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## MORTALITY FROM RHEUMATIC HEART DISEASE IN PHILADELPHIA DURING 1936<sup>1</sup>

## By O. F. HEDLEY, Passed Assistant Surgeon, United States Public Health Service

Rheumatic heart disease is the forgotten health problem. Despite the intense interest shown by clinical investigators and pathologists, health officers have been slow to recognize its importance. In this article an attempt is made to appraise rheumatic heart disease as a cause of death, to compare it with other public health problems, and to study the distribution of deaths from this cause during 1 year in a city of 2,000,000 population.

The chief difficulties in determining the death rate from rheumatic heart disease arise from the failure of physicians to report deaths as "rheumatic heart disease" and because the International List of Causes of Death does not contain rubrics for the tabulation of deaths from rheumatic heart disease when reported on the basis of etiology. It has been pointed out that this could be largely overcome by providing additional subtitles under titles 90–95 of the International List for recording deaths from this condition (1).

In December 1935 cards were sent to physicians, practicing in Philadelphia, requesting that they report deaths due to rheumatic heart disease as such. Notices were also sent to the hospitals. The coroner's office agreed to participate in the study. Cooperation of the Philadelphia County Medical Society was obtained and notices were inserted in the Weekly Roster, with suggestions concerning criteria for diagnosis. These efforts were superfluous in many cases as the local medical profession as a whole is alert to this problem. Most of the hospitals and out-patient clinics use the American Heart Association nomenclature, and a large number of physicians diagnose heart disease on the basis of etiology in their daily practice.

#### DEATH RATE

Three hundred and fifty-seven deaths were reported as due to rheumatic heart disease, subacute bacterial endocarditis superimposed on rheumatic heart disease, rheumatic fever, and chorea in 1936 (table 1). Of this number, 334 deaths, or 93.6 percent, were reported as rheumatic heart disease, and 18 deaths, or 5.0 percent, were reported as subacute bacterial endocarditis superimposed on rheumatic heart

<sup>&</sup>lt;sup>1</sup> From the Office of Heart Disease Investigations, National Institute of Health. Branch office, 133 S. 8th St., Philadelphia, Pa.

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disease. As most cases of subacute bacterial endocarditis occur as a complication of rheumatic heart disease, they were included in this series when so reported. Four deaths reported as rheumatic fever were included. Deaths in rheumatic fever from causes other than pancarditis are extremely uncommon as are also deaths from Sydenham's chorea, one of which was included in this series.

 
 TABLE 1.—Reported causes and mean ages at death in 357 deaths from rheumatic heart disease in Philadelphia during the year 1936

Cause of death	Num- ber	Per- cent	Mean age (years)
Rheumatic heart disease	334	93.6	87. 2
	18	5.0	27. 7
	4	1.1	14. 0
	1	.3	8. 0
	857	100.0	36. 5

In addition to these 357 deaths attributed to rheumatic heart disease this condition was mentioned on 5 other death certificates as a secondary or contributory cause. These deaths were not included in any of the computations. Based on an estimated population of 2,028,511 the mortality rate was 17.6 per 100,000 population.

This is probably an underestimate of the total mortality as there were 195 other deaths not reported as rheumatic heart disease but regarded as presumably due to that cause (table 2). Sixty-eight of these were reported as subacute bacterial endocarditis, exclusive of cases superimposed on congenital cardiovascular defects or associated with septic conditions such as criminal abortions. Deaths from acute bacterial endocarditis, gonococcal endocarditis, and hemolytic streptococcal endocarditis were not included. White (2) states that 80 percent of cases of subacute bacterial endocarditis have a rheumatic background. Furthermore, rheumatic infection in childhood not infrequently runs a rapidly fulminating course terminating in subacute bacterial endocarditis, and is likely to be diagnosed as such at the time of death.

 
 TABLE 2.—Reported causes and mean ages at death in 195 deaths not reported as rheumatic heart disease but regarded as presumably due to that cause in Philadelphia during 1936

Cause of death	Num- ber	Per- cent	Mean age (years)
Subacute bacterial endocarditis. Endocarditis—under 30 years of age. Endocarditis—ver 30 years of age—of long duration Mitral stenosis (alone or in combination with other lesions) Adherent pericardium—of long duration Aortic stenosis (2 cases under 50 years; 1 over 50 years, of long duration)	68 28 41 52 8 8	84.9 14.4 21.0 26.7 1.5 1.5	85.7 17.4 47.2 58.2 40.7 39.7
	195	100.0	41.8

There were 28 deaths of persons under 30 years of age reported as due to endocarditis or valvular lesions. At least 90 percent of heart disease among persons under 30 years of age is due to rheumatic heart disease, the remainder to congenital defects and other less frequent causes. Since deaths from congenital cardiovascular defects are computed separately, practically all of the fatal cases of heart disease occurring under 30 years of age are probably of the rheumatic type.

Forty-one deaths in persons over 30 years of age were reported as endocarditis or valvular disease of several years' duration. While the opportunities for error are greater with each succeeding decade of life, most of these deaths are attributable to rheumatic heart disease. For example, a death at 35 years of age from valvular disease of 17 years' duration is probably the result of rheumatic infection. All of these deaths followed illness of at least 3 years' duration.

Fifty-two deaths were listed as mitral stenosis, either alone or in combination with other valvular lesions. Rheumatic infection is by far the leading cause of mitral stenosis. There is reason however to question the validity of some of these diagnoses. The mean age was 58.2 years, much older than is usually encountered for rheumatic heart disease. Auricular fibrillation and gallop rhythms in coronary arteriosclerotic and hypertensive heart disease are sometimes mistaken for mitral stenosis. White states that mitral diastolic murmurs occasionally result from a relative stenosis due to cardiac dilatation (2). Osler (3) and Cabot (4) point out that mitral stenosis is not necessarily incompatible, especially in women, with a ripe old age. Most of these deaths past 60 years were among white females. Mitral stenosis not infrequently develops insidiously, or else older persons forget about rheumatic episodes in childhood. Furthermore, there were probably more undiagnosed than misdiagnosed deaths from mitral stenosis. Cabot states that not over 50 percent are recognized during life.

There were also three deaths from adherent pericardium and three from aortic stenosis—two in persons under 50 years of age and one over 50 years—all of long duration.

There are undoubtedly included among these 195 deaths presumably due to rheumatic heart disease a number of deaths from other causes. Contrariwise, there were probably many other deaths from rheumatic heart disease not included in this series. These include deaths certified as pericarditis, adherent pericardium, endocarditis of undetermined duration, aortic stenosis which may be due to rheumatic infection or calcareous changes, aortic insufficiency due to rheumatic infection, syphilis, arteriosclerosis or hypertension, and deaths certified as myocarditis in which the valvular lesion is overlooked. Often it is impossible to obtain any history, and physical examinations on very jll patients are likely to be unsatisfactory. A number of deaths not included in the presumably rheumatic group were certified as "mitral insufficiency." Many were probably due to rheumatic heart disease, especially deaths occurring between 30 and 59 years of age.

When these two groups are combined, a total of 552 deaths is obtained. This represents a conservative estimate of the annual mortality from rheumatic heart disease in Philadelphia, a death rate of 27.2 per 100,000 population. This point is stressed because with

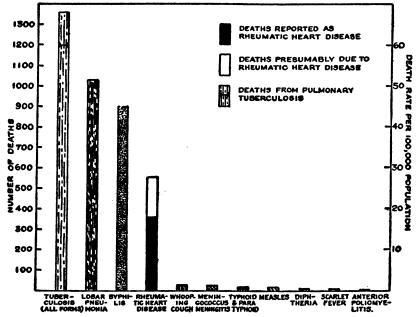


FIGURE 1.—Comparison of number of deaths at all ages and death rates per 100.000 population from rheumatic heart disease with other infectious diseases in Philadelphia during 1936.

better reporting the death rate will probably show an apparent increase. Several years will be required to stabilize the reported death rate.

## IMPORTANCE OF RHEUMATIC HEART DISEASE AS & PUBLIC HEALTH PROBLEM

A comparison is made in figure 1 of deaths from rheumatic heart disease with deaths from other important infectious diseases in Philadelphia during 1936. Tuberculosis (all forms) leads with 1,349 deaths, a mortality rate of 66.4 per 100,000 population. Pulmonary tuberculosis accounted for 1,194 deaths, a mortality rate of 58.8 per 100,000 population. Pneumonia (excluding bronchopneumonia) followed with 1,085 deaths, or 53.4 per 100,000 population. Of these, 1,032 were due to lobar pneumonia.

The death rate from syphilis is unknown. During 1936, 305 deaths were reported as due to syphilis, a death rate of 15.0 per 100,000

population. This is probably far below the actual rate. The Division of Venereal Diseases of the United States Public Health Service estimates that the death rate in the United States from cardiovascular syphilis alone is 30.7 per 100,000 population (5). Since Philadelphia has about the same ratio of colored population as the entire United States, this estimate is roughly applicable to this city. There were also 100 deaths, or 4.9 per 100,000 population, due to locomotor ataxia and general paralysis of the insane. Aneurysms other than of the heart, most of which are due to syphilis, were reported as the cause of 215 deaths, a mortality rate of 10.6 per 100,000 population. In addition, there were many still-births and neonatal deaths attributable to syphilis. It is therefore believed that the mortality rate from syphilis is greater than that from rheumatic heart disease in this locality.

There were 30 deaths, 1.5 per 100,000 population, from whooping cough; 26 deaths, 1.3 per 100,000, from meningococcus meningitis; 18 deaths, 0.9 per 100,000, from typhoid and paratyphoid fevers; 15 deaths, 0.7 per 100,000 population, from measles; 10 deaths, 0.5 per 100,000 population, from diphtheria; 7 deaths, 0.4 per 100,000, from scarlet fever; and 2 deaths, 0.1 per 100,000 population, from anterior poliomyelitis.

Among all the infectious diseases, rheumatic heart disease ranks fourth as a cause of death in Philadelphia, exceeded only by pulmonary tuberculosis, lobar pneumonia, and syphilis. Among the essentially chronic infectious diseases, only tuberculosis and syphilis cause more deaths. In some of the New England cities rheumatic heart disease probably exceeds syphilis, while in the South syphilis is a much more important factor.

Excluding lobar- and broncho-pneumonia, which result in an extremely high reported mortality under 2 years of age, and the diarrheas and enteritis of infancy, the mortality from rheumatic heart disease in persons under 20 years of age was exceeded only by all forms of tuberculosis and was greater than that for pulmonary tuberculosis (fig. 2). During 1936 there were 138 deaths in this age period from all forms of tuberculosis, of which 86 were due to pulmonary tuberculosis. There were 94 deaths reported as due to rheumatic heart disease. In the group regarded as presumably due to rheumatic heart disease there were 28 deaths under 20 years of age. These totaled 122 deaths, which is believed to be a very close approximation of the actual mortality from rheumatic heart disease for this age period.

Whooping cough ranked third, with 30 deaths in persons under 20 years of age. Twenty-nine deaths were reported as due to syphilis. Doubtless there were many more deaths from syphilis during the first two decades of life than are shown in mortality tables. It is improbable that syphilis results in as many deaths in this age group as

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rheumatic heart disease, especially if neonatal deaths from syphilis are excluded. Measles, resulting in 15 deaths under 20 years of age, meningococcus meningitis, with 12 deaths, diphtheria, with 10 deaths, scarlet fever, with 6 deaths, and anterior poliomyelitis, with 2 deaths, followed in the order listed. The year 1936 was a nonepidemic year for all of the acute communicable diseases, and these low figures do not always obtain. None of these conditions in recent years has exceeded the mortality from rheumatic heart disease during 1936.

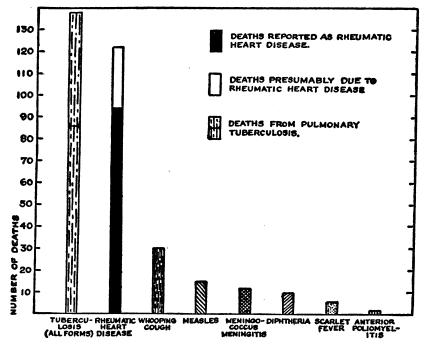


FIGURE 2.—Comparison of number of deaths in persons under 20 years of age from rheumatic heart disease with other leading infectious diseases during that period (excluding pneumonia and broncho-pneumonia, syphilis, and diarrhea and enteritis of infancy) in Philadelphia during 1936.

In figure 3 it is noted that rheumatic heart disease was responsible for more deaths in 1936 than the total for whooping cough, measles, meningococcus meningitis, diphtheria, scarlet fever, and anterior poliomyelitis. Doubtless this relationship does not exist in years when these diseases are very prevalent. Whether it holds over a number of years largely depends on the annual variations in the death rate from rheumatic heart disease. While the number of cases of rheumatic fever appears to vary from year to year, it is extremely doubtful whether the annual death rates fluctuate greatly, since the disease is essentially chronic and deaths usually occur after several years of illness.

# 1913

#### DISTRIBUTION OF DEATHS BY AGE, SEX, AND COLOR

The mean age at death of the 357 deaths reported as due to rheumatic heart disease was 36.5 years. This is 3 to 8 years above the average among hospital patients. Coombs (6), in Great Britain, in a

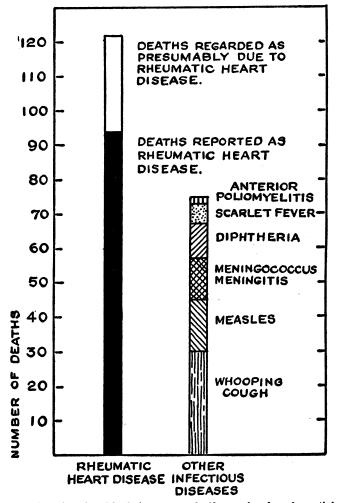


FIGURE 8.—Comparison of number of deaths in persons under 20 years of age from rheumatic heart disease with the total number of deaths during that period from other infectious diseases (excluding tuberculosis, pneumonia and broncho-pneumonia, syphilis, and diarrhea and enteritis of infancy) in Philadelphia during 1936.

series of 98 deaths from rheumatic heart disease examined post mortem found that the mean age at death was 28.3 years. The average age at death in Washington (D. C.) hospitals was 29.0 years (7). The mean age at death among hospital cases in Philadelphia during 1936 was 33.4 years, while among hospital cases in which death certificates were based on necropsy findings it was 33.0 years. The higher mean age at death is in part due to the relatively large number of deaths reported as "rheumatic heart disease—mitral stenosis" among white females past 60 years of age. Mean ages at death based on hospital statistics are usually a few years younger than among the general population. Older persons balk at "going to the hospital to die." Their sickness is frequently of long duration and the expense of hospitalization is almost prohibitive, especially in view of the prognosis.

