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PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

January 29-February 25, 1939

The accompanying table summarizes the prevalence of eight important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the Public Health Reports under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4-week period ending February 25, the number reported for the corresponding period in 1938, and the median number for the years 1934-38.

DISEASES ABOVE MEDIAN PREVALENCE

Smallpox.—Although the number of cases (1,554) of smallpox reported for the 4 weeks ending February 25 was only about 70 percent of the number reported during the corresponding period in 1938, it was still approximately 75 percent greater than the 1934–38 median figure for this period. In the North Central region, Indiana reported 449 cases, Iowa 196 cases, and Ohio 134 cases, while in the West South Central region Texas and Oklahoma reported 148 and 134 cases, respectively—more than two-thirds of the total number of cases occurring in those five States. In the Atlantic Coast regions the incidence remains about normal, no cases being reported from the North Atlantic States and only 7 from the South Atlantic States.

DISEASES BELOW MEDIAN PREVALENCE

Influenza.—There was a sharp increase in influenza cases from approximately 13,000 cases during the 4 weeks ending January 29 to approximately 24,000 cases during the 4 weeks ending February 25.¹ The number of cases was almost twice the number recorded for the corresponding period in 1938, but it was about 10 percent below the 1934–38 median figure for this period. Some increase in the number of influenza cases is to be expected, since the disease is usually quite prevalent at this time of the year, and since the highest incidence of

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¹ Later reports appear on pp. 445-446.

the season has normally been reached during the period corresponding to the one under consideration. The disease has apparently been most prevalent in the South Atlantic and East North Central regions, with slight increases over the normal expectancy in the North Atlantic and Mountain regions. The West North Central and Pacific regions reported the lowest incidence in recent years, and in the West South Central region the number of cases was slightly below the average incidence.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period Jan. 29-Feb. 25, 1939, the number for the corresponding period in 1938, and the median number of cases reported for the corresponding period 1934-381

Division	Cur- rent peri- od	1938	5-year me- dian	Cur- rent peri- od	1938	5-year me- dian	Cur- rent peri- od	1938	5-year me- dian	Cur- rent peri- od	1938	5-year me- dian
	Diphtheria		Īr	fiuens	ensa i Measles i Menin meni		ningoco	occus tis				
United States 1	1, 994	2, 436	2, 436	23, 994	12, 990	28, 549	53, 546	131, 829	91, 6 67	227	378	525
New England Middle Atlantic East North Central. West North Central. South Atlantic East South Central. West South Central. Mountain Pacific	39 331 411 168 383 139 299 56 138	36 414 553 196 397 178 400 116 146	53 394 531 222 422 192 400 71 176	122 840 5,016 793 9,184 2,196 4,322 1,170 351	76 131 397 836 2, 995 1, 996 5, 299 585 675	76 220 1, 212 1, 285 8, 337 3, 630 5, 058 812 2, 390	6, 201 5, 590 5, 799 8, 274 9, 968 1, 494 2, 117 3, 251 10, 852	2, 410 42, 069 51, 204 6, 372 14, 954 8, 767 2, 338 2, 628 1, 087	5, 686 11, 383 10, 255 6, 372 9, 232 4, 589 1, 667 2, 628 2, 764	12 50 19 13 41 51 20 11 10	12 60 38 28 79 98 38 9 16	12 60 98 54 93 92 43 23 18
	Pol	iomyel	itis	Sci	rlet fe	ver	8	imalipo	r	typ	hoid fe	ver
United States 1	66	89	80	22, 169	24, 290	26, 877	1, 554	2, 241	883	433	523	52 1
New England Middle Atlantic Rest North Central West North Central. Bouth Atlantic. East South Central. West South Central Mountain. Pacific.	0 7 9 4 17 14 6 4 5	2 5 9 4 17 19 9 8 16	2 6 11 6 12 9 6 3 16	1, 539 5, 100 8, 659 2, 507 1, 067 623 764 586 1, 304	1, 893 5, 682 8, 245 3, 765 1, 034 615 844 897 1, 815	1, 585 6, 539 9, 057 3, 765 1, 034 614 654 897 1, 406	0 749 254 7 273 134 95	0 803 661 11 193 208 193 412	0 0 179 413 5 9 185 125 148	12 50 52 18 74 47 144 13 23	16 54 86 36 85 37 153 27 29	14 55 73 36 85 58 141 19 29

¹48 States. Nevada is excluded and the District of Columbia is counted as a State in these reports. ²44 States and New York City.

344 States and New York City.
346 States. Georgia and Mississippi are excluded.

Measles.—While the number of cases of measles rose from approximately 37,000 for the preceding 4-week period to more than 53,000 for the 4 weeks ending February 25, the incidence was only about 45 percent of that for the corresponding period in 1938 and was approximately 38,000 cases less than the 1934–38 average number for this period. The incidence in relation to recent years was especially high in the Pacific area, considerably above normal in the West North Central, West South Central, and Mountain States, and about 10 percent above the seasonal average in the New England and South Atlantic regions. On the other hand, the Middle Atlantic and East North Central areas reported only about one-half of the 5-year average number of cases for this period, and in the East South Central area the number of cases was only about one-third of the 1934-38 median figure.

Poliomyelitis.—The incidence of poliomyelitis was about 20 percent below the average incidence for this season of the year. For the 4 weeks ending February 25 the number of reported cases totaled 66, as compared with 89, 80, and 66 for the corresponding period in 1938, 1937, and 1936, respectively. In the South Atlantic and East South Central regions the incidence was slightly above the seasonal average, while in the Pacific area the number of cases was relatively low; other regions reported about the normal seasonal incidence.

Diphtheria.—For the 4 weeks ending February 25 there were 1,994 cases of diphtheria reported, as compared with 2,436, 2,069, and 2,369 for the corresponding period in 1938, 1937, and 1936, respectively. With the exception of a slight interruption in 1938, there has been a steady decline in the incidence of diphtheria. The current incidence is the lowest for this period in the 11 years for which these data are available; in 1929 the cases for this period totaled approximately 6,100 cases.

Typhoid fever.—The typhoid fever incidence was relatively low. The number of cases (433) reported for the current period was only about 80 percent of the average incidence for recent years. In the West South Central region the number of cases was approximately the same as the 1934-38 average figure for this period, but in all other regions the incidence was below the seasonal expectancy.

Scarlet fever.—The scarlet fever incidence was comparatively low— 22,169 cases as compared with approximately 24,000 cases in 1938, and an average of approximately 27,000 cases for the years 1934–38. Very significant decreases in the numbers of cases as compared with the experience of recent years were reported from the Middle Atlantic, North Central, and Mountain regions. For the country as a whole the number of cases reported during the current period was the lowest reported for the corresponding period in 10 years.

Meningococcus meningitis.—For the 4 weeks ending February 25 there were 227 cases of meningococcus meningitis reported, as compared with 378, 678, and 800 for the corresponding period in 1938, 1937, and 1936, respectively. Each geographical division of the country shared in the favorable situation, the incidence in each region, except the New England, being considerably below the normal seasonal occurrence of this disease. For the country as a whole the current incidence is the lowest since 1934, when a total of 227 cases was then reported for the corresponding period.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ending February 25, based on data received from the Bureau of the Census, was 13.2 per 1,000 inhabitants (annual basis). The rates for the corresponding periods in the years 1938, 1937, and 1936 were 12.0, 14.3, and 13.8, respectively. While the current rate is considerably above that of last year, it is lower than the rates for the corresponding period of the two preceding years. The low rate of 1938 was no doubt largely due to the low incidence of influenza during the winter months. While the reported influenza incidence is not relatively high this year for the country as a whole, it might be a contributing factor to the increased death rates in some large cities located in regions from which large numbers of influenza cases have been reported.

MOUTH LESIONS ASSOCIATED WITH DIETARY DEFICIENCIES IN MONKEYS

By N. H. TOPPING, Assistant Surgeon, and H. F. FRASER, Passed Assistant Surgeon, United States Public Health Service, National Institute of Health

INTRODUCTION

The etiological factors responsible for some of the various types of soft tissue lesions occurring in the oral cavity are not clearly understood. The infectious theory, with the spirals and fusiforms implicated as the causative agents, has been investigated by numerous workers (1) since the original papers of Plaut (2) and Vincent (3). Recently, in aphthous stomatitis, still another agent has been suggested—the virus of herpes simplex (4). This type of stomatitis has yielded the virus by inoculations into rabbits' corneas, and the recovered animals were subsequently immune to inoculations with a known strain of herpes simplex virus (4). A vesicular stomatitis has been described in animals and the virus has been recovered (5). The recovery of these viruses from vesicular lesions has placed this type of infectious agent on a sound basis as the etiological factor in these lesions.

The etiology of the periodontal diseases, including gingivitis, chronic, subacute and acute Vincent's infections, and pyorrhea alveolaris, remains for the most part unsettled. For some time various dietary deficiency diseases in their milder forms have been suggested as possible factors in the production of these conditions. The effect on the oral tissues of dogs depleted of vitamin A was studied by Mellanby (β); the effects of vitamin C deficiency have been studied both clinically (7) and experimentally (8); and recently certain components of the B₂ complex have been implicated, both from experimental work (9) and from clinical observations (10). However, many of these were supplementary observations made during the course of experiments designed for other purposes, and therefore were not adequately controlled from the viewpoint of a study planned primarily for an investigation of stomatitis. With this in mind, an experiment was designed to determine the occurrence of oral pathology in monkeys on selected dietary deficiencies.

EXPERIMENTAL

Selection of animals and preliminary observations.—Macacus rhesus monkeys were selected for this work because their dental formula is similar to that of man, and because this primate is the only species except man known to require both vitamin C and nicotinic acid, two of the nutritional factors which it was desired to test.

Fifty monkeys were examined clinically, with special attention to the condition of the oral cavity. In addition, a series of stool cultures, one complete blood count, and one blood serum cevitamic acid determination ¹ were made on each prospective experimental animal.

Following such initial observations, 14 used monkeys which had been maintained on a stock diet and 26 fresh stock monkeys were selected and placed in individual screen-bottom cages in 2 rooms connected by an open doorway and 1 large open window.

During a subsequent 3-week standardization period on stock diet 512, 2 of the original 40 animals were discarded because of anemia and replaced with animals observed in a preliminary manner for only 2 days. The monkeys were then divided into groups, being equalized as evenly as possible, taking into account age as estimated from the teeth,² sex, the results of stool cultures, and whether or not they were used or fresh stock animals.

Diets served monkeys.—The various groups were then placed on control and selected deficiency diets. The details of these diets, their preparation, and concurrent check assays of the monkey diets in rats are presented below.

Nukint	Diet number		
Nutrients	482	483	
Corn meal ¹ Cowpeas ¹ Cottonseed oil ¹ Sucrose ¹ . Salt mixture (Osborne & Mendel) ¹ Casein, leached Yeast, dried brewers'	Grams 320 50 50 45 24 60 60	Grams 350 50 50 45 24 83	

Composition of monkey diets 482 and 483 with supplements

¹ These items are stirred into water in a double boiler of enamelware and cooked for about 1½ hours. Then the other ingredients are well stirred in, the completed diet is dried in cakes for 24 hours and fed so that each monkey receives approximately 800 calories per day.

¹ We are indebted to Dr. Mary E. Reid for the blood serum cevitamic acid determinations throughout the course of this study.

³ Age estimated from "The Anatomy of the Monkey," by Carl Hartmann.

Supplements

Dist 482

A. Control animals:

- 1. Vitamin A (carotene in cottonseed ofl (S. M. A. Corp.)). 1 cc. (7,500 units) given by mouth once a weak to each monkey.
- 2. Vitamin D (crystalline in propylene glycol (Winthrop Chemical Co.)). 0.33 cc. (12,300 units) given by mouth once a week to each monkey.
- 3. Vitamin C (crystalline ascorbic acid (Merck) neutralized with sodium bicarbonate). 30 mg. 5 days a week and 60 mg. 1 day a week given by intramuscular injection.

B. Deficient animals:

Vitamin A, D, or C deficient animals received the vitamins described for the controls with one vitamin deleted according to the deficiency desired.

Diet 483

A. Control animals:

- 1. Riboflavin, synthetic (Winthrop Chemical Co.). 0.5 mg. given intramuscularly once a week in distilled water.
- 2. Nicotinic acid, 20 mg. given once a week, dissolved in distilled water.
- 3. Vitamins A. D. and C were given as outlined for diet 482.

B. Deficient animals:

- 1. B₂ complex deficient animals did not receive ribofiavin or nicotinic acid. (When ingredients comparable to those of basal diet 483 are assayed on rats for Be (11), adequate amounts are present. According to work on chicks (12, 13) and rats (14), it probably contains an insufficient amount of the filtrate factor. No filtrate factor concentrate was supplied in these experiments).
- 2. Nicotinic acid deficient animals received a supplement of flavin as described for controls.
- 3. Flavin deficient animals received a supplement of nicotinic acid as described for controls,
- 4. Vitamins A, D, and C were given as outlined for dist 482.

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Irish potatoes (cooked)	Bread (whole wheat)	Carrots (raw)	Carrots (cooked)	Irish potatoes (cooked)	Bread (whole wheat)	Bananas
Milk	Milk	Milk	Milk	Milk	Milk	Two eggs (cooked)
Lettuce	Bananas	Bananas	Lettuce	Cabbage (cooked)	Cabbage (raw)	
	One egg (cooked)	Lettuce				

Composition and schedule of monkey diet 495a

Approximately 1,000 calories were provided for each monkey daily. Since they ate about 800 calories daily, this permitted some freedom of choice in quantity of individual foods consumed. The monkeys ate all of the above diet very well except the cooked carrots. The food was prepared as follows:

Milk was made from whole milk powder daily and the equivalent of about 250 cc. of whole milk was Eggs were boiled approximately 5 minutes. Excessive cooking of vegetables was carefully avoided.

