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THE PICTURE OF HEART DISEASE MORTALITY OBTAINED FROM VITAL STATISTICS IN WASHINGTON, D. C., DURING 1932 [1]

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According to vital statistics, heart disease is the leading cause of death. These statistics unfortunately are not based on the etiology of heart disease but largely on organic changes. As a result it is difficult to interpret heart disease mortality according to current clinical concepts. In this article an attempt is made to show the incidence of heart disease in various age groups and races, by sex, and to point out how the various etiological factors operate, even though not mentioned specifically in the official tabulation of heart disease mortality.

Heart disease mortality statistics are far from perfect. Many well-recognized forms of heart disease, such as congenital malformations and deaths from heart disease in rheumatic fever and acute rheumatic carditis, are not tabulated as heart disease. Deaths from syphilitic heart disease, thyrotoxic heart disease, the myxedema heart, and other less common forms of heart disease are recorded as due to their respective etiological factors when reported using etiological terminology. Furthermore, it is quite likely that about 25 percent of the reported deaths from heart disease are due to other conditions.[2]

Vital statistics represent the picture of mortality determined by the mass of practicing physicians filing death certificates and the method of tabulation by official agencies. To this extent they depict a cross-section of American medicine, both private and official.

A total of 7,949 death certificates filed with the registrar of vital statistics in Washington, D. C., during 1932 was examined.[3] Abstracts were made of 1,631 certificates, these having been classified by the registrar as heart disease (titles no. 90–95) according to the International List of Causes of Death.

[1] From Office of Heart Disease Investigations.

[2] Hedley, O. F.: Heart disease mortality. Pub. Health Bull. (In press.)

[3] The writer is indebted to Dr. W. C. Fowler, health officer, and to Mr. John H. Milligan, registrar of vital statistics, of the District of Columbia, for permission to examine and abstract these death certificates. Much information used in this survey was obtained from the Annual Report of the Health Officer to the Commissioners of the District of Columbia for the year ending June 30, 1933. The report of the health officer covered the calendar year of 1932.

MORTALITY FROM RECORDED HEART DISEASE AMONG THE GENERAL POPULATION

There were 1,129 deaths recorded as being due to heart disease among the white and 502 among the colored population. The estimated population of the District of Columbia in 1932 was 493,000, of which 357,000 were white and 136,000 were colored. The mortality rate from all causes was 1,616 per 100,000. The death rate among the white population was 1,406, while that among the colored was 2,155 per 100,000 population. The death rate from recorded heart disease was 330 per 100,000. The reported death rate from heart disease among the white population was 316 per 100,000, while that among the colored was 362. The reported death rate due to heart disease is considerably higher than that for the United States registration area and even higher than the reported death rate from heart disease in most cities and states,[4] due most probably to the large Negro population, many adult Government workers, the presence of elderly parents of Government employees who come to Washington to live with their children, the transient nature of certain elements of the population, the large number of retired Government personnel who continue to reside in the District, and the numerous Federal hospitals and institutions.

MORTALITY RATES FROM RECORDED HEART DISEASE IN SPECIFIC AGE GROUPS

In table 1 a study is made of deaths per 100,000 in various age groups. Despite the fact that only a small proportion of deaths from heart disease occurs in persons under 25 years of age, it is still a problem of considerable public health significance. There is a tendency to minimize the importance of heart disease during this period, because, in contrast with other age groups, relatively few deaths occur. This is a mistake. It should be compared with other disease entities, especially those occurring within this age group. There were 52 deaths reported as due to heart disease in this period; nearly as many deaths as due to typhoid fever, paratyphoid fever, typhus, measles, scarlet fever, whooping cough, and diphtheria combined and at all ages. When it is considered that most deaths from congenital heart disease and only slightly over 50 percent of the mortality from rheumatic heart disease are tabulated as heart disease, the importance of heart disease as a cause of death under 25 years of age becomes even more significant.[5]

[4] Whitney, Jessamine S.: Heart disease mortality statistics. (U. S. registration area.) Am. Heart Assoc., 1934.

[5] Hedley, O. F.: A critical analysis of heart disease mortality. Jour. Am. Med. Assoc., 105: 1405 (1935).

TABLE 1.—*Population distribution, number of deaths, and specific death rates per 100,000 from deaths recorded as being due to heart disease, by color and sex, in each age group of the population in Washington, D. C., during 1932—population distribution based on 1930 census*

WHITE

Age group	Male			Female			Total		
	Population	Deaths from heart disease	Rate per 100,000	Population	Deaths from heart disease	Rate per 100,000	Population	Deaths from heart disease	Rate per 100,000
0–14	35,428	8	22	34,848	10	29	70,276	18	25
15–24	28,619	10	35	30,533	5	16	59,152	15	25
25–34	31,929	19	59	33,937	17	50	65,866	36	55
35–44	27,748	43	155	30,448	20	65	58,196	63	108
45–54	21,250	84	395	24,192	29	120	45,442	113	248
55–64	14,559	154	1,058	17,222	85	493	31,781	239	752
65–74	7,134	180	2,523	9,207	134	1,456	16,341	314	1,922
75 and over	2,536	140	5,520	4,035	190	4,650	6,621	330	4,984

COLORED

0–14	14,748	4	27	15,546	2	13	30,294	6	20
15–24	11,229	5	45	13,852	8	57	25,081	13	52
25–34	12,883	16	125	14,466	21	144	27,349	37	135
35–44	10,290	42	408	11,427	41	359	21,717	83	382
45–54	7,680	56	728	8,134	68	835	15,824	124	784
55–64	3,240	50	1,546	3,546	60	1,690	6,786	110	1,621
65–74	1,357	40	2,948	1,708	45	2,635	3,045	85	2,791
75 and over	444	12	2,700	782	30	3,836	1,226	42	3,426

TOTAL, MALE AND FEMALE, WHITE AND COLORED

Age group	Population	Deaths from heart disease	Rate per 100,000	Age group	Population	Deaths from heart disease	Rate per 100,000
0–14	100,640	24	24	45–54	61,306	237	386
15–24	84,193	28	33	55–64	38,567	349	905
25–34	93,215	73	78	65–74	19,406	399	2,060
35–44	79,913	146	183	75 and over	7,847	372	4,731

In the group 35–44 years of age the importance of heart disease becomes felt to a greater extent. It is responsible for deaths at the rate of 155 per 100,000 among white males as compared with 65 per 100,000 among white females. In the colored population it results in 408 deaths per 100,000 among males and 359 deaths per 100,000 among females. The death rate in this period is nearly four times as great among the colored as among the white population and nearly six times greater among colored females than among white females.

In the group 45–54 years of age the death rate is 395 per 100,000 among white males as compared with 120 per 100,000 among white females, or over three times as high. Among the colored males the death rate is 728 per 100,000, while among colored females it is 835. The death rate during this period is three times higher among the

colored than among the white population and nearly seven times higher among colored than among white females.

In the group 55–64 years of age the incidence continues to mount. That among white males is 1,058 per 100,000, while that among white females is 493 per 100,000. Among colored males the rate is 1,546 per 100,000 as compared with 1,690 per 100,000 among colored females. The rate for the colored population is twice that for the white and that among colored females over three times that among white females.

In the 65–74 age period the mortality rate from deaths recorded as heart disease is 2,523 per 100,000 among white males as compared with 1,456 among white females. Among colored males it is 2,948 per 100,000, while among colored females it is 2,635. The death rate in this age group among the colored population is still well above that among the white population. The differences between the sexes of the two races is less marked, although the rate among colored females is still nearly twice that among white females.

Among those surviving 75 years of age, the rate among white males is 5,520 per 100,000, while that among white females is somewhat less, 4,650. Among the colored population the rates are 2,700 and 3,836 among male and female sexes, respectively.

Heart disease increases in importance as a cause of death among each race and sex with each successive decade. This becomes more manifest in adult life, especially after 35 years of age. Between 35 and 64 years of age the incidence of deaths recorded as being due to heart disease is three times greater among white males than among white females. In the colored population the rate is slightly higher among females. This difference is noteworthy. To a certain extent it is probably more apparent than real, owing to cardiovascular syphilis, which results in many cardiac deaths among colored males, only part of which are recorded as heart disease. The greater frequency of hypertensive heart disease among colored females counterbalances this to a certain extent, however. The greater incidence of fatal heart disease among colored than among white women is explained in a large measure by the more sheltered lives of the latter and the greater prevalence of hypertension and syphilis among the former. Syphilitic heart disease is not, however, as prevalent among colored females as might be expected on the basis of studies of cardiovascular syphilis conducted on colored males.[6]

[6] Hedley, O. F.: A study of 450 fatal cases of heart disease occurring in Washington, D. C., hospitals during 1932, with special reference to etiology, race, and sex. Pub. Health Rep., 50: 1127-1153 (Aug. 23, 1935).

