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THE ABSORPTION AND EXCRETION OF LEAD ARSENATE IN MAN[1]

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INTRODUCTION

In view of the large amounts of lead arsenate in common use as insecticide spray material, further information seems desirable regarding the behavior of lead arsenate in the human organism. Hitherto, sufficient information has been lacking with respect to (1) the extent to which lead arsenate is absorbed, (2) the route by which the absorbed material is eliminated from the system, (3) the systemic effect of small amounts on man, and (4) the effect of ingested ammonium chloride plus a low calcium diet on possible storage following ingestion of lead arsenate.

The following investigation was undertaken in an attempt to throw further light on these questions and especially to determine, if possible, any ill effects from small quantities of ingested lead arsenate. It was thought that some aid in the solution of this problem might be afforded by the determination of the lead and arsenic balance following the ingestion of known, weighed quantities of pure lead arsenate.

Careful experimental control is necessary in such an investigation, from the initial ingestion to the completion of the chemical analyses, in order to prevent loss or contamination. For this reason alone animal experimentation was out of the question, and it was thought that the fullest possible cooperation, such as could be obtained only by human experimentation, was necessary.

EXPERIMENTAL PROCEDURE

Two individuals, A and B, were selected and given a careful medical examination. The results of the examinations showed both subjects to be in good physical condition. The two men were placed on an adequate diet which could be kept under analytical control throughout the experimental period and which was low in natural lead and arsenic content. It consisted of milk, graham crackers, and apples. With

[1] From the Division of Industrial Hygiene, National Institute of Health. This is the first of a series of investigations concerning lead arsenate.

the exception of the milk, the material was purchased in bulk, sampled and analyzed for lead, arsenic, and calcium. This diet was continued for 16 days. No restriction was placed upon the quantity of food eaten, but a careful record was made of the quantity of and variation in food at each meal. Care was taken to clean and pare the fruit used, so that contamination from the surface was minimized. Analysis of the fruit used, as prepared for eating, showed that (assuming that the apples had been previously sprayed with lead arsenate) the amounts of lead or arsenic absorbed through the skin of the fruit and retained in the pulp were negligible. From the beginning of the experiment to the end of the first 16 days no deviation from this diet occurred.

In view of the increased lead excretion that occurs with acidosis in lead poisoning, according to the literature, it was felt that in case any storage of lead had occurred during this experiment it could be revealed by inducing an experimental acidosis. Therefore, beginning with the seventeenth day, the subjects were placed on a low calcium diet and in addition to this they received ammonium chloride during the first 4 days of this period. This low calcium diet was composed of meat, potatoes, soda crackers, and black coffee.

The experimental period was arranged so that, following a preliminary period of 3 days, each individual received daily at one meal a carefully weighed quantity (10 mg) of lead arsenate ($PbHAsO_4$). This was continued for 10 consecutive days, or until each individual had received 100 milligrams of lead arsenate. The lead arsenate ingestion period was followed by a rest period of 3 days and then, beginning the low calcium diet, by a period of 4 days during which each individual received 2 grams of ammonium chloride 4 times daily, or a total of 8 grams of ammonium chloride per day.

The total day's output of urine and of feces from each individual was collected in lead- and arsenic-free containers and quantitatively transferred without delay, for ashing and analysis. This procedure was maintained throughout the entire experimental period.

In spite of the simplicity of the experimental diet, individual preferences were noted. Thus, A consumed three times as much milk as B, to whom milk was rather distasteful, and B regularly consumed graham crackers, which A, with few exceptions, omitted from his diet. Apples, however, were common to both diets, and about 2½ pounds per day were consumed by each individual. Owing to the difference in the amounts of milk consumed, the difference in calcium content of the diets of the two individuals for the 16-day period is marked, as shown in table 1.

TABLE 1.—*Analysis of an average day's supply of food during the first 16 days of the experimental period*

Subject	Food			Calcium	Lead	Arsenic	Iron
	Apples	Milk	Graham crackers				
		Grams	*Grams*	*Grams*	*Milligrams*		*Milligrams*
A	1 kilo	1,440		1.649	0.009	0	7.2
B	1 kilo	480	250	.676	.006	0	14.2

It will be noted that the average lead and arsenic content of a total day's intake of food for both individuals is insignificant or negative. On the other hand, the average calcium intake of A differed from that of B in the proportion of 2½ to 1, whereas the average iron intake of A was only one-half that of B.

In view of the well-known effect of lead on the hemopoietic system as associated with lead poisoning, it seemed profitable to follow the blood picture of A and B carefully throughout the experimental period. For this reason, samples of blood were taken almost daily throughout the experiment; and, furthermore, the blood was examined at longer intervals for 4 months after the termination of the experiment. This examination included red blood count, white blood count, hemoglobin, Schilling's differential, reticulocyte and stipple cell estimations. In a few instances blood calcium determinations were made. In addition to the chemical analysis of the urine for lead and arsenic, chemical and microscopic examinations of the urine were made at intervals for any clinical evidence of possible genitourinary injury.

Symptoms, if any, were noted daily throughout the experiment.

In the analytical determinations which were made on the urine and feces, particular care was taken to avoid loss or contamination of the constituents sought. Both the urine and feces were acid ashed in order to avoid arsenic losses. Care was taken to insure separating lead from the urine and fecal specimens, and the residues were tested in all cases to insure complete removal The fecal and urine residues were negative or indicated negligible traces with dithizone after this treatment.

The methods of analysis used for the determination of lead were adapted to the quantities involved in the determination. When the quantities of lead were large or did not fall below the ordinary clinical range, the chromate titration method of analysis was used. For the determination of lead below these amounts, the dithizone method of determination was employed. The arsenic determinations were made by a modification of the Gutzeit method.

EXPERIMENTAL RESULTS

The amounts of lead, arsenic, and calcium excreted daily by both individuals are summarized in table 2 and are shown graphically in figures 1 and 2.

The results obtained in the clinical and microscopic examinations of the blood during the initial period of the experiment, the lead arsenate ingestion period and later periods, as well as at later infrequent intervals, are shown in table 3 and figure 3.

Certain features of the excretion of calcium, arsenic, and lead are common to both subjects. In general the calcium, arsenic, and lead excretion run parallel in the two subjects except with respect to urinary calcium. The average values for urinary calcium differ in that the total output of A is much greater than B. Furthermore, following the period of ammonium chloride ingestion, A's urinary calcium rose to a high level, while that of B showed but very little increase, even though both individuals were on a low calcium diet at this time. Blood calcium determinations on both A and B were made at the end of the lead arsenate period and at the end of the ammonium chloride period; however, these revealed no significant departure from normal blood calcium figures.

LEAD

The fecal excretion of lead occurred mostly within the period of lead arsenate ingestion and dropped at the end of this period. From this point on a small but steady excretion of lead occurred and was maintained to the end of the sampling period. During the actual ammonium chloride-low calcium diet period, no pronounced increase in fecal lead output was apparent, but it should be noted that following this period a progressively greater fecal lead excretion occurred, which reached a maximum on the third day following the termination of the ammonium chloride-low calcium diet period. This later slow excretion of lead possibly represents lead that had been retained by the tissues and re-excreted.

The urinary lead on the other hand was small in amount as compared with the quantity ingested, and was sporadic in output. In the case of B, with the exception of 1 day the lead output was not marked. In fact, several times during the lead arsenate ingestion period and following it on a number of occasions the daily urinary lead output of B amounted to only a few micrograms. In the case of A the urinary lead output increased from a normal of a few micrograms to an average well above that of B. The maximum quantity of urinary lead excreted during the experimental period in any given day amounted to 0.193 milligram in the case of A and 0.138 milligram in the case of B. Toward the end of the experiment both values tended to drop to normal.

TABLE 2.—*The excretion of lead, arsenic, and calcium during the lead arsenate ingestion experiment*

Days of exposure	Period of experiment	Amount ingested (milligrams)	Gastrointestinal tract				Genitourinary tract					
			Weight feces (grams)	Lead excreted (milligrams)	Arsenic excreted	Calcium excreted (grams)	Volume of urine (cc)	Specific gravity	Hydrogen ion	Lead (milligrams)	Arsenic (milligrams)	Calcium (gram)
						SUBJECT A						
1	Normal		147	0.005		0.505	2,920	1.008	6.7	0.017	0	0.236
2	do		181	.015		1.079	2,200	1.006	6.5	.014	0	.320
3	do		25	.004		.323	2,300	1.005	6.3	.005	0	.252
4	Lead arsenate	10	30	.34		.517	2,170	1.007	6.6	.010	.540	.254
5	do	10	250	1.89		1.438	1,950	1.010	6.6	.025	.975	.229
6	do	10	230	1.76		1.458	2,430	1.006	6.5	.097	2.187	.223
7	do	10	30	1.37		.210	1,960	1.008	6.0	.054	1.960	.243
8	do	10	149	6.15		1.329	2,250	1.008	6.3	.018	1.912	.305
9	do	10	165	2.63		.796	2,300	1.005	6.3	.078	1.725	.289
10	do	10	212	5.80		1.980	3,830	1.003	6.6	.047	1.608	.278
11	do	10	91	2.96		1.107	2,080	1.005	6.2	.122	1.456	.208
12	do	10	39	1.66		.521	3,070	1.002	6.4	.108	1.166	.209
13	do	10	0				3,960	1.002	6.6	.071	2.098	.199
14	Normal		260	6.00		1.635	2,120	1.006	6.3	.110	.933	.153
15	do		105	1.08		1.184	2,620	1.004	6.6	.134	.681	.201
16	do		135	.56		1.321	2,440	1.004	6.3	.100	.512	.187
		Grams										
17	Ammonium chloride	8	70	1.06		.945	3,200	1.006	6.1	.193	.512	.157
18	do	8	80	.68		.739	3,860	1.006	5.3	.117	.380	.194
19	do	8	45	.72		.271	3,910	1.009	5.3	.143	.39	.265
20	do	8	200	1.13		.416	3,300	1.006	5.3	.071	.33	.238
21	Low calcium diet		60	1.23		.202	2,000	1.012	5.5	.056	.20	.323
22	do		402	1.64		.315	2,330	1.008	5.6	.030	.23	.477
23	do		420	3.09		.566	2,580	1.010	6.0	.119	.25	.447
24	do		202	.86		.444	2,000	1.011	6.3	.015	.20	.388
25	do		105	.36		.287	3,040	1.010	6.5	.025	.15	.504
26	do		302	1.35		1.159	2,600	1.008	6.0	.010	.13	.466

TABLE 2.—*The excretion of lead, arsenic, and calcium during the lead arsenate ingestion experiment*—Continued

Days of exposure	Period of experiment	Amount ingested (milligrams)	Gastrointestinal tract				Genitourinary tract					
			Weight feces (grams)	Lead excreted (milligrams)	Arsenic excreted	Calcium excreted (grams)	Volume of urine (cc)	Specific gravity	Hydrogen ion	Lead (milligrams)	Arsenic (milligrams)	Calcium (grams)
					SUBJECT B							
		Milligrams										
1	Normal		360	0.002		1.123	2,180	1.009	6.7	0.006	0	0.096
2	do		170	.010		.537	1,410	1.010	6.6	.007	0	.049
3	do		262	.002		.356	1,520	1.012	6.4	.006	0	.044
4	Lead arsenate	10	443	5.69		.586	1,770	1.008	6.8	.018	.442	.051
5	do	10	649	4.22		.598	1,140	1.012	6.2	.002	.969	.042
6	do	10	467	4.15		.574	3,700	1.005	7.2	.018	1.850	.062
7	do	10	275	1.42		.323	1,620	1.006	6.7	.138	1.134	.041
8	do	10	325	8.20		.804	1,980	1.006	6.6	.030	1.485	.042
9	do	10	520	5.55		.606	1,520	1.012	6.4	.026	2.280	.040
10	do	10	210	1.03		.154	620	1.010	7.0	.010	.434	.039
11	do	10	490	5.07		.792	1,060	1.011	6.0	.025	.848	.052
12	do	10	632	7.78		.930	1,400	1.007	6.0	.069	1.274	.061
13	do	10	275	4.80		.534	1,460	1.011	6.6	.010	1.59	.071
14	Normal		470	1.44		1.006	1,430	1.008	6.0	.010	1.015	.019
15	do		325	.09		.546	1,210	1.007	5.5	.010	.653	.047
16	do		540	.28		.646	1,740	1.008	6.8	.071	.644	.055
		Grams										
17	Ammonium chloride	8	115	.36		.057	3,120	1.003	5.3	.010	.530	.023
18	do	8	125	.22		.376	1,840	1.011	5.3	.010	.27	.059
19	do	8	105	.08		.287	2,240	1.014	5.3	.095	.33	.075
20	do	8	245	.55		.262	2,320	1.011	5.3	.015	.23	.013
21	Low calcium diet		175	1.22		.287	1,200	1.014	5.3	.025	.18	.073
22	do		155	1.20		.351	1,770	1.009	5.5	.056	.17	.029
23	do		170	2.34		.242	1,480	1.009	5.5	.018	.07	.066
24	do		245	.89		.299	1,240	1.014	6.3	.040	.12	.072
25	do		265	.76		.679	1,660	1.010	6.7	.030	.08	.083
26	do		185	.90		.376	1,900	1.010	6.1	.020	.09	.085