Composition of monkey stock diet 512

Each monkey received 6 days a week approximately 125 gm. of whole wheat bread, 250 cc. of whole milk, and three bananas. One day a week each monkey received two hard-boiled eggs.

Concurrent check assays of monkey diets in rats.—A group of 75 rats, including males and females, ranging in age from 21 to 25 days, were used to test each diet as prepared for monkeys, except the diet deficient in vitamin C and diet 495a composed of natural foods. They were observed for rate of growth, symptoms, ability to reproduce, and success in lactation. Rats on control artificial diet 482 with supplements of vitamins A and D grew well and were successful in reproduction and lactation. Rats on basal diet 482 plus vitamin D but minus vitamin A grew rather poorly and did not reproduce; but since they maintained their weight fairly well and did not develop xerophthalmia, the presence of some vitamin A in the basal diet was suggested. Rats on B₂ complex control diet 483 with supplements of vitamins A, D, nicotinic acid, and flavin grew quite well, reproduced, and some were partially successful in lactation. Two red blood cell and two white blood cell counts were made at varying intervals from five rats in each of the four groups of the B₂ complex and showed no definite evidence of anemia or leucopenia. (The satisfactory response of control B₁ complex rats on basal diet 483, plus supplements of vitamins A, D, nicotinic acid and flavin, is noteworthy in view of the poor response of monkeys on a similar diet.)

Routine observations on monkeys.—Once weekly, or more frequently when necessary, routine observations were made on each animal's weight, general condition, and particularly the condition of the oral cavity. Determinations of hemoglobin content and total white and red cell counts were made less frequently. In addition, blood serum cevitamic acid³ determinations were made to ascertain the extent of depletion in deficient animals and the adequacy of the vitamin C supplement.⁴

Smears were made from the gums of all the animals every other week. The same method for making these smears was followed throughout, and consisted of passing a small cotton swab over the gums, several millimeters away from the cervical margin. These swabs were streaked on slides, stained with gentian violet, studied under oil immersion at once, and then saved for final study when the entire set was completed upon the death of the monkey.

Stool cultures were made every 2 weeks on all the monkeys and more frequently on those developing diarrhea. Both eosin methylene blue and desoxycholate citrate agar were used as primary isolation media and *Shigella paradysenteriae* (Flexner) was recovered many times. Blood cultures were taken on two of the monkeys developing noma, and both were negative.

In the planning of the experiment it was considered desirable that all animals proceed to natural death without treatment,⁵ and this plan was rigidly adhered to, with the following exceptions: (1) Animals obviously *in extremis* were killed with chloroform in order that postmortem autolysis would not interfere with the pathological studies. (2) Seven control animals and one A-deficient animal were killed with chloroform after about 210 days in order to terminate the experiment.

Photographs were taken of the mouths of all the monkeys before the experimental diets were begun, of those in which pathology was observed, and, finally, of most of the animals at death. These photographs were carefully reviewed in evaluating the clinical progression of the lesions, and particularly in weighing the severity of lesions at death.

Since it is impracticable to publish the series of photographs for each animal, typical examples have been selected of the mouths of two normal animals, following 209 and 210 days on the control diets, and other pictures demonstrating each type of lesion (as listed in table 1) observed at death. These photographs are presented in figures 1 to 8, inclusive, and illustrate the descriptive terms employed.

A routine autopsy was performed and the various tissues were preserved for histopathological studies by Passed Assistant Surgeon T. H. Tomlinson, of the Division of Pathology, National Institute of

³ Pure synthetic vitamin O.

⁴ A dose of 30 mg. of vitamin C given 5 days a week and 60 mg. 1 day a week was considered sufficient, since the average blood serum cevitamic acid of monkeys on the artificial diet plus vitamin C was higher than that of monkeys on diet 495a composed of foods with a high vitamin C content.

[•] A preliminary experiment was conducted on 20 monkeys using the same basal diets reported in this experiment, but with the onset of deficiency symptoms, specific therapy was instituted.

	Cause of desth and autopey findings	Obloroform. Do. Obloroform; tuberculo- Sis Obloroform.	Chloroform. Not definitely estab- lished.	Chloroform. Tuberculosis. Do. Acuto gastro-enteritis of unerplained origin Tuberculosis.	Chloroform. Tuberculosts. Do. Not definitely estab- lished.
5/	Survival time in day	210 211 209 209	210 211 124	146 7 1280 146 7 1280	22 19 19 8 19 19
	smoN	000 O	0 00	~ ~ ~ ~	00 00
	Ulcerations in buc- cal mucosa	000 O	0 00	~ ~ ~ ~	00 00
lesi	Necrotic gingivitis	000 O	0 00	~ ~ ~ ~	00 00
thut	Necrosis of inter- dental papillae		0 00	<u> </u>	00 70
dea dea	Gingivitis	000 0	0 00	<u> </u>	00 70
ptior	Localized recession	•• <u></u>	0 00	<u> </u>	00 40
escri	noitsamafinI	000 0	0 00	<u> </u>	00 40
9	Soft tartar	00 00 0	o o‡	<u></u>	0# 40
e in	દ્યક્રમાંવુરિ		Tenpo- rary	÷	
Bmears fro increase	arrolizu I	H	Tempo- rary	*	
	Progress of lesions	None do Chronio gingivitis→subsided. None	Cleared Note: Oleared	None Cleared Doubtful progression None	Cleared. No progression
{	Description of mouth le- sions when first noted	None. do. Some localized glogivitis None.	Blight localized gingivitis. None. Slight eingivitis in upper Jett premolar and molar	Note vors slight fingivits in upper right and left pre- noar avea. I alt pre- some localized recession. Note Very slight gingivits in Upper premokr segon.	siight localized gingfyltia. Siight gingfyltia with hard tatar. Very alight gingfyltia. None
verobeq	Month lesions dev after — days	0 H	2 °3	6 0 13 80 0	37 8 8 P
18 8010	beginning of experi-	328 k	* *	88 7 88	88 38
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	Monkey number	885 8	8 48	<u>22</u> 28 28 28	88 28
Vitamin deficiency		(sontrols) None	iving vita- d D)	None (controls rece mins A, C, a	A deficient
	Diet No.	490a1		53	

TABLE 1.-Summarization of pertinent data

Perforation of gut.	Tuberculosis.	00. 00.	Sourvy; tuberculosis.	Scurvy.	Scurvy; tuberculosis.	Scurvy.	Not definitely estab- lished.	Tuberculosis. Do.	Noma.	Tuberculosis. Bacillary dysentery.	Noms; tuberculosis.	Tuberculosis.
8	8	101 127	111	75	120	76	8	132 146	8	\$ \$	22	8
•	0	00	0	•	•	•	•	••	\$	00	\$	•
•	•	00	0	•	•	•	•	00	\$	••	\$	•
Ŧ	#	00	#	‡	•	*	‡	<u>_</u>	\$	°‡	<u>+</u>	•
2+	2+	••	4	‡	2+	34	‡	° ,	*	04	\$	•
2+	2+	<u>+</u> +	\$	‡	2+	‡	1	*# *#	#	°. °.	\$	•
34	5	°‡	1	‡	3+	‡	≿	- <u></u>	*	00	*	•
5	;	5 ⁺⁺	1	‡	3 +	*	1	*# *#	\$	°.	\$	•
34	2 +	5++ 5++	*	3+	*	<u></u>	•	74	<u></u>	••	*	*
2+	7	(Ŧ)	8+	3+		*		2+	3+	2	*	
2 <u>+</u>		(†)	*	5		3+		2+	3+	2+	<u></u>	
<pre>#ingivitis → recession → local- /acd normals</pre>	Died before progress could be	Some progression Further accumulation of tar- tar and localized separation of gum at cervical border.	Generalized redness→hemor- rhage→ulcerations→necro-	ous. 	Some progression	Generalized redness-hemor- rhage-detachment of gums ->necrosis.	Gingivitis → generalized red- ness at cervical margin→	Vary little progression Redness→hypertrophy→local- ized necrosis.	Hemorrhagic gingivitis → ne- crosis of gums→extension to mouth mucoss→noma.	None Very little progression	Redness-hemorrhagic gingi- vitis-hecrotic gingivitis- extension to cheeks-bilat-	eral noma. None
Very slight hypertrophic C	Early necrosis of inter-	Very slight gingivitis Boft tartar and some gin- givitis at cervical mar- gin.	Moderate redness in up- per right and left pre-	Very slight redness in upper right and left	Some gingivitis and slight hemorrhage at cervical margin	Some gingivitis with dif- fuse reddening of gums at cervical margin.	Areas of hemorrhagic gin- givitis.	Slight localized gingivitis. Generalized redness of gums with some hyper-	Areas of hemorrhagic gin- givitis.	None. Moderate redness at cer-	vical margan. Generalized diffuse red- ness of gums.	None
8	6	4 8	12	15	3	14	8	23	ន	08	8	<u> </u>
47	8	35	8	ĸ	8	\$	ង	89	8	\$ 8	31	8
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(43	\$	35	9	47	22	7	8	8 8	8	3 8	3	²⁸
C deficient D deficient)	ziois nin oin	sn (cont ving fi itopin	None None Dns Dis	-ge	d s niva Jusio	u		
				1	-	3						

Bee footnotes at end of table.

	Cause of death and autopsy findings	Not definitely estab- lished.	Tuberculosis. Possibly bacillary dys-	entery. Bacillary dysentery.	Noma; tuberculosis.	Not definitely estab- lished.	Tuberculosis.	Do.	
5.	zab ni əmit lavivus	11	85 38	69	134	103	8	8	
. 43	8moN	0	00	•	1	•	•	•	
ons a	Ulcerations in buc- cal mucosa	0	<u></u> +.	+	4	34	•	•	
1 lesi	Necrotic gingivitis	2	00	++	+	+	3+	0	
thouth	Necrosis of inter- dental papillae	3	00	++	+	‡	3+	5	
of m deat	dingivitis	*	00	#	4	#	*	5+	
otion	Localized recession	37	00	3+	+	2+	3+	5+	
escri	Inflammation	3+	50	#	1‡	#	#	5 +	
Å	Soft tartar	5	00	3+	5	3+	2+	5	
mgums in-	glstiqS			2+	+		2+	2+	
Smears fro increase	maolisuA			5 +	2+		2+	2+	
	Progress of lesions	Redness→ necrosis of interden- tal papillae→necrotic gingi-	vitis. Very little progression None	Hemorrhage → hemorrhagic gingivitis→necrosis of gums →extension to cheeks.	Severe gingivitis-mecrosis of gums-extension to buccal	mucosa-noma. Gingivitis-necrosis of inter- dental papillae - necrotic gingivitis-extension of buc-	cal mucosa. Redness->superficial ulcera- tions in sulci->hemorrhagic	gineivitis→necrosis. Redness-necrosis of some in- terdental papillae→hemor- rhagic gingivitis.	
	Generalized redness of gums.	Slight localized gingivitis. None	Hemorrhage from gums	Faint red line at cervical margin.	Gingivitis at cervical mar- gin.	Generalized redness hy- pertrophy of upper	interdental papillae. Re†ness at cervical mar- gin.	ural foods.	
pədoja	Mouth lesions developed after — days			13	8	11	8	35	f nat
JIS SUJU	beginning of experiment			39	\$	36	88	8	sed o
	xəS	0+	55	ъ	6	0+	0	б	mpoç
	Monkey number			72	35	62	4	1	is coi
	Vitamin deficiency	¢ Pl	inic act	Nicoti Nicot	0	nlcotini cient	b ns t ñeb b	nivalii 198	Diet 495a
	Diet No.				483				[

TABLE 1.—Summarization of pertinent data—Continued

¹ The control monkeys in the vitamin B₁ complex group with a basal diet containing vitamin B₂ and receiving surplements of nicotinic acid flavin vitamins A, D, and C responded in such a manner that they may have required one or more additional factors (contained in the yeast of diet 432) for normal nutrition. (See supplements diet 433.) 1 = Slinet. 2 = Mid. 3 = Moderate. 4 = Bart earter. 4 = Bart earter.

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Health. The pathological findings are made the basis of a concurrent report in this issue of the Public Health Reports.⁶ In the great majority of animals our clinical impression of the oral lesions was confirmed by these pathological studies.

The records of all the experimental animals and a summary of pertinent data is presented in table 1.

DISCUSSION OF TABLE 1

General.—Observing the table as a whole, it is at once evident that the control animals on both stock diet 495a and control diet 482 failed to develop as many or as severe mouth lesions as those animals on deficient diets. Individual monkeys, however, in all deficient groups show considerable variation regarding the extent of oral pathology.

Although table 1 shows tuberculosis as a significant cause of death in only 20 monkeys, gross or microscopic lesions typical of tuberculosis were found at autopsy in 26 out of 40 animals. However, this does not appear to invalidate observations on the development of mouth lesions, since table 1 shows that there is apparently no correlation between the incidence of clinical tuberculosis and mouth pathology.

Control monkeys on stock diet 495a.—All of these four animals lived to the termination of the experiment on the two hundred and tenth day. Three of the four animals, 34, 60, and 65, had no lesions of the oral cavity at any time, and at death their gums were in as good condition as, or even better than, at the beginning of the experimental period. One monkey, 61, developed a mild, localized gingivitis, which subsided to the mild chronic form. There was no progression of the lesions. He was diagnosed clinically as having tuberculosis on August 9, 1938, 2 months before the experiment was terminated.

