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PROVISIONAL MORTALITY RATES FOR THE FIRST SIX MONTHS OF 1938

The mortality rates in this report are based upon preliminary data for 42 States, the District of Columbia, Alaska, and Hawaii for the first 6 months of 1938. Comparative data for 39 States (District of Columbia included as a State) are presented for the first 6 months and by the 2 quarters of 1938 and 1937.

This report is made possible through a cooperative arrangement with the respective States, which voluntarily furnish provisional quarterly and annual tabulations of current birth and death records. These reports are compiled and published by the United States Public Health Service.

Because of lack of uniformity in the method of classifying deaths according to cause, and because a certain number of certificates were not filed in time to be included, these data may differ in some instances from the final figures subsequently published by the Bureau of the Census.

In the past, these preliminary reports have provided an early and accurate index of the trend in mortality for the country as a whole. Some deviation from the final figures for individual States is to be expected, because of the provisional nature of the information. It is believed, however, that the trend of mortality within each State is correctly represented. Comparisons of specific causes of death among different States are subject to error because of differences in tabulation procedure and completeness of reporting. Comparisons of this nature should be made only from the final figures published by the Bureau of the Census.

Unless there is a marked reversal of trend, the mortality rate from all causes of death during the current year will be the lowest on record with the possible exception of 1933, when the death rate was 10.7 per 1,000 population. The rate for the first 6 months of 1938, 10.8 per 1,000 population, is only slightly higher than the low rate for 1933 and represents a decrease of 8.5 percent from the rate for 1937. Every State for which data are available reported a lower rate than for 1937.

Although this decrease in the mortality rate is reflected in nearly all the important causes of death, about 60 percent is accounted for by the decreased prevalence of influenza and pneumonia, especially during the first quarter of the year. The death rate from influenza for the first half of 1938 is only one-third of the rate for 1937, and that

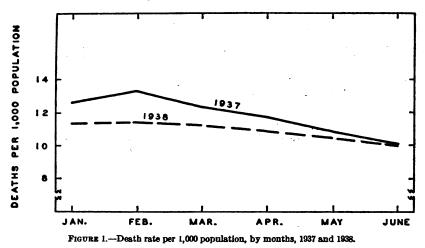
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from pneumonia is only three-fourths the rate for the corresponding period last year.

The downward trend of the death rate from tuberculosis continues unchecked and the current rate is 10 percent less than the corresponding rate for 1937. It is possible that the rate for 1938 will drop below 50 per 100,000 for the first time.

The decline in maternal mortality has been even greater than that from tuberculosis; the current rate, 4.4 per 1,000 live births, is 15 percent less than the corresponding rate for 1937.

The widespread efforts to prevent traffic accidents are apparently achieving success, since the mortality rate from automobile accidents for the half year is 20 percent less than the rate for the similar period of 1937. This decrease is widespread, only 4 of the States reporting a higher rate than for last year.



The only important cause of death for which the current rate is greater than that for 1937 is cancer, for which an increase of about 3 percent is reported.

The serious outbreak of measles which occurred last winter continued into the second quarter of this year. As a result, the death rate, 4.5 per 100,000 population, is more than 4 times the corresponding rate for 1937. Slight increases also occurred in the mortality rates for whooping cough and for diarrhea and enteritis under 2 years of age.

Another outstanding feature of the mortality record for the first 6 months of 1938 was the widespread decline in the infant mortality rate. Only 5 States reported a higher rate than for 1937, and the current rate is nearly 9 percent less than that for last year.

The birth rate for 1938 has continued slightly above that for 1937. This increase, combined with a lower death rate, has resulted in a crude rate of natural increase of 6.0 per 1,000 population, compared with the corresponding rate of 4.3 per 1,000 population for 1937. **Provisional m**ortality rates from certain causes in the first 6 months of 1938, with comparative provisional data for the corresponding period in preceding years

¹ These data are taken from the Monthly State program of a set of the first set up, 0.0, 0.00.
¹ These data are taken from the Monthly State program of the first set of the first

Provisional mortality rates from certain causes in the first 6 months of 1938, with comparative provisional data for the corresponding period in preceding years—Continued

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1	Automobile accidents (206, 208, 210) 1		818 20.3	EE	88. 3 4.8	27.9	16.8 21.8	×8	10.1 26.7	87 77
	All accidents (176-195, 201-214) 1		20 . 2	227.3	80.9 111.0	80. 7 87. 4	67.3 67.3	73. 1 106. 6	67.0 81.0	90.3 107.5
	Nephritis (130–132)		78.9 79.4 82.4	16.0 23.5	88.7 91.8	87.8 86.0	87.1 87.6 96.5	120.1	112 1 98.7 102.7	101.0
	Districts and enteritis, under 2 years (119)		19.6 13.1 13.2	6 1 1	1.104 4.04	6.4 8.0	0000 0000	6.2	7.8 5.8 8.8	15.6 13.4 8
	Diseases of the diges- tive system (115-129)		69.1 57.0 59.7	51.2 63.8	75.4 83.8 77.3	74. 1 81. 2	8.9 9.9 9.0	57. 7 66. 4	1.2 2.2 2.2	95.0 94.5 83.5
s)	Pneumonis, all forms (107–109)		91.5 117.0 139.2	284.9 198.3	89.8 160.4 105.3	117.0 244.8	838	97.8 129.8	108.9 166.3 177.4	79.9 81.1 105.3
Death rate per 100,000 population (annual basis)	Diseases of the heart (30-95)		167.7 164.9 162.2	262.9 369.7	389.0 478.3 378.0	246.3 242.0	261.9 246.1 247.2	384. 1 385. 5	353. 4 346. 4 370. 7	273.3 253.6 257.6
1 (annu	Cerebral hemorrhage, boplexy (82s, b)		71.2 68.5 69.3	182.4 60.5	101.6 8.6 8.8	91.0 92.8	91.6 86.0	110.8	87.6 104.2 111.1	106.1 108.1 108.9
ulatior	Diabetes (59)		11.9 11.0 12.6	9	8.88 8.99 8.99	18.8	31.9 34.8 32.0	31.6	32.5	28.2
dod D0	Cancer, all forms (45- 53)		54.8 57.1 56.1	96.0 67.2	144 0 144 0 141 3	119.1	137.0 126.6 128.7	127.0 108.9	137.2 144.4 132.5	95.0 96.7 87.9
er 100,0	Tuberculosis, all forms (23-32)		57.2 65.3 68.2	582.6 379.8	71.7 91.3 83.4	67.2 73.2	37.9 38.8 40.6	43.9	74.9 102.0 114.4	55.59 54.9
rate p	Epidemic cerebrospinal meningitis (18)		3.1 5.2 1.1	E.a	8.9 8 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9		1.32	s. 1 8.1	2.5 14.6	1.0 8.0 8.0 1.1
Death	Encephalitis, epidemic or lethargic (17)	-	0.8 .3 .1	EE_	4.00	1.3	808	E E	1.0	0.4 8.4 7 2
	Acute poliomyelitis and polioencephalitis (16)		0.4.	EE	<u>6</u> , – 6	1.9	<u>ເຄີ</u>	EE	<u>ເຄ</u>	4.0.
	(11) szasuftal		8578 8578 8578 8578 8578 8578 8578 8578	67.2 53.8	7.6 69.3 27.3	17.6 86.0	20.0 13.4	16.2 36.3	6.4 13.0 13.0	29.6 54.2 79.4
	Diphtheria (10)		5055 143	ΞE	2.1.9		0.0.M	1.5 .8	1.0 3.2 6.5	257 78 78 76 76 76 76 76 76 76 76 76 76 76 76 76
	Whooping cough (9)		0000 1700	96. 0 37. 0	9.45 1.45 1.04 1.04 1.04 1.04 1.04 1.04 1.04 1.04	3.0 6.2	2.1. 2.9	8.5	6.5.6	4.0.1.
	Scarlet fever (8)		0.7 .3 .5	3.2	2.1.0	1.3	1.2	°.	1.0	0.1
	(7) 29 [209 M		1.2	97 99	1.9.4	5.1		1.5 2.3	0.00 CP 17 CP	04.4.
	Typhoid fever (1, 2)		1.22	3.2	1.8.72	1.3	8.e.	.8	1.0	8.8.8 444
per 1,000 births	Vilatiom lanteski		තු හ තු හ තු තු තු	59 19	54 kg	4 5 8 8	ରାର୍ଦ୍ଦ ଜାର୍ଦ୍ଦ	00 00	ස හ හ ත් ත් ත් ත්	7.7.0
Rate per 1,000 live births	Total infant mortality		325	E.	74 88 21	22 73	8 24 74	67	285 29	322
[BUUUR)	Births (exclusive of still per 1,000 population (pasis)		21.2 20.8 21.3	31.6 22.7	15.6 14.3 13.0	18. 7 17. 9	13.4 12.6 12.5	15.7 15.9	19.7 19.0 18.4	16.1 15.9 15.1
	All causes, rate per 1,000 tion (annual basis		10.7 11.4 11.6	24.0 18.8	13.1 16.3 13.6	11.9 14.3	10.5 10.9	12.6 14.4	13.1 15.2 15.8	12.9 13.1 13.3
	state and period	JANUARY-JUNE	Alabama: 1988 1987	8 - 3	Cautorna: * 1938- 1937- 1936-	Colorado: 1938 1937	Connecticut: 1838	Delaware: 1938 1837	District of Columbia: 1837.	riorida: 1938 1937

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109. 9 101. 1 103. 5	60.8 20.8 60.5	85.8 85.8 86.98	97.8 108.7 100.2	20 20 20 20 20 20 20 20 20 20 20 20 20 2	8.278 8.248	00.3 01.2 01.2 01.2 01.2 0	88 48	106.1	85.1 95.9	137. 4 152. 3 155. 4	58.4 20.24 20.24
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101.4 107.8 151.4	62.1 73.6	92.0 93.9	69.3 69.3 92.8 10).1	79.1	73.4 84.1 91.3	88.5 86.1 119.0	80.4 116.1	100.6 129.1 150.9	95. 2 128. 7	102.5 144.0 143.5	69.9 112.7 115.2
165.9 167.4 173.5	111.8 115.2 127.9	180.7 174.2 198.6	322.0 309.3 331.2	242.5 264.3 281.6	249.7 225.8 251.5	238.5 251.7 250.3	178.0 176.8	216.0 213.4 215.3	335.4 395.1	340. 2 332. 1 326. 3	297.3 291.2 297.0
85.8 81.7 30.1	51. 5 89. 6 41. 6	73.0	75.4 75.7 84.4	122.5	104.8 105.9	96.1 103.6 113.4	95. 5 86. 8	68 1 70.4 8	113.3	101.4 117.6 119.2	90.8 91.1 98.7
13.4	13.33	10.1	8888 8	17.0 18.1	104	***	12.3	17.5 18.1 18.1	9 % 8 8	20. 0 27.1 29.8	27.7
5.214	47.09 8.28 8.09	181	132.9 128.4 131.3	111.4 104.8 108.7	127.5 119.1 125.5	116.2 114.8 112.3	8 .8 1.8	200	142.4 145.6	134. 1 131. 6 131. 6	116.7
52.8 40.9 55.4	61.2 78.2 76.4	22.5 25.5 25.5	47.9 53.6 54.0	40.2 49.2	200 200 200 200	33 0 0 2 33 0 0 2 35 0	69.8 67.8	70.8 74.0	20.6 34.6	81.5 88.5 82.7	0444 1-00
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10.7 10.9 11.8	8003	10.9	11.9	012121 004	9.8 10.1 10.5	10.2 11.1 12.4	9.2 10.6	11.4 12.3 12.7	12.3 14.2	12.7 14.0 13.9	10.6 11.8 11.8
											10.6 1937 11.8 11.8 11.8 11.8
Georgia: 1938 1936 Hawaii:	1938 1937 1936 1036	1988- 1986- 1986- Difnols:	1988 1987 1986 1986 Indiana:	1938 1937 1936 Iowa:	1938 1937 1936 Kansas :	1938 1937 1936 Kentucky:	1938 1937 Louisiana:	1938 1937 Maine:	1938 1937 Maryland:	1938 1937 1936 Michigan:	1938 1937 1936

¹ Data not available. ¹ No deaths reported. ¹ January to March.