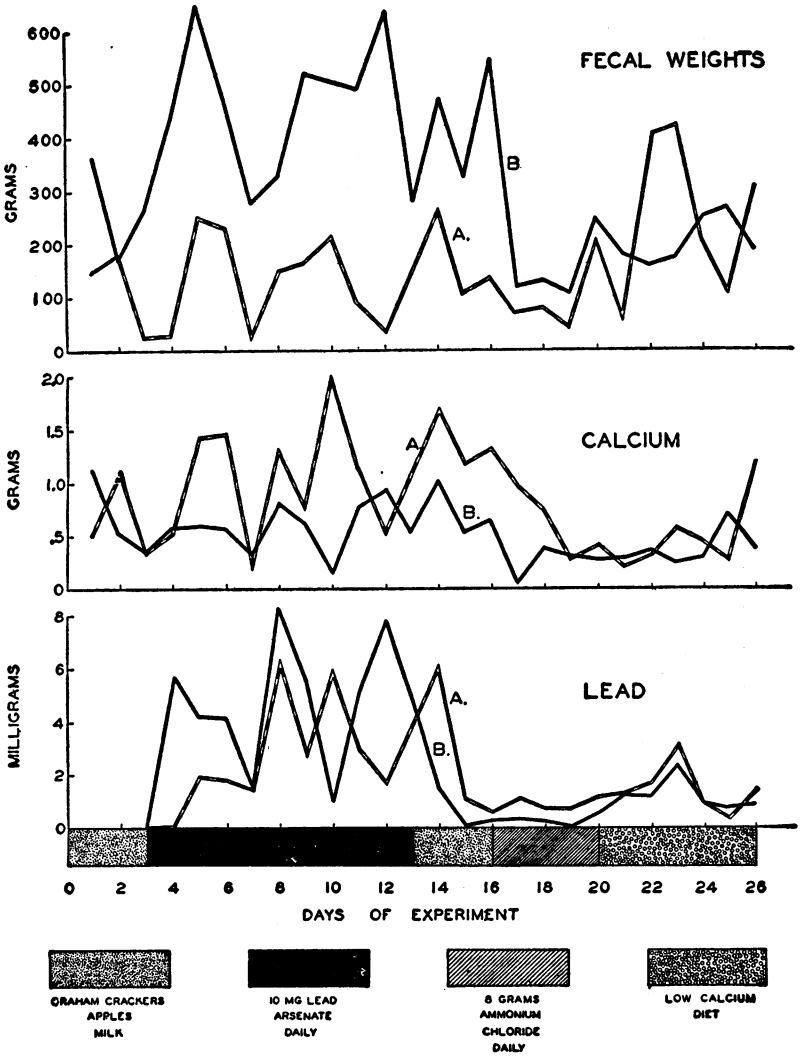

FIGURE 1.—The fecal excretion of lead and calcium following lead arsenate ingestion.

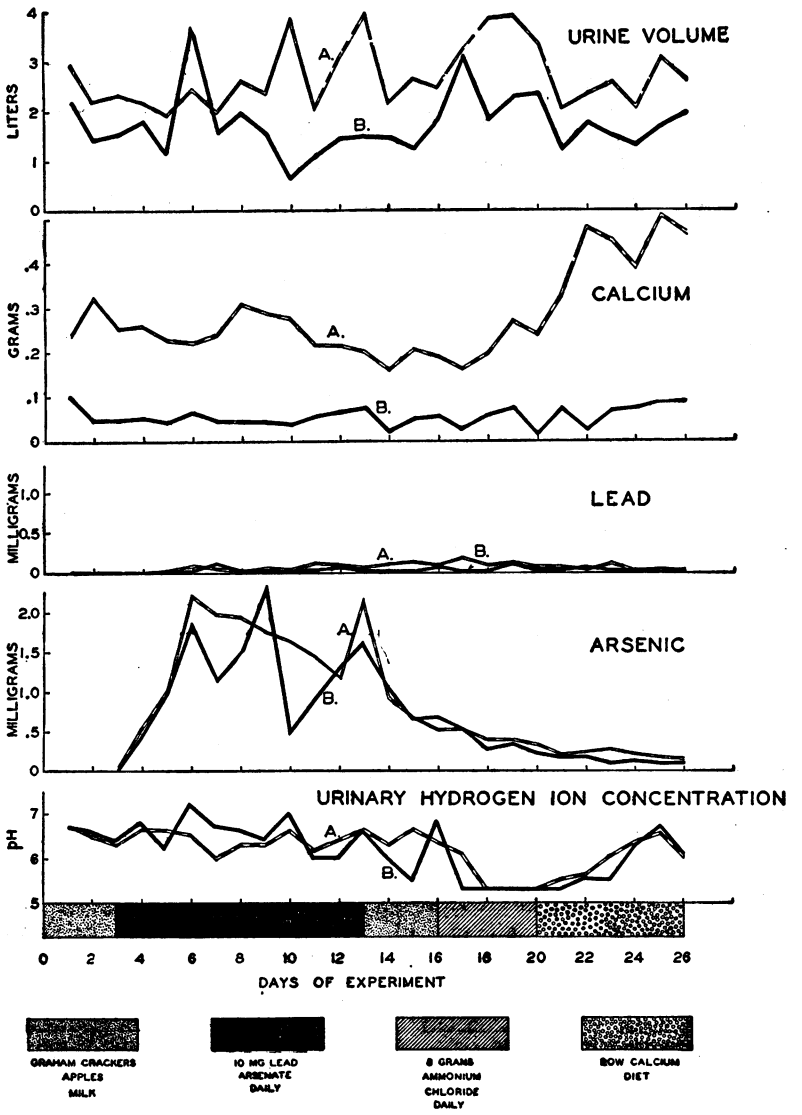

FIGURE 2.—The urinary excretion of lead, arsenic, and calcium following lead arsenate ingestion.

TABLE 3.—*Blood findings in two men following the ingestion of 10 mg of lead arsenate daily for 10 days*

Day of exposure[1]	Period of experiment	Red blood cells										White blood cells													
		RBC		Hemoglobin (percent)		Reticulocytes (percent)		Polychromasia		Basophilic stippling		WBC		Schilling's differential								Monocytes		Eosinophiles		Basophiles	
														Segmented neutrophils		Juveniles		Bands		Lymphocytes					
		A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B
1	Normal	5.14	5.67	84	97	0.23	0.26	0	0	0	0	10.9	8.4	69	71	0	0	6	1	21	21	2	6	2	1	0	0
2	do	5.11	5.73	84	95	.22	.23	0	0	0	0	11.3	7.7	62	57	1	0	10	1	21	33	5	6	1	2	0	1
3	do					.26	.34	0	0	0	0																
4	Lead arsenate					.60	.53	0	0	0	0																
5	do					.35	.41																				
6	do					.40	.50																				
8	do	5.29	5.58	84	94	.27	.30	0	0	0	0	11.7	8.2	53	59	0	0	11	5	27	21	7	8	1	4	1	3
9	do					.40	.60																				
10	do					.44	.48																				
12	do					.46	.51																				
13	do					.48	.54																				
15	Normal	4.82	5.62	81	93	.44	.41	0	0	0	0	10.5	8.6	57	53	1	0	12	6	20	30	8	6	2	3	0	2
16	do					.47	.57	0	0	0	0																
17	Ammonium chloride					.63	.40	0	0	0	0																
18	do					.83	.45	0	0	0	0																
19	do	5.48	5.99	78	90	.61	.46	0	0	0	0	12.2	9.4	59	51	1	0	11	3	18	30	9	9	2	2	0	5
20	do	5.52	5.91	78	87	.58	.71	0	0	0	0																
23	Low calcium diet	4.90	5.71	74	89	.69	.72	0	0	0	0																
24	do	5.03	5.52	75	87	.74	.47	0	0	0	0	12.5	8.3	66	61	0	0	6	0	22	29	6	9	0	0	0	1
26	do	4.96	5.51	74	86	.74	.52	0	0	0	0	11.6	9.3	61	62	0	0	6	1	28	24	4	7	1	5	0	1
	(End of experiment.)																										
29		5.06	5.58	78	87	.68	.47	0	0	0	0	12.8	8.3														
32		4.97	5.71	81	87	.50	.75	0	0	0	0	13.6	8.0	50	61	0	0	6	0	29	32	4	4	1	2	0	1
36		5.11	5.49	81	86	.76	.88	0	0	0	0	9.7	7.1	52	65	0	0	6	1	38	27	2	4	2	3	0	0
39		5.23	5.68	81	89	.77	1.04	0	0	0	0	9.4	8.5	73	63	0	0	1	0	17	29	6	6	3	2	0	0
44		5.21	5.68	81	89	.65	.70	0	0	0	0	10.6	7.6	69	65	0	0	3	0	22	29	3	4	2	1	1	1
47		5.32	5.10	82	91	.78	.56	0	0	0	0	11.3	7.7	74	59	0	0	4	0	17	37	5	2	0	2	0	0
52		5.4	5.74	81	90	.78	1.00	0	0	0	0	12.1	8.6	53	66	2	0	12	3	28	23	4	5	0	3	1	0
60		5.69	5.81	85	93	.45	.66	0	0	0	0	10.2	8.1	46	65	1	0	9	3	32	22	8	6	3	3	1	1
167		5.33	5.44	87.4	89	.29	.23	0	0	0	0	11.5	8.5	55	57	0	0	8	4	30	30	4	6	3	0	0	0

[1] Blood work was omitted on 7th, 11th, 14th, 21st, 22d, and 25th days of experiment; therefore these days are omitted from the table.

Thus, it would appear from the excretion figures for lead that the bulk of the metal passes through the gastrointestinal tract unabsorbed and that a small portion (1.3 to 3.2 percent) of the total ingested lead is absorbed and enters the systemic circulation, as shown by the urinary lead output.

From the fact that ammonium chloride did not cause an immediate response, that the urinary output of lead was very small, and that there was a diminished but steady output of fecal lead following the

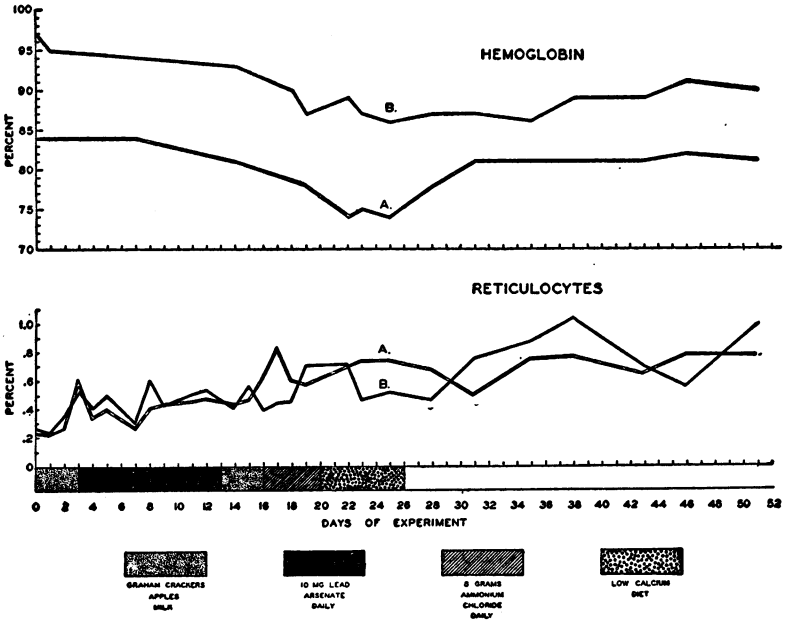

FIGURE 3.—Hemoglobin percentages (Newcomer) and reticulocyte counts expressed as percentages of total erythrocyte counts.

period of lead arsenate ingestion, one is inclined to consider the question of retained but mobile lead as differentiated from lead storage.

