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STUDIES ON THE EPIDEMIOLOGY OF POLIOMYELITIS

By C. C. DAUER, M. D., Epidemiologist, District of Columbia Health Department

Part I

To paraphrase a recent statement on science in general, the aim of epidemiology is to find out and expose, as far as is humanly possible, why diseases are as they are, and to learn the rules under which they act. Studies on the epidemiology of poliomyelitis have resulted in numerous hypotheses which have been altered by obtaining and studying additional data.

The purpose of this section of the paper is to present certain information on the prevalence of poliomyelitis in the United States since 1916, and to review the epidemiology of the disease as it was presented in 1916 and its developments during the succeeding 20 years. The year 1916 was selected as the starting point of this study because the epidemic of that year was intensively studied and reported upon, and the history of the disease previous to that time has been adequately reviewed. The second part of the paper presents in detail the distribution of the disease by counties during the 5-year period from 1933 to 1937, inclusive.

Death rates rather than case rates are used in this section of the paper to show the prevalence of poliomyelitis in the various States because they probably are more nearly accurate. A study of case fatality rates disclosed the fact that there were wide fluctuations from year to year in some States, suggesting that cases were well reported in epidemic years and poorly reported in the intervals between epidemics. In other States case fatality rates fluctuated within fairly definite limits, which might be regarded as an indication of fairly uniform reporting of cases. The decline in case fatality rates in the past 10 to 15 years might be explained on the basis of more complete reporting of cases, the inclusion of more abortive and nonparalytic cases among those reported, of a difference in the virulence of the virus, a more resistant host, or by a combination of two or more of the factors mentioned.

The death rates presented in table 1 are based on data taken from Mortality Statistics Reports of the Bureau of the Census. The data shown in figure 1 are from the same source.

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GEOGRAPHICAL DISTRIBUTION

The epidemic of 1916, the most severe in the history of poliomyelitis in the United States, was most intense in New York and New Jersey. It extended into New England, where it was less severe, into the East North Central States, and as far south along the Atlantic coast as North Carolina. Montana also experienced a sharp outbreak in 1916. From 1917 to 1920, localized outbreaks occurred in a few scattered States located in different sections of the country. Judging by the death rates, the disease became more prevalent again in 1921, at which time outbreaks occurred in the New England, Middle Atlantic, East North Central, and West North Central States. The State of Washington in that year experienced the most severe outbreak of poliomyelitis, but none of the neighboring States had an unusual number of deaths from this cause. In 1924 and 1925 several of the Pacific Coast and Rocky Mountain States had outbreaks of the disease.

In 1927 and 1928 death rates from poliomyelitis were higher than average in most parts of the country. In the Middle Atlantic States death rates were below the average for these 2 years, and no outbreaks occurred in the South Atlantic States south of North Carolina, in the East South Central States except for Kentucky, nor in the West South Central States.

Most of the Rocky Mountain and Pacific Coast States experienced high death rates from the disease in 1930. A widespread outbreak occurred in the northeastern section of the country again in 1931. The New England and Middle Atlantic States were most severely affected. Outbreaks of less intensity occurred in several of the East North Central and West North Central States.

In 1932 and 1933 a few localized outbreaks occurred in various sections of the country. In 1934 a severe outbreak of poliomyelitis occurred in the extreme northwestern part of the country. The States of Washington, Idaho, and Montana were most severely affected. In California and Arizona the disease was also epidemic.

In 1935, localized outbreaks occurred in Virginia, North Carolina, and Kentucky, and in several of the New England States. In 1936 an epidemic broke out in the West South Central States. The outbreaks which occurred in the South Atlantic and East South Central States in 1935 and 1936 have been described in more or less detail in various reports, and their geographical distribution will be presented in detail in the second part of this paper.

The mortality data for the year 1937 are not yet available for the various states. Other data show a widespread outbreak of poliomyelitis in the West South Central, West North Central, and several of the Rocky Mountain States. TABLE 1.—Poliomyelitis death rates per 100,000 population, by States, 1915-36

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1918	1.38 1.38 1.38 1.38	1.25.8	3.5882 3.13882	<u> </u>	-0	11.2	
1917	0044111		1.1			1.2	
1916	ふらふごてぬ マアオアオト		24 1.84 0.5	*	പ്പം പ്പ	2.1	
1915	001200	1.0	1.9 1.2 1.2		1.6 1.1 1.7	1.0	
Division and State	New England States: Maine	Middle Atlantic States: New York New Jersey	East North Central States: Dho	West North Contral States: Minnesota	South Atlantic States: Delaware Dataryland. District of Columbia. Virginia. North Carolina. North Carolina. Bouth Carolina. Foreita.	Bast South Central States: Kontucky Tennessee Alabama Mististippi	West South Central States: Arkansas. Louislana Oklahoma. Texas-

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TABLE 1.—Poliomyelitis death rates per 100,000 population, by States, 1915-36—Continued

Division and State	1915	1916	181	1918	1919	1920	1921	1923	1923	1924	1925	1926	1927	1928	1929	1930	1881	1932 1	1933	1834	1935	1936
Mountain States: Montana	02	5.0	1	1.3	0.0	0.9	0.4	-i-	1,1	4	11		0.7	1 18 18	0	1.1	-100 (i)	0.4	•	00	0 P	0.0
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California.	80	6.	.7				1.5		1.0	1 00						2.4					11	•

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From 1916 to 1937, inclusive, outbreaks of poliomyelitis have shown two characteristics. In 1916, 1921, 1927–28, 1931, 1934, and 1937 they were widespread and affected fairly large areas. In the intervals between these widespread outbreaks, localized epidemics occurred in more or less restricted regions. In adjacent areas to these localized epidemics the disease appears to have been no more than endemic in character.

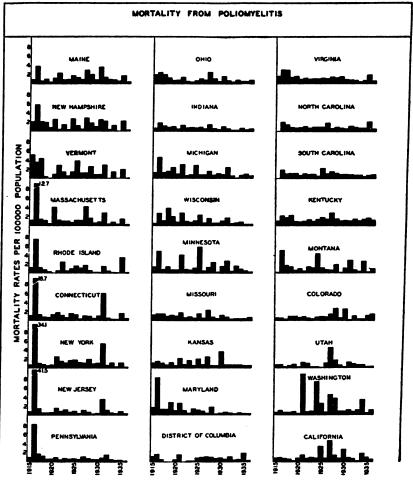


FIGURE 1.-Poliomyelitis death rates per 100,000 population for certain States, 1915 to 1936.

The widespread outbreaks since 1915 have occurred at varying intervals from 3 to 5 years. The same geographical area is not always affected in successive outbreaks. In 1916 the epidemic was most intense in the Middle Atlantic and New England States; in 1921 the East North Central and West North Central States were most severely affected. In 1927 and 1928 the outbreak was most severe in the Rocky Mountain and Pacific Coast States. In 1931 the greatest intensity

was again found in the Middle Atlantic and New England States. The widespread outbreak in 1934 occurred in the Rocky Mountain and Pacific Coast States, and in 1937 in the West North Central and West South Central States. The localized epidemics in the intervening years showed even more irregularities as regards geographical distribution and intensity.

Although death rates are not available for the 48 States from 1916 to 1936, inclusive, the available data clearly indicate that the disease occurs in every section of the country each year either in endemic or epidemic form. Judging from the available data for southern States, poliomyelitis seldom broke out in epidemics in this region previous to 1935. In the past 3 years, 1935, 1936, and 1937, as will be shown in the later section of this paper, there has been a definite increase in outbreaks in the South.

The most definite change in the occurrence of poliomyelitis since 1916 has been in the age distribution of persons affected. In 1916 and previous to that time, from two-thirds to three-fourths of the cases reported were in children under 5 years of age, while one-fifth or less were in persons 10 years of age and over. Since 1930, from one-third to one-half of reported cases have been in children less than 5 years of age, and one-fourth to one-third in persons 10 or more years of age. This change has been a gradual one over the past 20 years. In large cities there is still to be found a greater proportion of cases in children under 5 years than in rural regions, but the shift in the age distribution is apparent.

Except for this change in age distribution, there seems to have been little change in the behavior of the disease in recent years. Any reduction in case fatality rates could be explained by more complete reporting of cases; better diagnostic facilities, a more complete knowledge of the disease, and the development of public health activities no doubt have led to better reporting.

EPIDEMIOLOGY OF POLIOMYELITIS

One may summarize as follows the prevailing views concerning the epidemiology of poliomyelitis twenty years ago: ¹

1. The generally accepted view was that poliomyelitis was caused by a filterable virus and could be transmitted to monkeys experimentally. Aside from man and certain monkeys, no other animals were known to be susceptible. Nasal and intestinal secretions of persons suffering with the disease usually contained the virus. It was believed that carriers were responsible for widespread infection.

2. Monkeys could be infected with the virus by injecting it into the brain and nerve sheaths, and by introducing it into the nasal mucous

¹ Epidemiologic Studies of Poliomyelitis in New York City and the Northeastern United States During the Year 1916. By C. H. Lavinder, A. W. Freeman, and W. H. Frost. Public Health Bullstin No. 91.

membrane. It had also been reported that monkeys had been infected by biting insects but this method of experimental infection was open to question.

3. The disease was widespread and widely scattered, but it occurred most frequently in localized outbreaks which were irregular and scattered. It was noted that epidemics usually were more intense in villages and rural areas than in densely populated cities. The relatively small proportion of the population contracting the disease during an epidemic in contrast to other epidemic diseases was noted.

4. Although the characteristic seasonal prevalence was recognized, it was known that outbreaks of poliomyelitis occasionally began in the spring or extended into the fall months. Isolated outbreaks were known to occur in the winter time. Even though it was the general belief that secretions from the upper respiratory tract were infectious, it had not escaped the attention of observers that poliomyelitis had a seasonal prevalence very different from all other acute respiratory diseases.

5. The preponderance of cases in children under 5 years of age was noted, and also the fact that there was a greater concentration of cases under 5 years in cities than in rural areas.

6. Poliomyelitis was generally regarded as a contagious disease mainly spread from one human being to another. It was observed in some outbreaks that the infection apparently spread from case to case only in a small proportion of instances. No direct or indirect association could be traced in the majority of cases even after the most careful and searching investigations. However, it was observed that a greater proportion of contact children in families affected with poliomyelitis developed the disease than the child population as a whole. It was generally believed that an immunity or resistance to infection developed in a large proportion of persons following exposure to infection and that carriers were instrumental in spreading the infectious agent from one susceptible individual to another. The occurrence of abortive cases and of subclinical infections was also suggested.

7. The widespread occurrence of poliomyelitis and the apparent, although not proved, spread from case to case in a few instances suggested that man was the only source of infection and that lower animals were not the reservoir of infection. The possibility of a lower animal being the primary source of the disease was recognized, but investigations failed to show any positive evidence of such a source. The possibility that the disease might be spread by an insect vector was also recognized, but no convincing epidemiological evidence of such transmission was revealed. A few instances of insect transmission to monkeys had been reported, but these experimental results had not been confirmed. Following the 1916 outbreak, the opinion most generally held seemed to be that poliomyelitis was a contagious disease and that man was the only source of infection. This view is the one which is most generally accepted at the present time. It is quite evident, however, that no one in 1916 had any definite information regarding the mode of transmission of the disease; and the question of the mode of transmission is still unanswered.

From time to time attempts have been made to explain why only certain individuals develop poliomyelitis on exposure to infection. Draper suggested that morphological characteristics in certain persons were determining factors in the development of the disease. His views have never been widely accepted, nor have subsequent investigations substantiated his theories. Aycock has suggested that variations in physiological activities of the body determine whether or not a person will develop poliomyelitis. These variations, according to Aycock, are the result of environmental influences, principally seasonal. It has also been suggested that persons belonging to certain blood groups are more susceptible to poliomyelitis than others. Inheritance factors, vitamin imbalance, and other hypotheses have also been mentioned.

In recent years the possibility of the existence of several immunologically distinct strains of poliomyelitis virus has been brought to light. Not only is the possibility of different strains of virus admitted, but the ability of various strains to produce the disease experimentally by different routes or portals of entry is also suggested. These possibilities are important in studying the epidemiology of the disease.

It is quite evident that little has been added to our knowledge of the epidemiology of poliomyelitis in the past 20 years. More studies have been conducted in the experimental laboratory than in the field. It appears that the final solution of the epidemiological problems of poliomyelitis will have to come from studies of the behavior of the disease as it occurs under varying conditions from year to year. These studies will have to be conducted in such a manner that all possible factors which may be concerned in the causation of the disease will be brought to light.

Part II

In the preceding section the prevalence of poliomyelitis since 1916 in the United States was discussed and certain aspects of the epidemiology of the disease were reviewed. This section presents in detail the distribution of poliomyelitis by counties in the 5-year period 1933-37, inclusive, for the entire country. A number of papers have described and discussed certain of the outbreaks which have occurred since 1932, and, of these, Lumsden's recent paper presents the geographical distribution of the disease in greatest detail.² Other

² Lumsden, L. L.: Poliomyelitis: Facts and fallacies. Southern Medical Journal, May 1938.

phases of the epidemiology of poliomyelitis are discussed in considerable detail by Lumsden, and should be read by everyone interested in this problem.

The number of cases by counties was secured from records of or through the cooperation of the United States Public Health Service. Case rates rather than death rates are used in this part of the study because the former are more readily available and probably are more significant for the present purpose. It is recognized that reporting of cases may vary in periods of low prevalence as compared with epidemic periods, and that the proportion of abortive and nonparalytic cases may also vary. However, it appears that such differences do not seriously interfere with the main purpose of this presentation.

