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PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

March 27—April 23, 1938

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 ended April 23, the number reported for the corresponding period in 1937, and the median number for the years 1933–37.

DISEASES ABOVE MEDIAN PREVALENCE

Measles.—The number of cases of measles (147,707) reported for the weeks ended April 23 represented a decrease of approximately

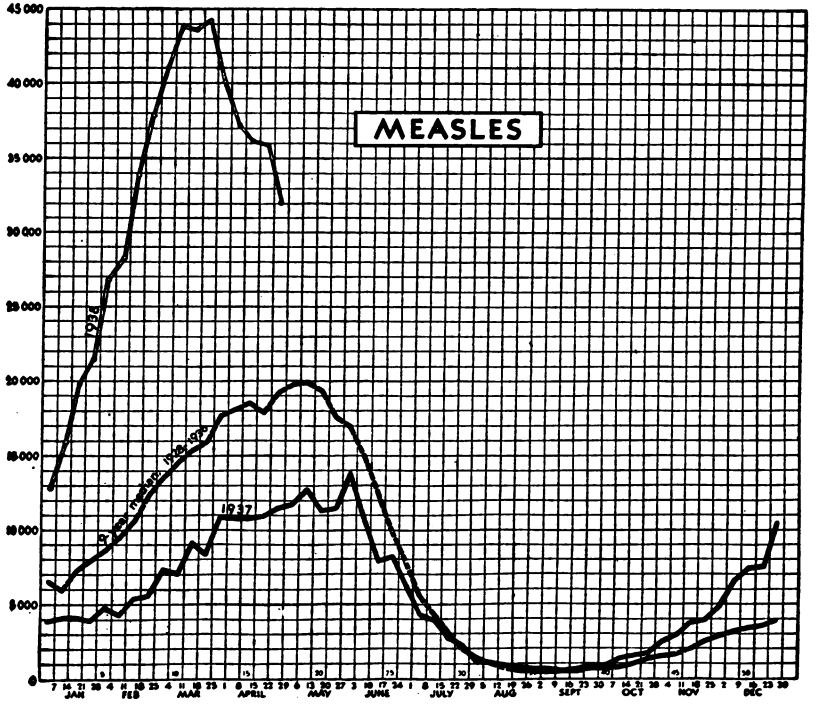

FIGURE 1.—Numbers of cases of measles reported by weeks for the first 17 weeks of 1938, for the year 1937, and for the median weeks of the 9 years 1928–36.

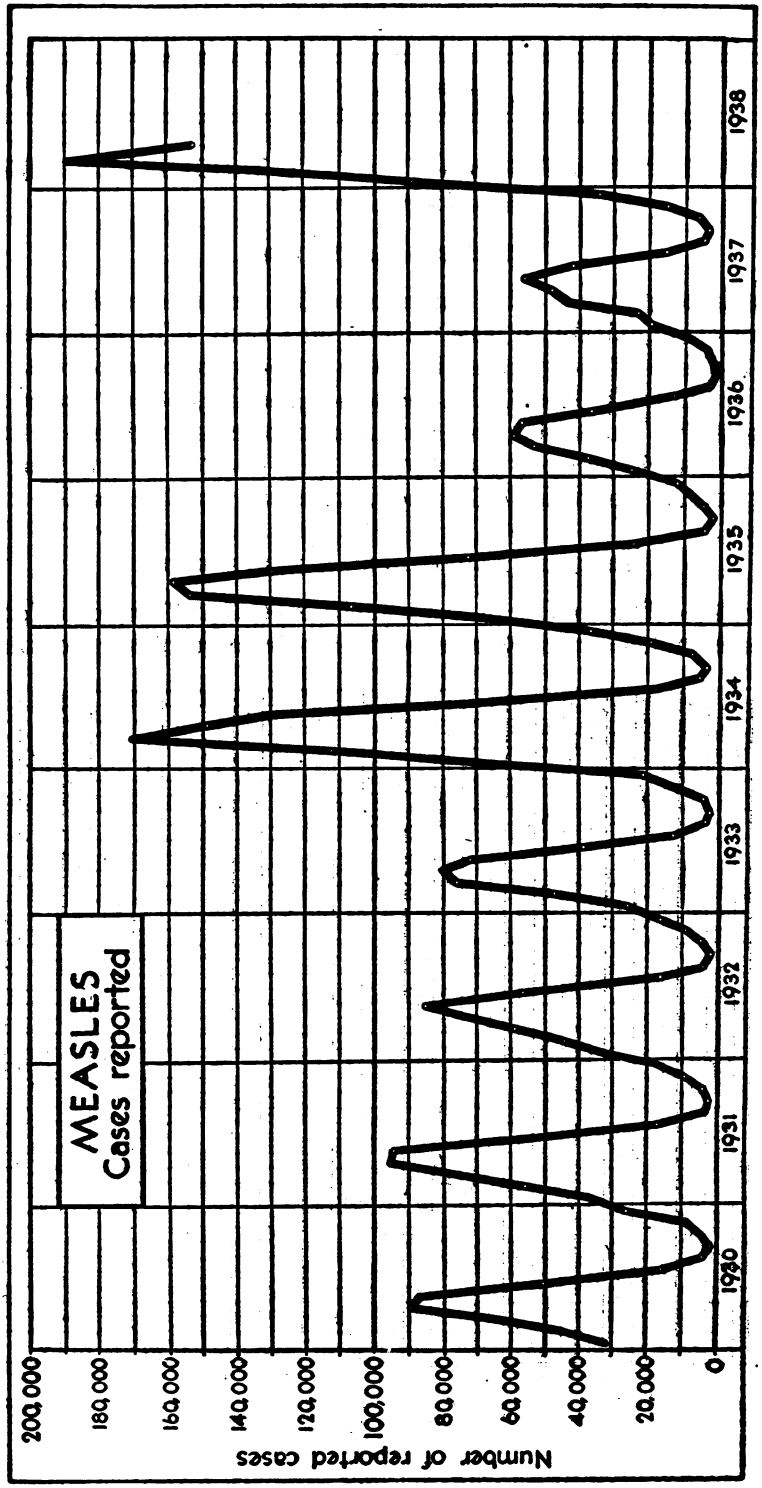

FIGURE 2.—Number of cases of measles reported, by months, January 1930–April 1938.

24,000 cases from the number reported for the preceding 4 weeks. In preceding years the seasonal peak of measles has not generally been reached until the latter part of April or the first of May, but apparently the peak of the current high incidence for the country as a whole was reached during the week ended March 26, the last week of the preceding 4-week period.

The current measles incidence has been the highest on record. While the incidence has declined considerably, the number of cases for the period under consideration is approximately three times that reported for the corresponding period in each of the 2 preceding years, and more than two times the median number reported for the corresponding period during the years 1933–37. In 1934 and 1935 the time of the peak corresponded with the current one, with a total of approximately 132,000 and 142,000 cases, respectively.

While each section of the country reported more than the average incidence for this season of the year, the highest incidence in relation to the 1933–37 median was reported from the East North Central and East South Central regions, where the incidence was more than four times the seasonal expectancy, and from the South Atlantic region, where the number of cases was almost three times the seasonal average.

Smallpox.—The number of cases of smallpox reported for the 4 weeks ended April 23 was 1,882, as compared with 1,443, 878, and 739 for the corresponding period in 1937, 1936, and 1935. The current incidence is the highest on record since 1931, when the number of cases for this period totaled 4,068. The present geographic distribution of smallpox is very uneven. The North Atlantic States are practically free from the disease and the South Atlantic States reported only six cases, which is only about 50 percent of the average incidence for that region, while the States in the West and Mississippi River Basin continue to report a relatively high incidence. Increases over the 1933–37 median range from one and one-third times the median in the West South Central region to more than six times the median in the East South Central region.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—The diphtheria incidence is the lowest on record for this period. The reported number of cases for the 4 weeks ended April 23 was 1,601. This is about 95 percent of the incidence for the corresponding period in 1937 and about 75 percent of the 1933–37 median. In the Mountain and Pacific regions the current incidence stood at about the average seasonal level, but all other regions reported a relatively low incidence.

Number of reported cases of 8 communicable diseases in the United States during the 4-week period Mar. 27–Apr. 23, the number for the corresponding period in 1937, and the median number of cases reported for the corresponding period 1933–37 [1]

Division	Current period	1937	5-year median	Current period	1937	5-year median	Current period	1937	5-year median	Current period	1937	5-year median
	Diphtheria			Influenza [2]			Measles [3]			Meningococcus meningitis		
United States [1]	1,601	1,724	2,193	4,761	14,019	7,139	147,707	45,102	72,322	275	690	659
New England	45	32	64	58	53	64	2,753	6,609	8,997	15	44	13
Middle Atlantic	336	373	394	79	125	125	42,909	18,818	26,117	52	104	104
East North Central	317	320	385	183	1,176	935	59,327	4,753	12,855	35	77	115
West North Central	111	171	231	329	577	555	7,498	575	7,122	19	28	40
South Atlantic	265	278	299	954	3,740	2,447	18,828	6,677	6,677	54	194	108
East South Central	117	111	131	455	2,460	871	6,555	1,494	1,494	62	156	53
West South Central	203	268	326	2,013	4,360	2,142	3,305	3,524	3,524	19	41	41
Mountain	64	52	60	314	270	436	3,777	1,648	1,648	9	28	11
Pacific	143	119	135	376	1,318	493	2,755	1,014	5,429	10	23	23
	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fevers		
United States [1]	72	96	77	22,199	29,478	29,478	1,882	1,443	815	457	443	568
New England	1	1	2	2,406	2,154	1,829	0	1	0	10	20	21
Middle Atlantic	11	8	8	6,845	8,162	8,361	0	1	0	63	58	58
East North Central	9	10	13	6,341	9,638	10,017	508	321	154	61	48	83
West North Central	6	5	5	2,823	5,683	2,450	558	739	271	31	18	20
South Atlantic	8	18	9	946	871	1,052	6	4	12	71	84	125
East South Central	19	15	4	469	415	415	63	5	10	58	56	56
West South Central	13	12	5	671	799	571	196	44	146	117	112	118
Mountain	1	9	2	619	677	677	144	154	96	15	19	17
Pacific	4	18	18	1,139	1,079	1,079	407	174	88	31	28	30

[1] 48 States. Nevada is excluded, and the District of Columbia is counted as a State in these reports.
[2] 44 States and New York City.
[3] 46 States. Mississippi and Georgia are not included.

Influenza.—The influenza incidence continued at a low level, with 4,761 cases reported as compared with 14,019, 31,791, and 6,922 cases for the corresponding period in 1937, 1936, and 1935. The current incidence is about 65 percent of the 1933–37 median (7,139 cases), which is the figure for this period in 1934, a year exceptionally free from influenza. The situation is very favorable in all parts of the country, each section reporting a decrease from the average seasonal expectancy.

Meningitis.—For the current period the number of cases of meningitis totaled 275, as compared with 690, 1,169, and 659 for the corresponding period in 1937, 1936, and 1935. The years 1935, 1936, and 1937, within which period the 5-year median falls (1935) were years of rather high meningitis incidence, and probably a better comparison is with the average (313 cases) for the years 1932–34, the current incidence being less than 90 percent of that figure. In the North Central and Pacific regions the incidence was the lowest on record for this period, while the Middle Atlantic, South Atlantic, West South Central, and Mountain regions reported the lowest incidence since 1924. Meningitis was unusually prevalent in the East

South Central and South Atlantic regions during this period in 1937 and 1936, and the incidence in the East South Central region is now somewhat above the seasonal average.

Poliomyelitis.—Poliomyelitis (72 cases) continued at about the average seasonal level, with the exception of the South Central regions, which reported definite increases over the normal seasonal incidence. The Pacific region has a comparatively low incidence for this season of the year, while in all other geographic areas the cases closely approximated the 1933–37 median.

Typhoid fever.—The typhoid fever incidence (457 cases) was slightly higher than during the corresponding period in 1937, but it was low in relation to the seasonal expectancy. The West North Central region reported a slight excess over the average incidence, and the South Atlantic States reported a very definite decrease; but in all other regions the incidence was about normal for this season of the year.

Scarlet fever.—The incidence of scarlet fever was below normal, with 22,199 cases reported for the 4 weeks ending April 23. More cases were reported from the New England, West North Central, West South Central, and Pacific regions than are normally expected at this season, but in all other areas the reported number of cases was relatively low. The Middle Atlantic and East North Central regions reported the lowest numbers in recent years.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ending April 23, based on data received from the Bureau of the Census, is 11.8 per 1,000 estimated population (annual basis). The current rate is the lowest since 1933, when the average rate for the corresponding period was 11.3. In 1937 and 1936 the rates for this period were 12.8 and 13.3, respectively.

STUDIES ON DENTAL CARIES

I. DENTAL STATUS AND DENTAL NEEDS OF ELEMENTARY SCHOOL CHILDREN[1]

By HENRY KLEIN, *Associate Dental Officer*, CARROLL E. PALMER, *Passed Assistant Surgeon*, and JOHN W. KNUTSON, *Passed Assistant Dental Surgeon, United States Public Health Service*

INTRODUCTION

Dental caries is generally recognized as a physical impairment which exceeds in prevalence all others known to occur in children of the United States. Although the use of the toothbrush, dietary regulation, prophylactic odontotomy, and the application of chemiotherapy (silver nitrate) have been suggested and advocated, no effective

[1] From Child Hygiene Investigations, Division of Public Health Methods, National Institute of Health, United States Public Health Service.

method for the control and prevention [2] of caries has been accepted other than the replacement of the carious tooth structure by chemically stable filling materials. The great importance of the caries problem, particularly from a public health standpoint, stimulates inquiry concerning the possibility of developing, through research, other methods of control which are less expensive, more effective, and better adapted for widespread application by practicing dentists.

The need for the development of such methods has led to a detailed study of dental disease in a representative urban community. This paper, the first in a series, presents the results of a dental survey of essentially the entire elementary grade school population of the community. The findings are interpreted as demonstrating that the magnitude of the caries problem, in grade school children, is of such order as to make difficult its immediate practical handling with existing facilities and knowledge. This identification of the magnitude of accumulated carious defects in school children has led to a detailed analysis of the manner in which dental defects accumulate in the permanent teeth of children of grade school age. The results strongly suggest that a redistribution of dental services is indicated if school children, who represent approximately 15 percent of the population, are to receive adequate and effective dental care.

GROUP SURVEYED AND METHODS

The small city, Hagerstown, Md., whose grade school children were studied is located in the eastern section of the United States and has a population of approximately 30,000 persons (1930). Somewhat over 90 percent of the inhabitants are native white, and of these, 95 percent have native parents. The city contains several small manufacturing and industrial units and includes the usual retail and wholesale commercial establishments. In general, the population is representative of the broad middle range of socio-economic groups in the United States.

Approximately 95 percent of the children of grade school age attend the municipal elementary schools. The enrollment of white children in the first eight grades of these schools was, in October 1936, approximately 4,700. Of this number 4,416 received, in the spring of 1937, complete dental examinations by dental officers in the employ of the United States Public Health Service. The children examined, therefore, include 94 percent of the enrolled elementary school population. The distribution of these children, with respect to age and many other characteristics, may be considered representative of many urban communities in the United States.

[2] Control and prevention as used in this connection is taken to mean the limitation of extension of caries and the prevention of tooth mortality rather than the prevention of the initiation of carious lesions.