AVERAGE AGES AT DEATH

The average ages at death were as follows:

	Number	Age (years)
Total whites	1,129	64.9
Male	638	62.7
Female	491	67.7
Total colored	502	53.5
Male	227	52.4
Female	275	53.3
Total both races	1,631	61.4

In evaluating the significance of heart disease as a public health problem on the basis of the officially recorded statistics, it should be taken into consideration that the average age at death among the white population occurs in the age period 60–69 years, and that among the colored in the 50–59-year age group. Among white females the average age at death is nearly 70 years. DePorte,[7] in New York State, noted that the average age at death was slightly higher. He found that it was 67.6 years—66.7 years and 68.7 years for males and females, respectively. Practically all of his deaths were among white persons. In each racial and sex group the average age at death exceeds the life expectancy for that particuar group and the average age among white deaths recorded as being due to heart disease exceeds that of the general population. There is a distinct difference in ages at death between the two races and a less marked difference between the sexes.

AGE DISTRIBUTION

Not only does the average age but also the distribution of the ages at death vary with race and sex. The maximum number of white male deaths recorded as being due to heart disease occurs in the age period 60–69 years, as shown in figure 1. That among the white females occurs in the age period 70–79 years. The greatest incidence in the colored race occurs somewhat earlier, among the males between 40 and 49 years and among the females between 50 and 59 years. The reason that the peak number of reported cardiac deaths occurs in earlier decades among the colored population is due to the greater prevalence of cardiovascular syphilis and the greater havoc wrought by the degenerative, particularly hypertensive, heart diseases among Negroes.

[7] DePorte, J. V.: Heart disease in general medical practice. Amer. Heart Jour., 8:476, 1933.

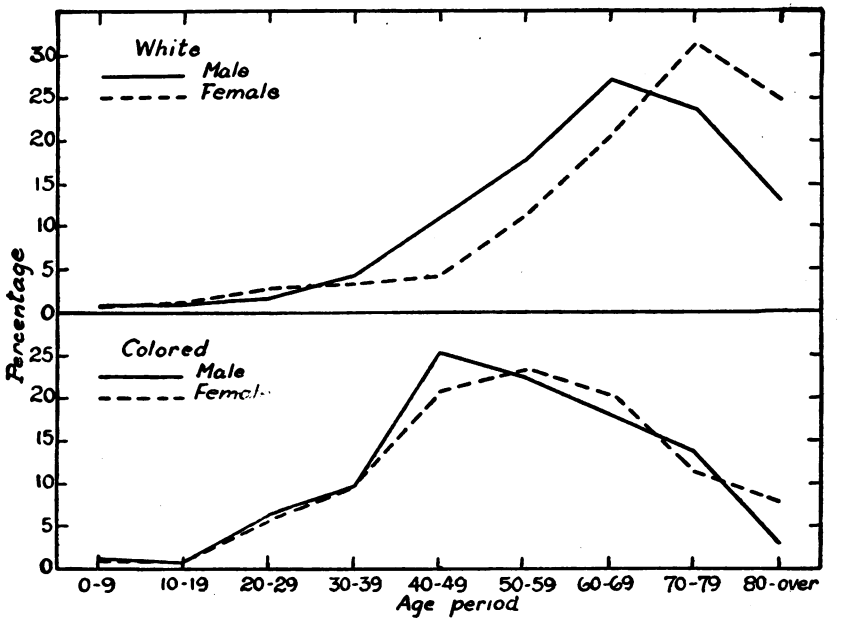

FIGURE 1.—Comparative percentage distribution between white and colored races of 1,631 deaths recorded as due to heart disease in Washington, D. C., 1932.

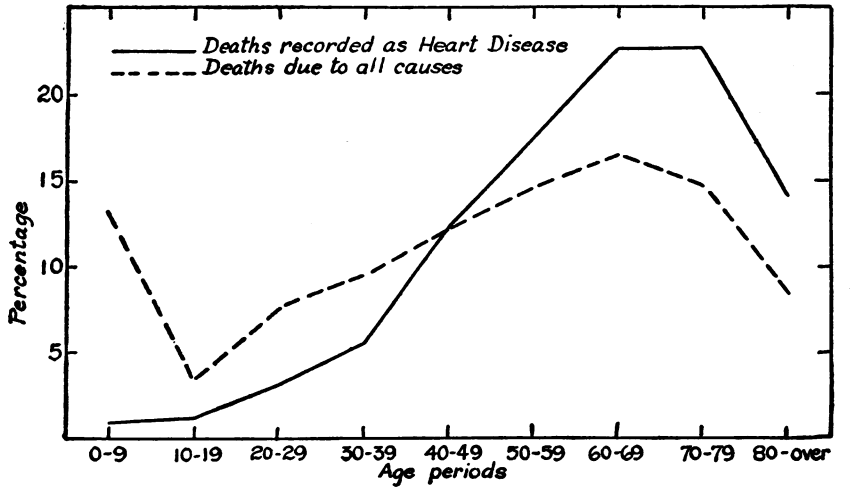

FIGURE 2.—Percentage of deaths in each age group among 1,631 deaths recorded as being due to heart disease as compared with 7,949 deaths due to all causes in Washington, D. C., 1932.

COMPARISON OF THE RECORDED MORTALITY FROM HEART DISEASE WITH THE GENERAL MORTALITY

A comparison between the age distribution of deaths recorded as being due to heart disease and that of deaths due to all causes, as shown in figure 2, indicates that heart disease plays a relatively unimportant role as a cause of death during the first decade. During the next 3 decades it is responsible for an increasingly higher proportion of deaths, but lags behind the general mortality. Deaths from heart disease are far more heavily distributed among the age brackets above 40 years than are deaths from all causes. The diver-

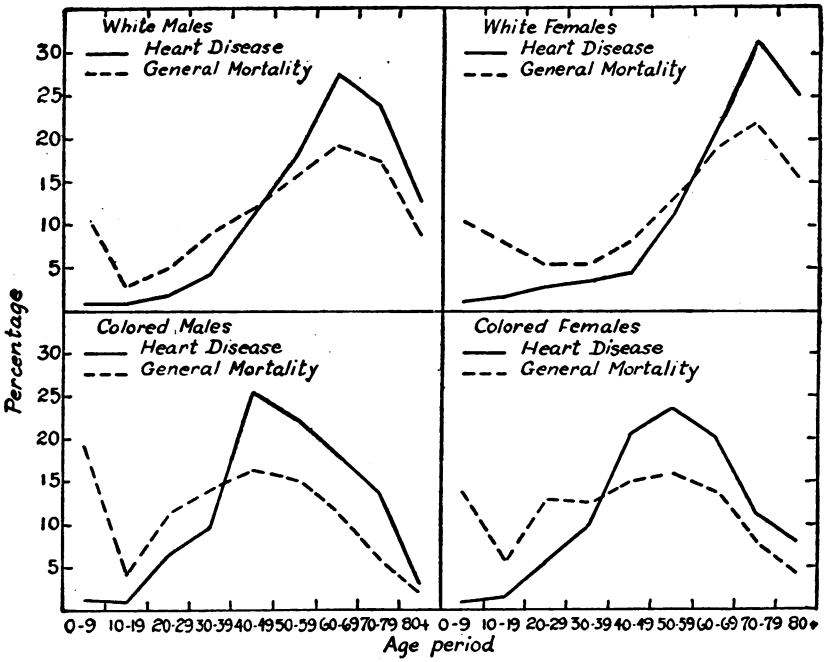

FIGURE 3.—Percentage of deaths in specific age groups among 1,631 deaths recorded as being due to heart disease as compared with 7,949 deaths due to all causes, by color and sex, in Washington, D. C., 1932.

gence of the two curves before and after the age group 40–49 years would probably be much less marked if it were not for the fact that the present system of recording tends to eliminate from the heart-disease curve many deaths of congenital, syphilitic, thyrotoxic, and rheumatic etiology.

Additional information is elicited by comparing the distribution of deaths from heart disease in each race and sex with the general mortality. (See figure 3.) In the age period between 10 and 29 years heart disease results in a relatively higher proportion of deaths among the white population than among the colored, due to the lower incidence of deaths due to infectious diseases, especially tuberculosis.

The peak incidence of deaths due to heart disease and that of deaths in general occurs in identical decades in each race and sex group, and the distribution of deaths in ages beyond these points tends to run parallel. There is a sharp increase in the percentage of deaths recorded as being due to heart disease among white males beginning with the age period 40–49 years, while that among white females does not increase until a decade later. In both of the colored groups there is a considerable increase in mortality from heart disease in the 30–39-year age period, with an almost precipitous increment in the 40–49-year bracket. There are two factors to account for this; namely, the increase in deaths due to hypertensive heart disease and to cardiovascular syphilis, the latter being not infrequently reported as valvular lesions or as myocarditis, and as such finding expression in the mortality statistics as being due to heart disease.