One hundred and sixty-nine deaths from rheumatic heart disease, or 47.3 percent, were among males and 188, or 52.7 percent, among In reviewing 4,653 admissions to Philadelphia hospitals females. from 1930 to 1935 for rheumatic fever, chorea, rheumatic heart disease, and subacute bacterial endocarditis, it was found that 58 percent were females. Most writers agree that there is a slight but appreciable predominance among females. White and Jones (8) noted that 54.9 percent of 956 clinical cases were females. Wilson, Croxford, and Lingg (9) reported that 60 percent of 500 juvenile cardiac patients were girls. Both Coombs (6) and Findlay (10) in Great Britain. observed a slight predominance among females. Mackie (11), in this country, however, noted an equal distribution. The higher incidence of rheumatic heart disease among females, although slight, is noteworthy. Most forms of heart disease, especially in middle life, occur more frequently in males.

A lessened incidence of deaths from rheumatic heart disease among males is not related to the ability of the cardiovascular system to withstand disease. The mean age at death among males was 35.4 years, while among females it was 37.2 years. The increased hazard on account of childbirth is not reflected in the mean ages at death among females. It is more than compensated by the increased vulnerability of the male cardiovascular system, to which may be added the factor of a greater output of energy in attempting to maintain his place in a competitive economic field.

Contrasted with the white population, Negroes succumb to rheumatic heart disease much earlier. The mean age among 61 colored deaths was 28.0 years, as compared with 38.1 years among 296 white persons. This lower age at death is observed in almost every form of cardiovascular disease. Here it may be attributed to greater racial susceptibility, ignorance of the significance of juvenile rheumatism, failure to apply to or cooperate with physicians, lack of proper medical facilities, especially in cases originating in smaller towns and rural areas, unfavorable economic conditions resulting in overcrowding, undernutrition, and a poor environment in general, and the not infrequent necessity for engaging in manual or domestic labor despite severe cardiac handicaps. When each race-sex group is considered separately practically no difference is noted in the mean ages at death among white males and females, averaging 37.8 and 38.3 years, respectively. Among Negroes the difference is more evident, the average being 24.2 years for males and 31.6 years for females.

When the ages at death are subdivided into decades (table 3 and fig. 4) the percentage of deaths in each decade assumes a somewhat dromedary shaped curve with peaks in the 10-19-year age period and in the 40-59-year age group. Findlay noted a similar distribution of deaths from rheumatic heart disease in Great Britain.

Comparatively few deaths occur during the first attack of rheumatic fever. Atwater (12) estimated the case fatality rate at 1.7 percent,

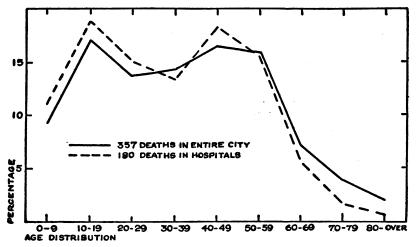


FIGURE 4.—Comparative percentage distribution in specific age groups between all deaths from rheumatic heart disease and deaths from rheumatic heart disease occurring in hospitals in Philadelphia during 1936.

and Swift at 1 to 4 percent (13). Ash (14) reports that in Philadelphia, 11 percent of deaths occurred within 2 years after the onset of rheumatic infection. Since the peak incidence of initial attacks is at about 7 to 10 years not many deaths occur before the second decade. Deaths during this period are usually due to cardiac insufficiency from repeated or continuous rheumatic infection.

A tendency about the age of puberty toward a subsidence of rheumatic infection is generally accepted, although contested by some authorities. Patients who have survived adolescence often do remarkably well until the late thirties or the 40-49-year age period. About this time there is a tendency for old smouldering infections to light up, resulting in cardiac insufficiency in the form of congestive failure. Other patients develop subacute bacterial endocarditis, due to the superimposing of *Streptococcus viridans* infection on probably slightly active rheumatic lesions. The onset of auricular fibrillation tells its story—death in a few years at most. While the mechanical effects of distorted valves play an important role, it is becoming increasingly recognized that congestive failure at any age is usually initiated by reactivation of the rheumatic infection. Premature arteriosclerotic changes, accelerated by rheumatic infection, further weaken the heart. Coronary occlusion, however, is not a common complication. Despite this tendency toward a dromedary mortality curve, rheumatic heart disease maintains a steady attrition of its victims at all ages.

In figure 5 a comparison is made of the distribution by age decades of deaths among white persons and Negroes from rheumatic heart

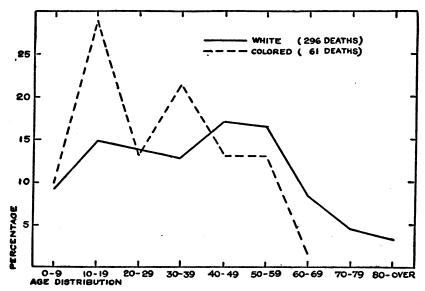


FIGURE 5.—Comparative percentage distribution in specific age groups between white and colored races of 357 deaths from rheumatic heart disease in Philadelphia during 1936.

disease. The mortality did not differ greatly during the first decade; apparently the proportion of deaths from fulminating pancarditis was quite similar. In the second decade, however, 27.9 percent of deaths among Negroes were caused by rheumatic heart disease, but only 14.9 percent of the deaths among white persons. This is indicative of a lower resistance to the infection among Negroes during this period. While it is dangerous to draw conclusions on the basis of a comparatively small series, attention is invited to the earlier secondary rise in mortality from this disease among Negroes. The secondary peak occurred precipitously in the 30-39-year age group among Negroes while among white deaths it is more gradual, reaching its maximum in the 40-49-year group. Comparatively few deaths were reported among Negroes past 50 years of age, while among white persons, especially females, a considerable number of deaths occurred (table 3).

In figure 6 a comparison is made of the distribution of deaths by decades according to sex. Under 30 years of age there was practically no difference. The secondary peak occurred earlier among males. More females survive 60 years.

#### DEATH RATES ACCORDING TO COLOR AND SEX

As stated previously, the crude death rate of the 357 deaths reported as rheumatic heart disease was 17.6 per 100,000 population, based on the estimated population for the year 1936 of 2,028,511 persons.

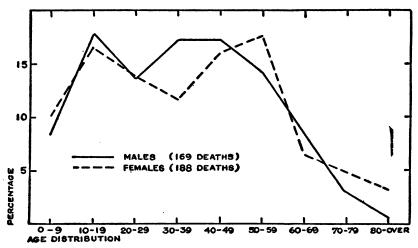


FIGURE 6.—Comparative percentage distribution in specific age groups between males and females of 357 deaths from rheumatic heart disease in Philadelphia during 1936.

Based on the 1930 census figures the death rate among white persons was 17.1, while among Negroes it was 27.8 per 100,000 population (table 3). Among males the death rate was 17.5, while among females it was 19.1 per 100,000 population. It was slightly higher among white females than white males, while the death rates among colored males and females were practically the same.

Rheumatic heart disease did not show the precipitous rise seen past 40 years of age for heart disease in general, which is due principally to deaths from hypertensive and coronary artery disease. The slight rise in age specific mortality is due to a smaller surviving population.

Under 60 years of age the mortality was consistently lower in the white than in the colored population. It was about 50 percent higher among Negroes during the first decade and nearly four times as high during the 10-19-year age group. Thereafter the increased age specific mortality was not so marked. Deaths from rheumatic heart disease are apparently more common among Negroes and occur at an earlier age period. Dublin (15) observed that this held for heart disease during the younger age periods according to the experience of the Metropolitan Life Insurance Co., while Atwater (12) noted that it holds for deaths from rheumatic fever.

 TABLE 3.—Ages at death and age specific death rates by decades according to color and sex from rheumatic heart disease in Philadelphia during 1936 (based on 1930 census)

		Male			Female			Total	
Age group	Popula- tion	Deaths	Rate per 100,000	Popula- tion	Deaths	Rate per 100,000	Popula- tion	Deaths	Rate per 100,000
		<u>.</u>			White	-			
0 to 9 10 to 19	141, 331 152, 879	10 20	7.1 13.1	137, 780 154, 265	17 24	12.3 15.6	279, 111 307, 144	27 44	9.7 14.3
20 to 29 80 to 39 40 to 49	150, 417 144, 099 116, 808	19 23 25	12.6 16.0 21.4	155, 922 141, 224 112, 855	22 15 26	14. 1 10. 6 23. 0	306, 339 285, 323 229, 663	41 38 51	13.4 13.3 22.2
50 to 59 60 to 69 70 to 79 80 and over	82, 046 49, 327 18, 878 3, 597	23 13 5 1	28.0 26.4 26.5 27.8	85, 270 54, 119 23, 551 6, 107	26 12 9 6	30. 5 22. 2 38. 2 98. 2	167, 316 103, 446 42, 429 9, 704	49 25 14 7	29.3 24.2 83.0 72.1
Total	859, 382	139	16. 2	871, 093	157	18.0	1, 730, 475	296	17.1
					Colored				
0 to 9	18, 875 14, 493 23, 115 24, 316 16, 117 8, 039 2, 526	4 10 4 6 4 1 1	21. 2 69. 0 17. 3 24. 7 24. 8 12. 4 39. 6	19, 083 16, 941 27, 138 23, 419 14, 007 6, 608 2, 573	2 7 4 7 4 7	10. 5 41. 3 14. 7 29. 9 28. 5 105. 9	87, 958 31, 434 50, 253 47, 735 30, 124 14, 647 5, 099	6 17 8 13 8 8 1	15. 8 54. 1 15. 9 27. 2 26. 6 54. 6 19. 6
70 to 79 80 and over	733 162			921 299			1, 654 461		
Total	108, 376	30	27.7	110, 989	31	27.9	219, 365	61	27.8
				White and	l colored	combine	d		
0 to 9 10 to 19 20 to 29 30 to 39 40 to 49 50 to 59 60 to 69 70 to 79 80 and over	160, 206 167, 372 173, 532 168, 415 132, 925 90, 085 51, 853 19, 611 3, 795	14 30 23 29 29 24 14 5 1	8.7 17.9 13.3 17.2 21.8 26.6 27.0 25.5 27.8	156, 863 . 171, 206 183, 060 1 164, 643 126, 862 91, 878 56, 692 24, 472 6, 406	19 81 26 22 30 33 12 9 6	12. 1 18. 1 14. 2 13. 4 23. 6 35. 9 21. 2 36. 8 93. 7	317, 069 338, 578 356, 592 333, 058 259, 787 181, 963 108, 545 44, 083 10, 165	83 61 49 51 59 57 26 14 7	10. 4 18. 0 13. 7 15. 3 22. 7 31. 3 24. 0 31. 8 68. 9
Total	967, 758	169	17.5	982, 082	188	19. 1	1, 949, 840	357	18.3

The impression that rheumatic heart disease is less common among Negroes is probably due to a failure to weight statistics on the basis of proportionate populations.

## DEATHS FROM RHEUMATIC HEART DISEASE IN HOSPITALS

Of the 357 deaths, 180, or 50.4 percent, were among regular admissions, excluding coroner's cases, to hospitals approved for interneship by the American Medical Association. This proportion would probably be reduced to a certain extent by more complete reporting of outside cases.

The mean age at death among 65 white males in hospitals was 34.5 years; while among 76 white females it was 36.1 years. Among 141 white patients the mean age at death was 35.4 years. Nineteen colored males and 20 colored females died in hospitals, at a mean age of 22.0 and 30.7 years, respectively. The mean age at death of colored patients was 26.2 years. The mean age at death of all hospital patients was 33.4 years. Deaths in hospitals occurred, in each group, a few years earlier than in the city as a whole.

In figure 4 a comparison is made of the percentage distribution by age decades of deaths from rheumatic heart disease in the city as a whole with deaths in hospitals. The curves are quite similar, and it is believed that the accuracy of deaths reported as rheumatic heart disease compares favorably with other certified causes of death. Both are dromedary shaped with peaks in the 10–19-year age period and in the 40–49-year age group. The chief difference is the larger number of deaths past 60 years from the city at large than in hospitals.

Seventy-four death certificates were based on necropsy findings. The mean age was 33.0 years, not dissimilar to that in the hospital series. Although these diagnoses are more accurate, they represent a highly selected group. Many racial, economic, and social factors are related to the obtaining of permission for necropsy.

## MISCELLANEOUS DATA

Percentage of total mortality due to rheumatic heart disease.—During 1936, deaths reported as rheumatic heart disease constituted 1.4 percent of all deaths in Philadelphia. With more uniform reporting it would probably account for 2.5 percent of the total mortality. In the 0–9year period it accounted for 1.7 percent of deaths, in the 10–19-year period for 11.5 percent, in the 20–29-year age group for 4.6 percent, in the 30–39-year age group for 3.0 percent, in the 40–49-year age group for 2.1 percent, in the 50–59-year group for 1.4 percent, in the 60–69-year period for 0.5 percent, in the 70–79-year age group for 0.3 percent, and at 80 years and over, for 0.3 percent. A higher proportion of the total deaths occurred during youth and comparatively early adult life. Unlike most forms of heart disease it results in a smaller percentage of the total number of deaths in each successive decade.

*Racial stocks.*—An attempt was made to compare deaths from rheumatic heart disease with deaths picked at random during similar age decades for controls. Deaths from rheumatic heart disease did not appear unduly frequent in any racial stock.

Coroner's cases.—Fifty-seven deaths, or 16.0 percent of the mortality from rheumatic heart disease, were reported by the coroner's office. Nonresident deaths.—Only 29 deaths, or less than 8 percent, occurred among nonresidents.

Influence of pregnancy.—In only five deaths, all among white persons, was pregnancy mentioned as a contributory factor.