Control monkeys on diet 482 with appropriate supplements.—There were eight animals in this group. One of these, monkey 73, will not be considered, as he died on the seventh day of the experiment. Three of the remaining seven animals were killed to terminate the experiment on the two hundred and tenth day. The average survival time for the group was 165+' days. Two monkeys, 41 and 55, at no time had any lesions in the oral cavity. Four, 39, 50, 58, 79, had at some time very slight, transient gingivitis, which cleared before death. Three of these, 50, 58, and 79, had a slight amount of soft tartar along the cervical gum margin at death. Only one monkey out of the seven, 69, developed a moderate degree of inflammation about the gingival margin, which became chronic before death. This

[•] The next article in this issue.

^{&#}x27;The plus mark following days of survival means that one or more monkeys in the group were chloroformed in order to terminate the experiment, and so it is impossible to calculate the complete survival time of the group.

animal had positive stool cultures for S. paradysenteriae (Flexner) before the experimental diet started. On several occasions there was blood and mucus in his stools, and during the experimental period practically all of his stools were positive for Flexner organisms. Paralysis of an undetermined cause developed in the left leg of the animal about 1 month after the experiment was started and continued until death. Gross lesions of a widely disseminated tuberculosis were found at autopsy. All of these facts make it extremely difficult to evaluate this monkey's condition.

"A" deficient monkeys on diet 482.—Of the four monkeys on diet 482 deficient in vitamin A, one was alive at the termination period of 210 days. The average survival period for the group was 162 + days, which compares favorably with the control group on the artificial diet. A simultaneously conducted rat experiment and observations of other workers on cowpeas (15) suggest that although this diet contains some vitamin A, it is nevertheless deficient in this respect. One animal in this group, 68, at no time developed lesions of the oral cavity. Two monkeys, 36 and 46, manifested some transient gingivitis which cleared up by the time of death. One animal, 46, developed hard tartar on his incisor teeth, the only 1 of all 40 animals upon which this was observed. The remaining monkey in the group, 51, showed a chronic gingivitis which was present at death.

"D" deficient monkeys on diet 482.—Of the four monkeys on diet 482 deficient in vitamin D, none was alive on the two hundred and tenth day. The average survival time for the group was 85 days. All four animals in this group developed some pathology of the gums. Monkeys 53 and 75 developed only a slight gingivitis. Monkeys 43 and 44 developed a gingivitis which proceeded to necrosis of some of the interdental papillae and finally to a slight generalized necrotic gingivitis.

"C" deficient monkeys on diet 482.—Of the four monkeys on diet 482 deficient in vitamin C, none was alive on the two hundred and tenth day. The average survival time for the group was 95 days. Three of the four monkeys developed a severe gingivitis, while the other, 57, had only a mild gingivitis. The latter monkey received 60 mg. of cevitamic acid by error shortly after manifesting gum lesions, which then cleared and did not recur with the same severity as those of the other three members of the group. In this monkey the lesions progressed only to a necrosis of the interdental papillae, while in the other three, 40, 47, and 71, all developed a generalized necrotic gingivitis, which in two, 40 and 47, was extremely severe. The diagnosis of scurvy was confirmed at autopsy by the findings of hemorrhage into the joints, subperiosteum, and in one instance, into the gastrointestinal tract.





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Figure 3.—Postmortem photograph Monkey 51, illustrating 2+ soft tartar, 2+ inflammation, 2+ localized receision, 1+ necroisis of interdental papillae, and 1+ mercofic gingivitis. (A deficient diet.)

FIGURE 4.—Postmortem photograph Monkey 47, illustrating 3+ soft tartar, 4+ inflammation, 4+ localized recession, 4+ gingivitis, 4+ necrosis of interdental papillae, and 4+ necrotic gingivitis. (C deficient diet.)



Figure 5.-Postmortem photograph Monkey 40, illustrating 3+ soft tartar. 4+ inflammation, 4+ localized recession, 4+ gingivitis, 4+ necrosis of interdental papillae, and 4+ necrotic gingivitis. (C deficient diet.)

Figure 6.—Postmortem photograph Monkey 72, illustrating 3+ soft tartar. 4+ inflammation, 3+ logitizer teesson, 4+ gingivitis, 4+ necrosis of interdental papillae, 4+ necrotic gingivitis, and 4+ ulcerations of buccal mucosa. (Note arrows.) (Nicotinic acid deficient diet.)

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PLATE IV





FIGURE 7.—Postmortem photograph Monkey 35, illustrating 2+ soft tartar, 4+ inflammation, 4+ localized recession, 4+ gingivitis, 4+ necrois of interdential papillae, 4+ necrotic gingivitis, 4+ ulcerations in buccal mucosa, and noma. (Flavin and incontinc acid deficient dist.)



FIGURE 8.—Photograph taken shortly before death of Monkey 63, illustrating a case of noma. (B₃ complex control diet, containing flavin and nicotinic acid.)

 B_2 complex deficient monkeys.—It was hoped at the beginning of the experimental period that some light would be thrown on the B_2 complex requirements of the monkey. However, the development of intercurrent infections so complicated the results that little knowledge could be gleaned of the requirements of this species. Nevertheless, certain characteristic mouth lesions developed in this group with sufficient regularity to be significant, more especially since this type of lesion was not observed in any of the other groups.

There were 4 groups of 4 monkeys each, making a total of 16, on some phase of the B_2 complex deficiency. None of these monkeys was alive at the end of the experimental period of 210 days. The average survival time for the 4 groups was 73 days. All of the 16 developed a severe watery diarrhea, yet in only 2 of these was a possible etiological cause demonstrated. In many of these monkeys some anemia associated with a moderately severe leucopenia developed which was not observed in other groups of monkeys. On B_2 complex deficient diets, anemia (16) (20), leucopenia (9c) (20), and diarrhea (9a) (20) have been observed in monkeys; and diarrhea has been described in other species (17).

Twelve of the 16 animals developed gingivitis; 11 had necrosis of the interdental papillae; 10 had generalized necrotic gingivitis, 6 developed ulcerative lesions in the buccal or labial mucosa where it came in contact with the gingival margins; and, finally, 3 in the group progressed to complete gangrenous necrosis of the cheek which was similar in appearance to the lesions described in human beings, as "noma." Only 2 monkeys, 45 and 67, had no lesions of the oral cavity at any time. Both of these died very early of an intercurrent infection in 27 and 38 days, respectively.

Ulcerative lesions of the gums have been described recently in monkeys on B_2 complex deficiencies (9a) (9c) (16) (20). However, to our knowledge, the extension of these lesions to the buccal or labial mucosa has not been recorded. These mucosal lesions observed in monkeys vary considerably from those described in canine black-tongue (18).

From our data it would appear that neither nicotinic acid nor flavin, either individually or together, would prevent these lesions from developing under the conditions of this experiment; however, it must be pointed out that the groups of monkeys receiving either or both of these substances survived longer than the group of monkeys receiving neither.

Sebrell and coworkers (19) have maintained dogs in an excellent condition for a period of 6 months on a basic diet comparable to diet 483 of this experiment when the dogs' diet was supplemented with cod liver oil, nicotinic acid, and flavin. In a concurrent rat experiment, here reported, using diet 483 and supplements of A, D, nicotinic acid, and flavin, a fair rate of growth and successful reproduction have been obtained. The poor response, therefore, of the control monkeys in the B₂ complex group receiving diet 483, plus supplements of A, D, nicotinic acid, and flavin, is noteworthy. These observations and those of Day, Langston, and Darby (9c) in monkeys suggest that our control B₂ complex diet, as compared with the same diet with yeast added, was still deficient for normal nutrition in the monkey.

A significant observation is that only the monkeys on the B_2 complex deficiencies developed primary ulcers at sites where the teeth and buccal mucosa approximate, yet the C-deficient group had just as severe or even more extensive lesions in the gums proper.

Results of smears from the gums.—Spirals and fusiforms were present in varying numbers in the smears from all the monkeys during the preliminary observation period. Some of the monkeys (41, 44, 45, 47, 51, 55, 59, 61, 67, 69, 71, 72, 73, 77, 78, 79) had rather large numbers of these organisms at this time, while the others had but few. Table 1 shows that there is apparently no correlation between the original number of organisms and subsequent development of mouth lesions. The data regarding the increase of spirals and fusiforms refers to gum smears made from the entire mouth. It shows that as ulcerative lesions develop in the gums the number of spirals and fusiforms increases to some degree; but this was not consistently observed. However, when smears were made directly from ulcerative lesions instead of the entire mouth large numbers of spirals and fusiforms could be demonstrated in almost every instance.

Condensations of table 1.—For purposes of clearness and emphasis, the data of table 1 have been condensed somewhat in tables 2 and 3.

Group	Number in group	Number in group killed after 210 days	Number in group dying	Survival time in days
Controls on stock diet Controls on artificial diet A deficient. D deficient. C deficient. B ₁ complex deficient. B ₂ complex controls Nicotinic acid deficient Flavin deficient.	4 57 4 4 4 4 4 4	4 3 1 0 0 0 0 0 0 0	0 4 3 4 4 4 4 4 4	• 210+ 165+ 162+ 95 76 98 67 54

 TABLE 2.—Survival time in days of monkeys on various diets tested (Condensation of data presented in table 1)

 The plus mark following days of survival means that one or more monkeys in the group were chloroformed in order to terminate the experiment, so it is impossible to calculate the complete survival time of the group.
 1 animal died on the seventh day and was therefore excluded from consideration in this table.

Table 2 summarizes the survival time for the various groups, regardless of the cause of death. Since all of the monkeys were kept in individual cages in the two rooms, the effects of the various diets may have had some effect on their cage-age at death. If this is true, it

•							
Group	Total num- ber in group	Number in group affected and weighted severity of lesions	Gingi- vitis	Necro- sis of inter- dental papillae	Ne- crotic gingi- vitis	Ulcer- ations of buc- cal mucosa	Noma
Controls on stock diet	4	Number in group affected. Weighted severity of le- sions.		0 0	0	0	0
Controls on artificial diet.	• 7	Number in group affected. Weighted severity of le- sions.	1 0.1	1 0.1	0	0	0
"A" deficient	4	Number in group affected. Weighted severity of le- sions.	1 0.8	1 0.3	0 0	0	0
"D" deficient	4	Number in group affected. Weighted severity of le- sions.	4 1. 5	2 1.0	2 0. 5	0	0
'C" deficient	4	Number in group affected. Weighted severity of le- sions.	4 8.5	4 3. 8	3 2.5	0 0	0
B ₁ complex deficient+ supplements of flavin and nicotinic acid.	} 4	Number in group affected. Weighted severity of le- sions.	4 8.8	8 1.8	8 2.3	1 1.0	1 1.0
B ₂ complex deficient+ supplement of nico ^t inic acid.	} 4	Number in group affected. Weighted severity of le- sions.	2 1.8	2 1.5	2 1. 8	1 1.0	1 1.0
B ₃ complex deficient+ supplement of flavin.	} 4	Number in group affected. Weighted severity of le- sions.	2 1.8	2 1.8	2 1.5	9 1.3	0 0
B ₂ complex deficient	4	Number in group affected. Weighted severity of le- sions.	4 8.5	4 3. 3	3 2.8	2 1.8	1 1.0

TABLE 3.—Mouth lesions observed at death in monkeys on various diets tested

(Condensation of data presented in table 1)

• 1 animal died seventh day; therefore excluded from consideration in this table.

perhaps was at play as a factor in host resistance to spontaneous infection. As striking as these results are, it must be remembered that the groups are small, and before much reliance can be placed on such a possible correlation many more experiments of this type must be made.

In table 3 the data on mouth lesions have been condensed. The less severe lesions (i. e., soft tartar, localized recession, and inflammation) were deleted so that differences between the groups might be more evident. In addition, in order to take into account both the number of animals affected and the severity of the lesions present in those animals involved, a process of "weighting" has been made. This has consisted of adding the numbers denoting degree of severity in each animal and dividing by the number of animals in the group. From this table it is apparent that both the control diets protected the monkeys from developing mouth lesions, and the diet supposedly deficient in vitamin A was about as efficient in this respect as the artificial control diet. In the remaining deficiency groups, however, there is a definite difference in the numbers developing lesions and the severity of these lesions. Again the point is stressed that only in some part of the B_2 complex deficiency did lesions in the buccal mucosa develop, and in these cases only did noma appear.

ATTEMPTS AT TRANSMISSION OF ORGANISM FROM INFECTED LESIONS TO NORMAL MONKEYS ON A STOCK DIET

In order to test the infectivity and the virulence of the spirals, fusiforms, and other organisms occurring in the mouths of those monkeys developing lesions, it was decided to try several direct transfers of this material to the mouths of normal monkeys. The general plan was thoroughly to swab the lesions with a saline-soaked cotton swab, emulsify this immediately in about 3 cc. of saline, and then to use this suspension at once to inoculate the mouths of normal monkeys on stock diet 495a. Following this, the suspension was studied in stained smears and under the dark field to establish the fact that large numbers of viable (by motility) organisms were actually inoculated. The animals were all observed daily for evidence of infection. The individual protocols are presented in table 4, where it is noted that no gingivitis or stomatitis resulted from this procedure.

		Source of i	noculum				Inoculated			
Date	Monkey No.	Deficiency	Description of mouth lesion on date of transfer	Spirals and fusiforms ¹	Motil- ity	Transferred to monkey No.	Preparation of gums for transfer	Results		
June 14, 1938	52	B ₂ complex.	Noma of right cheek. Be- g i n i n g noma of left	}2+	Motile	451 373 345 70	Deep swabbing of socket of re- cently extracted superior central incisor. Laceration of buc- cal mucosa. Laceration of in- terdental papil- lae. No trauma	Negative after 2 weeks. Do. Do. Do.		
June 17, 1938	35	B ₂ complex.	Acute gingivi- tis with ne- crotic area upper cen- tral incisor area.	 }₄+	Motile	452 453 454	No trauma. Laceration of buc- cal mucosa. Laceration of in- terdental papil- lae.	Negative after 2 weeks. Do. Do.		
June 17, 1938	40	"C" <u></u> {	Acute general- ized gingivi- tis.	}3+	Motile	455 456 457	No trauma Laceration of buc- cal mucosa. Laceration of in- terdental papil- lae.	Negative after 2 weeks. Do. Do.		

