fall River	0	1	0	Des Moines	0	0	10
New York:				District of Columbia:			
Buffalo	0	0	2	Washington	1	0	0
New York	2	0	1	South Carolina:			
Rochester	0	0	8	Charleston	0	0	1
New Jersey:				Georgia:			
Trenton	0	0	1	Savannah	0	0	1
Pennsylvania:				Kentucky:			
Philadelphia	0	0	4	Ashland	0	0	1
Pittsburgh	0	0	3	Louisiana:			
Ohio:				New Orleans	1	0	0
Cincinnati	0	0	1	Oklahoma:			
Cleveland	0	0	2	Oklahoma City	0	0	1
Illinois:				Utah:			
Chicago	1	0	2	Salt Lake City	0	0	3
Michigan:				California:			
Detroit	0	0	3	Sacramento	0	0	2
Wisconsin:				San Francisco	1	0	1
Milwaukee	1	0	0				
Minnesota:							
Minneapolis	0	0	2				
St. Paul	1	1	2				

Encephalitis, epidemic or lethargic.—Cases: New York, 1.

Pellagra.—Cases: Boston, 1; Wilmington, N. C., 2; Charleston, S. C., 1; Savannah, 1; Miami, 1; Birmingham, 1; Little Rock, 2.

Typhus fever.—Cases: New York, 2; Raleigh, 2; Charleston, S. C., 1; Atlanta, 3; Savannah, 2; Miami, 1; Nashville, 8; Mobile, 2; Fort Worth, 2; Houston, 2; Los Angeles, 1.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended November 4, 1939.—During the week ended November 4, 1939, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis			1	1	2					4
Chickenpox		21		104	205	51	70	67	104	622
Diphtheria		1	1	54	3	7	13	3		82
Dysentery					1				1	2
Influenza		21			55				6	82
Measles		4		86	110	31	2	2	24	259
Mumps		1		22	68	4		4	4	103
Pneumonia		8			19	3	1		5	36
Poliomyelitis		1		2	6		1			10
Scarlet fever		13	8	88	157	32	9	18	17	342
Trachoma							2		1	3
Tuberculosis		10	11	47	45	52	6	1		172
Typhoid and paratyphoid fever			5	11	8	1	5	1	2	33
Whooping cough		25		102	53	22	15	16	8	241

NOTE.—No cases of the above diseases were reported from Prince Edward Island for this period.

JAMAICA

Communicable diseases—4 weeks ended October 28, 1939.—During the 4 weeks ended October 28, 1939, 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		5	Leprosy		2
Diphtheria	3	2	Tuberculosis	39	89
Dysentery	1	2	Typhoid fever	9	58

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

NOTE.—A cumulative table giving current information regarding the world prevalence of quarantinable diseases for a six-month period appeared in the PUBLIC HEALTH REPORTS of November 24, 1939, pages 2106-2119. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China—Tsingtao.—During the period September 3 to October 14, 1939, 122 cases of cholera with 93 deaths were reported in Tsingtao, China.

Plague

Hawaii Territory—Island of Hawaii—Hamakua District.—Four rats found on October 19, 1939, in Hamakua Mill Area, 1 rat found on October 18, and 1 rat found on October 26, 1939, in Paauhau Sector, Hamakua District, Island of Hawaii, T. H., have been proved positive for plague.

Typhus Fever

Straits Settlements—Singapore.—During the week ended September 16, 1939, 1 case of typhus fever was reported in Singapore, Straits Settlements.

Yellow Fever

Nigeria.—Yellow fever has been reported in Nigeria as follows: Jos, 1 suspected case on November 7; Odochin, 1 case on November 4, 1939.

Niger Territory—Dosso.—On November 5, 1939, 2 suspected cases of yellow fever were reported in Dosso, Niger Territory.

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