PUBLIC HEALTH REPORTS

VOL. 51

SEPTEMBER 18, 1936

NO. 38

BRAIN REACTION IN GUINEA PIGS INFECTED WITH EN-DEMIC TYPHUS, EPIDEMIC (EUROPEAN) TYPHUS, AND ROCKY MOUNTAIN SPOTTED FEVER, EASTERN AND WESTERN TYPES ¹

By R. D. LILLIE and R. E. DYER, Surgeons, United States Public Health Service

Since 1928 we have been interested in the presence of brain lesions in endemic typhus and the Rocky Mountain spotted fevers in guinea pigs, particularly in view of their reported absence or extreme scarcity in those diseases. About 1930 the problem of a possible differential diagnosis between endemic typhus and spotted fever on the basis of histologic examination of the brains of infected guinea pigs became of interest. It soon became apparent that, in spotted fever, focal lesions tended to occur with relatively greater frequency in the mid and hindbrain, whereas in typhus, cerebral cortical involvement was predominant. However, individual variations prevented accurate differential diagnosis on this relatively crude basis.

In the early stages of this work we adopted a series of five standard transverse sections of the guinea-pig brain for routine examination. The first passed through the frontal cortex and the corpora striata, in the neighborhood of the anterior commissure. The second included parietal and temporal cortex, hippocampus, and thalamus, at about the posterior margin of the internal capsule. The third passed through the oculomotor roots and the anterior colliculi. The fourth included pons, cerebellum, and brachia pontis. The last was made through the enlargement of the medulla.

As the problem of regional distribution became of diagnostic importance it became necessary to record the position as to major divisions of the brain, and the type of each individual focal lesion.

Some 700 such records have now accumulated and an analysis was undertaken in the hope that interesting facts might be revealed by a rough statistical study. This report is the presentation of the results of this analysis.

The brain lesions in typhus and in spotted fever in guinea pigs have been previously described, those of typhus many times. In spotted fever and endemic typhus the lesions were briefly described in our previous reports (1931). They consist of more or less compact nodes or clumps of glia cells, often situated adjacent to small vessels,

¹ From the National Institute of Health.

87898°-36-1

and of various vascular lesions. The glia cell nodes are usually composed of small cells with rounded or elongate (rod) nuclei and little evident cytoplasm, sometimes of large round cells of amoeboid glia or monocyte type compactly grouped. They lie in the brain substance, either gray or white, possibly more often in the gray. Not infrequently they occur in the molecular layer of the cerebral or cerebellar cortex. The vascular lesions consist of cellular infiltration of the vessel sheaths, usually by lymphocytes, of swelling and proliferation of adventitial fibroblasts, and of swelling and concentric proliferation of vascular endothelial cells. Thrombosis and endothelial necrosis are rarely seen. In endemic typhus 10 such lesions were noted in over 400 guinea-pig brains in which over 10,000 focal lesions were counted. In epidemic typhus 5 were found in 72 guinea-pig brains showing over 10,000 focal lesions. In the Bitterroot (western) strain of Rocky Mountain spotted fever 2 thrombi were found in 50 guinea-pig brains with 164 focal lesions, and in 160 guinea pigs infected with eastern strains of spotted fever 8 thrombi were found among 1.680 focal lesions.

The character of the individual lesions does not vary appreciably between the various types of typhus and spotted fever. No such characteristic lesion as the arteriolar thrombonecrosis with microinfarcts seen in human spotted fever (Pinkerton and Maxcy 1931, Lillie 1931, Harris 1933) and probably in Malayan scrub typhus (Lewthwaite 1936) has been encountered in that disease in guinea pigs. However, important differences in topographic distribution of focal lesions in the various parts of the brain are found.

Similar focal nodal and vascular lesions have been repeatedly described in epidemic (European) typhus in guinea pigs (Grzywo-Dabrowski 1918, Pick in Otto and Dietrich's report 1918, Ceelen 1919, Doerr and Kirschner 1919, Wolbach, Todd, and Palfrey 1922, Hach 1925, Barikin, Kompanejez, Zacharoff, and Barikina 1927, and Tichomirov 1931); in Manchurian typhus by Kodama and Takahashi (1930), in tabardillo by Mooser (1928), in Sao Paulo typhus by Meyer in Gomes' report (1932), in Malayan shop typhus and scrub typhus by Lewthwaite and Savoor (1936), by the writers (Lillie 1931) in eastern and western strains of Rocky Mountain spotted fever and in endemic typhus (Dyer, Ceder, Lillie, Rumreich, and Badger 1931).

In epidemic typhus, these lesions have been numerous or variable (Wolbach et al.) in number; in tabardillo, Malayan shop typhus, endemic typhus, and apparently also in Manchurian typhus, they have been few. Focal lesions are also recorded as scarce in eastern and western strains of Rocky Mountain spotted fever, Sao Paulo typhus, and Malayan scrub typhus. The meninges and the chorioid plexus of the several ventricles usually show more or less focal infiltration, usually by lymphocytes. In the pia this infiltration is often perivascular. Vascular endothelial swelling and proliferation occur as in the brain, and occasionally necrosis and thrombosis are seen. In view of the statements of Wolbach, Todd, and Palfrey, and of Hach that, in epidemic typhus in guinea pigs, lesions of the chorioid plexus are never found, the accompanying tabulation of the guinea pig brains in which chorioid plexus was studied is presented.

 TABLE 1.—Lesions of the chorioid plexus in epidemic and endemic typhus and eastern and western strains of Rocky Mountain spotted fever

Disease	No lesions	Very slight infiltra- tion	Slight reaction	Moderate reaction	Total guinea pigs
Epidemic typhus. Endemic typhus, male Endemic typhus, female Eastern spotted fever Western spotted fever	11 41 4 17 4	5 73 2 	7 122 7 25 7	11 86 2 19 9	34 272 15 61 23

This accords with the findings of Ceelen and of Lupu and Petrescu in epidemic typhus in man and with those reported by one of us (RDL) in human cases of the eastern type of Rocky Mountain spotted fever. Wolbach and coworkers failed to find plexal lesions in 37 human typhus cases.

When the general average of all the guinea-pig brains examined in each group is considered without reference to other factors, it is seen that in typhus over half of the focal lesions occur in the cerebral cortex (epidemic about 55 percent, endemic about 60 percent), about 16 to 21 percent (epidemic 21, endemic 16) in the thalamus and basal ganglia, and 25 to 26 percent in the midbrain, pons, medulla, and cerebellum. In the eastern type of Rocky Mountain spotted fever about 54 percent of the focal lesions occur in the midbrain, pons, cerebellum, and medulla, about 15 percent in the basal ganglia and thalamus, and only 32 percent in the cerebral cortex. The percentages for the Bitterroot strain of (western) spotted fever are cerebral cortex 52, basal ganglia and thalamus 11, and midbrain, pons, medulla, and cerebellum 37 percent. Here also a significantly high proportion of the lesions is found in the mid- and hind-brain, though less than with the eastern strains.

It is interesting to note (table 2) the relatively small number of focal lesions in the cerebellum in typhus and the preponderance of nodes over vascular lesions. In the cerebellum the greater part of the nodes occur in the molecular layer of the cortex; the vascular lesions, on the other hand, tend to be more frequent in the central nuclei and white substance. It seems also to be generally true that TABLE 2.—Detailed topographic distribution of various focal brain lesions in typhus and spotted fever in 648 guinea pigs

			¥	Endemic typhus (391 guines pigs)	s typhu	16E) 81	guinea	pigs)					E	pidemi	Epidemic (European) typhus (51 guinea pigs)	pean)	typhu	s (51 gr	uines r	(23)		
	0	Cerebral cortax	cortex		Cerebral nucleí	ei ei	Mid-	Mid- and hindbrain	Indbra			වී	Cerebral cortex	orter		Cerebral nuclei		Mid-	Mid- and hindbrain	odbrat		
	Frontal	Parietal	Temporal	suqmaaqqiH	Corp. stri.	eumsiadT	Midbrain	Pons	slinbəM	Cerebellum	Total	Frontal	Parietal	Temporal	Hippocampus	Corp. strt.	anmaladT	Midbin	Pons	sliubeM	Cerebellum	Total
Perivascular lymphocyte	1, 083 319	734 184	48 223 48	520 106	571 66	509 141	262 115	269 56	215 78	82 86 41	4, 451 1, 179	208 208	88 1880 1980	ន្តឆ	<u></u>	38	128	571	187	813	ន្ទរុឌ	8 855 963
Glin "nodes"	1, 4 02 1, 002	918 864	270 328	626 485	637 171	4 10 4 10	377 378	815 288	888	324 4	5, 630 4, 517 1,	285 100	879 879	208 208	892	12.08	431	370	236	สีส์	3190	4 80 4 80 7
Total focal lasions	10	1, 772	598	1, 111	808	1, 060	755	603	573	88	10,147 1,	1, 939, 1,	1,400	362 1.	1,042	786	1, 036	212	2	43	8	8, 711
			West	Western spotted fever (50 guines pigs)	tted for	rer (50	guinea	pigs)						East	Eastern spotted fover (166 guinea pigs)	tted for	ver (15	6 guine	ea pigs			
Perivascular lymphocyte	81 8	-100	64	99	64	101	24	20	33	60	5 3	41	88	2	58	88	288	នន	88	41	ននី	5 8
Total vasonlar	% 9	15 14	64 CO	41	••	600	12 6	14	91 6	12	117 47	115	28	28	38	64	67	23	201 202	28	8 <u>8</u> 8	58
Total focal lesions	×	8	13	20	0	•	18	16	ส	8	164	240	136	4	132	114	132	202	8	141	874	1, 716
Norm21 European typhus records excluded. 10 because this saries was from a strain never antely identified, the others because of lack of detailed semication of cerebral contributions.	ecords	exclude	d 01 .b	ecense	this ser	AS WAS	from a	strain	DAVAL	maly	identifi		othere	have	lac lac	10 20	- tollat		-			

extr.—T. Intropend to prove accurate accurate or the series was from a strain never smell, identified, the others because of lack of detailed segregation of carebral corrected leaves. The according to the other spectrate accurate in the other spectrate is the other tabulation, as all the animals according to the time of completion of the proper are included from the animals that is used in the other tabulation, as all the animals studied up to the time of completion of the paper are included to the time of animals that is used in the other tabulation, as all the animals studied up to the time of completion of the paper are included here.)

the gray substance of the caudate nuclei contains relatively few glia "nodes" as compared with the number of vascular lesions, and that the frontal area of the cerebral cortex contains larger numbers of focal lesions, both nodal and vascular, than do the other portions of the cerebral cortex.