Provisional mortality rates from certain causes in the first 6 months of 1938, with comparative provisional data for the corresponding period in preceding years—Continued

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	Automobile accidents (206, 203, 210) 1		19.4	22	88	25	8. 3.	23 X	31.	23
	All accidents (176-195, 201-214) 1		62.4 65.9	67.6 80.3	101. 4 91. 3	48. 2 52. 1	102.8 135.8	54. 1 74. 0	86.5	60.4 68.0
	Nephritis (130-133)		44 49 49 49 49 49 49 49	107.5	36.6 76.3 76.5	62 70.0 0 0	37.6 45.9	76.0 74.0 82.4	70.2	78.2 82.8 84.9
	Diarrhea and enteritis, under 2 years (119)		1919	86 40	8-19 6-19	004 004	£0	890 80 10 10 10 10 10 10 10 10 10 10 10 10 10	22.0	50 53 50 52 50 52
	Diseases of the diges- tive system (115-129)		52.1 55.3 60.7	57.4 58.3	66.6 67.7 81.8	40.64 84.04 84.04	57.3 37.9	57.1 57.4 56.0	84.1	63.3 69.5 99.5
-	Preumonia, all forms (107-109)		76.9 98.2 100.7	114.5	98.0 142.9 149.2	72.6 94.8 00.0	128.5	72.8 92.1 88.7	113.2	81.6 119.9 117.9
Death rate per 100,000 population (annual basis)	Diseases of the heart (30-95)		250.4 241.4 250.9	276. 7 282. 5	222. 7 227. 1 198. 4	230.9 230.9 251.7	276. 7 257. 6	335.7 331.7 331.7 315.7	146.7	382.4 389.7 375.9
(annu	Cerebral hemorrhage, spoplery (82a, b)		58.2 91.6 84.7	91.2 99.8	80.2 96.9 91.3	867 86 80 80 80 80 80 80 80 80 80 80 80 80 80	83.0 83.8	81.4 79.6 85.0	51.1	68.3 79.6 85.0
ulation	Disbetes (59)		26.1 27.3	24.0 25.1	2802	8000 888	15.8 10.0	30.5 32.8 32.8 32.8	7.2	38.1 38.1 38.1
idod ()	Cancer, all forms (45- 53)		140.0 141.2 129.0	125.3	108.5 108.5	124.0 108.6 110.7	100.8	128.0 122.2 122.5	64. 5	155.9 150.6 146.2
r 100,00	Tuberculosis, all forms (23-32)		30.4 37.7	51.8 60.6	50.0 45.0 47.0	17.9 24.1 19.9	67. 2 71. 9	46.7 50.5 51.6	92. 7	53.9 62.2 61.5
rate pe	Epidemic cerebrospinal meningitis (18)		0110 804	1.8	10190 1019	2.1	εe		1.0	3.050
Death	Encephalitis, epidemic or lethargic (17)		400	æ.æ.	÷€	-41	εe	00 1- 00	ε	0.178
	Acute poliomyelitis and polioencephalitis (16)			64	999	61 C 4	EE		1.0	ÊÊE
	(II) sznsufini		10.5 18.1 18.1	88 88	20.22 20.29 4 20.09	80.0 80.0 80.0	2.0 16.0	6.4 16.8 11.3	20.5	17.1 9.5
	Diptheria (10)		0.40	10 4 0	3058 3078	400	6.8 0	010	3.8	404
	(9) dguos gaiqood W		1.22	803	4.1.4	0 2 2 0 i - i 3 3	4 5	1.4.1	21.6	1.11
	Scarlet fever (8)		1.01.1	4.1	1 40	9.2 9.2 9.2	£.	1.064	1.45	2.3
	(L) sieses M		1. 0	.12	1.5	1.50	EE	404	22.4	1.08
	Typhoid fever (1, 2)		2.9.9	41	1.10.7	10.01	36	2015	2.42	
1,000 ths	Maternal mortality		88.44 10.41	00 00	80 4 50 00 70 00	4054 954	10.3	11-10 14 16 16	2	0 - 10 0 + 10
Rate per 1,000 live births	Total infant mortality		40 4 5 5 4 0	88	4 1 57 51	4884	48	444	88	443
IBUDIA	Births (exclusive of still per 1,000 population (basis)		17.6 18.4 16.9	15.4	19.2 19.2	15.7 15.7 16.3	17.0 13.6	12.23	34. 2	14.4 14.5 14.0
-Blugoc ((2d11id	Births (exclusive of still) Births (exclusive of still)		9.8 10.7 10.8	11.6	10.7 12.6 12.1	9.2 10.9	11.9	10.4 10.8	13.3	12.0 12.8 12.7
	Btate and period	JANUART-JUNE-continued	Minnesota: 1988 1887	1938. 1938.	maturatias. 1887 1887	NeDraska: ¹⁰ 1838	Nevsda: 1938 1937	New Jersey: 1837. 1936.	New Mexico: 1936 New V.C.	1938. 1937. 1936.

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North Carolina:	-	-	-	-	-	-	-	-	-	-	-	-											
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North Dakota:	7.1	1	8		0				0	~	•	i	8	23	13	ε	ε	3	E	6			
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1938	12.7 13.9 12.8	15.2 15.0 15.0	\$ 82	0000 0000	ΞĒ	EE.		100	*** ***	333			*80 \$25	5 157. 0 162. 3 135.	4 48	801	8.5 F	1 29. 146.	885	0000 0000		2,4 80	11
1938. 1937. 1936. Bouth Dakota.	10.6 10.8	19.0 18.4 18.7	92 85	040 040	50 4 30 1 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2	12.9	<u>64.0</u> 50.000	406	384 284	600		10000	\$445	844 844	119	288	178.1	4 100.2 5 109.4 8 137.2	8 2 0 8 8 2 0 8	ర్ష ల శ	8 888	88 01-	241
1938 1937 1936 Tennessee	9.7	ର ଅନ୍ତ୍ର ଅନ୍ତ୍ର	51 49	8008 1909	4.04		1. 4 16. 4. 7 1.	000	<u>666</u>	333	£	3 .	288	8.2.8	522	833 8	4 128	0 65.8 5 72.1 87.8	3.4.8 4.60	2000 1000	8780 84180	50.4 51.6	11.9 13.1
1938 1936 Trah	9.6 10.6	15.9 15.5 15.9	86788	6 8 7 8 8 9	1.81.6	1 1 1 1	1.00 1.00 1.00 1.00 1.00 1.00 1.00 1.00	666 000 000	8.88 8.783			00 4	4 9 9 1 9	080	0.1.6	522 5379	4 159.5	9 92.9 2 120.2 159.3	8888 8988 8778	17.7 10.3 7.5	8.50 8.50 8.40	7 8	16.3 21.4
1938- 1937- Vermont:	9.3 10.1	88 88	43	40 40	£.	3.9	2.0 3.0 3.			. 3		54 55	នន	33.	ูลีส์ 	5 51. 62. 7	251.4	4 73.3	60. I 71. 5			88.8 76.5	4 -
1938 1937. Virginia:	11.0 12.0	14. 7 13. 1	262	3.5	e°:	6.3 ()	(). (). (). (). (). (). (). (). (). ().	1.0	<u>≋.</u> 83 82,53	3 [.]	<u>.</u>	<u>ء</u>	- 1	8 122.5 9 135.5	ର୍ଷ ର୍ଷ ® ଉ	07 IS	3 306.7	7 103.4	88 88	3.1	83.6 74.8	50.5 50.5	14.6 15.8
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¹ Data not compiled for these causes prior to 1937. Data not available. • No deaths reported. ¹⁰ January to May. ¹¹ Less than 0.1 of 1 per 100,000 population.

Provisional mortality rates from certain causes in the first 6 months of 1938, with comparative provisional data for the corresponding period in preceding years—Continued

14, 1938			1824			
	Automobile seccidents (206, 208, 210) 1		23.8 26.6 31.7	20. I 20. I	16.9 24.2	80.3 8.6
	All accidents (176-195,		81. 1 87. 3 91. 4	88	62. 3 75. 0	102.1 105.5
	Nephritis (130–132)		71.6 79.8 80.8	71.6	67.3 74.8 73.1	80.2 35.2
	Diarrhea and enteritis, under 2 years (119)		11.0	6.7.4 8.9	8.4.4 0.00	7.6
	Diseases of the diges- tive system (115-129)		58.2 54.7 66.0	40.8 54.9 52.7	EEE	71.5
(s)	Pneumonia, all forms (107-109)		81.6 88.5 90.6	8.27 8.27 8.27 8.28 8.27 8.28	8.99 8.99 8.88 8.88	87.6 149.3
Desth rate per 100,000 population (annual basis)	Diseases of the heart (30-95)		286.5 304.8 291.3	166.0 174.3 186.6	282.0 282.0 282.0	8 210.2 1 270.3
annu	Сегергаl пешогглаge, b) Сегергаl пешогглаge,		112.9 107.9 110.2	55.53 80.4	228	82
ulatior	Disbetes: (59)		28:25 28:25 28:25	16.3 15.2 15.2	***	12.8 8.6
dod 00	Cancer, all forms (45- 53)		135.2 120.3 128.8	88.9 7.4	134.9 136.7 133.9	8 92.7 4 73.8
er 100,(Tuberculosis, all forms (23–32)		45.6 46.0 44.0	40.05 26.05 20.08	2 31.7 0 36.9 5 37.0	19.
rate p	E pidemic cerebrospinal meningitis (18)		0.7 1.8 1.8	2,0,0 8,0,0 8,0,0	.44	3.4
Death	Encephalitis, epidemic or lethargic (17)		12121 12121 12121			1.78
	Acute poliomyelitis and polioencephalitis (16)		0.11	1.8.8.	7 5 (*) . 1 5	9 9 9 9
	(II) s znsufinI		15.3 39.3 39.3	8884 1403	74.7	8.2
	Diphtheris (10)		1.1	2 2 2 3 2 3 2 3 2 3 2 3 2 3 3 2 3 3 3 3	• • •	3.1
	Whooping cough (9)		5 5 6 8 6 8 6	810 910 910 910 910 910 910 910 910 910 9		8 375
	Bearlet fever (8)		0.1 2.1.6 4			. e.
	(7) 29[289 M		0.2 1.3 3.7	11.4		€°.
	Typhoid fever (L, 2)		0.6	2.0		€°.
r 1,000 irths	Maternal mortality		بد بتری به تا ۲۵	3.9 6.4 7.2	0,0,4, 0,000	4. 5 5. 1
Rate per 1,000 live births	Total infant mortality		88 48 89	222		82
(sdrid [gunns]	Births (exclusive of still per 1,000 population (basis)		15.1 14.0 13.8	883	17.	18.7 18.3
(1 000, i 194 9345, rate per l,000 i sized lannas) noit		11.3 12.0 12.2	9.6 10.6 8.01 8.01	10.6 11.9	9.5
	State and period	JANUARY-JUNEcontinued	Washington: 1938- 1987- 1987- Woot Viterials	W 68/ V IR KLILLE: 1938	Wisconsin: 1988. 1987. 1986.	W yoming: 1988 1987

¹ Data not compiled for these causes prior to 1937. ⁷ Data not available. ⁸ No deaths reported.