ARSENIC

With respect to arsenic the situation is exactly the reverse of that of lead. In this experiment the arsenic was almost completely absorbed and was excreted only through the kidneys. Although special care was used to prevent any possible loss in ashing, and repeated attempts were made to detect arsenic in the fecal specimens, the total amount of arsenic found to be excreted through the gastrointestinal tract amounted to but a few hundredths of a milligram. It should be pointed out that, while complete absorption of arsenic apparently occurred as shown by its presence in the urine and absence in the feces, it is quite possible that long-continued ingestion of small amounts of arsenic may result in fecal excretion of this substance.

This unexpected finding, in view of the low solubility of lead arsenate in water, is direct evidence that the ingested lead arsenate was broken down in passing through the gastrointestinal tract. The urinary arsenic output response was immediate following the ingestion of lead arsenate and the greater part was excreted during the ingestion period. From this point on there was a steady, constantly decreasing output of urinary arsenic, which was not affected by the ammonium chloride-low calcium diet regime. A small quantity of arsenic, rising to a maximum of 3 micrograms per gram, was found in the hair and nails of both subjects over a period of 4 months.

CALCIUM

During the course of the first 16 days of the experiment both subjects showed a slight positive calcium balance, but there was a marked difference between the calcium intake of A as compared with that of B. The average daily calcium intake of B amounted to but 40.9 percent of that of A, while the average daily calcium excretion of B represented but 41.9 percent of that of A. It is of interest, furthermore, that with respect to the urine the average daily urinary calcium output of B over the entire experiment was less than one-tenth that of A.

Since this marked difference occurred between A and B with respect to calcium intake, and since calcium is said to favor the accumulation of lead, it is, therefore, not surprising that during the experimental period the total lead output of A was somewhat less than that of B.

MEDICAL STUDY

Following the preliminary physical examination by a physician experienced in the diagnosis of lead and arsenic intoxication, each individual was carefully observed daily for evidence of any subjective or objective findings relative to lead or arsenic. During the experimental period and in the final physical examination the findings were negative except for a slight loss of weight (2 pounds in one case, and 4 pounds in the other) due to the monotonous diet and to the later low calcium diet and acidosis induced by the ingested ammonium chloride.

The chemical and microscopic examinations of the urine that were made at frequent intervals throughout the experiment were negative for any evidence of genitourinary injury.

As will be noted in table 3, the leucocyte count and band count in individual A, before beginning the experiment and continuing throughout, were above the average, and the leucocyte count and differential count were at all times within the average range in individual B. No change in the leucocyte or differential counts attributable to lead or arsenic could be ascertained.

Red blood cell counts were made at frequent intervals on each individual and were found to remain within normal limits before, during, and after the experimental period.

The hemoglobin percentages in each individual decreased gradually from the first day of the experiment to the last day of the milk, apple, and graham-cracker diet. Although the most marked decrease occurred during the period of acidosis, almost immediately upon changing to a meat diet the hemoglobin percentages began rising and eventually reached approximately the same level as before the experiment began. The hemoglobin of A dropped from 84 percent at the beginning of the experiment to 74 percent on the last day of the experiment, over a period of 26 days; but on the third day after terminating the experiment, it was back to 81 percent, and from this period on the hemoglobin gradually increased to 87.4 percent. This was greater than before the experiment began. The hemoglobin of individual B decreased from 97 percent at the beginning of the experiment to 86 percent on the last day of the experiment, and then rose slowly to 91 percent. It will be noted that B had approximately twice the iron intake in the experimental diet as A, and for this reason one would not expect B's hemoglobin curve to respond so spectacularly to the later diet.

The reticulocyte counts of both A and B remained well within normal limits throughout the experiment. A's reticulocyte count ranged from 0.23 percent on the first day of the experiment to 0.74 percent on the last day of the experimental diet, and B's count ranged from 0.26 to 0.52 percent during the same period. His reticulocyte count was 1.04 percent 2 weeks after the experiment and in 2 weeks more it was 0.66 percent. The reticulocyte counts in each individual about 4½ months after the termination of the experiment were 0.29 percent for A and 0.23 percent for B. There was a slight increase in the reticulocyte count within the normal range in each individual during the experimental period. It would be interesting to know whether these slight increases were due to lead, arsenic, or to the deficient diet.

Osgood and Ashworth, in the Atlas of Hematology, state that a reticulocyte count of 4 percent or more in persons over 4 years of age indicates an increased rate of erythrocyte formation. It should be noted that our results fall far below this value. Using this as a criterion, it does not appear that A and B had a definite increase in erythrocyte formation.

The reticulocyte counts and all blood and urine examinations were made by the same individual, using the same equipment and supplies. The Newcomer instrument and technique were used for the hemoglobin determination and Osgood's method was used for the reticulocyte staining and counting.

DISCUSSION

One significant fact is outstanding with reference to this experiment, i. e., the extent to which lead arsenate is broken down within the human organism. Furthermore, the behavior of the component parts of the molecule within the body varies, since the lead is largely excreted in the feces whereas the arsenic is almost completely eliminated in the urine.

A second fact of significance is that by careful physical examination and medical observation throughout, no evidence of injury to the two individuals from the ingestion of 100 milligrams of lead arsenate taken over a period of 10 days was apparent.

It should be noted, however, that these observations are strictly confined to a certain quantity of lead arsenate taken over a period of 10 days by two adult individuals, and does not relate to the possibility of toxicity from massive doses of lead arsenate or from the continued daily ingestion of small amounts of lead arsenate over long periods of time.

The analytical results obtained in this study are summarized in table 4. Analysis of the lead arsenate used showed a lead content of 59.7 percent and an arsenic content of 21.59 percent. Therefore, the 100 milligrams of ingested lead arsenate represented 59.7 milligrams of lead and 21.59 milligrams of arsenic. Of this ingested lead, 74.4 percent was recovered in the feces in the case of A and 97.5 percent in the case of B.

TABLE 4.—*100 milligrams of lead arsenate ($PbHAsO_4$) ingested in 10-milligram doses over a period of 16 days*

Individual	Total lead consumed	Total arsenic consumed	Total lead excreted in—				Total arsenic excreted in—				Average daily output of calcium	
			Urine		Feces		Urine		Feces		Urine	Feces
	Mg	*Mg*	*Mg*	*Percent*	*Mg*	*Percent*	*Mg*	*Percent*	*Mg*	*Percent*	*Mg*	*Mg*
A	59.7	21.59	1.92	3.2	44.42	74.4	20.79	96.4	0.03	-------	278	906
B	59.7	21.59	.82	1.3	58.24	97.5	16.69	77.5	.09	-------	26	512

The excretion of lead in relation to calcium intake and output is worthy of attention. For the first 16 days of the experiment the average daily calcium intake of A amounted to 1.649 grams, while that of B amounted to 0.676 gram. The average daily calcium output of A during this period amounted to 1.267 grams, while that of B was 0.683 gram. Therefore, the calcium balance in A was positive throughout this period, while that of B was only very slightly negative. Comparison of the above figures shows that both the calcium intake and output of A were double that of B. However, a comparison of

the fecal lead excretion of A and B shows a lower lead output for A that for B. It should be noted that the fecal excretion of the total ingested lead was high in the case of A (74.4 percent) and was nearly quantitative (97.5 percent) in the case of B.

The effect of the ingested ammonium chloride and the low calcium diet upon the lead output was not as great as was anticipated. Although a low calcium content diet was purposely selected for this period, and although the urinary hydrogen ion concentration promptly increased from a pH of about 6.5 to 5.3 and remained at this latter figure throughout the ammonium chloride period, the response of fecal lead showed only a delayed rise, and there is some question as to whether the rise could be attributed to the low calcium diet and the ammonium chloride or to increased gastric response following the change to a more appetizing diet. The urinary calcium output of A during this period showed immediate response to the acid regime and attained a higher figure than at any other time during the experiment.

This fecal excretion of lead does not necessarily indicate lack of absorption. It is evident that partial absorption occurs, and this is indicated by the fact that a small amount of the ingested lead was recovered in the urine. It is also possible that further absorption of lead may have occurred as it passed through the gastrointestinal tract, and that this lead entering the portal circulation would be arrested by the liver and excreted in the bile.

With arsenic the absorption is practically quantitative, as indicated by the fact that the feces contained only traces of this element. This would further indicate not only complete breaking down of the lead arsenate, but also rapid absorption of the arsenic component and a preferred path of excretion.

The difference in the paths of excretion followed by the arsenic and the lead derived from lead arsenate at once gives important information regarding this material in the organism. Complete solution of lead arsenate occurs at some stages in passing through the gastrointestinal tract. Until further solubility data are secured,[2] however, it will be difficult to state exactly at what stage in its passage through the alimentary canal the compound is sufficiently dissolved to permit absorption to occur.

Outstanding in importance is the fact that lead arsenate, although broken down in the organism, can be so rapidly and effectively eliminated.

Of the total amount of lead excreted by the gastrointestinal tract, 71 percent in one case and 85 percent in the second was excreted within the period of ingestion of lead arsenate, allowing for the necessary time lag in excretion. The lead that was excreted from this time on probably represented lead that had been removed by the liver and

[2] Solubility studies of lead arsenate are in progress at the present time and will be published shortly.

later re-excreted. This prompt excretion of the major portion of the lead in all probability represents that fraction that was merely mechanically carried through the alimentary tract.

It is of interest that the remaining portion—which appears to be absorbed lead—was not rapidly affected by the low calcium diet and the change in acid condition of the body following the ingestion of ammonium chloride in the same sense that these factors are reported to increase the excretion of stored lead in lead poisoning. On the other hand, increased elimination of lead occurred eventually following the change to low calcium diet and acidosis produced by ammonium chloride.

With respect to the arsenic, the elimination began at once following ingestion of the lead arsenate and remained at a high level during this period. Following the period of lead arsenate ingestion the arsenic excretion approached the normal rather slowly but quite regularly in both cases. The low calcium diet and acidosis produced by the ammonium chloride caused no change in the rate of arsenic excretion.

This difference in the urinary excretion of lead and arsenic may possibly be related to the difference in the solubility relationship of the two substances in body fluids. One would expect the arsenical compounds to be more or less freely soluble and lead to be more insoluble in the tissues. This is well shown in those instances where the urinary lead dropped to a normal of only a few micrograms per day during the period when it was definitely known that absorption had occurred. Otherwise it is difficult to account for the somewhat uneven excretion of urinary lead that occurred in individual B.

SUMMARY

One hundred milligrams of lead arsenate were ingested by two individuals over a period of 10 days while on a controlled diet. The degree of absorption, path of excretion, and toxicity of this dosage were evaluated. Variations of the calcium content of the diet and the effect of change in hydrogen ion concentration as induced by ammonium chloride were noted. While the lead arsenate was completely broken down in the body, no untoward effects on the well-being of these two individuals attributable to this quantity were noted. The greater part of the lead and arsenic derived from the ingested lead arsenate was directly recovered, and it was found that the lead was excreted with the feces and that the arsenic was excreted in the urine.

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THE PERSISTENCE OF THE VIRUSES OF ENDEMIC (MURINE) TYPHUS, ROCKY MOUNTAIN SPOTTED FEVER, AND BOUTONNEUSE FEVER IN TISSUES OF EXPERIMENTAL ANIMALS[1]

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The purpose of this paper is to report observations on the survival of the viruses of endemic (murine) typhus, Rocky Mountain spotted fever, and boutonneuse fever in the brain, spleen, blood, and the tunica exudate of white rats, white mice, guinea pigs, and the Columbian ground squirrel (*Citellus columbianus*).