In epidemic typhus in guinea pigs focal lesions were recorded as most numerous in the floor of the fourth ventricle, hippocampus, and cerebral cortex by Pick (Otto and Dietrich, 1918), in the cerebral cortex by Wolbach, Todd, and Palfrey, in the brain stem and then cerebral cortex, hippocampus, medulla and cord by Hach (1925). Doerr and Kirschner found them throughout the brain, most numerous in the medulla. Ceelen (1919) noted a predilection of lesions for the cerebral cortex and medulla, and found rather numerous cerebellar foci in one guinea pig. Pick found no cerebellar lesions, Wolbach and coworkers found few, and Hach noted the cerebellum as containing the least lesions. In Manchurian typhus, Kodama and Takahashi (1930) found lesions most numerous in the posterior part of the cerebral cortex.

The proportion of the various types of focal lesions varies also. The nodal, focal glioses make up over half of the focal lesions in the endemic and epidemic typhus and in the eastern strains of spotted fever (59, 51, and 53 percent, respectively), whereas with the Bitterroot (western) strain only 29 percent of the lesions were classed as nodes. In both epidemic and endemic typhus over three-fourths of the vascular lesions are perivascular lymphocyte infiltrations of the vessel sheaths and the balance include endothelial and adventitial swelling, proliferation, and thrombosis. For endemic typhus the proportions are 32 percent perivascular lymphocyte infiltration to 9 percent proliferative lesions, and for epidemic typhus 39 percent perivascular lymphocyte infiltration to 10 percent proliferative lesions. In spotted fever, proliferative lesions make up one-third to one-half of the vascular lesions. For the eastern strains the proportions are 26 percent perivascular lymphocyte infiltration to 21 percent proliferative lesions, and for western strains 44 to 27 percent.

Probably the most important modifying factor in the pictures just described is the lapse of time. Time may be counted either from the date of inoculation or from the onset of fever to the date of death. The relative significance of these two modes of estimating the time factor depends obviously on whether or not the evolution of lesions proceeds at a relatively uniform rate during the incubation period.

Accordingly, the records of 280 guinea pigs infected with endemic typhus were distributed according to the length of the incubation period. This period varied from 1 to 18 days, according to the inoculum used. As may be seen from table 3, there is no significant influence of the length of the incubation period on the number of brain lesions to be found in the standard series of sections. Consequently the logical mode of grouping appeared to be according to the time interval between onset of fever and death of the animal. When so grouped, these 280 guinea pigs showed low average numbers of lesions during the first 7 days of fever, a gradual rise on the eighth and ninth days, a peak period from the tenth to the thirteenth day, and a decrease thereafter (table 4).

 TABLE 3.—Influence of duration of incubation period on intensity of brain reaction

 in endemic typhus in 280 guinea pigs

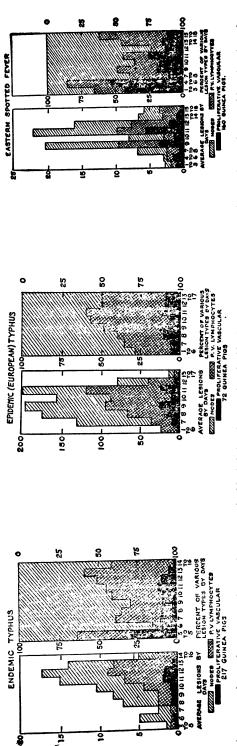
Days duration of incubation period	1	2	3	4	5	6	7	8	9	10	11	12	13–18	Mean, 5
Number of guines pigs Average number of focal lesions	8 4. 0		66 15. 0		45 24. 0	45 14. 5	15 6. 8			13 10. 6	7 9. 1	4 57. 7	3 4. 3	Total, 280. Average, 15.0.

 TABLE 4.—Influence of duration from onset of fever on intensity of brain reaction in endemic typhus in \$20 guinea pigs

Day from onset of fever	8	4	5	6	7	8	9	10	11	12	13	14-17	
Number of gninea pigs	8	1	6	9	27	43	33	60	49	18	17	14	Total, 280.
Average number of focal lesions	0. 0	0.0	2.3	6.4	2. 5	7. 0	8. 5	19. 3	18. 9	21. 1	25. 9	5. 5	Average, 15.0.

As might be expected from the lack of significance of the length of incubation periods in regard to intensity of brain reaction, grouping of the same series of 280 guinea pigs by duration from date of inoculation to date of death shows a much prolonged period of relatively marked reactions, with a much lower average number of focal brain lesions during the peak period. As this mode of grouping appeared to be less significant than that according to duration from onset of fever, it was not used in the study of the other strains of typhus and spotted fever.

In epidemic typhus (Breinl strain) the number of lesions seen in the standard series of sections remained low during the first 6 days, rose sharply on the seventh and eighth days, remained high on the ninth to eleventh days, and fell sharply thereafter. A group of 72 guinea pigs was used for this classification. In the remaining animals studied (28) either the data or the sections were too incomplete for use. The average number of lesions was far higher than in endemic typhus strains, though some animals with endemic typhus showed more lesions than some with epidemic typhus.





In eastern strains of Rocky Mountain spotted fever the first low period lasted to the sixth day, the rise occurred on the seventh and eighth days, the high or plateau period lasted from the ninth to the twelfth days and the number of lesions decreased from the thirteenth day on. The series charted for this study included 160 guinea pigs.

The small series of 50 guinea pigs infected with the western bitterroot strain of spotted fever showed a similar low number of lesions during the first week, a higher, though still small, number during the eighth to tenth days, and a fall in number of lesions in the few animals surviving the tenth day.

The relative paucity of focal brain lesions during the early stages of epidemic typhus in guinea pigs has been commented upon by Pick (Otto and Dietrich), who stated that the examination of several sections might be necessary to demonstrate them in the febrile period. and that later they became more numerous. Doerr and Kirschner found that focal brain lesions appeared in guinea pigs at about the time of onset of fever and persisted through the febrile period to as late as 11 days after defervescence. Hach also found focal brain lesions as early as the onset of fever and noted their increase in number up to the seventh day, a plateau period lasting to the third or fourth postfebrile day and a subsequent recession. Tichomirov's tabulation apparently shows a tendency to more marked reactions from the sixth to tenth day of fever, with fewer marked reactions before and after that period. Positive reactions were found as late as the fifth week. Wolbach, Todd, and Palfrey commented particularly on the presence of numerous brain lesions in a guinea pig killed on the eleventh or twelfth day from onset of fever. Barikin and coworkers also noted a greater frequency of nodes on the sixth to ninth days of fever, and appreciable numbers of "negative" brains before the sixth day and after the second postfebrile day.

In Manchurian typhus, Kodama and Takahashi found nodes most numerous on the third to seventh day of fever, and noted them as constantly present during the first 2 weeks if sufficiently exhaustive search was made.

In tabardillo, Mooser (1928) stated:

In animals killed before the fourth day of fever only the early vascular lesions accompanied by meningeal and perivascular infiltration were found. After the fourth day the typical nodular lesion was present in all animals, but as a rule their number was so small that a whole brain had to be cut in serial sections in order to find them.

The time factor shows a similar influence on the proportion of guinea pigs in which a definite diagnosis of typhus or spotted fever on histologic grounds is possible. Animals showing no focal lesions in the standard series of sections were considered as negative, those with 1 to 2 lesions as inconclusive, those with 3 to 4 as probable, those with 5 to 25 as positive (+), 26 to 50 (++), 51 to 100 (+++), and 101 or over (++++).

Among 10 guinea pigs killed on the third to fifth day of fever in endemic typhus, 1 showed a positive (+) reaction, and 9 inconclusive and negative reactions. On the sixth day there were 2 positive (+ and + +) and 7 negative or inconclusive reactions, on the seventh day 9 probable or positive and 19 negative or inconclusive; and the proportion thereafter rose during the peak period and fell late in the disease, as shown in table 5.

Reaction					Da	y of di	sease				Total
	3-5	6	7	8	9	10	11	12	13	14-17	
- and Ŧ ± and + ++ to ++++.	9 1	7 1 1	19 9	28 11 4	11 19 3	22 25 13	11 22 16	8 5 5	5 7 5	8 6	128 106 47
Total	10	9	28	43	33	60	49	18	17	14	281

 TABLE 5.—Number of negative and inconclusive brain reactions and of positive reactions of varying grades in endemic typhus by day of disease

The period in which approximately two-thirds of the animals show recognizable positive reactions (ninth to thirteenth day) corresponds fairly well with the period in which the average number of brain lesions is highest (tenth to thirteenth day).

In the eastern strains of spotted fever a similar influence was evident. Of 29 guinea pigs taken on the first to fourth days of fever, 21 showed negative or inconclusive reactions and 8 were positive. Of 28 taken on the fifth to eighth days, 14 were positive and 14 were negative or inconclusive. During the period ninth to twelfth day, in which the average number of brain lesions was highest, 65 of 82 guinea pigs showed definite brain reactions and only 17 were negative or inconclusive. Among 21 guinea pigs taken on the thirteenth to eighteenth days, 8 were negative and 13 positive.

In the western (Bitterroot) strain of spotted fever, 2 of 25 animals taken on the second to seventh days showed a positive reaction, 14 of 18 taken on the eighth to tenth days were positive, and 2 of 5 taken on the fourteenth to eighteenth days showed definite brain reactions. No animals were taken from the eleventh to thirteenth day.

Among the 72 guinea pigs infected with the Breinl strain of epidemic typhus only 7 showed negative or inconclusive brain reactions. These occurred in animals taken respectively on the second, eighth, eighth, tenth, tenth, thirteenth, and fourteenth days of fever, and are probably largely assignable to complications.

It has been stated that the reason brain reactions were slight or lacking in endemic typhus strains was that the testicular reaction localized the virus and protected the brain (Mooser, Pinkerton). If this were true, male animals showing no testicular reaction after intraperitoneal inoculation, male animals inoculated by the subcutaneous route, and female guinea pigs should show more marked brain reactions than male guinea pigs showing scrotal redness and swelling after intraperitoneal inoculation.