EFFECT OF SODIUM SELENITE AND SELENATE ON THE OXYGEN CONSUMPTION OF MAMMALIAN TISSUES

By C. I. WRIGHT, Pharmacologist, United States Public Health Service, Division of Pharmacology, National Institute of Health

That selenium is highly toxic has been known for some time (1), but renewed interest in its toxicology has developed with the demonstration (2) of a relationship between the selenium content of forage and the cattle poisoning known as "alkali disease." Consequently, there have appeared a number of reports (2, 3) of extensive pathological changes in animals following the feeding and injection of selenium, but the fundamental cause of the injuries developed is still obscure.

There is evidence that selenium interferes with respiratory metabolism. Collett (4) showed that selenite inhibited succinic dehydrogenase of minced muscle. More recently Labes and Krebs (5) have shown that the oxygen consumption of a muscle powder suspension is inhibited by selenite; and Potter and Elvehjem (6), using sugars as substrate, have found that selenite inhibits the oxygen consumption of yeast, but has little effect on the oxidation of lactic or pyruvic acids.

There has been no systematic study of the effects of selenium on the metabolism of different tissues; and, with the exception of yeast cells, the interference with oxidations has been noted on tissue "brei" or extracts. In all cases relatively high concentrations of selenium were used. In order to assume that the toxic effects of selenium are a result of interference with oxidations it seems essential to demonstrate that such interference is possible in intact cells and at concentrations within the range of those existing in the poisoned animal. Therefore, freshly sliced organs were used in determining the sensitivity of different tissues to selenite and selenate. The inhibiting action of these selenium salts was also determined in the presence of several substrates, and attempts were made to counteract the depression of oxygen consumption.

METHOD

Measurements of oxygen consumption were made with the Barcroft differential type manometers fitted with two side-arm flasks of approximately 20 cc capacity. The carbon dioxide was absorbed by 0.2 cc of 7 percent KOH in central wells containing rolled filter paper. After introduction of the tissue slices the manometer flasks were immersed in a water bath at 37.5° C., flushed with water-saturated oxygen, and shaken at a rate of 110 oscillations per minute. The suspending medium was a phosphate buffered (pH 7.3) physiological salt solution containing 0.2 percent glucose as described by Dickens and Greville (7). The total volume of fluid in each flask was 3.0 cc.

With the exception of muscle and tumor the tissues were taken from 3- to 5-month old male and female white rats (Wistar strain) weighing on the average 151 (males) and 135 (females) grams. The rats were fed an adequate semisynthetic diet the ingredients of which have been given in an earlier publication (3). Mouse diaphragm was used for determinations on muscle, and tumor slices were obtained from rats ¹ given an intramuscular inoculation of Walker 256 mammary carcinoma 2 weeks previously. The nonnecrotic portions of the tumor mass were chosen for slicing.

The animals were decapitated and the organs quickly removed and sliced with a straight razor to a thickness less than 0.4 mm. Approximately 30 minutes elapsed between the time the animal was killed and the first reading of the manometers. Readings were made at 15minute intervals. At the completion of the experiment the slices were dried for 17 hours in an oven at 100° C. and the oxygen consumption calculated per mg of dry weight.

The solutions to be tested were placed in side arms at 10 times final concentration and tipped into the main compartment containing the tissue after a control period. All concentrations given are the final dilutions after tipping. The dl-lactic, pyruvic, and citric acids were carefully neutralized and diluted with physiological salt solution. Determinations were repeated on different rats at least once and usually several times, and so each curve given represents a number of experiments.

RESULTS

Sodium selenite.—The effect of sodium selenite on the oxygen consumption of five different tissues is shown in figure 1. The rate of oxygen consumption in mm³ per mg per hour is plotted against the time in minutes. The selenite was tipped from the side arm after a control period of 45 minutes, as indicated by the arrows on the time axis. Under the same conditions untreated tissues maintain 85 to 90 percent of the initial rate of oxygen consumption for 5 hours.

The effect of sodium selenite common to all the tissues studied is a depression of the rate of oxygen consumption. If the selenite is in sufficient concentration, the consumption of oxygen eventually falls to a value representing 5 percent or less of the initial rate in all tissues tested except tumor tissue. The oxygen consumption of the Walker tumor was reduced only 70 percent at concentrations as high as M/1,000, representing more than 100 times the minimal effective concentrations.

There are other distinct differences in the action of selenite on different tissues, the most striking of which is a marked increase in the rate of oxygen consumption of liver slices cut from livers of well-nourished rats. This increase is sharp and of short duration at the higher concentrations (M/1,000) but less intense and prolonged as the

I am indebted to Dr. W. R. Earle of this laboratory for the tumor-bearing animals.

concentration is decreased. The excess consumption of oxygen is variable at a single concentration and apparently depends on the nutritional condition of the animal. If food is withheld from the rat for a period of 24 to 48 hours immediately preceding the removal of the liver, the increase in oxygen consumption no longer occurs on addition of selenite to the suspending medium. Such periods of fasting reduce the glycogen content of the rat liver to 0.1 percent (8). Tumor and muscle frequently show a slight increase in the rate of oxidation following the addition of selenite but never as marked as liver.

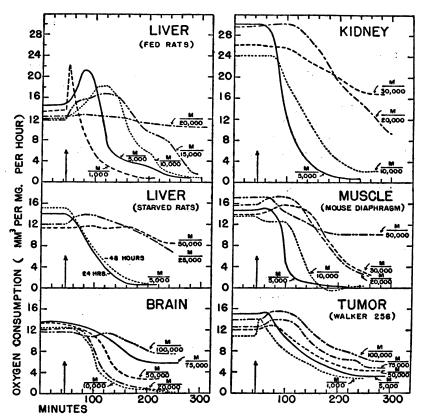


FIGURE 1.—The effect of sodium selenite on the oxygen consumption of tissue slices. The vertical arrows indicate the time of addition of selenite. Molar concentrations are given for each curve.

The tissues also vary in their sensitivity to selenite. The minimal molar concentrations affecting oxygen consumption are approximately as follows: Liver (fed) M/20,000, liver (unfed) M/25,000, kidney M/30,000, muscle M/50,000, brain cortex M/100,000, and tumor less than M/100,000. At all concentrations there is a fairly long induction period (30-60 minutes) during which the selenite has no depressant effect on oxygen consumption.

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Sodium selenate.—The main difference between the action of sodium selenite and sodium selenate (fig. 2) is in the concentration required for equivalent depression of oxygen consumption. Selenate causes an initial increase in oxidation when added to liver slices from well nourished rats and the increase disappears when food is withheld. At sufficiently high concentrations selenate also practically abolishes oxygen consumption. The order of sensitivity of the different tissues is not the same for selenate as for selenite. Thus, brain slices are but little affected by M/5,000 selenate, while the oxygen consumption of liver slices is definitely altered by half that concentration. Kidney and muscle are slightly more sensitive than brain but less so than liver.

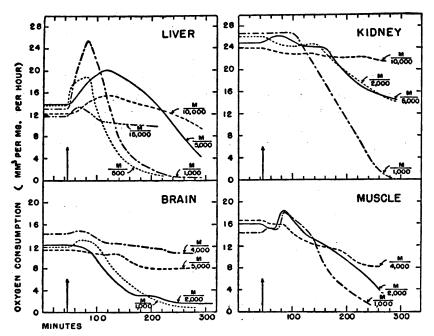


FIGURE 2.—The effect of sodium sclenate on the oxygen consumption of tissue slices. The vertical arrows indicate the time at which the sclenate was added. Molar concentrations are given for each curve.

The increased oxygen consumption of liver slices following the addition of selenite or selenate is probably an indirect result of an increased rate of glycolysis, resulting in a higher substrate concentration, or may be due to an interference with the normal glycolytic process and formation of more readily oxidizable substrates. This is indicated by the fact that fasting eliminates the stimulation and that iodoacetate, by inhibiting glycolysis (θ), can greatly diminish, and, under some circumstances, completely prevent the rise in oxygen consumption.

Figure 3 (A) shows the effect of adding iodoacetate and selenite to four liver slices taken from one animal. Addition of the selenite alone (curve 1) caused an increase in oxygen consumption to 20.6 mm³ per mg per hour. Addition of the iodoacetate alone (curve 2) had but little effect on the oxygen consumption for a period of an hour or more. Addition of the selenite and iodoacetate together at 30 minutes (curve 3) limited the rise in oxygen consumption to a maximum of 15.5 mm³ per mg per hour, and when the selenite was added 30 minutes after the iodoacetate (curve 4) there was practically no increase.

If selenite and selenate in some manner increase the rate of breakdown of glycogen in livers from fed rats and, through greater substrate formation, increase oxidations, it should be possible to simulate these conditions by adding substrates and selenite to fasting rat liver slices. Graph B of figure 3 shows that M/5,000 selenite alone added to starved rat liver slices caused no increase in oxygen

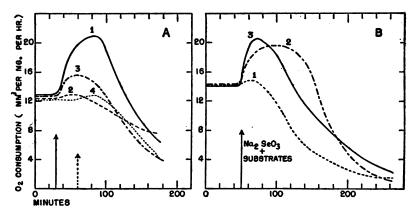


FIGURE 3.—Graph A shows the oxygen consumption of liver slices from a well-nourished rat after addition of Na₂SeO₃ (M/5,000) and iodoacetate (M/10,000). 1=selenite alone; 2=iodoacetate alone; 3=selenite and iodoacetate together at 30 minutes; 4=iodoacetate at 30 minutes followed by selenite at 60 minutes. Graph B shows the effect of selenite (M/5,000) and selenite plus substrates added to liver slices from a 24-hour fasted rat. 1=selenite; 2=selenite and pyruvate (0.02M); 3=selenite and succinate (0.02M).

consumption (curve 1). If pyruvate or succinate is added with the selenite (curves 2 and 3), the resulting rate of oxygen consumption is quite comparable to the curve resulting from the addition of selenite alone to liver slices from a fed rat (fig. 1). Furthermore, when the addition of pyruvate alone has raised the oxygen consumption of liver slices from unfed rats the subsequent addition of selenite does not further increase the oxidative rate. The latter fact is another indication that the stimulating action of selenite or selenate is effected through increased glycolysis rather than catalysis of oxidation.

However, it should be mentioned that up to the present time it has not been possible to demonstrate any effect of selenite on anaerobic glycolysis (glucose substrate) of liver, brain, kidney, or tumor slices. Mammalian tissues appear to differ from yeast in this respect (10).

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Substituting glycogen for glucose in the Ringer solution did not result in increased oxygen consumption on addition of selenite to starved liver slices. The glycogen probably failed to penetrate into the tissue cells.