Distribution by city wards.—Although no definite conclusions can be drawn, it did not appear that deaths occurred with significantly greater frequency in any part of the city.

Monthly variation.—The greatest number of deaths occurred in February, the smallest in September. The monthly distribution is similar to that for the general mortality, especially for heart disease.

#### COMMENT

The interest of the public in almost any disease is usually proportional to the excitement it causes and not necessarily dependent on its consequences either to the individual or the community. To the lay mind, the explosiveness of an outbreak has a dramatic appeal. Some diseases are better publicized than others. A suspected case of leprosy causes more excitement if not panic than a hundred deaths from malaria, pellagra, syphilis, pulmonary tuberculosis, or rheumatic heart disease. While there is little danger of leprosy becoming a major health problem in the United States, year in and year out these other diseases relentlessly take their toll.

If as many cases of anterior poliomyelitis developed in a city in the northern part of the United States as are normally found in the hospitals alone due to rheumatic heart disease in children, it would result in scare headlines and serious attempts to close down the schools. Yet the problem of rheumatic heart disease is at least equally serious. The person who contracts anterior poliomyelitis is often much more fortunate. He is at least immune to other attacks and can anticipate varying degrees of return of function. In rheumatic heart disease reactivation and recrudescences are more nearly the rule than the exception.

Rheumatic heart disease is one of mankind's most undramatic afflictions. Hardly any ailment is regarded with less concern by the laity than rheumatism. In the stress and strain of family and business life, the average adult pays little attention to joint and muscle pains until they become incapacitating. Consequently rheumatism in childhood is too often regarded with apathy. Its relatively slow progression often causes people to lose sight of its downward course. Compared to the pace set by most diseases of childhood it plays the successful, though more ominous, role of the tortoise in the fable of the tortoise and the hare. It is difficult for parents, school authorities, and others to visualize rheumatic infection in childhood as a systemic disease in which the heart bears the brunt of the attack. As a public health problem is has not received the attention it deserves. While in the present study there were doubtless imperfections in reporting deaths, and improvements could be made in the method of presenting these statistics, the results leave no doubt that rheumatic heart disease ranks as one of our major health problems from the standpoints of total mortality and age at death. Rheumatic heart disease not only results in loss of manpower to the Nation from deaths in childhood, adolescence, and the most economically productive period of adult life, but it lessens its manpower per man by causing various degrees of physical incapacity in about 1 percent of the wage earning population.

The International List of Causes of Death should be changed to permit the tabulation of deaths from rheumatic heart disease as an entity. Additional subtitles should be added under "Heart disease" for "Rheumatic heart disease." Physicians should be encouraged to report deaths from this cause on the basis of etiology. As demonstrated in Philadelphia, this need not prove difficult. Clinicians are aware of the problem, especially in the Northern States. By adding deaths reported as rheumatic heart disease to those reported as rheumatic fever, the mortality can be determined. This plan has received the approval of the Bureau of the Census (16), which unofficially put it into effect in the United States in 1937,<sup>2</sup> and of the committee on accuracy of certified causes of death, of the American Public Health Association.<sup>3</sup>

Better vital statistics are essential as a means and not as an end. They serve as an index for evaluating the effects of a disease and for studying its trends. Statistics, however accurate, will not solve the problem. Too much clinical and public health research begins at the morgue or with the registrar of vital statistics and gets no further. Concerted action among hospitals and clinics, bacteriological and pathological laboratories, epidemiological investigations of the houseto-house variety, sickness and industrial surveys, better health examinations of school children, and like measures are needed.

#### CONCLUSIONS

1. In response to a request to the practicing physicians, coroner's office, and hospitals, 357 deaths were reported from rheumatic heart disease, rheumatic fever, chorea, and subacute bacterial endocarditis in Philadelphia during 1936. The mortality rate from rheumatic heart disease reported during this year was 17.6 per 100,000 population.

<sup>&</sup>lt;sup>2</sup> Personal communication from Dr. Halbert C. Dunn.

<sup>\*</sup> Personal communication from Dr. Haven Emerson.

2. In **Sid**ilition, 195 deaths regarded as presumably due to rheumatic heart disease were reported. The total mortality from rheumatic heart disease during 1936 is estimated at 25 to 30 per 100,000 population.

3. Among the infectious diseases, rheumatic heart disease was exceeded as a cause of death by tuberculosis, lobar pneumonia, and syphilis. Among the essentially chronic infectious diseases it ranked third, exceeded only by tuberculosis and syphilis. It is believed that this is the usual relationship in this locality.

4. Rheumatic heart disease resulted in considerably more deaths than whooping cough, meningococcus meningitis, typhoid and paratyphoid fevers, measles, diphtheria, scarlet fever, and anterior poliomyelitis, which followed it in the order listed.

5. In persons under 20 years of age rheumatic heart disease was the cause of more deaths than pulmonary tuberculosis, but fewer deaths than all forms of tuberculosis. Excluding pneumonia and bronchopneumonia and the diarrheas and enteritis of infancy, it was the second largest cause of death from infectious diseases.

6. In the year under study, rheumatic heart disease resulted in more deaths under 20 years of **ug**e than whooping cough, measles, meningococcus meningitis, diphtheria, scarlet fever, and anterior poliomyelitis combined. These diseases followed rheumatic heart disease in the order mentioned. While rheumatic heart disease probably does not result in more deaths than all of these diseases during years in which epidemics of one or more of them occur, there is little doubt that over a course of years it is the cause of more deaths than any one of them.

7. The mean age at death was 36.5 years. Among all deaths from rheumatic heart disease occurring in hospitals approved for interneship by the American Medical Association the mean age was 33.4 years. Among necropsy cases it was 33.0 years.

8. Both in the number of deaths and the death rate per 100,000 population, females slightly exceeded the males. Among the males, especially Negroes, the mean age at death was somewhat younger.

9. The mortality was appreciably higher among Negroes than white persons, particularly under 40 years of age. It was especially high in Negro males in the 10-19-year age period.

10. A plea is made for increased recognition of the importance of rheumatic heart disease by health officials, for improved reporting and recording of deaths from this cause, and for greater cooperation among various agencies which may contribute to its study.

#### **ACKNOWLEDGMENTS**

The writer is especially indebted to Dr. Frank P. Strome, director of the bureau of vital statistics of the Pennsylvania State Department of Health, for permission to abstract the death certificates which form the basis of this paper. Statistics concerning the incidence of diseases other than rheumatic heart disease were obtained from the Annual Report of the Health Department of Philadelphia. The writer is also indebted to Dr. C. C. Wolferth, of the Robinette Foundation of the University of Pennsylvania, for a number of helpful suggestions in the preparation of this paper.

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## THE FAMILY AS A UNIT FOR NURSING SERVICE 1

By HELEN BEAN, Associate Public Health Nursing Analyst, and GEORGIE S. BROCKETT, United States Public Health Service

Perhaps one of the chief advantages of home visiting in a public health nursing program is the opportunity presented for ascertaining the health needs of various members of the family and for assisting in the solution of special problems. The nurse observes the social and

<sup>&</sup>lt;sup>1</sup> From the Division of Public Health Methods, National Institute of Health, in cooperation with Division of Domestic Quarantine. Special recognition is due to Dr. Mayhew Derryberry for his advice and guidance in the preparation of this paper.

environmental conditions in the home, and with this first-hand information she can adapt her advice so that it is directly applicable to the limitations within the family. Then, too, there is the situation demanding immediate attention which has not been recognized by the family. Many of these needs can be uncovered only by a personal visit to the household. Therefore, when a nurse goes into a home it is expected that she will not only give the nursing care or health instruction which occasioned her visit, but that she will also obtain an estimate of the health needs of the entire family and work out a constructive health plan for its several members.

The advantages of the home visit as a method of rendering a complete family health service have been repeatedly stressed by nurses and other public health workers (1, 2, 3, 4). In view of the emphasis that has been placed on the principle of "the family as the unit" for nursing service, it seemed appropriate to investigate the degree to which nurses in actual practice broaden the scope of their visits to include members of the family other than the specific individual whose condition initiated the home call. If many of their visits are confined to service for a single individual, then the alleged values of home visiting in terms of constructive health service to the entire family are not attained. Therefore, the extent to which other members of the household are served becomes one measure for determining the achievements of field nursing service.

## THE DATA

During the study of county health department practice, the United States Public Health Service obtained, among other data, duplicate records of all the work of the personnel in three rural health departments covering a period of 1 year. It has been possible to utilize the records of field nursing as a means of ascertaining the amount of family health service in the three counties. The conclusions are, therefore, limited by the recording policies of the department and the faithfulness with which all the services to a family were entered on the record.

The procedure for recording home visits followed commonly accepted practice. Each nurse made a daily report of all services rendered. An individual case record and a family folder showing the roster of the household members were opened whenever the nurse found a condition suggesting the necessity for a permanent record or the desirability of further nursing attention ( $\delta$ ). Since no evidence pertaining to the composition of the family was available for households where visits were recorded on the daily report only, the analysis has been limited to families for whom the nurse opened a family folder. The volume of nursing service excluded from the analysis by limiting the material in this manner may be judged from the data in table 1. In approximately one-third of the families given home care the problems found were considered of such slight importance that no family record was opened. It would seem safe to assume that in these families little or no service was rendered to members of the household other than to the specific individual whose name the nurse entered on her daily report. It is likely, therefore, that, by excluding the 1,498 families without folders, and limiting the analysis to the 3,074 with folders, the data are weighted in favor of a more complete family service than would be given on an initial visit or on an average of all visits to a family.

TABLE 1.—Distribution of families served in the home and number of these for whom family folders were not opened by the nurses

	Familie	s served in tl	he home	
County	Total	Families for whom no folders were opened		
	families	Number	Percent	
AB	552 1, 476 2, 544	266 1 575 657	48. 2 39. 0 25. 8	
Total	4, 572	1, 498	32.8	

16 families having home visits in county B for whom folders were opened were included in this group because the information thereon was inadequate for this study.

In making this analysis, it is recognized that information concerning the health needs of all members of the household is limited by the entries nurses made on the records. However it is doubtful that any constructive health plan was contemplated for the family if no record was made of it, for without a record the plan would not be likely to function. Therefore, the frequency with which nurses described problems in the home, other than the one that initiated the visit, may be considered indicative of the degree to which they gave attention to the "family as a unit."

There were a number of other factors which may have limited the amount of service the nurses rendered each family. In the first place, the population load per nurse was much higher than is ordinarily recommended for an adequate nursing service. In county A each nurse was required to serve a population of over 13,000 individuals, while in counties B and C the load was about 10,000 and 7,000, respectively. Furthermore, many of the families were large, oftentimes including a total of 12 and sometimes as many as 18 individuals. A third factor to be considered was the distance to be traveled. Approximately one-fourth of the population lived in the open country, while the remaining three-fourths resided in small villages scattered throughout the county. As the same limitations affect the field nursing service of most county health departments, the results obtained from an analysis of the records of these three organizations are more than likely indicative of the nursing service in many other counties where conditions are similar.

## PROPORTION OF THE FAMILY SERVED AT THE TIME OF NURSING VISITS

There were only 11 of the total of 3,074 families studied in which all members were served at every visit; and 3 of these consisted of but 1 member each. The remaining eight families were visited because of either tuberculosis or other communicable disease. In such situations all exposed individuals are contacts, and it is essential that nurses consider the possibilities of disease in the whole family. Even though all members were served in these homes, it is unlikely that a constructive health plan for the entire family was worked out, since a return nursing visit was made to only one of the eight households.

It may be argued, however, that there were few households in which every member had a health problem which required the assistance of a nurse. Accordingly, the 3,063 families in which not all members were served at each visit were divided into two groups—those in which only one individual was served on each visit, and those in which more than one person was served during one or more of the total visits to the family. The number of families in each of the classifications is shown by county in table 2. In over one-third of the households only one individual was served by the nurse on every visit to the home. The proportions varied from 31 percent in county B to 45 percent in county C.

		of famili	es served	Percentage of families served			
Classification of families	County A	County B	County C	County A	County B	County C	
All families	286 5	901 6	1, 887	100. 0 1. 7	100. 0 . 7	100.0	
total visits to the families <sup>1</sup>	152	617	1, 223	53. 2	68. 5	64.8	
families	129	278	664	45. 1	30. 8	35. 2	

 
 TABLE 2.—Distribution of families according to the number of individuals served during home visits

<sup>1</sup> Does not include families with all members served on every visit.

One might expect more health problems in larger families, hence the smaller families should predominate in the group in which only a single member was served. That this tendency existed to a slight degree is suggested by the data in table 3. The average size for the families with one individual served was somewhat smaller than the average size for all families receiving nursing care. However, in each county the difference is less than one individual per family. Moreover, in more than a fifth of the larger families—those with eight or more members—only one person was given service.

 TABLE 3.—Comparison of the average size of all families served with that of the families having a single member served

+	Average siz	e of families
County	All families served	Families hav- ing only one member served
ABC	6.4 6.5 6.0	5.7 5.7 <b>5</b> .1

In order to summarize the extent to which the nurses approximated a total family service for those households in which more than one individual was seen, the number of individuals served in the home was expressed as a percentage of the total number in the family. For example, if there were six individuals in a family and the nurse served two of them, then 33 percent of the family was reached. If she made two visits, serving two individuals at the first home call and one on the second, the average percentage served in a family of six would be 25.<sup>2</sup>

The median percentage of the family served during each visit was 29 in county A, 33 in county B, and 32 in county C. Thus it is evident that less than a third of the household members were served in the course of visits to over one-half of the families. Furthermore, in only 2 percent of the families were as many as three-fourths of the household members served during a nurse's visit.

The small number of families in which every member was given attention, the relatively large group in which a single individual was served at each visit, and the low percentages of the family served, even when more than one member was included, indicate definitely that little consideration was given to the needs of all the family when the nurse entered the home.