TABLE 4.—Data on attempts at transp	mission
-------------------------------------	---------

¹2+=small number. 3+=moderate number. 4+=large number.

It would seem from these 3 separate attempts at transmission to 10 normal animals on a stock diet that even though spirals and fusiforms were present in relatively large numbers, they were incapable of initiating primary or secondary lesions regardless of whether the tissues had been previously traumatized. Furthermore, other organisms present in the saline suspension were also incapable of initiating lesions in the mouths of normal monkeys.

One monkey, 451, deserves an added comment. In this monkey the inoculation was made by a deep swab of the socket of a superior central incisor which had been removed several hours before. This should have furnished adequate anaerobiosis for the growth of the organisms, yet healing went on normally and at no time were any unusual reactions noted.

SUMMARY AND CONCLUSIONS

Although the number of animals is relatively small, observations on Macacus rhesus monkeys are presented which indicate that certain of the dietary deficiencies tested are associated with the development of varying manifestations of gingivitis, stomatitis, periodontal disease, and noma. Comparable animals maintained on adequate diets showed little or no evidence of such oral pathology. In addition, monkeys maintained on a stock diet showed no evidence of gingivitis or stomatitis when material from affected monkeys was transferred directly to them.

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ORAL PATHOLOGY IN MONKEYS IN VARIOUS EXPERI-MENTAL DIETARY DEFICIENCIES *

By T. H. TOMLINSON, JR., Passed Assistant Surgeon, United States Public Health Service

INTRODUCTION

Topping and Fraser¹ have stated the objectives and outlined the methods of procedure followed in their experiment. This paper will

^{*}From the Division of Pathology, National Institute of Health.

¹Topping, N. H., and Fraser, H. F.: Mouth lesions in monkeys associated with dietary deficiencies. Preceding article in this issue.

report the oral pathology found in the 39 Macacus rhesus monkeys included in their study.

No reports have been found in the literature giving the histopathology of oral lesions in monkeys from experiments conducted simultaneously relating to the influence of vitamins A, C, and D, and nicotinic acid and flavin.

MATERIAL AND METHODS

Since changes in the soft tissues were to be studied rather than those occurring primarily in the teeth or bone, ground sections were not made. The questions of bone resorption, osteoclasia, new bone formation, and tooth development will be omitted from this paper and may be reported later when additional studies with ground sections are made.

In securing the blocks for histopathological study, the entire head was split in the median sagittal plane and fixed in 10 percent formalin. The jaws were disarticulated and, with each half held firmly in a clamp, a small, fine to medium toothed hack-saw was used to take a transverse section about 5 mm. in thickness through the alveolar process from both the right and left sides of the mandible and maxilla. Some sections were taken through the erupted teeth and others through the interdental regions. When gross lesions were evident, the sections were taken at these points.

These blocks were decalcified in 22.5 percent formic acid in 10 percent sodium citrate solution. Each was placed in 30 cc. of this decalcifying solution for 1 week, at the end of which time the acid was poured off and fresh acid added for a period of another 7 to 10 days, depending on the hardness of the tissue at that time. It was found that routine decalcification for 3 weeks or longer resulted in poor cellular detail as regards infiltrating leucocytes, while less than 2 weeks' treatment left the section too hard for satisfactory sectioning. Sixteen to eighteen days appeared to give the most satisfactory results.

It was felt that minute cellular detail was a little better in this decalcified material when the paraffin sections were stained with hematoxylin-eosin than when the hematoxylin-Romanowsky technique was used. In addition, sections stained by Van Gieson's picrofuchsin were studied on each animal.

In recording findings the sections were divided into four parts the free gingiva, the periodontal membrane, the intercancellous tissue, and bone. The free gingiva was subdivided into the tip, the lateral mucosa, the part lining and subjacent to the crevice, and the epithelial attachment with its subjacent fibrous tissue.

CONTROL ANIMALS

The changes found in the four animals on stock diet No. 495a consisted of slight to moderate lymphocyte and plasma cell infiltration of the corium of the free gingiva, usually beneath the crevice and the epithelial attachment, with occasional small foci of lymphocytes and neutrophils in the crevice epithelium. In 3 animals, 4 areas out of 16 showed slight thickening of the epithelial attachment accompanied in 1 area by a few rete pegs entering the corium.

In the 7 animals on basic diet No. 482 with appropriate supplements to render it presumably adequate, there was moderate to marked focally dense round cell infiltration of the corium of the free gingiva which generally involved the tip as well as the crevice and attachment Animal No. 58 showed in two areas pericellular edema and regions. neutrophil infiltration of the epithelium with loss of superficial cells from the tip to the cemento-enamel junction. Nos. 50 and 69 each showed ulceration in a similar location, while the latter also showed necrosis of the entire free gingiva on the labial side in one section. Monkey 39 showed marked focal perivascular round cell infiltration in the walls of the tooth follicles. This was not seen in any of the The epithelial attachment showed in 3 monkeys other 38 animals. occasional areas of necrosis and in 3 others slight to moderate thickening accompanied in 2 of them by numerous rete pegs.

Since most of the reported work on oral pathology as related to vitamin deficiencies had been carried out on guinea pigs, rats, and dogs, it was felt that considerable attention should be directed at the beginning of this work toward the determination of the possible significance in *Macacus rhesus* of round cell infiltration in the corium of the free gingiva. In addition to the 11 controls on this experiment, a few monkeys, most of which had been used in virus work, were examined, from 4 to 12 sections being taken from each. Occasional sections were obtained from almost all of these animals showing a thin, compact epithelial attachment with no rete pegs and no leucocytes in the corium of the free gingiva, or, at the most, a dozen or so scattered lymphocytes. However, about one-half or more of the sections from every monkey showed cellular infiltration as described above either in the lateral or medial free gingiva, occasionally in both.

In view of these findings it was not felt that much significance could be attached to round cell infiltration beneath the crevice and attachment epithelium when occurring alone, at least not in *Macacus rhesus* as fed, housed, and handled in our laboratory.

VITAMIN A DEFICIENCY ³

Here there was no more than a very slight keratosis of the alveolar epithelium. There were occasional small foci of edema and lymphocyte and neutrophil infiltration in the epithelium of the crevice and attachment. In animal 51 the crevice and attachment epithelium was necrotic, and No. 46 showed ulceration and marked cellular infiltration of the epithelium of one interdental papilla. Beneath the areas of maximum epithelial involvement there were many round cells and often a few neutrophils. The plasma cell and lymphocyte infiltration of the corium of the free gingiva was moderate in two animals and marked in the others. There was slight to moderate thickening of the epithelial attachment in three animals accompanied in two of them by a few rete pegs.

D DEFICIENCY

Three out of the four monkeys showed in one-fourth to one-half of the sections taken from each animal complete necrosis of the free gingiva, the term complete necrosis meaning loss of cellular detail. usually with, but occasionally without, preservation of stroma outline. In one of these there was also moderately extensive necrosis of the periodontal membrane and intercancellous tissue and some focal bone This monkey also showed hemorrhage and much free and necrosis. phagocytosed blood pigment in the areas of necrosis and infiltration. In the other two there was moderate to marked focal lymphocyte and neutrophil infiltration of the intercancellous tissue. In the gingiva of the fourth monkey (No. 53) and in the surviving gingiva of the others there were occasional foci of neutrophils and usually moderate, often dense, lymphocyte and plasma cell infiltration of the corium often extending from the tip to the cementoenamel junction as a wide zone beneath the epithelium. This animal also showed slight thickening of the epithelial attachment with a few rete pegs.

C DEFICIENCY

In this group all four animals showed extensive necrosis of the gingiva with, in one of them, moderately extensive necrosis of the periodontal membrane, intercancellous tissue, and bone. In the surviving areas there was marked neutrophil, lymphocyte, and plasma cell infiltration of the gingiva occasionally extending into the periodontal membrane and the intercancellous tissue. The areas of gingival involvement often showed occasional to numerous, small to large hemorrhages, much free and phagocytosed golden brown granu-

² That the depletion of vitamin A in this group may have been a relative one is pointed out by Topping and Fraser in their paper.

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FIGURE 1.—No. 61. Stock diet 495a. Thickening of epithelial attachment with rete pegs. Round cell infiltration beneath attachment and bottom of crevice. X 42. (N. I. H. 994.)



FIGURE 2.—No. 40. C deficiency. Necrosis and focal suppuration in free gingiva with subjacent plasma cell and lymphocyte infiltration, fibroblast proliferation, little free blood, and free and phagocytosed blood pigment. X 42. (N. I. H. 992.)



FIGURE 3.—No. 43. D deficiency. Necrosis of free gingiva. Subjacent round cell and neutrophil infiltration and hemorrhage extending into the intercancellous tissue, peridental membrane, medial alveolar mucosa, and periosteum of medial alveolar bone plate. Focal necrosis of alveolar bone crest. Much blood pigment. X 16. (N. I. H. 1000.)



FIGURE 4.—No. 72. Nicotinic acid deficiency. Necrosis of free gingiva with subjacent round cell and neutrophil infiltration. Focal hemorrhage above alveolar bone crest on labial side. \times 16. (N. I. H. 1004.)

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PLATE III



FIGURE 5.—No. 30. B₂ complex deficiency plus nicotinic acid and flavin. Extensive labial gingival necrosis extending into peridental membrane. Hemorrhage in gingiva and peridental membrane. \times 16. (N. I. H. 989.)

lar pigment giving the prussion blue reaction, and some darker ironfree pigment. This pigment was more abundant in the free gingiva, but there were scattered and clumped pigmented macrophages in the corium of the lateral alveolar mucosa some distance down from the alveolar crest. A little free and phagocytosed blood pigment with occasional small hemorrhages were seen in the intercancellous tissue.

B₂ COMPLEX DEFICIENCY PLUS NICOTINIC ACID AND FLAVIN³

In this group one monkey showed slight focal superficial necrosis of an interdental papilla and in the other three there was variably extensive necrosis in all parts of the gingiva. In No. 30 the necrosis was focally suppurative but more often hemorrhagic and involved to a moderate degree the periodontal membrane. There was also extensive hemorrhage and much blood pigment in the gingiva and periodontal membrane. Monkey No. 63 showed a large perforating lesion of the left cheek, a section through the margin of which showed complete necrosis of all tissues with little marginating inflammatory reaction. In a cross section of the upper jaw beneath this lesion there was complete necrosis involving the internal free gingiva, the external gingiva, the maxillary mucosa, and the bucco-maxillary sulcus. The periodontal membrane, the intercancellous tissue in areas, and the bone throughout most of the section were necrotic.

FLAVIN DEFICIENCY

Monkey No. 52 showed an area of necrosis in the right cheek 32 mm. in diameter and a similar-8 mm. area on the left side, both centrally perforated. Marginating the necrotic areas were diffuse round cell and neutrophil infiltration and moderate edema. Beneath both of these areas the upper jaws showed complete necrosis of the gingiva and of the intercancellous and peritrabecular tissues, the periodontal membrane, and most of the bone throughout the distal half of the alveolar process, including often the lateral and medial alveolar plates. In one section there was an old hemorrhage in the region of the tooth socket. The other three animals showed usually slight to moderate, sometimes focal, lymphocyte and plasma cell infiltration of the corium, usually beneath the crevice epithelium and the epithelial attachment, less frequently in the tip of the free gingiva. Two of these three showed very slight focal superficial necrosis of the gingiva and the other one showed a moderately thickened epithelial attachment with numerous rete pegs.

³ The adequacy of this diet as a B₂ complex control for monkeys was discussed by Topping and Fraser in their paper. The extensive pathology found in these animals suggests that one or more other deficiency factors may be involved.

NICOTINIC ACID DEFICIENCY

In this group No. 38 and No. 72 showed extensive, usually complete, occasionally suppurative necrosis of the free gingiva involving also the lateral alveolar mucosa. The subjacent surviving soft tissues showed round cell and neutrophil infiltration. Both monkeys showed focal extension of the infiltration and the necrosis into the periodontal membrane and the intercancellous tissue, with slight marginal necrosis of adjacent bone in No. 38. Monkey No. 72 showed on the inner surface of the lower lip a small circumscribed area of complete necrosis of the epithelium and subjacent corium, marginated by lymphocyte and neutrophil infiltration. The other two animals in this group showed slight to moderate, often focal round cell infiltration of the corium of the free gingiva, with moderate thickening of the epithelial attachment.

FLAVIN AND NICOTINIC ACID DEFICIENCY

One monkey (No. 77) from this group showed in one section ulceration of the tip of the free gingiva. The free gingiva of the other three showed extensive suppuration and necrosis, restricted in one animal (No. 62) to the labial free gingiva but involving the gingiva of the entire alveolar process in the other two and extending in areas below the alveolar bone crest. In these two (Nos. 35 and 64) there was moderately extensive necrosis of the periodontal membrane, and in No. 35 the intercancellous tissue, in areas throughout the distal half of the section, was necrotic and surrounded a few fragments of dead Nos. 62 and 77 showed moderate to marked round cell infiltrabone. tion of the corium of the surviving gingiva with many rete pegs entering from the attachment. Monkey 35 showed on the oral surface of the upper lip a 1 cm. penetrating ulcer and on the lower lip a similar 3½ by 2 cm. lesion extending to the mucocutaneous border. Sections from these areas showed complete necrosis of the oral mucosa extending about one-half way through the subjacent fibrous tissue and muscle except at the crest of the lips, where it extended completely through and involved the epidermis. Marginating inflammatory reaction was very slight. Animal No. 62 showed ulceration of the buccal mucosa opposite the areas of gingival necrosis.