THE OXIDATION OF **P-PHENYLENEDIAMINE AND SUBSTRATES**

Collett (4) showed that selenite inhibits the succinic dehydrogenase of minced tissue, and this finding has been confirmed repeatedly (5, 11, 12). Labes and Krebs (5) found that a suspension of muscle powder poisoned with selenite was able to catalyze the oxidation of p-phenylenediamine. The latter finding indicates that the cyto-

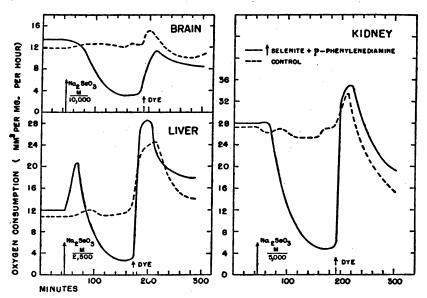


FIGURE 4.—The oxidation of p-phenylenediamine by tissue slices poisoned with sodium selenite. The scienite was added at 45 minutes and the p-phenylenediamine (0.02M) about two and a half hours later as indicated by small arrows.

chrome-indophenol-oxidase system (13) is intact and that the effect of selenite might be limited to succinic dehydrogenase.

Figure 4 shows that brain, liver, and kidney slices are able rapidly to oxidize p-phenylenediamine after being heavily poisoned with selenite. The solid lines represent the oxygen consumption of tissues treated with selenite and p-phenylenediamine, and the broken lines the tissues treated with p-phenylenediamine only. The selenite was added after 45 minutes, and approximately 2 hours later freshly prepared and neutralized p-phenylenediamine hydrochloride was added to the side arms and subsequently tipped on the tissues. The oxygen consumption of selenized tissues rose to the level of the nonpoisoned tissues, showing that the cytochrome oxidase system had not been impaired. If the oxidative mechanism consists of dehydrogenase, cytochrome, and oxidase (13), and selenite does not destroy the cytochrome nor the oxidase, then the action must be on the enzymes that activate the substrate hydrogen. The work of Potter and Elvehjem (6) on selenite poisoning in yeast shows that the oxidation of some substrates is not affected by selenite. There is some indication, then,

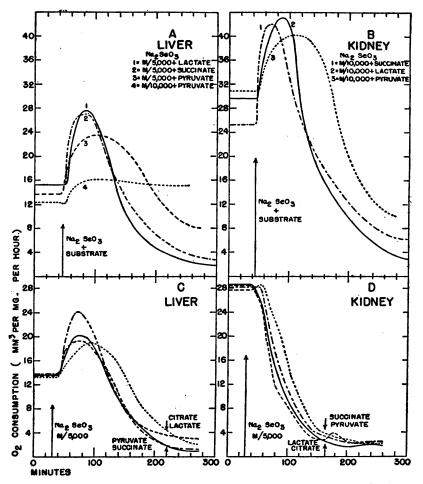


FIGURE 5.—The effect of addition of substrates to tissues poisoned with sodium selenite. Graphs A and B show the addition of selenite and substrates at the same time (45 min.). Graphs C and D show the inability of substrates to restore oxidation after poisoning with sodium selenite. The selenite was added at 30 minutes and the substrates (0.02M) at 220 minutes (liver) and 165 minutes (kidney).

that selenite is not a general dehydrogenase poison. In order to test this effect for mammalian tissues a number of substrates were added to the tissues after poisoning with sodium selenite (fig. 5, C and D). The selenite was added after 30 minutes and the substrate tipped from a second side arm after 160 minutes (kidney) and 220 minutes (liver). Neither lactate, pyruvate, citrate, nor succinate at con-

centrations of 0.02 molar caused any increase in oxygen consumption. It seems likely that selenite is a general poison for dehydrogenating enzymes.

However, substrates added at the same time as the selenite afford some protection to the tissue against the poisonous action of the latter. Thus in figure 5 (A and B) the fall in oxygen consumption due to M/5,000 and M/10,000 selenite is delayed by the addition of pyruvate and to a lesser degree by succinate and lactate. In fact, liver oxygen consumption can be completely maintained for 5 hours after adding M/10,000 selenite if pyruvic acid (0.02M) is added at the same time as shown in figure 5A, curve 4.

Results similar to those just discussed were also obtained on addition of substrates to tissues poisoned with sodium selenate.

REDUCED GLUTATHIONE AND SELENITE

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Since selenite and selenate catalyze the oxidation of glutathione (14), and the latter substance plays an important role in the metabolism of tissues, it was thought possible that a loss of reduced glutathione might offer at least a partial explanation of the toxic action of selenium. Attempts were therefore made to offset the effects of selenite by the addition of reduced glutathione.²

Reduced glutathione (M/1,000), when added to the suspending medium at the same time as the selenite (M/5,000), completely protects liver slices against the toxic effects of the latter (fig. 6A, curve 3). The excess oxygen consumption does not appear and there is no delayed fall in the rate of oxidations. The same concentration added to kidney slices (fig. 6B, curve 1) prevents the marked depression of oxygen that invariably follows the addition of M/5,000 molar selenite alone. If, however, the concentration of glutathione is reduced one-half (fig. 6A, curve 1) the protective action practically disappears. Reduction to one-half of both the glutathione and selenite concentrations (fig. 6B, curve 2) results in a fall of oxygen consumption almost as great as with the addition of M/10,000 selenite alone. Thus the ratio of glutathione to selenite of 5:1 appears adequate to protect the tissues at one concentration, but the same ratio has little protective action at a lower concentration level.

Reduced glutathione also offers partial protection to brain slices against M/20,000 selenite (fig. 6C).

The addition of glutathione to the tissues 30 to 60 minutes after the selenite will decrease somewhat the rate of fall of oxygen consumption (fig. 6A, curve 2, and 6B, curve 3). However, it is not possible to restore oxidation once lost through the action of selenite.

³ The glutathione was made available through the courtesy of Dr. J. M. Johnson who prepared it in this laboratory.

This is shown by graphs D and E of figure 6. The selenite was added to the kidney slice after 45 minutes; 150 minutes later 0.02 M reduced glutathione was added. The oxygen consumption temporarily increased to a value above the normal rate but fell sharply to a level below that preceding the glutathione addition. The same

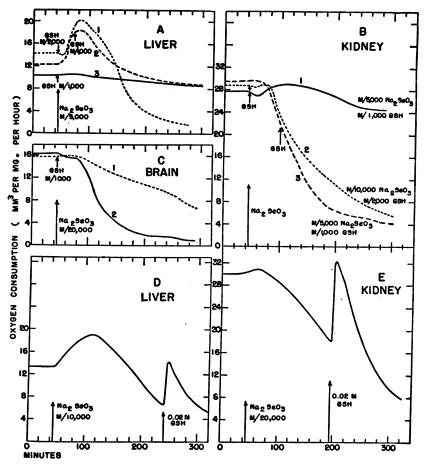


FIGURE 6.—The effect of reduced glutathione (G8H) on scienite-poisoned tissues. In A, B, and C the scienite was added at 45 minutes and the addition of G8H is indicated by small arrows. Molar concentrations are given for each curve. The lower graphs D and E show the effect of addition of 0.02M G8H after 50 percent reduction of 0₃ consumption by scienite.

result was obtained with liver (fig. 6D). The selenite was added after 45 minutes and the glutathione after 240 minutes. The increase in oxygen consumption after the glutathione addition was not maintained and in all probability represents the oxidation of a portion of the added glutathione.

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DISCUSSION

Mammalian tissues suffer an apparently irreversible loss of ability to oxidize naturally occurring substrates after contact with sodium selenite or selenate. The inhibition of oxygen consumption can be demonstrated in some tissues at a concentration of 10^{-5} molar and, assuming an equal distribution between tissue and solution, this represents roughly the concentration to be expected in an animal receiving a minimal lethal dose (3). It is possible, then, that some of the toxic effects of injected selenium might be due to injury to the oxidative mechanism in the tissues.

Since the cytochrome-indophenol-oxidase system is not impaired, and neither glucose, succinic, lactic, pyruvic, nor citric acid is oxidized, the injury is probably due to a general poisoning of dehydrogenating enzymes. The possibility remains, however, that succinic acid and its oxidation products serve as essential hydrogen carriers coupled with cytochrome, as postulated by Szent-Györgyi (15). In that case destruction of the succinic dehydrogenase alone would suffice to block the oxidation of the other substrates.

The fact that reduced glutathione can protect tissues against selenite, if added before the loss of oxygen consumption begins, has at least two possible explanations: The glutathione might reduce the selezate to the relatively innocuous colloidal selenium (16) before it penetrates the tissues and thus reduce the effective concentration of selenite or the reduced glutathione might serve to prevent the oxidation of sulfydryl groups that probably form an essential part of the oxidative mechanism. The first theory seems the more plausible, especially since glutathione cannot reverse the action of selenite. However, there are factors that argue against it: First, the ratio of glutathione to selenite that affords protection at one concentration does not protect at a lower concentration of selenite. Second. substances such as ascorbic acid and glucosamine, which rapidly reduce selenite in vitro, do not prevent the fall in oxygen consumption at concentrations as high as 0.02 molar. Neither does ascorbic acid reduce dithio groups in tissue (17).

Since the studies just reported were completed, Hopkins and Morgan (18) have shown that succinic dehydrogenase can be inactivated by oxidized glutathione and reactivated by reduced glutathione. In the light of their findings an attempt was made to reverse the action of selenite by exposing poisoned tissues to reduced glutathione in nitrogen for 30 minutes and then remeasuring the oxygen consumption with glucose and succinic acid as substrates. The poisoned tissues did not regain their oxidative ability through this procedure.

The delayed action of the selenite and the selenate may be due to a relatively slow penetration of the tissues. Cooper et al. (19) found

that valonia was impermeable to selenite. On the other hand, the rise in oxygen consumption of liver slices is quite prompt and indicates that the liver at least is readily permeable to selenite and selenate. Smith and co-workers (20) also found the selenium content of kidneys higher than blood 5 minutes after an intravenous injection of selenite.

The possibility that the inhibition of the oxygen consumption of veast by selenite might be an indirect result of interference with glycolysis is indicated by the results of Potter and Elvehjem (6). This does not seem to be true for mammalian tissues, since manometric measurements of anaerobic glycolysis of liver, kidney, brain. or tumor are not influenced by the presence of selenite. These results will be published later.

SUMMARY

1. Sodium selenite and selenate inhibit the oxygen consumption in vitro of liver, kidney, brain, muscle, and tumor slices. The initial effect on liver slices from well-nourished rats is a stimulation of oxygen consumption followed by a fall. The stimulation disappears if iodoacetate is added with the selenite, or if the rat is fasted 24 hours before removing the liver.

2. Selenite-poisoned tissues are not able to oxidize glucose or succinic, lactic, pyruvic, or citric acids, but rapidly oxidize p-phenylenediamine.

3. Reduced glutathione added with the selenite protects the tissues against the depressant action of selenite. Pyruvic acid added in sufficient concentration will also maintain oxygen consumption if added at the same time as the selenite. Delayed addition of glutathione or pyruvic acid does not restore oxygen consumption lost from contact with selenite.