In table 6, the brain reactions in 21 male guinea pigs not showing a scrotal reaction after intraperitoneal inoculation are compared individually with the averages of guinea pigs with scrotal reaction and killed on the same day of fever, with similar duration of fever. It is seen that there are more brain lesions in the group with the scrotal reactions.

	Scro	tal redn	ess and	swelling	absent	B	crotal r	edness ar	ıd swelli	ng prese	nt
Pathology no.	Day	Dura-	Br	ain react	ions	Num- ber of	Day	Dura-	Brain	erages	15, 87-
	of fever	tion of fever	Nodes	Vascu- lar le- sions	Total focal lesions	guinea pigs	of fever	tion of fever	Nodes	Vascu- lar le- sions	Total focal lesions
1267 8762 6188 8343 5127 5581 7836 8900 8688 9023 9258 1470 4018 9026 9309 8469 9029 8466 5340 9304	5 6 7 7 7 7 8 9 9 10 10 10 11 11 11 11 11 11 13 14 15 17	Days 1 2 6 8 9 2 0 10 1 6 8 9 2 0 10 1 1 2 9 13 12 5 1 1 6 8 9 2 0 1 1 6 8 9 2 0 1 1 1 6 8 9 2 0 1 1 1 1 1 1 1 1 1 1 1 1 1	0 8 3 8 0 5 0 2 4 0 0 0 1 0 5 3 8 6 9 0	0 13 8 2 4 1 0 0 0 0 0 0 0 0 0 0 23 6 0 6 27 0	0 21 11 4 6 0 2 4 4 0 0 0 1 0 0 28 9 9 0 9 8 8 16 0	2 3 19 7 3 19 9 8 6 0 10 10 12 14 6 6 3 2 8 4 8 5	5 6 7 7 7 7 7 8 9 9 9 9 10 10 10 10 10 10 11 11 11 11 11 11 11	Days 1-8 5-7 0-3 5-7 8 0-4 0-3 5-7 1-4 8-10 12-13 1-4 1-4 3-7 14-16	$\begin{array}{c} 1.0\\ 10.3\\ 1.4\\ 8.1\\ 1.0\\ 1.4\\ .9\\ 1.2\\ 6.7\\ 14.2\\ 5.5\\ 14.2\\ 12.8\\ 12.8\\ 12.8\\ 12.8\\ 12.8\\ 12.8\\ 0\\ 5.0\\ 4.0\\ .8\\ 2.6\\ \end{array}$	0.0 1.3 .6 1.6 0.6 1.1 24.1 24.1 24.9 84.0 18.7 14.0 0 2.8	1.0 11.7 2.0 1.0 2.0 1.0 2.0 1.0 2.0 1.0 2.0 1.0 2.0 1.0 2.0 1.0 2.0 1.0 2.0 2.0 1.0 2.0 2.0 1.0 2.0 2.0 1.0 2.0 2.0 2.0 2.0 2.0 2.0 2.0 2.0 2.0 2
Total, 21 guinea pigs Average			52 2. 5	72 3. 4	124 5. 9				135. 0 6. 4	210. 4 10. 0	845. 7 16. 5

 TABLE 6.—Endemic typhus: Brain reactions in intraperitoneally inoculated male guinea pigs in regard to presence or absence of scrotal redness and swelling

Seventeen female guinea pigs were killed 14 days after inoculation in December 1935. These showed an average of 23 focal brain lesions. Eleven male guinea pigs with scrotal reactions, also killed 14 days after inoculation, and in December, to eliminate any seasonal influence, showed an average of 32.1 focal brain lesions. No "protection of the brain" by the testicular reaction was evident in this series.

Eleven male guinea pigs were killed 19 days after subcutaneous inoculation on the sixth to seventeenth days of fever. They showed an average of 2.8 focal brain lesions in the standard series of sections. When these animals are compared individually with the averages of

September 18, 1936

guinea pigs with scrotal reactions, killed on the corresponding days and showing a similar duration of fever (table 7), it is seen that animals inoculated subcutaneously also show less marked brain reactions than male guinea pigs inoculated intraperitoneally and showing scrotal reactions.

<u> </u>	Sut	cutaneo rea	ous inoci ctions a	ulation (bsent)	scrotal	Intraj	peritone	al inocul pres	ation (s ent)	crotal rea	ctions
Pathology no.		Dura-	в	rain lesie	ons	Num-			Brain	lesions, a	verages
	Day of fever	tion of fever	Nodes	Vascu- lar lesions	Total focal lesions	ber of guinea pigs	Day	Dura- tion of fever	Nodes	Vascu- lar lesions	Total focal lesions
9107	6 9 9 9 10 10 10 10 13 17 17	Days 6 5 2 1 1 2 8 9 9 9 9 9 9 9 9 9 10 13	0 5 0 0 8 0 0 1 0 0 1	2527800200	2 10 2 7 6 0 0 8 0 0 1	4 7 28 26 19 12 5 4 7	6 9 9 9 9 9 9 9 10 10 10 13 14-17 14-17	Days 6 5 2 0-2 2 3 5-10 1-3 5-7 7-11 11-15	0.0 4.7 20 3.7 20 15.7 3.9 14.1 21.6 4.7 4.6	0.5 8.6 .5 23.1 3.6 23.9 26.8 4.0 4.3	0.5 8.3 2.5 4.0 2.5 38.8 7.6 38.1 48.4 8.7 8.9
Total, 11 guines pigs Average			10 0.9	21 1.9	31 2.8				77.0 7.0	91. 1 8. 3	168. 3 15. 3

TABLE 7.—Endemic typhus:			and intraperito-
neally	inoculated male	e guinea pigs	-

It would appear from the foregoing data that the presence of a testicular and scrotal reaction is not the factor which determines the relative scarcity of brain lesions in endemic typhus as compared with epidemic typhus.

However, the small series of guinea pigs infected with epidemio typhus which developed a testicular reaction did show brain reactions comparable with those of endemic typhus in number of lesions, and much reduced in comparison with other animals not showing scrotal involvement. Ten guinea pigs with scrotal reactions taken on the ninth to thirteenth days of the disease showed an average of 33.6 lesions, while 11 guinea pigs infected with epidemic typhus and not showing scrotal involvement, taken on the eighth to twelfth days, in the same year, 1930, showed an average of 279 lesions.

This series, occurring in the course of routine transfers of the Breinl strain of epidemic typhus in February and March 1930, gave scrotal reactions typical of endemic typhus, which was transferrable by testicular washings and was carried through several passages and then lost, never reverting to typical epidemic typhus. It appears probable that this group of guinea pigs may have been infected with an endemic strain through some laboratory accident.

1303

The concept that the presence of a testicular reaction prevented the appearance of focal brain lesions originated with Mooser, who (1928) noted that the one female guinea pig inoculated with tunica emulsion showed the most numerous nodules in the brain of his series of animals, and in the discussion of his paper on rickettsiae in Mexican typhus presented before the American Association of Pathologists and Bacteriologists in 1928 he stated that the scrotal swelling was probably responsible for the rarity of brain lesions—"The testicle protects the brain from being involved." Mooser's series was apparently too small to give him a true concept of the great variation in number of brain lesions possible in male guinea pigs inoculated intraperitoneally with the same material. Zinsser and Castañeda make the bare statement that the characteristic brain lesions are present in subcutaneously inoculated guinea pigs without scrotal reaction.

Pinkerton (1931) noted that brain lesions were practically absent in intraperitoneally inoculated male guinea pigs infected with the Wilmington strain of endemic typhus or with Mooser's strain of tabardillo, when the characteristic scrotal sac reaction had occurred. In female guinea pigs and in subcutaneously inoculated males, brain lesions were usually fairly easily found, though not as numerous as in the Wolbach and Breinl strains of epidemic typhus. In one series of six males inoculated subcutaneously with a small amount of scrotal sac exudate from the Wilmington strain, brain lesions were fully as numerous as in the epidemic strains.

Pinkerton further states: "In order to obtain numerous brain lesions in the American strains it would seem that we must prevent the occurrence of a severe local reaction."

It was found that in endemic typhus in guinea pigs the brain reaction was influenced by the clinical course of the disease. On the average, greater numbers of lesions were found in animals showing the typical picture of uncomplicated clinical typhus than in those showing complications or mild clinical reactions.

Of 5 guinea pigs showing febrile complication without clinically diagnosable typhus, 4 showed no brain lesions and 1 showed 11—an average of 2.2. Typical typhus followed by late complications gave an average of 3.5 lesions in 59 guinea pigs. Early febrile complications followed by typical typhus reactions gave an average of 4.6 lesions in 14 animals. Uncomplicated "mild" typhus reactions gave an average of 11.4 lesions in 22 guinea pigs. Typical uncomplicated clinical typhus with the usual scrotal reaction gave an average of 19.3 lesions in 180 guinea pigs. The maximum number of lesions for an individual animal was for typical typhus 220* for "mild" typhus 79, for early complications 39, for late complications 63, and

^{*} We have since seen 3 brains with counts of 229, 275, and 238.-Authors.

for masked complicated typhus 11. The influence of the lack of scrotal reaction in intraperitoneally inoculated males discussed above is probably of the same nature as that involved in the association of "mild" or atypical clinical reaction with a less marked brain reaction. It should be noted, however, that brain reaction may occur in the absence of a febrile reaction. Among 5 such guinea pigs, 3 showed no focal lesions, 1 showed 3 focal lesions (1 node and 2 vascular), and 1 showed a total of 7 nodes, giving an average for the group of 2.0 lesions.

Both Hach and Tichomirov have noted in epidemic typhus in guinea pigs that severe clinical reactions were associated with the most marked brain lesions and mild clinical reactions with slight histologic changes in the brain. Hach extended this correlation to strains of typhus virus, some showing severe clinical reactions and extensive brain lesions, with others showing mild clinical reactions and less extensive brain lesions. However, Hach has noted the presence of brain lesions in animals with slight or no febrile reactions.

In man, Grzywo-Dabrowski was unable to determine any relationship between the duration of epidemic typhus and the number and character of brain lesions.

Cultures of the heart blood were made at autopsy in 219 guinea pigs infected with endemic typhus in which histologic examination of the brain was done. Positive cultures were obtained in 30, killed on the sixth to the sixteenth days. These showed an average of 7.1 brain lesions in the standard series, while in 189 guinea pigs in which the heart blood culture was sterile the average number of brain lesions was 11.2.