Division and State	1933	1934	1935	1936	1937
New England States:					
Maine	. 8.1	2.7	19.0	5.0	16. 1
New Hampshire	. 2.8	.8	9.5	.8	4.9
Vermont		1.6	17.7	21	7.6
Massachusetts		171	32.0	1.3	7.9
Rhode Island		.i	51.5	.7	3.2
Connecticut		.8	23.4	.9	6.2
Middle Atlantic States:					
New York	3.2	1.7	22.2	1.5	4.9
New Jersey	5.5	1.4	11.8	.61	3.6
Penneylvania	1 4 1	I.4	22	13	8.3
Pennsylvania East North Central States:		[
Obio	5.3	4.3	1.3	5.1	7.9
Indiana		ĩği	1.4	1.5	4.2
Illinois.		4.8	30	8.8	9.9
Michigan		4.91	13.0	3.2	9.0
Wiccomein	22	17	22	1.5	11.4
Wisconsin West North Central States: Minnesota			**!	1.3	11, 1
West North Central States.	144	4.0	8.6	1.2	12.6
Ja Lineso La		1.5	2.5	3.0	9.4
Iowa					9.9
Missouri		.9	1.3	27	
North Dakota		1.4	1.7	2.7	.9
South Dakota		6.3	21	1.9	5.7
Nebraska	. 1.9	1.5	.9	1.7	16.0
Kansas		4.3	1.9	5.0	12.9
outh Atlantic States: Delaware					
Delaware	- 28	1.2	2.0	.4	3.1
Maryland	_ 2.6	1.5	6.4	2.2	4.8
District of Columbia		2.0	14.3	1.1	4.8
Virginia	. 1.5	3.0	25.7	2.2	2.4
West Virginia	. 5.21	4.7	2.2	8.4	3.7
North Carolina	9	1.4	19.8	1.5	3.1
South Carolina	1.6	19	21	1.2	1.2
Georgia		.8	.8	4.8	2.7
Florida		LÖ	1.0	2.5	1.8
ast South Central States:					
Kentucky	1.6	4.2	11.5	3.1	4.4
Tennessee	43	22	3.2	13.2	4.4
Alabama		1.8	21	14.6	29
Mississippi		17	.8	9.5	21.0
Vest South Central States:				0.0	
Arkansas	4	.4	.8	27	16.2
Louisiana		1.2	4.8	1.6	6.2
				5.0	18.1
Oklahoma			.5		10.7
Texas	8	2.5	10	1.1	10.7
Iountain States:					
Montana		60.3	11	2.6	5.8
Idaho		33.0	.9	4.3	3.9
Wyoming	4.8	3.5	.9	3.0	16.7
Colorado	6	1.9	2.1	6.3	19.4
Colorado	. 2.1	4.2	2.4	7.4	6.1
Arizona	. 7.5	32.0	6.1	3.4	6.8
Utab	4.4	2.9	2,1	1.3	6.4
Nevada	. 2.1	16.3	2.0	2.0	5.0
acific States:	1			1	
Washington	. 5.6	45.8	2.4	4.7	5.3
Oregon	3.6	8.1	4.6	3.6	6 .0
California		56.6	13.7	6.4	11.5
egistration area	4.3	5.9	8.6	3.5	7.3

TABLE 2.—Poliomyelitis case rates per 100,000 population, by States, 1933-37

Case rates by counties are shown on the maps for each of the 5 years from 1933 to 1937, and case rates by States are shown in table 2.

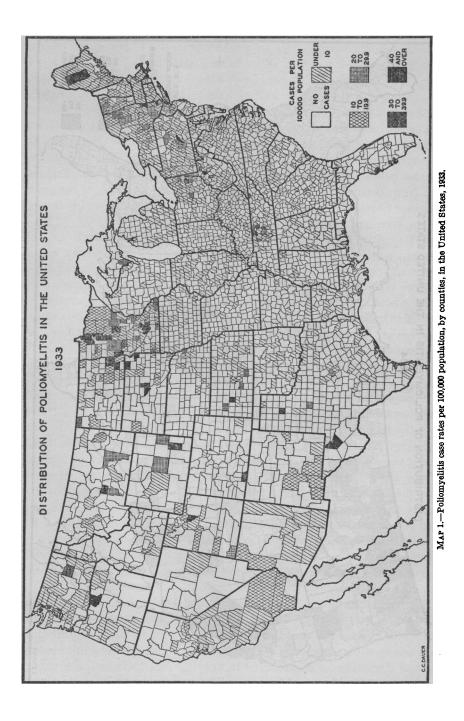
1933.—In the country as a whole, the incidence of poliomyelitis was comparatively low in 1933. (See table 2 and map 1.) The incidence was comparatively high in only one area—an area including most of Minnesota and the eastern half of North Dakota and South Dakota. Several localized outbreaks of milder intensity occurred in central Tennessee and Kentucky, in southeastern Ohio and northern West Virginia, in central Pennsylvania, in southeastern New York, and in northern New York, northern Vermont, and central Maine. In the greater part of southern California and the southern parts of Arizona and New Mexico the incidence was slightly above average for the year. In other States very small localized outbreaks occurred, as evidenced by scattered counties with case rates of 30 or more per 100,000.

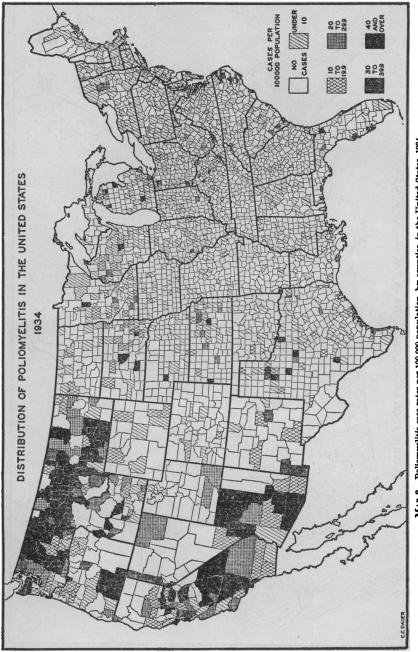
1934.—An extensive outbreak of poliomyelitis occurred in the western part of the United States in 1934. As shown in map 2, the outbreak was extensive and intensive in a large area extending from central Washington through northern Idaho, and to the eastern part of Montana. In a group of counties in central Idaho, and another in western Oregon and western Washington, the incidence of the disease was lower, but above the average for the country as a whole. A moderately severe outbreak occurred in a group of counties in north central California, and a more severe one in south central California. The greater part of Arizona was also the seat of a severe epidemic.

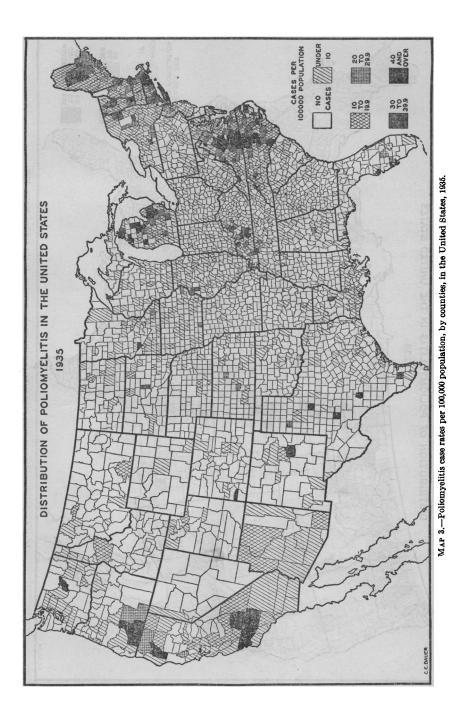
The remainder of the country had a fairly low incidence of poliomyelitis except the northwestern part of Texas, where a few scattered counties had a high incidence of the disease.

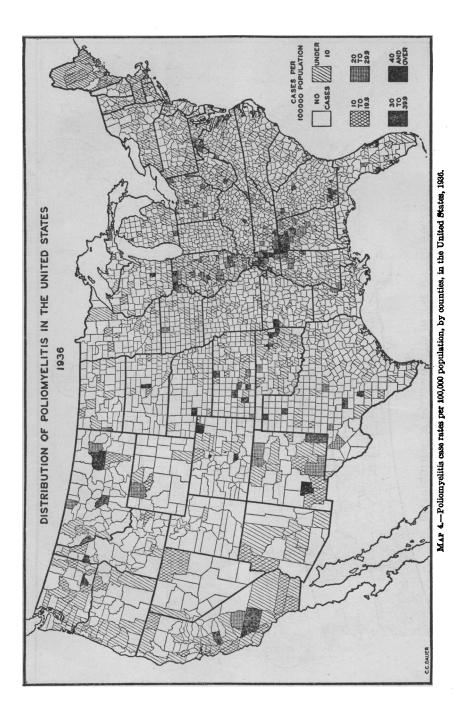
1935.—A number of outbreaks of poliomyelitis occurred in 1935. In southwestern Oregon and the extreme northern part of California, where the incidence was fairly low in 1934, an outbreak occurred. The disease was prevalent again in south central California but less severe than during the preceding year. The incidence was high in a number of counties in western Kentucky, and also in Michigan. An epidemic of unusual proportions occurred in two adjacent groups of counties in the east central section of North Carolina and Virginia. In smaller groups of counties in New York and in New England, poliomyelitis also occurred in epidemic proportions.

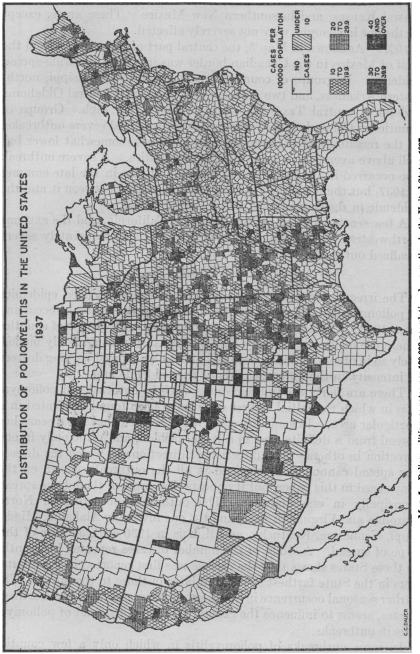
1936.—A number of small localized outbreaks occurred in various sections of the country during 1936. The most extensive epidemic occurred in an area comprising the northwestern part of Alabama, northeastern Mississippi, west central Tennessee, the extreme western part of Kentucky, and extending in varying degrees from the southern to the northern border of Illinois. The epidemic was most intense in Alabama, Mississippi, and Tennessee. A small outbreak occurred in a tri-State area consisting of a few counties in northern Virginia,











eastern West Virginia, and western Maryland. Others occurred in southern Georgia, in western Arkansas and eastern Oklahoma, in central Kansas, and in southern New Mexico. These areas, except for the one in Georgia, were not severely affected.

1937.—As shown in Map 5, the central part of the country from the Gulf of Mexico to the Canadian border was involved in a wide-spread epidemic. In groups of counties in southwestern Mississippi, northeastern Arkansas, and two adjacent groups in south central Oklahoma and north central Texas, the incidence was very high. Groups of counties in Colorado and Wyoming also experienced severe outbreaks. In the remainder of the area the case rates were somewhat lower but well above average in a large number of counties. A severe outbreak also occurred in the Province of Ontario, Canada, in the late summer of 1937, but there is no evidence of any association between it and the epidemic in the central part of the United States.

A few scattered areas in New England, California, and the extreme northwestern part of the country also experienced moderately severe localized outbreaks.

DISCUSSION

The irregularity of distribution which has characterized epidemics of poliomyelitis in the past has been exhibited in outbreaks occurring since 1932. Certain of them were confined to small groups of counties where the incidence was very high in some instances and only moderately so in others. Widespread epidemics also showed varying degrees of intensity in the different parts of the epidemic area.

There are fairly accurate accounts of some outbreaks of poliomyelitis in which the initial or first cases have been found or located in a particular area. In some instances it has seemed that the disease has spread from a definite area in a radial fashion, and principally in one direction in others. The direction or directions in which the disease has spread cannot be determined in all the outbreaks shown on the maps used in this paper, but it appears that some of them have spread principally in one direction. The outbreaks occurring in North Carolina and Virginia in 1935, and the one involving Alabama, Mississippi, Tennessee, Kentucky, and Illinois in 1936 are examples of this type of spread. A study of the number of cases reported by months in these States shows that the disease was first reported in large numbers in the State farthest south, and later in those to the north. The earlier seasonal occurrence in Southern, as compared with the Northern States, seems to influence the general direction of spread of poliomyelitis in outbreaks.

In some outbreaks of poliomyelitis in which only a few counties have been involved, the spread of disease has seemed to have been very definitely limited. Numerous examples of this may be found on the maps presented with this paper.

There have been a number of statements in the literature regarding the tendency of poliomyelitis to spread along lines of human traffic. However, there is no evidence of such manner of spread to be found in the data used here. Geographical barriers, such as mountains, valleys, and large rivers, do not seem to have influenced the direction in which the disease spreads.

It has been stated frequently that poliomyelitis outbreaks have been more intense in small towns and rural regions than in large cities. On the maps presented here it can be seen that case rates are lower during outbreaks for counties in which large cities (250,000 or more population) are located than in the surrounding, less thickly populated counties. It is also apparent that, during inter-epidemic periods, case rates are slightly higher in the counties with large cities than in rural areas. Cases occur each year in each of the large cities, but frequently none occur in rural areas between outbreaks.

High case rates (30 or more per 100,000 population) for 2 or more successive years occurred infrequently in a single county or groups of counties from 1933 to 1937. However, the occurrence of high case rates in one group of counties of a State or region during 1 year and in another group within the same State or in the same general region in a succeeding year was not infrequent. In some instances high rates for 2 successive years occurred in single counties with small populations, in which case the existence of one or two cases naturally resulted in high case rates. Under such circumstances one cannot eliminate the element of chance. Two adjacent counties in California had high case rates during 4 of the 5 years from 1933 to 1937, but this is said to be due to the fact that an unusual number of "abortive" cases have been reported even in the years when the disease had not seemed to be epidemic in adjacent counties.

Case rates by States do not always give a true picture of the prevalence of poliomyelitis within certain States, and this is more likely to be true in States with large populations. For instance, Pennsylvania and Tennessee did not have high case rates in 1933, nor Michigan in 1934, nor Georgia in 1936 (see table 2). However, localized epidemics occurred within these States during the respective years mentioned. In these instances the increased numbers of cases due to the localized outbreaks were not sufficiently large to cause more than a moderate rise in the case rates for the States as a whole. On the other hand, the case rates in the North Central States in 1937 were fairly high, but there was no evidence of localized or general epidemics within any State except in northwestern Illinois, where a

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group of counties had rates only moderately high, between 20 and 30 per 100,000 population.

CONCLUSION

The distribution of poliomyelitis in the United States from 1933 to 1937, inclusive, was extremely irregular in several respects. The regions involved in outbreaks of the disease showed wide variations in extent. In some outbreaks only a few counties were involved, and in other outbreaks there were a large number. The severity of the disease as evidenced by case rates showed considerable variation from year to year and from place to place.

The data presented in this paper confirm the statement that the disease is found in all parts of the country.

In this, as well as in the preceding section of this paper, the aim has been to present only factual material. No attempt has been made to explain any of the facts presented. This cannot be done because of many unsolved problems in the epidemiology of poliomyelitis. The solution of these problems awaits more intensive and extensive studies of the disease in the laboratory and, most important of all, in the field.

ACKNOWLEDGMENTS

The author wishes to thank all who aided in collection of the data used in this paper, especially Assistant Surgeon General Robert Olesen, of the Public Health Service, and his staff. Acknowledgment is also made to Medical Director L. L. Lumsden for his constructive criticisms in the preparation of the manuscript.