## SERVICES TO THE FAMILY UNDER SPECIFIC CONDITIONS

In making the above analysis, consideration was given to the proportion of the family served irrespective of the specific condition which stimulated the call or of the needs for care which the nurse may have encountered. Of course, the nurse should render service or give advice only when there is a need for such assistance. There are,

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<sup>&</sup>lt;sup>3</sup> To arrive at a percentage for each family, the number of individuals served on each visit was added and used as the numerator and the number of individuals in the family was multiplied by the number of visits and used as the denominator.

however, some situations in which the need for service to more than one individual in the family can be predicated from the condition which caused the visit. In order to measure the extent to which the nurses actually met the needs of these families, an analysis of service in the households presenting such problems has been made.

When there is tuberculosis in a family, a nurse makes a visit presumably to advise the patient in regard to his own care and to urge examination of all contacts. Hence, if nurses were guided by these purposes when they visited homes in which tuberculosis was a problem, the proportion of the household given service would approach 100 percent. Actually among families with a case of tuberculosis the average proportion of the members served on a visit was 29 percent in county B and 27 percent in counties A and C. Furthermore, in over one-third of the households where tuberculosis was a problem, only one individual was given attention. According to these data the nurses failed to consider the family as a unit when they made visits for tuberculosis, thus neglecting an important procedure for preventing the spread of the disease.

Infant health supervision is another service which nurses are expected to give whenever an infant is found in the home, regardless of the original purpose of the visit. However, it was found that the infant was not served in 21 percent of the families where there was the possibility of such supervision. Also, in the families where there were preschool children, no service was given them in 28 percent of the situations. The proportions for the separate counties appear in table 4. Despite the fact that nurses conducting a generalized program are expected to give special attention to infants and preschool children when they make a visit, these nurses failed to take advantage of this opportunity to render a preventive health service.<sup>3</sup>

	I	Households visi	ted by the nur	58
County	Number containing infants	Percentage in which infants were not served	Number containing preschool children	Percentage in which preschool children were not served
All counties	1, 106	21. 1	2, 054	28. 2
A B O	89 481 536	24. 8 21. 2 18. 7	165 660 1, 229	62, 4 33, 3 20, 9

 
 TABLE 4.—Number of households visited by the nurse in which there were infants or preschool children, and percentage of these groups receiving no service

<sup>9</sup> Since the preparation of this paper, Randall has reported the following: "An analysis of the (New York City) Health Department nurses' home visits to this sample of families revealed that in 31 percent of household visits to families in which there was an infant, no infant service was recorded; that 45 percent of the visits to families with preschool children gave no record that the preschool child had been visited" (7).

#### PROPORTION OF FAMILY SERVED DURING STUDY YEAR

The extent to which nurses perform a family service may be considered from a somewhat different point of view. Conditions may be such during a given visit that it is better to limit service to one individual and to consider other members of the family at a later visit. In such instances the percentage of the individuals in the families served over an extended period of time might be greater than the percentage served on any one visit. The possibilities of such consideration by the nurses are somewhat limited by the fact that 30 percent of the families were visited only once during the study year.

Furthermore, the data presented thus far have been confined to services in the home. If a nurse renders additional service to certain members of a family in her office or in the school, she may not need to continue service for them in the home. Hence the proportion of the family served might be increased if the services rendered at other places than the home were included. In order to investigate the extent to which the nurses rendered service to all members of the family regardless of place or time, the total number of individuals served during the study year was expressed as a proportion of the aggregate number of individuals in all the households visited by the nurse. The resulting proportions are 43.7 percent for county A, 43.2 percent for county B, and 51.2 percent for county C. Thus, less than half of the individuals in the families visited by the nurses were given a direct service or were advised concerning a health problem.

## FACTORS ASSOCIATED WITH MULTIPLE SERVICES TO THE HOUSEHOLD<sup>6</sup>

In the preceding tables it has been shown that the nurses did not give a complete family service at the time of their visits, but that they did serve more than one individual in about two-thirds of the families included in their clientele.

According to table 5 there was a tendency to give multiple services to the white families more frequently than to the Negro families. This trend was most pronounced in county A.

	County							
Number of individuals served in family		A	1	B	c			
	White	Negro	White	Negro	White	Negro		
1 individual served More than 1 individual served	43. 5 56. 5	59. 5 40. 5	28.4 71.6	33.6 66.4	34. 9 65. 1	38. 7 61. 3		

 TABLE 5.—Percentage distribution of white and colored families according to number

 of individuals served on a home visit

• The term "multiple services" is used in this paper to mean that more than 1 individual in a family was served during a single visit.

Previous studies (6) have shown that economic status is associated with the selection of families for home visiting. It is also related to the rendering of services to a family when a nurse visited the home. Among those families considered in the comfortable economic status group for all three counties, a single individual was served in 48 percent of the households, whereas this percentage was only 31 among the families of low economic status. The percentages for each county appear in table 6.

			per nom			
	P	ercentage o	of families	with one or	more serv	ed
<b>Re</b> onomic status	Cou	nty A	Cou	nty B	Cou	nty C
Reponomic status	1 indi- vidual served	2 or more individ- uals served	1 indi- vidual served	2 or more individ- uals served	1 indi- vidual served	2 or more individ- uals served
Comfortable Moderately comfortable Poor and very poor	63. 6 59. 3 40. 3	36. 4 40. 7 59. 7	33. 3 36. 4 28. 9	66. 7 63. 6 71. 1	51. 3 39. 2 29. 6	48.7 60.8 70.4

 TABLE 6.—Percentage distribution of families in different economic groups according to number of individuals served per home visit

The nurse may have given more service to the lower economic group because she recognized the greater need for service in homes where overcrowded conditions, inadequate clothing, and lack of proper food made the entire family more susceptible to disease, or again there may have been disease conditions already present in several members of the household.

#### SUMMARY

Records of the services rendered by public health nurses in three rural counties have been analyzed to ascertain the extent to which the cardinal principle of "the family as the unit" was observed in spreading service within families selected for home visits. The conclusions are necessarily qualified in terms of the faithfulness of the nurses in recording all the services.

The nurses whose records were analyzed were working under conditions commonly encountered in rural situations. Each nurse was required to serve a large population, most of which was distributed throughout the county. Thus it was necessary to travel considerable distances in order to reach the families.

Little or no evidence could be found that the nurses were rendering a complete family service. In one-third of the families only one person was served at any visit to the homes. Even when more than one person was served, relatively few of the family were included. The proportion of the family given care and advice was not increased when the visit was made for the purpose of tuberculosis control: neither was consideration given to the health needs of a very high percentage of the infants in the homes visited by the nurses. Even when all services rendered to a family during the period of a year were included, less than a half of the individuals were given attention. Evidently, from the data assembled, the nurses did not render a diversified service when they made a home call. Their visits seemed to be confined to services for one or two members in the family, with very little attention given to the development of constructive programs for the family as a social unit. Such limitations in the spread of nursing service considerably decrease the possible value of home visiting. It is hoped, therefore, that this analysis may stimulate health departments to appraise their nursing activities and to ascertain to what degree, in actual practice, stated objectives are really being attained.

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## DIBENZANTHRACENE TUMORS IN MICE

#### The Production of Subcutaneous and Pulmonary Tumors by 1: 2: 5: 6-Dibenzanthracene Adsorbed on Charcoal

By H. B. ANDERVONT, Biologist, and EGON LORENZ, Biophysicist, United States Public Health Service

The appearance of lung and liver tumors in mice (1) injected subcutaneously with 1:2:5:6-dibenzanthracene is evidence that the compound is capable of producing tumors in tissues which are distant from the site of injection. The problem arises as to whether the carcinogenic agent produces in the injected animal a constitutional change of which pulmonary and liver tumors are a local manifestation, or whether the tumors are the result of a direct action of the compound. or a derivative, upon the tissues of those organs. The results of one experiment performed to elucidate this problem have been reported (2). Pulmonary tumors were induced in mice by inserting into their

lungs threads coated with the hydrocarbon. In the present communication are included the findings of other experiments in which the carcinogenic agent was brought into contact with the lung tissues of mice. In order to place the compound in the lungs without inciting the irritation which accompanied the thread experiments, it was decided to adsorb it onto a substance which, when injected intravenously, is known to localize in the lungs.

During the course of previous investigations (9) in this laboratory in which efforts were made to detect the presence of 1: 2: 5: 6-dibenzanthracene in mouse tumors produced subcutaneously by the compound, it was found <sup>1</sup> that charcoal had a tendency to adsorb the hydrocarbon. This report deals with experiments in which 1: 2: 5: 6-dibenzanthracene adsorbed on charcoal was injected into mice both subcutaneously and intravenously.

#### PREPARATION OF 1:2:5: 6-DIBENZANTHRACENE ADSORBED ON CHARCOAL

Different kinds of powdered charcoal were tested for impurities of tarry nature by shaking them with ether <sup>2</sup> and comparing the ultraviolet absorption spectra. With the exception of medicinal charcoal, the filtered ether showed strong continuous absorption, beginning at approximately  $\lambda$ =3000 A, indicating the presence of impurities, probably of a tarry nature. This charcoal was used in the experiments.

To remove larger particles, the charcoal was shaken with ether in a separatory funnel, and the larger particles, after settling, were drained off. After drying at about 40° C. and heating in a porcelain dish at approximately  $300^{\circ}$  C., the charcoal was activated by heating it under vacuum (as produced by a Cenco Hyvac pump) to a temperature of  $270^{\circ}$  C. for 8 hours. Much difficulty was encountered when the gas escaping from the particles caused them to fly all over the vacuum system. This was avoided by inserting a cone of platinum sheet in the glass tube containing the charcoal.

Five hundred mg of charcoal thus activated were added to a solution of 400 mg of purified 1: 2: 5: 6-dibenzanthracene in 500 cc of ether, and this mixture was shaken for 8 hours at room temperature. The adsorption process is, however, almost complete after shaking for 10 minutes. Then the mixture was filtered and the charcoal washed with ether. Quantitative spectrographic analysis (9) of the 1: 2: 5: 6-dibenzanthracene content of the filtrate, as well as weighing the charcoal after drying, gave 0.5 mg of the hydrocarbon adsorbed on 1 mg of charcoal.

<sup>&</sup>lt;sup>1</sup> Experiments performed in collaboration with Dr. M. J. Shear.

<sup>&</sup>lt;sup>2</sup> The purity of the ether used was tested by evaporating 100 cc of ether to 1 cc and comparing its ultraviolet transmission with that of the original ether. Except for the short ultraviolet ( $\lambda$ <2300 A) no difference in transmission was observed.

No attempt was made to determine the saturation content, since 0.5 mg of 1: 2: 5: 6-dibenzanthracene per mg of charcoal was sufficient for the performance of biological experiments. However, it can be assumed that the value for the saturation of adsorption will not be much higher than the value given here. Assuming an active surface of 200 m<sup>2</sup> per gram of charcoal (10), and assuming the diameter of the 1:2:5:6-dibenzanthracene to be of the order of magnitude of 10 A, 0.5 grams of the hydrocarbon adsorbed on this surface will form a monomolecular layer. This monomolecular layer also explains why dibenzanthracene charcoal fails to show the characteristic fluorescence of this compound when irradiated with ultraviolet light.

The question arises as to whether the 1: 2: 5: 6-dibenzanthracene adsorbed on charcoal is held on the surface by physical forces or whether a chemical bond exists between the carbon and the hydrocarbon. It seems unlikely that chemical bonds exist, considering the fact that 1: 2: 5: 6-dibenzanthracene is chemically extremely inert and that most of the hydrocarbon can be brought into solution again by an excess of the solvent.

The forces that hold the 1:2:5:6-dibenzanthracene on the surface of the charcoal are illustrated by the fact that the equilibrium of the hydrocarbon for the system dibenzanthracene charcoal-ether (e. g., 20 mg of dibenzanthracene charcoal in 50 cc of ether) is 0.5 mg per mg of charcoal and  $1 \times 10^{-2}$  mg per cc of ether (determined spectrographically) for the preparation as described above.<sup>3</sup> This equilibrium is reached rapidly after mixing and does not change after standing or vigorous shaking.

Likewise the equilibrium of the hydrocarbon was measured for the system dibenzanthracene charcoal-horse serum. It was found that less than  $1 \times 10^{-4}$  mg of the hydrocarbon per cc of serum ( $1 \times 10^{-4}$  mg per cc being the limit of sensitivity of the spectrographic method in this case) are released from the charcoal into the serum.<sup>4</sup> Mouse serum differs little from horse serum with respect to solubility of 1: 2: 5: 6-dibenzanthracene.

The 1:2:5:6-dibenzanthracene adsorbed on charcoal is designated as dibenzanthracene charcoal throughout this communication.

## EXPERIMENTAL

As a preliminary experiment, mice of strain  $C_3H$  were injected with dibenzanthracene charcoal, or control material, in order to determine whether the compound retained its carcinogenic power after being adsorbed. The protocol of this experiment is presented below.

<sup>\*</sup> A saturated solution of 1: 2: 5: 6-dibenzanthracene in ether will contain approximately 0.75 mg of the hydrocarbon per cc of ether at room temperature.

<sup>&</sup>lt;sup>4</sup> A saturated solution of 1: 2: 5: 6-dibenzanthracene in horse serum contains 1.5×10<sup>-3</sup> mg of the hydrocarbon per co of serum.

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#### Experiment 1

Sixty-five mice of strain  $C_3H$  were used. These were divided into four groups and injected subcutaneously in the right axilla on July 9, 1936, as follows:

Group A.—To 5 cc of a 50 percent solution of glycerin in water were added 150 mg of dibenzanthracene charcoal. Since each mg of charcoal contained 0.5 mg of the carcinogenic agent, there were 50 mg of 1: 2: 5: 6-dibenzanthracene in the suspension. The preparation was used to inject 11 female mice, each animal receiving 0.2 cc of the glycerin suspension subcutaneously; hence, each received 2 mg of 1: 2: 5: 6-dibenzanthracene adsorbed on 4 mg of charcoal.

Group B.—To 5 cc of a 50 percent solution of glycerin in water were added 50 mg of crystalline 1: 2: 5: 6-dibenzanthracene. This suspension was used to inject 10 female mice, each of which was given 0.2 cc of the preparation containing 2 mg of the compound. These mice served as controls for those of group A.