ENDEMIC (MURINE) TYPHUS

It has been observed that the virus of endemic typhus persists in the brains of certain animals following clinical recovery. Among investigators reporting this fact may be mentioned Nicolle and Laigret (*1*) who recovered the virus from the brains of white rats 60 days after fever and 68 days after inoculation, but found that it had disappeared from the brains of guinea pigs by the 30th to the 40th day.[2] These periods, however, were much longer than in concurrent similar tests of the survival of the viruses of louse-borne (European, epidemic or historic) typhus and of Rocky Mountain spotted fever (western Montana strain). Lépine (*3*) demonstrated the presence of endemic typhus virus in white rats' brains for varying periods after inoculation up to the 87th day; he further reported the survival of European typhus virus for at least 64 days. On the other hand, Laigret and Jadin (*4*) found that the latter virus did not survive longer than 10 days in brains of white mice, whereas that of endemic typhus was recovered up to the 40th day. Violle (*5*) reported recovery of murine typhus from the brain of a rabbit 60 days after inoculation. Combiesco et al. (*6*) obtained murine virus from the brain of a "spermophile" 30 days after inoculation, and Lépine and Sautter (*7*) extended this observation to the remarkable period of 374 days, using the Macedonian spermophile (*Citellus citellus*). The latter authors considered the virus to be consistently present in these animals up to the 118th day and often to the 226th day. This suggested to them that this phenomenon might have a possible important relationship to the epidemiology of the disease.

Our observations have extended the previously observed periods of survival of endemic typhus virus in the tissues of white rats,

[1] Contribution from the Division of Infectious Diseases, National Institute of Health, Rocky Mountain Laboratory, Hamilton, Montana.

[2] Lewthwaite (*2*), using white rats inoculated with Malayan urban typhus, a flea-borne infection of the "X19 type" which is at least closely related to endemic typhus, failed to recover virus from the brains of one of these animals 30 days, and of 2 each 60 and 180 days after inoculation.

white mice, and guinea pigs, but were completely negative for the local ground squirrel (*Citellus columbianus*). The Wilmington strain of endemic typhus virus, obtained from the National Institute of Health at Washington, D. C., was used. Tunica exudate of passage strain guinea pigs, obtained by washing the parietal and visceral tunicas in physiological saline, was used as the inoculum. Duration tests were made at desired intervals by sacrificing the test animals and by transferring to duplicate normal male guinea pigs brain and spleen tissue (triturated in physiological saline), whole blood, and tunica exudate (in the case of males). Each transfer animal received intraperitoneally 1 cc of inoculum. Temperatures were taken daily for 15 to 21 days, and shortly thereafter the survivors were injected with controlled virus of the Wilmington strain. In reporting the experimental results, "positive" means that the test injection was followed by definite fever and characteristic swelling and reddening of the scrotum and that the animal was subsequently immune to controlled virus; "negative" indicates that there was no apparent response to the test material during at least 2 and more often 3 weeks, and that the subsequent virus injection was followed by typical evidence of a murine typhus infection.

TEST DATA

On September 28, 1935, 30 white rats and 6 Columbian ground squirrels received 1 cc of virus each, and 30 white mice ¼ cc each. Guinea pig controls, injected at the same time, developed typical fevers and scrotal lesions. Two each of the white rats and mice were sacrificed approximately monthly, and one ground squirrel bimonthly for transfers as described above.

Transfers from white rats.—Positive results followed blood transfer from both donors at 31 days, spleen transfer of one donor each at 31, 125, and 153 days, and brain transfer from both donors at 31, 60, 97, 125, 153, and 249 days. All transfers by tunica washings and all blood transfers after the thirty-first day were negative, as well as spleen transfers from both donors at 60, 97, 218, and 304 days, and from one donor at 31, 125, 153, and 249 days; the two brain transfers at 304 days were both negative also. The tests not listed above were invalidated by intercurrent infection.

Supplemental brain transfers from miscellaneous donors at 25, 30, 42, 50, 147, 161 (2), and 370 (2) days also were positive, but at 463 days were negative.

The above data as regards survival of endemic typhus virus in spleens and brains of white rats are summarized in the following table (valueless tests omitted).

Days after virus injection	Brain tests		Spleen tests	
	Number of rats tested	Number of brains positive	Number of rats tested	Number of spleens positive
25	1	1	0	----
30	1	1	0	----
31	2	2	2	1
42	1	1	0	----
50	1	1	0	----
60	2	2	2	0
97	2	2	2	0
125	2	2	2	1
147	1	1	0	----
153	2	2	2	1
161	4	2	0	----
218	2	1	2	0
249	2	2	1	0
304	2	0	2	0
370	2	2	0	----
463	1	0	0	----
Total	28	22	15	3

These data show that brain transfer was positive for 22 of 28 rats (78.57 per cent) tested at varying intervals up to 463 days. The tests were 100 per cent positive to the 153d day, but only 53.85 per cent positive (7 of 13 donors tested) thereafter.

Transfers from white mice.—Positive tests included the spleen transfers from both donors at 30 days, and from one at 132 days, and brain transfers from one donor each at 132 and 150 days. All other transfers, including blood and tunica washings, up to and including 218 days were negative, except for one doubtful blood test each at 101 and 218 days. Transfers at 249 days were valueless.

Transfers from ground squirrels of the various tissues at 60, 125, 185, 249, and 304 days were all negative.

Transfers from guinea pigs.—Brain transfers resulted positively from one animal each at 60, 90, and 120 days, the duplicates being negative. All tests of the other tissues of both donors at these periods were negative, as well as those including the brains at 150, 277, 287 and 360 days. Miscellaneous observations include positive transfers by brain 10, 11, and 14 days and by tunica exudate 16 days following cessation of fever.

ROCKY MOUNTAIN SPOTTED FEVER

In two similar series of concurrent tests of Rocky Mountain spotted fever, using a highly virulent strain of western Montana virus, transfers were made from duplicate rats and mice at 30-day intervals up to, and including, 210 and 270 days, respectively, after inoculation (except for technical difficulties which prevented the 60-day tests), and from the ground squirrels at 60-day intervals to 240 days.

The only definitely positive test was of the brain of a white rat killed 30 days after inoculation. One of each pair of test animals

receiving spleen emulsion from ground squirrels 1 and 2 (30 days) remained afebrile both before and after the immunity test. This was suggestive of the possible presence of virus, but it cannot be stated with certainty that the absence of fever following the immunity test was of specific significance. Of the other 192 test guinea pigs to which tissues were transferred, 169 were negative and in 23 the tests were valueless.

In a supplemental experiment, incidental to other studies, the brains of 3 recovered guinea pigs sacrificed at 45, 20, and 20 days, respectively, after inoculation, and of one white rat killed 24 days after inoculation, all animals having received Montana virus, were transferred to duplicate test guinea pigs. Three of the latter died of intercurrent infections; the remainder (at least one animal in each test) were negative. The donor guinea pigs had each shown definite fever and scrotal lesions, and transfers were made 34, 14, and 14 days, respectively, following termination of fevers.

BOUTONNEUSE FEVER

The plan of this experiment (using our boutonneuse fever strain from Morocco) duplicated exactly that with spotted fever, except that the 60-day tests with white rats and mice were included, while tests with the mice were discontinued after 180 days and those with the Columbian ground squirrel were extended to 300 days. No definite transfer of virus was accomplished in any test. In three instances, however, namely, the 60-day spleen transfer of rat 4, the 180-day spleen and blood transfers of mouse 10, and the 240-day spleen transfer of ground squirrel 4, one of each pair of the test guinea pigs remained afebrile after both the brain tissue transfer and the later injection of boutonneuse fever virus. Of the 238 other test animals used, 224 were negative and in 14 tests were valueless.

Incidental tests of four recovered passage guinea pigs (two each of our Greek and Morocco strains) were made by brain transfer alone. One of each strain was sacrificed 30 days after inoculation, and the other two, 220 days after inoculation. Two normal guinea pigs were injected with brain tissue from each of the animals with completely negative results.

DISCUSSION

These data suggest that white rats are more favorable laboratory animals for testing the survival of endemic typhus virus than either white mice or guinea pigs, and that in all three animals the brain is the most favorable tissue. It is entirely possible that the virus may persist for a longer period than shown by these tests.

Endemic typhus virus was not constantly persistent over the same periods in the same species of test animal, but varied with different

individuals. On several occasions this virus was recovered in only one of two guinea pigs receiving duplicate samples of the same tissue.

In view of our results and those of other investigators showing the presence of frankly infectious endemic typhus virus in the brains and occasionally in the spleens of laboratory animals long after clinical recovery, it would be of value to know to what extent latent infections occur and if such infections are important epidemiologically, particularly as regards the availability of virus in the blood for ectoparasitic transfer. The virus has repeatedly been recovered from the brains of domestic rats in several countries but not under conditions that have permitted any significant conclusions with respect to elapsed time after infection. It has been recovered only once from other rodents, namely, from the brain of an old-field mouse, *Peromyscus polionotus polionotus*, trapped in southeastern Alabama (*8*).

Since Lépine and Sautter (*7*) found the ground squirrel, *Citellus citellus*, highly susceptible, and readily recovered the virus from the brain up to the two hundred and twenty-sixth day after inoculation, and occasionally thereafter up to 374 days, our completely negative results with the Columbian ground squirrel (*Citellus columbianus*) are of interest.

The fact that the viruses of Rocky Mountain spotted fever and boutonneuse fever could not be demonstrated in the brain, spleen, blood, or tunica exudate of white rats (except for one 30-day brain transfer) suggests that the absence of virus in brains of recovered rats may be a useful additional criterion for differentiation of these viruses from that of endemic typhus.

SUMMARY

Endemic typhus virus, capable of producing frank infection in guinea pigs, was found to persist in white rats and white mice more consistently and for longer periods than in the same tissue of guinea pigs, or than the viruses of either Rocky Mountain spotted fever or boutonneuse fever in these animals. Of the tissues tested, namely, blood, spleen, tunica exudate, and brain, the last seemed to be the most favorable site for persistence.

In four different series of white rats, sampled periodically, endemic typhus virus was recovered from brain tissue as long as 147, 153, 161, and 370 days after noculation, respectively. It was also obtained from the blood and spleen at 31 days, and from the spleen at 125 and 153 days. In white mice, the virus was recovered sporadically from brain tissue up to 150 days after inoculation and from the spleen on the one hundred thirty-second day. It was less consistently found in the mice than in the rats. The virus was recovered from the brains of guinea pigs 60, 90, and 120 days after inoculation, and once from tunica exudate 16 days after fever, but not from other tissues. All

tests of the several tissues of Columbian ground squirrels were negative from 60 to 304 days after inoculation.

Tests for persistence of Rocky Mountain spotted fever virus were made in series of white rats, white mice, and ground squirrels over periods of 270, 210, and 240 days, respectively. The only definitely positive test was with the brain of a white rat sacrificed 30 days after inoculation. No definite, positive result was obtained in any of the similar boutonneuse fever tests.

It is suggested that persistence of endemic typhus virus in the brain of the white rat may be used as a supplemental aid in the differentiation of endemic (murine) typhus from certain other rickettsial infections.

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ENDEMIC TYPHUS VIRUS IN MICE[1]

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The virus of typhus fever was reported by Nicolle and Lebailly (*1*) in 1919 to give an apyretic or inapparent infection in white mice. In 1925–26, Nicolle (*2*) (*3*) found in this species that the epidemic type could not be passed from mouse to mouse. However, the virus survived at least 15 days in the brain. Lépine (1932) (*4*) reported the recovery of the epidemic virus from white mice 64 days after inoculation. Then Laigret and Jadin (1933) (*5*) found that, in white mice

[1] Read before the laboratory section of the American Public Health Association at the sixty-sixth annual meeting in New York, N. Y., October 8, 1937.

by the brain to peritoneal method, the epidemic virus was lost after two passages at 10-day intervals, while the endemic virus survived 16 passages. They reported also that the endemic virus could be recovered up to the 40th day after inoculation. The passage work was confirmed in America by Savoor and Velasco (1934) (*6*), who passed the endemic virus through 11 sets of white mice at 10-day intervals with no loss of virulence. Kligler, Aschner, and Levine (1936) (*7*) successfully transferred by the brain-to-brain method the endemic virus through 31 generations of white mice at 6- to 10-day intervals and noted no changes in the characteristics of the strain.

On the isolation of typhus virus from mice, one finds that Mooser, Castaneda, and Zinsser (1931) (*8*) failed to obtain the virus from mice trapped at points where infected rats were found. Lépine (1934) (*9*) reported the absence of the endemic virus from mice caught in Athens. Sparrow (1935) (*10*), utilizing 300 mice trapped in Tunis, isolated two strains of the endemic virus. Again, in 1936, Lépine and Lorando (*11*) failed to recover the virus from mice trapped in infected areas. In 1937, Brigham (*12*) reported the isolation of a strain of endemic typhus virus from a native field mouse trapped in the infected area of Alabama. This observation suggested a possible explanation for the occurrence of human cases of typhus in the rural areas of Alabama and encouraged further investigations of the susceptibility of other native rodents. Since two species of rodents have been shown to act as reservoirs of the disease in this country, it seems probable that other susceptible species may act as reservoirs of the infection in the future if not at the present time.