The type of inoculum used was also found to influence the intensity of the brain reaction in endemic typhus. This was manifested both in a higher proportion of brains showing diagnostic lesions and in a higher average number of focal lesions with the one inoculum, testicular washings, than with the other, blood.

In table 8 the total percentages of negative, positive, and marked reactions for all time periods are misleading, because of the higher proportion of the testicular washing group killed in the 3- to 8-day period. The really significant figures are found in the 9- to 13-day period, both in percentages of positive reactions and in average number of lesions.

In tabardillo, Mooser (1928) found very few "nodes" in the brains of guinea pigs infected with blood, but found them fairly numerous in those inoculated with an emulsion of tunica vaginalis. Later (1929) he recorded the presence of focal brain lesions in 5 of 10 guinea pigs inoculated with blood, 7 of 10 inoculated with brain showed very few lesions, and all of 10 inoculated with tunica emulsion showed lesions, which were also more numerous.

September 18, 1936

1306

TABLE 8.—Comparison of average intensity and proportion of diagnosable positive brain reactions in endemic typhus according to inoculum used

	Neg	ative	Pos	itive	Ma	rked	Total	Average
Day of fever	Num- ber	Percent	Num- ber	Percent	Num- ber		number of animals	number
8 to 8 9 to 13 14 to 17	40 22 7	70 27 64	13 88 4	23 46 36	4 22 0	7 27 0	57 82 11	5. 8 27. 6 5. 6
Total	69	46	55	37	26	17	150	17.5

REACTIONS FROM TESTICULAR WASHINGS INOCULUM

REACTIONS FROM BLOOD INOCULUM

8 to 8	22	69	9	28	1	8	82	5.0
9 to 13	29	35	35	43	18	22	82	21.8
14 to 17	1	50	1	50	0	0	2	5.5
Total	52	45	45	39	19	16	116	15. 4

CONCLUSIONS

1. The character of the individual focal brain lesions in guinea pigs is not notably different in endemic and epidemic typhus and in eastern and western strains of Rocky Mountain spotted fever.

2. A notably higher proportion of the focal lesions is found in the midbrain, pons, medulla, and cerebellum in Rocky Mountain spotted fever, especially in the castern strains, then in either of the typhus fevers.

3. Proliferative types make up a higher proportion of the vascular lesions in spotted fever than in typhus.

4. The length of the incubation period has no discernible influence on the intensity of the brain reaction in endemic typhus.

5. All the typhus and spotted fever strains studied show an early period of relatively low intensity of reaction in the first week, a period of rapid rise in number of lesions, about 2 days, a "plateau" period of maximum brain reaction lasting 3 to 4 days in the second week, and a late subsidence period. In typhus, perivascular lymphocyte infiltration is relatively more frequent in the "plateau" period of maximum reaction, "nodes" and proliferative vascular lesions earlier and later. The frequency of recognizable brain reactions in endemic typhus and the spotted fevers is also greatest in the "plateau" period.

6. The presence of a scrotal and testicular reaction in endemic typhus does not decrease the intensity of the brain reaction, but rather the reverse.

7. Clinically mild or complicated endemic typhus shows a less intense brain reaction than uncomplicated typhus with definite scrotal reaction.

8. The presence of cultivable microorganisms in the heart blood at autopsy is associated with a lowered intensity of the brain reaction.

9. The use of testicular washings as an inoculum in endemic typhus gives a greater frequency and intensity of brain reactions during the 9 to 13-day period than does blood.

REFERENCES

- Barikin, W., Kompanejez, A., Zacharoff, A., and Barikina, O.: Studien über den Flecktyphus. Centralbl. f. Bakt. etc., I Abt. Orig., 102: 329-338 (1927).
 Ceelen, W.: Die pathologische Anatomie des Fleckfiebers. Erg. d. allg. Path. u. path. Anat. d. Mensch. u. d. Tiere. I Abt. 19: 307-350 (1919).
 Doerr, R., and Kirschner, L.: Beitrag zur Diagnose der Fleckfieberinfektion beim Meerschweinchen. Med. Klin. 15: 894-897 (1919).
 Dyer, R. E., Ceder, E. T., Lillie, R. D., Rumreich, A., and Badger, L. F.: Typhus fever. The experimental transmission of endemic typhus fever of the United States by the rat flea (Xenopsylla cheopis). Pub. Health Rep., 46: 2481-2499 (1931).
 Gomes, L. S.: Estudo experimental do typho exanthematico de S. Paulo in
- Gomes, L. S.: Estudo experimental do typho exanthematico de S. Paulo, in Typho exanthematico de São Paulo. Piza, Meyer, and Gomes, Soc. Impress.
- Paulista, São Paulo, 1932. Grzywo-Dabrowsky, W.: Untersuchungen über die pathologische Anatomie des Fleckfiebers. Virch. Arch., 225: 299-319 (1918).
- Hach, I. W.: Beiträge zur experimentellen Pathologie des Fleckfiebers. II Über pathologisch-histologische Veränderungen in den Organen des Fleckfiebermeerschweinchens. Virch. Arch., 256: 495-517 (1925).
 Harris, P. N.: Histological study of a case of the eastern type of Rocky Mountain and the pathologie des Fleckfieber 1 102 (1920).
- tain spotted fever. Amer. Jour. Path., 9: 91-103 (1933). Kodama, M., and Takahashi, K.: Ueber die Häufigkeit der Knötchenbildung im
- Gehirn von Fleckfiebermeerschweinchen und ihre diagnostische Bedeutung.
- Gehirn von Fleckfiebermeerschweinchen und ihre diagnostische Bedeutung. Jour. Orient. Med., 12:55 (1930). (German abstract.)
 Lewthwaite, R.: The pathology of the tropical typhus (rural type) of the Federated Malay States. Jour. Path. and Bact., 42: 23-30 (1936).
 Lewthwaite, R., and Savoor, R. S.: The typhus group of diseases in Malaya. Part I: The study of the virus of rural typhus in laboratory animals. Part II: The study of the virus of the urban typhus in laboratory animals. Part II: The study of the virus of the urban typhus in laboratory animals. Br. Jour. Exp. Path., 17: 1, 15, 23-34 (1936).
 Lillie, R. D.: Pathology of the eastern type of Rocky Mountain spotted fever. Pub. Health Rep., 46: 2840-2859 (1931).
 Lupu, N. G., and Petrescu, M.: Recherches sur l'histopathologie du système nerveux central dans le typhus exanthématique. Bull. et Mém. Soc. Méd. d. Hôp. d. Bucarest, II: 220-241 (1929).
 Mooser, H.: Experiments relating to the pathology and the etiology of Mexican typhus (tabardillo). I: Clinical course and pathologic anatomy of tabardillo

- Mooser, H.: Experiments relating to the pathology and the ethology of Mexican typhus (tabardillo). I: Clinical course and pathologic anatomy of tabardillo in guinea pigs. Jour. Infect. Dis., 43: 241-260 (1928).
 Mooser, H.: A contribution to the pathology and etiology of Mexican typhus, Sci. Proc. 28 Ann. Meet. Amer. Assoc. Path. and Bact. Amer. Jour. Path., 4: 270.2 (1928).
- 652-3 (1928). Mooser, H.: Tabardillo, an American variety of typhus. Jour. Infect. Dis., 44: 186-193 (1929). Otto, R., and Dietrich: Beiträge zur experimentellen Fleckfieberinfektion des

- Otto, R., and Dietrich: Debrage zur experimentenen Fleckneberinfektion des Meerschweinchens. Centralbl. f. Bakt. etc., I Abt. Orig., 82: 383-400 (1918).
 Pinkerton, H.: Typhus fever. I. A comparative study of European and American typhus in laboratory animals. Jour. Exp. Med., 54: 181-186 (1931).
 Pinkerton, H., and Maxcy, K. F.: Pathological study of a case of endemic typhus in Virginia with demonstration of Rickettsia. Amer. Jour. Path., 7: 07 (102) (102) 95-104 (1931).
- 95-104 (1931).
 Tichomirov, D.: Beitrag zur pathologischen Anatomie des experimentellen Fleckfiebers. Zentralbl. f. Bakt. etc., I Abt. Orig., 122: 261-267 (1931).
 Wolbach, S. B., Todd, J. L., and Palfrey, F. W.: The etiology and pathology of typhus. Harvard Univ. Press, Cambridge, 1922.
 Zinsser, H., and Castañeda.: Studies on typhus fever. II. Studies on the etiology of Mexican typhus fever. Jour. Exp. Med., 52: 649-659 (1930).

TIME CHANGES IN THE RELATIVE MORTALITY FROM ACCIDENTAL BURNS AMONG CHILDREN IN DIFFERENT GEOGRAPHIC REGIONS OF THE UNITED STATES, 1925–32¹

Studies on the Fatal Accidents of Childhood No. 3

By WILLIAM M. GAFAFER, Senior Statistician, United States Public Health Service

Data pertaining to fatalities from different accidents among children under 15 years of age published in a recent paper (1) showed that for the death registration area of 1930 accidental burns (conflagration excepted) was the leading cause of death at ages 1, 2, and 3 years, and for the age group under 5 years, the deaths per 100,000 children being 22, 20, 18, and 17, respectively. The total number of deaths from burns among children under 5 years of age was 1,876, which is approximately one-third more than the number of deaths suffered by children in the same age group from automobile accidents.

Because of the importance of this accidental cause of death among children, and particularly among children of preschool age, it is purposed in this paper to study certain time changes in the mortality from accidental burns among children of different geographic regions of the United States. As in the previous papers (1, 2) the mortality data are specific for the single years of age under 5, and for the age groups 5 to 9 and 10 to 14 years; and, as in the paper immediately preceding, the time period extends from 1925 through 1932. Comparable figures are available in published volumes of the Bureau of the Census; and in the absence of accurate annual population enumerations, the mortality is measured in terms of relative mortality, namely, in terms of the ratio of the number of fatalities from burns to the number of fatalities from all accidents.