The dental examinations were made with number 3 plain mirrors and fine pointed pig-tail explorers under favorable lighting conditions. Observations were made on *all* teeth present in the mouths and, in addition, unerupted and extracted permanent teeth were noted. Pits and fissures in which the explorer caught and which after thorough inspection were not considered definitely carious were noted as separate items and were not counted as caries. Teeth designated as carious were those which showed actual, although frequently small, cavities. The lesions recorded are those which are readily found on careful clinical dental examination. The extent of caries in any single tooth was measured in terms of tooth surfaces involved. When such areas extended from one surface to others, the involved surfaces were counted separately as carious surfaces. Remaining roots were considered as equal to five carious surfaces. Records for filled teeth were made in a similar manner, that is, filled surfaces were considered as past carious surfaces. Full crowns, of which few were encountered, were considered equal to five filled surfaces (five surfaces affected by past caries).

FINDINGS

Preliminary to the presentation of findings it is essential to consider briefly certain constituents which make up the caries problem and the manner in which these affect the measurement of dental status and needs in children. The first of these considerations concerns the numbers and kinds of teeth present in, or absent from, the mouths of the individuals examined. The range of age of elementary school children, approximately 6 through 15 years, covers the period during which the deciduous is being replaced by the permanent dentition. Since entirely different teeth and numbers of teeth are involved in the description of status in older as contrasted with younger children, it is essential that findings be given for specific age and sex groups and given separately for deciduous and permanent teeth. The populations of children and teeth which are utilized for a quantitative description of status and needs are given in table 1.

TABLE 1.—*Numbers of children, numbers of permanent teeth erupted, and numbers of permanent and of deciduous teeth present in the mouth, by age and sex groups among 4,416 elementary school children in Hagerstown, Md.*

Item tabulated	Sex	Age										
		6	7	8	9	10	11	12	13	14	15	All ages
Number of children	Boys	171	197	231	253	270	262	299	267	199	83	2,232
	Girls	156	206	256	240	259	269	297	278	165	58	2,184
Number of permanent teeth erupted	Boys	734	1,537	2,486	3,352	4,436	5,380	7,121	7,023	5,364	2,270	39,703
	Girls	860	1,824	2,987	3,595	4,758	6,120	7,490	7,423	4,533	1,594	41,184
Number permanent teeth present in the mouth	Boys	734	1,532	2,479	3,325	4,366	5,314	7,030	6,885	5,253	2,203	39,121
	Girls	859	1,818	2,973	3,558	4,698	6,049	7,371	7,253	4,420	1,548	40,547
Number deciduous teeth present in the mouth	Boys	2,886	2,794	2,644	2,237	1,610	724	379	121	44	9	13,448
	Girls	2,498	2,729	2,718	1,650	1,123	456	223	81	14	0	11,492

A second major consideration which influences the choice of methods of presenting the findings concerns the fact that dental caries is a cumulative disease. A precise description of status of the disease in a population group demands, therefore, a quantitative evaluation of the complete caries experience. Such an evaluation requires a full consideration of what may be termed *past* and *present* caries. Present caries may be defined as actual carious lesions which are present in the mouth at the time of examination. The term "past caries" may be used in designating those teeth that are filled and those that are missing (extracted) because of caries. For purposes of precision and brevity, the term "DMF" (decayed, missing, or filled) is introduced and used in this paper to designate the complete caries experience. This measurement of complete caries experience is obtained readily for the permanent teeth, since missing permanent teeth in children may be considered, at least for purposes of the present discussion, as the result of severe caries. An equivalent measurement of the complete caries experience in the deciduous teeth is not possible in the data collected since a definite decision may not be made as to whether a missing deciduous tooth has or has not been carious. These latter considerations emphasize again that quantitative facts regarding dental caries in children must be presented separately, and in different ways, for the deciduous and permanent dentitions.

Total caries experience (DMF) in the permanent teeth.—Details of the caries experience in the permanent teeth, expressed in terms of DMF, are given in tables 2, 3, and 4. The first of these tabulations, including the percents of children having one or more permanent teeth classified as DMF, indicates that the disease, dental caries, has attacked one or more permanent teeth in 19.9 percent of 6-year-old girls and 12.3 percent of boys of the same age. As age increases, caries in the permanent teeth becomes more prevalent until at age 14 or 15, approximately 95 percent of the children have one or more DMF permanent teeth. Of the 4,416 grade school children ages 6 through 15 years, 3,156, or 71.5 percent show a history of caries in one or more permanent teeth.

The spread of the carious condition through the population of permanent teeth is shown in table 2, which presents, by age and sex groups, the numbers of erupted permanent teeth which are DMF. Of 39,703 erupted permanent teeth in boys, 5,932, or 15.0 percent are decayed, filled, or missing; of 41,184 erupted permanent teeth in girls, 6,340, or 15.4 percent show evidence of attack by caries; of 80,887 erupted permanent teeth in the 4,416 children, therefore, 12,272 are designated as DMF. Further details of the extension of the caries experience in the population of permanent teeth are shown by the fact (table 2) that a total of 23,753 DMF surfaces are present

TABLE 2.—*Percents of children with 1 or more DMF [1] permanent teeth, numbers of DMF permanent teeth, and numbers of DMF permanent tooth surfaces, by age and sex groups. (4,416 elementary school children, Hagerstown, Md.)*

Item tabulated	Sex	Age										
		6	7	8	9	10	11	12	13	14	15	All ages
Number of children with 1 or more DMF permanent teeth.	Boys	21	57	116	174	213	218	263	229	188	82	1,561
	Girls	31	89	144	174	201	217	260	266	159	54	1,595
Percent of children with 1 or more DMF permanent teeth.	Boys	12.3	28.9	50.2	68.8	78.9	83.2	88.0	85.8	94.5	98.8	69.9
	Girls	19.9	43.2	56.3	72.5	77.6	80.7	87.5	95.7	96.4	93.1	73.0
Number of DMF permanent teeth.	Boys	43	115	255	452	646	722	1,068	1,065	1,012	554	5,932
	Girls	52	178	328	542	663	788	1,111	1,413	986	359	6,340
Number of DMF permanent tooth surfaces.	Boys	50	169	365	826	1,301	1,334	2,094	2,216	2,004	1,191	11,559
	Girls	66	237	470	1,038	1,274	1,424	2,161	2,852	1,850	822	12,194

[1] Decayed, missing, or filled as defined in the text.

TABLE 3.—*Numbers and percents of DMF [1] permanent tooth surfaces contributed by specified corresponding permanent teeth, all ages (4,416 elementary school children, Hagerstown, Md.)*

Corresponding teeth	Number of surfaces		Percent of surfaces	
	Boys	Girls	Boys	Girls
Upper jaw:				
Central incisors	592	605	12.0	12.6
Lateral incisors	400	471	8.1	9.8
Canines	39	30	.8	.6
1st premolars	251	261	5.1	5.4
2nd premolars	244	228	4.9	4.8
1st molars	3,226	2,996	65.2	62.5
2nd molars	195	199	3.9	4.2
All teeth	4,947	4,790	100.0	100.0
Lower jaw:				
Central incisors	87	63	1.3	.9
Lateral incisors	66	41	1.0	.6
Canines	3	5	0	.1
1st premolars	46	55	.7	.7
2nd premolars	176	221	2.7	3.0
1st molars	5,776	6,456	87.4	87.2
2nd molars	438	563	6.9	7.6
All teeth	6,612	7,404	100.0	100.0

[1] Decayed, missing, or filled as defined in the text.

TABLE 4.—*Numbers and percents of DMF [1] permanent tooth surfaces contributed by specified tooth surfaces, all ages, all teeth (4,416 elementary school children, Hagerstown, Md.)*

Specified tooth surfaces	Number of surfaces		Percent of surfaces	
	Boys	Girls	Boys	Girls
Occlusal	4,951	5,264	42.8	43.2
Mesial	2,014	2,088	17.4	17.1
Distal	1,657	1,698	14.3	13.9
Buccal	1,503	1,624	13.0	13.3
Lingual	1,434	1,520	12.4	12.5
All surfaces	11,559	12,194	100.0	100.0

[1] Decayed, missing, or filled as defined in the text.

in the 12,272 DMF permanent teeth. The different kinds of permanent teeth which contribute DMF surfaces are indicated in table 3 which gives the numbers and percents of DMF permanent tooth surfaces by specified corresponding permanent teeth.[3]

Study of this table indicates that certain individual types of teeth contribute more carious surfaces than others. For example, 21,521, or nearly 91 percent, of all DMF surfaces in the permanent teeth of the grade school children are found in the upper and lower first molars, the lower second molars, and the upper central and lateral incisors. More specifically, 64 percent of the caries experience occurring in the upper jaw and 87 percent of that in the lower jaw is contributed by the first molars. Additional details regarding the location of carious defects in the permanent teeth are shown in table 4 which gives the numbers and percents of specific tooth surfaces which are affected. The fact may be derived from this table that 43 percent of the 23,753 DMF surfaces are found in occlusal surfaces, 31 percent in distal and mesial surfaces, and 26 percent in buccal and lingual surfaces.[4]

DENTAL NEEDS

For purposes of the present discussion the term dental needs is restricted to those professional requirements which are directly and obviously the immediate consequence of dental caries. Under this definition tooth defects which make up dental needs are contributed by present unfilled cavities in the deciduous and permanent dentitions and by missing permanent teeth. In the deciduous teeth, the measurement of dental needs is limited, for reasons given previously, to the consideration of unfilled carious teeth actually present in the mouth at the time of the examination.

Table 5 presents the details of the status of unfilled carious teeth in the deciduous dentition. In the 4,416 children observed, 2,331, or 53 percent, have one or more unfilled carious deciduous teeth. Of a total of 24,940 deciduous teeth present in the mouth, 9,943 have unfilled cavities which involve 21,191 tooth surfaces. Nearly 40 percent of the deciduous teeth present, therefore, contain unfilled cavities.

In the permanent dentition the conditions which, together, constitute dental needs as defined in this report, include all teeth with clinical histories which indicate untreated defects due to caries. The conditions which fall into this category are actual unfilled cavities, remaining roots, and missing permanent teeth. The status of these

[3] The data are presented for corresponding teeth, since caries occurrence is bilaterally almost equal.

[4] Data given here must not be interpreted as measuring the relative susceptibility of the various teeth and surfaces to attack by caries. In explanation of this caution it need only be pointed out, as an example, that the first molar teeth contribute a large proportion of the total caries experience primarily because these teeth have erupted into the mouths of nearly all of the 4,416 children examined while the second molars, for instance, have erupted into the mouths of a much smaller proportion of children.

TABLE 5.—*Status of teeth requiring fillings in the deciduous dentition, by age and sex groups (4,416 elementary school children, Hagerstown, Md.)*

Item tabulated	Sex	Age										
		6	7	8	9	10	11	12	13	14	15	All ages
Number of children having 1 or more carious deciduous teeth requiring fillings.	Boys	139	171	214	227	220	138	86	25	17	6	1,243
	Girls	126	176	221	198	172	101	60	28	6	-----	1,088
Percent of children having 1 or more carious deciduous teeth requiring fillings.	Boys	81.3	86.8	92.6	89.7	81.5	52.7	28.8	9.4	8.5	7.2	55.7
	Girls	80.8	85.4	86.3	82.5	66.4	37.5	20.2	10.1	3.6	-----	49.8
Number of carious deciduous teeth requiring fillings.	Boys	862	958	1,198	1,042	741	354	165	44	24	8	5,396
	Girls	728	1,013	1,158	751	501	226	119	44	7	-----	4,547
Number of carious deciduous tooth surfaces requiring fillings.	Boys	1,781	2,003	2,647	2,278	1,575	711	344	104	60	25	11,528
	Girls	1,374	2,138	2,595	1,571	1,057	517	283	105	13	-----	9,663

several components of the problem of dental needs is summarized in table 6. The section of the table giving the numbers and percents of children having one or more unfilled cavities in the permanent teeth shows that as age advances the proportion of children having such cavities increases. Of the 4,416 children examined, a total of 2,617, or nearly 60 percent, require one or more fillings in the permanent teeth. A total of 7,989 permanent teeth require the treatment of 11,802 tooth surfaces by fillings. Additional details of the status of dental needs shows that a total of 330 permanent teeth have remaining roots which require extraction and that a total of 1,219 permanent teeth are missing (extracted). When these are combined (9,538 teeth), it is indicated that 76.6 percent of all teeth giving a clinical history of caries must be considered in a broad discussion of the problem of dental needs in the permanent dentition.

TABLE 6.—*Status of teeth requiring fillings in the permanent dentition, by age and sex groups (4,416 elementary school children, Hagerstown, Md.)*

Item tabulated	Sex	Age										
		6	7	8	9	10	11	12	13	14	15	All ages
Number of children having 1 or more carious permanent teeth requiring fillings.	Boys	19	56	102	148	178	180	223	190	162	76	1334
	Girls	27	81	124	146	151	169	199	206	135	45	1283
Percent of children having 1 or more carious permanent teeth requiring fillings.	Boys	11.1	28.4	44.2	58.5	65.9	68.7	74.6	71.2	81.4	91.6	59.8
	Girls	17.3	39.3	48.4	60.8	58.3	62.8	67.9	74.1	81.8	77.6	58.7
Number of carious permanent teeth requiring fillings.	Boys	40	106	204	346	456	484	671	630	688	406	4033
	Girls	41	151	254	386	419	462	644	799	569	231	3956
Number of carious permanent tooth surfaces requiring fillings.	Boys	56	141	264	545	709	727	1028	996	962	594	6022
	Girls	47	134	327	601	641	638	917	1215	847	363	5780
Number of permanent teeth having only remaining roots.	Boys	-----	-----	3	8	18	11	30	22	37	31	160
	Girls	-----	-----	1	18	8	15	35	39	31	23	170
Number of children having 1 or more missing (extracted) permanent teeth.	Boys	-----	5	5	22	49	50	63	88	78	40	400
	Girls	1	6	10	27	42	51	76	107	68	28	416
Percent of children having 1 or more missing (extracted) permanent teeth.	Boys	0.0	2.5	2.2	8.7	18.1	19.1	21.1	33.0	39.2	48.2	17.9
	Girls	0.6	2.9	3.9	11.3	16.2	19.0	25.6	36.5	41.2	48.3	19.0
Number of permanent teeth missing (extracted).	Boys	-----	5	7	27	70	66	91	138	111	67	582
	Girls	1	6	14	37	60	71	119	170	113	46	637

Summarizing the dental needs for both dentitions, it is found that approximately 10,000 deciduous and 8,000 permanent teeth contain unfilled cavities. Defects in these 18,000 teeth affect approximately 21,000 deciduous and 12,000 permanent tooth surfaces. In addition to the total of 33,000 defective untreated surfaces, 7,745 permanent tooth surfaces have been lost because of severe caries.

Fillings in the permanent and deciduous teeth.—The data previously presented indicate that all except a very small segment of a representative elementary school population shows attack by caries in the permanent teeth and that approximately three-fourths of all teeth attacked show no objective evidence of treatment. Tables 7 and 8 give the amount and extent of fillings in the permanent and the deciduous dentitions. Among the 4,416 children, 436 boys and 547 girls have one or more permanent teeth filled. Of 5,932 DMF permanent teeth in boys and 6,340 DMF permanent teeth in girls, 23.4 percent and 29.2 percent, respectively, have been filled. Of 11,559 DMF surfaces in boys, 1,860 surfaces are filled. Of 12,194 DMF surfaces in girls, 2,422 are filled. Approximately 16 and 20 percent of the DMF surfaces in boys and girls, respectively, are replaced by fillings. In the deciduous dentition, 270 teeth and 396 surfaces are filled in 111 boys; 248 teeth and 353 surfaces are filled in 102 girls. It is thus apparent that in all of the children examined less than one-fourth of the permanent tooth surfaces which have been affected by caries experience show objective evidence of reparative treatment, and that a much smaller proportion of carious deciduous teeth present in the mouth show a similar evidence of treatment.