The susceptibility of the house mouse (*Mus musculus musculus*), the meadow mouse (*Microtus pennsylvanicus pennsylvanicus*) and the white-footed mouse (*Peromyscus leucopus noveboracensis*) to endemic typhus virus was reported by Dyer (1934) (*13*). Brigham (*14*) showed, in 1937, that three species of field mice, the old-field mouse (*Peromyscus polionotus polionotus*), the cotton mouse (*Peromyscus gossypinus gossypinus*) and the golden mouse (*Peromyscus nuttalli aureolus*), were susceptible to the endemic virus.

At this time are presented the results of the passage and survival of the endemic typhus virus in native field mice. The two species utilized, the cotton mouse and the old-field mouse, are exceedingly prevalent in the fields of southeastern Alabama.

The mice used [2] were all obtained from this one area in Alabama and supplied to the laboratory as needed. A known strain of endemic typhus was used as the source of the virus in all of the experiments.[3] Two mice were used for each generation of a passage except as will be noted later. All of the animals were inoculated by the intraperitoneal

[2] All mice were identified by courtesy of the National Museum, Washington, D. C.
[3] Wilmington strain, isolated by Maxcy in 1928.

route. The brains of the mice were removed, pooled, ground in a mortar, and saline was added; then the material was injected into fresh mice and guinea pigs. The guinea pigs were observed for the customary clinical picture—fever and scrotal involvement—and later tested for immunity to a known typhus strain. As a further check, the brains [4] of a few guinea pigs were examined for the typhus lesions.

To rule out natural infections as far as possible, a few mice of each lot received from the field were killed and their brains injected into guinea pigs. No strains of typhus were thus obtained.

COTTON MICE

The experiment on cotton mice was started in September 1936 and continued through June 1937. The original set of mice were given 0.25 cc of testicular washings from a guinea pig at the height of the infection. At all subsequent transfers in the series, 0.2 cc of the mouse brain emulsion was injected into each mouse and 4 to 5 cc were injected into each of two fresh guinea pigs. Every 14 days a passage was made except in the 6th, 9th, and 10th generations, in which the time interval was 15, 16, and 16 days, respectively. The serial transfer was terminated in the 20th generation because the guinea-pig controls of the 17th, 18th, 19th, and 20th generations had shown no clinical signs of typhus fever. These animals were found to be susceptible to typhus when reinoculated with a known strain. Out of 33 guinea-pig controls through the 16th passage, 24 had produced typical reactions; 5, fever only; 1, no fever; and 3 had died of secondary infections. Twenty-six guinea pigs were proved to be immune, and typical brain lesions were noted in four animals.

During the passage the virulence of the strain, as judged by the incubation period, was variable. The incubation period in guinea pigs showed a gradual shortening from an average of 8 days in the first 5 generations to 7 days through the 10th, then it dropped to 5 days through the 13th, when the time lengthened to 9 days through the 16th, the last generation to show a reaction. Of all of the 40 mice utilized, none showed any signs of illness, although 5 were found dead on the 14th day after inoculation—1 each in the 1st, 8th, 11th, 14th, and 15th generations of the series.

A new experiment was begun in the cotton mouse with the inoculations performed as before except that transfers were made at 10-day instead of at 14-day intervals. This passage was terminated voluntarily in the 5th generation, since the virus was recovered from the first set of mice only. At least, no clinical picture was shown by the control guinea pigs of the other 4 sets of mice. The guinea pig controls of the first set had incubation periods of 6 and 11 days.

[4] All brain examinations were made by Dr. Lillie of the National Institute of Health, Washington, D. C.

One of the two control guinea pigs of the second set died, while the other had either an inapparent infection or a natural immunity, as it was immune on reinfection with the Wilmington strain. The remaining 6 guinea pigs gave no signs of infection and were not immune. It is very possible that the time interval of 10 days between transfers was not sufficient to allow for the development of the virus in the brain of this species of mouse. A similar experience was reported by Laigret and Jadin (1933) (*5*) in their work on the white mouse.

OLD-FIELD MICE

In the old-field mice the passages were carried out so that the time interval between transfers alternated between 10 and 11 days. The amount of inoculum to each original mouse was 0.15 cc of testicular washings from a routine transfer of the Wilmington strain. The subsequent inoculations of brain emulsion were 0.15 cc to each mouse and 4 to 5 cc to each of 2 fresh guinea pigs. This series is now in the 36th generation. Two mice were used in each set until the 11th generation, when 3 mice were utilized. Starting at the 8th passage, 1 mouse was found dead, and 1 in each subsequent passage through the 14th. The mice were usually found dead on the 8th or 9th day after inoculation. One mouse in each of the next 6 sets of mice (through the 20th) has appeared ill. Deaths of one mouse occurred in each of the 26th, 27th, and 28th passages, and fatalities occurred again in the 33d, 34th, 35th, and 36th generations. Dyer (1934) (*13*) and Brigham (1937) (*14*) reported that this species of mouse shows evidence of infection by a listless and ruffled appearance. An almost sure sign of subsequent death is evidenced by the animal appearing on top of the litter in the jar during the day.

In the control guinea pigs, 62 animals showed reactions typical of typhus, 3 guinea pigs produced fever only, 1 gave no reaction, and 6 died of intercurrent infections. Fifty-five of the guinea pigs have been immune on reinoculation with a known typhus strain. The brains of 3 guinea pigs showed the typical typhus lesions. The virulence of the strain as indicated by the incubation period in guinea pigs has been retained. The time interval averaged 6 days in the first 7 passages, 8 days in the next 4 generations, and 6 days up to the last transfer reported, the 36th. In the old-field mouse the virus apparently multiplies with sufficient rapidity within the 10-day interval between passages to allow the infection to be carried on indefinitely at 10-day intervals. The virus has not as yet shown in the control guinea pigs any tendency towards a loss or gain in any of its original characteristics, even though it has now been maintained exclusively in old-field mice for over 1 year (384 days).

For a further control on the possible presence of natural infections in old-field mice, a serial passage, using supposedly normal mice of this species, has been carried on simultaneously and in an identical manner as in the infected series. However, it was not started until the time of the 15th passage of the infected series. Twenty transfers have been made, and no mice have shown any signs of illness although 4 have died. All of the deaths were apparently due to mechanical injury, as they occurred within 3 days of the inoculation. The control guinea pigs of this uninfected group have produced no signs of infection and have all been susceptible to the virus on reinfection with the Wilmington strain.

PRESERVATION OF VIRUS

Studies have been conducted to determine the length of time after inoculation that the virus can be recovered from the brains of the various species of mice. In one experiment, similar to the experiments reported, the virus was recovered from the brains of cotton mice 18 days, 22 days, and 23 days after the inoculation. In a later experiment, a group of cotton mice were inoculated with 0.2 cc each of testicular washings from a guinea pig at a routine transfer of the Wilmington strain. One of these mice was killed 51 days after inoculation and a strain was recovered from its brain. Another mouse of this group was killed 141 days after the inoculation and a strain was recovered. This strain was passed through 12 generations, 46 guinea pigs being used, and no changes were noted in the characteristics of the virus, when compared with the Wilmington strain. Out of the group, one mouse lived 160 days but was found dead on the 161st day. No strain was recovered from the brain of this mouse.

In the old-field species, mice were inoculated with testicular washings as in the case of the cotton mice. The virus was recovered from their brains twice after 23 days, once after 25 days, once after 63 days, and once after 141 days. Here also the typical clinical picture was obtained in the guinea pig.

From a less prevalent species of field mouse, the golden mouse (*Peromyscus nuttalli aureolus*), the virus was isolated 76 days after inoculation of the infective material.

SUMMARY

Endemic typhus virus can be maintained in native field mice which are prevalent in the typhus area of Alabama. In the cotton mouse, the virus was transferred through 16 generations before it was lost. In old-field mice the virus has been carried through 36 passage generations. The virus has been found to survive 141 days in the cotton mouse, 141 days in the old-field mouse, and 76 days in the golden mouse, the extent of our experiment.

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A RAPID METHOD FOR DRYING THICK BLOOD FILMS*

By MARTIN D. YOUNG, *Junior Zoologist, United States Public Health Service*

The time required for drying thick blood films before they can be moved or handled often inconveniences and delays the workers. This is especially true on very humid days, when a thick film may require over an hour to dry.

Recently a method of rapid drying has been instituted at the Williams Malaria Research Laboratory, Columbia, S. C., which has proved very satisfactory. An ordinary hot-air hair dryer is used. The dryer is held at a distance above the blood film, about 24 inches, so that the blood is not agitated by the current of air. The heated air is directed against the film, which thus is dried much more rapidly than it would be under ordinary circumstances. The staining reactions of the rapidly dried films are not affected.

*Contribution from the Williams Malaria Research Laboratory for Field Investigations of Malaria, of the National Institute of Health, United States Public Health Service, located at the South Carolina State Hospital, Columbia, S. C.

On a series of tests, the films dried under the hair dryer in one-tenth of the time required for films dried under ordinary room conditions. Considerable time is thus saved when it is desirable to handle and stain films as expeditiously as possible.

DEATHS DURING WEEK ENDED JULY 2, 1938

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

	Week ended July 2, 1938	Corresponding week, 1937
Data from 88 large cities of the United States:		
Total deaths	7,577	[1] 7,468
Average for 3 prior years	7,647	
Total deaths, first 26 weeks of year	221,943	243,106
Deaths under 1 year of age	519	[1] 547
Average for 3 prior years	536	
Deaths under 1 year of age, first 26 weeks of year	13,820	15,137
Data from industrial insurance companies:		
Policies in force	69,248,240	70,021,076
Number of death claims	11,389	11,729
Death claims per 1,000 policies in force, annual rate	8.6	8.7
Death claims per 1,000 policies, first 26 weeks of year, annual rate	9.7	10.7

[1] Data for 86 cities.

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

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

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

Cases of certain diseases reported by telegraph by State health officers for the week ended July 9, 1938, rates per 100,000 population (annual basis), and comparison with week ended July 10, 1937 and 5-year median

Division and State	Diphtheria				Influenza				Measles			
	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–37 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–37 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–37 median
NEW ENG.												
Maine	0	0	1	0	6	1	------	------	79	13	21	21
New Hampshire	0	0	1	0	------	------	------	------	317	31	47	6
Vermont	0	0	1	0	------	------	------	------	408	30	6	41
Massachusetts	0	0	6	6	------	------	------	------	273	232	215	299
Rhode Island	8	1	0	0	------	------	------	------	23	3	20	20
Connecticut	3	1	4	2	6	2	------	1	60	20	43	78
MID. ATL.[2]												
New York	8	21	39	33	[1] 0.7	[1] 1	------	[1] 1	523	1,299	637	697
New Jersey	10	8	10	17	8	7	2	2	249	207	517	442
Pennsylvania	14	28	18	30	------	------	------	------	323	630	630	630
E. NO. CEN.[2]												
Ohio	6	8	6	16	------	------	2	2	191	246	483	387
Indiana	6	4	15	7	------	------	8	8	197	131	152	34
Illinois	17	25	35	35	3	4	10	10	117	177	326	326
Michigan[3]	6	6	20	10	------	------	------	------	771	714	280	179
Wisconsin	4	2	8	1	18	10	18	8	1,524	855	51	127
W. NO. CEN.												
Minnesota	2	1	1	1	------	------	------	------	265	135	2	31
Iowa	0	0	2	3	------	------	------	------	249	122	6	7
Missouri	18	14	3	14	8	6	24	11	47	36	16	39
North Dakota	7	1	0	1	7	1	------	------	303	41	------	1
South Dakota	8	1	0	2	------	------	------	------	------	------	------	5
Nebraska	4	1	2	3	------	------	------	------	111	29	7	14
Kansas	8	3	5	5	------	------	------	------	87	31	4	34

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 9, 1938, rates per 100,000 population (annual basis), and comparison with week ended July 10, 1937 and 5-year median—Continued