DISCUSSION

The epithelial attachment showed some alteration in most of the animals, varying from slight focal thickening to complete necrosis; however, six monkeys showed no changes in any of the sections examined. Types of involvement and group distribution are shown in table 1.

Diet and number of animals	Normal	Thickening	Rete pegs	Necrosis
Stock 495a (control): (4)	65	34 7 60± 61+	61+	
482 plus supplements (control): (7)	55 79	39++ 41± 50++	39++ 50++	50± 58∓ 69+
A deficient: (4)	68	36 7 46+ 51++	46+ 51+	51+
D deficient: (4)		53+	53+	43+++ 44++ 75+++
C deficient: (4)				40+++ 47± 57+++ 71+++
B ₂ complex deficient plus nicotinic acid and flavin: (4)	48	63++		30+++ 63++ 56+
Flavin deficient: (4)	78	45++	45++	49+ 52+++
Nicotinic acid deficient: (4)		38++ 67++	59±	38+++ 72+++
Flavin and nicotinic acid deficient: (4)		77++	62++ 77++	35+++ 62++ 64+++ 77+

TABLE 1.—Epithelial attachment 1

1 Figures are animal identification numbers and + and - signs indicate degree of pathological condition.

Since all animals on this experiment showed cellular infiltration of the free gingiva, no attempt is made to evaluate this finding. There was round cell infiltration of the intercancellous tissue without necrosis in three monkeys, and perivascular round cell infiltration in the walls of the tooth follicles in one.

Blood pigment or hemorrhage was present, and occasionally both, in sections from all of the C deficiency animals, one of the D deficiency, one of the flavin deficiency, and one of the group on B_2 complex deficiency plus flavin and nicotinic acid. Sections showing brown pigment in the tissues were stained for hemosiderin by the Perls-Abbott method, sometimes with negative results, but frequently a positive prussian blue reaction was obtained. This variability was expected in accord with Lillie's ⁴ recent studies on the solubility of hemosiderin in acids.

Necrosis (table 2) was not seen in any of the animals on stock diet 495a and was present in the gingiva of only 3 on diet 482 plus appropriate supplements. Among the remaining 28 monkeys, 16 showed

⁴ Lillie, R. D.: Experiments on the solubility of hemosiderin in acids and other reagents during and after various fixations. In press.

extensive gingival necrosis and 6 others showed ulceration, either of the free gingiva or the interdental papillae. There was slight to extensive necrosis of the periodontal membrane and intercancellous tissue in 7 of these and of the periodontal membrane alone in 2 others. Necrosis of bone was seen in 6 animals and always in association with inflammation and necrosis of adjacent soft tissues. The necrosis of the gingiva and related structures was fairly evenly distributed throughout the various deficiencies with the exception of the (partial?) A deficiency group where the animals showed only slight focal necrosis of the gingiva. In all of the other groups from 2 to 4 animals showed rather extensive gingival necrosis with 1 to 2 monkeys from each group showing in addition necrosis of the periodontal membrane, intercancellous tissue, and bone.

Diet and number of animals	Gingiva	Periodontal membrane	Intercancel- lous tissue	Bone	Cheeks and lips
Stock 495a (control): (4)					
482 plus supplements (control): (7)	58∓ 50± 69++				
A deficient: (4)	46± 51+				
D deficient: (4)	43+++ 44++ 75+++	43++	43++	43+	
C deficient: (4)	40+++ 47+++ 57+++ 71+++	40++	40++	40++	
B ₁ complex deficient plus flavin and nicotinic acid: (4)	30+++ $48\pm$ 56++ 63+++	30++ 63+++	63+++	63+++	63 (Noma)
Flavin deficient: (4)	49± 52+++ 78±	52+++	52+++	52+++	52 (Noma)
Nicotinic acid deficient: (4)	38+++ 72+++	38+ 72++	38+ 72+	38+	72
Flavin and nicotinic acid deficient: (4)	35+++ 62+++ 64+++ 77±	35++ 64++	35+++	35++	35 (Noma) 62

TABLE 2.-Necrosis 1

Figures are animal identification numbers and + and - signs indicate degree of pathological condition.

Among the 16 monkeys fed a B_2 complex deficient diet with variations only in the flavin and nicotinic acid factors, 1 showed marked ulceration of the buccal mucosa, 1 a large circumscribed area of necrosis on the oral surface of the lower lip, and 3 showed perforating necrotic lesions of the cheeks or lips.

SUMMARY

The feeding of 39 monkeys on diets deficient in certain vitamins resulted in the development of necrosis of the periodontal tissues and oral mucosa of varying extent, qualitatively similar in the B_2 complex deficiencies with variations in the flavin and nicotinic acid factors, in the C deficiency, and in the D deficiency, and probably greatest in extent in the B_2 deficient groups.

A STUDY OF EXPERIMENTAL PERTUSSIS IN THE YOUNG RAT*

By J. W. HORNIBROOK and L. L. ASHBURN, Passed Assistant Surgeons, United States Public Health Service

Burnet and Timmins (1) showed that the intranasal instillation of *Haemophilus pertussis* in the mouse produced pneumonia and death. This has been confirmed by Bradford (2) and Culotta (3). One of us found that young rats more uniformly developed a fatal pneumonic infection than did mice when inoculated in this manner, and that the required infecting dose was considerably smaller for rats than for mice. Thus approximately 8,500,000 phase I organisms cause infection in the rat as compared with 200,000,000 in the mouse. Those rats which do not die early of pneumonia usually develop paroxysms in which air is forcibly expelled from the mouth or nose. This resembles a cough or sneeze. Though adult rats seldom die from the inoculation, they develop a more vigorous cough than the younger animals. The cough can be heard a distance of 10 to 20 feet. Vomiting was not observed.

INFECTION IN THE RAT

Method of inoculation.—A loopful of a 24-hour growth of phase I H. pertussis from Bordet Gengou media was suspended in saline. Using Burroughs Wellcome opacity tubes and the H. influenzae opacity table as a standard, an estimate was made of the number of organisms per cc. This suspension was then diluted so as to give an estimated 170,000,000 organisms per cc. The young rat (25-40 grams) was anesthetized with ether and held on its back, and the suspension was dropped into the nose with the aid of a syringe and blunt needle, the material being drawn into the respiratory tract during inspiration. The capacity of the lungs of young rats of the size mentioned rarely permits the instillation of volumes greater than 0.1 cc. The practice was to instill 0.05 cc. into each rat. If rhonchi were not felt, the rat was reanesthetized and the dose repeated.

Both Wistar albino and hooded rats were used in these experiments.

Results of inoculation.—In all, 51 animals were inoculated. Six were killed on the 2nd day and five on the 4th day for pathological examination. Twenty-six of the remaining 40 animals died on or

[•]From the Division of Infectious Diseases and the Division of Pathology, National Institute of Health, Washington, D. C.

before the 9th day, a mortality of 65 percent. The surviving animals were killed on or after the 10th day for section. Those animals dying of the disease survived an average of 5.2 days, with 9 days as the longest survival time and 3 days as the shortest. None of the rats showed any evidence of toxicity until a few hours before death. Animals which appeared perfectly well, except possibly for an increased respiratory rate, showed rather extensive lung changes at autopsy.

With a given strain of H. pertussis comparable results were obtained in each series of rats inoculated. On the other hand, when mice were used as the test animal the results were very irregular. A dose which killed in one series would often fail in another, though the technique and number of organisms used were the same.

A record of coughing was kept on 12 rats. The cough appeared in an average of 8.3 days following inoculation. The termination of the cough was difficult to estimate, as it disappeared so gradually. In some cases cough continued for 60 days.

Isolation of H. pertussis from the infected rat.—The lungs of 35 rats were cultured, with the following results:

Time inoculation to death, in days	Number of animals	Number of animals posi- tive for per- tussis
1-5	6 16 1 5 3 4	3 9 1 0 0 2

H. pertussis cultures were recovered from only 43 percent of the 35 rats. This low percentage may be explained in part by the fact that many cultures were overgrown with spreading colonies so as to obscure any H. pertussis which might have been present. The tabulated results also include earlier work when cultures were made by means of tracheal swabs. Subsequently one lung from each animal was ground and used as the inoculum. This produced a higher percent of positive cultures.

The organism isolated from the 60-day animals seemed to grow more rapidly than usual. With phase I antiserum this organism agglutinated in a dilution of 1:540, while fresh phase I bacilli agglutinated in a dilution of 1:1620 with the same serum.

Pathology.—The absence of post mortem changes made 31 animals suitable for pathological study; and of these, all but five showed microscopic evidence of lung changes. As is to be expected, the microscopic findings in these rats showed progressive changes with prolongation of the infection. The rat lung reacts to the introduction of H. pertussis by a process generally described as interstitial bronchopneumonia. In the early stages the exudate in the alveoli is by far the most striking feature; later, as this is resolved, the interstitial reaction becomes prominent. The number of consolidated areas in one lobe, the size of these areas, and the number of lobes involved are quite variable; also, the separate foci frequently have their inception at varying intervals, and consequently many stages of the developing process may be seen in the same lobe or different lobes of the two lungs.

The earliest reaction was seen about 48 hours following inoculation. There is slight congestion of the alveolar capillaries and slight to moderate serum exudation into alveoli. The areas involved are generally small, are in a peri- or para-bronchial location, and usually near the hilus. Not all alveoli in a given area are involved; some are solidly filled, although most show incomplete filling, and few have only a thin coating on the surface of septa. Interspersed with these are varying numbers of air-containing and occasionally dilated alveoli. In few scattered, and occasionally grouped, serum-containing alveoli there are occasional to few neutrophils, occasional small macrophages, and rarely a little fibrin. Alveolar septa show very slight neutrophil infiltration, with slight proliferation and enlargement of septal cells, few of which appear as macrophages. Small vessels show narrow surrounding zones of adventitial edema, occasionally with very few infiltrating neutrophils. The small bronchi show similar infiltration but without edema. Rarely a neutrophil is seen in the epithelium of small bronchi and bronchioles. A few small Gram-negative bacilli are scattered throughout alveolar serum and rarely in small groups on the surface of bronchial epithelium.

By the third day areas of involvement have become much larger, reaching nearly to the pleural surface, and occasionally an entire lobe is consolidated. Edema fluid is quite dense, and fewer alveoli con-Neutrophils in the exudate are much more numerous and tain air. occasionally show pyknosis and karyorrhexis; in some alveoli they are rather densely packed; generally they occur in moderate numbers; however, numerous scattered and grouped alveoli contain only serum, even in the older central area of the lesion. This irregular distribution of cells is also striking in more advanced stages. A few small and large macrophages are admixed with the neutrophils, but they are slightly more prominent in the less cellular exudate. Occasionally their ample cytoplasm contains oxyphilic hyalin globules. The septa show moderate irregular thickening, congestion, variable though usually moderate proliferation and swelling of septal cells, with few scattered neutrophils and fairly numerous macrophages and large mononuclear cells. These latter cells often protrude as knob-like projections from septa. The large vessels near the hilus show moderate perivascular serum exudation with moderate infiltration by neutrophils and few lymphocytes, mast cells, and macrophages. A similar but less prominent reaction is present around some smaller vessels. Around bronchi very small amounts of serum occur with occasional to few neutrophils. The epithelium of small bronchi and bronchioles is about twice its normal thickness, cells are considerably enlarged, particularly in height, and nuclei are oval, or round, and leptochromatic. The cilia-bearing surface is frayed and quite uneven. Little serum, few neutrophils, and occasional red cells are present in the lumen. Infrequently the lumen is completely filled with serum. At this stage bacteria are present in very great numbers, in clumps, and scattered throughout the alveolar exudate, and less numerous in bronchial exudate and on the surface of the epithelium. Few are phagocytosed.

From the third to fourth day there is simply an accentuation of the changes already established. More neutrophils show degenerative changes, serum is thinner and thready, macrophages in alveoli are more numerous and larger, and few to many binucleate or occasionally multinucleate cells are present. These latter cells have little basophilic cytoplasm and their large nuclei are deeply stained and vesicular. Septa show slightly greater thickening and in many areas are lined by discrete or coherent, large, occasionally vacuolated epithelial cells, few of which lie free in the alveoli. Occasionally septal cell proliferation and swelling of epithelial cells completely obliterate alveolar spaces. The perivascular and peribronchial reaction shows little progression. Cells are slightly more numerous, with relative and actual increase in mononuclear cells, mainly macro-The fibroblasts are swollen but show no significant proliferaphages. Edema fluid is no longer seen in the adventitia of smaller tion. vessels. Bronchial exudate and epithelium show no additional changes except a little focal serum exudation between epithelium and supporting tissues.

By the fifth to sixth day the alveolar exudate shows much necrosis and is decreased in amount; however, macrophages are still moderately numerous and usually of the large foamy type. Septa show no significant additional change. The striking feature is the increased prominence of the peribronchial and perivascular reaction, particularly near the hilus. In addition to considerable increase in number of cells, there is moderate fibroblast proliferation. The infiltration and proliferation occupy a wider zone than that previously seen and extend to involve the loose tissue about the lung roots, and occasionally extend along the pulmonary vessels to the base of heart. Perivascular and peribronchial edema is no longer present. Epithelial cells of small bronchi and bronchioles still show hypertrophy and marginal fraying, and cells of larger bronchi are quite tall. In an



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FIGURE 1.—Rat lung 2 days following intranasal inoculation with H. pertussis showing alveoli filled with serum and few emphysematous air spaces. Edema fluid seen in upper right part of photomicrograph is perivascular in location. \times 250. (N. I. H. 1084.)



 $\label{eq:Figure 2.--Three-day lung lesion showing cellular exudate in alveoli, mainly neutrophils, also a little serum and fibrin. <math display="inline">\,\times\,$ 250. (N. I. H. 1052.)