STUDIES ON DENTAL CARIES

IV. TOOTH MORTALITY 1 IN ELEMENTARY SCHOOL CHILDREN³

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

INTRODUCTION

Recent widespread activity in the promotion of dental health programs in the United States is evidence that dental health has become recognized as a major subdivision of the more general problem of national health conservation. Since the loss of teeth resulting from carious processes constitutes an important physical defect found in elementary school children, one of the primary objectives of dental health programs becomes the prevention of tooth loss. Inasmuch as the etiology of dental caries is unknown, prevention of the disease causing these defects is still in the experimental stage. It is generally acknowledged, however, that the treatment of early carious lesions by the proper placement of chemically and physically stable filling materials will largely prevent carious teeth from terminating in tooth loss, or tooth mortality. A primary purpose of dental health programs becomes, therefore, the promulgation of procedures whereby the early detection and treatment of carious teeth is accomplished and tooth mortality thereby prevented.³

This perspective on dental programs, and on the problem of dental caries in children, suggests an analogy between the public health approach to dental disease and that which has already been applied to other diseases. In a general way the loss of permanent teeth in children, or tooth mortality in childhood, bears somewhat the same relation to the problem of dental caries as specific death rates bear to the problem of other disease entities. While it has been pointed out many times that death rates alone are not satisfactory indicators of

The preceding papers of this series are as follows:

I. Dental status and dental needs of elementary school children. By Henry Klein, C. E. Palmer, and J. W. Knutson. Pub. Health Rep., 53: 751-765 (May 13, 1938).

II. The use of the normal probability curve for expressing the age distribution of eruption of the permanent teeth. By Henry Klein, C. E. Palmer, and M. Kramer. Growth, 1: 385-394 (1937).

¹ Although the term "tooth mortality" has been infrequently used in the dental literature, it appears to possess definite value in designating deaths among a population of permanent teeth. The term includes, therefore, not only extracted permanent teeth but also those which are indicated for extraction and still present in the mouth. It is clear that "tooth mortality" does not apply to missing deciduous teeth lost through exfoliation, since exfoliation is considered a normal biological process.

² From Child Hygiene Investigations, Division of Public Health Methods, National Institute of Health, U. S. Public Health Service.

III. A method of determining post-eruptive tooth age. By C. E. Palmer, Henry Klein, and M. Kramer. (In press.) Growth.

³ The immediate objective for placing fillings in many instances may not be the prevention of tooth loss but to perform such functions as the following: The prevention and allaying of toothache; the prevention of apical abscesses and their sequelae, focal infections; the maintenance of normal occlusion and esthetics; and the maintenance of a normally functioning masticatory unit. However, the performance of these functions operated directly or indirectly to prevent tooth loss.

the health of a community or nation, the study of such rates has assisted enormously in recent mass attacks on disease.

Recently, Wisan (1) has developed a "lost permanent teeth index" based on counts of extracted permanent teeth plus those indicated for extraction, and has proposed that this index of missing teeth, for 7th and 8th grade children or those between 12 and 14 years of age, be used for the purpose of evaluating dental health programs. Before practical use is made of such an index it is essential that very careful study be made of the basic elements which make up the index. Thus it is imperative that detailed information be made available regarding those factors which may be associated with the mortality of permanent teeth in children. The present paper represents an attempt to study certain of these factors as they are found in a representative elementary school population.

MATERIAL AND METHODS

The findings to be presented are concerned with an analysis of tooth mortality rates in the grade school population of Hagerstown, Maryland, a representative urban community in the eastern section of the United States. The enrollment of white children in the first eight grades of the municipal elementary schools of that community was, in October 1936, approximately 4,700. Of this number, 4,416 received, in the spring of 1937, complete dental examinations ⁴ by dental officers 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 such characteristics as age and sex, may be considered representative of many urban communities in the United States.

The tooth mortality rates are determined by counting missing (extracted) permanent teeth and permanent teeth showing only remaining roots (severely decayed); this sum divided by the number of children and multiplied by 100 gives a tooth mortality rate of the number of lost teeth per 100 children. The analysis of the data is made in the following order: First, the distribution of tooth mortality in terms of specified kinds of permanent teeth is described for the elementary grade school population as a whole; second, the tooth mortality rates are considered for children having one or more decayed or missing teeth but no fillings; finally, the tooth mortality rates are discussed for children having one or more decayed or missing teeth and, in addition, one or more filled teeth.

⁴ Details of the manner in which the examinations were made and a general analysis of the findings have been presented in a recent publication (2).

FINDINGS

The age and sex distribution of tooth mortality in grade school children studied in terms of numbers of missing teeth per 100 children are presented in table 1. Girls, for each age, have a significantly higher tooth mortality rate than boys. This finding parallels the observation previously reported (2) that girls, for each age, have a significantly higher caries rate than boys. This table also shows the manner in which tooth mortality rates increase directly with chronological age. For boys age 6 the rate per 100 children is 0, for age 7, it is 2.5, and it increases gradually until age 15, when the rate has become 118.1. Because of this cumulative characteristic, the yearly increments of numbers of missing teeth per 100 children are readily calculated. For example, since boys age 6 have no missing teeth, the rate at age 7 may be considered as equal to the increment which has accumulated between the sixth and seventh years. It follows, therefore, that the total yearly increment of tooth mortality in the entire elementary school population is the sum of all the annual increments for each age-sex group from 6 through 15 years.

Аgə	6	7	8	9	10	11	12	13	14	15	All ages
						Boys					
Number of cases Number of teeth missing Number of teeth missing per 100 cases	171 0 0	197 5 2.5	231 10 4. 3	253 35 13. 8	270 88 32. 6	262 77 29. 4	299 121 40. 5	267 160 59. 9	199 148 74. 4	83 98 118. 1	2,232 742 83. 2
					·	Girls					•••••••••••••••••••••••••••••••••••••••
Number of cases Number of teet hmissing Number of teeth missing per 100 cases	156 1 0.6	206 6 2.9	256 15 5. 9	240 55 22. 9	2:9 68 26. 3	269 86 32.0	297 154 51. 9	278 209 75. 2	165 144 87. 3	58 69 119.0	2,184 807 37.0

TABLE 1.—Number of permanent teeth missing and tooth mortality rates (4,416 children, Hagerstown, Md.)

Table 2 shows the distribution of tooth mortality by specified kinds of permanent teeth. For all ages in 2,232 boys, the specific contribution of the teeth, in increasing order of their numerical importance to a total tooth mortality of 742 is as follows: 3 canines, 3 second molars, 5 second bicuspids, 8 first bicuspids, 8 lateral incisors, 18 central incisors, and 697 first molars. Similar findings as to the identity of the kinds of teeth contributing to tooth mortality may be noted in the data given for girls.

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TABLE 2.—Number of specified kinds of permanent teeth missing (4,416 children, Hagerstown, Md.)

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Age	6	7	8	9	10	11	12	13	14	15	Allages
			BO	YS							
Number of cases	171	197	231	253	270	262	299	267	199	83	2, 232
						Upper	•				
Central incisors Lateral incisors Canines First bicuspids Second bicuspids First molars Second molars		0 0 0 0 0 1 0	1 0 0 0 0 1 0	3 0 0 0 0 3 0	2 0 0 1 25 0	0 0 0 1 14 0	· 4 3 2 0 23 0	5 2 1 1 0 46 0	2 0 0 8 1 36 0	1 2 0 3 0 29 1	18 7 3 7 3 177 1
						Lower					
Central incisors Lateral incisors Canines First bieuspids First molars Second molars	0 0 0 0 0 0	0 0 0 0 4 0	0 0 0 0 8 0	00000290	000000000000000000000000000000000000000	0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 1 89 0	0 1 0 1 1 1 101 1	0 0 0 0 105 1	0 0 0 0 62 0	1 0 1 2 520 2
Total	0	- 5	10	35	88	77	121	160	148	98	742
		·	GIF	LS							
Number of cases	156	206	256	240	259	269	297	278	165	58	2,184
				-		Upper					
Central incisors Lateral incisors Canines First bicuspids Second bicuspids First molars Second molars	0 1 0 0 0 0	000000000000000000000000000000000000000	0 0 0 0 1 0	00000090	0 0 0 0 0 4 0	1 1 0 0 7 0	8 1 0 1 25 0	0 1 0 0 41 0	1 1 0 3 2 35 1	8 1 0 1 16 0	8 6 0 3 4 138 1
 The design of the second second]	Lower					
Central incisors Lateral incisors Oanines. First bicuspids. Second bicuspids First molars	0 0 0 0 0 0	0 0 0 0 6 0	0 0 0 0 14 0	0 0 0 0 46 0	0 0 0 0 64 0	0 0 0 2 75 0	0 1 0 2 1 119 1	0 0 0 1 166 0	0 0 0 0 100 1	0 0 0 0 48 0	0 1 0 2 4 638 2
Total	. 1	6	15	55	68	86	154	209	144	ð9	807

When the percentages of the total tooth mortality contributed by the first permanent molars (specific for age and sex) are computed, it is clearly indicated that, in the permanent teeth of a representative grade school population, tooth mortality is largely a problem of mortality of the first molars. In no age-sex group do they account for less than 90 percent of the total tooth mortality, and for all ages they account for 93.9 percent in boys and 96.2 percent in girls. These

findings are shown in table 3, which presents the percentages of the total tooth mortality contributed by the first molars.

Адө	6	7	8	9	10	11	12	13	14	15	All ages
						Boys					
Number of cases Number of teeth missing Number of first molars missing Percent of teeth missing that are	171 0 0	197 5 5	231 10 9	253 25 32	270 88 85	262 77 76	299 121 111	267 160 147	199 148 141	83 98 91	2, 232 742 697
first molars Number of first molars missing per 100 children	•••••	100. 0 2. 5	90. 0 3. 9	91. 4 12. 7	96.6 31.5	98. 7 29. 0	91. 7 37. 1	91. 9 55. 1	95. 3 70. 9	92.9 109.6	93. 9 31. 2
						Girls					
Number of cases Number of teeth missing Number of first molars missing	156 1 0	206 6 6	256 15 15	240 55 55	259 68 68	269 86 82	297 154 144	278 209 207	165 144 135	58 69 64	2, 184 807 776
Percent of teeth missing that are first molars	0	100. 0 2. 9	100. 0 5. 9	100. 0 22. 9	100. 0 26. 3	95. 4 30. 5	93. 5 48. 5	99. 0 74. 5	93. 8 81. 8	92.8 110.3	96. 2 35. 5

 TABLE 3.—Percentages of total tooth mortality contributed by first permanent molars (4,416 children, Hagerstown, Md.)

A further analysis of the manner in which specified teeth contribute to the total tooth mortality indicates that, although all first permanent molars contribute from 90 to 100 percent of the mortality for any given age, the lower first molars *alone* contribute 70 percent of the total tooth mortality in boys and approximately 80 percent of that occurring in girls (all ages). The percentages of the total number of missing teeth contributed by lower first molars are shown in table 4.

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Age	6	7	8	9	10	11	12	13	14	15	All ages
						Boys					
Number of cases	171 0	197 5	231 10	253 35	270 88	262 77	299 121	267 160	199 148	83 98	2, 232 742
missing	0	4	8	29	60	62	89	101	105	62	520
Percent of teeth missing that are lower first molars	0	80.0	80.0	82.9	68.2	80.5	73.6	63.1	71.0	63. 3	70.1
Number of lower first molars missing per 100 cases	0	20	8.5	11.5	22.2	23.7	29.8	37.8	52.8	74.7	23, 3
						Girls					
Number of cases	156 1	206 6	256 15	240 55	259 68	269 86	297 154	278 209	165 144	58 69	2, 184 807
Number of lower first molars missing	0	6	14	46	64	75	119	166	100	48	638
Percent of teeth missing that are lower first molars		100.0	93. 3	83.6	94.1	87.2	77.3	79.4	69.4	69.6	79.1
Number of lower first molars missing per 100 cases	0	2.9	5. 5	19. 2	24.7	27.9	40. 1	59. 7	60. 6	82.8	2 9. 2

 TABLE 4.—Percentages of total tooth mortality contributed by lower first permanent molars (4,416 children, Hagerstown, Md.)

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In a recent report on the dental status of the grade school population under discussion (2) it is shown that, in the permanent dentition, the lower first molars account for 87 percent of the total caries experience in the teeth of the lower jaw, and the upper first molars account for 64 percent of the total caries experience in the teeth of the upper jaw. Since, in children, the loss of permanent teeth may be presumed to be largely or almost entirely due to neglected carious processes, the kinds of teeth affected by the highest mortality rates may be postulated to be those having the highest level of caries experience. Clinical observations (3), together with the above findings, clearly indicate that the first permanent molars experience the highest tooth mortality rates.

It is generally recognized that tooth eruption and the occurrence of caries in teeth are symmetrical and bilaterally equal. That tooth mortality is also bilaterally equal has been pointed out by Brekhus (3). This investigator found that the number of specified kinds of missing teeth in males and females, in both upper and lower jaws, is only one-fourth to one-half of one percent higher on the left than on the right side of the mouth. These slight differences are well within the probable error of his observations. The marked consistency with which tooth mortality tends to be bilaterally equal in the lower first permanent molars of the Hagerstown children is shown in table 5, which presents the number of specified right and left lower first molars missing for each age and sex group. For all ages, the total number of specified lower first molars missing in boys is 260 lower left, 260 lower right, and in girls, 321 lower left, 317 lower right.

 TABLE 5.—Number of lower left, number of lower right, average of numbers of left and right, lower first permanent molars missing, and their mortality rates. (4,416 children, Hagerstown, Md.)

Age	6	7	8	9	10	u	12	13	14	15	All ages
						Boys					
Number of cases Lower left first molars missing Lower right first molars missing	171 0 0	197 2 2	231 3 5	253 16 13	270 27 33	262 33 29	299 47 42	267 50 51	199 56 49	83 26 36	2, 232 260 260
Average of left and right lower first molars missing Average of left and right lower	0	2	4	14. 5	30.0	31. 0	44.5	50. 5	52. 5	31. 0	260
first molars missing per 100 cases	0	1.0	1.7	5.7	11.1	11.8	14.9	18.9	26.4	37.4	11.7
						Girls					
Number of cases Lower left first molars missing Lower right first molars missing	156 0 0	206 3 3	256 8 6	240 23 23	259 31 33	269 38 37	297 60 59	278 84 82	165 51 49	58 23 25	2, 184 321 317
Average of left and right lower first molars missing Average of left and right lower	0	3	7	. 23	32	87. 5	59. 5	83	50	24	319
first molars missing per 100 cases	0	1.5	2.7	9.6	12.4	13.9	20.0	29. 9	30. 3	41.4	14, 6

Findings presented thus far have been concerned with the tooth mortality rates of an entire grade school population. However, it is apparent that the tooth mortality of this grade school population is based on the total tooth mortality experience of three separate groups of children: First, those children having one or more DMF 5 (decayed, missing, or filled) teeth, but no evidence of reparative treatment; second, those children having one or more DMF teeth, one or more of which show objective evidence of treatment (fillings); and third, those children having no DMF teeth. Since only the first and second groups of children contribute to the tooth mortality problem, and since the only criterion ⁶ which differentiates these two groups is past history of dental treatment, a comparison of their tooth mortality rates should reveal the manner in which fillings affect the characteristic of tooth mortality. However, since evidence indicates that tooth mortality is affected by the level of caries experience, it becomes necessary to examine and compare the DMF rates, or caries attack rates, in these two subdivisions of the school population.