Division and State	Diphtheria				Influenza				Measles			
	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–37 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–37 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–37 median
SO. ATL.[2 4]												
Delaware	20	1	0	1					40	2	2	6
Maryland[3]	3	1	3	3	3	1		1	137	44	16	32
Dist. of Col	17	2	3	3					100	12	34	20
Virginia	50	26	6	6					291	151	78	78
West Virginia	11	4	2	11	31	11	4	4	137	49	34	34
North Carolina	7	5	6	6	9	6			503	337	127	74
South Carolina	28	10	1	1	250	90	53	53	278	100	18	18
Georgia	15	9	7	3								
Florida	16	5	6	4			2		31	10	1	9
E. SO. CEN.[4]												
Kentucky	4	2	5	5	5	3	1	1	80	45	96	53
Tennessee	13	7	3	5	14	8	14	5	83	46	69	35
Alabama	16	9	6	7	18	10	2	3	76	42	25	24
Mississippi[3]	8	3	4	4								
W. SO. CEN.[4]												
Arkansas	10	4	4	3	25	10	3	3	115	45	9	5
Louisiana	27	11	9	10	22	9	43	9	10	4	5	11
Oklahoma	8	4	3	5	55	27	4	6	35	17	17	17
Texas	11	13	14	21	76	90	60	44	34	40	147	147
MOUNTAIN[2 4]												
Montana[3]	0	0	0	1							8	10
Idaho	11	1	0	0			2	1	11	1	3	3
Wyoming	22	1	0	0					67	3		1
Colorado	58	12	2	3					268	55	36	36
New Mexico	25	2	1	2			1	1	62	5	14	13
Arizona	0	0	1	3	139	11	6	4	215	17	15	15
Utah[3]	70	7	0	0					975	97	41	27
PACIFIC[4]												
Washington	0	0	2	1					35	11	60	69
Oregon	5	1	1	1	36	7	6	5	71	14	3	14
California	23	27	22	24	9	11	10	15	334	394	84	343
Total	12	292	288	347	16	326	275	253	268	6,523	4,385	5,642
27 weeks	19	12,479	11,937	15,878	82	44,016	273,110	140,369	1,133	745,963	229,118	331,471

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median
NEW ENG.												
Maine	0	0	0	0	12	2	2	0	49	8	3	6
New Hampshire	0	0	0	0	0	0	0	0	20	2	6	6
Vermont	0	0	0	0	27	2	0	0	82	6	2	2
Massachusetts	0	0	3	1	1	1	3	1	112	95	73	74
Rhode Island	0	0	0	0	0	0	0	0	46	6	16	6
Connecticut	0	0	0	0	0	0	2	0	69	23	25	23
MID. ATL.[2]												
New York	0.8	2	15	11	4	10	6	6	57	141	212	212
New Jersey	0	0	2	1	0	0	1	1	37	31	43	43
Pennsylvania	1	2	6	4	0	0	0	0	63	122	131	177

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 9, 1938, rates per 100,000 population (annual basis), and comparison with week ended July 10, 1937 and 5-year median—Continued

Division and State	Meningitis, meningococcus				Poliomyelitis				Scarlet fever			
	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median
E. NO. CEN.[2]												
Ohio	1.5	2	2	2	0.8	1	9	1	69	89	100	113
Indiana	0	0	1	1	1.5	1	1	1	45	30	28	27
Illinois	0.7	1	3	8	1.3	2	2	2	88	133	149	190
Michigan[3]	1	1	1	0	0	0	1	0	146	135	237	158
Wisconsin	0	0	0	1	1.7	1	1	1	82	46	83	87
W. NO. CEN.												
Minnesota	2	1	0	0	0	0	0	1	49	25	39	39
Iowa	2	1	0	0	4	2	0	0	37	18	23	23
Missouri	0	0	1	1	1.3	1	22	1	39	30	19	19
North Dakota	0	0	1	1	0	0	0	0	37	5	8	4
South Dakota	0	0	0	0	0	0	0	0	23	3	9	9
Nebraska	4	1	1	0	0	0	0	0	19	5	13	13
Kansas	0	0	0	0	0	0	3	1	70	25	40	23
SO. ATL.[3 4]												
Delaware	0	0	0	0	0	0	1	0	40	2	2	2
Maryland[3]	0	0	0	0	0	0	0	1	37	12	12	18
Dist. of Col.	0	0	0	0	0	0	0	0	42	5	2	7
Virginia	2	1	3	3	0	0	3	1	21	11	6	13
West Virginia	0	0	6	1	0	0	0	1	28	10	11	18
North Carolina	1.5	1	2	2	1.5	1	4	0	25	17	15	14
South Carolina	3	1	0	0	0	0	0	0	17	6	3	2
Georgia	0	0	0	0	1.7	1	6	0	14	8	10	4
Florida	3	1	3	0	0	0	0	0	3	1	-------	-------
E. SO. CEN.[4]												
Kentucky	1.8	1	3	2	1.8	1	7	1	21	12	15	14
Tennessee	5	3	3	2	0	0	11	5	23	13	7	11
Alabama	9	5	4	1	9	5	4	2	20	11	5	7
Mississippi[3]	0	0	4	0	8	3	20	0	18	7	1	5
W. SO. CEN.[4]												
Arkansas	0	0	5	1	0	0	36	0	5	2	4	2
Louisiana	0	0	2	0	0	0	8	1	20	8	4	4
Oklahoma	4	2	1	1	2	1	55	1	18	9	8	7
Texas	0	0	4	1	0	0	36	3	35	41	26	26
MOUNTAIN[3 6]												
Montana[5]	0	0	0	0	0	0	1	0	-------	-------	12	9
Idaho	0	0	1	0	0	0	0	0	-------	-------	15	2
Wyoming	0	0	0	0	0	0	0	0	44	2	5	7
Colorado	5	1	0	0	0	0	0	0	88	18	11	11
New Mexico	0	0	0	0	0	0	1	0	111	9	3	3
Arizona	13	1	1	0	13	1	2	0	51	4	12	3
Utah[3]	0	0	0	0	0	0	0	0	100	10	6	6
PACIFIC[4]												
Washington	0	0	0	1	0	0	0	0	41	13	10	17
Oregon	0	0	0	0	5	1	0	0	56	11	5	10
California	0	0	5	2	3	4	8	8	53	63	81	81
Total	1.1	28	83	78	1.7	41	256	156	52	1,283	1,550	1,550
27 weeks	2.9	1,926	3,792	3,708	0.9	589	1,071	1,071	197	131,647	158,823	158,823

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 9, 1938, rates per 100,000 population (annual basis), and comparison with week ended July 10, 1937 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough	
	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases
NEW ENGLAND										
Maine	0	0	0	0	12	2	0	0	146	24
New Hampshire	0	0	0	0	0	0	0	0	-------	-------
Vermont	0	0	0	0	0	0	0	0	218	16
Massachusetts	0	0	0	0	1	1	1	2	108	92
Rhode Island	0	0	0	0	0	0	0	0	230	30
Connecticut	0	0	0	0	12	4	2	0	237	79
MIDDLE ATLANTIC [2]										
New York	0	0	0	0	2	5	19	8	163	406
New Jersey	0	0	0	0	4	3	3	3	285	237
Pennsylvania	0	0	0	0	3	5	13	13	108	211
EAST NORTH CENTRAL [3]										
Ohio	2	3	1	1	10	13	10	10	121	156
Indiana	24	16	3	1	11	7	17	5	23	15
Illinois	7	11	4	2	9	13	6	12	193	292
Michigan [3]	2	2	0	0	2	2	3	5	324	300
Wisconsin	5	3	1	9	4	2	0	2	330	185
WEST NORTH CENTRAL										
Minnesota	10	5	2	2	2	1	0	2	59	30
Iowa	33	16	19	7	0	0	1	1	22	11
Missouri	13	10	4	2	22	17	14	12	76	58
North Dakota	96	13	11	2	0	0	0	0	96	13
South Dakota	45	6	0	0	0	0	0	0	105	14
Nebraska	8	2	1	6	0	0	2	0	11	3
Kansas	0	0	0	0	11	4	6	6	344	123
SOUTH ATLANTIC [2 4]										
Delaware	0	0	0	0	0	0	0	0	140	7
Maryland [3]	0	0	0	0	19	6	6	6	112	36
District of Columbia	0	0	0	0	17	2	4	0	33	4
Virginia	0	0	0	0	60	31	16	16	466	242
West Virginia	0	0	0	0	3	1	10	10	179	64
North Carolina	1.5	1	0	0	43	29	19	19	421	282
South Carolina	0	0	0	0	72	26	21	21	578	208
Georgia	0	0	0	0	74	44	38	38	127	75
Florida	0	0	0	0	3	1	1	1	50	16
EAST SOUTH CENTRAL [4]										
Kentucky	7	4	2	1	50	28	26	26	100	56
Tennessee	0	0	0	0	52	29	24	24	150	83
Alabama	4	2	2	0	34	19	19	20	70	39
Mississippi [3]	0	0	0	0	34	13	20	16	-------	-------
WEST SOUTH CENTRAL [4]										
Arkansas	0	0	0	0	43	17	34	21	92	36
Louisiana	0	0	0	0	37	15	21	20	113	46
Oklahoma	6	3	0	0	8	4	16	19	18	9
Texas	1.7	2	6	6	69	82	61	61	221	261
MOUNTAIN [2 6]										
Montana [3]	319	33	18	9	10	1	1	2	-------	-------
Idaho	21	2	4	0	11	1	0	0	-------	-------
Wyoming	0	0	0	1	0	0	0	0	67	3
Colorado	0	0	3	2	19	4	2	2	131	27
New Mexico	25	2	0	0	86	7	2	5	358	29
Arizona	25	2	0	0	38	3	4	3	620	49
Utah [3]	0	0	1	0	20	2	0	0	663	66

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended July 9, 1938, rates per 100,000 population (annual basis), and comparison with week ended July 10, 1937 and 5-year median—Continued

Division and State	Smallpox				Typhoid and paratyphoid fever				Whooping cough	
	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases	July 10, 1937, cases	1933–1937 median	July 9, 1938, rate	July 9, 1938, cases
PACIFIC [4]										
Washington	60	19	2	6	0	0	1	1	60	19
Oregon	132	26	10	2	0	0	0	6	208	41
California	8	10	2	3	5	6	11	6	182	215
Total	8	193	96	96	18	450	454	454	173	4,208
27 weeks	18	12,130	7,466	4,976	7	4,749	4,245	5,009	176	115,995

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended July 9, 1938, 24 cases as follows: New Jersey, 4; Indiana, 1; Illinois, 1; Virginia, 9; North Carolina, 7; Idaho, 1; Colorado, 1.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended July 9, 1938, 66 cases as follows: Maryland, 1; North Carolina, 2; South Carolina, 2; Georgia, 28; Florida, 1; Alabama, 12; Louisiana, 3; Texas, 16; California, 1.
[5] Montana.—Delayed report: Diphtheria. 2; measles, 28; poliomyelitis, 1; scarlet fever, 4; whooping cough, 50. These figures are included in the 27-weeks totals but not in the reports for the current week.
[6] Colorado tick fever, week ended July 9, 1938, 10 cases as follows: Wyoming, 6; Colorado, 4.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Meningitis, meningococcus	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
May 1938										
Alaska	0	0	12	----	----	----	0	0	0	0
Colorado	5	50	149	----	1,179	----	0	150	36	11
Wisconsin	3	12	89	----	10,632	----	2	569	15	5
June 1938										
Arkansas	1	18	40	712	505	197	2	17	22	70
Connecticut	1	13	14	----	277	----	1	297	0	5
Delaware	0	3	----	2	?3	----	0	20	0	4
Idaho	0	1	18	----	37	----	0	20	46	9
Iowa	3	5	2	8	1,036	----	1	180	114	3
Michigan	3	31	2	4	8,369	----	1	1,034	5	17
Nebraska	2	15	----	----	611	----	0	65	0	0
West Virginia	7	16	25	----	582	2	1	67	1	17

Summary of monthly reports from States—Continued

May 1938

	Cases
Chickenpox:	
Alaska	23
Colorado	305
Wisconsin	1,350
Dysentery:	
Colorado (amoebic)	2
Colorado (bacillary)	2
German measles:	
Wisconsin	57
Mumps:	
Alaska	10
Colorado	67
Wisconsin	1,027
Rocky Mountain spotted fever:	
Colorado	3
Scabies:	
Alaska	2
Septic sore throat:	
Alaska	3
Colorado	2
Wisconsin	8
Trachoma:	
Wisconsin	1
Tularaemia:	
Wisconsin	3
Undulant fever:	
Wisconsin	7
Whooping cough:	
Colorado	127
Wisconsin	795