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THE ASSAY OF URINE IN CANINE BLACKTONGUE BY THE **USE OF** Shigella paradysenteriae (SONNE)

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

Early in 1937 Knight (1) showed that vitamin B₁ plus nicotinic acid could completely replace the "staphylococcus growth factor" for 12 strains of Staphylococcus aureus in a medium of known chemical composition. Since the appearance of Knight's original paper, several workers have shown that other organisms require nicotinic acid. Among these, Koser and his associates (2) found that Shigella paradysenteriae (Flexner) and Shigella paradysenteriae (Sonne) require nicotinic acid.

Shortly after Knight's paper, Elvehjem et al. (3) showed that nicotinic acid would cure canine blacktongue, and Sebrell et al. (4), in 1938, reported that canine blacktongue could be prevented by adequate doses of nicotinic acid for a period of six months. Woollev et al. (5) later investigated a large number of compounds closely related to nicotinic acid for their blacktongue-curative activity. They found nicotinamide, diethyl nicotinamide, nicotinuric acid, etc., to be active in the cure of blacktongue. Dorfman et al. (6) observed that nicotinic acid, nicotinamide, methyl nicotinate, trigonelline amide, ethyl nicotinate, nicotinuric acid, etc., had growth supporting properties for certain bacteria in a synthetic basal medium.

It is conceivable that dogs on an adequate diet might excrete appreciable amounts of nicotinic acid, its derivatives, or closely

related compounds, while dogs on a blacktongue-producing diet might excrete much smaller amounts of these substances. Therefore, it was decided to use a modification of Knight's medium and technique for a biological assay for one or more substances in urine which would support growth of one of the organisms requiring nicotinic acid or related compounds in a basal synthetic medium complete except for nicotinic With this in mind various cultures were tested both for their acid. nicotinic acid requirement and for their ability to produce a turbidity of even distribution in order to facilitate readings. A culture of Shigella paradysenteriae (Sonne N. I. H. 741) was found to be suitable for this purpose.

EXPERIMENTAL

Six healthy young adult dogs (five males and one female) were selected for the experiment. Three of these animals (376, 381, 401) were given varying amounts of crystalline thyroxin while on diet 123.¹ This did not appear to influence the results of this experiment.

Technique of urine collection.—The dogs were walked for approximately 15 minutes or until they voluntarily voided. The animal was then given, by stomach tube, as much water as it appeared to be able comfortably to retain (400-700 cc) and placed in a metabolism cage, and urine was collected for a period of 4 hours. The five male dogs were catheterized and all residual urine was withdrawn at the end of the 4-hour period. The collected urine was measured for total volume and specific gravity. It was then filtered (Berkfeld N) and an aliquot portion stored in a refrigerator for the biological assay.

¹ Composition of blacktongue-producing diets:

		Diet numb	er
Nutrients	123	502	503
Corn meal*	Grams 400	Grams 65	Grams 65
Cornstarch* Cowpeas* Sugar	50 32	420 50	6 50
Casein, leached	C0	60 6	60 177
Cottonseed oil* Cod liver oil Salt mixture	30 15	6 12 26	6 12 26
Calcium carbonate Sodium chloride*	3 10		

Supplements:

Supplements:
0.2 mg synthetic vitamin B₁ bi-weekly, diets 502 and 503.
0.05 mg synthetic vitamin B₁ daily, diet 123.
0.5 mg riboflavin to each dog for 3 weeks beginning June 8, 1938, on diets 502 and 503.
Stock diet 326 consists of the following: graham flour,* 380 grams; whole milk powder, 120 grams; dried pork liver, 60 grams; brewers' yeast, 20 grams; cod liver oil, 10 grams; calcium carbonate, 6 grams; and sodium oblicit de constant. chloride,* 6 grams.

Each day the food served to every dog is weighed, the following day the residue deducted and the net food intake recorded.

^{*}These items are stirred into water and cooked in a double boiler of enamelware for about 1½ hours, except for cornstarch, which is cooked for 20 minutes. Then the other ingredients are well stirred in, the total weight being brought to 2.400 grams with water (so that 1 gram represents 1 calorie), and this finished mixture is fed to the dog ad libitum.

Experimental status of animals at the time of urine collection.-A sample of urine was collected from each dog at the beginning of the experiment while on stock diet 326.² The animals were then placed on blacktongue-producing diet 123² and a second sample of urine was collected from five dogs when they had definite symptoms of blacktongue (from the sixth dog after 52 days on the experimental diet). A third specimen was obtained from four dogs after the symptoms of blacktongue had entirely disappeared following return to stock diet 326. Two of these dogs were also given nicotinic acid therapy. Later, another 4-hour urine specimen was obtained from these four dogs 1 hour following the administration of 5 mg per kilo of body weight of nicotinic acid in distilled water given intramuscularly. A final urine specimen was then collected from 48 to 72 hours later.

Bacteriological technique.-The sample of urine to be tested was added to the basal media ³ in ascending dilutions from 0.1 cc of undiluted urine through the dilutions 1:10, 1:20, etc., to 1:1280. This basal medium, plus the dilutions of urine, was then autoclaved for 15 minutes at 15 pounds' pressure. The tubes, after cooling, were inoculated with a small loopful of a suspension of Shigella paradusenteriae (Sonne) prepared by touching a 24-48 hour agar slant of the organism with a straight wire and suspending in 5 cc of saline.

² See footnote 1.

³ Preparation of media. -- Nutrient solution I is prepared as follows:

Substance	Amount/1,000 cc	Substance	g/1,000 cc
KH ₁ PO ₄	4.5 g 550.0 ml 26.0 ml .12 g .15 g .17 g .05 g .07 g .08 g	S-aspartic acid d-glutamic acid S-methionine. S-phenylalanine. 1-tyrosine. d-arginine HCl 1-histidine HCl S-lysine HCl	0. 18 . 09 . 07 . 08 . 05 . 05 . 05 . 05 . 09

The amino acids are dissolved, the pH is brought to 7.4, and the volume is adjusted to 600 ml. The solution is placed in 200-cc flasks, stoppered, autoclaved, and stored in a refrigerator. The number of tubes, each finally to contain 1 ml, is computed. For example, say 100 tubes; this will amount to 100 cc of media, which will be composed of the following:

Substance Nutrient solution I	Concentration	Amount 60 ml
Vitamin B.	10-6 M in H.O	10 ml
Ferrous ammonium sulfate ^b Dithiodiglycollic acid (Na salt) ^e	M/10 SH	2.0 ml
	M2 Triple distilled	2.5 ml 13.0 ml
	Total	

This solution is then divided into the 100 tubes, each tube receiving 0.9 ml. Finally, the tubes each receive 0.1 ml of urine dilutions, or nicotinamide 4 (1×10-4) dilutions. Certain control tubes receive 0.1 ml of triple distilled water.

The tubes are then plugged, autoclaved for 15 minutes at 15 pounds' pressure, and, after cooling, are ready for inoculation.

· Modification of basal media originally developed by Fildes et al. (Brit. J. Exp. Path., 17; 481 (1936)).

Prepared freshly for each test.
 Prepared by Dr. Floyd S. Daft. Division of Chemistry, National Institute of Health.
 Specially purified and furnished by courtesy of Merck & Co.

After the tubes had been inoculated, they were incubated at 37° C. for about 20 hours, and the presence or absence of growth was read at once. The readings were made by comparing the gross turbidity produced by the growth of the organism in the tubes containing media plus urine dilutions with tubes containing media plus dilutions of either nicotinic acid or nicotinamide. Controls were included in each test for the sterility of the urine, sterility of the basal medium, and for the nicotinic acid requirements of the organism. In no test has there ever been perceptible growth of the Sonne bacillus in the basal medium without the addition of one of the growth supporting substances (i. e., nicotinic acid, nicotinamide, or urine). The results are summarized in table 1.

DISCUSSION

Table 1.—This table reveals a marked and consistent decrease in the bacterial growth promoting properties of the urine collected from all six dogs after 31 to 52 days on the blacktongue-producing diet. In one instance (dog 401) this was observed even though the animal showed no clinical signs of blacktongue after 52 days on the blacktongue-producing diet. This animal was then returned to stock diet 326 for a period of 15 days. Two dogs with blacktongue (358, 376) were treated with nicotinic acid and then returned to stock diet 326 for 12 and 20 days, respectively. Urine collected from these three dogs at that time showed bacterial growth promoting properties comparable to that observed before starting the blacktongue-producing diet. One dog (381) that developed blacktongue was returned to stock diet 326 for a period of 14 days without nicotinic acid treatment. Urine collected at that time demonstrated less bacterial growth promoting value than the specimen collected before starting the experimental This is consistent with the observation that the urine of dogs diet. 358 and 376, receiving both nicotinic acid and stock diet 326, gave a titration comparable to that at the beginning of the experiment.

Four of the animals (358, 376, 387, 401) were given an intramuscular injection of 5 mg of nicotinic acid per kilo of body weight and the urine manifested a marked increase in bacterial growth promoting properties, considerably above that shown by animals when on stock diet alone. Another specimen taken from the same animals from 48 to 72 hours later demonstrated a return to a level comparable to that on stock diet previous to the administration of nicotinic acid.

In order to avoid possible differences in rate and amount of bacterial growth at different times, all of the samples from any one dog were run simultaneously. In addition, the first and second urine samples from all of the dogs were run concurrently, and, as a further check, all urine samples from all dogs, except the two that died of blacktongue, were run at the same time under identical conditions.

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incubation
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1.—Results
TABLE

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ANCED NG UE NG UE	~	0	0	0		0	7/21 FIRST SIGN OF BLACKTONGUE	400 400	400 230 1 000 400 300 1 001	400 230 1 000 400 300 1 001
NOLE	ŧ	+ +	0 +	0	0	0				
NCED	• +	0	0 0	0			T/21 FIRST SIGN OF BLACKTONGUE.	600	900 900	600 600 1.000
NCED	‡ ‡	ŧ	+	0	6		1			<u>}</u>
	• +	0	0	0		0	7/21 FIRST SIGN OF BLACKTONGUE	001	8	700 600 1.000
326/12/8/24 NORMAL HHH	+ ‡	+	0 +	0	0	0	8/18	001	002	100 100 1.002
326 19 8/31 NORMAL	‡ ‡	ŧ) 	+	+		OIVEN 6 MG/KILO (62 5 MG) MIC ACIO LUG RECOL LIGIL È COL		310	500 310 1.000
326 22 9/3 NORMAL HIH	+ ‡	+	0 +	0	0	0				1.001
326 99 6/23 NORMAL HHH	‡ ‡	ŧ	+++++++++++++++++++++++++++++++++++++++	0	0	0		000	940	500 440 I.005
123 46 8/9 {EARLY	+	0	0	0	_		BAL BIVEN AD ME BAP AND AAR AND AND A AND A		600 460 1.001	00.
326 20 8/30 NORMAL HIH	+ + +	+	+	0	0	0			000	000.1 000 000
326 21 9/1 NORMAL HIH	‡ ‡	+	+ + + + + + + + + + + + + + + + + + + +	+	+			600	222	eoo 222 1.000
32623 9/3 NORMAL HHH	+ +	+	• •	0	1 0		WITH WE AND AT HIS NIC. ACID INR. BEFORE URINE COL.	E COL 600	465	600 465 1.000
326 98 6/23 NORMAL HHH	‡ ‡	1	+	+-	┿	+		600	505	600 505 1.004
123 46 8/9 (EARLY	+	0	0	1 0			8/2 FIRST SIGN OF BLACKTONQUE.	500	200 330 1 001	1001
	‡	0	0	0			e /i0	460	265	460 265 1.002
326 21 8/31 NORMAL	ŧ	\$	+		-			200	500 145	000 1
NORMAL HI	#	: +	+ c	нc	-		BIVEN D MG./KILO (33 MG.) NIC. ACID I MR. BEFORE URINE COL. 500 255 1 003	4E COL. 500	255	1 003
	ŧ	1 ‡	+	-	+	+		500	380	500 380 1.004
1	-	: 0	+ C					550	455	550 455 1 002
	- 1	> =) (BUME ANOREXIABEGINS STOCK DIET 326	490	210	490 210 1000
		= =) = =	.				500	460	500 460 1 000
		ŧ :		+ •		р (н (GIVEN SMA/KILD (41 MG.) NIC. ACID I HR BEFORE URINE COL 500 250 1 002	15 COL. 500	250	1 002
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Supplementary observations on animals on stock diet and other blacktongue-producing diets.—Even though the urine of depleted dogs on blacktongue-producing diet 123 produced the results given in table 1, it is possible that other blacktongue-producing diets would show no correlation between the clinical condition and urinary findings. Therefore, the urine of dogs on a high carbohydrate and on a high fat blacktongue-producing diet was investigated. A random sample of urine was taken from each of two dogs (384, 405) in an acute attack of blacktongue after 34 and 37 days, respectively, on diet 503.⁴ These samples showed definitely less bacterial growth promoting ability than a 24-hour sample from two control dogs (353, 388) on stock diet 326, run simultaneously.