TABLE 7.—*Status of filled teeth in the deciduous dentition, by age and sex groups (4,416 elementary school children, Hagerstown, Md.)*

Item tabulated	Sex	Age										
		6	7	8	9	10	11	12	13	14	15	All ages
Number of children having 1 or more filled deciduous teeth.	Boys	17	20	15	15	26	13	3	1	1	-----	111
	Girls	15	17	16	15	22	11	3	3	-----	-----	102
Percent of children having 1 or more filled deciduous teeth.	Boys	9.9	10.2	6.5	5.9	9.6	5.0	1.0	0.4	0.5	-----	5.0
	Girls	9.6	8.3	6.3	6.3	8.5	4.1	1.0	1.1	-----	-----	4.7
Number of filled deciduous teeth.	Boys	51	54	41	37	57	24	4	1	1	-----	270
	Girls	45	43	45	32	54	21	4	4	-----	-----	248
Number of filled deciduous tooth surfaces.	Boys	85	77	67	46	78	35	6	1	1	-----	396
	Girls	67	62	64	38	79	33	6	4	-----	-----	353

An analysis of DMF surfaces in the permanent teeth of those boys and girls who have one or more fillings in these teeth shows (table 8) the presence of 3,244 DMF surfaces in 436 boys and 4,537 DMF surfaces in 547 girls. Since it is shown also that a total of 1,860 and 2,422 permanent tooth surfaces are filled in these boys and girls, respectively, it is revealed that reparative treatment has been supplied for 57.3 percent of the DMF surfaces in this particular group of boys

and 53.4 percent of the DMF surfaces in this group of girls. This analysis of the distribution of reparative treatment indicates that a relatively small group of children (983) receive all of such treatment given; yet only one-half of their needs are supplied. Of all the children examined, 3,160 have one or more permanent teeth showing a history of caries. Of these, 983 have one or more filled teeth, the remaining 2,197 children exhibit no evidence of treatment for their carious defects. It may be stated, therefore, that carious defects in the permanent teeth are treated fairly adequately in one-fifth and are entirely neglected in one-half of the 4,416 grade school children.

TABLE 8.—*Status of filled teeth in the permanent dentition by age and sex groups (4,416 elementary school children, Hagerstown, Md.)*

Item tabulated	Sex	Age										
		6	7	8	9	10	11	12	13	14	15	All ages
Number of children having 1 or more filled permanent teeth.	Boys	2	2	22	32	49	68	89	86	64	22	436
	Girls	4	10	27	45	68	85	110	118	58	22	547
Percent of children having 1 or more filled permanent teeth.	Boys	1.2	1.0	9.5	12.6	18.1	26.0	29.8	32.2	32.2	26.5	19.5
	Girls	2.6	4.9	10.5	18.8	26.3	31.6	37.0	42.4	35.2	37.9	25.0
Number of filled permanent teeth.	Boys	3	3	45	86	128	183	320	307	228	86	1,389
	Girls	10	22	65	127	219	272	364	463	225	85	1,852
Number of filled permanent tooth surfaces.	Boys	3	3	51	109	155	227	466	426	311	109	1,860
	Girls	14	24	71	164	297	364	479	600	293	116	2,422
Percent of DMF[1] permanent teeth that are filled.	Boys	7.0	2.6	17.6	19.0	19.8	25.3	30.0	28.8	22.5	15.5	23.4
	Girls	19.2	12.4	19.8	23.4	32.1	34.5	32.8	32.8	25.4	23.7	29.2
Percent of DMF[1] permanent tooth surfaces that are filled.	Boys	5.1	1.8	14.0	13.2	11.9	17.0	22.3	19.2	15.5	9.2	16.1
	Girls	21.2	10.1	15.1	15.8	23.3	25.6	22.2	21.0	15.8	14.1	19.9
Number of DMF[1] permanent tooth surfaces among children having 1 or more filled permanent teeth.	Boys	3	5	80	138	268	349	784	753	627	237	3,244
	Girls	15	25	89	249	420	612	908	1,186	724	309	4,537
Percent of DMF[1] permanent tooth surfaces filled among children having 1 or more filled permanent teeth.	Boys	100.0	60.0	63.8	79.0	57.8	65.0	59.4	56.6	49.6	46.0	57.3
	Girls	93.3	96.0	79.8	65.9	70.7	59.5	52.8	50.6	40.5	37.5	53.4

[1] Decayed, missing, or filled as defined in the text.

ANALYSIS

The data just presented offer quantitative support for the frequently encountered observation that defects due to dental caries in childhood constitute a health problem of major proportions. The problem of caring for these defects is of such complex character and magnitude as to justify attempts to clarify the more important issues involved. A clarification of one aspect of the problem may be facilitated by an analysis of the manner in which carious defects develop and accumulate in the permanent teeth.

Caries in the permanent teeth of children 6 years of age may be viewed as an accumulation of defects which have been added each year up to age 6. Carious defects at 7 years of age may be considered equal to those which have accumulated at age 6, plus the *increment* of new defects which have appeared between the 6th and 7th years of age. For example, in the 6-year-old girls examined during the survey, 42.3

DMF surfaces per 100 girls had accumulated in the permanent teeth. In the 7-year-old girls, 115.0 DMF surfaces per 100 girls were observed. The difference in caries experience, 72.7 DMF surfaces per 100 children, represents the increase or increment of defects which appeared between the 6th and 7th years of age. From this point of view, a total yearly increment of defects for the entire elementary school population may be visualized as equal to the sum of annual increments for each age-sex group from 6 through 15 years. Such a total yearly increment may be interpreted as a reasonably accurate measurement of new carious defects which appear each year in the population of school children. Estimates [5] of the increments for each separate age-sex group, and the total yearly increment for the entire school population, are shown in table 9. The results indicate that 5,859 carious tooth surfaces represent the estimated increment of new defects in the permanent teeth which may be expected to appear each year in a representative group of 4,416 grade school children. This annual increment, it is postulated, would account for the finding (table 2) of 23,753 DMF surfaces in the elementary school children at the time of the survey. Expressed in somewhat more general terms the analysis indicates that a representative group of grade school children have an average of nearly five and one-half carious permanent tooth surfaces per child (23,753 DMF surfaces in 4,416 children) and that this accumulation of defective surfaces is maintained, as children enter and leave the school group, by a yearly increase of approximately one and one-third new carious surfaces per child per year (approximately 6,000 new DMF surfaces per year in 4,416 children).[6]

According to the perspective of this analysis, it is clearly implied that the basic problem of giving care for carious defects in the permanent teeth is the problem of caring for the yearly increments of defects. In a parallel manner, and more fundamentally, the problem of controlling the initiation of dental defects is the problem of eliminating these yearly increments.

On the basis of the analysis just made, it becomes pertinent to relate the yearly increment of new defects to an analogous estimate of the amount of care now given each year for carious defects in the permanent teeth of the children. Such an estimate of dental care may be obtained by the same method of analysis as was used to determine the total yearly increment of new defects. Basic data which may be used for the calculation of an annual increment of filled surfaces are given in table 6. The analysis, although not shown in detail here, indicates that the filling of approximately 1,000 permanent tooth sur-

[5] It is appreciated by the authors that some influence in depressing the tendency towards new caries, or extensions of caries, may be contributed by the process of filling carious teeth.

[6] Insofar as the children studied may be considered representative of grade school children in the United States generally, and insofar as this estimate of the total yearly increment may be considered accurate, these figures furnish the basis for estimation of dental requirements for the permanent teeth of that segment of the population which attends the elementary schools.

faces per year in the mouths of 4,416 children would result in the finding of 4,282 filled surfaces at the time of the survey. Since, as was shown in table 9, approximately 6,000 permanent tooth surfaces develop caries each year it becomes apparent immediately that the filling of permanent tooth surfaces is being accomplished at a rate which is about one-sixth of the rate at which the defects are accruing. Identification of this disparity between the rate of development of defects and the rate of placement of fillings largely explains, in quantitative terms, the existence of the present accumulated dental *needs* of the children studied and leads to the conclusion that if such an accumulation of untreated defects in the permanent teeth is to be avoided in the future some provision should be made to give elementary school children (in the form of fillings alone) approximately six times the amount of service that they now receive

TABLE 9.—*Data for the calculation of the increment of new carious (DMF)* [1] *surfaces which may be expected to arise annually in the permanent teeth of 4,416 elementary school children in Hagerstown, Md.*

Item tabulated	Sex	Age										
		6	7	8	9	10	11	12	13	14	15	All ages
Observed number of DMF[1] surfaces present in the mouth, per 100 children.	Boys	34.5	85.8	158.0	326.5	481.9	509.2	700.3	830.0	1,007.0	1,434.9	-----
	Girls	42.3	115.0	183.6	432.5	491.9	529.4	727.6	1,025.9	1,121.2	1,417.2	-----
Difference between age specified and previous age in observed number of DMF[1] surfaces present in the mouth, per 100 children.	Boys	34.5	51.3	72.2	168.5	155.4	27.3	191.1	129.7	177.0	427.9	-----
	Girls	42.3	72.7	68.6	248.9	59.4	37.5	198.2	298.3	95.3	296.0	-----
Expected number of new carious (DMF)[1] surfaces between age specified and previous age (item tabulated directly above times number of children in specified age groups).	Boys	59.0	101.1	166.8	426.3	419.6	71.5	571.4	346.3	352.2	355.2	2,869
	Girls	66.0	149.8	175.6	597.4	153.8	100.9	588.7	829.3	157.2	171.7	2,990

[1] Decayed, missing, or filled as defined in the text.

Information afforded by the above analysis may be considered in the light of professional dental facilities available in the community. At the time of the survey there were 32 dentists practicing in the city. It may be assumed that some of these dentists allocate more time for the treatment of children than others. However, a number of useful purposes may be achieved by expressing dental service in terms of an equal distribution of work by the entire group of 32 practitioners. For example, on the basis of the estimate of 1,000 permanent tooth surfaces filled per year, it appears that, on the average, each dentist fills approximately 30 permanent tooth surfaces each year in the mouths of the children. On the basis of the estimate that approximately 6,000 new carious surfaces appear in the permanent teeth each

year, it may be postulated that the care of the yearly increment of defective surfaces would require the filling of approximately 190 surfaces per year per dentist in order to *prevent the accumulation* of untreated defects. An attempt to care, during the course of one calendar year, for the present accumulation of 11,802 defective surfaces [7] in the permanent teeth of 4,416 children (table 6) would require the placement of fillings in approximately 370 surfaces by each dentist. In connection with this last estimate, if an attempt were made to care for the present accumulation of defects, *plus* those which it is estimated would accrue during the year, it may be postulated that over 500 permanent tooth surfaces would need to be filled during the course of the year by each dentist.

Perhaps additional information of value may be derived from this analysis of professional dental services if the estimates are expressed in terms of *time* requirements. For this purpose, it seems reasonable to make the assumption that 1 hour,[8] on the average, of professional time would be required to care adequately for each defective permanent tooth surface. In addition, it may be satisfactory for present purposes to assume that each dentist works a total of 1,800 [9] hours per year.

The acceptance of these two assumptions makes possible the following general estimates of dental services for the permanent teeth of grade school children:

1. The filling of 500 surfaces, which represents the estimated number of defective surfaces that are present now plus those that would be expected to accrue during 1 year, means that nearly 30 percent of the professional services of the community would be required during 1 full year if an attempt were made to provide the present elementary school population with complete dental treatment in the form of fillings for the permanent teeth.

2. The filling of 30 surfaces per year per dentist, which is the estimate of service now given the children, means that somewhat less than 2 percent of the total professional time of the dentists practicing in the community is devoted to the filling of permanent teeth in that 20 percent of the population which attends the elementary schools.

3. The filling of 190 permanent tooth surfaces per year per dentist, which is equivalent to the estimated increment of defects which appear each year, means that approximately 10 percent of the professional time available in the community would be needed to prevent

[7] No provision being made to extract remaining roots or to restore missing permanent teeth.

[8] It is recognized that the location of surfaces to be filled may considerably influence the time required for placing fillings or for providing other indicated treatment. Since the immediate purpose of this discussion is chiefly the dissection of the problem of supplying dental needs due to caries, the estimates of professional services required are arbitrarily stated. Thus, if more or less than 1 hour is estimated for the treatment of each carious surface, the total services required would be proportionately changed.

[9] This estimate of working hours per year is used by Strusser in a recent publication (New York State Journal of Dental Hygiene, 8: 51 (1938)).

the accumulation of defects in the permanent dentition of the school children.

This summary, although obviously based on arbitrary estimates of professional time requirements, provides some clarification of the problem of supplying dental services for the permanent teeth of school children.[10] First, it seems necessary to conclude that the magnitude of the present accumulation of dental needs in the permanent teeth alone is of such order as to make exceedingly difficult its immediate practical handling with existing facilities. Second, it seems reasonable to expect that provision should be made for handling the yearly increment, the care of which is estimated to require approximately 10 percent of the professional time of the dental practitioners of the city. Third, care for the yearly increase in new defects appears to involve a six-fold increase over the time now given to the filling of permanent teeth in the school children.

SUGGESTIONS FOR PROVIDING MORE NEARLY ADEQUATE DENTAL CARE

The analysis given in the preceding statements may be used as the basis for suggesting procedures by means of which it *may* be possible to give more nearly adequate dental treatment for the permanent teeth of grade school children. One such procedure may be predicated on two assumptions: First, that it is highly desirable to *prevent the accumulation* of untreated defects; and, second, that each dentist of the community be willing to assume a proportionate share of the professional labor involved.

On the basis of these assumptions, the following suggestions are offered in connection with certain aspects of the operation of a rational plan [11] designed for the specific purpose of preventing the accumulation of dental defects in the permanent teeth of grade school children. During the initial year of operation of the plan complete dental treatment would be supplied for all carious defects in the permanent teeth of all children in the first grade of the elementary schools. During the following year, complete care would be provided for caries appearing in the permanent teeth of all new first-grade children and for the increment of new carious defects appearing in the second-grade pupils who were treated the preceding year. During each of the succeeding third, fourth, fifth, sixth, seventh, and eighth years, accumulated defects in first-grade children entering the school popula-

[10] An equivalent analysis of the problem of supplying care for the deciduous teeth is difficult, since in many instances the short life expectancy of these teeth does not justify extensive reparative dental work. It is appreciated, of course, that a considerable amount of professional service is necessary if the deciduous teeth are to be given adequate dental care.

[11] This plan includes no provision for the treatment of accumulated defects in the *present* group of elementary school children nor does it include a provision for the treatment of defects in the deciduous teeth. Furthermore, the plan is not designed to control those unknown yet basic factors which give rise to carious defects.

tion during these respective years would be treated and new increments of defects, contributed by each grade treated in earlier years, would be given dental care. It is apparent, therefore, that the suggested plan provides that complete dental care be given for each group of new first-grade children plus treatment for new increments of caries appearing in the permanent teeth of the children, as they progress from lower to higher school grades. After the operation of the plan for 8 years, all grades of the elementary school population will have received, systematically, treatment for yearly increments of defects.