June 1938

	Cases
Actinomycosis:	
Michigan	1
Chickenpox:	
Arkansas	40
Connecticut	329
Delaware	43
Idaho	15
Iowa	173
Michigan	1,000
Nebraska	117
West Virginia	105

June 1938—Continued

	Cases
Conjunctivitis:	
Connecticut	1
Idaho	3
Dysentery:	
Arkansas (amoebic)	7
Arkansas (bacillary)	290
Connecticut (bacillary)	16
Iowa (amoebic)	1
Michigan (amoebic)	1
Michigan (bacillary)	11
Encephalitis, epidemic or lethargic:	
Idaho	1
Iowa	2
German measles:	
Connecticut	21
Delaware	1
Idaho	6
Iowa	4
Michigan	323
Hookworm disease:	
Arkansas	3
Lead poisoning:	
Connecticut	1
Idaho	1
Mumps:	
Arkansas	35
Connecticut	391
Delaware	41
Idaho	27
Iowa	92
Michigan	506
Nebraska	121
West Virginia	10
Paratyphoid fever:	
Arkansas	4
Connecticut	6
Michigan	1
West Virginia	3
Puerperal septicemia:	
Arkansas	1
Rabies in animals:	
Arkansas	22
Connecticut	8
Iowa	3
Michigan	2

June 1938—Continued

	Cases
Rocky Mountain spotted fever:	
Delaware	1
Idaho	13
Iowa	1
West Virginia	1
Septic sore throat:	
Arkansas	12
Connecticut	12
Idaho	4
Iowa	1
Michigan	5
Nebraska	10
West Virginia	3
Tetanus:	
Connecticut	3
Michigan	5
Trachoma:	
Arkansas	12
Michigan	2
Trichinosis:	
Connecticut	1
Michigan	1
Tularaemia:	
Arkansas	9
Typhus fever:	
Connecticut	1
Undulant fever:	
Arkansas	1
Connecticut	9
Iowa	12
Michigan	8
Vincent's infection:	
Idaho	3
Michigan	8
Whooping cough:	
Arkansas	167
Connecticut	420
Delaware	44
Idaho	27
Iowa	102
Michigan	1,344
Nebraska	55
West Virginia	348

PLAGUE INFECTION IN GROUND SQUIRRELS AND FLEAS IN BEAVERHEAD COUNTY, MONT.

Under date of July 8, 1938, Senior Surg. C. R. Eskey reported plague infection proved in pooled tissue from 2 ground squirrels, *Citellus richardsoni*, and in a pool of 98 fleas from 28 ground squirrels of the same species, all taken on June 24, 1938, 10 miles north of Wisdom, on Plempton Creek, Beaverhead County, Mont.

PLAGUE INFECTION IN GROUND SQUIRRELS AND FLEAS IN UINTA COUNTY, WYO.

Under date of July 8, 1938, Senior Surg. C. R. Eskey reported plague infection proved in tissue from 1 ground squirrel, *Citellus armatus*, and in a pool of 19 fleas from 9 *Citellus elegans* all taken on June 27, 1938, 15 miles northwest of Evanston, Uinta County, Wyo.

This is stated to be the first positive demonstration of plague existing among wild rodents in the State of Wyoming.

CASES OF VENEREAL DISEASES REPORTED FOR MAY 1938

These reports are published monthly for the information of health officers in order to furnish current data as to the prevalence of the venereal diseases. The figures are taken from reports received from State and city health officers. They are preliminary and are therefore subject to correction. It is hoped that the publication of these reports will stimulate more complete reporting of these diseases.

Reports from States

	Syphilis		Gonorrhea	
	Cases reported during month	Monthly case rates per 10,000 population	Cases reported during month	Monthly case rates per 10,000 population
Alabama	2,030	7.01	274	0.95
Arizona	79	1.92	76	1.84
Arkansas	1,305	6.37	262	1.28
California	2,127	3.46	1,300	2.11
Colorado [1]				
Connecticut	260	1.49	111	.64
Delaware	252	9.66	55	2.11
District of Columbia	230	3.67	233	3.72
Florida [2]	2,110	12.63	260	1.56
Georgia	2,820	9.14	381	1.24
Idaho	24	.49	31	.63
Illinois	2,203	2.80	1,020	1.29
Indiana	278	.80	132	.38
Iowa [1]				
Kansas	163	.87	83	.45
Kentucky	649	2.22	296	1.01
Louisiana	999	4.69	120	.56
Maine	57	.67	43	.50
Maryland	1,177	7.01	273	1.63
Massachusetts	507	1.15	366	.83
Michigan	1,224	2.53	485	1.00
Minnesota	255	.96	191	.72
Mississippi	2,883	14.25	2,648	13.09
Missouri	642	1.61	217	.54
Montana [1]				
Nebraska	48	.35	67	.49
Nevada	47	4.65	16	1.58
New Hampshire	17	.33	12	.24
New Jersey	1,060	2.44	293	.67
New Mexico	80	1.90	34	.81
New York [1]				
North Carolina	3,915	11.21	522	1.49
North Dakota	37	.52	27	.38
Ohio [2]	1,826	2.71	404	.60
Oklahoma [2]	431	1.69	278	1.09
Oregon	100	.97	155	1.51
Pennsylvania [2]	1,497	1.47	185	.18
Rhode Island	113	1.66	43	.63
South Carolina [1]				
South Dakota	47	.68	20	.29
Tennessee	1,030	3.56	310	1.07
Texas [2]	1,050	1.70	381	.62
Utah	23	.44	19	.37
Vermont	10	.26	29	.76
Virginia	1,052	3.89	295	1.09
Washington	300	1.81	337	2.03
West Virginia [2]	370	1.98	108	.58
Wisconsin	34	.12	100	.34
Wyoming [2]	7	.30	4	.17
Total	35,368	3.21	12,496	1.13

See footnotes at end of table.

Reports from cities of 200,000 population or over

	Syphilis		Gonorrhea	
	Cases reported during month	Monthly case rates per 10,000 population	Cases reported during month	Monthly case rates per 10,000 population
Akron, Ohio [1]				
Atlanta, Ga	340	11.84	137	4.77
Baltimore, Md	700	8.48	171	2.07
Birmingham, Ala	375	13.28	48	1.70
Boston, Mass	175	2.21	128	1.62
Buffalo, N. Y	140	2.37	56	.95
Chicago, Ill	1,542	4.32	723	2.03
Cincinnati, Ohio	232	4.98	77	1.65
Cleveland, Ohio	278	2.99	80	.86
Columbus, Ohio	117	3.83	26	.85
Dallas, Tex	240	8.29	64	2.21
Dayton, Ohio [1]				
Denver, Colo. [1]				
Detroit, Mich	624	3.60	230	1.33
Houston, Tex. [1]				
Indianapolis, Ind	23	.61	24	.64
Jersey City, N. J. [1]				
Kansas City, Mo	87	2.07	3	.07
Los Angeles, Calif	687	4.80	409	2.86
Louisville, Ky	305	9.41	124	3.83
Memphis, Tenn	382	14.31	77	2.88
Milwaukee, Wis. [4]				
Minneapolis, Minn	67	1.38	46	.95
Newark, N. J	342	7.38	98	2.11
New Orleans, La. [1]				
New York, N. Y. [1]				
Oakland, Calif	83	2.74	33	1.09
Omaha, Nebr	19	.86	14	.64
Philadelphia, Pa	546	2.75		
Pittsburgh, Pa	219	3.20	16	.23
Portland, Oreg	46	1.47	103	3.28
Providence, R. I	71	2.74	28	1.08
Rochester, N. Y	37	1.10	37	1.10
St. Louis, Mo	284	3.40	111	1.33
St. Paul, Minn	34	1.21	20	.71
San Antonio, Tex	120	4.77	57	2.27
San Francisco, Calif	191	2.85	156	2.33
Seattle, Wash	136	3.58	133	3.50
Syracuse, N. Y	57	2.62	9	.41
Toledo, Ohio [1]				
Washington, D. C. [5]	230	3.67	233	3.72

[1] No report for current month.
[2] Incomplete.
[3] Only cases of syphilis in the infectious stage are reported.
[4] No report during present fiscal year.
[5] Reported by Social Hygiene Clinic.

WEEKLY REPORTS FROM CITIES

City reports for week ended July 2, 1938

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

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	146	48	17	2,586	359	858	10	387	57	1,261	----
Current week [1]	102	20	10	2,139	300	491	6	335	22	1,290	----
Maine:											
Portland	0	----	0	6	1	0	0	0	0	4	10
New Hampshire:											
Concord	0	----	0	0	1	0	0	0	0	0	5
Manchester	0	----	0	0	1	0	0	0	0	0	15
Nashua	0	----	0	0	0	0	0	0	0	0	10
Vermont:											
Barre	0	----	0	0	0	0	0	0	0	0	1
Burlington	0	----	0	1	0	0	0	0	1	1	5
Rutland	0	----	0	0	0	0	0	0	0	0	3
Massachusetts:											
Boston	0	----	0	143	11	24	0	8	0	13	205
Fall River	0	----	0	1	0	1	0	3	0	3	34
Springfield	0	----	0	146	0	0	0	0	0	12	30
Worcester	0	----	0	2	1	4	0	1	0	7	52
Rhode Island:											
Pawtucket	0	----	0	0	0	1	0	0	0	0	7
Providence	2	----	0	0	4	4	0	3	0	13	62
Connecticut:											
Bridgeport	0	----	1	3	1	2	0	1	0	5	28
Hartford	0	----	0	5	2	2	0	1	0	6	35
New Haven	0	----	0	1	0	1	0	0	0	15	30
New York:											
Buffalo	2	----	0	4	5	10	0	5	0	16	121
New York	13	2	1	681	56	79	0	57	2	250	1,259
Rochester	0	----	0	33	3	4	0	2	0	5	58
Syracuse	0	----	0	84	2	8	0	0	0	12	33
New Jersey:											
Camden	3	----	0	0	2	2	0	3	0	3	32
Newark	0	----	0	5	1	8	0	13	0	36	83
Trenton	0	----	1	1	0	0	0	1	0	2	29
Pennsylvania:											
Philadelphia	1	----	0	127	9	30	0	21	4	47	419
Pittsburgh	5	1	0	8	6	16	0	6	0	17	134
Reading	0	----	0	6	0	0	0	2	0	4	15
Scranton	0	----	----	2	----	2	0	----	0	0	----
Ohio:											
Cincinnati	2	----	0	4	8	6	0	10	0	15	131
Cleveland	4	3	0	98	7	18	0	14	1	50	173
Columbus	0	----	0	1	2	2	0	6	0	0	77
Toledo	1	----	0	16	1	7	0	3	0	6	51
Indiana:											
Anderson	0	----	0	0	0	0	1	0	0	0	5
Fort Wayne	0	----	0	0	3	0	0	0	0	0	26
Indianapolis	5	----	0	21	5	6	5	9	0	3	101
Muncie	0	----	0	0	1	0	0	0	0	0	13
South Bend	0	----	0	2	0	1	0	1	0	3	19
Terre Haute	0	----	0	2	0	0	0	0	0	0	21
Illinois:											
Alton	0	----	0	0	0	0	0	0	0	1	14
Chicago	14	----	0	46	20	82	0	43	1	139	641
Elgin	0	----	0	0	1	2	0	0	0	0	8
Moline	0	----	0	0	0	1	0	0	0	3	8
Springfield	0	----	0	0	0	3	0	0	0	6	25
Michigan:											
Detroit	11	----	0	41	17	48	0	8	0	133	239
Flint	0	----	0	23	4	6	0	0	0	19	18
Grand Rapids	0	----	0	50	1	5	0	0	0	0	23
Wisconsin:											
Kenosha	0	----	0	19	0	0	0	0	0	1	9
Madison	0	----	0	73	0	0	----	0	0	1	18
Milwaukee	0	----	0	12	4	15	0	2	0	87	82
Racine	0	----	0	9	0	0	0	0	0	15	13
Superior	0	----	0	9	0	0	0	0	0	0	12