The third and fourth revisions of 1920 and 1929 of the "Manual of the International List of Causes of Death" include under the title, "Accidental burns (conflagration excepted)", the following: Burn (conflagration excepted, of any organ or part), by boiling liquid, boiling water, coal oil, corrosive substance, fall with lighted lamp, fire, gasoline, kerosene, molten metal, petroleum, steam, sulphuric acid, and vitriol; Dermatitis actinica and ambustionis; Effects of corrosives, radium, and X-ray; Explosion of gasoline, kerosene, and lamp; Fall into fire; Fire (conflagration excepted); Lamp accident; Playing with fire; Scald of any part of body by steam; and Sunburn.

For the purpose of this inquiry, the death registration States of 1925, consisting of 40 States and the District of Columbia, are divided into 4 broad groups each comprising a geographic region as indicated: A Northeastern (Connecticut, Delaware, Maine, Maryland,

¹ From the Office of Child Hygiene Investigations, U. S. Public Health Service.

Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont, and District of Columbia), a North Central (Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, West Virginia, and Wisconsin), a Southeastern (Alabama, Florida, Kentucky, Louisiana, Mississippi, North Carolina, South Carolina, Tennessee, and Virginia), and a Western (California, Colorado, Idaho, Montana, Oregon, Utah, Washington, and Wyoming).

TABLE 1.—Number of deaths from accidental burns (conflagration excepted) per 100 deaths from all accidents among children under 15 years of age in different geographic regions of the United States, 1925–32, white and colored combined

				A	ge in yea	13			
Year	All ages	Under 1	1	2	8	4	Under 5	5 to 9	10 to 14
1925 1928 1927 1929 1929 1930 1941 1932	20. 0 19. 5 19. 0 17. 5 16. 3 11. 5 10. 6 10. 1	10. 7 11. 5 12. 2 12. 7 13. 5 10. 1 8. 7 10. 4	88. 4 88. 4 89. 9 88. 3 80. 9 32. 9 28. 0 26. 4	42. 6 42. 4 41. 3 41. 1 30. 5 26. 4 26. 1 29. 0	39. 7 36. 8 32. 6 31. 4 32. 0 21. 9 17. 1 16. 6	31. 6 30. 9 31. 5 28. 1 28. 3 18. 1 16. 1 13. 8	31. 8 81. 2 31. 4 29. 9 26. 8 21. 2 19. 0 18. 7	13.6 11.9 11.9 11.6 11.7 7.2 6.4 6.7	8.9 5.5 4.7 8.8 4.1 8.1 8.1 2.2
		NO	RTH CI	ENTRA	L				
1925	16. 0 16. 1 15. 4 15. 1 14. 7 10. 0 9. 8 11. 2	7.8 7.2 6.9 6.3 10.7 5.5 5.8 7.6	27. 8 30. 1 28. 2 26. 3 26. 3 21. 1 21. 4 25. 2	38 . 3 33 . 3 33 . 1 31 . 0 27 . 9 22 . 5 24 . 8 27 . 9	28. 7 34. 2 29. 7 27. 6 30. 0 18. 6 18. 1 19. 4	26. 9 25. 9 28. 5 23. 5 24. 8 17. 8 13. 3 18. 5	24. 6 24. 1 23. 4 21. 8 22. 9 16. 5 16. 4 19. 5	10. 6 11. 2 10. 3 10. 7 10. 0 6. 9 6. 1 8. 6	4.8 5.7 5.0 6.7 4.5 3.1 8.6 3.6
		80	UTHEA	STERN					
1925 1926 1927 1928 1929 1930 1931 1932	27. 9 28. 9 25. 1 24. 3 23. 0 18. 6 17. 5 18. 6	14. 5 14. 5 15. 1 11. 7 11. 5 11. 3 13. 2 12. 6	33. 6 57. 5 32. 1 34. 3 32. 9 28. 9 27. 9 33. 2	47. 5 47. 3 39. 9 47. 3 40. 8 32. 4 35. 7 35. 5	52. 7 56. 6 47. 3 39. 9 39. 9 32. 9 32. 7 36. 6	46. 6 44. 8 42. 6 41. 4 39. 3 32. 2 27. 0 30. 7	35. 0 36. 2 32. 3 31. 0 30. 1 25. 4 25. 8 27. 7	26. 3 27. 7 23. 0 23. 7 21. 2 16. 6 14. 3 15. 7	10. 1 10. 3 9. 3 7. 7 8. 4 6. 8 5. 4 5. 4 5. 3
		W	ESTER	N					
1925 1926 1927 1928 1929 1930 1931 1932	13.8 11.8 12.3 11.2 12.2 9.0 7.9 8.6	7.2 9.0 8.8 6.7 7.0 5.6 7.0 3.5	21.5 14.1 14.6 19.1 17.3 19.1 13.4 16.0	28. 2 33. 3 24. 1 17. 0 26. 0 15. 8 14. 7 19. 6	27.6 20.3 14.6 23.5 23.3 14.7 13.2 14.1	18.5 14.8 21.8 21.1 17.6 11.0 16.1 19.2	19. 8 17. 3 16. 3 17. 0 17. 4 12. 9 12. 5 13. 6	10. 3 8. 0 10. 3 8. 1 9. 9 6. 4 5. 2 5. 5	4.3 6.1 6.3 4.3 4.4 5.1 8.7 8.2

NORTHEASTERN

TABLE 1-A.—Number of deaths from accidental burns (conflagration excepted) among children under 15 years of age in different geographic regions of the United States, by age, 1925-32, white and colored combined

NORTHEASTERN

				A	ge in yea	FB			
Year	All ages	Under 1	1	2	8	4	Under 5	5 to 9	10 to 14
1925	1,200	66	214	204	215	177	876	277	47
1928	1,131	71	204	208	193	161	837	231	63
1927	1.049	59	175	200	173	156	763	233	63 53 49 50
1928	968	63	172	· 181	149	133	698	221	49
1929	850	64	126	122	141	138	591	209	50
1930	772	53	134	130	121	102	540	182	50
1931	715	41	129	131	102	103	506	161	48
1932	598	42	109	109	84	76	420	145	83
	L	NOI	RTU CH	ENTRA	ւ ւ				L
1925	926	55	174	188	143	121	681	181	64
1928	898	56	173	161	153	101	644	182	72
1927	865	48	156	162	140	124	630	171	64
1928	827	42	159	138	131	102	572	169	86
1929	816	72	153	134	135	99	593	165	- 58 54
1939	721	41	132	129	106	94	502	165	54
1931	660	37	131	131	92	72	463	135	62
1932	663	43	144	129	89	78	483	123	57
		SOUT	THEAS	FERN					
1925	1,024	96	132	172	187	137	724	225	75
1928	1, 110	96	163	183	193	137	772	259	. 79
1927	924	90	140	130	156	121	638	215	71
1928	891	78	137	159	125	115	612	220	59
1929	801	62	122	133	121	105	543	194	64
1930	776	69	109	117	120	96	511	200	65
1931	694	69	109	121	100	78	477	164	53
1932	710	67	114	110	117	85	493	166	53 51
		W	ESTER	N	I	(1	
1007		1 1				~	110	40	 1-
1925	215	14	46	44	32	22	158	42	15
1926 1927	184 204	17	28 27	41 42	26 20	12 26	124 132	37 48	23 24
1841	184	ii l	33	25	32	20	132	42	17
1000 1			35	40	32 24	21	133	42	17
1928	101				22	21	100 1		17
1929	191	13			02	18		56 1	~
1929 1930	176	12	33	29	23	15	112	39	25
1929		13 12 15 7	33 27 34		23 23 24	15 22 20		39 32 29	25 20 15

Table 1, which presents the essential data of the study, gives for 1925-32 the geographic distribution of the number of deaths from accidental burns per 100 deaths from all accidents, together with the number of deaths from accidental burns, for children under 15 years of age, white and colored combined. The table immediately discloses that the number of deaths from accidental burns has been decreasing in each region during the 8 years under observation. In general, however, the percentage age distribution varies but little from year to year as among the different regions. For example, a calculation shows that for each region during 1925-32 approximately 70 percent of all deaths from accidental burns occurring annually among children under 15 years of age were suffered by children under 5 years of age. Similar percentages for ages 1, 2, 3, and 4 are 18, 18, 15, and 13, respectively.

RELATIVE MORTALITY BY AGE, SPECIFIC FOR REGION

Figure 1 shows the time changes by age in the relative mortality from accidental burns for the different geographic regions from 1925 through 1932. It will be observed that the magnitudes of the lower and upper limits of the distribution of the relative mortalities regardless of age vary from region to region. In each region the lower limit is given by the age group 10 to 14 and the upper limit by the children of age 2, save the Southeastern region, where this limit is given by the 3-year-olds. When the ranges of relative mortality are placed in order of decreasing magnitude, the regions with their ranges read as follows: Southeastern (51.3), Northeastern (40.4), North Central (35.2), and Western (30.1). The lower limits of the ranges are of a similar order of magnitude, being in the neighborhood of 3 percent. The upper limits, on the other hand, read 56.6, 42.6, 38.3, and 33.3, with the regions in the same order as given immediately above. These upper limits indicate that, in the Southeastern region, accidental burns accounted for more than one-half of the deaths from fatal accidents in 1926 at 3 years of age; in the Northeastern region the proportion in 1925 was nearly one-half, but at 2 years of age: in the North Central region the proportion in 1925 was more than one-third and at 2 years of age, and in the Western region the proportion in 1926 was one-third at 2 years of age.