According to the records of the survey, approximately 400 permanent tooth surfaces in the first-grade children are estimated to require repair. During the initial year of operation of the plan, fillings would need to be placed in these children's teeth at a rate of 12 permanent tooth surfaces per dentist. During the second year, the placement of fillings in 30 permanent tooth surfaces by each dentist would be required to care for the carious defects in new first-grade pupils and for the estimated 600 new carious permanent tooth surfaces which would be expected to appear in the second-grade pupils. By the end of the eighth year, services equivalent to the placement of fillings in 200 surfaces per year per dentist would be necessary to care for all of the yearly increments of defects appearing in the children of all eight grades.

If it be assumed that 1 hour is required for the filling of each carious surface, it may be estimated that the order of only two-thirds of 1 percent of the available professional dental services of the community would need to be utilized in giving complete care for defects in the permanent teeth of the first-grade pupils. During the second year 1.67 percent of the professional services would be required to care for the new first-grade pupils and the second-grade children. Each successive year of operation of the plan would involve the use of a slightly higher percentage of the professional facilities until the eighth year, when 10 percent of the existing professional services would be required to care for the annual increment of the entire grade school population. Thus, one feature of this plan is that the demands on the existing dental services are gradually increased, beginning at a very minimum (two-thirds of 1 percent) and gradually increasing until the eighth year, when 10 percent is required to care for that 15 percent of the population which attends the elementary schools.

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EXPERIMENTAL VANADIUM POISONING IN THE WHITE RAT[1]

By ESTHER PETERSON DANIEL, *Associate Nutrition Chemist, Bureau of Home Economics, U. S. Department of Agriculture,* and R. D. LILLIE, *Surgeon, U. S. Public Health Service, Division of Pathology, National Institute of Health*

As part of a study conducted for the purpose of determining the nutritional significance of vanadium, observations were made of the tolerance level of this element in the diet and symptomatology and pathology of vanadium fed animals.

Sodium metavanadate was incorporated into the stock ration of rats in amounts calculated to bring the level of vanadium to 12.5, 25, 100, 200, and 400 parts per million, respectively. Upon subsequent chemical analysis,[2] the sodium metavanadate (C. P. grade) was found to be 92 percent pure, the impurity being practically all absorbed CO_2. Thus, the actual amounts of vanadium in the diets fed were 11.5, 23, 92, 184, and 368 parts per million, respectively. The stock ration into which the vanadate was mixed consisted of whole wheat 650 parts, whole milk 250, meat scraps 100, and sodium chloride 13 parts. These diets were fed *ad lib* to healthy young rats 3 to 4 weeks old, weighing between 34 and 50 grams. Five of these young animals originally receiving the diet containing 23 p. p. m. of vanadium were transferred at the end of 12 weeks to the ration supplemented with 368 p. p. m. of vanadium. Control animals, taken from the same litters, were maintained at the same time on the unsupplemented ration. Food records were kept in all cases.

The diets supplemented with higher levels of vanadium proved very disagreeable to the animals and their normal food intake was

[1] Vanadium is used as a mordant in the dye industry and more extensively as a hardener in the manufacture of certain steels. The important ores are vanadinite, carnotite, and descloizite.

Anemia, cachexia, respiratory irritation, diarrhea, and emaciation have been reported as due to vanadium poisoning in workers in these industries. There is little information as to the frequency of industrial poisoning. (Bull. U. S. Bur. Lab. Stat. No. 582.)

Very few previous studies have been made on the toxic action of vanadium salts, and most of these have been confined to pharmacological and gross anatomical findings.

This paper gives certain basic data as to tolerance levels for ingestion of vanadium salts and as to histologic effects of acute and subacute poisoning.—ED.

[2] The vanadium was reduced to the quadrivalent state with SO_2 followed by expulsion of this gas with CO_2 and titration of the hot solution with $KMnO_4$.

markedly affected. In order to determine whether the restricted growth of animals on these diets was due entirely to inanition caused by a lowered food consumption or in part to the effects of the vanadium itself, animals, paired as to litter, weight, and sex with the vanadium-fed rats, were given the normal ration without vanadium and the food intake was so controlled that both groups received the same quantity.

The results of experiments in which the vanadium was incorporated in the ration at different levels are summarized in tables 1 and 2.

TABLE 1.—*Response of rats to different amounts of vanadium incorporated in the ration as $NaVO_3$*

Vanadium in diet	Number of rats		Initial age	Length of test period [1]	Gain in weight [2]		Deaths		Total vanadium ingested [2]		Observations
	Male	Female			Male	Female	Male	Female	Male	Female	
P. p. m.			*Weeks*	*Weeks*	*Gm.*	*Gm.*			*Mg.*	*Mg.*	
11.5	1	2	3-4	9	217	116	0	0	8.3	6.1	Apparently normal. No significant evidence of stimulation by V. All lived until end of experiment.
23	9	5	3-4	10	192	114	0	0	17.5	14.4	Apparently normal. 3 males and 2 females placed on 368 p. p. m. V at end of 12 weeks. Others killed at end of 10th week.
92	5	1	3-4	10	138	66	0	0	67.3	43.0	Slight symptoms of poisoning. 1 male and 1 female placed on stock ration at end of 12th week for later feeding of V by stomach tube. 4 killed at end of 16th week.
184	7	2	3-4	10	107	86	0	0	107.5	91.4	All but 1 male showed definite symptoms of poisoning. 2 males and 2 females killed at end of 10th week. 4 males killed at end of 16th week. 1 male retained for further experiment.
368	5	4	3-4	[3] 10	−14	−11	6th day	6th day	4.1	4.2	Marked symptoms of poisoning. 2 males and 2 females lived until end of 10th week.
					−6	−11	12th day	6th day	17.3	5.2	
					−9	42	7th week		69.2	134.6	
					24	54			133.6	144.3	
					21				139.0		
	[4] 3	[4] 2	16	[3] 10	−100	−68	7th week	7th week	122.2	108.2	Marked symptoms of poisoning. 1 male and 1 female lived until end of 10th week.
					−103	−10	9th week		185.4	251.1	
					−62				325.0		
0	8	7	3-4	10	202	117	0	0	0	0	Normal controls.

[1] The length of the test period considered in computing average gains in weight and total vanadium ingested. Some of the animals were kept on the experiment for a longer time for various reasons, as indicated under the heading "Observations".
[2] Averages except in the case of animals receiving 368 p. p. m. V, in which case individual values are given.
[3] Or until time of death.
[4] Animals formerly receiving 23 p. p. m. V in the diet for 12 weeks.

On vanadium levels of 11.5 and 23 p. p. m. all of the animals appeared normal throughout the experiment. Higher levels of vanadium caused loss in weight and gross pathological symptoms, the severity of which progressed with the greater doses of the element. This is

shown by the fact that on vanadium levels of 92 and 184 p. p. m. the animals exhibited definite symptoms of vanadium poisoning, but all of them lived to the end of the experimental period while 5 of the 9 young rats given the ration containing 368 p. p. m. of vanadium died. Three of the five older animals transferred to this diet after previously receiving 23 p. p. m. of vanadium for 12 weeks also succumbed (table 1).

TABLE 2.—*A comparison of the average weight gains of paired rats on normal and on vanadium supplemented rations after the ingestion of the same quantities of food*

[All quantities given in grams]

Food intake	11.5 p. p. m. V group and controls				23 p. p. m. V group and controls				184 p. p. m. V group and controls				368 p. p. m. V group [2] and controls			
	Male (1 pair) [1]		Female (2 pairs)		Male (6 pairs)		Female (3 pairs)		Male (2 pairs)		Female (2 pairs)		Male (2 pairs)		Female (2 pairs)	
	On V diet	On control diet	On V diet	On control diet	On V diet	On control diet	On V diet	On control diet	On V diet	On control diet	On V diet	On control diet	On V diet	On control diet	On V diet	On control diet
50	24	21	24	20	21	18	18	15	12	13	14	13	0	7	5	9
100	48	42	44	42	42	37	35	32	21	27	27	27	5	21	14	16
150	68	59	53	57	55	56	45	43	34	40	36	39	6	29	23	26
200	85	77	68	68	70	71	58	53	48	51	47	51	-----	-----	30	40
300	123	106	88	90	108	106	78	71	70	86	55	68	-----	-----	38	52
400	152	136	104	108	136	136	95	84	82	110	68	82	-----	-----	-----	-----
500	182	166	115	116	157	158	111	103	102	120	-----	-----	-----	-----	-----	-----
600	202	181	-----	-----	177	175	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----
700	214	-----	-----	-----	192	191	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----

[1] The control rat could not be induced to eat as much food during the same period of time as the animal receiving the vanadium supplement.

[2] 5 pairs could not be included in this summary because of the deaths of 3 males and 2 females on the vanadium supplemented diet. None of the control animals died, indicating that starvation was not the primary cause of death.

Table 2 shows a comparison of the average weight gains of the vanadium-fed animals and their paired controls at different levels of food intake. From these data it may readily be observed that, with greater ingestion of vanadium, the animals made smaller gains for a given level of food intake. This evidence, together with the fact that none of the paired control rats died, indicates that inanition was not the primary cause of death of animals receiving diets containing 368 p. p. m. of vanadium.

Proescher, Seil, and Stillians (*4*) have reported that one or two administrations of tolerated doses of vanadium either *per os* or injection produced a rapid increase in body weight. With a continuation of these doses they found that the animals began to lose weight, although this loss was never sufficient to bring the weight below that recorded for the animal at the beginning of the experiment. Several French investigators, cited by Lyonnet, Martz, and Martin (*3*) have reported on the therapeutic effects of small doses of vanadium in a number of diseases. In the amounts which were fed in the present

experiments, there is no significant evidence to indicate a stimulating effect of small quantities of this element. For a time it was felt that the appetites of the rats on the low levels of vanadium were more pronounced, and on occasions it was difficult to get the paired control animals to eat the same quantity of food. However, experiments on a larger number of animals would be necessary before the slight differences which were obtained in these experiments could be considered significant.

The incorporation of the supplement into the ration has certain distinct advantages, chief of which is that it offers the most natural introduction of the material for a nutritional consideration. However, since the amount of supplement ingested depends upon the quantity of food eaten, and no two animals eat the same amount, the results are difficult to summarize. Furthermore, in cases such as the present one, in which a disagreeable ration limits food intake, the quantity of supplement ingested falls below the desired level. For these reasons, solutions of sodium metavanadate (found by analysis to have a purity of 92 percent (see footnote 2, p. 764)), and sodium orthovanadate (not analyzed) were fed to a number of rats by stomach tube. The solutions were made up (account being taken of the determined purity of $NaVO_3$) so that 1 cc contained 2 mg of vanadium, and daily doses of 2 and 4 mg, respectively, of vanadium were given to a total of 83 rats, ranging in weight from 40 to 350 gm. Pathological examinations were made of a large number of these animals; and to avoid post mortem changes in the tissues, many of them were killed in a moribund condition. Since these animals were not expected to live more than a few hours longer, their records have been summarized with those which were left to die from the poisoning.

Table 3 gives a summary of the results obtained when solutions containing 2 or 4 mg of vanadium were administered daily by means of a stomach tube to rats of different weights. Of the 83 animals in these experiments, only one (a male receiving sodium ortho-vanadate) lived to the end of the 10-week experimental period. Another male died during the sixth week, two others during the fourth week, while another lived until the third week of the experiment. All of the remaining animals either died or would have succumbed (had they not been killed in moribund condition) some time between the second and thirteenth day. Most of them died on the second or third day.

TABLE 3.—*Effects of 2 and 4 milligrams of vanadium administered daily to rats of different weights by means of stomach tube*

Form of vanadium administered	Weight groups	Number of rats		Time of death[1]				Total vanadium[2] administered				Percentage of initial weight lost			
				Male		Female		Male		Female		Male		Female	
		Male	Female	Range	Average	Range	Average	Range	Average	Range	Average	Range	Average	Range	Average
Aqueous solution $NaVO_3$	*Grams*			*Days*	*Days*	*Days*	*Days*	*Mg*	*Mg*	*Mg*	*Mg*	*Pct.*	*Pct.*	*Pct.*	*Pct.*
	0-100	5	8	3- 4	3	2- 4	3	4.0- 8.0	6.0	4.0- 8.0	5.5	20-25	22	8-24	20
	100-200	[2] 13	26	2- 5	3	2-13	3	5.5-20.0	11.2	8.0-36.0	11.0	7-26	14	2-19	12
	200-300	[3] 19	2	2- 8	3	2	2	8.0-28.0	10.6	8.0	8.0	8-28	16	6- 9	7
	300-350	4	0	3-11	7	-----	-----	12.0-38.0	24.5	-----	----	16-29	22	-----	-----
Na_3VO_4	0-100	[4] 4	2	3- 4	3	3- 6	4	6 - 8	7.3	4 -16	10	21-26	23	20-35	28

[1] In a number of instances this summary includes animals which were killed in moribund condition for pathological examination.

[2] 2 from this group were very irregular and were not included in the summaries. 1 died during the fourth week after a total vanadium intake of 64 mg and a gain of 7 gm, the other died during the sixth week after a total vanadium consumption of 84 mg and a gain of 4 gm.

[3] 1 from this group died during the fourth week after a total vanadium intake of 82 mg and 25 percent loss in weight. A second died during the third week after consuming a total of 82 mg of vanadium and losing 16 percent in weight.

[4] 1 from this group was killed the tenth week after taking 130 mg of vanadium and gaining 190 gm in weight.

On an average it required approximately 6 mg of vanadium given daily in 2-mg doses to cause the death of animals weighing less than 100 gm, and about twice this amount to exceed the tolerance level of those weighing 100 to 300 gm. The heavier animals, 300 to 350 gm, lived for a longer period, during which they received a greater total quantity of vanadium. The loss of weight in this latter group averaged about 22 percent and was very similar to the percentage weight loss in the young animals. Those weighing from 100 to 200 gm and from 200 to 300 gm averaged a smaller percentage weight loss, although in all groups there was great individual variation.

It is customary to determine toxicity by injection of the material under investigation instead of administering it *per os*. The latter method is more difficult to control and gives such variable results that it is generally considered unsuitable. However, from the standpoint of a nutritional problem it is desirable to introduce the substance orally. Again, repeated doses given over a period of time may act differently from single massive doses and the repeated ingestion of small amounts more nearly approaches the way in which an animal naturally receives harmful substances in its food. Proescher, Seil, and Stillians (*4*) reported the lethal dose of sodium ortho-vanadate for rats injected subcutaneously to be 50–60 mg V_2O_5 per kilogram (2.8 to 3.4 mg V per 100 gm); of ammonium metavanadate to be 20 to 30 mg V_2O_5 per kilogram (1.1 to 1.7 mg per 100 gm); and of sodium vanadate 10 to 20 mg V_2O_5 per kilogram (0.6 to 1.1 mg V per 100 gm). They found the toxic dose of ammonium metavanadate

and sodium ortho-vanadate for rabbits injected intravenously to be 1.5 to 2 mg V_2O_5 per kilogram (0.43 to 1.12 mg V per 100 gm) and 2 to 3 mg V_2O_5 per kilogram (1.12 to 1.68 mg V per 100 gm), respectively. Franke and Moxon (*1*) observed that intraperitoneal injections of sodium metavanadate caused the death of 75 percent of their animals (young, healthy rats weighing between 125 and 175 grams) within 48 hours when administered in doses equivalent to 0.4–0.5 mg of the element per 100 grams and that toxic symptoms were produced by feeding (*2*) in amounts of 50 parts per million of sodium metavanadate. Priestley (*5*) found the fatal dose of the pentoxide administered subcutaneously in rabbits in the form of sodium ortho-vanadate to be between 9.18 and 14.66 mg per kilogram (0.5 to 0.8 mg V per 100 gm). From a comparison of these observations with those of the present study there is evidence that vanadate given orally is better tolerated than intravenous or intraperitoneal injections.