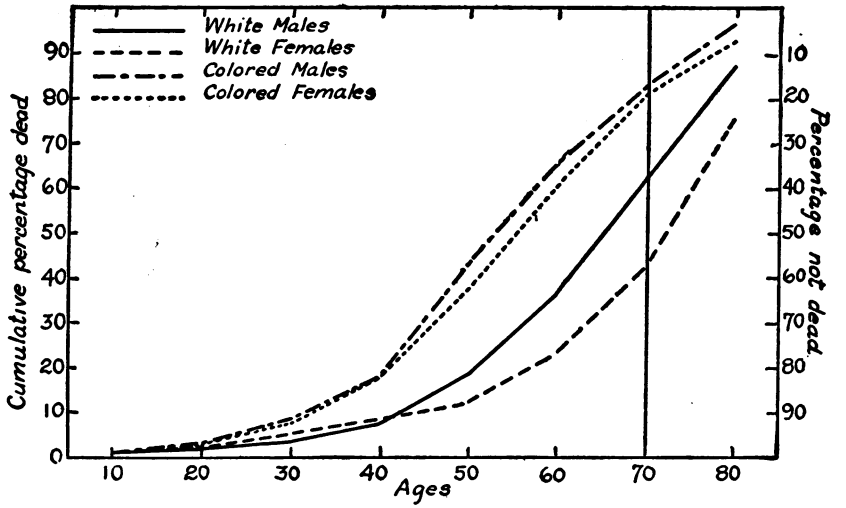

FIGURE 4.—Cumulative percentage, by color and sex, of 1,631 deaths recorded as being due to heart disease either prior to or after various ages, with special reference to those surviving 70 years of age, in Washington, D. C., 1932.

A comparatively small proportion of the Negro population survives 60 years of age. This is clearly seen by studying both the curves of deaths due to all causes and those recorded as being due to heart disease. Britten [8] has shown that the mortality rate from all causes and the rate for a number of conditions, including heart disease, are higher among wage earners in the United States than in England. Since American Negroes are for the most part in the occupational class of unskilled laborers (including domestics), this may account for the increased and earlier mortality from heart disease among this group. Certainly it cannot be said categorically that there is any

[8] Britten, Rollo H.: Mortality rates by occupational class in the United States. Pub. Health Rep., 49: 1101-1111 (Sept. 21, 1934).

racial predisposition among the Negroes to cardiovascular deterioration.

TOTAL RECORDED MORTALITY FROM HEART DISEASE OCCURRING PRIOR TO VARIOUS AGES

In figure 4 are shown by means of a cumulative curve the total percentages of deaths reported as being due to heart disease both prior to and after various ages. Less than 10 percent of the deaths recorded as being due to heart disease occur before the age of 30 years. Racial differences become more pronounced after that period. By 40 years of age nearly 20 percent of the total deaths recorded as due to heart disease will have occurred in Negroes, while among the white population less than 10 percent will have occurred. Colored males die the earliest, while colored females follow with only a slightly delayed rate. Deaths from recorded heart disease occur considerably earlier among the colored than among the white and among white males as compared with white females.

Of the total recorded cardiac deaths, 37.2 percent occur at the age of 70 years or older. Among white males 36.3 percent die during this well-advanced period, while among white females 56.1 of those listed as having died of heart disease survive 70 years of age. The Negroes are not so fortunate. Only 16.4 percent of the total deaths recorded as being due to heart disease among Negro males occur in the seventies or older, while only 18.9 percent of colored females to whom heart disease is attributed as the cause of death survive three score and ten years.

That such a large proportion of these deaths occurs during these advanced ages cannot be attributed to any peculiarity of the population distribution of the District of Columbia. In Chicago, Ill., in 1930, 30.8 percent of the deaths recorded as due to heart disease occurred at 70 years or older,[9] while in Virginia, during 1932, 41.6 percent occurred during this period.[10]

It is quite significant that approximately a third of the deaths reported as heart disease occur in individuals of 70 years or older. The question arises to what extent these deaths are really due to cardiac affections. Frequently heart disease is only part of a general clinical picture due to conditions either produced by or associated with degenerative changes. Generalized arteriosclerosis and arteriolosclerosis, diabetes, prostatic hypertrophy, senile encephalopathies, and other afflictions attending advancing years not infrequently occur concomitantly with heart disease and with each other. In a way these deaths are due to heart disease by default, in that infections,

[9] Report of Board of Health, Chicago, Ill. 1930.

[10] Report of the Bureau of Vital Statistics for the year ended Dec. 31, 1932. Virginia Health Bulletin, vol. 26, no. 4, April 1934.

trauma, and neoplasms have not resulted in death at an earlier period. One cannot escape the impression after extensive perusal of death records that many old people are recorded as having died of heart disease principally, if not solely, on the grounds that they died when their hearts stopped beating.

It is doubtful, however, that any system of reporting and recording deaths could be devised which would eliminate this effect. Certainly there is nothing to be gained by reporting these deaths as due to senility. The trouble lies not in considering such deaths as due to heart disease but in assuming that, because there has been an increase in heart disease among the older age periods, this holds for all types of heart disease. If there were less emphasis on heart disease in general and more importance attached to the various etiological components, much of the present-day hysteria on the subject would be greatly reduced, and it might be possible to formulate plans for attacking certain aspects of this problem.

An understanding of the biological principles attending old age is necessary for a proper interpretation of heart disease occurring during this period. Death due to circulatory failure in such cases is but the clinical reflection of wide-spread senile changes. As there is little likelihood of adding to the span of human life, while on the other hand life expectancy at birth has greatly increased, a crowding of the population into the older age brackets has resulted. Cohn[11] has invited attention to the fact that the increase in heart-disease mortality did not occur simultaneously with the beginning of the decline in deaths from infectious diseases, but about 17 years later. Those who would have died of infectious diseases lived until the effects of senescence became manifest. It is felt that this will continue, and with it there will be a greater incidence of heart disease of a sort. It is the result of benign rather than malevolent influences and should be considered in the light of a victory for medical science.

Percentage of total mortality recorded as heart disease

Group	Total deaths	Deaths recorded as due to heart disease	Percent of deaths recorded as due to heart disease
Total whites	5,018	1,129	22.4
Male	2,770	638	22.7
Female	2,248	491	21.8
Total colored	2,931	502	17.1
Male	1,528	227	14.2
Female	1,403	275	19.6
Total both races	7,949	1,631	20.5

[11] Cohn, Alfred E.: Heart disease from the point of view of the public health. Am. Heart Jour., 2: 275, 386 (1927).

The lower percentage of deaths due to heart disease among the colored population is likely to be misleading. The reason that a relatively smaller proportion of the total mortality is due to cardiac affections is that there is a greater number of deaths from infections, particularly tuberculosis, among Negroes during the earlier decades. As a result, relatively fewer survive to die subsequently of heart disease. With the prevention of those factors causing deaths at earlier ages it is expected that a higher proportion of deaths among colored people will result from diseases of the heart. It has been remarked rather cynically that medical science saves people from dying of tuberculosis to have them succumb later to cancer. This appears even more poignantly true of heart disease.

Another factor resulting in a lower percentage of the total deaths being listed as heart disease (titles nos. 90–95 of the International List of Causes of Death) among colored than among white deaths is that syphilitic heart disease when reported as such is not recorded as a cardiopathy but as title no. 34, syphilis, or as title no. 96, aneurysms. Furthermore, it appears that in certifying deaths due to cardiovascular syphilis more attention may be given to the venereal disease aspects when occurring in Negroes than when occurring in white persons.

The extent to which heart disease participates in the total mortality in each decade is shown in figure 5. In general, heart disease accounts for a greater percentage of the total mortality in each succeeding decade until over 35 percent of deaths from all causes are recorded as being due to that cause. It is a mistake, however, to assume that heart disease is a relatively unimportant mortality factor in youth and early adult life. In spite of the limitations imposed by the International List of Causes of Death, heart disease is accredited with 8 percent of the total deaths during the period between 10 and 29 years.