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

City reports for week ended July 2, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0	------	0	26	1	0	0	1	0	9	23
Minneapolis	0	------	0	40	2	6	0	2	0	1	88
St. Paul	0	------	0	20	7	2	0	3	0	6	58
Iowa:											
Cedar Rapids	0	------	--------	10	-------	1	0	-------	0	2	--------
Davenport	0	------	--------	0	-------	0	-------	-------	0	0	--------
Des Moines	0	------	0	3	0	6	2	0	0	0	32
Sioux City	0	------	--------	47	-------	3	0	-------	0	4	--------
Waterloo	0	------	--------	5	-------	0	0	-------	0	1	--------
Missouri:											
Kansas City	1	------	0	0	3	3	0	4	0	3	95
St. Joseph	1	------	0	0	1	2	0	0	0	0	12
St. Louis	3	------	0	2	3	4	0	9	0	0	184
North Dakota:											
Fargo	0	------	0	2	0	0	0	0	0	3	8
Grand Forks	0	------	--------	2	-------	0	0	-------	0	0	--------
Minot	0	------	0	5	0	0	0	0	0	1	12
South Dakota:											
Sioux Falls	0	------	0	0	0	0	0	0	0	0	10
Nebraska:											
Omaha	0	------	0	28	4	0	0	2	0	0	58
Kansas:											
Lawrence	0	------	0	2	0	0	0	0	0	5	5
Topeka	0	------	0	7	1	0	0	0	0	32	12
Wichita	0	------	0	21	0	0	0	1	0	7	37
Delaware:											
Wilmington	0	------	0	0	3	3	0	0	0	7	30
Maryland:											
Baltimore	1	1	1	9	10	11	0	8	1	42	198
Cumberland	0	------	0	9	0	0	0	1	0	0	11
Frederick	0	------	--------	0	-------	0	0	-------	0	0	--------
Dist. of Col.:											
Washington	7	------	0	19	4	8	0	6	0	5	139
Virginia:											
Lynchburg	1	------	0	0	1	0	0	0	2	4	11
Norfolk	3	------	0	0	3	0	0	0	0	2	28
Richmond	0	------	0	69	0	1	0	3	0	0	48
Roanoke	0	------	0	0	0	3	0	0	0	4	13
West Virginia:											
Charleston	0	------	0	0	3	2	0	0	0	0	13
Huntington	0	------	--------	0	-------	0	0	-------	0	0	--------
Wheeling	0	------	0	6	1	0	0	1	0	5	16
North Carolina:											
Gastonia	0	------	--------	0	-------	1	0	-------	0	3	--------
Raleigh	0	------	0	5	1	0	0	1	0	6	17
Wilmington	0	------	0	0	0	1	0	1	1	3	15
Winston-Salem	0	------	0	25	0	1	0	0	0	10	13
South Carolina:											
Charleston	0	5	0	1	0	0	0	1	0	0	12
Florence	0	------	0	0	1	0	0	0	1	0	4
Greenville	0	------	0	4	0	0	0	0	2	2	8
Georgia:											
Atlanta	0	------	0	0	6	0	0	5	2	12	78
Brunswick	0	------	0	2	2	0	0	0	0	0	5
Savannah	0	------	0	7	1	0	0	0	0	11	26
Florida:											
Miami	0	------	0	0	4	0	0	2	0	2	27
Tampa	0	------	0	2	0	0	0	0	0	0	18
Kentucky:											
Ashland	1	------	0	0	0	0	0	0	0	1	--------
Covington	2	------	0	0	0	1	0	0	0	8	13
Lexington	0	------	0	1	0	0	0	0	0	0	21
Louisville	0	------	0	7	1	3	0	3	1	3	61
Tennessee:											
Knoxville	2	3	1	0	2	1	0	0	0	49	26
Memphis	2	------	0	0	5	2	0	1	1	3	75
Nashville	1	------	0	5	4	2	0	3	0	7	57
Alabama:											
Birmingham	0	------	0	0	0	1	0	2	1	0	43
Mobile	0	------	0	0	0	1	0	0	1	0	25
Montgomery	0	------	--------	18	-------	0	0	-------	0	16	--------

City reports for week ended July 2, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Arkansas:											
Fort Smith	0			3		0	0		0	1	
Little Rock	0		0	0	1	0	0	2	0	0	6
Louisiana:											
Lake Charles	0		0	0	0	0	0	1	0	0	7
New Orleans	4	2	1	10	9	2	0	8	2	42	135
Shreveport	0		0	0	3	0	0	1	0	0	37
Oklahoma:											
Muskogee	0			4		2	0		1	0	
Oklahoma City	1		0	0	0	2	0	1	0	0	39
Tulsa	0			5		0	0		0	2	
Texas:											
Dallas	1		0	2	3	0	0	3	2	5	47
Fort Worth	0		0	0	1	2	0	0	1	1	34
Galveston	1		0	0	1	0	0	2	0	0	19
Houston	1		0	0	6	1	0	3	1	0	101
San Antonio	0		0	0	4	0	0	6	0	9	61
Montana:											
Billings	0		0	0	0	0	0	0	0	5	8
Great Falls	0		0	0	0	0	0	0	0	13	12
Helena	0		0	0	0	0	0	0	0	0	1
Missoula	0		0	0	1	0	0	0	0	0	6
Idaho:											
Boise	0		0	1	1	0	0	0	0	1	5
Colorado:											
Colorado Springs	0		0	1	0	1	0	0	0	4	5
Denver	8		0	6	2	8	0	2	0	5	66
Pueblo	0		0	22	1	1	0	0	0	5	11
New Mexico:											
Albuquerque	0		0	0	1	0	0	1	0	0	5
Utah:											
Salt Lake City	0		0	112	2	4	0	0	0	4	35
Washington:											
Seattle	0		1	7	2	2	0	7	0	25	95
Spokane	0	1	1	1	2	0	0	0	0	2	30
Tacoma											
Oregon:											
Portland	0		1	9	1	5	0	2	0	2	81
Salem	1	1		1		0	0		0	0	
California:											
Los Angeles	7	5	2	59	17	28	0	19	0	18	311
Sacramento	1		0	18	0	0	0	1	0	5	16
San Francisco	0		0	4	5	2	0	6	0	26	159

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
New York:				Kentucky:			
Buffalo	1	1	0	Lexington	1	0	0
New York	5	2	1	Tennessee:			
Pennsylvania:				Memphis	0	0	1
Pittsburgh	1	0	0	Alabama:			
Illinois:				Birmingham	3	0	2
Chicago	0	0	2	Mobile	0	0	1
Michigan:				Montgomery	0	0	1
Detroit	0	1	0				
Virginia:							
Richmond	0	0	1				

Encephalitis, epidemic or lethargic.—Cases: Rochester, 1; St. Louis, 2.
Pellagra.—Cases: Columbus, 1; Baltimore, 1; Charleston, S. C., 3; Atlanta, 5; Savannah, 1; Birmingham, 1; Mobile, 3; Montgomery, 3; New Orleans, 1; Dallas, 1.
Typhus fever.—Cases: New York, 1; Savannah, 1; Mobile, 1; Montgomery, 1; Lake Charles, 1.

FOREIGN AND INSULAR

AUSTRALIA

Notifiable diseases—1937.—According to a report issued by the Commonwealth Department of Health, the following cases of notifiable diseases were reported in Australia during 1937 (population given as 6,833,375):

Disease	Cases	Disease	Cases
Ancylostomiasis	14	Lethargic encephalitis	18
Anthrax	1	Malaria	20
Beri-beri	7	Measles	239
Cerebrospinal fever	43	Mumps	107
Chickenpox	1,231	Poliomyelitis	1,857
Coastal fever	2	Puerperal fever	399
Dengue	56	Scarlet fever	5,939
Diphtheria	10,530	Tetanus	13
Dysentery	30	Trachoma	3
Erysipelas	113	Tuberculosis	3,672
Filariasis	1	Typhoid fever	346
Helminthiasis	1	Typhus fever (endemic)	118
Hydatid	6	Undulant fever	4
Influenza	197	Weil's disease	20
Leprosy	12	Whooping cough	826

Poliomyelitis.—An epidemic of poliomyelitis occurred in Australia during the latter part of 1937 and extended into 1938. Provisional quarterly figures for 1937 and the first quarter of 1938, exclusive of Western Australia, which did not share in the outbreak, are shown in the following table. The largest numbers of cases were reported in the State of Victoria. As these figures are provisional, they do not agree with the total in the preceding table, but they show the seasonal incidence of the epidemic.

Year and quarter	Cases	Year and quarter	Cases
1937		*1938*	
1st quarter	8	1st quarter	1,974
2d quarter	17		
3d quarter	463		
4th quarter	1,487		

CANADA

Provinces—Communicable diseases—2 weeks ended June 18, 1938.—During the 2 weeks ended June 18, 1938, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada, as follows:

Disease	Prince Edward Island	Nova Scotia [1]	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				6	4				2	12
Chickenpox		24	16	162	633	255	116	11	185	1,402
Diphtheria		3		72	4	2		2		83
Dysentery				3	1				8	12
Erysipelas		1		6	4	2		1	1	15
Influenza		2		2	1	11			1	17
Lethargic encephalitis					1	1			2	4
Measles		82	7	128	1,652	15	16	26	8	1,934
Mumps		40	54		197	93		12	26	422
Paratyphoid fever					6					6
Pneumonia					38				15	53
Poliomyelitis				1	5			1	1	8
Scarlet fever		32	15	147	181	40	49	38	58	560
Smallpox						8		12		20
Trachoma							1		7	8
Tuberculosis		56	46	128	130	39	13	1	35	448
Typhoid fever			1	18	6	2		2	3	32
Undulant fever				3	5				1	9
Whooping cough		24	8	[2] 115	387	39	11	61	102	747

[1] For 2 weeks ended June 22, 1938.
[2] For the 2 weeks ended June 4, 1938, the cases of whooping cough for Quebec should read 144 instead of 114.

ITALY

Communicable diseases—4 weeks ended April 24, 1938.—During the 4 weeks ended April 24, 1938, cases of certain communicable diseases were reported in Italy as follows:

Disease	Mar. 28–Apr. 3	Apr. 4–10	Apr. 11–17	Apr. 18–24
Anthrax	6	16	15	10
Cerebrospinal meningitis	51	56	31	34
Chickenpox	621	567	414	411
Diphtheria	599	578	479	479
Dysentery	35	32	35	19
Hookworm disease	9	17	13	12
Lethargic encephalitis	1	4		
Measles	4,554	4,343	3,312	3,331
Mumps	438	332	258	226
Paratyphoid fever	29	35	36	33
Pellagra	17	28	16	34
Poliomyelitis	20	18	18	20
Puerperal fever	46	33	22	25
Scarlet fever	327	359	303	278
Typhoid fever	211	243	229	194
Undulant fever	126	168	125	114
Whooping cough	512	532	363	344

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

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for June 24, 1938, pages 1049–1064. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

China.—During the week ended July 2, 1938, cholera was reported in China as follows: Canton, 2 cases; Hong Kong, 25 cases; Macao, 76 cases, Shanghai, 301 cases.

French Indochina.—During the week ended July 2, 1938, cholera was reported in French Indochina as follows: Annam Province, 117 cases; Tonkin Province, 177 cases; Hanoi, 3 cases.

Plague

Argentina—Salta Province—Vespucio.—For the period June 15–30, 1938, 4 cases of plague with 1 death were reported in Vespucio, Salta Province, Argentina.

Tunisia—Tunis.—During the week ended July 2, 1938, 2 plague-infected rodents were reported in Tunis, Tunisia.

United States.—A report of plague-infected fleas and ground squirrels in Beaverhead County, Mont., and plague-infected fleas and a ground squirrel in Uinta County, Wyo., appears on page 1263 of this issue of PUBLIC HEALTH REPORTS.

Smallpox

Brazil—Porto Alegre.—During the week ended May 7, 1938, 1 death from smallpox was reported in Porto Alegre, Brazil.

Japan—Kobe.—During the week ended June 25, 1938, 1 case of smallpox was reported in Kobe, Japan.

Yellow Fever

Gold Coast—Atua Manya.—During the week ended June 25, 1938, 2 suspected cases of yellow fever were reported in Atua Manya, Gold Coast.

Ivory Coast—Bognoa.—On July 4, 1938, 1 suspected case of yellow fever was reported in Bognoa, Ivory Coast.

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