After oral administration of vanadium to a human subject, Proescher, Seil and Stillians (*4*) observed that the elimination of this element took place quickly and quantitatively; about 87.6 percent was passed through the feces, and the remainder was voided in the urine. From these observations it appears that only about 12 percent of the vanadium given orally is ever absorbed, a possible explanation for the apparently increased tolerance of oral doses over those administered by injection.

The effects of oral ingestion of vanadium are not cumulative as evidenced by a comparison of the total vanadium intake of the various animals shown in tables 1 and 3. The daily dose of vanadium for those fed by stomach tube was sufficient in most cases to exceed the tolerance level, and death of the animal resulted in a very short time. On the other hand, the daily quantity of vanadium which the animals received as a part of their food mixture was a tolerated amount. However, after a period of several weeks many of the rats in this latter group had ingested a total quantity of vanadium far in excess of the amount which produced death in the animals fed by stomach tube. Had the toxic effects of the element been significantly cumulative, these animals could not have tolerated any such total quantity as many of them did with few or no outward signs of poisoning. It was also observed that rats poisoned by toxic levels of vanadium in the diet over a long period of time very soon appeared normal again when placed on the stock diet. Subsequently they showed no difference in reaction to large doses of vanadium from other animals of approximately the same weight that had never received this element.

Acute symptoms of vanadium poisoning appeared almost immediately in rats receiving vanadium solutions by stomach tube. These animals appeared extremely nervous, violently clawed their cages, and rubbed their noses in evident distress. On the second day, those

rats receiving the larger doses of vanadium (4 mg daily) showed a hemorrhagic exudate from the nose and marked diarrhea, which continued to grow worse until death. Frequently there was a hemorrhagic discharge from the intestine. Often paralysis of the hind legs occurred. After labored respiration and a series of convulsions, death usually resulted in these animals on the second or third day.

Chronic symptoms were observed in animals receiving vanadium as a part of their food mixture. They were substantially the same as the acute symptoms, although much less marked in degree. Since toxic levels of vanadium definitely retarded appetite, the severity of these symptoms was restricted by a reduced food intake and at times would almost completely disappear only to reappear when a state of inanition once more compelled an increased food intake. This state of inanition superimposed on the condition occasioned by the poisoning left extremely weakened, emaciated animals which generally died in a state of coma.

Examination of the animals immediately after death regularly revealed a stomach, cecum, and intestinal tract greatly distended with gas. Very frequently there was evidence of inflammation in the ileum, often accompanied by a number of ulcers ranging in diameter from 1 to 2 mm. The spleen appeared abnormally small; otherwise there were no outstanding gross abnormalities observed.

Priestley *(5)*, in 1875, described visceral and intestinal congestion in dogs, cats, and rabbits, sometimes with focal mucosal hemorrhage and occasionally small injected intestinal ulcers. Heimberger *(6)* also noted polypnea, lassitude, and cramps and soiling of mice parenterally injected with $NaVO_3$ or $H_4V_6O_{17}$, followed by intestinal congestion and bloody stools at autopsy.

Tissues from 57 rats were examined in the course of this study, together with similar tissues from 9 rats on the same diets which had received no vanadium salts. Most of the animals died, or were killed when moribund, 2 to 4 days after the first daily dose of sodium vanadate. Nine were taken 5 to 12 days after the first daily dose, while 5 lived 3 to 8 weeks.

Tissues were fixed in Orth's fluid and hardened in alcohol, also in 10 percent formalin for study of fatty changes. The following organs were studied routinely: Brain and spinal cord, often spinal ganglia, bone marrow of tibia and vertebrae, spleen, liver, kidney, adrenal, stomach, small and large intestines, pancreas, heart, lung, and testicle or ovary and adnexa. Orth-fixed tissues were sectioned in paraffin and stained with iron chloride hematoxylin and picrofuchsin and with alum hematoxylin and eosinate of polychrome methylene blue. Spleen sections were also stained for iron by the Perl-Abbott ferrocyanide procedure. Bones were decalcified briefly in 5 percent formic acid, which allowed satisfactory marrow staining. Frozen sections

of heart, liver, kidney, and adrenal were stained with alum hematoxylin and Sudan IV and with Nile blue sulphate, and were also examined under polarized light.

In 8 control animals and 1 rat that died 30 minutes after its first dose of sodium vanadate the proventriculus and fundus of the stomach were normal. One control rat and 10 vanadium rats showed purulent inflammation of the proventriculus characterized by the formation of intraepithelial vesicles and pustules leading sometimes to ulceration and often accompanied by subjacent polymorphonuclear infiltration and edema. In 42 vanadium rats the proventriculus was normal. The fundus often showed a little eosinophil leucocyte infiltration of mucosa and submucosa near the proventricular border, both in vanadium and in control rats. This was pronounced in only 8 vanadium rats and in 1 control. Some congestion of superficial vessels in the fundus mucosa was noted in 9 vanadium rats, all from the 2- to 4-day period.

The incidence of the purulent proventriculitis seen does not appear to be significantly greater than in the control series.

One of the most interesting findings in the animals dying after short periods of administration is an acute desquamative enteritis. The villi are moderately to intensely congested, their stroma sometimes contains numbers of leucocytes, and the columnar epithelial cells on their tips are partially separated, rounded, and evidently desquamating. Sometimes suppurative ulceration of the tips of the villi is seen, but more often small pyogenic ulcers appear overlying the agminated lymphoid follicles. When present in the sections, these follicles quite regularly show germinal center hyperplasia with rather marked accumulation of free and phagocytosed nuclear debris during the acute stages. There is a more or less copious surface exudate composed of desquamated epithelial cells, leucocytes, clumps of pus, and blood in decreasing frequency in the order mentioned. Table 4 shows that this enteritis is commonest and most severe in animals dying after 2 to 4 days of vanadium administration.

TABLE 4.—*Incidence of enteritis*

Day of death	Number of rats	Normal	Slight	Moderate	Severe	With ulcers
Second to fourth	40	6	4	10	20	7
Fifth to twelfth	8	5	1	2	0	0
Thirteenth to eighty-second	5	2	1	1	1	0
Controls	10	9	1	0	0	0

Dowdeswell *(7)* noted only congestion of the intestinal mucosa.

The colon is often normal, sometimes shows slight to moderate congestion of the mucosa, infrequently some rounding and desquama-

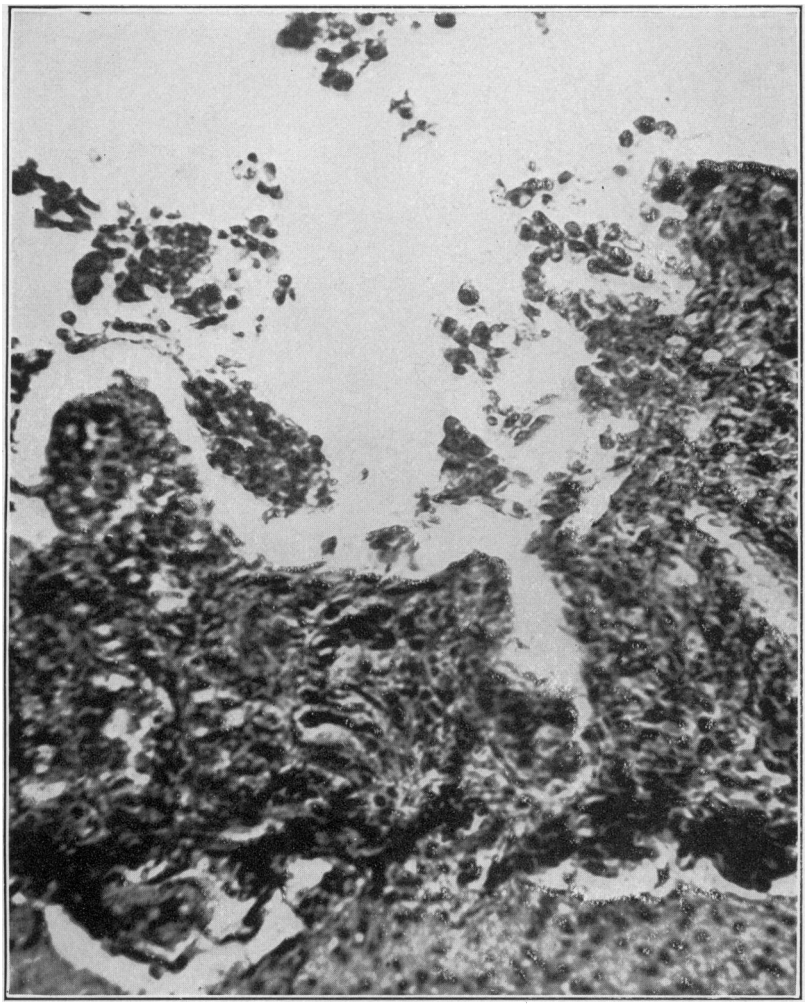

FIGURE 1.—Mucosa of small intestine; epithelial desquamation on villi. (Approx. × 300.)

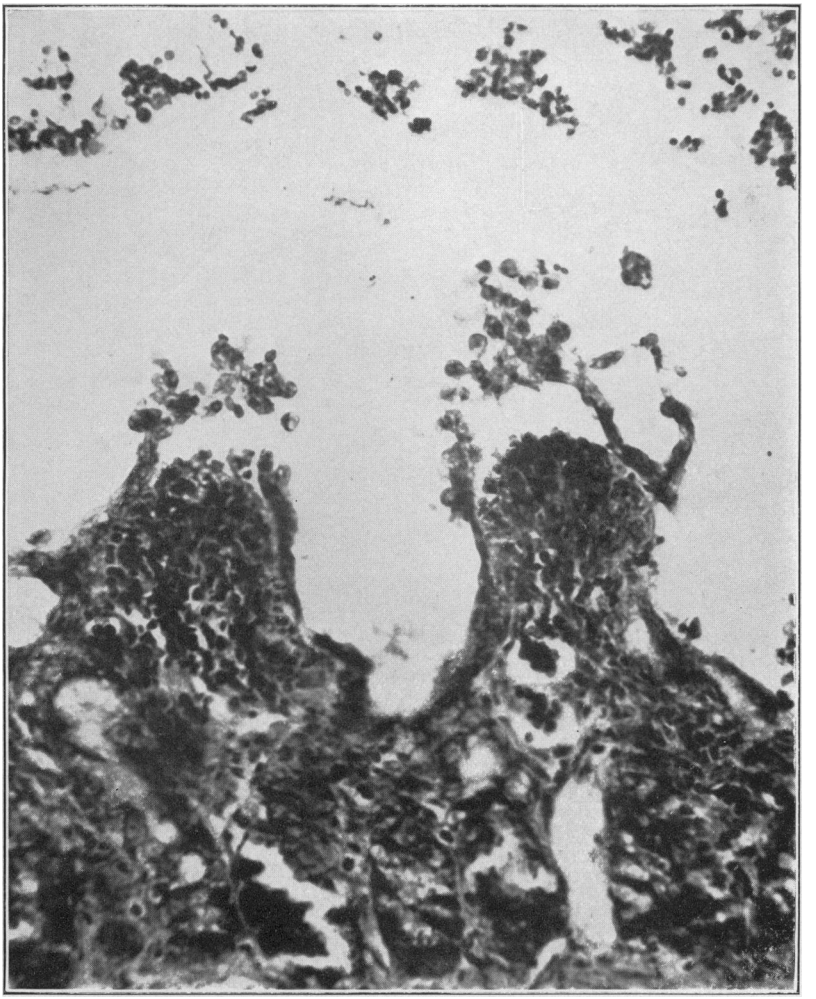

FIGURE 2.—Mucosa of small intestine; epithelial desquamation, mixed purulent, sanguinous and epithelial exudate. (Approx. × 200.)

tion of the surface epithelium, and often some increase in mucus secretion.

In the liver the two significant findings were congestion and the appearance of fine droplets of neutral fat in the liver cells. Congestion was more marked in the centers of the lobules and most frequent in animals dying after 2 days, decreasing sharply after the third day. Fatty changes in the liver cells were variable in intensity and extent. Fine fat droplets were often confined to the peripheral part of the cytoplasm, extending to involve the entire cytoplasm but not the nucleus. In many animals fatty changes were localized in the periportal zones, in others in the centers of the lobules and in others in irregularly distributed areas. Severity of these changes was greatest on the third day.

TABLE 5.—*Incidence of congestion and fatty metamorphosis in the liver by days*

Day of death	Number of rats	Normal, fat-free	Congestion				Fatty metamorphosis			
			−	±	+	++	−	±	+	++
Second	25	0	4	5	12	4	4	9	6	6
Third	12	0	2	6	3	1	1	3	4	4
Fourth to sixth	8	2	3	2	1	0	0	2	2	2
Seventh to twelfth	5	0	3	0	2	0	0	3	2	0
Thirteenth to eighty-second	6	1	3	1	1	0	1	1	3	0
Control	10	8	1	1	0	0	1	0	1	0

Dowdeswell has also noted a fatty metamorphosis in the liver after administration of vanadium salts in various animals. This was accompanied by congestion of capillaries, decrease in liver pigment, and apparently by karyolysis of liver cells with granular cytoplasmic degeneration (in carnivores).

The pancreas was studied in 26 vanadium-treated and 4 control rats and was normal in all.

The heart muscle showed an inconstant cloudy swelling, usually slight to moderate in grade, more frequent and more pronounced in animals dead 2 and 3 days after beginning vanadium treatment than later. Muscle fibers were often cloudy and contained fine eosinophilic granules in their cytoplasm, sometimes totally, more often partially, obscuring the cross striations. In the majority the cross striations were quite distinct at 300 X magnification. Severity of this change did not vary much with the stage of the intoxication, but was definitely greater than in control material. The presence of usually few fine fat droplets in the muscle fibers was noted in 4 vanadium-treated and 1 control rat, a not materially different frequency.

In about 70 percent of the rats dead in 2 to 4 days the lungs showed more or less marked congestion, with hemorrhages into the alveoli in a minor proportion. In the 5- to 12-day period, congestion is less frequent and less marked when present, and after 13 days it is slight or

absent. The control rats showed no pulmonary congestion. Noteworthy perivascular lymphocyte infiltration is probably less frequent than in normal rats. Changes such as purulent bronchitis, abscesses, peribronchial lymph follicle hyperplasia, and intrafollicular phagocytic activity appear to be at least no more frequent than in the control rats.

Renal changes are generally slight, are at their maximum in the 2-day group, and, though decreasing thereafter in frequency and intensity, persist throughout. They consist in swelling and finely granular degeneration of the epithelium of the convoluted tubules, with formation of fine basal droplets of largely neutral fat in a variable proportion of the tubules, more often in the proximal group, and in a decreasing proportion of rats with longer duration of the intoxication. In a few rats a few doubly refractile lipoid crystals were found. In many rats, particularly in the later stages, many basally striated, brush bordered tubules were present. In the control series some rats showed finely granular changes in part, but not all, of the convoluted tubules, but fatty changes were regularly absent. More severe degenerative changes did not occur at any stage.