The relative incidence by sex and color is given in figure 5. Among white females there is a rather considerably higher proportion of deaths recorded as being due to heart disease between 10 and 39 years of age than among white males during these periods. This is probably due to the higher incidence of rheumatic heart disease among white females, and possibly to childbearing. During the succeeding periods between 40 and 69 years the extent to which heart disease results in the general mortality among white males becomes more manifest. In the very advanced periods the percentage of deaths listed as heart disease tends to become approximately similar in the two sexes. Heart disease among colored females exceeds that among males as a recorded cause of death in nearly every age period, especially in the brackets between 40 and 69 years of age. As compared with the white population, heart disease results in about twice the

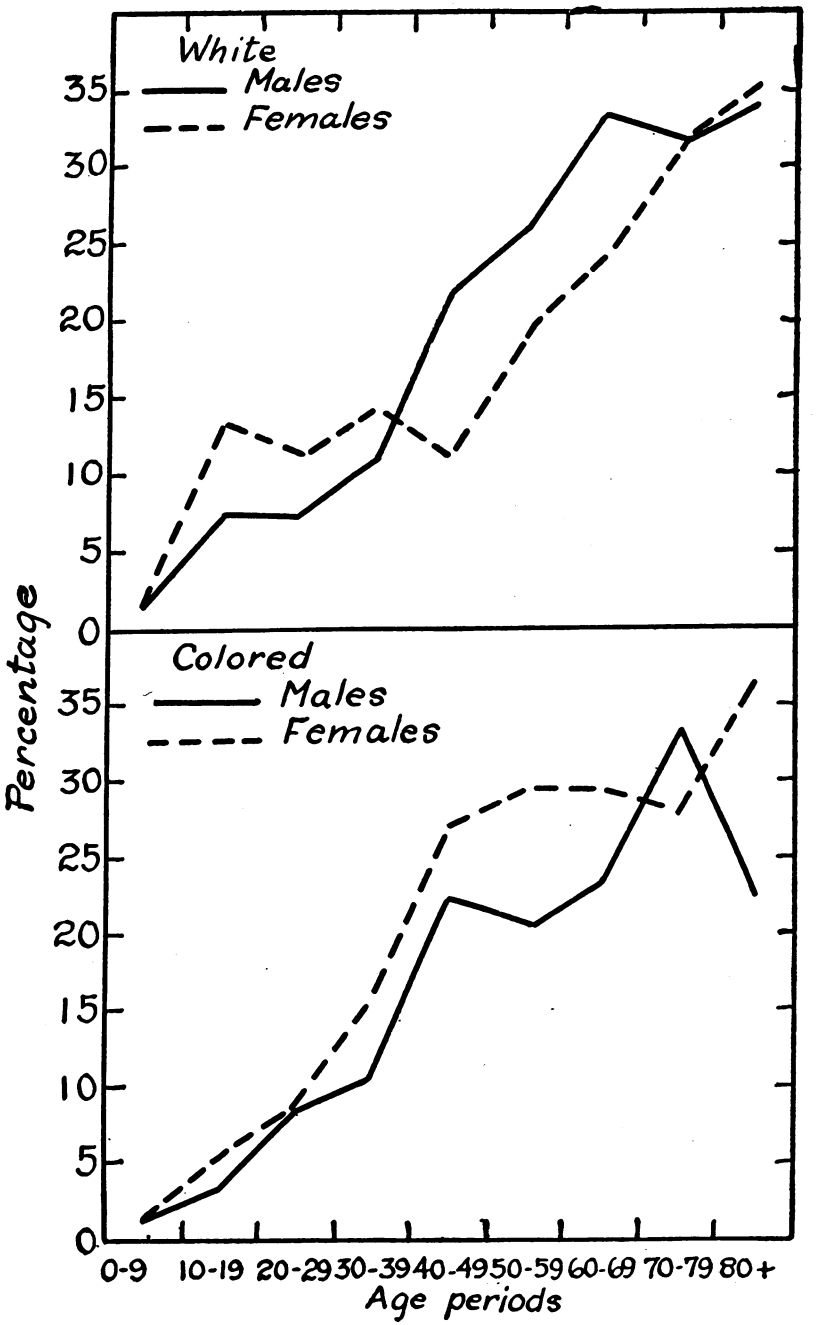

FIGURE 5.—Percentage of total deaths in each age period, by color and sex, recorded as being due to heart disease, in Washington, D. C., 1932.

percentage of total deaths in the colored population between 40 and 49 years of age as among the white population during that period. This has been noted by Dublin,[12] using the experiences of the industrial department of the Metropolitan Life Insurance Co. Dublin also noted that, in contrast with white females, the rate among colored females was as high as, if not higher than, that of colored males.

SEASONAL VARIATIONS IN MORTALITY

The incidence of deaths recorded as being due to heart disease is increased during the colder months and reduced during the warmer months, as shown in figure 6. There is a striking rise in the incidence of deaths from heart disease with the onset of cold weather, with an even greater secondary rise toward the end of winter and in the

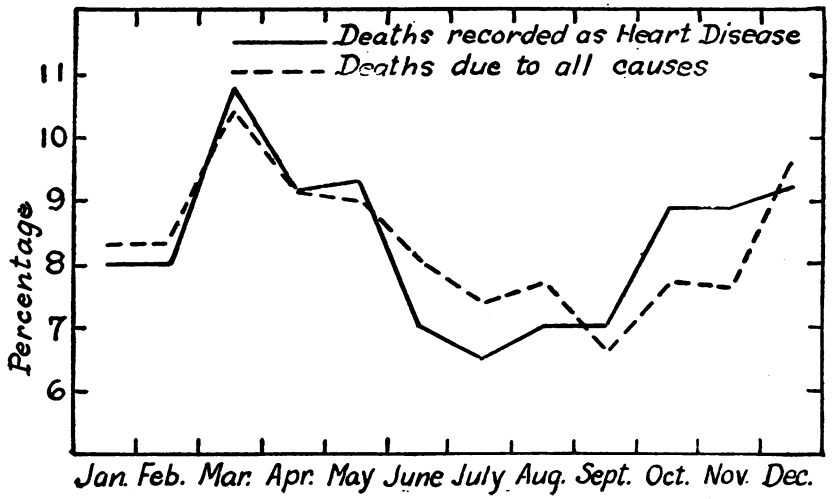

FIGURE 6.—Distribution, by months, of 1,631 deaths recorded as being due to heart disease and occurring in Washington, D. C., during 1932, as compared with 7,949 deaths due to all causes.

spring months. This approximates the seasonal distribution of the general mortality. Cardiac patients withstand the effects of respiratory infections very poorly. Conditions resulting only in temporary discomfort to individuals without circulatory impairments are frequently fatal to those suffering from heart disease, especially those in incipient stages of congestive heart failure.

SUMMARY AND CONCLUSIONS

An analysis has been made of deaths recorded as being due to heart disease in Washington, D. C., during 1932. The limitations of such a study are obvious, being due to vagaries in the reporting of deaths and to difficulties inherent in the present system of collecting and

[12] Dublin, L. I.: Statistical aspects of problems of organic heart disease. Amer. Heart Jour., 1: 359 (1926).

tabulating mortality statistics. The study is based, however, on the official records of what constitutes heart-disease mortality and as such should be given consideration.

Heart disease becomes of greater significance as a cause of death with each succeeding decade. It is of greater significance among white males than among white females and in the colored than in the white race. These differences are more pronounced in the periods between 35 and 64 years of age. The most marked differences in mortality in specific age groups occur in colored females as compared with white females. It is believed that the comparisons of the mortality incidence are reasonably accurate in that the same sources of error operate in each race and sex.

Over a third of the deaths recorded as heart disease occur at the age of 70 years or older. Among white females over 50 percent occur at 70 or older, while among white males about a third of the total recorded deaths from heart disease are in this period. Less than a fifth of the colored persons recorded as dying of heart disease survive three score and ten years.

Deaths in advanced years should not be considered in the same light with those due to infections or metabolic forms of heart disease, or even with certain of the arteriosclerotic and hypertensive forms occurring earlier, such as coronary arteriosclerosis and essential hypertension in middle age.

While only a small proportion of the aggregate number of deaths from heart disease occurs under 25 years of age, it is nevertheless a public health problem of considerable importance when compared with deaths from other causes, especially those occurring within this age period. It should be borne in mind that most of the deaths from heart disease under 25 years of age are due to the cardiac manifestations of rheumatic fever, only a part of which are tabulated as heart disease, the remainder being recorded as due to rheumatic fever.

DISTRIBUTION OF LYMPHOCYTIC CHORIOMENINGITIS VIRUS IN THE ORGANS OF EXPERIMENTALLY INOCULATED MONKEYS

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The presence of choriomeningitis virus in the brain, blood, spinal fluid, and urine of experimentally infected monkeys has been reported (1). The work here recorded constitutes an effort to determine further the distribution of the virus in the organs of infected animals (*Macacus rhesus*).

EXPERIMENT I

Monkey no. 754 was inoculated intravenously on December 19, 1934, with 6 cc of supernatant fluid from a 1:20 suspension of mouse brain virus in saline, which had been allowed to settle for 15 minutes.