Table 6 shows that, of the 2,232 boys examined, 1,127 have one or more DMF teeth but no evidence of treatment (fillings); 440 have

Age	6	7	8	9	10	11	12	13	14	15	All ages
]	Boys-	Withou	ıt filliv	gs	·		<u> </u>
Number of cases	19 40	55 111	94 192	142 356	164 472	150 485	174 613	143 625	124 633	62 427	1, 12 3, 95
Cases	210. 5	201.8	204. 3	250.7	287.8	323. 3	352. 3	437.1	501. 5	688.7	350.8
					Boys-	-With	filling	1			
Number of cases Number of DMF teeth	2 3	24	22 63	32 97	49 173	68 237	89 455	86 440	64 380	26 163	440 2, 015
Number of DMF teeth per 100 cases	150. 0	200. 0	286.4	303. 1	353. 1	348. 5	511. 2	511.6	593.8	626. 9	458.0
				C	irls—V	Withou	t fillin	ga			
Number of cases Number of DMF teeth Number of DMF teeth per 100	27 41	79 155	117 252	129 382	133 422	132 414	150 579	148 729	101 495	32 202	1, 048 3, 672
	151.9	196. 2	215. 4	296. 1	317.3	313.6	386. 0	492.6	490. 1	634. 4	350. 4
`					Girls-	With	fillings				
Number of cases	4 11	10 23	27 76	45 160	68 262	85 374	110 532	118 684	58 392	22 155	547 2, 669
	275. 0	230. 0	281. 5	355. 6	385. 3	440. 0	483.6	579. 7	675. 9	704.6	487.9

TABLE 6.—Number of decayed, missing, or filled (DMF) permanent teeth and DMF rates for 2,175 children without fillings and 987 children with fillings. (3,162 children, Hagerstown, Md.)

* Teeth showing objective evidence of caries, past decay (fillings), or which are missing due to extraction are classed as DMF teeth. For full description of DMF concept see reference (4).

[•] The criterion for that group with fillings is the presence of one or more fillings per child. This is no indication of the adequacy of dental treatment. However, a previous report (2) shows that the boys had had 57.3 percent and the girls 53.4 percent of all carious surfaces in the permanent teeth filled at the time of the examination.

one or more DMF teeth, one or more of which have been filled. Of 2,184 girls, 1,084 have one or more DMF teeth but no evidence of treatment, while 547 have one or more DMF teeth, one or more of which show evidence of fillings.⁷ It is evident from an examination of this table that both boys and girls in the group with fillings have a significantly higher DMF rate than the children in the group without fillings. For all ages, the boys and girls with fillings have, respectively, 457.9 and 487.9 DMF teeth per 100 children; comparable rates for the children without fillings are, respectively, 350.8 and 350.4 DMF teeth per 100 children. Roughly, the group with fillings has, for all ages, 100 more DMF teeth per 100 children than the group without fillings.

When the tooth mortality rates of these two groups are compared, it is shown (table 7) that the group without fillings (for each age and sex), has a markedly higher rate of lost teeth than the group with fillings. For all ages the boys and girls without fillings have, respectively, 53.2 and 55.3 missing teeth per 100 children, and the boys and girls with fillings have, respectively, 33.6 and 41.5 missing teeth per 100 children. Thus, the boys and girls in the group without fillings have, respectively, 58 percent and 33 percent more missing permanent teeth per 100 children than the boys and girls in the group having one or more DMF teeth filled. Since tooth mortality rates appear to be directly related to DMF rates, the finding that the group of children with fillings has a significantly higher DMF rate, yet a markedly lower tooth mortality rate, affords specific quantitative evidence that the placement of fillings markedly lowers tooth mortality rates.

DISCUSSION

Since the prevention of tooth loss due to carious processes is one of the primary objectives of a dental program, it follows that the adequacy and effectiveness of efforts directed toward accomplishing this objective may be measured by the reduction in tooth mortality rates achieved. A measure of such reduction may be obtained through a comparison of full and accurate counts of all missing teeth, specific for each age and sex, made at yearly intervals. However, it must be conceded that limitations of time, personnel, and funds, might limit the practicability of making such complete periodic counts. This latter consideration suggests that a study of the characteristics of tooth mortality and the component mortality rates of the several kinds of teeth might provide simpler, less time-consuming, yet relatively accurate alternative methods for measuring the effectiveness of a dental program.

⁷ For purposes of convenience, that group with one or more DMF teeth but no evidence of treatment will be designated *without fillings*, and that group with one or more DMF teeth, one or more of which show evidence of treatment will be designated *with fillings*.

TABLE 7.—Number of permanent teeth missing and tooth mortality rates for 2,175 children without fillings and 987 children with fillings. (3,162 children, Hagerstown, Md.)

Age	6	7	8	9	10	11	12	13	14	15	All ages
· · · · ·				I	Boys—\	Withou	ıt fillin	ga			
Number of cases	. 19 0	55 5	94 9	142 33	164 75	150 64	174 89	143 122	124 118	62 84	1, 12 59
Number of teeth missing per 100 cases	0	9.1	9.6	23.2	45.7	42.7	51. 2	85. 3	95.2	135. 5	53.
					Boys-	-With	fillings				
Number of cases	20	2 0	22 1	32 2	49 13	68 13	89 32	86 37	64 30	26 20	440 148
	• 0'	0	4.6	6.3	26.5	19. 1	36. 0	43.0	46. 9	76. 9	33.6
				C	irls—V	Withou	t fillin _i	zs			
Number of cases Number of teeth missing Number of teeth missing per 100	27 1	79 6	117 14	129 50	133 54	132 60	150 103	148 141	101 102	32 49	1, 048 580
Cases	. 8. 7	7.6	12.0	38.8	40.6	45. 5	68.7	95.3	101. 0	153. 1	55.3
الم				•••	Girls-	-With	fillings				
Number of cases Number of teeth missing	4	10 .0	27 1	45 5	68 14	85 26	110 51	118 68	58 42	22 20	547 227
Number of teeth missing per 100 cases	<u>0</u>	0	3. 7	11.1	20.6	30. 6	46. 4	57.6	72.4	90. 9	41.5

It has been shown (table 3) that the first permanent molars contribute no less than 90 percent of the total tooth mortality for each age and sex group of a representative grade school population. Since it is evident that any dental program which attempts to conserve teeth must. primarily, be concerned with reducing mortality rates in the first permanent molars, it follows that a relatively accurate measure of that reduction may be obtained through a comparison of periodic counts of missing first permanent molars. In this connection it is of interest to note that, when the total tooth mortality rates for boys in the group with fillings were compared with the rates for boys in the group without fillings (table 7), the latter were found to have, over all ages, a mortality rate 58 percent higher than the former.⁸ However, when the tooth mortality rates of the first permanent molars alone are used to make this comparison (table 8) the boys, over all ages, in the group without fillings have a 60 percent higher rate. This close agreement in findings is significant in view of the fact that the former method involved making observations on 32 tooth spaces per child and the latter on only 4 tooth spaces per child.

[•] The first permanent molars contributed no less than 90 percent of the total tooth mortality for each age and sex group of these specially composed groups, and for all ages in the group of boys and girls without fillings they contributed, respectively, 93.2 and 93.9 percent, and in the group of boys and girls with fillings they contributed, respectively, 94.5 and 97.4 percent.

Age	6	7	8	9	10	11	12	18	14	15	All ages
				1	Boy s —'	Withor	ıt fillin	gs			
Number of cases	19 0 0	55 5 9.1	94 8 8.5	142 30 21. 1	164 72 43. 9	150 63 42. 0	174 82 47. 1	143 115 80. 4	124 118 91. 1	62 78 125. 8	1, 127 566 50. 2
				ł	Boys-1	Withfi	llings				
Number of cases	2 0 0	2 0 0	22 1 4.6	32 2 6.3	49 13 26. 5	68 13 19. 1	89 29 32.6	86 82 37. 2	64 28 43. 8	26 20 76.9	440 138 31. 4

 TABLE 8.—Number of first permanent molars missing and tooth mortality rates for

 1,127 boys without fillings and 440 boys with fillings.
 (1,567 children, Hagerstown, Md.)

It has been shown (table 3) that the lower first permanent molars contribute 70 percent of the total tooth mortality in boys and 80 percent in girls. From this finding it appears reasonable to assume that any appreciable reduction in tooth loss must be reflected in these latter teeth, which account for approximately three-fourths of the total mortality. It follows that a measure of such reduction might be obtained by a comparison of periodic counts of only missing lower first permanent molars. Furthermore, since the distribution of tooth mortality is bilaterally equal, and since tooth mortality rates of lower first molars have a marked tendency to be equally distributed bilaterally in grade school children (table 5), it follows that a reliable measure of this reduction in mortality rates might be obtained through a comparison of periodic counts of missing lower right (or left) first permanent molars.

On the basis of this last postulate, the mortality rates of lower right first permanent molars were used to compare the boys in the group without fillings with the boys in the group with fillings. It is of interest to note (table 9) that by this method of comparison the boys in the group without fillings had (for all ages) a 61 percent higher mortality rate than the boys in the group with fillings. Since counts of all teeth missing gave a 58 percent difference, counts of first molars a 60 percent difference, and counts of lower right first molars a 61 percent difference, it is obvious that the results are very similar. Moreover, the first method involved making observations on 32 tooth spaces, the second, four tooth spaces, and the third, a single tooth space in a single quadrant of the mouth.

TABLE 9.—Number of	lower right first	permanent mol	ars missing	and tooth	mortality
rates for 1,127 boys	without fillings	and 440 boys u	with fillings.	(1,567	children.
Hagerstown, Md.)			•••		

Адө	6	7	8	9	10	11	12	13	14	15	All ages	
	Boys-Without fillings											
Number of cases Number of lower right first molars missing Number of lower right first molars missing per 100 cases	19	55	94	142	164	150	127	143	124	62	1, 127	
	0	2	5	12	27	24	80	43	40	28	211	
	0	3.6	5.3	8.5	16.5	16.0	17. 2	30. 1	32. 3	45. 2	18. 7	
	Boys-With fillings											
Number of cases Number of lower right first molars missing Number of lower right first molars missing per 100 cases	2	2	22	32	49	68	89	86	64	26	440	
	0	0	0	1	6	5	12	8	9	10	51	
	0	0	0	8.1	12. 2	7.4	13. 5	9. 3	14. 1	38. 5	11.6	

The employment of this third method for determining the level of reduction in tooth mortality should markedly reduce the time, funds, and personnel needed. Without disturbing ⁹ the seating arrangement of a classroom, an examiner could proceed from child to child merely counting the number of lower right first permanent molars affected by mortality. If the children have been instructed to retract the right cheek in such a manner as to expose the lower right first molar tooth space, the census would be greatly facilitated.

It has been shown (table 1) that tooth mortality is cumulative with respect to chronological age, and that the rate for any given age is equal to the sum of the increments of all previous ages. This characteristic of the age distribution of tooth mortality suggests that counts might be limited to a certain age group. For example, since the tooth mortality rate at age 12 is equal to the sum of the increments of all previous ages, it follows that this rate should be a reliable measure of the total tooth mortality experience through age 12. However, it is evident that the intervention of an influence (dental treatment) may have variable effects on the tooth mortality rates at different age levels, and that counts of only one specific age will not give an indication of such variability. Making counts at alternate ages might provide sufficient data to indicate a tendency for dental treatment to affect tooth mortality rates differently at different age levels. Although a number of other methods for limiting counts according to age selection seem possible, from a study of table 1 it is evident that tooth mortality counts must be specific for age and sex, since there is a marked difference between the sexes for each age.

[•] The amount of disturbance to routine classroom functions caused by the use of a particular dental public health procedure determines to a considerable extent the practicability of that procedure.

The various alternatives for making counts of tooth mortality in a grade school population are suggested to facilitate the making of such counts with limited personnel, funds, and time. All of these procedures, however, are subject to sampling variations; other factors being equal, their reliability is directly proportional to the number of cases studied. From these considerations it is evident that in small age-sex groups full counts of tooth mortality would appear necessary. It is also clear that the employment of the third alternative (making counts of missing lower right first permanent molars) should be limited to groups of such size that the bilateral occurrence of tooth mortality will not be appreciably affected by chance variations.

The foregoing interpretations of this study on tooth mortality in a representative school population indicate that tooth mortality rates specific for age and sex may be adapted for measuring the adequacy and effectiveness of dental care (fillings). Although it is apparent that total counts of permanent teeth affected by mortality, specific for each age and sex, should afford the most accurate measure of the status of tooth mortality in a community, several alternative methods for obtaining estimates of that status through sampling procedures have been suggested. It has been shown, for the grade school population studied, that these sampling devices afford a comparatively accurate estimate of the reduction in tooth mortality effected by dental treatment.

Since the proper placement of fillings prevents the extension of decay, it follows that through complete and adequate care the tooth mortality rate may be expected to be considerably reduced in children who may actually have a high incidence of dental caries. Therefore, periodic tooth mortality rates may be used as an index of the adequacy and effectiveness of dental care (fillings) in a specific community,¹⁰ but they should not be considered an index of dental health, since the problem of tooth mortality is only one factor in dental health.

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¹⁰ It is apparent that tooth mortality rates should not be used to compare the effectiveness of different types of dental programs in different communities without taking full account of the numerous variables which may affect those rates, such as differences in caries rates.

A STUDY OF PSEUDOTUBERCULOSIS RODENTIUM RECOVERED FROM A RAT

By V. H. HAAB, Passed Assistant Surgeon, United States Public Health Service

This report deals with a disease produced in laboratory animals by inoculation with tissue from an infected domestic rat (*Rattus norvegicus*). The lesions of the disease resemble those of plague, and the organism isolated from infected animals is similar in many ways to *Pasteurella pestis*.

SOURCE OF THE DISEASE

A rat trapped in a market was found to have several granules in its spleen. The lesions were about 2 mm in diameter, and were white and firm; they could be lifted from the spleen with a knife point. A smear made from the organ showed a number of bipolar bacilli bearing a morphologic resemblance to P. pestis.

GUINEA PIG INOCULATION EXPERIMENTS

Passage through guinea pigs.—A guinea pig inoculated cutaneously and killed on the fifth day was found to have an enlarged inguinal lymph node containing organisms similar to those found in the rat spleen. From this guinea pig the disease was passed to others, a total of 40 animals being infected, some by cutaneous inoculation and others by the subcutaneous route.

Thirty-five guinea pigs died of the disease and five were killed while definitely ill. Four animals did not become sick after inoculation. The causative organism was recovered repeatedly from infected guinea pigs, as will be described later.

Duration of the disease in guinea pigs.—In guinea pigs allowed to die of the infection, the length of life following inoculation was from 12 to 31 days in those infected by the cutaneous route, the average being 21 days, while in those inoculated subcutaneously the time was from 7 to 18 days, averaging 11. As passage through guinea pigs was continued, the disease assumed a somewhat more acute form than that seen at first.

PATHOLOGY IN GUINEA PIGS

The following summary of autopsy findings is based on the examination of 39 guinea pigs.

(1) Cutaneous lesions.—Ulcers of $\frac{1}{2}$ to 3 cm in diameter, covered by crusts which varied from thin flakes to thick layers, were present at the inoculation site in half of the animals.