The curves (fig. 1) for the four regions are similar in three respects. First, the trends of relative mortality, while with different rates of change, are decreasing at each age and for each age group. Second, the curve of relative mortality for all ages definitely separates all of the age curves in each region into two similar sets: The first set, which lies below the curve for all ages, consists of the curves for ages under 1 and the age groups 5 to 9 and 10 to 14; the second set, which lies above the curve for all ages, consists of the curves for ages 1, 2, 3, and 4. Thus, in each region during the 8 years 1925-32, the trend of the relative mortality from burns decreased at each age and for each age group. And during the same period, furthermore, in each region the relative mortality of ages 1, 2, 3, and 4 was consistently greater than the relative mortality for all ages; at under 1 year of age and for the age groups 5 to 9 and 10 to 14, on the other hand, the relative mortality was consistently less than that for all ages. The third point of similarity deals with the absence of order of the curves within the sets referred to above: The relative mortality at ages 1, 2, 3, and 4 in each region is such that it does not permit a definite ordering of the mortality with respect to these ages; with respect to the set comprising ages under 1 year and the age groups 5 to 9 and 10 to 14,

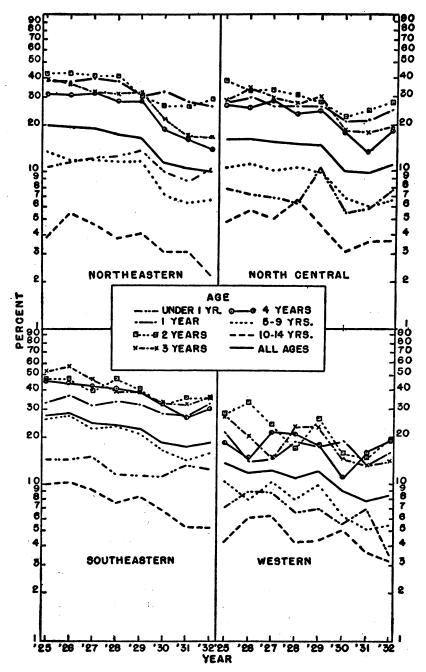


FIGURE 1.-Number of deaths from accidental burns (conflagration excepted) per 100 deaths from all accidents, by age, in different geographic regions, 1925-32, white and colored combined. (Logarithmic scale.)

the situation is only slightly different in that the mortality for the latter age group is generally the lowest in each of the regions.

With regard to the order of the ages in each region, for the period 1925-32, with respect to the magnitude of the rate of decline of relative mortality, measured by the slopes of the straight lines fitted to the appropriate curves of figure 1, the following facts emerge:

In all regions ages 2, 3, and 4 show the most rapid decline. As previously indicated, it was the children of these particular ages who suffered almost one-half of the burden of the mortality from burns among children under 15 years of age. When the rates of decrease for these ages are ordered by region according to decreasing magnitude, the Northeastern and the Southeastern regions show the same order. The first, second, and third places are occupied by the 3-, 4-, and 2-year-olds, respectively. For the Northeastern region the rates of decrease in percent are 28, 21, and 19; for the Southeastern, 25, 23, and 17. The corresponding percents for the North Central and Western regions are, respectively, 11, 15, and 17, and 8, 7, and 15. The remaining ages of the corresponding regions show lower percents, with no striking similarity of order as among the regions.

RELATIVE MORTALITY BY REGION, SPECIFIC FOR AGE

In figure 2 the curves of relative mortality for the period 1925-32 have been rearranged to show how the four regions compare when age is held constant. While the regions are not similarly ordered at each age, attention must be directed to certain other observable facts relating to order.

For all ages, ages 5 to 9 and 10 to 14, and at ages 3 and 4, the Southeastern region consistently shows the highest relative mortality. This is directly opposed to an earlier finding which disclosed that this region had the lowest relative mortality from automobile accidents at each age (2). For all ages and the age group 5 to 9, and at ages 2, 3, and 4 the regions tend to order themselves with respect to decreasing order of relative mortality thus: Southeastern, Northeastern, North Central, and Western. At ages under 1, both the Northeastern and Southeastern regions show a higher relative mortality than either the North Central or Western. At age 1 both the Northeastern and Southeastern are followed by the North Central and Western, respectively. For the age group 10 to 14 the Southeastern is high, with the remaining regions all lower and in no definite order.

With regard to the order of the regions at each age with respect to the magnitude of the rate of decline of relative mortality, measured as indicated in the previous section, figure 2 shows the following:

The largest rate of decrease among the various curves appears to be at age 3 for the Northeastern (28 percent) and Southeastern (25 percent) regions; at the same age the rates for the North Central

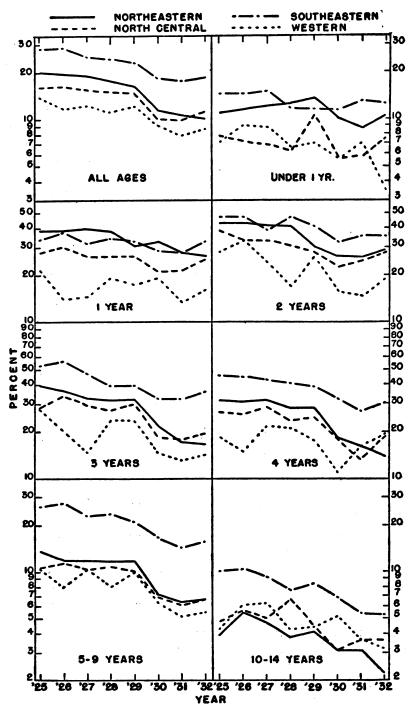


FIGURE 2.—Number of deaths from accidental burns (configuration excepted) per 100 deaths from all accidents, by geographic region, at different ages, 1925-32, white and colored combined. (Logarithmic scale.)

and Western regions are 11 and 8, respectively; and for all ages, while the rates of decrease for the corresponding regions are lower, the order of the regions is similar. At ages under 1 the rate of decline for all regions is in the neighborhood of 3 percent, while at age 2 the decline for all regions is approximately 17 percent. At age 1 the Northeastern region is highest, with a rate of 16 percent followed by the North Central (9 percent), Southeastern (8 percent), and the Western (4 percent). At age 4 the Northeastern and Southeastern regions lead with a rate of about 20 percent, followed by the North Central (15 percent) and Western (7 percent). The age groups 5 to 9 and 10 to 14 show similar orders, the Southeastern leading with 16 and 7 percent, respectively, and the other regions approximating 7 and 3 percent.

SUMMARY

This paper deals with time changes in the relative mortality from accidental burns (conflagration excepted) among children under 15 years of age in different geographic regions of the United States from 1925 through 1932. Relative mortality is defined as the ratio of the number of fatalities from accidental burns to the number of fatalities from all accidents.

The death registration States of 1925, consisting of 40 States and the District of Columbia, are divided into 4 broad groups, each constituting a geographic region: A Northeastern, a North Central, a Southeastern, and a Western.

The actual number of deaths from accidental burns decreased in each region during the 8 years observed. The percentage age distribution of the deaths, however, varied from year to year but little as among the different regions.

Relative mortality by age, specific for region.—In each region the lower limit of relative mortality, approximately 3 percent, is given by the age group 10 to 14 years and the upper limit by the children of age 2, save the Southeastern region, where this limit is at 3 years. The upper limits for the Southeastern, Northeastern, North Central, and Western regions are 57, 43, 38, and 33 percent, respectively. The trends of relative mortality for all regions, while with different rates of change, decrease for each age and age group. In all regions ages 2, 3, and 4 show the most rapid decline. The other ages of the corresponding regions show lower rates.

Relative mortality by region, specific for age.—The regions are not similarly ordered at each age. For all ages and the age group 5 to 9 years, and at ages 2, 3, and 4 the regions tend to order themselves with respect to decreasing relative mortality, thus: Southeastern, Northeastern, North Central, and Western. With respect to the rate of decline of relative mortality, the largest rate appears to be

at age 3 for the Northeastern (28 percent) and the Southeastern (25 percent) regions; at the same age the rates for the North Central and Western regions are 11 and 8, respectively.

REFERENCES

- Gafafer, W. M.: (1936) Mortality from automobile accidents among children in different geographic regions of the United States, 1930. Studies on the fatal accidents of childhood no. 1. Pub. Health Rep., 51: 1083-1090 (Aug. 7, 1936).
- (2) Idem.: (1936) Time changes in the relative mortality from automobile accidents among children in different geographic regions of the United States, 1925-1932. Studies on the fatal accidents of childhood no. 2. Ibid., 51: 1186-1194 (Aug. 28, 1936).

DEATHS DURING WEEK ENDED AUG. 29, 1936

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

	Week ended Aug. 29, 1936	Correspond- ing week, 1935
Data from 86 large cities of the United States: Total deaths. Deaths per 1,000 population, annual basis. Deaths under 1 year of age. Deaths under 1 year of age per 1,000 estimated live births. Deaths per 1,000 population, annual basis, first 35 weeks of year Data from industrial insurance companies: Policies in force. Number of death claims. Death claims per 1,000 policies, first 35 weeks of year, annual rate. Death claims per 1,000 policies, first 35 weeks of year, annual rate.	7, 845 10. 3 509 40 12. 5 68, 313, 576 11, 009 8. 4 10. 2	6, 691 9, 8 511 48 11, 6 67, 554, 445 10, 659 8, 2 10, 0

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

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

Reports for Weeks Ended Sept. 5, 1936, and Sept. 7, 1935

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Sept. 5, 1936, and Sept. 7, 1935

	Diph	theria	Influ	lenza	Me	asles	Mening meni	ococcus ngitis
Division and State	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935
New England States: Maine		1 2 1 3 2 4 4 1 11 15 32 32 32 32 7 1 1 2 3 2 3 2 3 2 3 2 1 1 3 2 3 2 3 2 3	3 14 4 2 7 3 2 2 7 3 3 2 2 3 1 4 4 4 4 2 7 3 3 2 2 1 1 1 1 2 1 1 1 5 2	13 4 1399 6 111 113 	3 221 6 60 857 5 100 2 13 8 8 4 3 2 10 1 2 13 13 1 1 8 17	8 4 13 2 1 20 19 3 16 21 36 21 36 4 4 4 4 5 5 1 1 5 1 4 6 6	000100 532 022331 1010000 000300	00001 1001 5223 7226221 202201000 043221
South Carolina 4 Georgia 4 Florida 4	4 11 8	20 26 7	50	94 2	2	1	1 1 1	0 0 0

See footnotes at end of table.