The animal developed fever on December 24 and ran a typical course of severe choriomeningitis, death occurring on January 1, 1935. The autopsy revealed slight engorgement of the surface vessels of the brain. The superficial lymph nodes were enlarged and congested, and the abdominal and pleural cavities contained a small amount of clear, yellowish fluid. The lungs were mottled and covered by a slight amount of fibrinous exudate, the heart was flabby, the liver was pale yellow in color, and the spleen was soft, red, and slightly enlarged. The adrenals were large and the kidneys pale. Other organs appeared normal.

Portions of various organs (table 1) removed with sterile precautions were stored in 50 percent glycerol at +4° to +6° C. Samples of blood and pericardial fluid were similarly stored, but without glycerol. On the following day, January 2, 1935, samples of each organ were drained of excess glycerol, weighed, ground in a mortar (without sand), and 1:20, 1:100, 1:500, and 1:2500 suspensions made in saline. Each suspension was allowed to settle for a few minutes, and 0.03 cc of the supernatant fluid, after being pipetted off, was immediately inoculated intracerebrally into each of four mice. The mice were observed for 15 days and deaths recorded.

The symptoms in mice are quite characteristic, but may be of short duration and are easily missed. Mice dying from an intracerebral inoculation of the virus, however, quite regularly die in tetanic convulsions with the hind legs fully extended. This position maintained after death offers considerable aid in arriving at a diagnosis where symptons have been missed. As a further check on the diagnosis, brains from random samples of dead mice were submitted to Surgeon R. D. Lillie for pathological confirmation. The results checked closely with conclusions drawn from symptoms and position at death and indicated that deaths occurring from the fifth to twelfth days, inclusive, following inoculation, were so regularly due to the virus that, for practical purposes, the few exceptions may be ignored. However, the few deaths occurring before and after this interval were found usually to be due to other causes.

By reference to table 1, it may be noted that virus was present in all the organs and fluids tested, in many instances in sufficient amounts to bring about the death of all 4 mice in the highest dilution employed.

Since the dosage employed was uniformly 0.03 cc, it is possible to make a rough estimate of the number of minimal fatal intracerebral

mouse doses of virus in a gram of each organ or fluid tested. The results are shown in table 1.

TABLE 1.—*Determination of virus content in organs of monkey no. 754*

Tissues examined	Day of death for mice inoculated with—				Culture results 1:20 suspension	Estimated number of minimal fatal doses of virus for mice in 1 gram of tissue (0.03 cc intracerebrally)
	1:20 suspension	1:100 suspension	1:500 suspension	1:2500 suspension		
Abdominal fluid	2, 4, 7, 9	6, 7, 7, 7	6, 7, 10, [1] S	2, 7, S, S	Sterile	12,000
Adrenal	2, 7, S, S	5, 6, 8, 10	7, 7, 7, 7	2, 4, 6, 7	Growth, base slant	82,000+
Axillary gland	7, 7, 7, 7	7, 7, 7, 7	8, 8, 8, 9	6, 7, 7, 8	Few colonies	82,000+
Blood, heart, defibrinated.	2, 4, S, S	4, 7, 7, 12	2, 2, 7, 8	8, 8, 8, 8	Sterile	82,000+
Cerebrum	7, 8, 10, S	1, 7, 7, 7	7, 10, S, S	8, 10, S, S	do	41,000
Kidney	7, 7, 7, S	7, 7, 8, 8	2, 8, 8, 8	8, 8, 12, S	do	62,000
Liver	4, 7, 8, S	7, 7, 7, 9	6, 7, 7, 10	7, 12, S, S	do	41,000
Lungs	7, 7, 7, 8	1, 7, 7, 8	6, 7, 8, 8	7, 8, 8, 8	Growth, base slant	82,000+
Marrow, red, femur	1, 1, 7, 7	5, 7, 8, S	6, 7, 7, S	7, 7, 8, 12	Sterile	82,000+
Muscle, voluntary	2, 8, 9, 10	7, 7, 8, 8	1, 2, 8, 8	5, 8, 9, 10	do	82,000+
Pancreas	4, 7, 7, 7	6, 9, 11, 14	7, 7, 7, 7	7, 8, 11, S	Growth, base slant	62,000
Pericardial fluid	7, 10, 13, S	4, 7, 7, 7	7, 7, 7, 7	2, 7, 7, 14	Sterile	82,000+
Spleen	4, 7, 7, 13	1, 7, 9, S	5, 8, 8, 11	6, 8, 8, S	do	62,000
Salivary gland	6, 7, 7, 7	6, 7, 7, 8	5, 7, 7, 8	7, 9, 9, 10	do	82,000+

[1] S=Survived.

EXPERIMENT II

Since the route of inoculation conceivably might influence the distribution of the virus, it was deemed desirable to test the virus distribution in animals inoculated by other than the intravenous route. Consequently, the organs of monkey no. 30, which died 13 days following a combined intracerebral and intraperitoneal inoculation given for another purpose, were tested for virus.

At autopsy, the brain was congested, the superficial glands were reddened and enlarged, the right lung showed a small area of consolidation in the lower lobe, and there was free fluid in the right pleural cavity. The left lung and other organs appeared normal.

Tissues taken with sterile precautions were handled as in experiment I, except that less concentrated suspensions were employed; namely, 1:1000, 1:5000, 1:25000, and 1:125000. The tissues examined and the results, which indicate a wide distribution of the virus, are shown in table 2.

TABLE 2.—*Determination of virus content in organs of monkey no. 30*

Tissues examined	Day of death for mice inoculated with—				Culture results 1:1000 suspension	Estimated number of minimal fatal doses of virus for mice in 1 gram of tissue (0.03 cc intracerebrally)
	1:1000 suspension	1:5000 suspension	1:25000 suspension	1:125000 suspension		
Adrenal	8, 10, [1] S, S	3, 5, 8, 8	8, 8, S, S	8, 8, S, S	Sterile	412,000
Axillary gland	5, 7, 7, 7	9, 9, 10, S	S, S, S, S	S, S, S, S	do	124,000
Bile	S, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	do	(?)
Blood, defibrinated	4, 7, 8, S	8, S, S, S	11, S, S, S	S, S, S, S	do	22,000
Cerebrum	7, 8, S, S	4, 9, 10, S	S, S, S, S	9, 4, S, S	do	110,000
Kidney	3, 3, 8, 9	1, 11, S, S	S, S, S, S	S, S, S, S	do	33,000
Liver	8, 9, 9, 10	8, 8, 9, S	7, S, S, S	11, S, S, S	do	124,000
Lungs	8, 8, 8, 9	9, 9, 9, S	S, S, S, S	S, S, S, S	2 colonies, slant	124,000
Marrow	8, 8, 9, 13	6, 7, S, S	8, 10, 13, S	S, S, S, S	Sterile	82,000
Muscle, heart	8, 8, 9, 10	8, 11, S, S	2, S, S, S	S, S, S, S	3 colonies	82,000
Muscle, voluntary	8, 9, 9, 10	2, S, S, S	S, S, S, S	S, S, S, S	Sterile	33,000
Pancreas	7, 8, 8, 9	8, 8, 8, 10	8, 11, S, S	10, S, S, S	do	412,000
Pleural fluid	3, 3, 3, 3	3, 3, 3, 3	2, 3, 4, 4	3, 5, 11, S	Many colonies	(?)
Salivary gland	9, 9, 10, S	4, S, S, S	S, S, S, S	S, S, S, S	Sterile	29,000
Spinal cord	8, 9, S, S	S, S, S, S	9, S, S, S	3, S, S, S	Growth, base slant	16,000
Testicle	9, 9, 9, 9	14, S, S, S	S, S, S, S	12, S, S, S	Sterile	33,000

[1] S=Survived. [2] None apparent.

EXPERIMENT III

Since monkeys no. 754 and no. 30 both had died of choriomeningitis, it was deemed desirable to test the organs of an animal sacrificed during the acute attack. Therefore, monkey no. 41 was inoculated subcutaneously with 6 cc of a 10 percent suspension of mouse brain virus on October 9, 1935. The animal developed symptoms of choriomeningitis and was etherized on October 18, 9 days after inoculation and on the fifth day of fever.

While the amount of virus in many organs of monkeys no. 754 and no. 30 appeared to be greater than could be accounted for by the presence of virus-containing blood in the vessels (tables 1 and 2), nevertheless it seemed desirable to remove the blood from monkey no. 41 insofar as was possible. The animal was etherized and bled from the heart, after which a cannula was inserted into the femoral vein of the left leg. The femoral artery of the right leg then was severed, and transfusion with saline was given until the heart ceased to beat and thereafter by manual manipulation of the chest until the effluent was clear.