FIGURE 3.—Five-day lung lesion showing thickening of alveolar septa, increase in number and size of septal cells, and large mononuclear and multinuclear cells in alveoli. X 340. (N. I. H. 1051.)



FIGURE 4.—Five-day lung lesion showing area in which some alveoli are obliterated by proliferation and exudation of large mononuclear and multinuclear cells. X 340. (N. I. H. 1053.)



 $\label{eq:Figure 5.-Six-day lesion showing marked peribronchial and perivascular mononuclear cell infiltration and fibroblast proliferation. <math display="inline">\times$ 125. (N. I. H. 1058.)



FIGURE 6.—Nine-day lung lesion. High-power photomicrograph of perivascular reaction, similar to that seen in figure 5. \times 250. (N. I. H. 1059.)

occasional animal a few epithelial cells of large bronchi contain mucus, and a few strands of mucus and a few neutrophils are inconstantly present adherent to the epithelial surface.

Usually by the tenth day certain areas show almost complete absorption of alveolar exudate with the exception of occasional to few remaining macrophages. Alveolar septa show less cell infiltration, and their increased thickness is due mainly to enlargement of cells lining alveoli, the alveoli being reduced in size. In this stage there is still slight to moderate peribronchial and perivascular fibroblast proliferation with slight infiltration by large mononuclear cells. Bronchial epithelium shows no significant alteration, although an occasional group of small Gram-negative bacilli occur among the cilia.

In an occasional animal unresolved pneumonic foci have been found as late as fifteen days. This may be due to the fact that these lesions have had their onset some time after inoculation. This factor makes it difficult to follow the chronological development of the lesions after the tenth day. Focal, peribronchial septal thickening with atelectasis was present in one animal 21 days after inoculation.

It seems that in animals that do not die of the pneumonia the lungs return to their normal appearance, except in an occasional case where one or more small abscesses occur. These were present in two animals killed on the tenth post-inoculation day and showed an incomplete peripheral layer of loosely disposed fibroblasts surrounding an area of suppuration in which occasional non-necrotic alveolar septa persist. Bacteria, usually in clumps and morphologically similar to those seen in earlier lesions were present in the necrotic exudate. No abscesses were seen in later stages, but it is evident that such a lesion would leave a residual scar.

The tracheal reaction is generally very slight and often absent. It consists of slight congestion of mucosal corium, with very few scattered neutrophils. Epithelium is not altered; occasionally, however, a clump of small gram negative bacilli was present among the cilia. Serum was rarely present in lumen.

Other organs examined, including heart, liver, kidney, thymus, and spleen, showed no significant alteration.

DISCUSSION

The lung reaction occurring in rats is similar to that described in mice, and as has been pointed out by Bradford (2) and others, is not specific for H. pertussis. An essentially similar lesion has been produced experimentally by bacterial toxins, viruses, and a variety of bacteria—B. typhosus (E. typhosa), B. bronchisepticus, B. lepisepticum, and H. influenzae.

In mice, Burnet and Timmins (1) found goblet cells in the bronchiolar epithelium, with a film of mucus on the surface, and Bradford

(2) noted considerable amounts of mucus and cellular exudate accumulated in alveolar spaces and much mucus on the surface of bronchial epithelium. However, Culotta and associates (3), also working with mice, make no mention of the occurrence of mucus but record the presence of "homogeneous pink staining material" in the lumina of bronchioles. This absence of mucus and the presence of serum as found by Culotta and associates is in agreement with the findings in Since the stagnation of mucus is usually stated to be the cause. rats. of spasmodic cough in human pertussis, the absence of mucus secretion in most rats (which also appear to cough) is interesting. Also, mechanical blockage of cilia, as suggested by Mallory and Hornor (4), cannot account for the cough in rats since so few bacteria were present among cilia.

SUMMARY AND CONCLUSIONS

The intranasal instillation of live phase I H. pertussis organisms into young rats produces a nonspecific interstitial bronchopneumonia which often leads to death (65 percent in our series).

Phase I H. pertussis organisms can be recovered from the involved lungs, particularly if the entire lung is ground and used as the inoculum.

The young rat is superior to the mouse as an experimental animal because of the fewer organisms required to infect and because of the higher and more uniform death rate. Furthermore, the rat so infected usually develops cough-like paroxysms, a phenomenon which in itself has experimental significance.

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INFLUENZA PREVALENCE

The week ended March 11, 1939, recorded another increase in the number of cases of influenza—18,135 cases being reported as compared with 14,288 for the preceding week.

The greatest prevalence is still confined principally to the four groups of Central States and the South Atlantic region, with a slight increase shown in the Mountain and Pacific States, especially New Mexico and Oregon. Considerable increases are noted during the current week, as compared with the preceding week, for Wisconsin (from 584 cases to 1,516), North Dakota (364 to 741), Virginia (1,509 to 1,991), North Carolina (97 to 386), Georgia (140 to 420), Kentucky (1,348 to 1,792), Tennessee (146 to 469), and Alabama (599 to 1,126). The New England and Middle Atlantic States remained comparatively free from the disease.

The effect of the mild epidemic prevalence of influenza is now reflected in the weekly reports from cities, which show an increase in the number of cases reported and in the number of deaths attributed to the disease, but it is not yet indicated by excess deaths from pneumonia. For the week ended March 4, 1939, a group of large cities scattered throughout the United States and having an aggregate population of approximately 33,000,000, reported 1,271 cases of influenza, with 190 deaths, as compared with 5-year averages of 736 and 128, respectively; these same cities reported 891 deaths from pneumonia as compared with a 5-year average of 989.

The seasonal peak of influenza will evidently come much later this year than in prior years. In the past 11 years there have been three more or less mild influenza epidemics—1928-29, 1932-33, and 1937. In the first two the rise was rapid in November and December, the earlier epidemic reaching the peak of incidence in January, while the 1932-33 outbreak reached its peak in December. In 1937 the sharp rise occurred early in January and the peak was reached during the week of January 30, when 37,101 cases were reported.

The accompanying table gives the numbers of cases of influenza reported by States, arranged by geographical divisions and by weeks, from the first of the year to and including the week ended March 11.

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Cases of influenza reported by weeks, Jan. 1-Mar. 11, 1939

						-				
Division and State	Jan. 7	Jan. 14	Jan. 21	Jan. 2	Feb. 4	Feb. 11	Feb. 18	Feb. 25	Mar. 4	Mar. 11
NEW ENGLAND										
Maine New Hampshire	1	8	2	10 1	4	1	8	25	46	103
Verbiolit	•									·
Dhodo Island	•				-	-				
Connecticut	10	6	13	4	7	26	22	29	30	141
MIDDLE ATLANTIC		_								
New York	. 44	57	37	155	159	183	137	101	91	57
New Jersey	. 14	24	12	19	1 56	61	99	44	24	19
Pennsylvania					-					
BAST NORTH CENTRAL										
Ohio										·
Indiana	12	11	22	4	21	21	363	1,085	607	35
Illinois	. 18	12	60	30	36	227	955	1, 478	1,241	838
Michigan			1	2			39	255	429	674
Wisconsin	62	65	52	47	68	65	56	346	584	1,516
WEST NORTH CENTRAL										
Minnesota		2	3	2	;-	1	3	24	12	40
10W8		- 1	10				2/	291	1,063	695
Missouri	1 24		12	33	1 2	15	13/		264	0/8
South Dekote	34		10		1 4	10	14	01	304	141
Nahaaka	0			1	1 1	10	3	0	1 "	00
Vopena	18								116	
Kausas	10		•	U	l v	• •			110	440
SOUTH ATLANTIC										
Delaware										
Maryland	4	5	12	10	61	103	182	209	124	53
District of Columbia	2	2	6		5	5	18	25	25	11
Virginia	454	420	282	617	1, 100	553	1,338	1,604	1,509	1,991
West Virginia	21	13	34	41	21	26	33	36	271	71
North Carolina	3	7	28	9	9	18	71	230	97	386
South Carolina	909	495	865	649	772	701	972	592	1, 181	1, 142
Georgia	133	136	143	110	131	118	139	110	140	420
Florida	1	1	2	5		1	1		9	3
EAST SOUTH CENTRAL										
Kentucky	5A	65	37	27	109	K1	479	ANK	1 949	1 709
Tennessee	36	64	87	100	58	75	63		146	460
Alabama	158	191	188	160	250	186	160	190	500	1 1 2 6
Mississioni				100		100	100	100	000	1, 120
WEST SOUTH CENTRAL										
Arkansas	181	203	145	139	159	87	113	182	1, 473	1, 532
Louisiana	~ ~ ~	36	12	8	10	20		9	30	82
Oklanoma	222	149	119	193	162	207	129	193	334	387
Texas	492	716	631	703	699	621	983	787	965	968
MOUNTAIN										
Montana		26	23	<i>K</i> 0	25	42	25	200 İ	196	125
Idaho	4	2	ī	ĩ	1		~]	12	~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~	14
Wyoming	-	- 1		-					il	1 A
Colorado	21	21	31	45	35	93	125	121	150	134
New Mexico	2	ī	21	ĩõ	6	ğ	ī		57	677
Arizona	138	117	132	81	68	114	82	94	144	191
Utah	7	1	2	9	20	24	16	4	53	119
		-	-	-						
PACIFIC										
Washington		4	1 .			1	8 .		8	8
Oregon	71	39	46	53	25	40	42	34	97	261
California	41	41	82	33	76	43	28	59	50	78
I										
Total	3, 255	3, 018	8, 097	3, 395	4, 310	3, 802	6, 895	8, 987	14, 288	18, 135

DEATHS DURING WEEK ENDED FEBRUARY 25, 1939

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

	Week ended Feb. 25, 1939	Correspond- ing week, 1938
Data from 88 large cities of the United States: Total deaths. Average for 3 prior years. Total deaths, first 8 weeks of year. Deaths under 1 year of age. Average for 3 prior years. Deaths under 1 year of age. Average for 3 prior years. Deaths under 1 year of age, first 8 weeks of year. Deaths under 1 year of age, first 8 weeks of year. Data from industrial insurance companies: Policies in force. Number of death claims. Death claims per 1,000 policies in force, annual rate. Death claims per 1,000 policies, first 8 weeks of year, annual rate.	10, 086 19, 713 75, 530 589 1 594 4, 433 68, 013, 875 12, 447 9, 5 10, 0	1 8, 608 72, 099 1 554 4, 332 69, 772, 226 12, 091 9, 0 10, 0

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 March 4, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median

		Diph	theria		Influenza					Measles			
Division and State	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, me- dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934 38, me- dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, me- dian	
NEW ENG.										•			
Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	36 0 6 0 0	6 0 5 0	8 0 2 5 1 8	0 0 1 8 1 4	278 89	46 30	3 2	6 21	60 91 308 1, 248 107 1, 454	10 9 23 1, 061 14 490	165 33 172 242 1 8	165 30 44 916 43 91	
MID. ATL.													
New York New Jersey Pennsylvania	9 12 19	23 10 38	31 14 51	42 16 51	1 63 29	¹ 91 24	1 18 29	1 32 29	490 54 92	1, 224 45 182	1, 848 1, 437 7, 508	1, 848 842 3, 823	
E. NO. CEN. Ohio Indiana Illinois Michigan ³ Wisconsin	37 25 21 13 0	48 17 32 12 0	22 38 41 33 13	30 27 41 12 6	902 813 453 1, 026	607 1, 241 429 584	22 14 2 74	103 89 66 2 98	24 34 15 338 1, 909	31 23 23 320 1, 086	2, 170 955 6, 933 3, 564 4, 316	421 528 1, 139 73 1, 136	
W. NO. CEN.													
Minnesota Iowa Missouri North Dakota South Dakota Nebraska Kansas	6 8 32 0 30 30 30	3 4 25 0 4 0 1	3 5 13 0 0 17 11	3 8 20 2 9 15	23 2, 192 828 2, 658 579 8 324	12 1, 083 644 364 77 2 116	5 1 146 2 2 42 21	3 6 355 12 2 	2, 171 389 18 1, 570 2, 104 160 28	1, 120 192 14 215 280 42 10	63 54 907 8 0 23 382	227 54 662 8 8 29 246	
SO. ATL.													
Delaware Maryland ³ Dist, of Col Virginia West Virginia North Carolina ³ Georgia ³ Florida	39 15 57 30 19 20 33 3 15	2 5 7 16 7 14 12 2 5	3 14 7 23 12 31 9 3 13	1 9 7 16 12 19 6 8 5	382 202 2, 828 728 142 3, 226 232 27	124 25 1, 509 271 97 1, 181 140 9	10 1 52 36 481 1	53 2 218 174 799 304 83	3, 321 154 472 35 2, 284 74 254 567	1, 077 19 252 13 1, 563 27 153 188	26 66 5 461 531 2, 659 610 404 569	66 146 25 461 73 787 72 102	

See footnotes at end of table.