The adrenals show a quite marked reduction in the fat and lipoid content of the cortex, moderate amounts being present in a minority of the animals in the 2-day group, and very little thereafter. In the 2- and 3-day groups most of the rats show a more less pronounced congestion of the inner part of the adrenal cortex, with focal hemorrhage in about one-third of the congested cortices. Some chromaffin persists in the medulla in some animals of the 2-day group, while it is absent thereafter.

Testis and epididymis were examined in 31 vanadium-treated and 5 control rats. They were generally normal with active spermatogenesis. Acute desquamative degeneration was found in 3 experimental rats and 1 control rat.

Ovaries and adnexa were studied in 17 vanadium-treated and 5 control rats. Adnexa were normal in all. In about half of the rats in the 2-to 4-day period corpora lutea were more or less congested, and one showed focal hemorrhage in lutein tissue.

Skeletal muscle was generally normal in both experimental and control rats.

Splenic follicles show an average decrease in size, most marked in the 5- to 12-day period, but the presence of germinal center proliferative activity is less altered, showing a little decrease in the 3- to 4-day period only. Nuclear fragmentation and phagocytosis of nuclear debris by follicle reticulum cells is slight or absent in control rats, while in many vanadium-treated rats it is quite prominent, particularly after 2 and 3 days, less later.

The blood content of the spleen pulp is quite definitely decreased in rats succumbing after 2 to 12 days of vanadium treatment. Later there seems to be an increase toward normal. The lymphocyte content of the spleen pulp is definitely reduced after 2 days, and more so in the 3- to 12-day period. At the same time there is a relative increase in prominence of pulp reticuloendothelium and a moderate to marked hemosiderosis (see table 6).

TABLE 6.—*Hemosiderin content of spleen pulp*

Day of death	Number of rats	−	Trace	±	+	++	+++
Second	25	0	1	2	4	11	7
Third to fourth	15	0	0	0	6	5	4
Fifth to twelfth	9	0	0	1	1	2	5
Thirteenth to eighty-second	5	0	0	2	1	0	2
Control	10	1	3	1	4	1	0

The bone marrow is usually congested after 2 days of vanadium treatment, less often after 3 days, and infrequently thereafter. Fat content of the upper tibial marrow is somewhat increased in animals treated 3 days or longer. In the 2- to 4-day period there is a fairly pronounced decrease in numbers of metamyelocytes and polymorphonuclear leucocytes, sometimes with the appearance of moderate numbers of myeloblasts. Later maturation of leucocytes is increased but does not reach normal. Similarly the numbers of eosinophil granulocytes is reduced, on the average, in the early period, and a less marked reduction in number of normoblasts is noted. Numbers of megakaryocytes are quite variable and apparently little altered. Numbers of tissue mast cells are seen in the marrow of the tibial shaft, sometimes very numerous, sometimes none. Variations in their numbers cannot be correlated with the vanadium poisoning.

The brain and spinal cord showed an inconstant, usually moderate, capillary congestion, particularly of the gray substance, which was most frequent in the 2- to 4-day period and decreased in frequency thereafter. Less often tigrolysis of nerve cells in stem ganglia of the brain and anterior horns of the cord was present, again more frequently in the 2- to 4-day period. Swelling and vacuolation of nerve cells was an infrequent finding in either brain or cord, and was usually seen in the early period.

Spinal ganglia were examined in 31 vanadium-treated and 6 control rats and were normal in all.

TABLE 7.—*Congestion and cell degeneration in central nervous system*

BRAIN

Vanadium treatment	Normal	Congestion				Tigrolysis stem ganglia			Nerve cell vacuolation			Total
		−	±	+	++	−	±	+	−	±	+	
2 days	9	0	5	8	3	8	5	3	13	2	1	25
3–4 days	4	0	5	6	0	10	0	1	10	0	1	15
5–12 days	4	1	1	3	0	2	2	1	5	0	0	9
13–82 days	2	0	2	0	1	3	0	0	3	0	0	5
Controls	10	0	0	0	0	0	0	0	0	0	0	10
Total	29	1	13	17	4	23	7	5	31	2	2	64

SPINAL CORD

Vanadium treatment	Normal	Congestion			Tirgolysis			Nerve cell vacuolation			Total	Spinal ganglia normal
		−	±	+	−	±	+	−	±	+		
2 days	12	1	3	10	5	3	4	12	1	1	26	15
3–4 days	6	2	0	8	7	0	1	10	0	0	16	11
5–12 days	6	0	1	1	1	1	1	2	0	0	8	3
13–82 days	4	1	0	0	0	0	1	0	0	1	5	2
Controls	9	0	0	0	0	0	0	0	0	0	9	6
Total	37	4	4	19	13	4	7	24	1	2	64	37

SUMMARY

Sodium metavanadate in amounts to give 11.5, 23, 92, 184, and 368 parts per million, respectively, of vanadium in the ration of rats produced toxic symptoms at the three highest levels. The animals receiving 11.5 and 23 p. p. m. showed no gross symptoms of poisoning. Deaths occurred in the group given 368 p. p. m. of vanadium.

Doses of 2 to 4 mg of vanadium administered daily by means of stomach tube as solutions of sodium metavanadate or sodium orthovanadate caused acute poisoning and death of rats weighing between 40 and 350 grams. Animals under 300 grams usually died sometime between the second and fourth day, while the heavier animals lived for a slightly longer period.

There was no evidence of a cumulative effect from vanadium ingestion. Provided the daily tolerance level was not exceeded, many animals receiving a total quantity of vanadium far in excess of the lethal dose lived for weeks, some in an apparently normal condition.

Symptoms accompanying acute vanadium poisoning are immediate nervous reactions indicative of intense distress, hemorrhagic exudate from the nose and intestines, marked diarrhea, often paralysis of the hind legs, labored respiration, and a series of convulsions followed by death. Chronic symptoms were much the same as the acute, although less marked. With higher levels of vanadium the food intake of the rats decreased and a state of starvation superimposed

on the condition of chronic poisoning frequently left extremely weakened, emaciated animals which generally died in a state of coma.

Pathologically, acute sodium vanadate poisoning in rats produces an acute desquamative enteritis, with congestion of villi and some leucocyte exudation; congestion and mild fatty degeneration of the liver; congestion and sometimes focal hemorrhage in the lungs; slight parenchymatous and often fatty degeneration of the renal convoluted tubules, more severe in the proximal group; congestion, often hemorrhage and lipoid decrease in the adrenal cortex; and chromaffinolysis in the adrenal medulla. The spleen is reduced in size; its follicles are small and show karyorrhexis and phagocytosis of nuclear debris; the blood and lymphocyte content of the pulp is reduced, and there is usually a more or less pronounced hemosiderosis. The bone marrow shows an early congestion and a decrease in maturation of leucocytes, sometimes with myeloblast increase. The brain and cord often show congestion in the early stages, less often tigrolysis in stem nuclei and anterior horn cells.

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THE INFLUENCE OF NONBREEDING AND FOSTER NURSING UPON THE OCCURRENCE OF SPONTANEOUS BREAST TUMORS IN STRAIN C_3H MICE [1]

By H. B. Andervont, *Senior Biologist*, and W. J. McEleney, *Laboratory Aide*, *United States Public Health Service*

The occurrence of spontaneous mammary gland tumors in breeding females of strain C_3H has been recorded in an earlier paper (*1*), in which the finding was reported that the mice have a high incidence of such tumors at an average age of 8 to 9 months. The present report deals with the occurrence of mammary gland tumors in nonbreeding

[1] From the Office of Cancer Investigations, U. S. Public Health Service, Harvard Medical School, Boston, Mass.

females of the same strain. The study was undertaken for two reasons: First, to determine the influence of pregnancy and lactation upon the incidence and the time of appearance of mammary tumors in mice of this strain; and, second, to ascertain the incidence of such tumors in nonbreeding females of the strain before using them as material for investigations in which it is desirable to eliminate any variables introduced by pregnancy.

The report also includes the results, to date, of a foster nursing experiment in which mice of strain C_3H were employed. The findings are presented here for comparison with the incidence of breast tumors in breeding and nonbreeding females of the same strain.

REPORTS OF OTHER INVESTIGATORS

The occurrence of mammary tumors in nonbreeding female mice has received attention from a number of investigators. Lathrop and Loeb (*7*), in 1913, came to the conclusion that breast cancer occurred in a larger number of breeding than nonbreeding mice, and that the tumors arose at a later age in the nonbreeding animals. Of special interest is their observation that the number of mammary cancers which arose in nonbreeding mice varied in different strains. In a later paper (*8*) the same authors again stated their conclusion as follows: "Breeding increases the tumor incidence in mice and makes the tumors appear at a lower age." Slye (*15*), in an abstract of a paper read before the American Association for Cancer Research, stated that "mammary neoplasms are about as common in unmated females as in the mated, and in this series they occur at a rather earlier age." Lynch (*9*) reported the breast tumor incidence in two strains of mice: In strain 1194 there was an incidence of 65 percent in breeding and 20 percent in nonbreeding, while in her line of Bagg albinos there was an incidence of 28 percent in the breeding and of 4 percent in the nonbreeding. Cori (*5*) found that "strain 3" mice showed a breast cancer incidence of 94 percent in breeding at an average age of 9 months and of 78.5 percent in nonbreeding at an average age of 14 to 15 months. Marsh (*10*) reported similar findings in "strain 3" breeding and nonbreeding mice.

Murray (*11*), in a comprehensive study of strain D (dilute brown) mice which have been inbred since 1909, found that the average tumor age in breeding females was 10.6 months. He mentioned 1,318 breeders which developed breast tumors and 934 which did not. These figures indicate a 58.52 percent tumor incidence for this strain. In a later paper (*13*) with Little, the breast tumor incidence in nonbreeding strain D mice was found to be 50.84 percent at an average age of 16.7 months (according to table II in their report). Murray and Little (*14*) also found that strain D, "after 20 years of inbreeding was developing tumors of the breast in 80 to 90 percent of the breeding

females," and Murray (*12*) has recently recorded the tumor incidence as 65 to 100 percent in breeding females and about 50 percent in the nonbreeding females. Korteweg (*6*), in a study of the same strain of mice which was under observation in Holland, stated "there is but little difference in frequency or date of the development of cancer between dil. brown females which have and those which have not had litters." Bittner (*2, 3*) concluded from his study of strain A mice, which have been inbred since 1921, that the breeding females exhibited a breast tumor incidence of 83.2 percent at an average age of 11.5 months and the nonbreeding females showed a breast tumor incidence of but 4.5 percent at an average age of 18.5 months.[2]

Suntzeff, Burns, Moskop, and Loeb (*16*) have also determined the breast tumor incidence of breeding and nonbreeeding females of strains D and A. For strain D they reported an incidence of 51.5 percent in breeding and 6.4 percent in nonbreeding at an average age of 9.9 months and 13.5 months, respectively. For strain A they recorded an incidence of 43.1 percent for breeding and 4.5 percent for nonbreeding at an average age of 12.6 months and 14.7 months, respectively. Of interest to this report are their observations of strain C_3H mice, in which they found an incidence of 60.8 percent in breeding and 2.5 percent in nonbreeding at an average age of 10.9 months and 9 months, respectively. Their publication is, apparently, the only previous report on the breast tumor incidence in strain C_3H nonbreeding females, and in this connection they concluded that, "In strain C_3H the incidence of tumors in nonbreeding mice was very low." It should be pointed out that in their table I, dealing with nonbreeding mice, is a column in which are included "Mice without tumors," and under this heading is included the "Average age living or dead." It is possible that all the mice had not lived their span of life before publication of the table.

From the preceding review, it is clear that the various investigators are not in agreement as regards the relative tumor incidence in breeding and nonbreeding female mice. Those who are inclined to believe that the tumor incidence is greater in the breeding than in the nonbreeding females attribute the difference to the added stimulation the mammary glands receive during pregnancy. The majority of the authors are agreed that the average age in which breast tumors appear is greater in nonbreeding than in breeding mice. The striking difference in the incidence of tumors in the nonbreeding mice of highly inbred strains, such as strain D (over 50 percent), and strain A (4.5 percent) is evidence of a variation in susceptibility according to the difference in strain.

[2] The average tumor age of nonbreeding females was kindly supplied by Dr. Bittner in a personal communication.

BREAST TUMOR INCIDENCE IN STRAIN C_3H NONBREEDING AND BREEDING FEMALE MICE

In a single experiment 117 female mice of strain C_3H were separated from males at the time the litters were weaned. They were kept under the same conditions as the breeding females but in different cages. Five died without tumor at the ages of 6.5, 13, 14, 18, and 18.5 months, respectively. All the remaining animals (112, or 95.7 percent) developed spontaneous mammary gland tumors at an average age of 11.5 months. The results obtained in mice of different generations are presented in table 1, where they can be compared with the tumor data of breeding mice of the same generation. The nonbreeding mice of the F_{14} and F_{15} generations compare favorably with the breeding mice so far as tumor incidence is concerned, but in both generations the average age at which tumors appeared was higher in the nonbreeding than in the breeding. It may be concluded that in strain C_3H mice pregnancy hastens the appearance but does not influence the incidence of spontaneous breast tumors.

TABLE 1.—*Summary of all breeding and nonbreeding females in completed generations F_9 to F_{15} of strain C_3H mice*

Generation	Total number of mice	Number that died without tumor	Number that developed spontaneous breast tumor	Percent that developed tumor	Average age at which tumor appeared (in months)
F_9 Breeding	50	11	39	78.0	9.90
F_{10} Breeding	70	9	61	87.1	10.50
F_{11} Breeding	79	6	73	92.4	9.26
F_{12} Breeding	85	2	83	97.6	9.98
F_{12} Nonbreeding	12	0	12	100.0	11.70
F_{13} Breeding	115	7	108	93.9	8.51
F_{13} Nonbreeding	6	0	6	100.0	13.33
F_{14} Breeding	65	3	62	95.3	8.72
F_{14} Nonbreeding	57	2	55	96.4	11.29
F_{15} Breeding	75	2	73	97.3	8.57
F_{15} Nonbreeding	42	3	39	92.8	11.42

The table also includes a summary of all breeding females of the F_9 to F_{15} generations which are now completed and which may be regarded as a continuation of the earlier report (*1*). It is seen that breeding strain C_3H females continue to exhibit a high incidence of spontaneous breast tumors at an average age of 8 to 9 months. There has not been any change in the breeding procedures, diet, or handling of these mice since publication of the previous communication.

In view of differences in the average age at which breast tumors appear spontaneously in mice of different lines of the same inbred strain (*1*), it is believed that in any experimental procedure dealing with such growths it is essential to use litter mate controls. Accordingly, 51 breeding litter mates were kept as controls for 71 of the

nonbreeding mice. These 142 mice represented the offspring from 43 litters. The results are summarized in table 2.