A 24-hour urine specimen from one dog (392), after 5 days on diet 503, showed bacterial growth promoting value comparable to urine from dogs on diet 326 (visible growth at dilution 1:160), but after 20 days on the diet, although there were no clinical signs of blacktongue, visible bacterial growth decreased to a dilution of 1:40. This result, together with that obtained on dog 401 (table 1), indicates that there is a decrease in the excretion of the bacterial growth promoting substances in urine preceding the development of the clinical signs of blacktongue.

A 24-hour urine specimen from one dog (409), taken 2 days after beginning the blacktongue-producing diet 502,⁴ showed a bacterial growth promoting value comparable to urine from two dogs on stock diet 326 (visible growth in dilution 1:320). Eighteen days later, bacterial growth was visible only in dilution of 1:80, and 28 days from the beginning of the experiment, when the dog showed early clinical signs of blacktongue, bacterial growth was visible only in dilution of 1:40. Two days later, although the clinical signs of blacktongue persisted, the urine gave visible bacterial growth in dilution of 1:160. However, this animal unexpectedly made a spontaneous recovery, and within 4 days the clinical signs of blacktongue had entirely disappeared without treatment or change of diet. Furthermore, although this animal was continued on the diet for an additional 40 days and then sacrificed, post-mortem examination revealed no gross evidence of blacktongue.

General.—We have not attempted to estimate nicotinic acid quantitatively in dogs' urine by this test because we have no direct evidence that the substances being tested in this experiment are nicotinic acid or its derivatives. However, the observation that there was a marked increase in the bacterial growth promoting power of the urine following the administration of nicotinic acid, as well as the marked decrease in the growth promoting value of the urine of dogs with blacktongue,

⁴ See footnote 1.

suggests that the principal factor concerned in this experiment is nicotinic acid or related compounds.

In every instance there has been a close correlation between the results of the biological assay and the clinical condition of the animal. Therefore, it appears that this test may be utilized to study some of the aspects of canine blacktongue.

CONCLUSIONS

1. A method for assaving the bacterial growth promoting properties of urine by the use of Shigella paradysenteriae (Sonne), which requires nicotinic acid or its related compounds, is presented.

2. Results obtained with urine from dogs on stock diet and blacktongue-producing diets indicate that this test may be correlated with the clinical condition of the animal.

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DEATHS DURING WEEK ENDED SEPTEMBER 24. 1938¹

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

•	Week ended Sept. 24, 1938	Correspond- ing week, 1937
Data from 87 large cities of the United States: Total deaths. Average for 3 prior years Total deaths, first 38 weeks of year Deaths under 1 year of age. Average for 3 prior years Deaths under 1 year of age. Death for industrial insurance companies: Policies in force Number of death claims	7, 321 37, 069 306, 874 496 3495 19, 933 68, 268, 220 10, 891 8, 3 9, 3	* 7, 597 330, 587 * 505 21, 340 69, 872, 337 11, 867 8. 9 9. 9

¹ The figures presented in the table appearing in the Public Health Reports for Sept. 30, p. 1748, were for the week ended Sept. 10 instead of Sept. 17 as published.

³ Data for 86 cities. ³ Data for 85 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

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 Oct. 1, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median

		Diph	theria			Inf	uenza			Me	asles	
Division and State	Oct.1, 1938, rate	Oct.1, 1938, cases	Oct.2, 1937, cases	1933 37 me- dian	Oct.1, 1938, rate	Oct.1, 1938, cases	Oct.2, 1937, cases	1933- 37 me- dian	Oct.1, 1938, rate	Oct.1, 1938, cases	Oct.2, 1937, cases	1933 37 me- dian
NEW ENG.												
Maine New Hampshire Vermont. Massachusetts Rhode Island. Connecticut.	6 0 4 0 3		1 0 1 0 3	1 0 8 1 3	6 9	1 3	 2		6 61 12	1 52 4	8 1 6 11 5	3 1 6 12 2 5
MID. ATL.									•			
New York New Jersey Pennsylvania	5 7 7	13 6 14	23 7 19	27 15 39	1 1 14 			¹ 11 9		60 5 46	115 24 234	60 18 30
E. NO. CEN.												
Ohio Indiana Illinois Michigan ³ Wisconsin	25 44 17 12 0	32 29 26 11 0	33 28 35 24 2	40 29 35 19 2	32 8 	21 12 23	15 14 9 2 35	19- 15- 9- 2 20	18 3 15 56 98	23 2 22 52 55	94 3 45 15 28	20 3 15 20 33
W. NO. CEN.												
Minnesota Iowa Missouri North Dekota South Dakota Nebraska Kansas	22 63 32 8 77 17	11 31 25 3 1 7 6	3 5 38 0 0 0 5	4 7 45 2 1 5 7	10 10 14 37 15 3	5 5 11 5 2 1	5 22 	 28 	73 12 4 465 75 8 17	37 6 3 63 10 2 6	6 3 15 4 2	7 3 15 4 1 1 3

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended Oct. 1, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

•		Diph	theria			Inf	uenza			м	083]05	
Division and State	Oct.1, 1938, rate	Oct.1, 1938, cases	Oct.2, 1937, cases	1933 37 me- dian	Oct.1, 1938, rate	Oct.1, 1938, cases	Oct.2, 1937, cases	1933- 37 me- dian	Oct.1 1938, rate	, Oct.1, 1938, cases	Oct.2, 1937, cases	1933- 87 me- dian
SO. ATL.												
Delaware Maryland ¹ ³ Dist. of Col Virginia West Virginia North Carolina ³ ⁴ Georgia ⁴ Florida ⁴	60 33 96 59 158 120 63 31	3 2 4 50 21 106 43 37 10	2	0 13 10 39 45 104 23 52 12	12 17 145 34 3667 93	4 2 75 12 2 240 55	3 5 1 122 3	3 7 5 142	24 12 14 70		10 9 21	8
E. SO. CEN.												
Kentucky Tennessee 4 Alabama 4 Mississippi 3	77 61 140 85	43 34 78 33	24 39 29 17	50 47 48 30	45 61 50	25 34 28	3 13 24	1 11 9 	21 4 13	2	12 47 1	9 14 5
W. SO. CEN.												
Arkansas ³ Louisiana ⁴ Oklahoma Texas ⁴	59 34 25 36	23 14 12 43	20 14 7 40	14 16 12 40	64 12 76 91	25 5 37 108	5 3 31 135	5 3 27 45	3 54 8 11	22	8 1 1 13	2 1 1 10
MOUNTAIN						- 1						
Montana Idaho Wyoming Colorado New Mexico Arizona Utah ²	0 0 44 122 37 25 10	0 2 25 3 2 1	0 0 18 2 6 2	2 0 0 4 2 0	39 32 202	4 8 16	4 18 10	8 2 10	203 11 133 34 37 38 20	21 1 6 7 8 3 2	16 8 7 3 95	4
PACIFIC						i						
Washington Oregon California	3 20 34	1 4 40	4 0 15	3 0 29	36 13	7 15	11 16	18 24	28 41 110	9 8 130	6 8 18	13 8 47
Total	34	854	784	984	40	800	534	534	33	799	918	672
89 weeks	19 1	8, 253	6, 979 2	2, 422	63 4	9, 189 2	76, 830	143, 202	804	764, 564	245, 396	344, 747
	Me	ningiti co	is, men ecus	ingo-		Polior	nyelitis		<u> </u>	Scarle	fever	
Division and State	Oct. 1, 1938, rate	Oct. 1, 1938, cases	Oct. 2, 1937, cases	1933- 37 me- dian	Oct. 1, 1938, rate	Oct. 1, 1938, cases	Oct. 2, 1937, cases	1933– 37 me- dian	Oct. 1, 1938, rate	Oct. 1, 1938, cases	Oct. 2, 1937, cases	1933 37 me- dian
NEW ENG.												
Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	0 0 0 1.2 0 8			000000000000000000000000000000000000000	0 0 2.4 0	0 0 2 0 5	0 2 16 0	6 0 2 13 0 7	24 31 95 47 23 30	4 3 7 40 3 10	4 8 3 64 8 19	4 8 64 10 13
MID. ATL. New York New Jersey Pennsylvania	0.8 1.2 1		9 0 4	Ō	1.2	1	45 12 31	45 12 15	50 30 36	125 25 71	128 45 149	130 38 168

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended Oct. 1, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