At autopsy the organs appeared essentially normal, except for marked pallor.

Dilutions of spinal fluid and defibrinated blood were inoculated at once intracerebrally into white mice. Other tissues were stored in glycerol until the following day, when organ suspensions in saline were made and inoculated as in experiments I and II. Saline suspensions were employed in dilutions of 1:100, 1:1000, 1:2000, 1:4000, 1:8000, and 1:16000. The inoculation results, as recorded in table 3, indicate further that the virus is distributed widely throughout the organs of the infected animal, even after the tissues have been freed largely of blood by washing out with saline.

TABLE 3.—*Determination of virus content in organs of monkey No. 41*

Tissues examined	Day of death for mice inoculated with—								Culture results 1:100 suspension	Estimated number of minimal fatal doses of virus for mice in 1 gram of tissue (0.03 cc intracerebrally)
	Not diluted	1:10 suspension	1:100 suspension	1:1000 suspension	1:2000 suspension	1:4000 suspension	1:8000 suspension	1:16000 suspension		
Adrenals			6, 6, 7, 7	4, 7, 8, 8	5, 8, [1] S, S	4, 8, 8, 8	3, 8, S, S	8, 9, 10, S	Growth, base tube	132,000
Axillary gland			7, 7, 8, 8	7, 8, 8, 13	7, 7, 10	7, 7, 8, 8	8, 9, S, S	6, 8, 9, 13	Sterile	132,000
Bile			S, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	do	([2])
Blood, defibrinated		7, 7, 7, 8			S, S, S, S	S, S, S, S	11, 14, 14	4, 14, S, S	do	330+
Cerebrum			1, 4, 10, S	S, S, S, S	9, S, S, S	1, 1, S, S	S, S, S, S	S, S, S, S	do	1,600
Kidney			7, 8, 8, 8	8, 8, 10, 10	8, S, S, S	8, 9, 9, S	2, S, S, S	11, S, S, S	Growth, base slant	33,000
Liver			6, 6, 7, 11	6, 6, 7, 7	8, 9, 9, S	7, 7, 8, 8	9, 9, 9, S	6, 7, 8, 10	Sterile	528,000+
Lungs			6, 7, S, S	8, S, S, S	3, 9, 10, 11	9, 11, S, S	2, 9, 11, S	10, S, S, S	Growth, base slant	1,600
Marrow, femur			7, 7, 8, 10	2, 8, S, S	1, 1, 13, S	8, 9, 13, 14	10, 10, S, S	7, S, S, S	6 colonies on slant	11,000
Muscle, heart			8, 9, 10, S	9, S, S, S	7, 7, S, S	1, 10, S, S	S, S, S, S	1, S, S, S	1 colony	2,500
Muscle, voluntary			6, 10, S, S	S, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	2, 4, S, S	4 colonies	1,600
Pancreas			6, 8, S, S	10, S, S, S	2, S, S, S	10, S, S, S	4, S, S, S	11, 14, S, S	Sterile	1,600
Salivary gland			7, 14, S, S	S, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	S, S, S, S	1 colony	1,600
Spinal cord			10, 10, 10, S	1, 1, 9, 13	1, 10, 13, S	13, S, S, S	9, S, S, S	S, S, S, S	Sterile	2,500
Spinal fluid	5, 7, 8, 10				S, S, S, S	S, S, S, S	S, S, S, S	3, S, S, S	do	33+
Spleen			7, 7, 7, 8	7, 7, 7, 8	1, 8, 8, 13	7, 7, 8, 9	7, 10, 11, S	1, 7, 8, 8	do	528,000+
Testicle			8, 8, 9, 11	9, 9, 9, S	8, 8, 11, S	S, S, S, S	2, 8, 10, S	S, S, S, S	Growth, base slant	50,000

[1] S=Survived.

[2] None apparent.

The presence of foci of lymphocytic infiltration in the organs of these (monkeys 754, 30, and 41) and of other choriomeningitis-infected monkeys during the acute stages of the disease, as well as the occasional occurrence of actual foci of necrosis in certain organs, as the liver, adrenals and parathyroids, as observed by Surgeon R. D. Lillie, furnishes confirmatory evidence of the wide distribution of the virus.

For a further consideration of the histopathology the reader is referred to the detailed pathological report by Surgeon R. D. Lillie, (2), based on the examination of tissues from 51 monkeys. (The article immediately following.—Ed.)

SUMMARY

Monkeys experimentally infected with the virus of choriomeningitis were shown, by mouse inoculation, to harbor the virus in large amounts in all the organs and tissues tested; namely: adrenals, cerebrum, blood, kidneys, liver, lungs, lymph glands, marrow, heart muscle, voluntary muscle, pancreas, spinal cord, spinal fluid, spleen, submaxillary glands, and testicle. This distribution of virus is not explainable by the presence of virus-containing blood in the vessels.

REFERENCES

(1) Armstrong, C., and Lillie, R. D.: Experimental lymphocytic choriomeningitis of monkeys and mice produced by a virus encountered in studies of the 1933 St. Louis encephalitis epidemic. Pub. Health Rept., **49**; 1019–1027 (1934).

(2) Lillie, R. D.: Pathologic histology of lymphocytic choriomeningitis in monkeys. Pub. Health Rept., this issue (following article).

PATHOLOGIC HISTOLOGY OF LYMPHOCYTIC CHORIOMENINGITIS IN MONKEYS

By R. D. Lillie, *Surgeon, United States Public Health Service*

The purpose of the present communication is to amplify the findings on the brain previously reported by Armstrong and Lillie, 1934 (1) and to report the lesions produced in the other viscera.

The material was derived from 51 monkeys used in Armstrong's experimental studies. In a considerable number of these only the brain was available, but in about half of the animals fairly complete material was studied histologically. Tissues were fixed routinely in Orth's bichromate formalin, imbedded and sectioned in paraffin, and stained routinely with our Romanowsky technique and with iron hematoxylin and picrofuchsin.

Forty-two monkeys died or were killed *in extremis* at intervals of 6 to 17 days after inoculation, the mean survival period being 11 days.

Nine monkeys survived for longer periods, 27 days up to 1 year, and were studied principally for the demonstration of the persistence of lesions over prolonged periods.

Survival periods

Days	6	7	8	9	10	11	12	13	14	15	16	17	27	32	34	40	55	81	7 months	1 year
Number of monkeys	2	2	1	5	7	4	6	4	2	3	1	3	1	1	1	1	1	1	2	1

The *brain* was studied histologically in 51 monkeys, the *spinal cord* in 35, and spinal *root ganglia* in 7. The *meninges* showed a slight to moderate focal to diffuse lymphocyte infiltration occurring generally, not especially more marked in sulci and fissures. It was somewhat more marked over the frontal cortex and cerebellum, and was slight on the spinal cord. In a few animals, plasma cells and macrophages were present as well. Congestion was common, and hemorrhage was frequent. The last finding was discounted considerably, as the brains were usually removed within a few minutes after death while the blood was still unclotted, and, hence, much of the hemorrhage was considered artefact. Cellular infiltration of the meninges was present in 39 of the 42 animals dying in less than 18 days, while of the 9 monkeys surviving for prolonged periods, 4 showed absence of meningeal cellular exudation. The latest survivor showing focal meningeal lymphocyte infiltration died 81 days after inoculation.

The *plexus chorioidei* almost constantly showed at least a focal lymphocyte infiltration. More often the plexus was swollen and densely and diffusely infiltrated by lymphocytes. In the 2 animals dying in the early period and not showing plexal infiltration, plexus of only one ventricle was present in the material, and infiltration may well have been present in other areas. Congestion, serous exudation, and participation of plasma cells and macrophages in the plexal infiltration occurred in some of the earlier cases. Serous and serocellular exudates in the ventricles were seen in 11 monkeys. Lymphocytes, red corpuscles, and macrophages were the usual cellular constituents, and polymorphonuclears were found only in one case.

The infiltration of the plexus chorioidei was not uniform. It was perhaps dense in one plexus, or part of one, and scanty or absent in other areas. Commonly, it was focal and sparser in the later survivors, perhaps limited to the base of the plexus. A slight focal infiltration was still present in the animal surviving 1 year after inoculation.

Subependymal edema and focal lymphocyte infiltration were present in a few animals. Ependymal desquamation, both from the walls and from the plexal villi, were noted in a number of animals.

In the *brain* substance there were only occasional small vessels mantled by lymphocytes and a few small nodes of glia cells. In one-third of the animals none was found, and in many only 3 or 4 focal lesions could be demonstrated in 12 to 20 sections of the brain.