(2) Subcutaneous reaction.—Subcutaneous vascular engorgement was present in slightly more than one quarter of the guinea pigs, being moderate in degree in most instances. The animals dying within relatively few days were more frequently involved than those living longer. Subcutaneous gelatinous edema was present twice.

(3) Mass at inoculation site.—In more than three quarters of the guinea pigs there was a mass of 2 to 4 cm in diameter at the site of inoculation. In the animals dying more acutely, this mass was indurated, and firm when sectioned, while in those dying more slowly it was caseous, and tended to become globular. Purulent masses were rare.

(4) Superficial lymph nodes.—One or both inguinal lymph nodes were involved in every infected animal. There was usually a globular mass in the inguinal region, within which the gland could be seen on section; the inguinal mass varied from less than 1 cm to 3 cm in diameter. In acute cases the lymph node was firm on section, but in the more chronic infections it was caseous. Hemorrhagic nodes, though they occurred, were rare. The axillary lymph nodes were affected in slightly over half the guinea pigs, showing changes similar to those seen in the inguinal nodes.

(5) Deep lymph nodes.—The sacroiliac nodes were involved in half the animals, being similar in all respects to diseased superficial nodes, though seldom as greatly enlarged. There were two instances of involvement of mesenteric lymph nodes, which appeared as yellowish white nodules studding the mesentery and omentum. In one of these cases the entire mesentery, omentum, and small intestine were matted together and adherent to the greater curvature of the stomach.

(6) Spleen.—The spleen was involved in all the guinea pigs. Characteristic changes were enlargement of one and one-half to three times normal size, dark red discoloration, and mottling with white granules, which varied from a few scattered lesions to a hundred or more. The granules were from pin-point size to 2 or 3 mm. In six guinea pigs the lesions were discrete enough to be lifted from the spleen by a knife point.

(7) Liver.—Liver lesions were present in all the infected animals, the lesions being very similar to those in the spleen.

(8) Lungs.—Lung lesions were found in all the animals, and were of 3 types. The most common were focal lesions of $\frac{1}{2}$ to 1 mm, consisting of white granules surrounded by dark red areolae; these granules varied in number from a dozen to more than a hundred. Next in frequency were irregular areas of red or grey consolidation varying in extent from 1 cm to an entire lobe. Least common were large caseous lesions, from $\frac{1}{2}$ to 2 cm in diameter, with grey centers and dark red concentric borders up to 5 mm in width.

(9) Serous cavities.—Pleural effusion was present in about one third of the animals, in quantities varying from 1 to 15 cc. It was almost always clear. Peritoneal effusion was present in only one-tenth of the cases, being tenacious in one instance only. A single guinea pig inoculated intraperitoneally died on the eighth day. This animal showed peritoneal exudate of 15 cc of thin red fluid, widespread mesenteric adhesions, and typical lesions of the lung, liver, and spleen.

Smears from tissues.—Organisms were found in the diseased tissues of all except two guinea pigs. There were two forms, viz, bipolar bacilli about half as long as *P. pestis* and rather wide in proportion to their length, and coccoid, solid staining forms. The organisms tended to appear in large aggregates or clumps, so that one microscopic field might show several dozen, while several adjacent fields would show none.

Isolation of causative organisms.—Cultures of a small cocco bacillus were readily obtained by rubbing diseased spleen or lung tissue on blood agar plates, or by inoculating heart blood of sick animals into infusion broth. The typical lesions of the disease were produced in 18 guinea pigs by inoculating them with these cultures; recovery of the organism from these latter animals fulfilled Koch's postulates.

CHARACTERISTICS OF THE ORGANISM

Colonies on blood agar plates: Abundant growth in 18 hours at 30° C.; colonies discrete, pin point, pale white, translucent, convex, and moist, with regular margins; not tenacious. Agar slants: Abundant growth in 18 hours at 30° C.; diffuse growth without distinct colonies; pale, white, translucent, moist, not tenacious. Bouillon cultures: Good growth in 18 hours at 30° C.; broth sometimes uniformly cloudy and sometimes clear with surface growth and slowly settling flakes.

Morphology.—Small rods and cocci on solid media. Rods usually stain bipolar and cocci stain solidly. Rods resemble P. pestis but are only one-half to three-fourths as long. No definite arrangement of organisms. In broth the organisms appear much the same as on solid media; there was no definite chain formation. On 3 percent salt agar there are involution forms; these are usually long, slender, slightly curved rods, or medium sized rods lying in bundles in a manner suggestive of the diphtheria bacillus; less frequently there are enlarged hollow staining organisms resembling "balloon" forms of involuted P. pestis.

Gram's stain.—The organism is Gram-negative.

Fermentations.—Gas is not produced in any of the common sugars. Acid is produced in dextrose, maltose, galactose, and rhamnose. No acid in lactose or saccharose. Litmus milk unchanged.

Motility.—Motile organisms were seen in two cultures grown at room temperature, but in all other cultures there was no motility, regardless of temperature.

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PATHOGENICITY FOR RATS

Four rats were inoculated with cultures, 2 cutaneously and 2 subcutaneously. One rat was inoculated subcutaneously with a diseased guinea pig spleen. None of these rats became ill, but two showed small masses at the site of inoculation.

One of the rats inoculated subcutaneously with bacterial culture was killed on the 14th day. Autopsy showed a small crusted ulcer at the inoculation site, with a subcutaneous mass 2 cm in diameter. The mass was firm and indurated, with considerable injection about it, but no hemorrhage. There was no inguinal adenopathy, but the right axillary node was rather hard, with a diameter of 0.5 cm, and the right sacroiliac node was 0.75 cm in diameter. The spleen was slightly enlarged, very dark, and firm; it contained 5 white granules of 1 mm diameter or less, which were discrete and raised above the surface of the organ. Liver dark red but not mottled; lungs normal; smears from the spleen showed no organisms. A guinea pig inoculated subcutaneously with the spleen of this rat died in 27 days with lesions typical of the disease under consideration, and a guinea pig inoculated from the latter died in 10 days, with typical findings.

The other 4 rats were apparently healthy 30 days after inoculation. They were then inoculated cutaneously with the spleen of a guinea pig dead of plague; one of the rats died on the fourth day with typical plague and the others remained healthy. The three surviving rats were inoculated again with plague on the eighth day following the original inoculation, the second inoculation being subcutaneous. One of these animals died of plague on the fifth day, while the other two remained healthy.

It is evident that the disease under study is only slightly pathogenic for rats, and that it confers on these rodents some degree of protection against plague.

AGGLUTINATION

The organism was agglutinated by the serum of a rabbit immunized against plague in dilution of 1 to 160. The same serum agglutinated P. pestis in a dilution of 1 to 1280. Blood from a guinea pig sick with the disease failed to agglutinate the organism. No immune guinea pig serum could be tested, as none of the sick guinea pigs ever recovered.

COMPARISON WITH PLAGUE

The disease under study is sufficiently similar to subacute or chronic plague that in any single guinea pig it would be impossible to differentiate with certainty on the basis of anatomical appearance. In a number of these animals, however, it is clear that the constancy of hepatic

lesions is a point of great value in distinguishing between the two diseases, as similar liver granules are seldom encountered in plague in guinea pigs.

The causative organism differs from P. pestis morphologically in that the bipolar forms are smaller and the coccoid forms more numerous than in the case of the latter organism. Culturally, the former organism grows faster on solid media than P. pestis; it usually produces diffuse cloudiness of broth and regularly forms acid in rhamnose, both of which are points of differentiation. There is also a difference in the appearance of the forms produced on salt agar. Motility is not constant in the organism under study, but it does occur, which is not the case with P. pestis.

Pathogenicity for rats is another point of differentiation between the two organisms, P. pestis being quite virulent for these rodents whereas the other organism is only slightly pathogenic.

IDENTITY OF THE DISEASE

The disease described above conforms with the available descriptions of pseudotuberculosis rodentium, and the organism isolated is like the causative organism of that disease. It therefore appears that the disease under study is pseudotuberculosis.

ATTEMPTS TO TRANSMIT PSEUDOTUBERCULOSIS BY FLEAS

Thirty female Xenopsylla cheopis were fed on guinea pigs infected with pseudotuberculosis at a time when the animals were known to have organisms in their blood, as demonstrated by cultures. These fleas were then allowed to feed on healthy guinea pigs for the remainder of their lives. Feces of the fleas were inoculated into guinea pigs at convenient intervals, and the dead fleas were also inoculated into guinea pigs. None of the guinea pigs ever contracted the disease, either by flea bites or by inoculation with flea feces or with dead fleas, although other groups of X. cheopis fed on plague-infected guinea pigs at the time when this experiment was going on became infected with plague and were found to be capable of transmitting the infection by biting.

SUMMARY

Pseudotuberculosis rodentium recovered from a rat was transmitted to guinea pigs and other rats. Pathologically and bacteriologically considerable similarity was found between this disease and subacute plague; the chief differential points have been discussed. Some writers believe that *Bacillus pseudotuberculosis* is a rough variant of *P. pestis*, and it has been claimed that the latter organism has been recovered from supposedly pure cultures of the former by special methods. In the study reported herewith, there has thus far been no indication that the organism of pseudotuberculosis might give rise to variants which would be identical with P. pestis; even in those instances in which guinea pigs died of pseudotuberculosis in a week or less, both disease and causative organism remained true to type and could be distinguished from plague and P. pestis, respectively.

Attempts to transmit pseudotuberculosis from guinea pig to guinea pig by Xenopsylla cheopis were not successful.

DEATHS DURING WEEK ENDED JUNE 4, 1938

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

	Week ended June 4, 1938	Correspond- ing week, 1937
Data from 87 large cities of the United States: Total deaths. Average for 3 prior years	7, 817 8, 164 190, 707 484 547 11, 781 68, 306, 548 10, 143 7. 7 9. 8	1 8, 128 211, 781 1 547 13, 062 69, 785, 134 10, 174 7. 6 10. 9

1 Data for 86 cities.

PREVALENCE OF DISEASE

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

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers. In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (.....) represent no report, with the implication that cases or deaths may have occurred but were not reported to the State health officer.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended June 11, 1938 and June 12, 1937

	Diph	theria	Infl	uenza	Me	asles		gococcus ingitis
Division and State	Week ended June 11, 1938	Week ended June 12, 1937	Week ended June 11, 1938	Week ended June 12, 1937	Week ended June 11, 1938	Week ended June 12, 1937	Week ended June 11, 1938	Week ended June 12, 1937
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut ¹ Middle Atlantic States:	2 0 0 1 0 5	0 1 0 3 2 3	2		105 101 96 526 2 59	75 2 634 69 130	0 0 3 1 0	0 0 0 2 0 0
New York New Jersey Pennsylvania East North Central States:	27 18 25	43 6 25	14 2	2 5 5	3, 665 477 1, 942	1, 586 1, 123 1, 727	· 4 1 6	7 2 7
Date Notifi Celutal States. Ohio Indiana Illinois Michigan ³ Wisconsin West North Central States:	13 6 26 7 0	11 4 39 13 3	1 10 1	14 15 18 19	997 279 753 2, 683 2, 822	2, 290 379 457 279 52	3 1 4 1 1	3 2 5 3 1
Minnesota Iowa 4 Missouri North Dakota South Dakota Nebraska Kansas	3 2 14 1 0 4 1	3 0 7 2 1 0 4	2 3 7 	2 23 11 7	412 298 98 84 154 257	3 76 1 2 10 25	1 2 0 0 0 1 1	0 0 1 0 1 0 1
South Atlantic States: Delaware. Maryland 1 * 4. District of Columbia Virginia. West Virginia 4. North Carolina South Carolina Georgia 1. Florida 1.	2 7 2 3 16 2 3 1	1 5 7 6 5 2 4 8	3 3 1 72 2	1 17 85 2	4 67 16 339 212 745 84 148 69	22 195 93 228 39 196 6 3	0 0 1 4 1 1 0 2	0 3 0 7 3 2 3 0

See footnotes at end of table.

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Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended June 11, 1938 and June 12, 1937—Continued

·		Diph	theria	Infi	uenza	Me	asles	Menin	gococcus ingitis
Division and State		Week ended June 11, 1938	Week ended June 12, 1987	Week ended June 11, 1938	Week ended June 12, 1987	Week ended June 11, 1938	Week ended June 12, 1987	Week ended June 11, 1938	Week ended June 12, 1937
East South Central States: Kontucky Tennessee		14	6	59	1	144	198 94	4	5
Alabama ¹ Mississippi ³ West South Central States:		10 6	10	16	9	121	24	6	47
Arkansas Louisiana ¹ Oklahoma		1 1	3 13 8	9 12 16	7 14 18 135	123 7 117	6 33 366	011	1 2 0 7
Texas ¹ Mountain States: Montana ⁴ Idaho ⁴		1	26 8 4	191 4	1	38 [.] 97 9	8	3 0 0	
Idaho 4 Wyoming 4 4 Colorado 4 New Mexico Arizona		0 18 8 4	1 8 1 2			19 143 72 3	21 21 60 53	000000000000000000000000000000000000000	
Utah ⁹ Pacific States: Washington		0 2 4	0 3 0			444 47 34	49 93 10	0	, 0 1
Oregon California 4		81	81	22	63	871	273	. 8	3
Total First 28 weeks of year		327 11, 359	335 10, 665	442 42, 366	512 271, 539	19, 890 702, 121	11, 121 202, 181	59 1, 749	88 3, 516
<u></u>	Polion	nyelitis	Scarle	st føver	Sma	llpox	paraty	oid and phoid ver	Whoop- ing cough
Division and State	Week ended June 11, 1938	Week ended June 12, 1987	Week ended June 11, 1988	Week ended June 12, 1987	Week ended June 11, 1938	Week ended June 12, 1937	Week ended June 11, 1938	Week ended June 12, 1937	Week ended June 11, 1938
New England States: Maine	0	0	15	13	0	0	0	1	29
New Hampshire Vermont	0 0 0 0 0	0 0 1 0 0	1 12 852 8 87	1 1 164 37 91	0000	0 0 0 0	0 1 1 0 2	0 0 1 0 1	1 17 104 26 123
New York New Jersey Pennsylvania East North Central States:	1 1 0	0 0 1	528 97 223	574 101 500	0 0 0	0 0 0	7 1 7	15 2 8	531 197 232
Dhio Indiana Illinois Michigan ¹ Wisconsin West North Central States:	1 0 2 1 0	0 0 1 0 0	182 34 291 276 99	\$10 63 892 591 189	1 25 15 1 0	8 7 15 12 2	7 7 4 8 3	5 1 5 2 2	104 17 232 336 197
Minnesota Iowa 4 Missouri. North Dakota South Dakota Nebraska Kansas	000000000000000000000000000000000000000	0 1 0 1	70 56 67 29 29 19 54	26. 94 107 13 15 28 72	16 28 38 19 17 4 25	14 30 16 7 0 0 5	0 0 0 1 0	0 4 7 0 2 1	28 37 19 14 11 14 13
South Atlantic States: Delaware Maryland 134 District of Columbia Virginia West Virginia 4 North Carolina South Carolina Georgia 1 Florida 1	0 1 0 0 0 0 1	0 0 0 1 2 2	6 41 6 16 12 16 9 10 8	7 21 6 10 34 16 	0 0 0 2 0 0 0		0 2 8 3 10 6 22 5	0 8 9 2 3 16 11 0	3 46 7 68 76 329 73 44 11

See footnotes at end of table.