TABLE 2.—*Summary of nonbreeding and breeding sister controls of 43 litters of strain C_3H mice*

	Total number of mice	Number that died without tumor	Number that developed spontaneous breast tumor	Percent that developed tumor	Average age at which tumor appeared (in months)
Nonbreeding	71	0	71	100	11.5
Breeding	51	0	51	100	9.5

In table 2 it is seen that the nonbreeding mice and their breeding litter mates developed breast tumors at an average age of 11.5 months and 9.5 months, respectively. It will be noted that the average tumor age of the breeding mice is somewhat higher than in the recent generations of strain C_3H. This may be due to the influence of delayed breeding. As stated previously (*1*), the breeding procedure is to keep the females of each litter in a cage with a brother from the time they are weaned, which usually results in their becoming pregnant at a relatively early age. The 51 breeding mice recorded in table 2 were removed from their brothers when weaned at 1 month of age and were not mated until 2 or 3 months had elapsed. They are not included in the figures of table 1. It is of interest to record that of the 43 mothers of the mice included in table 2, 40 developed spontaneous mammary gland tumors at an average age of 8.5 months. The influence of delayed breeding upon the age incidence of breast tumors in strain C_3H mice is receiving further consideration.

INFLUENCE OF FOSTER NURSING UPON THE INCIDENCE OF BREAST TUMORS IN STRAIN C_3H MICE

Bittner (*3, 4*) has shown that mother's milk may be responsible for the occurrence of spontaneous breast tumors in mice. In his experiments, newborn mice of a strain with a high incidence of breast tumors were removed from their mothers and suckled by mice belonging to strains with a low incidence of breast tumors, and it was found that in these fostered mice, and in their offspring, the incidence of breast tumors was low. Such findings suggest that some agent (or agents) is transmitted in the mother's milk which exerts a decided influence upon the occurrence of breast cancer in mice.

A preliminary report on the results of an experiment along similar lines is included in this paper, since strain C_3H mice were used and the results thus far obtained can be compared with the incidence of breast tumors in breeding and nonbreeding females of the same strain. Newborn mice of strain C_3H were foster nursed by strain C57 black

mice which, under ordinary conditions, are very resistant to the development of spontaneous mammary gland tumors (*13*). After the mice had been weaned they were bred to their brothers and treated the same as other mice in the strain C_3H colony. There were 51 strain C_3H in the experiment, and the results recorded herein are as of March 1, 1938, when all the surviving animals were from 13 to 13.5 months of age. Of the 51 mice, 11 or 21.5 percent, developed breast tumors at an average age of 9.9 months. Since practically all breeding C_3H females and 76 percent of nonbreeding C_3H females develop breast tumors when 13.5 months old, it is apparent that foster nursing exerted a pronounced influence upon the occurrence of mammary gland tumors in the experimental mice. The results, thus far, confirm Bittner's findings.

In the same experiment there were 44 C57 black mice which were nursed by strain C_3H mice; and of these, 7 have died without tumor. Of the remaining 37 mice, which were 13 to 13.5 months old, none had developed a tumor.

It is desirable to emphasize that the findings reported here are not final and are presented at this time to show that Bittner's work has been confirmed in part. The final results will be presented in detail in a future report.

SUMMARY AND CONCLUSIONS

Breeding females of strain C_3H have a high incidence of spontaneous breast tumors at an average age of 8 to 9 months. Nonbreeding females have the same high incidence of tumors, but they appear at an average age of 11.5 months. It is concluded that pregnancy hastens the appearance of spontaneous mammary gland tumors in this colony of strain C_3H mice.

The preliminary findings of a foster nursing experiment in which strain C_3H mice were nursed by C57 black mice show that, up to the age of 13.5 months, the strain C_3H mice exhibit a tumor incidence of approximately 21.5 percent, which is much lower than the tumor incidence in breeding or nonbreeding mice of the same age. It is concluded that foster nursing by C57 black mice exerts a decided influence upon the occurrence of spontaneous mammary gland tumors in strain C_3H mice.

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DEATHS DURING WEEK ENDED APR. 23, 1938

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

	Week ended Apr. 23, 1938	Corresponding week, 1937
Data from 86 large cities of the United States:		
Total deaths	8,661	9,072
Average for 3 prior years	9,131	
Total deaths, first 16 weeks of year	142,023	160,944
Deaths under 1 year of age	546	552
Average for 3 prior years	588	
Deaths under 1 year of age, first 16 weeks of year	8,693	9,961
Data from industrial insurance companies:		
Policies in force	69,642,337	69,763,872
Number of death claims	13,910	14,315
Death claims per 1,000 policies in force, annual rate	10.4	10.7
Death claims per 1,000 policies, first 16 weeks of year, annual rate	10.0	11.4

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) is to be interpreted to mean that no cases or deaths occurred, while leaders (----) indicate that cases or deaths may have occurred although none were reported.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Apr. 30, 1938, and May 1, 1937

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937
New England States:								
Maine	0	1	6	3	166	12	0	0
New Hampshire	0	0	----	----	13	85	0	0
Vermont	7	1	----	----	187	----	0	0
Massachusetts	4	4	----	----	386	667	2	4
Rhode Island	0	2	----	----	1	183	0	0
Connecticut	7	2	3	6	49	464	0	1
Middle Atlantic States:								
New York	30	47	[1] 15	[1] 13	4,234	1,281	8	13
New Jersey	20	16	7	8	1,211	2,392	1	8
Pennsylvania	33	32	----	----	3,009	1,113	2	13
East North Central States:								
Ohio	10	27	----	27	2,469	1,086	3	8
Indiana	19	9	17	14	979	332	2	0
Illinois [2]	35	27	18	41	2,455	282	1	7
Michigan [2]	8	13	----	3	3,930	160	4	0
Wisconsin	3	1	41	26	3,250	21	1	2
West North Central States:								
Minnesota	2	3	2	----	212	23	1	0
Iowa	2	3	----	6	268	15	0	1
Missouri	11	7	23	61	420	40	3	4
North Dakota	0	0	14	----	85	----	0	0
South Dakota	0	1	----	----	----	2	1	0
Nebraska	0	5	----	----	137	35	0	0
Kansas	4	9	6	24	605	42	0	1
South Atlantic States:								
Delaware	4	0	----	----	25	44	0	0
Maryland [2 3]	4	6	4	4	107	509	0	4
District of Columbia	5	11	1	2	20	75	0	1
Virginia	11	5	----	----	423	634	0	9
West Virginia	10	14	20	50	517	123	4	5
North Carolina	5	10	14	31	2,223	321	2	13
South Carolina [4]	5	7	125	264	223	120	1	1
Georgia [4]	6	2	----	53	248	----	1	3
Florida	4	4	1	----	259	2	3	0

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Apr. 30, 1938, and May 1, 1937—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937
East South Central States:								
Kentucky	20	10	5	16	405	551	2	15
Tennessee	3	4	30	37	296	44	2	6
Alabama [4]	10	9	39	93	619	12	6	13
Mississippi [3]	9	6	----	----	----	----	0	0
West South Central States:								
Arkansas	3	7	51	63	331	5	1	1
Louisiana [4]	12	15	11	63	17	2	0	0
Oklahoma [5]	1	9	53	41	127	62	0	0
Texas [4]	31	40	306	479	249	930	3	6
Mountain States:								
Montana	0	2	----	10	49	4	0	1
Idaho [2]	0	0	7	2	29	13	0	0
Wyoming [3]	2	0	----	----	42	25	0	2
Colorado [2]	9	5	----	----	356	19	0	2
New Mexico	22	3	----	----	84	74	0	0
Arizona	3	0	36	31	54	102	0	0
Utah [3]	0	0	----	----	334	23	0	0
Pacific States:								
Washington	1	0	----	----	39	53	0	0
Oregon [2]	1	2	27	29	52	11	2	1
California [4]	30	12	42	198	812	183	1	4
Total	406	393	924	1,698	32,006	12,176	57	149
First 17 weeks of year	9,308	8,477	39,027	266,608	555,979	127,959	1,416	2,857

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid and paratyphoid fever		Whooping cough
	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938
New England States:									
Maine	0	0	8	30	0	0	0	0	40
New Hampshire	0	0	6	4	0	0	1	0	----
Vermont	0	0	14	4	0	0	0	0	36
Massachusetts	0	0	332	238	0	0	2	2	120
Rhode Island	0	0	19	53	0	0	0	1	11
Connecticut	1	1	107	154	0	0	1	1	69
Middle Atlantic States:									
New York	0	0	675	985	0	0	5	9	535
New Jersey	0	0	126	246	0	0	2	3	211
Pennsylvania	0	0	270	747	0	0	3	5	165
East North Central States:									
Ohio	1	1	340	442	3	0	6	8	287
Indiana	0	2	109	177	87	10	9	0	28
Illinois [2]	2	1	487	725	19	25	5	4	126
Michigan [3]	0	0	412	765	5	16	6	5	336
Wisconsin	2	0	185	289	18	7	3	1	255
West North Central States:									
Minnesota	0	0	182	160	20	1	0	0	44
Iowa	0	0	166	230	36	80	2	2	28
Missouri	0	1	161	389	45	48	4	7	20
North Dakota	0	0	14	30	8	10	1	0	18
South Dakota	0	0	15	64	8	2	0	1	20
Nebraska	0	1	39	----	19	14	0	0	17
Kansas	0	0	105	326	13	18	1	1	183
South Atlantic States:									
Delaware	0	0	14	4	0	0	0	1	11
Maryland [2 3]	0	0	75	40	0	0	1	1	61
District of Columbia	0	0	18	12	0	0	0	0	13
Virginia	1	2	17	17	0	0	6	9	81
West Virginia	1	0	26	55	1	0	8	2	50
North Carolina	0	0	23	37	1	2	2	3	437
South Carolina [4]	3	1	1	3	0	1	5	4	107
Georgia [4]	3	0	6	10	1	0	10	0	28
Florida	1	0	6	15	2	0	5	2	24

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Apr. 30, 1938, and May 1, 1937—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid and paratyphoid fever		Whooping cough
	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938	Week ended May 1, 1937	Week ended Apr. 30, 1938
East South Central States:									
Kentucky	1	0	39	40	12	0	3	6	139
Tennessee	0	0	28	11	3	0	2	2	42
Alabama [4]	1	0	6	6	23	2	2	0	62
Mississippi [3]	0	0	5	4	4	1	5	2	-------
West South Central States:									
Arkansas	1	0	6	7	4	7	5	1	43
Louisiana [4]	1	1	9	17	1	0	16	6	1
Oklahoma [5]	0	0	24	27	20	3	9	4	99
Texas [4]	0	1	92	139	4	14	8	21	327
Mountain States:									
Montana	0	0	15	24	3	69	2	1	34
Idaho [2]	0	0	9	12	16	5	1	2	10
Wyoming [2]	0	0	3	7	2	1	0	0	23
Colorado [2]	0	0	58	62	3	2	1	0	47
New Mexico	0	0	11	25	0	0	1	2	15
Arizona	0	0	3	13	6	0	1	1	65
Utah [2]	0	0	32	13	1	0	0	0	60
Pacific States:									
Washington	1	0	35	35	27	12	0	2	196
Oregon [2]	1	2	32	31	16	18	0	1	18
California [4]	0	2	207	170	34	20	7	6	643
Total	21	16	4,577	6,904	465	388	151	129	5,185
First 17 weeks of year	347	355	100,393	117,155	9,053	5,485	2,046	1,880	71,886

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended Apr. 30, 1938, 8 cases, as follows: Illinois, 1; Maryland, 1; Idaho, 1; Wyoming, 3; Colorado, 1; Oregon, 1.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended Apr. 30, 1938, 16 cases, as follows: South Carolina, 1; Georgia, 5; Alabama, 5; Louisiana, 1; Texas, 3; California, 1.
[5] Figures for 1937 are exclusive of Oklahoma City and Tulsa.

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	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
January 1938										
New Hampshire	0	1	10	-------	-------	-------	0	86	0	0
February 1938										
New Hampshire	0	0	10	-------	-------	-------	0	84	0	2
South Carolina	-------	113	2,522	329	1,059	60	2	28	0	8
March 1938										
Hawaii Territory	0	19	35	-------	124	-------	8	2	0	1
Kansas	2	23	53	-------	2,071	-------	0	761	73	2
Ohio	19	96	120	1	12,023	-------	4	1,656	62	16
Puerto Rico	0	38	55	2,708	30	7	0	-------	0	39
Virginia	12	67	681	7	2,393	5	5	175	0	11
Wisconsin	3	15	175	-------	20,505	-------	1	810	17	2

February 1938

South Carolina:	Cases
Chickenpox	206
Dengue	1
Diarrhea	250
Dysentery (amoebic)	1
Hookworm disease	91
Mumps	134
Ophthalmia neonatorum	10
Paratyphoid fever	2
Rabies in animals	35
Septic sore throat	10
Tetanus	1
Tularaemia	2
Typhus fever	5
Whooping cough	254

March 1938

Anthrax:	
Puerto Rico	1
Chickenpox:	
Hawaii Territory	135
Kansas	797
Ohio	2,439
Puerto Rico	128
Virginia	412
Wisconsin	1,510
Conjunctivitis:	
Hawaii Territory (epidemic)	2
Diarrhea:	
Ohio (under 2 years; enteritis included)	7
Virginia	28
Dysentery:	
Hawaii Territory (amoebic)	1
Ohio (amoebic)	1
Puerto Rico	22
Virginia (amoebic)	1
Dysentery—Continued.	Cases
Virginia (diarrhea included)	28
Encephalitis, epidemic or lethargic:	
Kansas	5
Ohio	1
Wisconsin	5
Filariasis:	
Puerto Rico	3
German measles:	
Kansas	7
Ohio	43
Wisconsin	89
Hookworm disease:	
Hawaii Territory	6
Impetigo contagiosa:	
Hawaii Territory	38
Lead poisoning:	
Ohio	2
Leprosy:	
Hawaii Territory	5
Puerto Rico	1
Mumps:	
Hawaii Territory	67
Kansas	1,222
Ohio	1,132
Puerto Rico	10
Virginia	292
Wisconsin	1,326
Ophthalmia neonatorum:	
Kansas	1
Ohio	53
Puerto Rico	1
Paratyphoid fever:	
Ohio	2
Puerperal septicemia:	
Ohio	6
Puerto Rico	6
Scabies:	
Kansas	19
Septic sore throat:	Cases
Hawaii Territory	1
Kansas	5
Ohio	138
Virginia	80
Wisconsin	26
Tetanus:	
Hawaii Territory	2
Ohio	1
Puerto Rico	8
Tetanus, infantile:	
Puerto Rico	8
Trachoma:	
Hawaii Territory	2
Kansas	3
Trichinosis:	
Ohio	1
Tularaemia:	
Kansas	2
Ohio	2
Virginia	3
Wisconsin	3
Typhus fever:	
Hawaii Territory	2
Undulant fever:	
Kansas	6
Ohio	5
Wisconsin	9
Vincent's infection:	
Kansas	10
Puerto Rico	2
Whooping cough:	
Hawaii Territory	146
Kansas	524
Ohio	656
Puerto Rico	280
Virginia	382
Wisconsin	629
Yaws:	
Puerto Rico	2

PLAGUE INFECTION IN GROUND SQUIRRELS IN SANTA CRUZ COUNTY, CALIF.

Under date of April 28, 1938, Dr. W. M. Dickie, Director of Public Health of California, reported that plague infection had been proved, by animal inoculation, in one *beecheyi* squirrel from a ranch 4 miles east of Watsonville and in a pooled specimen of organs from five *beecheyi* squirrels from a ranch 6 miles east of Watsonville, Santa Cruz County, Calif. These specimens were submitted to the Hooper Foundation for Medical Research on April 13.