The *spinal cord* was normal in 18 of 35 monkeys. Congestion and focal hemorrhages in the gray substance were found in 7, and a few focal glioses or focal perivascular lymphocyte infiltrations in 10 monkeys.

The *spinal root ganglia* regularly showed slight to moderate sheath cell proliferation, focal lymphocyte infiltration, or both. The ganglion cells were normal.

The *heart* was studied histologically in 23 monkeys. In 10 the muscle was normal, in 3 there were slight granularity and indistinctness of the cross striations, and in 10 there was a slight to moderate focal lymphocyte infiltration in the myocardium or occasionally epi- or endocardium. One monkey showed a few sarcosporidia in the muscle fibers.

In 8 of 21 monkeys the *larynx* and *trachea* showed a slight to moderate diffuse or focal lymphocyte infiltration of the mucosa. In the remainder no significant lesions were seen.

In 9 of 22 monkeys the *lung* was normal. In two there was a nodular, purulent pneumonia, with congestion, edema, and fibrinopurulent pleurisy. Two monkeys presented a parasitic bronchiectasis and bronchitis. In five there were patchy to diffuse congestion, serous alveolar exudation, and nodular paravascular or perivascular lymphocyte infiltration. Two of these showed also a marked perivascular edema, and one of them a nodular hemorrhagic consolidation. In two other animals patchy and diffuse congestion and serous alveolar exudation, respectively, were seen, in the latter with perivascular edema and focal alveolar hemorrhages. The two remaining monkeys showed focal perivascular lymphocyte infiltration, alone in one and with alveolar hemorrhages in the other.

It appears that the congestion, serous exudation, edema, and perivascular lymphocyte infiltration of greater or less extent form the essential picture of the pulmonary reaction to the virus of lymphocytic choriomeningitis.

The *submaxillary gland* was examined in 17 animals. In 15 it was normal. In two there were several hyperplastic lymphoid follicles, and in one of these there was a concurrent focal interstitial lymphocyte infiltration.

The *esophagus* was normal in 14 monkeys and in 1 showed a slight focal lymphocyte infiltration of the mucosa.

The *gastric* mucosa was regularly more or less infiltrated by lymphocytes. The infiltration was slight in two, moderate in five, "patchy" in four, "superficial" in one, "normal" in two, and marked in four.

In one of the last group appreciable numbers of plasma cells were present as well. A catarrhal exudate was present in one monkey, and a patchy lymphocyte infiltration of the serosa in another.

Reticulum cell hyperplasia in lymph follicles was noted in the *small intestine* in 2 monkeys (13 and 15 days), slight edema of the mucosa in 2, and in the remaining 12 it was considered normal.

In 11 of 17 monkeys the *large intestine* was normal. Two showed catarrhal or mucopurulent colitis and parasitic abscesses (nematodes), with local peritonitis in the serosa. Two showed a simple catarrhal colitis. Two showed ulcerative colitis, acute necrotizing and subacute fibrosing, respectively. None of these changes appears significant in relation to lymphocytic chorimeningitis.

The *pancreas* was examined in 22 monkeys. It was normal in 17, acute intercurrent processes of dubious significance were present in 3, and in 2 there was an irregular periductal or interstitial lymphocyte infiltration.

The *liver* was examined histologically in 24 monkeys; in 3 it was normal. In 10 there was slight to marked fatty infiltration, without other changes in 5, with congestion in 1, with periportal lymphocyte infiltration in 2, with both in 1, and with midzonal foci of lymphocyte infiltration and Kupffer cell hemosiderosis in 1. In another monkey moderate periportal lymphocyte infiltration and an excess of lymphocytes in the capillaries were the only findings. Periportal lymphoid cell infiltration and irregular sinusoidal Kupffer cell swelling and proliferation, with accumulation of polymorphonuclear leucocytes and large and small lymphocytes, were seen in another. In two monkeys small midzonal nodules of lymphocytes with swollen endothelial or epithelioid cells were seen.

The foregoing findings are minor in character and probably not particularly significant. In the remaining seven monkeys there were focal necroses, and in four of them capillary thrombi. In one of these there was an acute pyogenic interstitial pancreatitis intercurrently fatal 55 days after the virus inoculation, and the scattered isolated coagulated liver cells, the diffuse fatty degeneration of the liver, and localized accumulation of hyaline and leucocytic capillary thrombi appear assignable to the intercurrent disease and not to virus action. In the remaining six the focal lesions appear to be assignable directly to virus action, though in two of them there was a concurrent infection with a not especially virulent vaccine virus. These six monkeys died 9, 11, 11, 11, 12, and 14 days after inoculation, just about the median death point (11 days) for all deaths due directly to the virus of lymphocytic choriomeningitis. The focal necroses were small, approximately 50 micra or less in diameter. They were composed variously of pale or oxyphil karyolytic liver cells, of nuclear and cellular debris, and of coarse-meshed fibrin

enmeshing nuclear fragments, variably numerous red corpuscles, and occasional coagulated liver cells. Only in one of these animals (9 days) was there a variable, irregular commixture of proliferating reticulum cells with the necrosis. In four monkeys there were also scattered to numerous isolated coagulated necrotic liver cells. Capillary thrombi were seen in three, some hyaline, some fibrinous, some of fragmenting leucocytes or caseous debris. Numerous mitoses in liver cells were present in one monkey.

No lesions of the *gall bladder* were encountered in the four monkeys in which it was studied histologically.

The *spleen* pulp in the earlier stages was usually more or less congested. Clumps of lymphoid cells were present in some animals, but absent in others. The follicles were hyperplastic in some, small and inactive in others. Sinus endothelial swelling was infrequent. Phagocytic activity of follicular or pulp reticulum cells was observed in a few animals 12 to 15 days after inoculation, but was inconstant. After the second week, the congestion disappeared and follicular activity was not common.

The femoral *bone marrow* was studied in 20 monkeys. In about half of these it remained predominantly fatty, the remainder showing more or less cellularity. Generally, neutrophil myelocytes were predominant, and fair numbers of metamyelocytes and leucocytes were present. Only occasionally were myeloblasts appreciably increased in numbers. Usually fair numbers of erythroblastic cells were present in the more cellular marrows. Megakaryocytes were present in average numbers and occasionally showed nuclear pyknosis. There were no focal lesions assignable to the disease.

The *lymph nodes* generally presented more or less hyperplasia of follicles and of the sinus reticulo-endothelium. Crowding of the sinuses with macrophages and erythrophagia were infrequent findings. Pulp and follicular reticulum cells were often swollen and contained ingested nuclear debris or hemosiderin. Focal granulomata were observed in the superficial regional nodes of two monkeys. One of these (9 days) showed a small, solid, epithelioid cell nodule, the other (34 days) caseopurulent foci bordered by polymorphonuclears, lymphocytes, plasma cells and proliferating epithelioid cells, and fibroblasts with some delicate collagen fibrils.

The *kidney* showed generally more or less finely granular swelling and marginal fraying of the epithelium of the convoluted and loop tubules, less often vacuolar or hydropic degeneration, and not infrequently more or less copious intratubular exudate. This exudate was usually a foamy or granular albuminous coagulum. Hyaline, granular, and, rarely, necrotic cellular casts occurred in a few animals.

In about two-thirds of the animals there was an exudative inflammation as well. This was most frequent and most marked in animals

dying 10 to 17 days after inoculation. The exudative cells were chiefly lymphocytes; sometimes some plasma cells and a few macrophages were present as well. The exudate occurred most often about arcuate vessels and as interstitial foci in the cortex. In about one-third of the monkeys there was a similar exudate beneath the parietal pelvic epithelium and in the pelvic fatty areolar tissues. Here there was inconstantly an accompanying serous exudation. The pelvic cellular exudate was partly diffuse, partly perivascular in distribution. Intraepithelial intercellular vesicles containing cell debris and macrophages were formed in the pelvic mucosa of one monkey (12 days). Lymphocyte infiltration of the cortex appeared as early as 9 days after inoculation and persisted as long as 34 and 55 days and 7 months. In 3 other monkeys it was absent at 81 days, 7 months, and 1 year after inoculation.

The inclusions in the nuclei of the convoluted and loop tubules described by Cowdry and Scott (2) were present in 7 of the 30 monkeys, or 23 percent. This proportion is not significantly different from that seen in 57 monkeys infected with poliomyelitis (21 percent). The significance of these inclusions is unknown.

In 13 of 22 monkeys the *adrenals* were normal. Two showed changes possibly assignable to intercurrent infections. In five there was more or less marked congestion, particularly in the zona reticularis, with focal hemorrhages in three. Focal lymphocyte infiltration of the zona reticularis and medulla was present in four monkeys, alone in one, and associated with congestion in three and with hemorrhages in two. One monkey dying 9 days after inoculation showed focal coagulation necrosis of cortical tissue, grading from isolated coagulated cells to focal necroses 0.1 mm in diameter. There was no marginal reaction about these necroses.