Group C.—To 25 cc of a horse-serum dispersion of 1:2:5:6-dibenzanthracene containing 1 mg of the hydrocarbon in each cc were added 100 mg of activated charcoal. The mixture was shaken, allowed to stand for 10 minutes at room temperature, and then injected into mice. Twenty-four male mice each received 1 cc of the mixture; thus, each was given 1 mg of 1:2:5:6-dibenzanthracene and 4 mg of charcoal.

Group D.—Each of 20 male mice was injected with 1 cc of a horseserum dispersion of 1: 2: 5: 6-dibenzanthracene. These mice were used as controls for those of group C.

Mice of groups C and D were employed to determine whether the presence of charcoal might influence the time of appearance of induced tumors. Previous work (1) had shown that 1 cc of horse-serum dispersion would induce subcutaneous sarcomas in 50 percent of strain  $C_3H$  mice within 24 weeks. The results of the experiment are presented in table 1.

In table 1 it is seen that dibenzanthracene charcoal produced subcutaneous tumors at the site of injection and that no pronounced difference occurred in the time of appearance of tumors induced by dibenzanthracene charcoal and crystalline dibenzanthracene. The table also shows that of the mice of group C which had received the horse-serum dispersion plus charcoal, 75 percent developed tumors within 24 weeks after injection, while of the control mice of group D which had received only the horse-serum dispersion, 50 percent developed tumors during the same period of time.

#### 1935

TABLE 1.—Experiment 1:	Time of ap	pearance of	subcutaneous	tumors induced
in strain C <sub>2</sub> H mice by 1:	2:5:6-diber	nzanthracene	when injected	as dibenzanthra-
cene charcoal, crystals, dispersion	ho <b>rse-</b> serum	dispersion	plus charcoal	, or horse-serum

	Preparation used	Num-		Num	ber	of tu	mors	, acc	ordin	ng to	time	, in v	veek	9	Total	
Group	and amount of	ber of mice used	14	16	18	20	22	24	26	28	30	32	34	36	num- ber of tu- mors	
A	Dibenzanthracene charcoal, 2 mg	11		,	3			2	,		2				10	
в	Crystals in glycerin,			-			1	-	1		<b>1</b>				10	-
-	2 mg	10	1		1			4	2						8	2
С	Horse-serum disper- sion plus charcoal,															
	1 mg	24		2	5	5	2	4	1	1	2	1			23	1
D	Horse-serum disper- sion, 1 mg	20		1	2	5		2		3	1	1	1	1	17	8

These results were probably due to the presence of charcoal in the mice of group C which may have held some of the hydrocarbon at the injection site, or may have acted as an irritant, thus accelerating the appearance of tumors. This latter possibility, however, is unlikely in view of the experiments by Berenblum (6), who has shown that an irritant tends to inhibit the carcinogenic action of 1:2:5:6-dibenzan-thracene and unpublished experiments performed in this laboratory in which the addition of kaolin or kieselguhr to a lard-solution of dibenzanthracene failed to bring about the earlier appearance of induced tumors.

When the tumor-bearing mice of groups C and D came to autopsy, additional evidence was found which suggested that charcoal kept the carcinogenic agent from spreading throughout the bodies of the group C animals, for of 9 mice of group D which developed subcutaneous sarcomas at the site of injection, 20 weeks or more after injection, 5 also had primary induced lung carcinomas, while of 11 mice of group C which developed tumors at the injection site during the same period of time none had developed lung growths. It has been recorded (1) that subcutaneous injection of a horse-serum dispersion of 1: 2: 5: 6-dibenzanthracene induces pulmonary tumors in strain  $C_3H$  mice and the findings in group D mice of this experiment confirm the previous observation.

Though it might seem unjustifiable to state that charcoal held the 1:2:5:6-dibenzanthracene at the injection site, yet the paucity of lung tumors, together with the earlier appearance of subcutaneous growths in group C mice, strongly suggests that this was the case.

Further results of interest were procured following subcutaneous injection of dibenzanthracene charcoal into mice of strain A. Since the lungs of strain A mice are known to be especially susceptible to the carcinogenic action of 1: 2: 5: 6-dibenzanthracene when injected subcutaneously as a lard solution (3), it was decided to inject dibenzanthracene charcoal into the subcutaneous tissues of these mice to ascertain whether the adsorbed compound would bring about the appearance of pulmonary tumors.

Strain A female mice, 3 months of age, were given dibenzanthracene charcoal or crystalline dibenzanthracene at the same time and in the same manner as mice of groups A and B of Experiment 1: 12 each received 2 mg of the hydrocarbon adsorbed on 4 mg of charcoal and 10 served as controls when each received 2 mg of crystalline dibenzanthracene. From July 9, 1936, which was the time of injection, to January 22, 1937, only 2 mice of each group developed induced subcutaneous sarcomas. In the dibenzanthracene charcoal mice these tumors arose 18 and 28 weeks after injection and neither mouse had lung tumors when autopsied, while in the mice injected with crystalline material the tumors were noted 26 and 28 weeks after injection, and both mice also had multiple lung tumors. The production of only 4 subcutaneous tumors in strain A mice indicates that these animals were far more resistant to the carcinogenic agent than mice of strain  $C_3H$ .

On January 22, 1937, more than 6 months after injection, all surviving strain A mice were sacrificed and their lungs examined carefully for the presence of macroscopic tumors. Of the 10 dibenzanthracene charcoal animals, 5 were found to be free from lung tumors and 5 had but a single lung tumor, while of the 8 crystal-injected mice, all had large multiple lung tumors. Previous investigations (2) had revealed that 1 mg of crystalline dibenzanthracene suspended in a glycerin solution and injected subcutaneously into mice of strain A induced multiple lung tumors in practically all the animals within 3 months. Hence, the scarcity of pulmonary tumors within the dibenzanthracene charcoal-injected mice suggests that the carcinogenic agent remained adsorbed on the charcoal at the site of injection, thereby preventing its spread through the body of the injected animal.

This explanation of the results is substantiated by the recent observations of Lettinga,<sup>5</sup> who found that large amounts of 1:2:5:6-dibenzanthracene induced many lung tumors in susceptible mice, while small amounts induced very few such tumors. Kennaway and Kennaway (8) interpret these results as suggesting "very strongly an overflow of carcinogenic material either from the actual site of injection or from some organ where these compounds undergo chemical change."

Having found that dibenzanthracene charcoal induced tumors at the site of injection, the next step was to carry out the projected lines of experimentation by localizing the material in the lungs of mice. It

<sup>&</sup>lt;sup>6</sup> Up to the present time, Lettinga's original publication has not been read and efforts to locate it have failed. The information concerning this work was obtained from the publication of Kennaway and Kennaway (8)

is known that a particulate substance such as charcoal, when injected into the blood stream, tends to localize in the lungs. After intravenous injection in these investigations, the lungs of the mice were black with charcoal and histological sections showed many small vessels engorged with the particulate material. The experiment in which charcoal was injected intravenously into mice is described in the following:

#### Experiment 8

Female mice of strain A, all of which were approximately 4½ months old were used. The mice were divided into four groups and injected on January 14, 1937, as follows:

Group A.—This group consisted of 28 mice, each of which received an intravenous injection of 0.5 cc of a horse-serum dispersion of 1: 2: 5: 6-dibenzanthracene containing 0.5 mg of the hydrocarbon.

Group B.—There were 36 mice, each of which received an intravenous injection of 1.5 mg of dibenzanthracene charcoal suspended in 0.5 cc of sterile physiological saline. Thus, each was given 0.5 mg of 1: 2: 5: 6-dibenzanthracene adsorbed on 1 mg of charcoal.

Group C.—This included 36 mice, each of which received an intravenous injection of 1 mg of charcoal suspended in 0.5 cc of sterile physiological saline.

Group D.—Twelve mice were set aside as normal untreated controls. In order to detect the appearance of lung tumors, two mice of each of the first three series were killed at weekly intervals, beginning 1 week after the intravenous injections. It has been noted during the course of experiments performed in this laboratory that when strain A mice of the same age are given injections of 1: 2: 5: 6-dibenzanthracene, lung tumors arise simultaneously in practically all the animals. This uniformity in the latent period of induced lung tumors presents an excellent opportunity for carrying out investigations pertaining to the time of appearance of these growths. It was believed, therefore, that weekly examination of the lungs of two mice in a given group would give an accurate idea as to when lung tumors appeared in most individuals of the same group and the results of the experiment proved this to be the case. In the interest of brevity the results of the weekly autopsies are presented in respect to the findings in each group. These were as follows:

Group A.—Injected with horse-serum dispersion. All lungs were negative until the fifth week after injection, when both showed several small nodules. From the sixth up to and including the fourteenth week following the injection, when the last two mice were killed, all the animals had multiple lung nodules.

Group B.—Injected with dibenzanthracene charcoal. Up to and including the eighth week, all the lungs were tumor-free, but the

ninth week both lungs showed a single lung nodule. From the ninth to the sixteenth week both sets of lungs contained several lung nodules, and from the sixteenth to the eighteenth week all showed multiple lung tumors.

Group C.—Injected with charcoal. These mice served as controls for those of group B. A single lung nodule was found in one of the two sets of lungs 7, 8, and 14 weeks after injection. No lung tumors were found in mice killed from the fourteenth to the eighteenth week after injection.

Group D.—Normal untreated controls. Three of these mice were killed 14 weeks after the beginning of the experiment; none had lung tumor. The remaining nine were killed 19 weeks after the experiment was started, and of these, six were tumor-free and three had a single lung tumor.

The results of the experiment may be summarized as follows: When 0.5 mg of 1: 2: 5: 6-dibenzanthracene was injected intravenously as a horse-serum dispersion, it evoked macroscopic lung tumors in strain A mice within 5 weeks; when 0.5 mg of the compound was adsorbed on 1 mg of charcoal and injected intravenously as a suspension in 0.5 cc of saline, it induced macroscopic lung tumors within 9 weeks; while 1 mg of charcoal suspended in 0.5 cc of saline had very little, if any, effect in producing lung tumors.

The findings show that 1: 2: 5: 6-dibenzanthracene is capable of inducing lung tumors in susceptible mice when it is adsorbed on charcoal and localized in the lungs via the blood stream. However, the fine particles of the compound present in the horse-serum dispersion were considerably more effective in this respect than was the dibenzanthracene charcoal. It will be noted that dibenzanthracene charcoal induced lung tumors in strain A mice within 9 weeks, though it failed to evoke tumors in the subcutaneous tissues of 10 mice of the same strain which were kept for more than 6 months after injection. This again shows that the lungs of strain A mice are more susceptible to the carcinogenic action of 1:2:5:6-dibenzanthracene than are their subcutaneous tissues.

Campbell (7) has found that the inhalation of road dust "minus tar, as removed by benzene" produces lung tumors in susceptible mice, but fewer lung tumors are induced than when tar is present in the inhaled dust. His observations are of interest when compared with the inability of intravenously injected charcoal to produce lung tumors in strain A mice. The difference in the results is probably due to the relatively short period of time (18 weeks) the mice were kept under observation in this laboratory, for Campbell allowed his mice "to live their allotted span of life." From the results of his experiments with both tar-containing and tar-free road dust there are excellent indications that the presence of charcoal alone might accelerate the appearance of lung tumors in strain A mice, provided the animals are kept for a sufficient period of time. Perhaps the special susceptibility of the lungs of some mice to induced tumors, which is known  $(4, \delta)$  to be inherited in accordance with genetic principles, is an important factor involved when lung tumors are produced by agents which are not considered to be carcinogenic.

The uniformity in the latent period of induced lung tumors in mice also presents an opportunity for histological studies of the development of induced growths. Accordingly, the lungs removed from all mice of Experiment 2, ranging from 1 to 18 weeks after injection of the carcinogenic compound or charcoal, have been prepared for histological studies which are now in progress.

#### SUMMARY AND CONCLUSIONS

When 1: 2: 5: 6-dibenzanthracene was adsorbed on charcoal and injected subcutaneously into strain  $C_{3}H$  mice, it produced tumors at the injection site.

When charcoal was added to a horse-serum dispersion of 1: 2: 5: 6dibenzanthracene and the resulting mixture injected subcutaneously into strain  $C_3H$  mice, tumors arose more rapidly at the injection site than in control mice injected subcutaneously with the horse-serum dispersion. Induced lung tumors were found in mice injected with the horse-serum dispersion but such tumors were not observed in mice injected with the horse-serum dispersion to which charcoal had been added. These results suggest that charcoal held the carcinogenic compound at the site of injection.

When dibenzanthracene charcoal was injected subcutaneously into strain A mice, it induced tumors at the site of injection in but a few mice, which indicates that the subcutaneous tissues of mice of strain A are more resistant than those of strain  $C_3H$  to the carcinogenic action of dibenzanthracene charcoal. Subcutaneous injection of dibenzanthracene charcoal into strain A mice induced very few, if any, lung tumors which is evidence that the dibenzanthracene was held firmly by the charcoal. This is regarded as further evidence that lung tumors are induced in mice by a direct action of the carcinogenic agent upon the lung tissues.

When dibenzanthracene charcoal was injected intravenously into strain A mice it induced lung tumors within 9 weeks, while intravenous injection of an equal amount of 1: 2: 5: 6-dibenzanthracene as a horseserum dispersion induced lung tumors in strain A mice within 5 weeks. Intravenous injection of charcoal into strain A mice failed to induce any appreciable number of lung tumors within 18 weeks after injection.

## 1940

The localization of dibenzanthracene charcoal in the lungs via the blood stream and the production of tumors by dibenzanthracene charcoal in the lungs show that lung tumors can be produced in strain A mice by placing the carcinogenic agent in contact with lung tissues.