	Polion	nyelitis	Scarle	Scarlet fever		llpox	Typh parat fe	Whoop- ing cough	
Division and State	Week ended June 11, 1938	Week ended June 12, 1937	Week ended June 11, 1938						
East South Central States:									
Kentucky	0	0	20	19	4	3	16	9	99
Tennessee	0	2	10	10	0	0	18	11	60
Alabama 1	2	1	9	5	4	1	4	11	75
Mississippi 3	4	7	2	5	1	0	8	5	
West South Central States:									
Arkansas.	0	4	4	13	4	0	7	6	39
Louisiana ¹	3	2	6	9	0	1	15	11	37
Oklahoma	0	3	20	9	18	1	9	11	78
Texas ¹	3	5	41	101	25	1	38	26	384
Mountain States:		•	8						
Montana 4 Idaho 4	0	0	85	11 20	3 23	6	0	0	59
W yoming 4 4	ŏ	ŏ	3	20		1 3	0 3	0	4
Colorado 5	ŏ	ŏ	37	10	4	ő	3 4	1	3 32
New Mexico.	ŏ	ŏ	10	32	3	ŏ	5	ō	32 6
Arizona	ŏl	ŏ	3	4	11	ŏ	10	3	17
Utah ³	ĭ	ŏ	ğ	15	ő	ŏ	10	ő	51
	-	v	-		Ŭ	v	v	Ŭ	
Pacific States: Washington	0	0	17	25	11	0	4	0	89
Uregon	Ó	Ō	19	30	12	0 7	ō	i	32
California 4	1	4	145	181	36	8	15	4	423
Total	23	38	2, 980	4,011	355	148	256	209	4, 545
First 23 weeks of year	450	506	123, 877	149, 164	11, 249	6, 898	3, 222	2, 815	98, 803

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended June 11, 1938 and June 12, 1937-Continued

¹ Typhus fever, week ended June 11, 1938, 34 cases as follows: Connecticut, 1; Maryland, 1; Georgia, 16; Florida, 5; Alabama, 7; Louisiana, 1; Texas, 3.
 ³ New York City only.
 ⁴ Period ended earlier than Saturday.
 ⁴ Rocky Mountain spotted fever, week ended June 11, 1938, 12 cases as follows: Iowa, 1; Maryland, 1; West Virginia, 1; Montana, 1; Idaho, 2; Wyoming, 5; California, 1.
 ⁴ Colorado tick fever, week ended June 11, 1938, 11 cases as follows: Wyoming, 1; Colorado, 10.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Menin- gococ- cus menin- gitis	Diph- theria	Influ- enza	Ma- laria	Mea- sles	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
May 1938 Maine Pennsylvania Tennessee West Virginia Wyoming	15 7 7 0	8 121 17 14 2	2 73 112	2 47 	554 16, 256 534 1, 565 96	 25 	0 2 0 1 0	99 2, 264 75 103 22	0 0 7 2 3	47 17 20 4

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Mey 1958		May 1958—Continued		May 1958—Continued	
Cae	ės 🛛	-	Cases		Cases
Actinomycosis:		Impetigo contagiosa:		Trachoma:	Cabig
Pennsylvania	2	Tennessee	1	Tennessee	0
Chickenpox:	-	Mumps:	-	Trichinosis:	4
	79	Maine	159	Pennsylvania	-
		Dependence			2
Pennsylvania 2, 7		Pennsylvania		Tularaemia:	
	38	Tennessee		Tennessee	4
	51	West Virginia		Typhus fever:	
	47	Wyoming	18	Tennessee.	2
Colorado tick fever:		Ophthalmia neonatorum:		Undulant fever:	-
Wyoming	4	Pennsylvania	8	Maine	1
Dysentery:	-	Tennessee	ī	Pennsylvania	
Pennsylvania (amoebic)	9	Paratyphoid fever:	•	Tennessee	0
Pennsylvania (bacil-	-	Tennessee	1	Vincent's infection:	1
_lary)	1	Rocky Mountain spotted		Maine	3
Tennessee (amoebic)	8	fever:	-	Tennessee	5
Tennessee (bacillary) 8	B5	Wyoming	7	Whooping cough:	
German measles:		Septic sore throat:		Maine	188
Maine 1	15	Tennessee	17	Pennsylvania	954
	33	West Virginia	2	Tennossee	
Tennessee	2	Wyoming	2	West Virginia	389
1 UIIIIWARU	-	Tetanus:	-	Wyoming	
				w Joining	15
		Tennessee			

PLAGUE INFECTION FOUND IN GROUND SQUIRREL AND FLEAS FROM GROUND SQUIBRELS IN IDAHO

Under date of June 9, 1938, Senior Surg. C. R. Eskey, reported plague infection found in ground squirrel, *Citellus armatus* and in fleas from ground squirrels in Bear Lake County, Idaho, as follows:

- Tissue obtained from one ground squirrel shot May 25, 1938, 2 miles south of Bern.
- Five fleas collected from one ground squirrel shot May 25, 1938, 2 miles south of Bern.

Eighty fleas collected from 67 ground squirrels shot May 27, 1938, 5 to 8 miles southeast of Montpelier.

PLAGUE INFECTION FOUND IN FLEAS FROM GROUND SQUIRRELS IN MONTANA

Under date of June 9, 1938, Senior Surg. C. R. Eskey reported plague infection found in 284 fleas collected from 88 ground squirrels, *Citellus elegans*, shot 9 miles northwest of West Yellowstone, Gallatin County, Mont.

PLAGUE INFECTION FOUND IN FLEAS FROM DESERT WOOD RATS IN UTAH

Under date of June 9, 1938, Senior Surg. C. R. Eskey reported plague infection found in 7 fleas collected May 20, 1938, from 15 desert wood rats, *Neotoma desertorum*, shot and trapped 11 to 14 miles northwest of Kanab, Kane County, Utah.

CASES OF VENEREAL DISEASES REPORTED FOR APRIL 1938

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

Reports from States

	9 y p	hilis	Gone	orrhea
	Cases re- ported during month	Monthly case rates per 10,000 population	Cases re- ported during month	Monthl case rate per 10,00 populatio
labama	2, 574	8.89	318	1
rizona 1				
rkansas	1,881	9.18	316	1.
alifornia	2, 108 220	3.43 2.05	1, 331 143	2
Colorado		1.22		
Connecticut	213		81	
Delaware	235 221	9.00	37	1.
District of Columbia		3.52	144	2
lorida ²	2,455	14.70	256	1.
leorgia	2,607	8.45	445	1.
daho	68	1.38	23	
llinois	1,952	2.48	931	1.
ndiana	431	1.24	80	
0W8 1	365	1.43	175	
Cansas	303	1.63	94	
Centucky	747	2.56	276	•
ouisiana	2, 164	10.15	105	
faine	75	. 88	40	
faryland	1, 130	6.73	260	1.
Assachusetts	516	1.17	399	
fichigan	1, 214	2.51	479	
(innesota	255	. 96	198	
Lississippi	2, 530	12.51	2, 234	11.
lissouri	923	2.31	114	
Iontana 1				
lebraska	82	. 60	81	
levada	24	2.38	4	
New Hampshire	22	. 43	8	•
lew Jersey	927	2.13	224	
lew Mexico	88	2.09	60	1.
lew York	4, 960	3.83	1, 590	1.
orth Carolina	3, 833	10.98	631	ī
forth Dakota	29	.41	. 38	•
hio ⁹	1, 856	2.76	428	
klahoma ³	635	2.49	287	1.
regon	85	.83	119	1.
ennsylvania ¹	1, 906	1.87	225	
hode Island	103	1. 51	52	•
outh Carolina '				
outh Dakota	33	.48	23	
ennessee	1, 376	4.76	401	1.
ezes	2, 950	4.78	832	1.
tah	24	. 46	25	•
ermont	19	. 50	16	
irginia	1, 125	4.16	253	
Vashington	244	1.47	199	1.
Vest Virginia ^a	433	2.32	143	
isconsin a	45	. 15	106	•
yoming 1	7	. 30	4	•
Total	45, 993	3.64	14, 260	1.

See footnotes at end of table.

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Reports from cities of 200,000 population or over

	Syp	hilis	Gond	orrhea
	Cases re- ported during month	Monthly case rates per 10,000 population	Cases re- ported during month	Monthly case rates per 10,000 population
Akron, Ohio 1				
Atlanta, Ga.1				
Baltimore, Md	778	4.43	158	1.91
Birmingham, Ala	318	11.26	46	1.6
Boston, Mass	191	2.42	149	
Buffalo, N. Y.	123	2.08	149 50	1.88
Chicago, Ill	1, 135	2.08 3.18	676	.84
Cincinnati, Ohio	250	5.36		1.90
Cleveland, Ohio ¹		0. 30	94	2. 02
Celumbus Ohio				
Columbus, Ohio	62	2.03	18	. 59
Dallas, Tex.	331	11. 43	78	2.69
Dayton, Ohio 1				
Denver, Colo	51	1.72	31	1.04
Detroit, Mich	596	3. 44	229	1. 32
Houston, Tex.4	321	9.58	83	2.49
Indienapolis, Ind	34	. 90	35	. 93
Jersey City, N. J. ¹				
Kansas City, Mo	85	2.02	6	. 14
Los Angeles, Calif	768	5. 37	466	3. 26
Louisville, Ky	352	10.86	87	2.69
Memphis, Tenn	428	16.03	90	3. 37
Milwaukee, Wis. ³				0.01
Minneapolis, Minn	76	1.56	58	1. 19
Newark, N. J	293	6. 32	129	2.78
New Orleans, La	54	1. 13	42	. 83
New York, N. Y.	3.428	4, 69	1, 144	1. 57
Oakland, Calif. ¹	0,		-,	1.01
Omaha, Nebr	47	2, 13	36	1.63
Philadelphia, Pa	628	3. 16		1.00
Pittsburgh, Pa	238	3.48	25	. 37
Portland, Oreg.1		0. 10	~	. 0/
Providence, R. I	45	1. 74	37	1 40
Dophersten N V				1. 43
Rochester, N. Y St. Louis, Mo	44	1.31	25	.74
	322	3.85	78	. 93
St. Paul, Minn	43	1.52	22	. 78
San Antonio, Tex	128	5.09	56	2.23
San Francisco, Calif	151	2.25	156	2.33
Seattle, Wash	152	4.00	93	2.45
Syracuse, N. Y	78	3. 58	19	. 87
Toledo, Óhio 1				
Washington, D. C.	221	3. 52	144	2.30

No report for current month.
 Incomplete.
 Only cases of syphilis in the infectious stage are reported.
 Reported by Jefferson Davis Hospital.
 No report during present fiscal year.
 Reported by social hygiene clinic.

WEEKLY REPORTS FROM CITIES

City reports for week ended June 4, 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.

	Diph-	Inf	uenza	Mea-	Pneu-	Scar-	Small-	Tuber-	Ty- pheid	Whoop-	Deaths
State and city	theria cases	Cases	Deaths	sles cases	monia deaths	fever cases	pox cases	culosis deaths	fover cases	cough cases	all causes
Data for 90 cities: 5-year average Current week	162 105	77 45	30 30	5, 268 5, 491	518 873	1, 8 24 1, 167	14 25	409 366	34 36	1, 318 1, 202	
Maine: Portland New Hampshire:	0		0	23	1	1	0	1	0	5	27
Concord Manchester Nashua	0000		0 0 0	0 0 0	0 1	0 2 0	0	0	0	0	8 15
Vermont: Barre Burlington	0		0	0	1 0 0	0	0	1 0 0	0 0	0 0 0	4
Rutland Massachusetts: Boston	ů o		ů 0	0 197	0	0 95	Ŭ 0	0	1 0 0	0	4 191
Fall River Springfield Worcester	0 0 1	 	000	197 0 47 4	1 0 3	4 2 32	000	5 1 0 2	0000	15 4 12 15	191 32 35 41
Rhode Island: Pawtucket Providence	0		0	0	0	2	0	03	0	13 0 20	17
Connecticut: Bridgeport Hartford	0		02	42	1	5 23	0	1 2	0	0	27 33
New Haven New York:	ŏ		ō	5	3	3	Ŏ	ī	ŏ	13	36
Buffalo New York Rochester	0 16 0 0	2	0 2 0 0	8 1, 971 59 96	9 91 7 1	36 206 19 5	0 0 0 0	1 80 2 1	0 1 0 0	6 233 4 6	153 1, 411 75 55
Syracuse New Jersey: Camden Newark Trenton	3 0 0	<u>-</u> 1	0	9 21 1	2 7 2	6 12 6	0	0 2 3	0 0 1	9 27 0	83 87 33
Pennsylvania: Philadelphia Pittsburgh	9	4	3	444 25	18 10	81 24	0	22 14	82	19 32	408 154
Reading Scranton	000		0	35 1	••••	1	0	1	0	71	29
Ohio: Cincinnati Cleveland Columbus Toledo Indiana:	· 3 1 0 0	5 2 1	0 0 2 1	14 239 12 69	5 13 4 2	6 33 5 6	0 0 0 0	8 7 5 3	0 1 0 0	7 46 0. 23	126 198 98 67
Anderson Fort Wayne Indianapolis Muncie	00000		0 0 0 0	4 40 130 0 33	1 3 5 2 0	3 8 13 0 0	0 0 1 0	0 0 10 0	0 0 1 0	0 0 6 0	14 29 96 10
South Bend Terre Haute Illinois: Alton	0 1 0		0 0	33 2 0	ŏ	1	01	0	0 0 0	0	6 18 5
Chicago Elgin Moline Springfield	17 0 0	5	5 0 0	165 1 5 5	25 1 0 2	167 4 4 3	0 0 0 1	43 0 0	0000	122 2 1 5	695 7 4 21
Michigan: Detroit Flint Grand Rapida.	4 0 0	1	0 0 0	162 134 126	12 4 2	95 10 8	0	24 0 0	0000	153 9 4	257 27 32
Wisconsin: Kenosha Madison Milwaukee Racine. Superior	0 0 0 0		000000000000000000000000000000000000000	63 207 20 116 33	0 0 2 0 0	0 3 31 12 0	000000000000000000000000000000000000000	0 0 4 0	0 0 0 0	4 6 53 13 2	9 8 105 11 14
Minnesota: Duluth Minnespolis St. Paul	0		000	21 282 5	2 4 0	0 20 5	0 13 0	0 2 1	1 0 0	4 8 16	26 94 67

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City reports for week ended June 4, 1938-Continued