September 18, 1936

1318

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Sept. 5, 1936, and Sept. 7, 1935—Continued

	Diph	theria	Infi	uenza	Me	asles	Menin men	goooccus ingitis
Division and Stata	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. δ, 1936	Weck ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935
East South Central States: Kentucky Tennessee Alabatna 4 Mississippi 4 West South Central States:	12 17 21 19	63 22 81 26	5 6 6	8 29 27	17 5	8 1 7	2700	0
Arkansas. Louisiana Oklahoma ¹ Teras ⁴ Mountain States:	9 10 8 28	35 23 19 76	2 15 8 81	7 17 12 16	 18 8	4 7 1 1	1 1 0 2	2 0 1 0
Montana Idaho	1 1 	1 7 2	4 1 14	6	1 1 3 8 1	8 	0 0 5 2	0001000
Utah ³ Pacific States: Washington Oregon California	1 		2 10 13		1 4 4 18	1 13 82 73	2 1 0 8	
Total	887	679	211	846	839	438	55	62
First 36 weeks of year	16, 189	19, 777	142, 334	105, 025	271, 308	697, 342	6, 123	4, 354
	Polion	yelitis	Scarle	t fever	Sma	llpox	Typho	id fever
Division and State	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935
New England States: Maine New Hampshire Vermont. Massachusetts Rhode Island. Connecticut Middle Atlantic States:	1 0 0 1 0 1	17 3 4 169 81 38	4 3 3 8 8 6 7	11 8 1 32 8 8	000000000000000000000000000000000000000	000000000000000000000000000000000000000	0 0 0 5 6 2	80 8701
New York New Jersey Pennsylvania East North Central States:	20 1 5	414 72 9	88 11 76	108 25 52	0 0 0	1 0 0	3 0 27 38	85 15 16
Date North Central States: Ohio Indiana Michigan Wisconsin West North Central States:	2 1 80 5 1	2 3 22 76 4	52 14 84 40 46	111 43 130 31 59	0 0 8 0 1	0 0 0 0 0	15 8 25 10 2	54 18 47 16 6
Minnesota Iowa Missouri North Dakota South Dakota Nebraska Kansas	0 3 2 0 2 1 0	δ δ 0 0 0 1	12 16 13 5 13 3 17	81 18 54 2 10 9 17	- 0 4 0 2 0 0 0	0 0 1 0 6 1	2 1 47 2 1 0 11	5 7 20 1 1 1 17
Bouth Atlantic States: Delaware ¹	0 1 0 4 3 1 0 5 2	0 11 8 16 3 11 1 0 0	15 6 17 12 27 3 1 4	4 18 10 19 45 36 7 9 4	0 0 0 0 0 0 0 0 0 0 0	0 0 0 0 1 0 0 0	1 11 0 15 16 25 4 24 5	1 16 4 11 16 16 24 16 1

See footnotes at end of table.

	Polion	yelitis	Scarle	t fever	Sma	llpox	Typhoid fever	
Division and State	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Weck ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935	Week ended Sept. 5, 1936	Week ended Sept. 7, 1935
East South Central States:								
Kentucky	4	42	27	57	0	0	52	96
Tennessee	22	8	21	27	0	1	25	50 15
Alabama 4,	5	2	13	4	0	0	12	15
Mississippi ^a	18	0	8	9	0	0	16	13
West South Central States:			1					
Arkansas	1	0	8	83	0	0	20	7
Louisiana	8	2	8	13	ŏ	1	83 26	20 21
Oklahoma		8	8	13 21	Ň	i	20	70
Texas 4		0		4 1	U U		"	
Mountain States: Montana		1	16	9	8	2	7	9
Idaho	ō	ō	10		ŏ	i	8	8
Wyoming \$	ŏ	ŏ	ā	i i	š	ō	ŏ	2
Colorado	ž	ň	Č	21	ŏ	ŏ	Ğ	35350
New Mexico	2	ō	Š	- 6	ŏ	ŏ	20	2
Arizona	ō	i	i	2	Ŏ	Õ	Ō	6
Utah ¹	ŏ	ī	2	14	Ō	Ó	1	Ó
Pacific States:	ė	-	_	_				
Weshington	7	1	9	8	8	18	2	1
Oregon	0	Ō	11	14	2	8	5	5
California	25	24	64	75	0	2	16	15
Total	183	1,007	865	1, 210	28	39	606	753
First 36 weeks of year	1, 847	6, 424	186, 465	183, 421	6, 345	5, 407	8, 687	11, 471

Cases of certain communicable diseases reported by telegraph by State health officers

New York City only.
 Rocky Mountain spotted fever, week ended Sept. 5, 1936, 2 cases, as follows: Delaware, 1; Virginia, 1.
 Week ended earlier than Saturday.
 Typhus fever, week ended Sept. 5, 1936, 44 cases, as follows: South Carolina, 1; Georgia, 18; Florida, 3; Alabama, 16; Teras, 6.
 Exclusive of Oklahoma City and Tulsa.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Menin- gocoo- cus menin- gitis	Diph- theria	Influ- enza	Malaria	Measles	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
May 1938									7	
California	25	126	2, 134	15	8, 031	12	19	1, 264	1	51
July 19 3 6										
Florida	13	13	5	62	19	7	1	11	0	4
August 1956										
Connecticut	- 4	8	2		43 3		8 1	23 8	0	8
Delaware District of Colum- bia	8 7	20			22 14	1	1	9	Q	5
Florida Maine	7	20 11 7	2	98	14 111	4	7 8 17	13 80	0	6 12
Pennsylvania	18	69		4	242	2	17	347	0	109

Summary of Monthly Reports from States-Continued

RODENT PLAGUE IN MODOC COUNTY, CALIF.

The Director of Public Health of California reported under date of June 15, 1936, that plague infection had been proved, by animal inoculation, in four ground squirrels received at the laboratory on June 11 from localities 4 miles east, and 5 miles east and 1 mile north of Hackamore, in Modoc National Forest, Modoc County, Calif.

WEEKLY REPORTS FROM CITIES

City reports for week ended Aug. 29, 1936

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. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

State and shire	Diph-	Inf	uenza	Mea-	Pneu-	Scar- let	Small-		Ty- phoid	Whoop- ing	Deaths, all
State and city	theria cases	Cases	Deaths	sles cases	monia deaths	fever cases	pox cases	culosis deaths	fever cases	cough cases	all causes
Maine:									0	· ·	14
Portland New Hampshire:	0		0	0	0	0	0	0	U	8	19
Concord	Q		ļ	ļ	0	0	0	0	0	0	8
Manchester Nashua	0		G	0	0	0	0	0	0	8	17
Vermont:	, v			v		v	v		v	ľ	
Barre											
Burlington Rutland	0		0	0	0	1	0	0 0	0	20	12
Massachusetts:				-	-				-	i i	
Boston	0		0	0	10	9 1	0	52	0	53	183
Fall River	0		ŏ	0	1	2	ŏ	ő	8	ō	22 23
Worcester	ŏ		ŏ	Ŏ	1 i	2	Ŏ	8	Ŏ	8	40
Rhode Island:	0		0	0	0	0	0	0	0	0	
Pawtucket Providence	ŏ		ŏ	ŏ	ŏ	ÿ	ŏ	4	ŏ	14	84
Connecticut:											
Bridgeport Hartford	0		1	1	1	0	0	1	0	12	26 25
Hartford New Haven	1	1	0	0	0	1	0	6	1	0 73	20
	Ű	-		Ĵ		Ţ	Ŭ	Ť	-		
New York: Buffalo	0		0	1	5	1	0	4	0	0	93
New York	8		8	44	53	24	ŏ	79	16	107	1, 125
Rochester	0		0	0	4	0	0	0	0	- 2	56
Syracuse	0		0	0	0	3	0	1	0	22	46
New Jersey: Camden	2		0	0	2	0	0	1	1	0	35
Newark	0		Ó	Ó	4	2	0	3	0	27	90
Trenton	0		0	0	2	2	0	8	0	4	81
Pennsylvania: Philadelphia	1		2	7	23	14	0	24	11	97 36	384
Pittsburgh	1		1	7	18	9	0	11	1	36	146
Reading	0		0	1	0	0	0	1	0	18 0	43
BCIAHOH	v			Ŭ		Ů	Ů		Ů	Ů	
Ohio:			0		8	3	0	10	8	1	164
Cincinnati Cleveland	4	3	1	8 2 3	ŝ	16	ŏ	15	ő	74	181
Columbus	3		0	3	8	4	0	2	0	74 6	79
Toledo	0		0	Ō	1	4	0	4	0	12	68
Indiana: Anderson	0		0	2	1	1	0	0	0	0	8
Fort Wayne	0		Ó	0 1	1 7	0	0	0	0	2	22
Indianapolis	0		2	1	7	1	0	4	2	1	107 14
South Bend Terre Haute	ŏ		ŏ	ŏ	ô	il	ŏ	ŏ	ŏ	ő	22
Illinois:	-			-							_
Alton	0 12		0	0	0 18	1 26	8	0	1 0 0	0 86	657
Chicago Elgin	1		ŏ	5	ĩ	õ	8	85 0 0	ŏ		
Moline	0		Ó	0	0	002	0	91	0		2
Springfield Michigan:	0		0	Ō	1	2	0	- 1	1	- 1	-
Detroit	2 0		0	3	8	10	0	17	0	88	222 26 18
Flint.			0	0	1	1	0	<u> </u>	. Ő	1	25
Grand Rapids. Wisconsin:	0		0	Ō	0	ī	0	0			
Kenosha	0		0	0	1	1	0	0	0	ŝ	. 9
Madison	0		0	0	0 2	1 2 6	0	1	9	9 51	6 18 76 10 13
Milwaukee Racine	0		0	1	ő	ő	8 I	ő	1	01	ið
Superior	ŏ		ŏ	ô	ŏ	ŏ	ŏ	ŏ	ŏ	ŏ	18
			1						- 1		
Minnesota: Duluth	0		0	1	0	0	000	1	0	41	18 94 47
	ŏ		ŏ	Ö 1	2	8	01	1	0	A 1	04
Minneapolis St. Paul	ŏ		ŏ	¥ 1		š	× 1		ŏΙ		