Cases of certain disease	s reported by telegrap	h by State heal	th officers for	the week
ended March 4, 1989,	rates per 100,000 popu	lation (annual)	basis), and c	omparison
with corresponding we	ek of 1938 and 5-year	median—Conti	nued	-

	Diphtheria					Influ	enza		Measles			
Division and State	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, me- dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934– 38, me- Gian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934– 38, me- dian
E. 80. CEN.												
Kentucky Tennessee Alabama ³ Mississippi ² ³	12 14 14 10	7 8 8 4	13 7 11 8	16 10 19 4	2, 343 257 1, 054	1, 348 146 599	41 75 195	80 215 889	97 141 401	56 80 228	602 725 1, 117	269 52 463
W. SO. CEN.												
Arkansas Louisiana ³ Oklahoma Texas ³	17 19 18 33	7 8 9 40	11 10 10 47	3 15 10 47	3, 654 73 672 800	1, 473 30 334 965	184 7 222 888	140 37 244 897	189 443 298 273	76 183 148 330	310 7 58 5 9 4	58 51 54 538
MOUNTAIN												
Montana Idaho. Wyoming Colorado New Mexico Arizona Utah ³	0 10 131 14 12 61 10	0 1 6 8 1 5 1	0 1 0 15 3 0 6	2 0 8 7 1 0	1, 180 10 22 722 704 1, 767 526	126 1 150 57 144 53	7 	29 3 8 85 	3, 398 806 2, 596 472 470 380 1, 291	363 79 119 98 38 31 130	49 3 17 620 60 9 281	49 28 17 188 60 37 24
PACIFIC												
Washington Oregon California	12 5 37	4 1 45	6 0 27	8 0 33	25 482 41	8 97 50	4 68 53	3 109 202	1, 085 298 3, 153	352 60 3, 845	8 33 398	132 116 564
Total	18	456	606	606	674	14, 288	2, 798	5, 727	643	15, 922	41,011	30, 806
9 weeks 4	22	4, 939	5, 803	5, 918	268	51, 047	27, 416	49, 965	477	106, 124	242, 887	166, 268

	Me	ningiti: coc	s, meni cus	ngo-	Poliomyelitis				Scarlet fever			
Division and State	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Miar. 5, 1938, cases	1934– 38, me- dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934– 38, me- dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, me- dian
NEW ENG.												
Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	0 0 2.4 0 0	0 0 2 0	0 0 8 0 1	0 0 3 0 1	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	163 41 121 269 92 291	27 4 9 229 12 98	12 33 16 279 18 90	20 14 13 224 18 89
MID. ATL.												
New York New Jersey Pennsylvania	2.4 0 8	6 0 6	10 8 8	13 8 6	0.4 0 0.5	1 0 1	0 0 1	1 1 1	255 233 205	638 196 404	905 125 608	948 182 720
E. NO. CEN. Ohio Indiana Michigan ³ Wisconsin	2.3 0.7 0.7 0	8 0 1 0 0	0 2 5 2 2	9 1 9 2 3	0 1.5 0.7 0	0 1 1 0 0	0 1 1 1 0	0 0 1 1 0	497 303 338 496 515	646 204 516 469 293	293 198 699 620 170	491 281 707 620 333

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended March 4, 1939, rates per 100,000 population (annual basis), and comparison with corresponding week of 1938 and 5-year median—Continued

-													
	м	Meningitis, meningo- coccus				Polior	nyeliti	5		Scarlet fever			
Division and State	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, m o - dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, me- dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, me- dian	
W. NO. CEN.													
Minnesota Iowa Missouri North Dakota South Dakota Nebraska Kansas So. ATL.	0 2 0 4 2 8	0 0 2 0 1 1	2 1 1 0 1 18 2	2 1 3 0 1 2 2	000000000000000000000000000000000000000	000000000000000000000000000000000000000	1 0 0 0 0 0 0 0	0 0 0 0 1 0	218 258 161 110 173 157 430	5 111 5 12 12 12 14 24 15	1 123 5 255 5 233 5 24 67 4 217	5 149 7 106 8 219 5 50 4 24 7 66 7 217	
Delaware Maryland ² Dist. of Col Virginia West Virginia North Carolina South Carolina ³ Georgia ³ Florida	0 16 0 29 27 0	0 0 2 0 0 2 0 0 2 1 0 0	042 52 51 10	0428251112	0 0 0 5 0 0 0 0 0	000000000000000000000000000000000000000	0 0 1 1 1 0 0	0 0 1 0 1 0 1 0	0 145 162 75 108 47 14 22 54	0 47 20 40 40 32 5 13 18	10 73 40 82 58 4 7 8	16 91 25 46 81 44 5 7 4	
E. SO. CEN.											1		
Kentucky Tennessee Alabama ³ Mississippi ³ ³	5 1.8 1.8 0	8 1 1 0	6 6 8 0	6 6 2 0	0 0 1.8 0	0 0 1 0	0 0 1 1	0 0 1 0	118 93 37 18	68 53 21 7	89 37 11 9	58 28 11 12	
W. SO. CEN.													
Arkansas. Louisiana ³ Oklahoma. Texas ³	000	0 0 4	0 0 1	1 1 3 5	0 2.4 0 0	0 1 0 0	0 0 1 2	0 0 1	22 15 91 74	9 6 45 89	9 11 31 127	9 12 81 113	
MOUNTAIN													
Montana. Idaho	0 0 5 0 25 0	0 0 1 0 2 0	0 0 0 0 0 1	1 0 0 1 0 1	0 10 0 12 0 0	0 1 0 1 0 0	0 1 0 0 1 0	000000	253 184 44 116 334 123 417	27 18 24 27 10 42	81 84 37 46 30 6 54	81 16 37 73 26 11 54	
PACIFIC							- 1						
Washington Oregon California	0 0 4	0 0 5	2 0 5	1 0 5	0 0 1.6	0	0 0 2	0 0 8	194 234 234	63 47 285	51 68 212	65 39 250	
Total	1.8	44	103	154	0. 5	12	18	21	215	5, 39 8	6, 224	7, 158	
9 weeks	21	481	858	987	0.6	145	192	192	213	48, 148	54, 300	57, 724	

See footnotes at end of table.

Cases of	' certain dise ase	s reported by tele	egraph by Stat	e health c	officers for	the week
ended	March 4, 1939,	rates per 100,000	0 population (a nn ual bas	sis), and co	mparison
with c	orresponding w	eek of 1938 and	5-year median	-Continu	ued	•

·····	Smallpox				Typl	hoid and fe	l paraty ver	Whooping cough			
Division and State	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 88, me- dian	Mar. 4, 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1938, cases	1934- 38, me- dian	Mar. 4. 1939, rate	Mar. 4, 1939, cases	Mar. 5, 1933, cases
NEW ENG.											
Maine. New Hampshire Vermont Massachusetts Rhode Island Connecticut	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	6 0 0 0 0	1 0 0 0 0	1 0 1 0 0	1 0 1 1 0 1	386 0 469 269 733 229	64 0 35 229 96 77	89 16 42 117 29 46
MID. ATL. New York New Jersey Pennsylvania	000000000000000000000000000000000000000	000000000000000000000000000000000000000	0 0 0	0 0 0	2 2 2	5 2 4	2 2 3	6 2 3	197 688 143	491 578 282	472 177 285
E. NO. CEN. Ohio Indiana Illinois Michigan ³ Wisconsin	17 128 10 14 9	22 86 15 13 5	23 50 81 16 4	1 8 6 1 15	2 8 1 1 0	2 2 1 1 0	2 1 4 6 1	2 8 4 0	122 21 176 218 480	159 14 269 206 273	116 22 121 177 141
W. NO. CEN. Minnesota Iowa Missouri North Dakota Bouth Dakota Nebraska Kansas	23 75 82 83 53	12 37 6 3 11 14	28 24 46 9 15 9	8 18 17 8 3 9 28	0 0 1 0 0 0	0 0 1 0 0 0	1 0 4 0 0 0	0 1 2 0 0 0	68 38 62 95 45 11 61	35 19 48 13 6 3 22	30 35 39 24 17 19 155
SO. ATL.		-		~						_	
Delaware Maryland ³ Dist, of Col Virginia West Virginia. North Carolina ³ Georgia ³ Florida	0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0 0	0 0 0 0 1 0 7	0 0 0 1 0 1 0	0 8 15 16 4 3 7 18	0 0 1 8 6 3 1 4 6	0 2 0 1 4 0 5 2	0 2 3 0 1 8 2	39 71 137 126 65 308 290 20 93	2 23 17 67 24 211 106 12 31	6 86 9 76 70 417 96 11 0
E. 80. CEN.											
Kentucky Tennessee Alabama ³ Mississippi ^{3 3}	7 12 0 0	4 7 0	80 10 0 1	0000	9 0 2 8	5 0 1 3	2 0 0 1	2 1 2 1	17 72 90	10 41 51	134 64 24
W. SO. CEN. Arkansas. Louisiana ³ Oklahoma. Teras ³	2 0 111 21	1 0 55 25	6 0 80 29	1 1 8 7	2 131 2 17	1 54 1 20	1 21 3 7	1 7 8 7	42 2 12 80	17 1 6 96	52 12 41 304
MOUNTAIN							4				
Montana Idaho Wyoming Oolorado New Mexico Arizona Utah "	28 163 0 0 74 0	3 16 0 0 6 0	23 9 10 0 8	84 1 8 1 0 0	0 10 22 10 49 0	0 1 2 4 0 0	0 0 1 2 0 0	0 0 2 4 0 0	66 0 22 169 556 233 507	7 0 1 35 45 19 51	28 9 19 8 36 40 28
PACIFIC											
Washington Oregon Oalifornia	12 45 31	4 9 38	64 16 12	11 1 1	6 0 2	2 0 3	1 0 0	1 0 3	99 65 133	32 13 162	124 7 403
Total	16	396	534	233	6	146	81	100	162	3, 999	4, 273
9 weeks	16	3, 597	5, 184	1, 851	5	1, 037	1,067	1, 067	171	38, 184	36, 089

New York City only.
Period ended eas lier than Saturday.
Typhus fever, week ended Mar. 4, 1939, 23 cases as follows: Bouth Carolina, 2; Georgia, 4; Alabama, 3; Mississippi, 1; Louisiana, 1; Teras, 12.
The discrepancy of 2 cases of diphtheria in the totals for 6 weeks and 7 weeks (Public Health Reports of Feb. 24 and Mar. 3, pp. 314 and 362) was a correction from 11 to 9 cases in the report for Florida for the week ended Jan. 21, 1939 (Public Health Reports Feb. 3, 1939, p. 193).

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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	Menin- gitis, menin- gococ- cus	Diph- theria	Influ- enza	Ma- laria	Mea- slos	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid and paraty- phoid fever
January 1939 Arizona California New Hampshire North Dakota Ohio Utah Virginia Wisconsin February 1939	2 11 1 0 1 8 1 9 1	41 142 14 10 149 0 96 7	549 199 135 54 107 19 1, 615 231	1 8 1 	11 7, 826 11 1, 103 101 109 333 2, 185	4 8 1 	0 4 1 0 2 0 2 1	16 968 1 2,126 112 162 1,154	85 77 0 22 142 0 42 42	8 19 2 2 19 1 1 15 1
Arkansas. Connecticut Delaware Texas	8 1 0 7	40 6 5 169	541 86 3, 030	74 	391 2, 159 4 572	20 76	2 0 0 1	56 412 31 405	11 0 0 148	11 1 0 44

January 1939-Continued

January 1999

Actinomycosis:	Cases
Hawaii Territory	1
Botulism:	-
California	
Chickennor:	•
A minopola.	100
California	8 900
	0,020
Hawall Territory	103
North Dakota	104
<u>Unio</u>	8,033
Utan	632
Virginia	459
Wisconsin	8, 140
Conjunctivitis:	
Hawaii Territory	3
Diarrhea.	
Ohio (under 2 vrs en-	
toritic included)	15
Dennis manuel)	10
Dysentery:	
Arizona (Dacillary)	32
California (amoebic)	10
Oalifornia (bacillary)	22
Hawaii Territory (amoe-	
bic)	5
Virginia (amoebic)	1
Virginia (bacillary)	85
Wisconsin (amoebic)	1
Encephalitis, epidemic or	-
lethargic:	
Arizona	1
California	- 1
Ohio	5
litah	1
Wiegonein	- 1
W ISCONSIN	1
rood poisoning:	
California	158
German measles:	
Arizona	7
California	126
North Dakota	- 4
Ohio	21
Utah	10
Granuloma, coccidioidal.	
California	11
Hookworm disease:	
Hawaii Territory	17
Impetigo contagiosa	
Hawaii Territory	14
Isundice infections.	12
California	

Lead poisoning: Ohio_____ Cases 8 1 16 Leprosy: 5 Bawaii Territory_____ 7 Obio_____ i 8 Mumps: Arizona 16 California 8,412 Hawaii Territory 129 North Dakota 32 Ohio 1858 Utab 619 16 Arizona_ 3 Utah_____ Virginia_____ Wisconsin_____ 619 313 £63 Ophthalmia neonatorum: California 2 2 Ohi Virginia 71 8 Puerperal septicemia: Ohio - 4 Rabies in animals: Virginia_____ Wisconsin_____ 143 127 14 Tetanus: California Bawaii Territory Virginia 2 2 ī Trachoma: Arizona California Hawaii Territory..... 43 -----23 8 Trichinosis: Arizona California Ohio 8 Š ĩ Tularaemia: California..... 1 Ohio_____ Virginia_____ Wisconsin_____ 20 17 1 Typhus fever: California Hawaii Territory 2

January 1959-Continued

I

Undulant fever:	Cases
California	9
Ohio	10
Virginia	1
Wisconsin,	3
North Dakota	
Whooping cough:	
Arizona.	36
California	424
Hawall Territory	4/
Ohio	018
Utah	56
Virginia	220
Wisconsin	1, 321
Fabra and 1099	
Anthrax:	
Delaware	3
Chickenpox:	
Arkansas	249
Connecticut	438
Tayog	1 635
Conjunctivitis, infectious;	4,000
Connecticut	7
Dengue:	-
Texas	
Arkanges (emoshic)	
Arkansas (bacillary)	Ŧ
Connecticut (bacillary)	ž
Texas (bacillary)	30
Encephalitis, epidemic or	
Hethargio:	4
Hookworm disease:	
Arkansas	1
Leprosy:	-
Teras.	2
Mumps:	ar
Connectiont	204
Delawara	123
Texas.	162
Ophthalmia neonatorum:	
Arkansas	2
Terrerol continentia	
Arkanses	