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## **DEATHS DURING WEEK ENDED DEC. 11. 1937**

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

	Week ended Dec. 11, 1937	Correspond- ing week, 1936
Data from 86 large oities of the United States:         Total deaths.         A verage for 3 prior years.         Total deaths, first 49 weeks of year.         Deaths under 1 year of age.         A verage for 3 prior years.         Deaths under 1 year of age, first 49 weeks of year.         Deaths under 1 year of age, first 49 weeks of year.         Deaths under 1 year of age, first 49 weeks of year.         Deaths under 1 nsurance companies:         Policies in force.         Number of death claims.         Death claims per 1,000 policies in force, annual rate.         Death claims per 1,000 policies, first 49 weeks of year, annual rate.	8, 552 8, 618 419, 590 558 546 26, 873 70, 452, 399 12, 820 9, 5 9, 7	8, 790 420, 674 521 

# **PREVALENCE OF DISEASE**

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

# **UNITED STATES**

#### **CURRENT WEEKLY STATE REPORTS**

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers. In these and the following tables a zero (0) is to be interpreted to mean that no cases or deaths occurred, while leaders (.....) indicate that cases or deaths may have occurred although none was reported.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Dec. 18, 1937, and Dec. 19, 1936

·	Diphtheria		Influenza		Measles		Meningococcus meningitis	
Division and State	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut Middle Atlantic States:		4 5 2		7	42 96 130 61 1 5	53 9 1 456 158 116	0 0 0 2 0	1 0 0 2 0 0
New York New Jersey Pennsylvania East North Central States:	43 16 37	32 16 55	1 10 11	1 23 20	128 660 2, 275	215 158 43	9 2 8	8 1 7
Ohio Indiana Illinois Michigan Wisconsin West North Central States:	22 17 30 17 1	45 19 37 20 1	6 57 17 51	5 45 113 	267 50 935 305 141	22 12 27 21 34	4 0 5 1 0	8 2 9 7 0
Minnesota Iowa. Missouri. North Dakota South Dakota Nebraska Kanssa.	1 12 35 1 	15 5 10 1  8 6	2 4 44 18  4	5 85 3  4	4 976 11  23	25 2 7  1 3 10	0 1 0 0 1	1 2 1 0 0 2
South Atlantic States: Delaware. Maryland <sup>1</sup> District of Columbia Virginia. West Virginia North Carolina <sup>1</sup> South Carolina <sup>1</sup> Georgia <sup>1</sup> Florida	22 10 24 18 35 7 25 20	12 10 36 19 70 8 19 12	16 	14  109 12 353 209 9	2 5 6 127 237 434 71 35	53 128 6 46 43 22 20 3	0 0 4 8 1 2 1 0	0 7 3 7 1 4 2 2 8

See footnotes at end of table.

#### December 31, 1937

## 1942

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Dec. 18, 1937, and Dec. 19, 1936—Continued

				nu Dec	. 19, 10				
Division and State		Diphtheria		Influenza		Measles		Meningococcus meningitis	
		Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936
East South Central States: Kentucky. Tennessee <sup>1</sup>		18 11 23 17	15 28 23 10	24 96 258	31 59 117	101 119 12	60 8 2	5 5 10 4	73311
Arkansas. Louisiana Oklahoma <sup>4</sup> Texas <sup>3</sup>		23 27 21 48	4 13 5 74	134 54 98 499	23 12 56 561	17 3 5 36	1 9 72	1 0 3 6	
Mountain States: Montana Idaho Wyoming Colorado New Marico		7	1 1 4 4	3	65 4 	1 11 1 61 49	86 1 44	0000	
Arizona Utah <sup>1</sup> Pacific States: Washington Oregon California		2 1 1 2	4  8 1	73	93  39	69 38 10	44 72 70 20 9	2 1 0 1 0	2
California Total			49 721	32	58 2, 225	71 7, 631	28 2,176	3 80	9
50 weeks of year		26, 697	27, 511	288, 665	153, 949	283, 762	280, 038	5, 226	7, 187
Poli		nyelitis	Scarle	Scarlet fever		Smallpox		Typhoid and paratyphoid fevers	
Division and State	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937
New England States: Maine New Hampshire Vermont Massachusetts Rhode Island Connecticut	1 0 0 0 0	000000000000000000000000000000000000000	45 7 35 207 31 77	24 3 2 178 38 57	0 0 0 0 0	0 0 0 0 0	2 1 1 4 1	1 0 1 0 8	25 9 17 217 43
Middle Atlantic States: New York New Jersey Pennsylvania East North Central States:	0 1 0	2 0 0	410 94 428	496 103 417	0 0 0	47 0 0	6 3 12	8 0 4	875 152 320
Ohio Indiana Illinois Michigan Wisconsin	1 0 0 0 0	5 0 1 1 1	274 167 512 363 140	274 172 423 370 257	2 102 28 0 5	2 1 0 1 6	1 0 1 4 0	4 2 5 8 1	64 11 73 179 153
West North Central States: Minnesota Iowa Missouri North Dakota South Dakota Nobraska Kansas	2 0 1 0 0 0 1	1 0 0 1 0 5	111 233 230 24 31 25 160	140 99 101 25 76 43 250	21 63 2 7 3 0 11	11 15 3 13 10 1 7	0 1 5 0 0 0	2 1 10 0 0 1 2	30 28 22 44 35 1 81

See footnotes at end of table.

	Polion	ayelitis	Scarle	et fever	ver Smallpox		Typhoid and paratyphoid fevers		Whoop- ing cough
Division and State	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937	Week ended Dec. 19, 1936	Week ended Dec. 18, 1937
South Atlantic States:	0	0	12	22				0	
Maryland <sup>2</sup>	1	ŏ	71	69	ŏ	ŏ	3	8	59
District of Columbia	o i	ŏ	16	16	ŏ	ŏ	l ő	3	10
	1	1	58	39	l ö	ŏ	2	6	67
Virginia West Virginia	i	1	08 71	77	ŏ	ŏ	1	6	44
North Carolina <sup>3</sup>	0	ŏ	50	65	1	2	4	1	192
South Carolina *	ŏ	ŏ	15	6	Ō	ő	2	2	23
	1	4		31	ő	ŏ	3	6	15
Georgia *	1		43 13	10	ő	ŏ	°7	ő	10
Florida.	1	1	13	10	U	0	· · · ·		
East South Central States:									
Kentucky	1	1	72	. 63	4	0	0	3	57
Tennessee 3	0	1	45	· 45	1	0	1	3	24
Alabama *	1	3	20	20	0	0	1	5	9
Mississippi <sup>a</sup> West South Central States:	1	0	9	13	1	0	0	3	
West South Central States:		_					-		
Arkansas	2	3	25	17	1	0	2	0	19
Louisiana	2	1	15	16	0	0	12	8	14
Oklahoma 4	1	4	76	11	0	0	1	2	16
Texas 3	3	0	99	117	8	4	24	9	123
Mountain States:									
Montana	1	0	23	61	15	28	1	0	37
Idaho	0	0	11	48	20	0	0	3	6
Wyoming	0	0	12	12	11	1	0	0	14
Colorado	0	0	43	21	6	7	3	1	11
New Mexico	Ó	0	15	22	0	0	3	2	9
Arizona	Ó	0	9	15	0	0	0	1	16
Utah 3	Ō	Ó	96	23	0	0	0	0	18
Pacific States:	-	-							
Washington	0	1	35	47	12	1	0	2	87
Oregon	å	2	76	43	9	29	2	2	20
California	5	6	172	305	14	2	13	6	308
			4 004	4 702	347	191	128	135	3, 106
Total	32	45	4, 806	4, 783					
50 weeks of year	9, 391	4, 452	21 <b>4, 311</b>	224, 182	10, 444	7, 144	14, 827	14, 387	

1 New York City only.
Week ended earlier than Saturday.
3 Typhus fever, week ended Dec. 18, 1937, 47 cases, as follows: North Carolina, 3; South Carolina, 1; Georgia, 30; Tennessee, 2; Alabama, 6; Texas, 5.
4 Figures for 1936 are exclusive of Oklahoma City and Tulsa.

#### SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Menin- gococ- cus menin- gitis	Diph- theria	Influ- enza	Mala- ria	Mea- sles	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
Nocember 1937 Alabama Florida Georgia Indiana Louisiana Minnesota Minnesota New York Ohio Pennsylvania Vermont	19 6 8 6 2 7 8 24 15 16 0	145 109 123 132 108 52 93 189 173 7	354 27 369 102 32 5 3 	172 59 291  76 9  11 2 2	18 98 168 97 2 407 22 390 908 4,409 295	20 2 16 	6 3 8 12 13 21 28 4 9 0	96 34 123 579 56 1, 714 518 1, 245 1, 391 1, 412 50	1 4 23 73 4 7 52 0 4 0 0	17 24 20 333 13 4 36 37 80 8 8 8 8

#### Summary of monthly reports from States-Continued

#### November 1937

A shin smarste	Cases		Cases	١.
Actinomycosis	1	German measles-Con.	62	Ľ
Michigan	1	New York	02 18	L
Anthrax:		Ohio Pennsylvania	86	Ł
Louisiana	1		90	L
New York	1	Hookworm disease:		
Pennsylvania	1	Florida	1, 154	L
Chicken pox:		Georgia		Ŀ
Alabama	93	Louisiana	326	
Florida	25	Jaundice, infectious:		
Georgia	129	Michigan	1	
Indiana	233	Lead poisoning:		Ŀ
Louisiana	12	Ohio	7	Ł
Michigan		Mumps:	•	
Minnesota	690	Alabama	22	1
New York Obio	1,747	Florida	16	
Pennsylvania	1,708	Georgia	57	L
Vormont	3, 088	Indiana	16	L
Vermont	201	Michigan	455	
Conjunctivitis, infectious:	_	Ohio	188	
Georgia	7	Pennsylvania		
Dengue:		Vermont	395	L
Florida	1	Ophthalmia neonatorum:		ľ
Georgia	1	Alabama	2	Ł
Diarrhea and enteritis:		Florida	2	Ł
Ohio (under 2 years)	17	Louisiana	ĩ	L
Dysentery:		New York 1	10	Ι.
Alabama (amoebic)	1	Ohio.	61	1
Florida (amoebic)	i	Pennsylvania	ň	
Georgia (amoebic)	5	Paratyphoid fever:		Ł
Georgia (bacillary)	8	Florida	1	
Louisiana (amoebic)	2	Michigan	4	
Louisiana (bacillary)	5	New York	8	
Michigan (amoebic)	6	Ohio.	ĭ	
Michigan (bacillary)	12	Puerperal septicemia:	•	
Minnesota (amoebic)	5	Ohio	5	
New York (amoebic)	8	Rabies in animals:		
New York (bacillary)	106			
Pennsylvania (amoe-		Alabama	59	1
bic)	1	Florida	8	
Pennsylvania (Dacii-		Indiana	40 36	
lary)	8	Louisiana	30	
Encephalitis, epidemic or		Michigan New York <sup>1</sup>	3	٦
lethargic:			•	
Alabama	1	Rabies in man:		
Georgia.	1	Ohio	1	
Louisiana	1	Septic sore throat:		
Minnesota.	27	Florida	1	
New York		Georgia	50	
Ohio	•	Louisiana	5	í.
German measles:	.	Michigan	36	
Alabama	3	Minnesota	7	
Florida	57	New York	57	
Michigan	0/ [	Ohio	65	

~	German measies—Con.	
1	New York	62
	Ohio Pennsylvania	18
1	Pennsylvania	86
1	Hookworm disease:	
1	Florida	1.154
	Georgia	2,359
93	Louisiana	326
25	Jaundice, infectious:	
29	Michigan	1
33	T and material and	- 1
12	Lead poisoning:	
39	Ohio	7
90	Mumps:	
47	Alabama	22
08	Florida	
88	Georgia	57
87	Indiana	16
	Michigan	455
	Ohio	188
7	Ohio Pennsylvania	2.041
	Vermont	395
1	Ophthalmia neonatorum:	
1	Alabama	2
i	Florida	2
17	Louisiana	ĩl
	New York 1	10
1	Ohio.	61
1 1 5	Pennsylvania.	ii l
ŝ	Paratyphoid fever:	
2		.
2	Florida	1
825	Michigan	4
8	New York	8
12	Ohio	1
5	Puerperal septicemia:	
8	Ohio	5
xā İ	Rabies in animals:	1
~	Alabama	59
1	Florida	8
- 1	Indiana	40
8	Louisiana	36
- I	Michigan	3
	Michigan New York 1	3
1	Rabies in man:	
ī	Ohio	1
ī	Septic sore throat:	- 1
$\overline{2}$	Florida	1
2 7 4	Georgia	50
4	Louisiana	5
-	Michigan	36
<u>a</u>	Minnesota	7
31	New York	57
źΙ	Ohio	65
	VM.V	~ 1

_		<b>^</b>
5	Tetanus:	Cases
2	Alabama	7
B	Georgia	2
6	Louisiana	5
	Michigan	3
4	New York	9
9	Ohio	2
6	Trachoma:	
	Alabama	6
1	Minnesota	1
•	Pennsylvania	2
_	Trichinosis:	
7	New York	- 4
	Pennsylvania	1
2	Tularaemia:	
2 8 7 8 5 8 1	Alabama	
7	Florida	
3	Indiana	
5	Louisiana	
ŝ	Minnesota	
i	Ohio	4
5	Pennsylvania	1
	Typhus fever:	
,	Alabama	
2	Florida	7
í	Georgia	72
5	Louisiana	3
í	Undulant fever:	
i	Alabama	2
•	Florida	3
1	Georgia	1
IJ	Indiana	2
H	Louisiana	
3	Michigan	3
	Minnesota	4
	New York	16
5	Ohio	6
	Pennsylvania	7
۱	Vermont	2
i	Vincent's infection:	
1	Florida	83
1	Michigan New York 1	16
1	New York 1	76
1	Whooping cough:	
1	Alabama	51
	Florida	
	Georgia	59
1	Indiana	109
. [	Louisiana	48
) [	Michigan	792
1	Minnesota	274
	New York	1, 595
1	Ohio Pennsylvania	579
1	Pennsylvania	1, 446
	Vermont	135

<sup>1</sup> Exclusive of New York City.

#### CASES OF VENEREAL DISEASES REPORTED FOR OCTOBER 1937

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.