State and city	Diph- theria	Inf	luenza	Mea-	Pneu- monia	Scar- let	Small- DCX	Tuber-	Ty- phoid	Whooping	Deaths,
State and city	Cases	Cases	Deaths	6105 C8365	deaths	fever CE 505	cases	deaths	fever cases	cough cases	all Causes
Iowa:											
Cedar Rapids	0			13		1	0		0	5	
Davenport Des Moines	0		0	0 27	0	0 14	09	0	0	0	
Sioux City	ŏ		v	105	۱ v	4	ð	l v	ŏ	12	22
Waterloo	ŏ			6		8	ŏ		Ő	2	
Missouri:	•			Ŭ		v	, v		·	-	
Kansas City	0		0	. 2	4	5	0	8	0	0	85
St. Joseph	Q	<u>i</u>	0	0	1	0	0	0	0	0	13
St. Louis	2	1	1	3	6	30	6	12	1	2	167
North Dakota: Fargo	0		o	1	1	. 0	0	0	0	1	Ι.
Grand Forks	ŏ		, v	9	I 1	ŏ	ŏ		ŏ	ō	8
Minot	ŏ		0	3	0	ŏ	ŏ	0	ŏ	š	4
South Dakota:	•		Ť	•	'	•			, i	•	•
Aberdeen	0			0		1	0		0	0	
Sioux Falls	0		0	0	0	1	0	0	0	0	11
Nebraska:	•			-						-	
Lincoln	0			30 132	<u>-</u> -	3	0		0	2	
Omaha Cansas:	0		0	132	4	0	0	1	0	1	65
Lawrence	0		0	6	!	0	0	1	0	1	
Topeka	ŏ		ŏ	45		ĭ	ŏ	ō	ŏ	13	1 27
Wichita	ŏ		ŏl	36	l il	3	ĭl	ŏl	ŏ	12	21
1					-	-	-	-	-		
)elaware:											
Wilmington	0		0	1	8	8	0	0	1	5	26
faryland:				~							
Baltimore Cumberland	0	1	0	22 9	11	32	0	13	0	85	199
The destals	0		0	ŏ	0 0	0 0		8	Ô	0	13
istrict of Colum-			•	v		, v			•	0	3
bia: Washington_	6	2	0	- 44	4	11	0	8	1	12	135
irginia:	° I	~	•		-		°	°	- 1		100
Lynchburg	0		1	0	0	. 0	0	0	0	2	15
Norfolk	ō		ōl	ŏ	i	4	ŏ	i	ŏ	2	2 0
Richmond	2		Ő I	170	2	i	Ŏ	4	ŏ	ō	44
Roanoke	0		0	0	2	2	0	1	Ó I	Ó	21
est Virginia:											
Charleston	0		0	0	4	0	0	0	2	0	16
Wheeling	0		0	9	0	0	0	0	0	8	21
orth Carolina: Gastonia	0			7		0	0		0	3	
Raleigh	ŏ		0	16	1	ŏ	ŏ	i	ŏ	15	22
Wilmington	ŏ		ŏ	6	ô	ŏl	ŏ	il	ŏ	5	13
Winston-Salem	ŏ		ŏl	36	ŏl	ŏl	ŏ	ô	ŏl	18	10
outh Carolina:	- 1		-		· ·	-	-	×		~	10
Charleston	0		1	1	2	1	0	0	0	0	22
Florence	0		0	3	1	0	Ő	0	Ó	0	8
Greenville	0		0	4	0	0	0	0	0	3	8
eorgia:											
Atlanta	0	2	1	1	2	1	0	2	3	9	58
Brunswick	0		0	21	0	0 0	0	Q	02	õ	3
lorida:	•			4	1		• 1	5	2	7	32
Miami	0	1	0	ol	3	ol	0	o	1	7	39
Tampa	š l	-	ŏ	Š I	ĭl	ŏ	ŏl	ĭ	il	il	22
			-			-	-	-	-	- 1	
entucky:			1								
Ashland	1.	-		0		0	0 .		0	0 .	
Covington	2		0	0	0	1	0	2	0	0	6
Lexington	02		0	.9	0	0	0	1	0	1	19
EDDessee:	2	1	0	31	6	7	0	6	0	4	65
Knorville	0		o	11		10	o		ol		90
Memphis	ŏl	3	2	- 4	ĩ	3	ŏ	3	ŏl	3	30 97
Nashville	ŏl.		ōl	13	5	ŏ	ŏ	2	ŏ	2	54
abama:	- T		-		-		- T	- 1	-	-	•••
Birmingham	1	5	0	11	6	1	0	0	0	0	47
Mobile	0 -		0	0	0	0	0	0	Ó	0	10
Montgomery	1.			3 -		0	0 -		1	8 -	
kansas:	1	1				1	1				
Fort Smith	0	1		3 _		0	0	1	0	0 -	
Little Rock	ŏ	-	0	1	5	ŏ	ŏ -	8	ŏl	ŏ	
	- i -		۲ ۲	- 1	"	1	°	°	v	- 1-	
ouisiana: Lake Charles	0	0 -		1	0	0	o	0	0	0	2
ouisiana:	030	0 8	30	1 11 0	0 7 2	000	000	0 11 2	0 2 2	0 9 0	2 129 36

City reports for	week ended June 4,	1958—Continued
City reporte jur	ween chucu v une 4,	

	Diph	• • • • • • •	luenza	Mea- ales	Pneu- monia	Scar- let	Small		Ty- phoid	Whooping	Deaths
State and city	Cases		Deaths	sues Cases	deaths	fever cases	pox cases	culosis deaths	' Comon	cough cases	all causes
Oklahoma: Oklahoma City Tulsa.	0		0	8 43	5	20	07	2	0	1 8	41
Dallas	1		4	5	4	11	0	2	3	5	69
Fort Worth Galveston	0		0	1 0	5 5	8 0	0	6 0	0		48
Heuston	1		0	0	4	3 0	1	5 7	1	07	15 70 82
Montana:	-										
Billings Great Falls	0		0	02	02	0 1	0	1	0	5 5	7 11
Helena. Missoula	0		0	0	Ö	5 1	0	0	0	0	63
Idaho: Boise Colorado:	0		0	0	0	1	0	0	0	0	5
Colorado Springs	0		0	0	0	0	. 0	0	0	3	21
Denver Pueblo	9		1	19 19	ő	16 0	0	4	0	9 5	73 4
New Mexico: Albuquerque	0		0	1	8	0	0	6	0	1	18
Utah: Salt Lake City.	0		0	193	3	5	0	1	0	10	34
Washington:											
Seattle	000		1 0 0	3 8 . 0	2 8 1	1 0 1	010	0 0 1	1 0 0	39 5 3	85 28 26
Oregon: Portland	0		2	26	-5	9	8	1	0	- 5	93
Salem California:	Ō			2		Ŏ	ŏ		ŏ	Ŏ	
Los Angeles Sacramento San Francisco	12 0 8	8 1	1 0 0	53 84 3	11 0 5	29 0 14	0 0 0	12 3 6	1 0 0	32 6 29	256 21 163
	1,	dening	coccus		<u> </u>				Mening	ococcus	
State and city		menin	gitis	Polio- mye-	H	State a	nd City		menir	gitis	Polio- mye-
	-	Cases	Deaths	litis Cases				Ţ.	Cases	Deaths	litis C3985
New York: New York		1	1	0	Tenn	essee:	le		1	0	0
Pennsylvania: Philadelphia		1	0		Alaba	<i>lemphi</i>	S		ō	ŏ	i
Ohio: Cincinnati		3	1	0	B		ham		1	0	0
Toledo		ŏ	ō	ĭ	Texas	lew Orl	eans		0	0	2
Chicago Springfield		2 1	8	0		louston			2	0	1
Lichigan:		- 1	0		H	lelena			1	1	0
Detroit		1		1	Color	enver_			0	0	1
St. Louis Maryland: Baltimore		2	0	0		ortland			1	1	0
District of Columbia:		0	1	0	Califo L		eles		1	0	0
Washington Kentucky:		2	2	0							
Louisville		1	0	0	1						

Encephalitis, epidemic or lethargic.-Cases: New York, 1. Pellegte.-Cases: Chicago, 1; Charleston, S. C., 1; Atlanta, 2; Savannah, 3; Birmingham, 3; Los Angeles, 1.

FOREIGN AND INSULAR

DENMARK

Notifiable diseases—January-March 1938.—During the months of January, February, and March 1938, cases of certain notifiable diseases were reported in Denmark as follows:

Disease	Janu- ary	Febru- ary	March	Disease	Janu- ary	Febru- ary	March
Cerebrospinal meningitis. Chickenpoz Diphtheria Epidemic encephalitis Erysipelas German measles. Gonorrhea Lymphogranuloma Malaria. Measles Mumps	5 1, 982 98 3 319 297 786 7, 152 3 6 6, 717 794	3 1, 806 72 2 305 657 6, 100 	10 1, 775 57 1 260 399 738 12, 311 	Paradysentery Paratyphoid fever Poliomyelitis Puerperal fever Scarlet fever Syphilis Tetanus, neonatorum Tetanus, traumatic Typhoid fever Undulant fever Weil's disease Whooping cough	15 2 7 28 1,009 58 6 3 46 3 1,122	20 4 5 22 694 54 3 47 1 1,022	28 6 581 34 3 45 1,076

ITALY

Communicable diseases—4 weeks ended March 27, 1938.—During the 4 weeks ended March 27, 1938, cases of certain communicable diseases were reported in Italy as follows:

Disease	Feb. 28- Mar. 6	Mar. 7-13	Mar. 14–20	Mar. 21-27
Anthrax Cerebrospinal meningitis. Chickenpox Diphtheria. Dysentery Hookworm disease Lethargic encephalitis. Mumps. Paratyphoid fever. Pellagra. Poliomyelitis. Puerperal fever. Scarlet fever. Typhoid fever. Undulant fever.	7 46 401 595 13 7 2 3,199 3,40 311 11 16 39 286 202 97	8 57 496 659 13 23 23 23 24 5 24 358 22 5 24 33 321 193 321 193	8 600 533 555 6 13 1 4,255 409 28 28 6 16 16 32 304 204 133	7 55 501 615 52 17 1 3,714 413 28 8 28 17 33 339 216 5128
Whooping cough	305	459	407	416

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

From medical officers of the Public Health Service, American consuls, International Office of Public Health, Pan American Sanitary Bureau, health section of the League of Nations, and other sources. The reports contained in the following table must not be considered as complete or final as regards either the list of countries included or the figures for the Faticular countries for which reports are given.

CHOLERA

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indicates cases:

			ares cas	O Indicates cases; D, deatns; F, present	auns; P.	preen	c]										
	Oet.		Ę							Week ended-	-beba						
Place	Nov.	ෂ් ^{සු} ස්	26, 1937- Jan. 29,	\$£		March 1938	1938			April 1038	1038			A.	May 1938	*	
	1937		90AT		10	13	91	8	~		8		8		1		8
Afghanistan. ¹ China: Kone	7		c										 				'
Kwangchow Wan	51		•														•
	29																
	101	ឌ។	со С										-			ca 🛥	89 4
Dutch East Indies: Macazzar. O	A 200	R 226	0 330	A 710	745		1				18					-	
	3, 737	25 24 24	4		82			<u>.</u>	5	125 2 373		<u> </u>	<u> </u>	<u> </u>	<u> </u> -		•
	23	89 8	262 141	<u>4</u> 8	12 %	22	84	- <u>8</u> 8	<u>ខ្ល</u> ាន:	258	88	28°5.	. <u>8</u> 8	28 28	-ន្មន	- 288.	•
Bombay Fresidency	363 171	161	97 P	4.64	18	40	80		148	- 4 01	<u> </u>				$\frac{1}{111}$	•	-
Calotta - Calott	8	8 8	21 22	226	8	118	35	ลี	33	134	<u>1</u> 21	8	27-	-18	0	5	102
Central Provinces and Berar.	866 1	113	\$	99 1	11	9	61	132	198	1200	643 9	963 1, 3	307 1, G	008 1, 007 17		28 7 28 7	1, 904
Jodhpur Madras Presidency	1, 204	2, 654 1, 178	8, 914 1, 804	967 466	128	ន្តន	23	84	38	22	305 5	262			~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~		
i Obolera reported present early in June in South Afghanistan. Afghanistan	nistan, /	Afghanis	an.					:	•		:						

3 Under date of June 7, 1938, the American Consul at Swatow reported approximately 200 cases of cholera with 50 deaths, in Swatow, China, for the period May 29-June 6, 1938. 8 Inported: 4 Imported:

FEVER —Continued	
YELLOW	
AND Y	
FEVER,	
TYPHUS	
PLAGUE, SMALLPOX,	
PLAGUE,	
CHOLERA,	

CHOLERA-Continued

/ [C indicates cases; D, deaths; P, prosent]

	Oet.	Nov.	, L	Jan.						Week	Week ended	1				;	
Place	Zov.	ងខ្ព័ន	26, 1937- Jan. 29,	şĘ.		March 1938	1938			Υb	April 1938				May 1938	88	
	1937	1937	90AT	1938	, ۵	12	19	*	8	•	16	ន	8		3	31	8
India—Continued. Madrae	55 F	91 04	8 8 8	16 8		6169	-		-				4.00		-		
	91	39	11	82	8	8	31	ន	83	2.	88	1	-85	58E	82 197	ន្តនទួ	8
Rantoon Sind State	-		6								1 -		3 7	64	8	<u>;</u>	4
			r.	1 0		1	4			-	9		6				
			•*	•	16	9	1	-1000	6	•							
Andornius (Franch): Annam Province. Tonkin Province.	2, 364 44	1 ⁵⁰ 4	88	338 55		CN 00	*8	170	28	28g	121	28	88 8	213 643	25	88	110
Biam. Banoi	361	39	~	~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~			2	7	28	11	13	8	3	3	8	8	8
On vareale:																	

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66444*

¹ Including plague in the United States and its possessions.
² Pneumonic.
³ Information dated May 9, 1938, states that an outbreak of bubonic plague has occurred in Kochow District, and on Hainan Island, China.
⁴ Information dated May 9, 1938, states that an outbreak of bubonic plague has occurred in Kochow District, and on Hainan Island, China.
⁴ Suspected.