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February 19 39 Continue	d	February 1939-Continu	February 1939-Continued			
Rables in animals: Arkansas Connecticut Delaware Teras Relapsing fever: Teras Septic sore throat: Arkansas Connecticut	Cases 31 4 1 12 10 47 19	Trachoma: Arkansas Connecticut Ternas Trichinosis: Connecticut Tularaemia: Arkansas Taras Typhus fever: Taras	Cases 4 1 30 2 2 8 19	Undulant fever: Arkansas Connecticut Teras Whooping cough: Arkansas Connecticut Delaware Teras	Cases 4 6 12 62 320 18 401	

WEEKLY REPORTS FROM CITIES

City reports for week ended February 25, 1939

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

	Diph-	Influenza		Mea-	Pneu-	Scar-	- Small-	Tuber-	Ty- phoid	Whoop-	Deaths,
State and city	cases	Cases	Deaths	SJ65 Cases	deaths	fever cases	pox cases	deaths	fever cases	cough cases	all causes
Data for 90 cities: 5-year average Current week	188 156	839 1, 339	139 159	6, 938 4, 417	994 943	2, 303 1, 546	24 82	412 371	18 47	1, 247 1, 264	
Maine: Portland	0		0	0	2	2	0	0	0	4	28
New Hampshire: Concord Manchester Nashua	000		0 0	0 0 0	8 2 0	0 8 0	0	1 0 0	000	0	10 15 4
Vermont: Barre Burlington	0		0	0	1	0	0	0	0	1	39
Massachusetts: Boston Fall River	0		0 2 0	0 167 1	80 6	57 3	0	9 1	0	21 2	2 252 33
Springfield Worcester Rhode Island:	Ŏ		Ŏ	21 4	45	2 18	Ŏ	8 1	Ŏ	4 53	49 56
Pawtucket Providence Connecticut:	00	4	00	2 9 0	01	8 11	0	02	000	5 80	17 46
Hartford New Haven	02	<u>-</u> 4	0 1	162 70	4	10 2	0	02	0	57	41 63
New York: Buffalo New York Rochester Syracuse	1 28 0 0	 101 4 	1 12 0 0	83 80 106 110	10 150 3 5	77 233 31 26	000000000000000000000000000000000000000	8 75 0 2	0200	23 151 16 38	123 1, 684 73 53
Camden Newark Trenton	4 1 0	1 4 1	2 1 0	0 5 0	2 10 6	3 46 5	0 0 0	0 5 7	0 0 0	0 93 5	39 126 44
Pennsylvania: Philadelphia Pittsburgh Reading Scranton	5 2 6 0	17 18 	10 8 0	29 8 2 3	46 19 2	76 48 1 30	0 0 0 0	24 10 0	2 0 0 9	106 16 0 9	592 212 36
Ohio: Cincinnati Cleveland Columbus Toledo	7 2 0 0	27 345 1 1	2 8 1 1	0 5 4 2	22 24 6 8	34 57 4 20	0 0 0 0	5 9 2 4	0 0 0 0	8 44 4 17	148 239 85 83
Indiana: Anderson Fort Wayne Indianapolis Muncie South Bend Terre Haute	0 1 1 0 0		0 0 5 0 2	0 1 2 0 1 1	0 2 29 5 4 2	6 10 38 4 4 1	0 0 67 1 0	1 0 2 0 1 0	0 0 0 0 0 0	1 0 9 0 6 0	14 26 132 17 22 27
Illinois: Alton Chicago Elgin Moline Springfield	0 15 0 0	851 56 2	0 34 0 0	0900	0 135 8 0 2	0 178 10 0 8	0 1 0 0	0 48 0 0	00000	0 128 1 4	5 1,092 16 8 28

City reports for	• week ended	February	25, 19 39 —	Continued
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State and city	Diph- theria cases	Inf Cases	Deaths	Mea- sles cases	Pneu- monia deaths	Scar- let fever cases	Small- pox cases	Tuber- culosis deaths	Ty- phoid fever cases	Whoop- ing cough cases	Deaths, all causes
Michigan: Detroit Flint Grand Rapids Wisconsin	16 0 0	82 82	15 1 2	9 156 2	67 8 8	122 18 16	000	19 1 0	1 0 0	96 0 2	398 81 47
Madison Madison Milwaukee Racine Superior	0 0 0 0	 57 	0 0 1 0 0	0 1 8 8 9	2 2 6 0 1	4 7 71 5 2	0 0 0 0	0 8 0 0	0 0 0 - 0	20 7 94 1 0	14 23 123 16 9
Minnesota: Duluth Minneapolis St. Paul Iowa:	0 2 1	20 	4	8 266 446	1 2 4	4 23 16	0 6 0	1 1 8	0 0 0	5 28 6	19 129 64
Cedar Rapids Davenport Des Moines Sioux City Waterloo	0 0 0 2	 		1 0 1 26 1	0 	0 6 23 1 13	0 2 1 0 1	 0 	0 0 0 0 0	8 0 2 1	84
Missouri: Kansas City St. Joseph St. Louis North Dakota: Fargo	0 0 2	1 14	6 0 1	100	26 3 12	13 8 27 7	0 0 1	5 0 8 0	0 5 0	1 3 89	137 13 208
Grand Forks Minot South Dakota: Aberdeen Sioux Falls	0 0 0 2		0 0	1 21 23	0 0	0 1 2	0 0 1 0	0 0	0 0 0	02000	4
Nebraska: Lincoln Omaha Kansas: Lawrence	000		0	10 5 0	4	0 8 1	000	2	000	8	50
Topeka Wichita Delaware:	Ŏ		Ŏ	Ŏ	4	24	Ŏ	Ŏ 1	ŏ	Ŏ	23
Maryland: Baltimore Cumberland Frederick	8 0 0	79 8	9 0 0	1, 010 0 0	44 0 0	27 0 0	000	9 0 0	0000	7 0 1	87 290 14 5
Virginia: Lynchburg	7	25	2	19 79	17 4	20 2	0	10 1	1	17 7	235 20
Richmond Roanoke West Virginia: Charleston	0 1 0	211 2	1 8 0	8 25 0	0 5 2	8	0	1 8 0	000	808	86 54 20
Huntington Wheeling North Carolina: Gastonia	1 0 1		0	0 2 1		0 1 0	Ŏ Ŏ O		Ŏ Ŏ O	Ŭ 4 0	23
Releigh Wilmington Winston-Salem. South Carolina:	0 0 1		0 0 1	1 0 214	4	800	0000	2 0 1	0 1 0	0 10 1	90 9 19
Greenville Georgia: Atlanta	1 0 0	 22	0 0 1	000	4	0 0 8	0	2 1 0 6		0 5 0	15 18 8
Brunswick Savannah Florida: Miami	0	17	0 0 1	50 0 1	2 2 2 2	Ŏ O ₽	Ŏ O Q	Ŏ Ŏ Q	Ŏ	0 8 9	6 85 56
Tampa Kentucky:	•	1	1	92	•	1	°	1	0	•	23
Ashland Covington Lexington Louisville		2 8 86	200	0020	10	0 12 5	8	0	0000	000	-17.01

State and city	Diph theri		fluenza	Mea- sles	Pneu- monia	Scar- let	Small- pox	Tuber	Ty- phoid	Whoop- ing	Deaths, all
	cases	³ Case	s Deaths	C8.565	deaths	Cases	Cases	deaths	cases	cases	causes
Tennessee: Knoxville Memphis Nashville		3	- 0 1 3	0 0 0	1 15 2	8 5 6	0	0 3 1	0000	0 9 1	31 79 42
Alabema: Birmingham Mobile Montgomery		6 	. 8	0 0 8	5 5	2 1 0	0 0 0	3	000	000	61 33
Arkansas: Fort Smith Little Rock		8		1 0	3	1 0	0		0	0	
Lonsmans: Lake Charles New Orleans Shreveport	1	7	0 2 0	63 49 5	2 17 10	0 7 6	0 0 0	0 9	0 31 0	0 10 0	15 156 52
Oklahoma City. Tulsa Texas:	0	14	0	2 2	6 	11 7	0	1	0	0	50
Dallas Fort Worth Galveston Houston San Antonio		14 14 1 3	1 0 1 1	10 1 0 16 6	11 6 2 15 7	5 7 0 5 5	2 3 0 0 0	1 1 0 4 11	0 0 1 0 0	3 0 1 0	60 27 13 99 80
Montana: Billings Great Falls Helena Missoula	2000		0	10 2 119 26	2 3 0 0	0 0 1 0	0 0 0 1	0 0 1 0	0 0 0	0 0 0 0	10 15 9 4
Idaho: Boise Colorado:	0		0	0	0	1	0	0	0	0	4
Springs Denver Pueblo	0 8 0		0 6 0	61 5 6	0 5 3	2 8 3	0 0 0	0 1 0	0 0 0	12 28 5	11 95 13
Albuquerque Utah: Salt Lake City.	0 0		0	0 2	1 1	0 14	0 0	0 1	0 0	5 2	12 38
Washington: Seattle Spokane Tacoma	0 0 0		2 0 0	53 62 0	9 4 0	7 3 2	0 0 0	2 0 0	0 1 0	6 0 0	109 37 29
Oregon: Portland Salem	1 0	1	1	1 2	5	6 1	8 0	1	0 0	2 0	99
Los Angeles Sacramento San Francisco	14 1 1	16 	1 0 3	213 135 412	21 8 10	47 0 16	2 2 0	17 8 13	1 0 0	18 1 3	369 33 212
State and city		Meningitis, meningococcus		Polio- mye-		State a	nd city		Mening	ngitis, ococcus	Polio- mye-
	[Cases	Deaths	cases				[Cases	Deaths	Cases
Massachusetts: Boston Rhode Island:		1	1	0	Mar j Dist	yland: Baltimo rict of C	re olumbi	a:	1	0	0
Pawtucket Providence New York:		1	10	0 Florida: Tampa					1 0	1 0	0 1
New York Pennsylvania: Philadelphia		2 2 1	2	0 0	Loui	aina: Birming siana: New Orl	ham		1 2	0	0 0
Pittsburgh Ohio: Cincinnati	•••••	ī 1	Ĭ O	Ŭ O	E Tera I	hrevep s: Dallas	ort		0 1	1	0 0
Illinois: Chicago		1	0	0	I I	Iouston			1	0	0

City reports for week ended February 25, 1939-Continued

Encephalitis, epidemic or lethargic.—Cases: New York, 1. Pellagra.—Cases: Wilmington, N. C., 1; Atlanta, 7; Savannah, 1; Dallas, 1; Los Angeles, 1. Typhus fever.—Cases: Charleston, S. C., 2; Houston, 1; San Antonio, 2.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—Week ended January 28, 1939.— During the week ended January 28, 1939, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Bruns- wick	Que- bec	Onta- rio	Mani- toba	Sas- katch- ewan	Alber- ta	British Colum- bia	Total
Cerebrospinal meningitis. Chickenpox Diphtheria Dysentery		5	25 8	206 29	1 384 2	25 3	83 8	10		1 764 45 2
Influenza Measles Mumps		8		55 116	164 933 24	27 18	5	1 8	26 2 1	190 1,028 167
Pneumonia Scarlet fever Smallpox		8 4 	14	104	29 195 	 83 1	1 81 1	 81 	10 8 1	43 420 1
Tuberculosis Typhoid and paraty- phoid fever		1	8	22 5	50	24 1	4	1	13	123 7
Whooping cough				286	343	16	5	1	73	724

FINLAND

Communicable diseases—January 1939.—During the month of January 1939, cases of certain communicable diseases were reported in Finland as follows:

Disease	Cases	Disease	Cases
Diphtheria Influenza Paratyphoid fever Poliomyelitis	304 8, 514 12 18	Scarlet fever Typhoid fever Undulant fever	741 6 2

LATVIA

Notifiable diseases—October-December 1938.—During the months of October, November, and December 1938, cases of certain notifiable diseases were reported in Latvia as follows:

Disease	Octo- ber	Novem- ber	Decem- ber	Disease	Octo- ber	Novem- ber	Decem- ber
Botulism Cerebrospinal meningitis. Diphtheria Erysipelas. Influenza Lead poisoning. Lethargic encephalitis. Measies. Mumps. Paratyphoid fever	11 171 47 55 1 17 52 12	1 6 244 36 54 3 1 16 145 10	1 4 240 40 49 6 76 347 6	Pollomyelitis Puerperal septicemia Scarlet fever Tretanus Trachoma Tuberculosis. Typhold fever Typhus fever Whooping cough	154 11 333 3 64 214 54 79	64 3 455 2 48 240 53 1 51	26 5 471 2 38 212 82

YUGOSLAVIA

Communicable diseases—4 weeks ended January 29, 1939.—During the 4 weeks ended January 29, 1939, certain communicable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax. Cerebrospinai meningitis. Diphtheria and croup. Dysentery. Erysipelas. Favus. Lethargic encephalitis.	23 48 585 8 159 9 3	4 10 43 5 2	Paratyphoid fever Poliomyelitis Scarlet føver Sepsis. Tetanus Typhoid fever Typhus fever	5 2 258 11 11 316 22	1 12 1 4 22 2

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 February 24, 1939, pages 322-333. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Plague

Egypt—Asynt Province.—During the week ended February 25, 1939, 26 cases of plague were reported in Asynt Province, Egypt.

Peru.—During the month of January 1939, plague was reported in Peru as follows: Lambayeque Department, 2 cases, 1 death; Libertad Department, 11 cases, 6 deaths; Lima Department, 1 case.

Yellow Fever

Brazil—Espirito Santo State.—Yellow fever has been reported in Espirito Santo State, Brazil, as follows: Alegre, January 27, 1939, 1 death; Sabino Pessoa, January 27, 1939, 1 death; Sao Felipe, January 25, 1939, 1 death.

Niger Territory-Say.-On February 23, 1939, 1 suspected case of yellow fever was reported in Say, Niger Territory.