	Syl	philis	Gon	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
Alabama	1, 995	6.97	442	1.54
Arizona	86	2.12	97	2.39
Arkansas	742	3.67	355	1.75
California Colorado <sup>1</sup>	1, 533	2. 53	1, 493	2.46
Connecticut	273	1. 57	148	.85
Delaware	205	7.92	27	1.04
District of Columbia	195	3. 15	146	2.36
Florida 1	2, 316	14.10	258	1.57
Georgia	1,981	6.47	473	1.55
Idaho	78 1,894	1.61	45	.93 1.86
Illinois Indiana	318	.92	1,401	37
Iowa	394	1.55	232	.91
Kansas	152	. 81	104	.55
Kentucky	634	2.20	363	1, 26
Louisiana	498	2.35	105	.49
Maine	56	. 66	61	.72
Maryland	1, 083 424	6.47	368	2.20 1.01
Massachusetts Michigan	722	1.51	538	1.01
Minnesota	307	1.17	312	1.18
Mississippi	1,908	9.50	2, 565	12.78
Missouri	811	2.05	420	1.06
Montana 1				
Nebraska	63	.46	121	. 89
Nevada 3	11	. 22	11	. 22
New Hampshire	887	2.05	415	. 24
New Mexico <sup>3</sup>	143	3.39	39	.92
New York	9, 753	7.54	2, 106	1.63
North Carolina	3,064	8.86	726	2, 10
North Dakota	25	. 36	45	. 64
Ohio	1, 253	1.87	444	.66
Oklahoma	677 106	2.68 1.04	481 235	1.90 1.31
Oregon Pennsylvania 4	1.860	1.84	289	.29
Rhode Island	.,000	1.28	36	. 53
South Carolina <sup>3</sup>	355	1.91	436	2.34
South Dakota	38	. 55	27	. 39
Tennessee	931	8.25	429	1,50
Texas.	743	1.21	279	.46
Utah	2 20	. 04 . 53	10 33	. 19
Vermont Virginia	1.062	. 53 8. 98	354 354	1.33
Weshington	347	<b>2</b> . 11	457	2.78
West Virginia <sup>3</sup>	437	2.39	183	1.00
Wisconsin	27	. 09	113	. 39
Wyoming *				
Total	40, 496	3.20	17, 855	1.41
& VW4	,			

#### **Reports from States**

#### Reports from cities of 200,000 population or over

		r	T	
Akron, Ohio 1				
Atlanta, Ga	254	8.85	136	4. 74
Baltimore, Md	702	8.51	227	2.75
Birmingham, Ala	800	10.63	104	3.68
Boston, Mass	151	1.91	153	1.93
Buffalo, N. Y	190	3. 21	103	1.74
Chicago, Ill	1,058	2.97	968	2.71
Cincinnati, Ohio	135	2,90	62	1.33
Cleveland, Ohio	215	2.31	94	1.01
Columbus, Ohio	65	2.13	24	. 79
	184	6.35	76	2.62
Dallas, Tex Dayton, Ohio	83	3. 95	18	. 86
Dayton, Unio	51	1.72	37	1.25
Denver, Colo	315	1.82	240	1.39
Detroit, Mich	910 1	1.96 1	ATO 1	

See footnotes at end of table.

	871	hilis	Gonorrhes		
	Cases re- ported during month	Monthly case rates per 10,000 population	Cases re- ported during month	Monthly case rates per 10,000 population	
Houston, Tex.4 Indianapolis, Ind.4		5. 58	29	.87	
Jersey City, N. J. Kansas City, Mo Los Angeles, Calif.	110	2. 61	12	. 28	
Louisville, Ky Memphis, Tenn Milwaukee, Wis. <sup>1</sup>	185 245	5. 71 9. 18	160 88	4. 94 8. 30	
Minneapolis, Minn. Newark, N. J. New Orleans, La.	80 336	1.64 7.25 1.61	82 145 47	1.69 8.13 .98	
New York, N. Y. Oakland, Calif. Omaha, Nebr.	8, 366 55	11.45 1.81	1, 518 44	2.08 1.45	
Philadelphia, Pa Pittsburg, Pa	515 114	1.72 2.59 1.67	53 	2. 41 . 44	
Portland, Oreg. <sup>1</sup> Providence, R. I. Rochester, N. Y	43 36	1.66 1.07	21 52	.81 1.54	
St. Louis, Mo St. Paul, Minn San Antonio, Tex	29 108	1. 34 1. 03 4. 30	88 14 80	1.05 .50 8.18	
San Francisco, Calif Seattle, Wash Syracuse, N. Y	139 94	2, 24 3, 66 4, 31	223 173 56	8.32 4.56 2.57	
Toledo, Óhio Washington, D. C. <sup>4</sup>	115 195	3. 78 3. 15	46 146	1. 51 2. 36	

#### Reports from cities of 200,000 population or over-Continued

No report for current month.
 Incomplete.
 Not reporting.
 Only cases of syphilis in the infectious stage are reported.
 Reported by Jafferson Davis Hospital.
 Reported by the Social Hygiene Clinic.

#### WEEKLY REPORTS FROM CITIES

City reports for week ended Dec. 11, 1957

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

				-									
State and city	Diph-		Diph- theria	Inf	luenza	Mea-	Pneu- monia	Scar- let		Tuber-	Ty- phoid	Whoop-	Deatos,
State and city	Cases	Cases	Deaths	Cases	deaths	fever cases	pox cases	culosis deaths	fever cases	cough cases	all causes		
Data for 90 cities: 5-year average Current week <sup>1</sup> .	<b>2</b> 81 160	837 233	95 55	879 2,060	747 616	1, 405 1, 165	10 60	369 303	83 16	979 900			
Maine: Portland New Hampshire:	0		0	1	3	0	0	0	0	19	17		
Concord Nashua Vermont:	0		0	11 0	1	0	0	0 	0	. 0 0	13 7		
Barre Burlington Rutland Massachusetts:	0 1 0	 	0 0 0	57 0 8	0 0 0	000	0 0 0	1 0 0	000	0 0 8	8 10 7		
Boston Fall River Springfield Worcester Bhode Island:	0 0 0 0		8 0 0	41 1 0 1	16 5 1 4	51 2 9 6	0 0 0	4 1 0 1	0 0 0 0	5 21 15 13	225 24 42 55		
Pawtucket Providence Connecticut:	0		0 0	0 1	0 4	4 21	0	0 2	0	0 13	19 61		
Bridgeport Hartford New Haven	0	1	000	1 0 0	3 3 1	10 17 8	000	0 1 1	000	8 0 5	28 89 47		

<sup>1</sup> Figures for Newark estimated; report not received.

City reports for week ended Dec. 11, 1937-Continued	
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	Diph-	Inf	uenza	Mea-	Pneu-	Scar- let	Small-	Tuber-	Ty- phoid	Whoop-	Deaths,
State and city	theria cases	Cases	Deaths	sles cases	monia deaths	fever cases	pox cases	culosis deaths	fever cases	cough cases	all causes
New York: Buffalo New York Rochester	0 29 1	 13 2	1 7 0	1 20 3	6 86 5	20 141 8	0 0 0	6 62 1	040	17 109 3	138 1, 397 71
Syracuse New Jersey: Camden	0		0	0	4	10 5	0	. 1	0	5	66 43
Newark											
Trenton Pennsylvania: Philadelphia	0	1	0	94 35	0 28	1 77	0	2 24	0 4	8 44	30 461
Pittsburgh Reading Scranton	4 0 0	4	4	332 0 13	23 1	28 2 0	0 0 0	9 3	0 0 0	26 1 1	211 21
Ohio:											
Cincinnati Cleveland Columbus Toledo	5 1 0 4	16 3 3	0 2 3 0	3 73 4 16	16 14 10 7	19 49 17 5	0 0 0	6 10 3 10	0 0 0 0	3 89 4 8	138 187 92 81
Indiana: Anderson	Q		0	0	2	8	0	0	0	1	14
Fort Wayne Indianapolis	2		0 1	06	4	1 26	0	0	0	0 5	30 96
South Bend Terre Haute	0		0	3 3	3	4	0	0	0	0	20 16
Illinois: Alton	1		0	61	0	3	0	0	0	3	9
Chicago Elgin	13 1	12	1 0	246 0	43	163 9	6	28 0	Ŏ	38	721 8
Moline Springfield	Ô		Ŏ	12 1		12 14	04	0	0	0	11
Michigan:	-			-	1 1			0	0	0	23
Detroit Flint	12 1		3 0	143 1	19 4	93 14	0	20 0	0	16 33	271 35
Grand Rapids Wisconsin:	0		0	4	3	23	0	1	0	11	36
Kenosha Madison	0		0	0		2 3	0	0 1	0	17	14 13
Milwaukee	Ŏ	1	1 0	48 0	11	22 7	0	1	Ó	27	109
Racine Superior	ŏ		ŏ	1	0	6	0	8	0	2 0	9 7
Minnesota:											
Duluth Minneapolis	0		0	23	0 1	227	1 0	0	0	6 13	27 113
St. Paul Iowa:	2		0	2	2	2	21	1	0	2	52
Cedar Rapids Davenport	0			0		1	0		0	0	
Des Moines	0			Ŏ		33	0		0	0	31
Sioux City Waterloo	0			0 1		03	0		8	0	
Missouri: Kansas City	0		1	0	9	25	0	7	0	3	119
St. Joseph St. Louis	1		0	0 708	6	3 41	02	07	02	0	17 231
North Dakota:	0		0	0	0	2	0	0	0	8	6
Grand Forks Minot	Ō			Ō		7	1		Ó	0	
South Dakota:	0		U I	0	0	1	0	0	0	4	2
Aberdeen Sioux Falls	0		0	0	0	02	0	0	00	2 0	14
Nebraska: Lincoln Omaha	1 2			0 1	5	0	0	0	0	0	60
Kansas: Lawrence	0		0	0	0	0	0	0	0	6	7
Topeka Wichita	Ŏ.	1	Ŭ 1	Ŭ 2	0 3	47	ŏ	ŏ	Ŏ	11 5	19 19
Delaware: Wilmington	1		0	0	7	3	0	1	0	,	37
Maryland: Baltimore	16	,	3	2	21	23	o	13	0		232
Cumberland	0 .		0	0	2	0	0	0	0	84 0	9
Frederick Dist. of Columbia:	<u> </u>		0	0	0	0	0	0	0	0	2
Washington	71	81	11	6	12	12 1	01	<b>#</b> 1	21	41	130

#### City reports for week ended Dec. 11, 1937-Continued

State and site	Diph-		uenza	Mea-	Pneu-	Scar- let	Small	Tuber-	Ty- phoid	Whoop	Death
State and city $f_{\rm for}$	theria cases		Deaths	sles cases	monia deaths	fever cases	pox cases	culosis deaths	fever	cough	all cause
Virginia:		·									
Lynchburg	1		0	0	2	1	0	0	0	0	
Norfolk	0		0	1	6	12	O O	8	Q	1	
Richmond	2		1	0	13	1	0	. 0	0	. 0	
Roanoke	Ĩ		0	1	3	2	0	1	0	0	
West Virginia:											
Charleston	1	1	0	0	8	1	0	1	0	0	:
Huntington	0			4		0	0		0	0	
Wheeling	1		0	1	8	0	0	1	0	6	
North Carolina:	_										· ·
Gastonia	1			0		0	0		0	3	
Raleigh	0		0	0	7	0	0	2	0	6	
Wilmington	0		0	0	0	0	0	0	0	10	1 1
Winston-Salem.	1		0	1	0	4	0	1	0	5	
outh Carolina:			-				_				
Charleston	0	89	1	- 4	6	0	0	0	0	0	8
Florence	0		0	0	1	0	0	0	0	1	1
Greenville	0		0	0	2	0	0	0	0	5	1
leorgia:			_							_	
Atlanta	1	41	3	26	0	7	0	4	0	7	8
Brunswick	0		0	0	0	0	0	0	0	0	
Savannah	1	20	0	0	7	0	0	1	0	0	8
lorida:	_										
Miami	1	1	0	16	8	1	0	2	0	1	1
Tampa	1	1	1	1	4	1	0	0	0	0	2
Kentucky:			1								
Ashland	0		0	0	1	0	0	1	0	0	
Covington	ĭ		ŏl	ŏ	ŝ	ŏ	ŏ	ō	ŏ	ŏ	1
Lexington	ō		ŏl	ŏĺ	3	ĭ	ŏ	2	ŏ	ŏ	i
Louisville	ŏ	4	ĭ	50	13	15	ŏl	5	ŏ	4	2
ennessee:	•	-	- 1	~			×			-	
Knoxville	1	8	1	3	8	ol	0	0	0	0	. 3
Memphis	ō	, v	ôl	85	7	¥	ŏl	3	ŏ	4	8
Nashville	3		ŏ	ĩ	8	2	ŏ	ŏ	ŏ	ō	6
labama:			۰ı	- 1	°	-	•	•	۷I	v	0
Birmingham	4	18	2	13	7	2	0	6	0	0	7
Mobile	ō		õ	ŏ	4	ő	ŏ	5	ŏl	ŏ	2
Montgomery	ĭ	5	•	ŏ	*	ĭ	ŏ		ŏl	ŏ	4
	-	•				- 1			•	•	
rkansas:									1		
Fort Smith	0			0		4	0		0	2	
Little Rock	0		1	13	3	8	0	8	0	1	
ouisiana:		- 1									
Lake Charles	0			0	8	0	0	0	0	0	
New Orleans	11	11	7	1	32	2	0	8	1	2	19
Shreveport	0		0	0	13	2	0	1	0	0	6
klahoma:		- 1				I		1			-
Muskogee	1	.		0		3	0		0	0	
Oklahoma City_	0		2	0	8	0	0	2	0	0	4
Tulsa.	1			0		5	1		01	8	-
exas:									-	-	
Dallas	2	1	1	01	3	13	0	2	0	2	5
Fort Worth	1		11	Ó	7	4	Ō	Ō	Ó	ō	4
Galveston	2		Ō	Ó	7	ī	ŏ	1	ŏ	ŏl	- Ž
Houston	ī		ŏ	3	12	ŝΙ	ŏl	ô	ŏl	ŏ	10
San Antonio	i		il	ĭ	4	ŏl	ŏl	8	ŏ	ŏ	6
1	-		- 1	- 1	- 1	-	-	- 1	- 1	-	•
Iontana:		1									-
Billings	<u>o</u>		0	0	3	0	.0	0	0	0	1
Great Falls	0		0	0	8	8	12	0	0	14	
Helena			0	0		0	0	0	0	4	
Missoula	0		0	0	0	0	0	0	0	0	
aho:							_				
Boise	0		0	0	1	0	9	0	0	0	
olorado:						- 1					
Colorado											
Springs	0		0		1	2	0	1	0	0	1
Denver	4		0	33	5	12	0	6	1	1	9
Pueblo	0		0	1	0	5	1	0	0	8	
ew Mexico:		1			.		_ 1	_			
Albuquerque	1		0	10	0	2	0	2	0	1	10
tah:	.						_		_ [	_ 1	
Salt Lake City_	0.		0	2	4	12	0	0	0	5	2
ashington:	I			1			1				
Seattle	0		0	1	8	8	0	2	0	21	78
Spokane	ŏ		ŏ	ð	4	2	i	ő	ŏ	14	40
Tacoma	ŏ		ŏ		i		8	öl	ŏl		
1 acoma	•  -		۲ <b>۲</b>	1	- 1	5	•	•	<b>ا</b> ۳	7	44
Portland		8	0	0	2	9	0	0	0		
Portland	6		0		z			٥l		4	65
Salem	0	2		1		1	0  -		0	0  -	
				- 1	~		0	9	2		
alifornia:		400									
alifornia: Los Angeles Sacramento	6	17 13		5	20	<b>3</b> 3	ŏ	i	ő	50 36	841 29 170

State and city		ococcus ngitis	Polio- mye- litis	State and city		gococcus ngitis	Polio- mye- litis
	Cases	Deaths	cases		Cases	Deaths	Cases
Connecticut: Hartford	1 1 2 1 0 1 0 0 0 0 1	0 0 1 1 6 0 0 0 0 0 0 0 0	0 0 1 0 0 1 2 1 1 1 1 0	Maryland: Baltimore	1 0 0 5 1 0 1 1 1 2	1 1 1 0 0 3 0 0 1	0 0 0 0 0 0 0 0 0 0 0

City reports for week ended Dec. 11, 1937-Continued

Encephalitis, epidemic or lethargic.—Cases: St. Louis, 1. Pellagra.—Cases: Philadelphia, 2; Baltimore, 1; Lynchburg, 1; Atlanta, 3; Savannah, 2; Memphis, 1; Birmingham, 2. Typhus fever.—Cases: Savannah, 3.