State and city	Diph- theria	Inf	luenza	Mea-	Pneu- monia	Scar- let	Small- pox	Tuber-	Ty- phoid	Whooping	Deaths,
	CRSES	Cases	Deaths	Cases	deaths	fever cases	cases	deaths	fever cases	cases	CAUSES
Iowa:											
Cedar Rapids Davenport	0						0		0		
Des Moines	ŏ			ŏ		1	ŏ		ŏ	ŏ	26
Sioux City	Ó			i i		1	2		ŏ	ĺ	
Waterloo	1			0		0	0		0	2	
Missouri: Kansas City	6		0	6	0	6	0	4	0	0	122
St. Joseph			ĭ								
St. Louis	.8		0	0	4.	2	0	6	4	13	262
North Dakota: Fargo	0		0	1	0	0	0	0	0	0	11
Grand Forks	ŏ			l õ		ŏ	Ιŏ		ŏ	ŏ	**
Minot	-0		0	Ó	0	Ó	Ó	Ö	Ō	Ŏ	6
South Dakota:	0			0							
Aberdeen Sioux Falls	ŏ		0	l ö	0	0	0	ō	0	O O	
Nebraska:	Ů		Ů	ľ	, v	v	ľ	, v	v	, v	
Omaha	5		0	0	5	0	0	2	0	8	58
Kansas:	0		0	0		•			•		
Lawrence Topeka	0		0	U	0	0	0	0	0	0	2
Wichita	Ō		0	0	2	ō	0	0	0	0	82
								-	-	-	
Delaware: Wilmington	0			0		0					
Maryland:	v		0	v	4	U	0	1	0	1	28
Baltimore	2		0	5	15	4	0	8	0	108	192
Cumberland	0		0	0	Ó	1	Ó	0	Õ,	0	11
Frederick	0		0	0	1	0	0	0	0	0	5
District of Col.: Washington	0		0	4	6	0	0	8	0	18	164
Virginia:	-		l l	-	Ů	v		°	v	10	101
Lynchburg	0		0	0	Q	0	0	0	2	- 4	17
Norfolk	2		0	2	2	4	0	8	<u> </u>	0	80
Richmond Roanoke	1		0	0	8	1	0	2 1	Ó	Ő	49 14
West Virginia:			Ŭ	Ů	Ů	v	v	· • I	Ň	v	11
Charleston	0		0	0	1	0	0	0	2	0	19
Huntington	1 0			0		0	0		1	0	
Wheeling North Carolina:	U		0	v	1	0	0	2	0	8	20
Gastonia	0		0	0	0	0	0	0	0	0	
Raleigh	0		0	Ŏ	1	0	0	0	1	0	18
Wilmington Winston-Salem	12	<u>i</u>	0	0	12		0	1	<u>Š</u>	0	11
South Carolina:	~	- 1	v	, v	-	° I	•	- 1	۳	- 1	17
Charleston	0		0	0	0	0	0	1	2	0	24
Columbia											
Florence Greenville	,0 O		0	0	Ô	0	0	1	8	0	9
Georgia:	v		v I	•	° I	۳		•	0	0	4
Atlanta	0		0	2	5	8	0	5	1	0	70
Brunswick	0		0	0	11	0	0	0	0	0	8
Savannah	8		0	0	0	0	0	8	8	8	83
Miami	0	1	1	0	1	2	0	0	0	0	21
Tampa	i		ō	i	i	ī	ŏ	ŏ	ŏ	ŏ	19
Ventuchan							1			[
Kentucky: Ashland	0			0		0	0		1	0	
Covington	Ó		0	Ó	4	0	0	ŏ	ô	0	19
Lexington	0		0	0	8	0	0	2	0	0	25
Louisville Tennessee:	2		0	0	8	1	0	2	0	13	41
Knozville	8		0	1	01	2	0	2	8	o	82
Memphis	i		ŏ	ō	2	ī	ŏ	ē	ŏ	2	95
Nashville	1		0	0	2	Ō	0	8	8	ō	67
Alabama: Birmingham	1		0	0	8	1	0	4	4	0	KO
Mobile	2		ŏ	ŏl	8	ôl	ŏ	2	5	ŏ	58 80
Montgomery	2			ŏ		Õ	ŏ.		ĭ	ŏ].	
Ankemaa			I	1						1	
Arkansas: Fort Smith			I		I						
Little Rock	0		0	ŏ	6	i	0	8	0	0	13
Louisiana:						1					
Lake Charles.	2		9	0	2	9	8	9	01	2 1 1	120
New Orleans Shreveport	1	•	į	8	8	i	ŏ	7	- 1	- 11	139 80
PUTO LODOT Prosent		{					• 1		• '	11	a u

City reports for week ended Aug. 29, 1936-Continued

	•	-	•								
State and city	Diph	-	luenza	Mea-	Pneu- monia	Scar-	Small-	Tuber	Ty-	Whoop	Deaths,
State and city	cases		Deaths		deaths	fever cases	cases	deaths		cough cases	causes
Oklahoma:											
Oklahoma City Texas:		2	0	0	1	1	0	1	8	0	42
Dallas Fort Worth				2	4	5	l o	0	1		60 36 21
Galveston	2		Ĭ	Ö	882	0	l ö	9	20	l ö	21
Houston	l é	3	l ĭ	ŏ	2	2	ŏ	25) š	Ŏ	81
San Antonio		2	. Õ	Ŏ		ō	Ŏ	8	Ŏ	Ŏ	68
Montana:			1								
Billings	0		0	0	1	0	0	1	0	0	12
Great Falls			0	0	1	Ő	0	0	1	0	8 2
Helena Missoula			0	0	0	0	03	0	0	8	12
Idaho:		,	l v	۱ ° ۱	U		Ů	v	1 .	ľ	
Boise	0)	0	0	1	2	0	0	0	0	7
Colorado: Colorado									ł		
Springs	0		0	1	0	0	1	2	0	0	10
Denver	ĬĬ		Ĭ	Ô	š	2	ō	6	l ŏ	34	96
Pueblo	Ō		Ō	ŏ	ĭ	2	Ŏ	Ŏ	Ŏ	1	10
New Mexico:				1 .1					Ι.		l
Albuquerque Utah:	0	'	0	0	0	1	0	2	1	0	14
Salt Lake City.	0)	0	1	3	6	0	1	1 0	5	37
Nevada:				-	Ŭ	Ű	Ŭ	-	· ·		
Reno		-									
Washington:									1		
Seattle											
Spokane Tacoma	0		8		0	8	0	0	0	20	24 25
Oregon:	0		U	0	2	0	0	0	0	0	20
Portland	0		0	l ol	1	8	0	1	0	13	62
California:					-	-	-				
Los Angeles	8 0	10	0	6	7	7	0	18	22	37	252 26
Sacramento San Francisco.	2			0	1 5	6 12	0	0 6	Ő	10	20 155
Dan Francisco.	-	1 *	ľ	· •	° I	12	۲ľ	v	Ů		100
	1				11			1			
		Mening	ococcus	Polio-				1		ococcus	Polio-
State and city		mení	ngitis	mye-		State a	nd city		meni	ngitis	mye-
Brase and City	ŀ			litis	11	Stard a	and truy	1-			litis
		Cases	Deaths	Cases					Cases	Deaths	Cases
					_						

City reports for week ended Aug. 29, 1936-Continued

State and city		gococcus ngitis	Polio- mye-	State and city	Menin meni	Polio- mye- litis	
	Cases	Deaths	litis cases		Cases	Deaths	cases
Massachusetts: Boston	0 0 8 2 1 1 1 1 1 1 2 1 0	1 0 2 0 1 1 1 1 0 0 1 1 1 1	2 1 0 1 0 7 2 1 0 0 1 1	Georgia: Atlanta Florida Tannessee: Memphis Nashville Alabama Birmingham Montgomery Louisisana New Orleans Shreveport Texas Dallas Montana Billings Colorado: Denver Washington Spotane California Los Angeles Barramento		0 0 10 0 0 2 0 1 0 0 10	0 0 2 1 1 1 1 0 1 1 1 2 2

Epidemic encephalitis.—Cases: Washington, 1; Birmingham, 1; Denver, 3. Pellagra.—Cases: Washington, 1; Savannah, 4; Dallas, 1; New Orleans, 3; Los Angeles, 3. Typhus fever.—Cases: Moline, 1; Charleston, S. C., 1; Atlanta, 1; Savannah, 2; Birmingham, 1; Mobile, 1; Montgomery, 2; New Orleans, 3.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended August 22, 1936.— During the 2 weeks ended August 22, 1936, 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 Bruns- wick	Quebec	Ontario	Mani- toba	Sas- katch- ewan	Alber- ta	British Colum- bia	Total
Cerebrospinal meningitis Chicken pox Diphtheria Dyseatery Erysipelas Lafuenza		2 6 1	6 8	41 27 1 4	1 71 11 2 18	5 8 3	 	12	1 22 1 8 5	2 173 58 8 12 24
Measles Mumps Paratyphoid fever	5	1 2	1	52 	74 63 5 2	44 3	28 5 2	8 5	83 28	240 105 5
Pneumonia Poliomyelitis Scarlet fever		7	5	6 66	12 93	32 62	2 27	1 85 1	2 14	15 55 809 1
Trachoma Tuberculosis Typhoid fever Undulant fever	2	87	25 10	79 47	96 16 5	6 3 1	1 7	1 2 2	5 23 	5 270 85 8
Whooping cough		12		171	167	2	4	5	29	890

CUBA

Habana—Communicable diseases—4 weeks ended August 29, 1936.— During the 4 weeks ended August 29, 1936, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria. Dysentery (bacillary) Malaria.	16 36 1 120	3 2	Poliomyelitis Tuberculosis Typhoid fever	19 14 170	1 1 18

1 Includes imported cases.

Provinces—Notifiable diseases—4 weeks ended August 22, 1936.— During the 4 weeks ended August 22, 1936, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Piñar del Rio	Habana	Matan- zas	Santa Clara	Cama- guey	Oriente	Total
Cancer Chicken pox	4	2		2 1 5		3 1 1	11
Hookworm disease Leprosy Malaria	198	2 102		1 394	221	i 677	1 3 1, 626
Measles Poliomyelitis Scarlet fever		3	 1 1		8	4	12
Tuberculosis Typhoid fever	12 22	19 68	· 7 38	35 62	37 31	34 30	144 251

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 August 28, 1936, pages 1214-1227. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued September 25, 1936, and thereafter, at least for the time being, in the issue published on the last Friday of each month.

Plague

Algeria—Oran.—On September 1, 1936, 1 suspected case of plague was reported at Oran, Algeria.

France—Marseille.—On August 30, 1936, 1 case of plague in a dock laborer was reported in Marseille, France. A report dated September 3, 1936, stated that 2 plague-infected rats were also reported in Marseille.

Hawaii Territory—Hawaii Island—Hamakua District—Paauhau Sector.—Two rats found on August 27, 1936, and 1 rat found on August 28, 1936, in Paauhau Sector, Hamakua District, Hawaii Island, Hawaii Territory, have been proved plague infected.

United States—California.—A report of plague-infected ground squirrels in Modoc County, Calif., appears on page 1320 of this issue of PUBLIC HEALTH REPORTS.

Yellow Fever

Nigeria—Kano.—On August 21, 1936, 1 suspected fatal case of yellow fever was reported in Kano, Nigeria.