					-							
	Me		s, meni cus	ngo-		Polion	yelitis			Scarle	t fever	
Division and State	Oct. 1, 1938, rate	Oct. 1, 1938, cases	Oct. 2, 1937, cases	1933 37 m e - dian	Oct. 1, 1938, rate	Oct. 1, 1938, cases	Oct. 2, 1937, cases	1933- 37 me- dian	Oct. 1, 1938, rate	Oct. 1, 1938, cases	Oct. 2, 1937, cases	1933- 37 me- dian
E. NO. CEN.												
Ohio Indiana Illinois Michigan ² Wisconsin	0.8 0 0 1.1 0	1 0 0 1 0	3 1 3 1 1	3 1 3 1 1	3 1.5 4 2.2 0	6	40 8 72 44 34	27 7 15 14 7	118 134 91 198 143	89 138 183	238 85 161 163 44	238 85 161 92 76
W. NO. CEN.												
Minnesota Iowa Missouri North Dakota South Dakota Nebraska Kansas	0 0 7 0 0 0	0 0 1 0 0 0	0 0 3 0 2 0	0 1 2 0 0 0 0	0 2 0 8 0 0	0 1 0 1 0 0	28 18 20 0 2 18 26	4 3 2 0 2 1 4	106 51 106 23 50 196	25 81 13 3	24 66 146 8 9 11 61	24 41 51 11 9 13 40
SO. ATL.												
Delaware. Maryland ¹³ Dist. of Col West Virginia. Worth Carolina ³⁴ South Carolina ³⁴ Georgia ⁴ Florida ⁴	0 9 0 2.8 0 0 0 0	0 3 0 1 0 0 0 0	0 3 1 1 3 1 2 0	0 1 0 1 0 2 0	0 3 0 2.8 0 2.8 0 0	0 1 0 1 0 1 0 0	0 7 2 1 2 2 0 2 0	0 5 1 2 4 2 0 0 0	40 25 67 71 134 124 36 39 25	2 8 8 37 45 83 13 23 8	10 36 4 23 57 88 7 27 27 27 2	9 36 14 34 73 88 8 20 2
E. SO. CEN.												
Kentucky Tennessee 4 Alabama 4 Mississippi 3	0 0 5 0	0 0 3 0	4 1 0 1	4 1 2 1	0 0 7 0	0 0 4 0	2 4 1 8	3 4 1 0	127 88 54 28	71 49 30 11	57 41 23 13	57 55 23 15
W. SO. CEN.												
Arkansas ³ Louisiana ⁴ Oklahoma Texas ⁴	0 2.4 2 0	0 1 1 0	3 0 1 2	0 1 1 0	0 0 4 1.7	0 0 2 2	12 3 21 26	1 1 1 3	23 12 41 43	9 5 20 51	15 4 14 50	13 5 12 31
MOUNTAIN												
Montana Idaho Wyoming Colorado New Mexico Arizona Utah ³	0 0 0 0 0 0	0 0 0 0 0 0	1 0 0 0 0 0	0 0 0 0 0 0	000000000000000000000000000000000000000	0 0 0 0 0 0	3 1 31 0 0 3	1 0 1 1 1 1	203 74 67 93 49 38 50	21 7 3 19 4 3 5	9 10 3 18 10 2 16	9 9 4 18 10 6 7
PACIFIC												
Washington Oregon California	0 0 0.8	0 0 1	0 0 1	0 1 1	0 0 6	0 0 7	6 3 30	6 3 26	31 117 94	10 23 111	18 10 119	19 22 118
Total	0.8	20	53	52	2. 1	52	603	316	76	1, 871	2, 125	2, 210
39 weeks	2.4	2, 337	4, 499	4, 499	1.4	1, 354	7, 724	5, 807	149	144, 157	172, 584	172, 584

See footnotes at end of table.

Cases of certain diseases reported by telegraph by State health officers for the week ended Oct. 1, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median—Continued

New Hampchire			Sma	llpox		Typl	noid and fe	l paraty ver	phoid		oping ugh
Maine 0 0 0 0 12 2 3 110 18 Vermont 0	Division and State	1, 1938,	1, 1938,	2, 1937,	37 me-	1, 1938,	1, 1938,	2 , 1937,	87 me-	1, 1938,	1, 1938,
New York 0 0 0 0 0 0 11 27 29 26 194 481 Pennsylvania 0 0 0 0 0 0 0 9 17 41 41 95 182 Pennsylvania 0 0 0 0 0 9 17 41 41 95 182 Ohio 1 1 1 1 0 13 17 49 49 177 228 183 Minnesota 0 0 1 1 0 4 2 2 2 2 2 2 2 2 2 2 11 13 14 12 13 13 12 13 12 <th13< th=""></th13<>	Maine New Hampshire Vermont Massachusetts Rhode Island	000000000000000000000000000000000000000	000000000000000000000000000000000000000	000000000000000000000000000000000000000	000	0 0 2 23	023	6 3 1	003	0 204 92 69	0 15 78 9
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	Washington Oregon California	20 2	42	1	0	5 17	1 20	4 12	4 12	46 89	9 105
39 weeks	Total 89 weeks		38 12, 932		33 5, 484						

 New York City only.
 Period ended earlier than Saturday.
 Rocky Mountain spotted fever, week ended October 1, 1938, 3 cases as follows: Maryland, 1; North ⁴ Typhus fever, week ended October 1, 1938, 64 cases as follows: North Carolina, 1; South Carolina, 9; Georgia, 24; Florida, 1; Tennessee, 1; Alabama, 10; Louisiana, 1; Texas, 17.

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- sles	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
July 1938 Hawaii Territory New Hampshire	0	5 1	17	1	17		0 0	6	0 0	73
August 1938 Arizona California. Hawaii Territory Louisiana Massachusetts Nevada New Hampshire New Hampshire North Dakota Oregon	1 12 0 7 4 1 0 2 0	24 78 55 8 0 2 3	61 49 14 39 	3 106 	31 675 5 19 261 3 	2 10 	0 29 0 3 4 0 3 0	4 282 120 1 18 34	3 38 0 0 0 0 7 17	12 52 11 72 11 1 1 1 0
Vermont Virginia Washington	0 1 8	1 77 8	155 5	20 1	22 108 33	22	2 12 1	9 34 43	0 0 29	5 60 42

July 1938

August 1938-Continued

August 1938—Continued

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Chickenpor.28 Dysentery (amoebic)	Hawaii Territory:	Case			Septic sore throat-Con,	Cases
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Hokworm disease	Dysentery (amoebic)		California		Massachusetts	. ç
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California			Sentic sore throat:		Virginia	
	California	89	California	9	Washington	

PLAGUE INFECTION IN CALIFORNIA

IN POOLS OF FLEAS FROM GROUND SQUIRRELS AND IN A GROUND SQUIRREL IN SAN BERNARDINO AND ELDORADO COUNTIES

Under date of September 7, 1938, Doctor W. M. Dickie, Director of Public Health of California, reported plague infection proved in a pool of 39 fleas from 14 *fisheri* squirrels collected August 18, from Running Springs, 2 miles south, 4 miles east of Lake Arrowhead, San Bernardino County; and under date of September 15, in one *beecheyi* squirrel shot August 8, one mile northwest of Tallac, Fallen Leaf Lake, Eldorado County, and in a pool of 11 fleas from 3 *beecheyi* squirrels collected August 15, 2 miles south of Tallac, Fallen Leaf Lake, Eldorado County.

WEEKLY REPORTS FROM CITIES

City reports for week ended Sept. 24, 1938

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

									· · · · · · · · · · · · · · · · · · ·		
State and city	Diph- theria	Inf	uenza	Mea- sles	Pneu- monia	Scar- let	Small pox	Tuber-	Ty- phoid	Whoop- ing	Deaths,
	cases	Cases	Deaths	cases	deaths	fever cases	cases	deaths	fever cases	cough cases	causes
Data for 90 cities:											
5-year average	153	69	17	121	325	438	3	341	87	924	1
Current week 1.	99	157	10	222	305	367	2	313	48	1, 344	
Maine:										İ	ĺ
Portland New Hampshire:	0		0	0	2	0	0	0	0	2	31
Concord	0		0	0	0	0	0	l ol	0	0	8
Manchester	Ó		Ó	Ō	ŏ	ž	Ŏ	ŏ	ŏ	ŏ	13
Nashua	0		0	0	0	0	0	0	0	Ó.	3
Vermont:											
Barre Burlington	0		0	1	0	3	0	0	0	0	1
Rutland	ŏ		0	0	0	0	C O	0	0	0	10
Massachusetts:				v		v	U	U	U	0	3
Boston	0		1	3	17	17	0	8	0	8	193
Fall River	1		Ō	Ó	1	ö	ŏ	i	ŏ	ĭ	36
Springfield	0		0	0	2	1	0	Ō	Ō	2	39
Worcester	0		0	2	3	0	0	1	0	13	42
Rhode Island: Pawtucket	0				· _					_	
Providence	ŏ		0	0	7	0	0	0	0	0	16 77
Connecticut:	۳		•		- 1		v	V I		13	
Bridgeport	0		0	0	1	0	0	1	0	2	29
Hartford	Ō		ŏ	2	õ	ĭ	ŏ	ō	ŏ	ī	31
New Haven	0	1	0	2	1	0	Ó	1	Ō	- 16	32
New York:				1							
Buffalo	1		0	1	3	7	0	5	0	22	127
New York	9	5	1	16	53	16	0	53	11	242	· 1, 331
Rochester	0		0	5	1 2	0	0	2	0	3	54
New Jersey:	•		U I		z	2	0	0	1	10	44
Camden	0		0	0	1	1	0	0	1	ol	24
Newark	Õ.		ŏ	2	4	7	ŏ	5	ôl	57	88
Trenton	0		0	0	5	0	ŏ	ŏ	ŏ	· 1	28
Pennsylvania:											
Philadelphia	0.		1	7	13	15	0	27	2	92	431
Pittsburgh Reading	5.		0	2	10	9	0	9	0	14	130
Scranton	il			ŏ	•	0	0	1	0	5	19
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Cleveland	1.	5	8	05	0	7	0	1	1	.9	110
Columbus	ō.	"	ŏ	î	2	8	0	16		- 36 3	142 80
Toledo	ŏĿ		ŏl	il	41	ŝ	8 I	5	ŏ	2	80 68
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¹Figures for South Bend, Ind., and Tacoma, Wash., estimated; reports not received.

Ginte and sit-	Diph-	Inf	luenza	Mea- sles	Pneu- monia	Scar- let	Small	Tuber- culosis	Ty- phoid	Whoop- ing	Deaths
State and city	theria cases	Cases	Deaths	8168 C8365	deaths	fever cases	pox cases	deaths	fever cases	cough cases	all causes
Indiana:											
Anderson	0		0	0	0	4	0	0	0	0	
Fort Wayne	0		0	0	1	0	0	0	1	0	2
Indianapolis	3		0	0	11	5	1	3	2	6	6
Muncie	0		0	0	0	2	0	0	0	0	
South Bend	;-		0			7	ō	0	0	1	
Terre Haute	1			0	0	•		۱ v	U	•	1 1
filinois: Alton	0		0	0	0	0	0	0	1	0	
Chicago	12	4	2	ğ	20	58	ŏ	37	5	249	6
Elgin	ō		ō	ŏ	ī	Ő	Ŏ	Ö	Ŏ	0	Ĩ
Moline	Ŏ		0	0	0	0	3	0	0	0	
Springfield	0		0	0	8	0	0	0	0	0	
Michigan:											
Detroit	3		0	3	8	34	0	9	0	135	1
Flint	0		0	2	6	14	0	0	0	9	
Grand Rapids.	0		0	1	1	15	0	2	0	0	
Wisconsin:	0		0	0	0	1	0	0	0	7	1
Kenosha Madison	ŏ		ŏ	ĭ	ŏ	4	ŏ	ŏ	ŏ	l i	1 '
Milwaukee	i		ŏ	3	l ĭ	19	ŏ	3	ŏ	141	
Racine	- Ō		ŏ	ŏ	Ô	4	ŏ	Ĭ	ŏ	29	
Superior	ŏ		ŏ	ŏ	ŏ	ō	Ŏ	ō	Õ	2	
			-			-					
Minnesota:											
Duluth	0		0	Q	1	1	0	0	0	6	
Minneapolis	0		0	3	4	7	0	0	0	M	6
St. Paul	0		. 0	3	6	3	0	2	0	15	
lowa:				0		0	0	0		1	
Cedar Rapids	0			ŏ		ŏ	ŏ	ŏ		Ó	
Davenport Des Moines	1 0		Ö	ŏ	0	ŏ	ŏ	ŏ	0	ŏ	2
Sioux City	ŏ		v	ĭ	l VI	2	ŏ	ŏ	•	ĭ	· · · · ·
Waterloo	. 7			ō		2	ŏ		0	ō	
Missouri:	•			•							
Kansas City	2		0	0	2	7	0	2	0	3	7
St. Joseph	Ō		0	0	0	2	1	2	0	1	1
St. Louis	8	1	0	0	6	2	0	10	5	10	12
North Dakota:											
Fargo.	0		0	24	. 0	1	0	0	0	0	:
Grand Forks	0			. 0		0	. 0		0	. 0	
Minot	1		0	. 0	0	0	. U	0		v	
South Dakota:		•		0		1	: 0		0	0	
Aberdeen Sioux Falls	ŏ		0	ŏ	0	2	ŏ	0	ŏ	ŏ	1
Nebraska:	v		, v	v	•		, v	Ť	•	•	-
Lincoln	0			1		. 0	0		. 0	8	
Omaha	i		0	Õ	8	Ó	Ó	1	Ó	0	ł
Kansas:				-							•
Lawrence	0		0	0	1	. 0	0	0	0	0	20
Topeka	0		0	0	1	8	0	0	0	6	1
Wichita	1		0	0	1	4	0	2	0	8	1
Delaware:	. 0		0	0	1	0	0	1	0	0	2
Wilmington			۰	v	-	° I	v	- 1			•
Baltimore	. 8	8	1	6	11	2	. 0	14	2	17	18
Cumberland	ő	•	ō	ŏ	Ö	õ	ŏ	Ö	ō	ö	
Frederick	ŏ		ŏ	ŏ	ŏ	ŏ	ŏ	ŏ	ŏ	Ó	
Dist. of Col.:	•		-	-							
Washington	4	2	0	1	8	7	0	8	8	7	, 12
/irginia			1	_		_					
Lynchburg	2		0	0	0	0	0	0	0	0	1
NOFICIE	0		0	0	1	- 2	0	1	0	<u> </u>	. 2
Richmond	4		0	2	2	- 4	0	1	1	1	3
Roanoke	1		0	U	0	1	0	0	1	v	
Vest Virginia: Charleston	, I	- + -	0	0	1	0	- ¹ 0	1	1	0	2
Huntington	1		۷I	ő		ĩ	ŏ	- 1	ő	ŏ	-
Wheeling	ő		ō-	2	i	ô l	ŏ	8	ŏ	ĭ	1
Forth Carolina:			•	- 1	•	۳I	۲ ۲	•		_	-
Gastonia	1	1		0		0	0		0	0	
Raleigh	1		ŏ	ŏl	il	Ó	0	0	0	8	1
Wilmington	i		ğ	0		0	0	0 2	0	i	1
Winston-Salem	ī					- 4 1	0'		0'		