#### FOREIGN AND INSULAR

#### **CUBA**

Provinces—Notifiable diseases—4 weeks ended November 13, 1937.— During the 4 weeks ended November 13, 1937, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matan- zas	Santa Clara	Cama- guey	Oriente	Total
Cancer Chicken pox	1	12	1	9		2	14 2
Diphtheria Leprosy	2	20 1	2	1	1	1	26 6
Malaria Measles	47	176	117 4	135	17	103 1	<b>5</b> 95 9
Poliomyelitis Tetanus		8				5	8
Tuberculosis. Typhoid fever	44 12	17 53	30 15	51 28	37 10	23 12 11	202 130 11
4 6 10							

#### DENMARK

Notifiable diseases—July-September 1937.—During the months of July, August, and September 1937, cases of certain notifiable diseases were reported in Denmark as follows:

Disease	July	August	September
Cerebrospinal meningitis Chicken poz	13	8 7 56	4 8 86
Diphtheria and croup Epidemic encephalitis Erysipelas	4	242	285
German measles	19 922	6 1,061	5 991
Influenza Malaria	<b>2</b> , 131 8	2, 562 <u>4</u>	<b>4,</b> 653 5
Measles Mumps	55 420	49 285 425	145 864 138
Paradysentery Paratyphoid lever Poliom velitis	13	423 50 272	138 20 567
Puerperal fever		18 911	13 1, 832
Scarlet fever Syphilis	514 59	705 50	1, 181 64
Tetanus neonatorum	31	3	5
Typhoid fever Undulant fever (Bact. abort. Bang)		5 50 2	88
Weil's disease Whooping cough	711	638	782

(1950)

#### ITALY

Communicable diseases—4 weeks ended October 10, 1937.—During the 4 weeks ended October 10, 1937, cases of certain communicable diseases were reported in Italy as follows:

	Sept.	13-19	Sept.	20-26	Sept. 2	7-Oct. 3	Oct.	4-10
Disease	Cases	Com- munes affected	Cases	Com- munes affected	Cases	Com- munes affected	Cases	Com- munes affected
Anthrax. Carebrospinal meningitis Chicken pox. Diphtheria. Dysentery. Hookworm disease. Lethargic encephalitis Meesles. Mumpe Paratyphoid fever Poliomyelitis Puerperal fever Scarlet fever Typhoid fever Undulant fever Whooping cough	33 15 53 575 77 14 2305 102 220 40 32 253 1,438 31 246	26 11 37 334 53 7 2 117 58 142 31 142 31 108 683 30 108 683 27 123	42 14 68 601 40 22 280 77 191 61 25 258 1, 316 36 252	87 12 51 304 32 12 12 12 12 12 12 12 12 12 12 12 12 12	32 10 49 667 41 26 1 334 52 151 64 28 266 1,242 26 178	26 4 35 329 30 10 1 145 36 106 49 27 134 584 23 89	31 8 80 659 68 23 298 60 156 43 43 289 1, 127 50 224	25 8 54 330 35 111 38 130 38 130 38 40 143 564 44 42 82

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 particular countries for which reports are given.

### CHOLERA

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

	Apr.	May	June							Weel	Week ended-						
Place	May 28	June 9	July 31	Aug. 1-28, 1037	ž	September 1937	ır 1937			Oetc	October 1937	~		4	November 1937	sr 1937	
	1937	1937	1937	5	4	п	18	35	8	8	16	ន	30	ø	13	30	21
China: Canon Ganon			8	8	Ş	5	16	2	~	-	•						
			5				00	-		4							
			3~ *	964	303	205	8 <u>1</u> 8	88	28	8	13	- 	-			- 0	1
Kwangchow Wan			•	, Ž			3	3	នន	13	"8		•	- 4	1 00 9		
************************				788 788	20	22	38	16	30	12	8	•		*1	•	-	
Datren									89	1	- 10				Ť		
								<u>~</u>		2				İİ	İİ		
Nanking										8 2 3	Ī	142					
Shanghai				00 0	8°	36	128	655	613	- 88: 89:	350	91	8:	5	<b>6</b>	99	9
Tientein				0	•	3	•		-	- 10 -				5		*	
Chosen Colored Calabas						-	Ī				T						
Federated Malay States			2														
	<b>1</b>	11, 113	13, 893	18, 259	4, 316		2, 834	2,200	1, 623	2, 254	2, <del>1</del> 88 10						
	.બન	282		88	9 8 9	22	1=-		3 2 2 2 2 2 2 2		04091	20	31	13	22	93	99
	ର୍ଚ୍ଚ <del>-</del> - ବି	1, 713 1, 713	2, 936 1, 098	4, 273	1, 143	890 374	762 118	571 282	391	<b>4</b> 30 212	311	ន្តន	81 113	12			
Calcutta. Coloutta. Contral Provinces and Berar	- <u>8</u> 33 -	173 318	-88,	176	<b>,</b> ≭%	28	20 20 20	52 52	154	218	113	211	25	20	83	18 102	8
	ਜੱਜ 	1,048 544	2, 796 1, 182	4, 210	898 378	475 208	393 393 167	12.02	35	82 82 82 82		83	222				

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$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	<ul> <li>ressels-Continued.</li> <li>S. Hatching at Hong Kong.</li> <li>S. Craina at Singapore from Hong Kong Kong, and S. Crainer at Singapore from Amoy, Hong Kong, and Bustow</li> <li>S. Tyndaret at Kole from Hong Kong and Dairen.</li> <li>S. Anting at Singapore from Hong Kong.</li> <li>S. Anting at Singapore from Hong Kong.</li> <li>S. Sphirz at Singapore from Hong Kong.</li> <li>S. Shiraptow at Hong Kong from Sharghal.</li> </ul>
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<sup>6</sup> For reports prior to Apr. 25, 1837, see previous issues of PUBLIC HEALTH REPORTS. <sup>6</sup> A report states that up to Sept. 30, cholera was reported in Japan, as follows: Hlogo Prefecture, 1 case, 1 death; Hiroshima Prefecture, 40 cases, 14 deaths; Yamaguchi Pre-fecture, 2 cases. 1 death. <sup>6</sup> In addition, for week anded July 28, 3 cases with 2 deaths in contacta.

PLAGUE

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

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December 31, 1937

<b>Haw</b> ali Territory: Plague-infected rats: Hawaii Island-Hamakua District: Hamakua Mill Sector <sup>4</sup>				80									2			 
Honokas Sector	*	4		1									1		3	 . <b>m</b>
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Plague-infected rats	- 35 2 363 63 2	~~~~~	18831	<b>3</b> 88	812	80.02	8280	141 141	17 13 31	18 10 121	8 12 12	11 <b>4</b> 8	86 e 23	120 80	84 183	1 1 1 2
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Imported.
 Pneumonic plague.
 For 2 weeks.

## **PLAGUE**—Continued

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

	Apr.	Mav	June							We	Week ended-	Ĩ					
Place	25- 30- May June	Jun Jun	July	Aug. 1-28,		September 1937	ber 1937			õ	October 1937	37			Novem	November 1937	
	29, 1937	26, 1937	31, 1937		4	11	18	52	2	6	16	8	30	•	13	8	2
United States: • California: • 10 11 Eldorado County - Plague-infected Fresno County 10 Plague-infected fless.					-			•									
Place Topuer uncould ground aquartes Placer County 11-D lagrue-infected fleas Ban Bernardino County-Plague-in- fected fleas. San Mateo County-Plague-infected fleas, lice, and ticks.						1 1 1 1 1		。 。									
Idado: V Bannock County-Flague-in- Idected ground squirrels Montanas: Betwerbead County-Plague-infected		<b>E</b>	-														
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Dragons County - Plague infected Gragons and Ilce.		4															
		1															
Utah: 9 Morgan County — Flague - infected Wastch County — Plague - infected envind southers				-													
Washington: <sup>9</sup> Adams County-Plague- infected fleas and lice.																	

August Bep. October 1937 1937 1937	880-10 E
Sep- tember 1937	\$\$\$ 10 a 2 \$
August 1937	880-1488 95-09 95-09 95-09 95 95-09 95 95-09 95 95 95 95 95 95 95 95 95 95 95 95 95
July 1937	841-1480
June 1937	884-0 -
M.ay 1937	2821-0 <b>60</b>
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August 1937	- 79 - 76 - 6 - 6 - 1 - 1 - 1 - 1 
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June 1937	4 4 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2
May 1937	
Place	Argentina: Cordoba Province

<sup>7</sup> Pneumcnic plague.

• Piscue miestion proved in insect hosts as follows: Catifornia-Eldorado County, Aug. 31; Fresno County, Oct. 7-Nov. 5; Placer County, June 22; San Bernardino County, July 12-Sept. 8; San Matso County, July Aug. 27. Idaho-Bannock County, July 8. Newda-Douglas County, July 29-31; Ormsby County, July 2-Aug. 20. Orgon-Lake County, May 7; Wallowe County, June 20. Utaho-Morgan County, reported Aug. 10. Washington-Adams County, Apr. 29, 1877. Nallowe County, June 22, San Bernardino County, May 7; Wallowe County, June 22. Utaho-Morgan County, reported Aug. 10. Washington-Adams County, Apr. 29, 1877. Nallowe County, June 26. Utaho-Morgan County, reported Aug. 10. Washington-Adams County, Apr. 29, 1877. Nallowe County, June 26. Utaho-Morgan County, reported Aug. 10. Washington-Adams County, Apr. 29, 1877. Nallowe County, June 26. Utaho-Morgan County, reported Aug. 10. Washington-Adams County, Apr. 29, 1877. Nallowe County, June 26. Utaho-Morgan County, reported Aug. 10. Washington-Adams County, Apr. 29, 1877. Nay 76. San Bernardin County, Reverse County, Apr. 29, 1877. Nallowe County, Take Reverse County, Apr. 29, 1877. Natheward County, Take Reverse County, Apr. 20, 1877. Natheward County, Take Reverse County, Apr. 29, 1877. Natheward In Pooled tissue from squirtels, chipmunks, and mice in Fresno County.

## SMALLPOX

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

	Apr.	May 30-	June 27-	Ane.						Weel	Week ended	1					
Place	May 29,	June 28,	July 31,	1-28, 1937		September 1937	er 1937			ő	October 1937	37		4	November 1937	er 1937	
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## **SMALLPOX**—Continued

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

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**SMALLPOX**-Continued

[C. indicates cases: D. deaths; P. present]

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	Place	Argentina. Belgian Comgo. La Par. Cohosen Manchuria-Harbin. Cohosen Manchuria-Harbin. Colombia (see also table above). Grance. Arause also table above). Didochina (French) (see also table above). Aguaselientes State. Conhuna State. Conhunanue State. Conhunanue State. Conhunano State. Conhunano State. Condulas State.	On vessels: 8. G. G. Pasquier at Singapore from Baigon 8. S. Changte at Thurstak Sland 8. S. Empres of Japan at New York from Rio de Janeiro 8. S. Northern Prince at New York from Rio de Janeiro

<sup>4</sup> July and August.

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

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# **TYPHUS FEVER-Continued**

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

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Place	25- May	June	27- July		August 1937	1937		Se	September 1937	r 1937			Octol	October 1937			November 1937	aber 1	31
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## YELLOW FEVER

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

	Apr.	May										Week ended-									
<b>Flace</b>	26, 29, 29, 20, 1	s <sup>2</sup> <sup>2</sup> <sup>2</sup> <sup>2</sup>	July 31,		August 1937	: 1937		σδ.	September 1937	er 1937			Octob	October 1937			Nov	November 1937	1937		₽ ₽
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<sup>1</sup> See also reports of yellow fever in Brazil on pp. 463, 536, 657, 683, 762, 818, 912, 1134, 1248, 1327, 1471, and 1637 of the PUBLIC HEALTH REFORTS. <sup>3</sup> Includes 1. <sup>4</sup> A dispatch dated case. <sup>5</sup> During the week ended Dec. 18, 1937, 1 suspected case of yellow fever was reported in the northwestern part of Paraguay. <sup>5</sup> During the week ended Dec. 18, 1937, 1 suspected case of yellow fever was reported in Baol, Senegal. <sup>6</sup> Imported.

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