WEEKLY REPORTS FROM CITIES

City reports for week ended April 23, 1938

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

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	178	212	73	7,234	784	2,481	23	421	24	1,420	------
Current week [1]	106	82	29	10,884	620	1,656	22	357	18	1,211	------
Maine:											
Portland	1	------	0	19	1	0	0	0	0	3	19
New Hampshire:											
Concord	0	------	0	0	1	0	0	1	0	0	13
Nashua	0	------	0	0	0	0	0	0	0	0	7
Vermont:											
Barre	0	------	0	0	0	0	0	1	0	0	7
Burlington	0	------	0	13	0	0	0	0	0	0	10
Rutland	0	------	0	0	2	0	0	0	0	0	6
Massachusetts:											
Boston	0	------	0	186	32	97	0	3	0	9	238
Fall River	0	------	1	1	3	0	0	0	0	4	28
Springfield	0	------	0	17	0	2	0	2	0	9	40
Worcester	1	------	0	1	6	15	0	1	0	9	44
Rhode Island:											
Pawtucket	0	------	0	0	0	1	0	0	0	0	20
Providence	0	------	1	0	9	10	0	1	0	10	73
Connecticut:											
Brigdeport	0	------	0	5	3	12	0	1	1	0	35
Hartford	0	------	0	2	4	30	0	1	0	2	42
New Haven	0	3	0	1	3	0	0	0	0	5	41
New York:											
Buffalo	0	------	0	2	11	58	0	8	0	13	155
New York	27	9	4	2,706	112	345	0	87	0	235	1,554
Rochester	0	1	0	13	5	29	0	2	1	2	71
Syracuse	0	------	0	60	5	13	0	1	0	1	57
New Jersey:											
Camden	0	------	0	46	1	7	0	0	0	0	38
Newark	1	------	1	20	11	5	0	6	0	38	138
Trenton	0	------	0	3	2	5	0	1	0	0	49
Pennsylvania:											
Philadelphia	2	3	1	1,215	29	132	0	26	2	16	538
Pittsburgh	3	3	2	123	13	27	0	8	0	28	145
Reading	0	------	1	17	3	2	0	0	0	1	32
Scranton	0	------	------	36	------	10	0	------	0	0	------
Ohio:											
Cincinnati	1	------	1	8	6	17	0	7	1	4	120
Cleveland	0	19	1	340	14	60	0	9	1	56	180
Columbus	4	1	1	194	4	8	2	2	0	2	90
Toledo	1	------	0	121	10	5	0	2	0	29	65
Indiana:											
Anderson	0	------	0	187	1	0	0	0	0	4	9
Fort Wayne	1	------	0	37	4	3	0	0	0	2	27
Indianapolis	2	------	0	361	9	19	0	2	0	7	98
Muncie	0	------	0	0	2	0	0	0	0	0	7
South Bend	0	------	0	119	2	3	0	0	0	1	19
Terre Haute	0	------	0	24	0	0	4	0	0	0	11
Illinois:											
Alton	0	------	0	0	4	2	0	0	0	0	10
Chicago	12	3	1	980	34	241	0	31	0	57	659
Elgin	0	------	0	15	0	4	0	0	0	0	4
Moline	0	------	0	11	0	2	0	0	0	0	8
Springfield	0	------	0	90	3	1	1	0	0	0	22
Michigan:											
Detroit	10	------	0	1,256	16	112	0	14	1	80	247
Flint	0	------	0	134	2	37	0	0	0	26	27
Grand Rapids	0	------	0	135	2	14	1	1	0	4	36
Wisconsin:											
Kenosha	0	------	0	57	0	3	0	0	0	1	6
Madison	0	------	0	152	0	1	0	0	0	5	10
Milwaukee	0	------	0	225	5	11	0	6	0	52	104
Racine	0	------	0	345	1	7	0	0	0	31	21
Superior	0	------	0	7	0	4	0	0	0	2	8

[1] Figures for Salt Lake City, Utah, and St. Joseph, Mo., estimated; reports not received.

City reports for week ended April 23, 1938—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0	----	0	3	3	6	0	0	0	3	35
Minneapolis	0	----	0	280	6	9	0	0	0	3	108
St. Paul	1	----	0	4	8	16	0	0	0	3	67
Iowa:											
Cedar Rapids	0	----	----	8	----	1	0	----	0	3	----
Davenport	0	----	----	1	----	0	0	----	0	0	----
Des Moines	0	----	0	19	0	19	4	0	0	0	33
Sioux City	0	----	----	12	----	4	0	----	0	0	----
Waterloo	0	----	----	103	----	6	0	----	0	0	----
Missouri:											
Kansas City	2	----	1	42	12	16	0	3	0	1	97
St Joseph	----	----	----	----	----	----	----	----	----	----	----
St. Louis	1	----	0	2	6	75	1	3	0	4	214
North Dakota:											
Fargo	0	----	0	1	2	1	0	0	0	3	16
Grand Forks	0	----	----	94	----	0	0	----	0	0	----
South Dakota:											
Aberdeen	0	----	----	0	----	0	0	----	0	6	----
Sioux Falls	0	----	0	0	0	0	1	0	0	0	10
Nebraska:											
Omaha	0	----	0	115	13	0	0	2	0	0	67
Kansas:											
Lawrence	0	----	0	59	0	0	0	0	0	1	3
Topeka	0	----	0	315	1	1	0	0	0	36	23
Wichita	0	----	0	33	4	2	0	0	0	4	30
Delaware:											
Wilmington	0	----	0	16	3	6	0	0	0	4	32
Maryland:											
Baltimore	0	3	1	20	23	33	0	13	0	33	223
Cumberland	0	----	0	5	1	2	0	0	0	0	19
Frederick	0	----	0	1	0	0	0	0	0	0	3
Dist. of Col.:											
Washington	4	----	0	23	15	27	0	8	0	13	163
Virginia:											
Lynchburg	1	----	0	1	4	1	0	1	0	2	16
Norfolk	0	----	0	28	6	7	0	3	0	1	36
Richmond	1	----	1	115	5	2	0	3	0	0	62
Roanoke	0	----	0	1	0	1	0	1	0	0	10
West Virginia:											
Charleston	0	----	0	0	0	2	0	0	0	0	10
Huntington	0	----	----	4	----	2	0	----	0	0	----
Wheeling	0	----	0	182	3	3	0	1	0	6	22
North Carolina:											
Gastonia	0	----	----	68	----	1	0	----	0	5	----
Raleigh	0	----	0	161	1	0	0	0	0	23	19
Wilmington	0	----	0	67	2	0	0	0	0	30	13
Winston-Salem	0	----	0	5	2	1	0	0	0	38	16
South Carolina:											
Charleston	2	18	0	5	2	2	0	2	0	0	21
Florence	0	----	0	13	2	0	0	0	0	0	10
Georgia:											
Atlanta	0	2	1	19	10	1	0	7	0	9	88
Brunswick	0	----	0	11	0	0	0	0	0	0	4
Savannah	1	2	0	74	2	1	0	2	0	1	38
Florida:											
Miami	0	----	0	52	2	0	0	1	1	6	39
Tampa	0	2	1	33	1	1	0	2	1	0	21
Kentucky:											
Ashland	0	----	0	8	4	0	0	1	0	5	20
Covington	1	----	0	0	0	0	0	1	0	3	16
Lexington	0	----	0	2	2	1	0	2	0	0	21
Louisville	2	----	0	174	10	25	0	5	0	7	76
Tennessee:											
Knoxville	0	1	0	20	0	3	0	2	0	3	22
Memphis	0	----	0	20	3	8	0	10	0	2	70
Nashville	0	----	0	72	8	7	0	1	0	7	48
Alabama:											
Birmingham	0	2	0	54	4	2	0	3	1	0	71
Mobile	0	2	0	5	2	0	0	1	0	0	19
Montgomery	0	----	----	80	----	0	0	----	0	1	----
Arkansas:											
Fort Smith	0	----	----	3	----	0	0	----	1	0	----
Little Rock	0	----	0	2	7	1	0	3	0	2	10

City reports for week ended April 23, 1938—Continued

State and city	Diphtheria cases	Influenza Cases	Influenza Deaths	Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
Louisiana:											
Lake Charles	0	----	0	0	0	0	0	1	0	0	9
New Orleans	6	1	1	0	14	5	0	8	4	32	132
Shreveport	1	----	0	3	5	0	0	5	0	0	52
Oklahoma:											
Oklahoma City	0	----	0	0	2	4	0	1	0	0	29
Tulsa	0	----	----	101	----	2	1	----	0	11	----
Texas:											
Dallas	1	8	2	7	5	11	0	3	0	17	56
Fort Worth	0	----	0	5	1	2	0	2	0	5	30
Galveston	0	----	0	0	2	1	0	2	0	0	20
Houston	3	----	1	0	4	3	1	4	1	2	66
San Antonio	0	----	1	0	10	1	0	5	0	0	61
Montana:											
Billings	0	----	0	0	3	0	0	1	0	0	13
Great Falls	0	----	0	0	4	2	0	1	0	10	13
Helena	0	----	0	2	0	0	0	0	0	0	2
Missoula	0	----	0	0	1	0	0	1	0	0	6
Idaho:											
Boise	0	----	0	0	0	0	1	2	0	0	6
Colorado:											
Colorado Springs	0	----	0	0	1	1	0	2	0	1	11
Denver	5	----	1	131	7	13	0	4	0	7	105
Pueblo	0	----	0	6	0	3	1	0	0	12	7
New Mexico:											
Albuquerque	0	----	0	3	3	1	0	3	0	4	15
Utah:											
Salt Lake City	----	----	----	----	----	----	----	----	----	----	----
Washington:											
Seattle	0	----	2	4	8	5	0	3	0	56	105
Spokane	0	1	1	0	5	1	1	0	0	11	43
Tacoma	1	----	0	1	5	2	0	0	0	4	30
Oregon:											
Portland	1	----	0	20	9	15	2	2	0	1	82
Salem	0	1	----	1	----	0	0	----	0	0	----
California:											
Los Angeles	7	9	0	39	16	40	0	21	1	17	349
Sacramento	2	----	0	21	2	2	0	1	0	56	34
San Francisco	1	----	0	3	13	6	0	6	3	47	174

State and city	Meningococcus meningitis Cases	Meningococcus meningitis Deaths	Poliomyelitis cases
Rhode Island:			
Providence	1	0	0
New York:			
Buffalo	3	3	0
New York	1	0	0
Pennsylvania:			
Pittsburgh	0	0	1
Scranton	1	0	0
Ohio:			
Cincinnati	4	0	0
Indiana:			
Indianapolis	1	1	0
Michigan:			
Detroit	4	0	0
Iowa:			
De Moines	1	0	0
Maryland:			
Baltimore	1	0	0
District of Columbia:			
Washington	2	0	0
Georgia:			
Atlanta	0	0	1
Kentucky:			
Lexington	1	0	0
Alabama:			
Birmingham	3	2	0
Louisiana:			
New Orleans	1	0	0
Shreveport	0	2	0
Texas:			
Dallas	1	1	0
Houston	1	0	0
Colorado:			
Denver	3	1	0

Dengue.—Cases: Charleston, S. C., 1.
Encephalitis, epidemic or lethargic.—Cases: New York, 2; Philadelphia, 1; Chicago, 2.
Pellagra.—Cases: Lynchburg, 1; Charleston, S. C., 3; Atlanta, 2; Savannah, 5; Birmingham, 4; Dallas, 1; San Antonio, 1.
Typhus fever. Cases: New York, 1.

FOREIGN AND INSULAR

CANADA

Provinces—Communicable diseases—2 weeks ended March 26, 1938.—During the 2 weeks ended March 26, 1938, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Nova Scotia [1]	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis			2	6	1				9
Chickenpox	14	25	551	462	93	41	10	252	1,480
Diphtheria	9	3	61	9	2	7			91
Dysentery			1	1				1	3
Erysipelas			24	3	7		2	8	44
Influenza	16		30	39	9			59	153
Measles	32	8	498	840	3	2	55	182	1,620
Mumps	31	11		308	252	1	26	60	689
Paratyphoid fever				1					1
Pneumonia	9			34		2		28	73
Poliomyelitis							1	1	2
Scarlet fever	38	6	260	213	30	93	91	92	823
Smallpox	2						8		10
Trachoma					1			3	4
Tuberculosis	8	17	100	107	22		4	23	281
Typhoid fever		5	50	1	4	2		1	63
Undulant fever			2	1					3
Whooping cough			168	209	31			101	509

NOTE.—No report was received from Prince Edward Island for the above period.

[1] For 2 weeks ended Mar. 30, 1938.

ESTONIA

Vital statistics—1937.—The following table shows the marriages, births, and deaths in Estonia for the year 1937:

Population	1,131,125	Number of births per 1,000 population	16
Number of marriages	9,585	Number of deaths	16,650
Number of births	18,190	Number of deaths per 1,000 population	15

SWEDEN

Notifiable diseases—March 1938.—During the month of March 1938, cases of certain notifiable diseases were reported in Sweden as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	3	Poliomyelitis	[1] 61
Diphtheria	5	Scarlet fever	2,103
Dysentery	17	Syphilis	29
Epidemic encephalitis	2	Typhoid fever	2
Gonorrhea	816	Undulant fever	16
Paratyphoid fever	15	Weil's disease	2

[1] Includes 15 cases nonparalytic at time of notification.

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 April 29, 1938, pages 685-700. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Cholera

India—Northwest Frontier Province—Dera-Ismail-Khan.—During the week ended April 30, 1938, 1 fatal case of cholera was reported in Dera-Ismail-Khan, Northwest Frontier Province, India.

Indochina (French).—During the week ended April 23, 1938, cholera was reported in French Indochina as follows: Annam Province, 76 cases; Tonkin Province, 425 cases; Hanoi, 39 cases.

Plague

On vessel—S. S. Ville de Tamatave.—On March 31, 1938, 1 case of plague was reported in a member of the crew of the S. S. *Ville de Tamatave* at Beirut, Syria.

United States—California.—A report of plague infection in ground squirrels in Santa Cruz County, Calif., appears on page 787 of this issue of the PUBLIC HEALTH REPORTS.

Smallpox

Mexico.—During the month of February 1938, smallpox was reported in Mexico as follows: Chiapas State, 1 case, 1 death; Mexico State, 1 case; Mexico, D. F., 4 cases, 2 deaths; Queretaro State, 4 cases, 3 deaths.

Typhus Fever

Mexico.—During the month of February 1938, typhus fever was reported in Mexico as follows: Mexico, D. F., 16 cases, 4 deaths; Queretaro, Queretaro State, 5 cases, 1 death; San Luis Potosi, San Luis Potosi State, 2 cases; Saltillo, Coahuila State, 1 death; Toluca, Mexico State, 7 cases, 2 deaths.

On vessel—S. S. Empress of Japan.—A case of typhus fever was reported on the S. S. *Empress of Japan* at Yokohama, Japan, on April 7, 1938.

Yellow Fever

Brazil.—Yellow fever has been reported in Brazil as follows: Minas Geraes State, February 17 to April 4, 1938, 9 deaths; Rio de Janeiro State, February 22 to April 3, 1938, 4 deaths; Santa Catharina State, March 28, 1938, 1 death.

Gold Coast—Keta.—On April 23, 1938, 1 fatal case of suspected yellow fever was reported in Keta, Gold Coast.

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