City reports for week ended Sept. 24, 1938-Continued

cases Cases Desites cases <	State and city	Diph- theria	Inf	hienza	Mea-	Pneu-	Scar- let	Small pox	Tuber-	Ty- phoid	Whoop-	Deaths,
Charleston 0 0 0 0 0 0 1 0 0 1 0	State and city		Cases	Deaths			fever Cases			fever cases	cough cases	CELLSES
Tiorance	South Carolina:						_					
Greanville	Charleston		3									17
Georgia: 7 10 0 2 7 0 5 0 5 Brunswick 6 0 0 0 1 0 0 1 6 Savannah 1 14 1 0 1 0 0 3 1 1 Florida: 1 0 0 0 1 0 0 3 1 1 Miamt 1 0 0 0 1 0 0 0 0 4 1 1 0	Greenville										1 1	12
Brunswick	Georgia:	-		1			-					-
Savannah	Atlanta		110			2						68
Florida: 1 0 0 3 0 9 2 0 3 Miand. 1 0 0 0 0 1 0 0 3 0 9 2 0 3 Ashland. 0 </td <td>Savannah</td> <td></td> <td>14</td> <td></td> <td></td> <td>l ĭ</td> <td></td> <td>ŏ</td> <td>3</td> <td>1</td> <td>1</td> <td>4 28</td>	Savannah		14			l ĭ		ŏ	3	1	1	4 28
Tampa	Florida:	_		1 -	-		-					
Ashland 0 0 0 1 0 </td <td>Miami Tampa</td> <td></td> <td>28 24</td>	Miami Tampa											28 24
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Lexington i	Ashland							0				7
Louisville 0 1 0 1 3 6 0 4 0 1 Tennesse: 0 1 1 0	Covington					0						13
Tennessee: Mamphis			i -									21 75
Memphis		v	•	•			-			-		10
Nash'ille 0 1 0 0 1 0 4 0 1 Alabama: 0 1 0 0 8 1 0 8 0 1 Birmingham 0 1 0 0 0 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 0 1 0 <									. 0			27
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State and city		ngitis, ococcus	Polio- mye-	State and city		ngitis, cococcus	Polio- mye-
	Cases	Deaths	litis cases		Oases	Deaths	litis cases
Massachusetts: Springfield New York: Rochester New Jersey: Camden Pennsylvania: Philadelphia Ohio: Cleveland Illinois: Alton Detroit	0 8 0 1 0 1 0	0 20 0 0 0 0 0 0 0	1 1 0 1 1 1 0	Minnesota: Duluth Minneapolis Missouri: St. Louis North Dakota: Minot District of Columbia: Washington West Virginia: Wheeling Alabama: Montgomery Montana: Great Falls	0 0 0 1 0 0	0 0 1 0 0 0	1 1 0 8 0 1 8

City reports for week ended Sept. 24, 1958-Continued

Encephalitis, epidemic or lethargic.—Cases: New York, 2; St. Paul, 2; St. Louis, 1; Minot, 2; Louisville, 1; Billings, 1. Pellagra.—Cases: Savannah, 3; Birmingham, 1; San Antonio, 1; Los Angeles, 1; San Francisco, 1. Typhus feor.—Cases: Wilmington, N. C., 3; Charleston, S. C., 3; Atlanta, 2; Savannah, 5; Tampa, 3; Mobile, 2; Lake Coarles, 1; Fort Worth, 2; Houston, 3.

FOREIGN AND INSULAR

CZECHOSLOVAKIA

1. 1.

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

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax. Cerebrospinal meningitis Chickenpox. Diphtheria. Dysentery. Influenza. Lethargic encephalitis Malaria.	3 42 176 1,714 11 19 2 962	70 70 1 2 2	Paratyphoki fever Poliomyelitis Puerperal fever Scarlet fever Trachoma Typhoid fever Typhus fever	10 5 19 1, 680 72 333 1	2 10 14 13

FRANCE

Vital statistics—First quarter 1938—Comparative.—The French Ministry of National Economy has recently published the following vital statistics for the first quarter of 1938, with the figures for the first quarter of 1937 for comparison:

	1938	1937		1938	1937
Number of marriages Number of live births Number of stillbirths	54, 639 157, 253 5, 804	55, 001 158, 605 6, 020	Number of deaths Deaths under 1 year of age	192, 937 10, 457	177, 671 11, 60 5

GREAT BRITAIN

England and Wales—Smallpox—1901-1936.—The British Ministry of Health has recently published the numbers of cases of, and deaths from, smallpox for the years 1901 to 1936, reproduced in the accompanying table. The figures relate to civilians only, and include both virulent and non-virulent forms of smallpox. In 1917 the number of cases of smallpox fell to the low figure of 7 and then rose almost uninterruptedly until 1927, when 14,767 cases were reported. The number of cases then declined to 1 in 1935.¹ Since 1920, the majority of the cases have been of the non-virulent type. It is stated that, in 1936, all the cases notified were of the virulent type,

¹ It is stated that the diagnosis in this case was probably incorrect.

which led to the belief that, in many instances, the infection had been introduced from abroad. The report states that all of the outbreaks were checked and finally extinguished by prompt and vigorous action on the part of the health authorities. It also states that experience proved the virulent type of smallpox to be more easily conquered than the mild form, because in the former case the public is alarmed and willing to aid the health authorities in all control measures.

Year	Cases	Deaths	Year	Cases	Deaths
1901 1962 1903 1904 1905 1906 1907 1906 1907 1908 1909 1909 1909 1909 1909 1910 1911 1912 1913 1914	1,980 13,923 7,383 5,766 1,020 1,020 127 22 87 108 295 123 115 64 90	356 2,464 760 567 116 21 10 12 21 19 23 4 10 4 13	1919 1920 1921 1922 1923 1924 1925 1925 1926 1927 1928 1929 1920 1923 1924 1925 1929 1920 1920 1923 1924 1925 1929 1920 1920 1921 1922 1923 1929 1920 1920 1921 1922 1923 1931 1931 1932	294 263 315 973 2,485 5,365 10,146 14,767 12,420 10,967 11,839 5,664 2,038 631	244 30 527 7 13 9 19 477 535 28 9 9 39 28 39 39 39 39 39 39 39 39 39 39 30 30 30 30 30 30 30 30 30 30 30 30 30
1915 1916 1917 1918	149 7 63	16 3 2	1933 1934 1935 1936	179 1 1 12	

NOTE.—The population for England and Wales in 1901 was 15,555,319; in 1920, 37,609,600; in 1936, 49,839,000. ¹ Diagnosis believed to have been incorrect.

CHOLEBA, 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 BEFORTS for September 30, 1933, pages 1759–1773. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REFORTS for the last Friday of each month.

Cholera

China.—During the week ended September 24, 1938, cases of cholera were reported in China as follows: Canton, 4; Hong Kong, 20; Shanghai, 113; Swatow, 2.

India-Rangoon.-During the week ended September 24, 1938, 1 case of cholera was reported in Rangoon, India.

Japan—Fukuyama.—During the week ended October 1, 1938, 3 cases of cholera were reported in Fukuyama, Japan.

Plague

Argentina.—For the period September 1–15, 1938, plague was reported in Argentina as follows: Ingenio Santa Ana, Tucuman Province, 2 cases, 1 death; Aguaray, Salta Province, 1 case, 1 death.

China—Manchuria.—According to information dated August 25, 1938, 17 cases of plague occurred in the Moli Tribe in South Hsingan Province, near Fengpu. Plague has also been reported at Wutaokoutzu in the Kaitung district, where 5 deaths occurred between August 13 and 17. Ten cases of plague with 10 deaths have also been reported in the Li Chin Yu Tribe near Heitimiao in northern Kirin Province between July 29 and August 10, 1938.

Peru.—During the month of August 1938, plague has been reported in Peru as follows: Trujillo, Libertad Department, 1 case; Canete, Lima Department, 3 cases, 2 deaths.

United States—California.—A report of plague infection in Eldorado and San Bernardino Counties, California, appears on page 1848 of this issue of PUBLIC HEALTH REPORTS.

Smallpox

Colombia.—During the month of July 1938, smallpox was reported in Colombia as follows: Departments—Antioquia, 27 cases; Caldas, 57 cases; Cundinamarca, 19 cases; Magdalena, 9 cases; Narino, 8 cases; Tolima, 5 cases, 5 deaths; Valle del Cauca, 2 cases, 1 death. Intendencias and Commissaries, 2 cases, 2 deaths.

Dutch East Indies—Batavia.—During the week ended September 17, 1938, 1 imported case of smallpox with 1 death was reported in Batavia, Dutch East Indies.

Siam.—During the week ended September 24, 1938, 33 cases of smallpox were reported in Siam.

Yellow Fever

Gold Coast-Salaga.-On September 23, 1938, 2 cases of yellow fever were reported in Salaga, Gold Coast.

Sudan (French)—Kouy.—On September 23, 1938, 1 suspected fatal case of yellow fever was reported in Kouy, French Sudan.