PLAGUE 1

June 24, 1938

EVER —Continued	
YELLOW F	
FEVER, AND	
X, TYPHUS	
SMALLPO	
A, PLAGUE,	
CHOLER	

PLAGUE-Continued (C indicates cases; D, desths; P, present]

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	Oet.		ž	Jan.					Þ	Week ended-	led-					
Place	81- Nov. 27.	ส่อี่ส่	26, 1987- Jan. 20,	ବ୍ଳ କୁ କୁ	-	March 1938	808			April 1938	888			Ma	May 1938	
	1687		9941	1988	20	12	19	8		8	8	8	-	1	8	8
Hawaii Territory:' Plague-infected rata: Hawaii IslandHamakua District: Hamakua Mill Bactor		Ċ														
Takasan Pasuhau Sector	9	64.0	80	60	-	. 69			8-	-					~	
Paraten Societ Maul Inland:										-	<u> </u>		<u> </u>	<u> </u>		
		0.	-	+					<u> </u>		<u> </u>	-		<u> </u>		<u> </u>
	1,874	446 658	1, 968 965	8, 482 1, 582	2657 2657	1022	1,003 1, 618	484 8	774 54 883 15	547 560 188 283				<u> </u>		
Automoted C			- 0	6	-											
	22 <u>5</u>	56g	42 3	838 2 - 1	స్లంజి	413 °11	gi Po œ	8 222	50°1	15 13 6 176 250 176		1.08 2	2	31		
Occhin. Cochine Presidency	-15.38	160 84	830 174	157 69	23	10-1	22	<u> </u>	35	1	<u> </u>	00 -				
			4 4			25		99 99	$\frac{11}{11}$		$\frac{11}{11}$	++			<u> </u>	<u> </u>
				~~~	64	-	•	ca				<u></u>				
Inq: Bapbdad-Plaqueinfortd rata Madamary. (Bea table below.) Niger Territory. (Bea table below.) Purr. (Baa table below.)			1				-	<u> </u>				<u></u>	<u> </u>	<u> </u>		<u> </u>
Trunken: Tunka: Plagra-Infocted rata		-	1.	13	-			3				<u></u>	-		- 18	

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During the week ended Nov. 20, 1837, plague infection was proved in 10 rats by mass inoculation in Omsopio, Makawaso District, Maul Island, Hawall Territory.
For 3 week.
For 4 week.
Plague infection proved in insect hosts as follows: California—Fresno County, Oct. 7-Nov. 5; Santa Cruz County, Feb. 3-Apr. 27, 1388: Idako—Bear Lake County, May 25-27, 1988: Montens—Gallatin County, May 1885; Newdor Clark County, Apr. 14-22, 1688; Oregon—Baker County, Apr. 23-407, 24, 1088; Honkington—Clark County, Apr. 14-22, 1688; Oregon—Baker County, May 24, 20, 1888; Montens—Gallatin County, May 13-21, 1938; Utaka—East County, May 25, 1768, Nontens—Gallatin County, May 13-21, 1938; Utaka—East County, May 26, 1888; Montens—Gallatin County, May 13-21, 1938; Utaka—Kane 00000, May 1800; Neaper Clark County, Apr. 14-22, 1688; Oregon—Baker County, Apr. 23, 1938; Grant County, May 13-21, 1938; Utaka—Kane 00000; May 1808; Nonden Clark County, Apr. 7-30, 1888; Oregon—Baker County, Apr. 24, 1938; Oregon of Clark County, Apr. 7-30, 1888; Oregon—Baker County, Apr. 25, 1938; Oregon of Clark County, Apr. 7-30, 1888; Oregon of Clark cla

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<b>FEVER</b> —Continued
YELLOW
AND
FEVER,
TYPHUS
SMALLPOX,
PLAGUE,
CHOLERA,

### PLAGUE-Continued

(C indicates cases; D, deaths; P, present)

Place	Novem- ber 1937	Novem- Decem- Janu- Febru- March April ber 1837 ber 1837 ary 1838 ary 1838 [1838]	Janu- ary 1938	Febru- ary 1938	March 1938	April 1938	Place	Novem- ber 1987	Decem- ber 1987	Janu- ary 1938	Novem- Decem- Janu- Febru- March April ber 1037 ber 1037 ary 1038 ary 1038 1938 1938	March 1938	A pril 1688
Brasil: • Pernambuco State 0 Madagaacar (central region) 0 Niger Territory	8 67 85 85	82 86 80	66 65	28	26		Peru Anceah Department C Anceah Department C Lambayeque Department C Libertad Department C Lima Department C	811 8 <u>1</u> 8	• •	0	110	019 -1	***

⁹ For the year 1937, 35 cases of plague with 15 deaths were reported in Bratel as follows: Bahla State, 5 cases, 5 deaths; Cears State, 3 cases, 5 cases, 1 death; For-nambuco State, 28 cases, 9 deaths. Information dated Apr. 19, 1838, states that since Mar. 24, 4 deaths from bubonic plague have been reported in the Novo Exu District, Pernam-buco State, 28 cases, 9 deaths. Information dated Apr. 19, 1838, states that since Mar. 24, 4 deaths from bubonic plague have been reported in the Novo Exu District, Pernam-buco State, 28 cases, 9 deaths. Information dated Apr. 19, 1838, states that since Mar. 24, 4 deaths from bubonic plague have been reported in the Novo Exu District, Pernam-buco State, 28 cases, 9 deaths.

### SMALLPOX

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[C indicates cases; D, deaths; P, present]

	të O			Jen.					P	Week ended-	1			-		
Place	Nov.	ෂ්ට්ස්	26, 1937- Jan. 20,	ଞ୍ଚଳିଛ		March 1938	1938			April 1938	888			May	May 1938	
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			12	29													-	**	3	23 23 23 23	22	5*	°8	-	2
	11		53	8-	3											3, 193	144	<b>\$</b>	<b>1</b> E	83 83 83	នដ	115	<b>2</b>	-	1
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	10		102	112	3						er					2, 927	-0	18	1		8¥			- 00	
	32		131	131	8								-	-	1	3, 833	33	81	88	83 2	88	191	25	N 100	2
	17	•	236	192	2								2			3, 307	22	22	753	212 212	201	5	* 8	~	2
	30		214	123	1						•					2,920		ž	842	28 8	135	3.	-8.		-
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Queboo Saskatohewan	China: Canton	Procise Contraction of the second sec	HAUKOW Bong Kong	Macoo	Shanghai	Plantein	Colombia (see also table below): Barranquilla	Dutch East Indies: Batavia.	Ecuador: Guayaquíl Fent	Alexandria	Port Said	France: (See table below.) Greet Britain: England and Wales-	Kent County-Gravesend	Leighton Buzzard. Port of London	Greece. (See table below.) Guatamata. (See table below.)	dia.	4 National	Assam	Bombay Presidency.	Bombay	Calcutta	Caminora	Central Provinces and Berar	Delhi	Karachi Veder Breiden

For 2 weeks. Imported. For 3 weeks.

June 24, 1938

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<b>FEVER</b> -Continued
YELLOW
AND
FEVER,
TYPHUS
SMALLPOX,
PLAGUE,
CHOLERA,

# SMALLPOX-Continued

[C indicates cases; D, deaths; P, present]

										Week	Week ended-	Ι.					I
Place Place	North Contract		26, 1087- Jan. 20,	ቔ፟፟፟ዿኇ፝ቔ		March 1938	1968			₫	April 1938				May 1938	8	
	1937				10	2	9	8	- 64	•	9	8	8	~		R	*
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Van Ora	<u>'</u>								Π			Π		Π	Ī		

37     4     5     7       1     1     28     1     1       28     37     1     28       21     1     28       21     1     28       21     1     28       21     1     28       21     1     28       21     1     28       21     1     1       22     1     1       23     2     1       23     1     1       23     1     1       24     1     1       25     1     1       26     1     1       27     1     1       28     1     1       29     1     1       21     1     1       26     1     1       27     1     1       28     1     1       29     1     1       21     1     1       21     1     1       28     1     1       29     1     1       21     1     1       21     1     1       21     1     1       21	On vessels.       On vessels.       On vessels.       On vessels.       Continued.       B. Hair Altray at Singapore from Amoy. Swatow, and From Karr. 9, 103         B. B. Arman at Singapore from Hong Kong.       B. B. Arman Altray at Singapore from Hong Kong.       B. B. Arman Altray at Singapore from Hong Kong.       B. B. Arman Altray at Singapore from Hong Kong.       B. B. Arman Altray at Singapore from Hong Kong.       B. B. Arman Altray at Singapore from Hong Kong.       B. B. Arman Altray at Singapore from Hong Kong.       B. B. Arman Altray at Singapore from Hong Kong.       B. B. Arman Altray Kong.       B. Arman Altray A
Morocco.(See table below.)C29136104Nystakind.IdentifiedC1183Nystakind.CC1183Nystakind.CC1183Norugress East Africa.Cee table below.)C324Salvador.CS2476Balvador.CSC324Sintal.Contagress East Africa.Cee table below.)C324Balvador.CSCS276Sintal.ContagressC12111413Southern Rhodesia.CSCS2Union of South Africa.Csee table below.)CS24Venesulai.Puerto Cabello.4SCS2114Union of South Africa.Csee table below.)CS211413Venesulai.Puerto Cabello.4SSS54	On vessels: 8. Erre at Rangcon from Calcutta. 8. Riteroral at Singapore from Hong Kong. 8. Riteroral at Singapore from Hong Kong. 8. Riteroral at Kannara. 8. Riteroral at Rangapore from Hong Kong. 8. Riteroral at Neuron Hong Kong. 8. Riteroral at Aden from Bounbay. 8. Riteroral at Riteroral from Pout Biology. 8. Seringe at Singapore from Hong Kong. 8. Seringe at Singapore from Amoy, Swatow, and 1 case. 8. Kon Huttar at Singapore from Amoy, Swatow, and 1 case. 1 case. 1 case. 8. Seringe at Riteroral at the stanalpox is present from Barqued in Paular at From Port Sata, Venezuela, and that smallpox is present from Barqued in Barquistimeto, Lara Stata, Venezuela, and that smallpox is present from Barqued in Barquistimeto, Lara Stata, Venezuela, and that smallpox is present from Barqued in Paula. 1 case. 1 case. 1 case. 1 case. 1 case. 1 case. 1 case. 1 case. 1 case.

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

## BMALLPOX-Continued

, [O indicates cases; D, deaths; P, present]

No- Vembe 1837	r Decem-	Janu- 1968 1968	Febru- 1988 1988	March 1938	April 1038	Place	No- Vember 1937	Decem- ber 1887	Janu- ary 1038	Febru- ary 1638	March 1988	April 1938
14	7	10	8 5			Mexteo-Continued: Hidalgo State	~				1	
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				•	<b>4</b> -	Nayarit State						
			1010	8		Queretaro State				-	<b>0</b> -1	
				-		Tabazoo State						
	4	10	1	7		Vera Cruz State	8-1					
					-		- 64					•
53	200 200 200	<b>2</b> 22	22	- -		Portugal (see also table above) O	g-••	<b>ğm</b>	34	2		
5			-			Salvador	•		16	42	2	
122				•		Union of South Africa: Cape Province				1	5	
×								4				

· For January and February.

TYPHUS FEVER

[C indicates cases; D, deaths; P, present]

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	April 1988	9	<b>4</b> 8 <b>6 1</b>
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	March 1988	10	
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		8	102-2 NB
	February 1958	10	0-1
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Oet.	81- Nov. 27,	1987	4 n 280% 200 200 200 200 200 200 200 200 200 2
	Place	-	Agerta: Algere Department. Algere Department. Constantine Department. Bons Prilipyerila. Bont Department. Prilipyerila. Bonthen Territor. Baltran (Se table Balov.) Britian East Africa: Kenya Concorpeden Frovince. Ontoepeden Frovince. Austran. Mallee Province. Mallee Province. Mallee Province. Mallee Province. Mallee Province. Datran. China (see also table below): Datran. Datran. Datran.

FEVER-Continued
YELLOW
<b>UND</b>
FEVER,
TYPHUS
SMALLPOX,
PLAGUE, 8
CHOLERA,

# **TYPHUS FEVER-Continued**

[C indicates cases; D, deaths; P, present]

	Oet:	Nov.	Å.							M	Week ended-								
Place	24. 204.	ෂ්රී් ස්	sğigs t		February 1938	y 1938			March 1938	1938			April 1938	1938			May 1938	888	1
	1937	1937	1938	10	13	10	38	8	13	10	8	-		16	<b>8</b>	8			
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Marcocoo (see also table below)	8	315 24	743 70	131	<b>42</b>	<b>208</b> <b>67</b>	240 17	<b>8</b> 38	411 70	372   78	36	57	199	583 283	100	165	162	17.5	173
		00	-											<u> </u>	<u> </u>	<u> </u>	-	<u>:</u> 	-
Poland O	81	3	5	131	-8	-8	150	-3	75	-5	176	130	3	!	69 g	1	<b>8</b>		-
Portugal. (See table below.) Rimaria (See table below.)		~	8		6		10	~	"	=	•			~		80			
Bierra Leone: Preetown. Bierra Leone: Preetown. Bitraite Settlementa: Binranora		-	e													_	_		
877a: Deires-Zor Trans-Jordan			_	•		-	2						-						
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Turkey. (See table below.)	161	22	1	5	22	123	123	. <u>6</u>	- 77	22	. <del>3</del>	22	-8	•	<b>"</b> %	°5	•å	:: 8≪	
Unput of south Africa. (See table below.) Yugoadayis: BelgradeC	-																		
B. B. Blackhill at Philippeville.			1	Ì										1					
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CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER-Continued

# **TYPHUS FEVER-Continued**

[C indicates cases; D, deaths; P, present]

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² For January and February. ³ Tropical typhus fever. YELLOW FEVER

[C indicates cases; D, deaths; P, present]

	č										Week	Week ended	1							
Place	81- 81- 80V.	N N N N N N N N N N N N N N N N N N N	27, 1937- Jan.	<b>P</b>	ebrua	February 1938			March 1938	1938			Υb	April 1938				May 1938	1938	
				20	12	19	8	2	13	61	8	3	•	16	8	R	7	14	21	8
Belgian Congo: Baratumba Zongo					17			==												
			16	16	* 0	-8	17	8	2	9	•			6			6.0			
Para State		1	-10	~~~	80 A	64	61		=	ğa	13	*-	69	-1 00	- 60		-	-		
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Bantander Department		2 2											-		-			Ī		
Gambia: Georgetown	00 <b>60</b> •	99	10.												===		-	= -		
			•	1		1			F			ÎÎ			:			•		
m ttions (near Binger-		1	4						64 6											
1 Suspected.									1 2 900						-					

³ See also reports of yellow fover in Brasil in preceding issues of the PUNISC HEALTE REPORTS for 1988 and 1937. ⁴ Includes 1 suspected case.

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<b>FEVER</b> —Continued
YELLOW
AND
FEVER,
TYPHUS
SMALLPOX,
PLAGUE,
CHOLERA,

# YELLOW FEVER-Continued

[C indicates cases; D, deaths; P, present]

	Oct.	N07.	Dec.			-				M	Week ended —	ded-								
Place	31- Nov. 27.	85.55 Dec	Jan. Jan	щ	February 1938	y 1938			March 1938	1938			μ	April 1938			4	May 1938	33	
	1937	1937	1938	5	12	19	26	5	12	19	26	2	a	16	23	30	7	14	21	82
Nigeria. C	- 75	11	8°3																	
Paraguay: AsuncionC		3	•			11														
Dakar. Diourbel.	57 F					11														
Rufisque C Thies C Sierra Leone: Kailahun	71																	11		
Sudan (French)	11	1																		
1 Suspected. a Tridindes 1 suspected	_	_					-	-	-	-	-	-	-	-	-	-	-		-	1

^a Includes 1 suspected case. ⁴ Includes 3 suspected cases. ^b Imported.

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