The *urinary bladder* was normal in 6 of 13 monkeys. One showed a patchy edema of the mucosa, one a patch of serosal mesothelial proliferation, submesothelial focal caseous necrosis and satellite perivascular lymphocyte and plasma cell infiltration, and two a focal perivascular and subepithelial lymphocyte infiltration of the mucosa. In three there was a hemorrhagic cystitis characterized by focal interstitial and subepithelial hemorrhages in the mucosa, concentric vascular endothelial swelling or proliferation, and perivascular and interstitial infiltration by lymphocytes in the mucosa. In one lymphoid follicles were formed, in one numbers of polymorphonuclear leucocytes were present in the epithelium, and in the third the epithelium contained scattered, swollen, clear epithelial cells grading into rounded clear spaces containing nuclear fragments and eosinophil globules and masses.

Testicles were studied in 15 monkeys and were normal in 6. In two the lesions present were probably assignable to intercurrent infection.

In five the testis was normal, with a focal lymphocyte infiltration of the cremaster and tunica vaginalis (two cases) or a more or less dense diffuse and perivascular lymphocyte infiltration in the epididymis (three cases). In two further monkeys there were, respectively, a slight focal (12 days) and a diffuse interstitial lymphocyte infiltration (20 days) of the testis, moderate diffuse and focal lymphocyte infiltration, slight edema, and a few plasma cells in the epididymis of the one monkey (12 days), and dense diffuse lymphocyte infiltration of epididymis, cremaster, and tunica vaginalis, with diffuse parietal and focal visceral stratifying proliferation of the tunical mesothelium (20 days). These exudative inflammatory changes appeared as early as 9 days and as late as 55 days after inoculation. In the last-noted monkey one testicle was normal. In the monkey showing the most severe changes in the testis excised 20 days after inoculation, the other testicle was normal at autopsy 2 months later.

Ovary, uterus, and *tube* were studied in six monkeys. All showed focal lymphocyte infiltration of greater or less extent and density. The mucosa of the tube was involved in five, the tubal muscularis as well in one, the myometrium in three, with the perimetrium as well in one, the ovarian stroma in one, and the broad ligaments in three. In one monkey there were necrosis and desquamation of thecal epithelium. The ovary was normal in four, the uterus in three, and the tube in one only.

Thymus was studied in four monkeys. None showed any lesions.

The *thyroid* was examined in 18 monkeys and showed no significant lesions in any.

Parathyroids were encountered in thyroid sections in nine animals. In five no lesions were seen, a single focus of acute karyorrhectic necrosis without marginal reaction was seen in one monkey (12 days), and focal perivascular lymphocyte infiltration was seen in three. In one of these last there were associated plasma cells and clumps of nuclear debris; but as a concurrent infection was present in this animal, the significance is doubtful.

Skeletal *muscle* was studied in 17 monkeys. In one there was a sparse focal lymphocyte infiltration. Three showed sarcosporidia and the remainder were normal.

SUMMARY

Lymphocytic choriomeningitis in monkeys is characterized by an almost constant, irregular, more or less pronounced lymphocyte infiltration of the plexus chorioidei of the cerebral ventricles, sometimes accompanied by serocellular exudation into the ventricles, by an almost constant, usually quite moderate, irregularly distributed lymphocyte infiltration of the leptomeninges, and by very few foci

of cellular gliosis and of vessel sheath lymphocyte infiltration in the brain and cord substance. Meningeal and plexus infiltration may persist for long periods after infection. Focal lymphocyte infiltration and sheath cell proliferation occur in the spinal root ganglia.

The lungs often present congestion, serous exudation, interstitial edema, hemorrhages, and perivascular lymphocyte infiltration. Pyelitis and a sometimes hemorrhagic cystitis occur in a number of animals and are characterized by focal and diffuse mucosal lymphocyte infiltration and edema. Foci of coagulative to fibrinoid hemorrhagic necrosis in the liver are seen in about one fourth of the animals, and focal necroses also occur occasionally in the adrenal and parathyroid. Splenic congestion, a variable grade of marrow hyperplasia and lymph node follicle hyperplasia, and sinus reticulo-endotheliosis are other frequent findings.

A focal interstitial or perivascular lymphocyte infiltration is frequent in the kidney, epididymis, uterus, tube, parathyroid, heart, lung, and tracheal mucosa, and occasional in esophageal mucosa, pancreas, adrenal, testis, ovary, and skeletal muscles.

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(2) Cowdry, E. V., and Scott, G. H.: Nuclear inclusions in the kidneys of *Macacus rhesus* monkeys. Am. Jour. Path., 11: 659–668 (1935).

DEATHS DURING WEEK ENDED FEB. 29, 1936

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

	Week ended Feb. 29, 1936	Corresponding week, 1935
Data from 86 large cities of the United States:		
Total deaths	10,573	9,473
Deaths per 1,000 population, annual basis	14.8	13.2
Deaths under 1 year of age	580	691
Deaths under 1 year of age per 1,000 estimated live births	52	64
Deaths per 1,000 population, annual basis, first 9 weeks of year	13.7	13.0
Data from industrial insurance companies:		
Policies in force	67,956,142	67,432,737
Number of death claims	16,328	15,011
Death claims per 1,000 policies in force, annual rate	12.6	11.6
Death claims per 1,000 policies, first 9 weeks of year, annual rate	10.8	10.8

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

Reports for Weeks Ended Mar. 7, 1936, and Mar. 9, 1935

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Mar. 7, 1936, and Mar. 9, 1935

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935
New England States:								
Maine	1	2	5	214	263	538	0	0
New Hampshire	----	----	----	----	55	16	0	0
Vermont	1	----	----	----	567	4	0	0
Massachusetts	2	5	----	----	819	471	8	2
Rhode Island	1	----	----	----	39	112	0	0
Connecticut	2	7	26	2	89	997	1	3
Middle Atlantic States:								
New York	37	21	[1] 109	[1] 20	2,368	2,226	30	15
New Jersey	14	17	89	14	148	1,058	5	3
Pennsylvania	48	60	----	----	776	5,103	14	4
East North Central States:								
Ohio [2]	29	41	39	28	258	810	9	9
Indiana	19	18	82	70	15	468	3	3
Illinois	35	45	63	71	57	2,709	20	19
Michigan	10	18	6	13	87	2,340	2	1
Wisconsin	3	1	89	98	76	2,290	2	0
West North Central States:								
Minnesota	3	5	----	----	272	1,813	1	3
Iowa	4	5	8	----	3	1,163	1	6
Missouri	21	28	618	243	63	873	7	7
North Dakota	1	9	6	27	1	33	2	0
South Dakota	3	1	----	5	14	38	0	0
Nebraska	3	6	----	----	88	336	0	1
Kansas	12	4	65	21	15	1,255	0	1
South Atlantic States:								
Delaware	3	2	----	2	69	2	1	0
Maryland [3]	8	9	70	72	195	141	15	2
District of Columbia	19	13	4	3	16	32	3	11
Virginia	15	11	2,046	----	84	1,216	24	4
West Virginia	14	21	135	234	8	518	11	3
North Carolina [4]	14	9	343	67	58	607	2	4
South Carolina [5]	2	5	1,005	425	16	62	1	15
Georgia [5]	13	15	1,544	387	----	----	13	0
Florida	4	9	61	39	4	36	1	0
East South Central States:								
Kentucky	15	15	73	103	62	1,141	16	4
Tennessee	24	13	477	228	79	89	29	9
Alabama [5]	13	8	2,140	761	63	433	1	4
Mississippi [3]	5	9	----	----	----	----	1	2

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Mar. 7, 1936, and Mar. 9, 1935—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935
West South Central States:								
Arkansas	8	8	346	118	1	41	0	2
Louisiana	18	22	141	27	70	175	0	3
Oklahoma [6]	11	20	298	505	5	188	11	9
Texas [5]	62	72	1,279	2,589	648	163	5	9
Mountain States:								
Montana	1	6	23	-------	18	111	3	0
Idaho	0	-------	1	4	20	46	0	0
Wyoming	-------	4	-------	-------	12	211	0	1
Colorado	8	9	-------	-------	8	708	0	1
New Mexico	2	7	9	9	44	-------	0	2
Arizona	4	1	213	105	81	26	0	0
Utah [3]	-------	1	-------	-------	20	23	0	2
Pacific States:								
Washington	1	3	11	5	329	207	2	2
Oregon	3	-------	269	144	564	95	1	1
California	32	42	2,099	377	2,729	598	11	7
Total	548	627	13,792	7,030	11,276	31,522	256	174
First 10 weeks of year	6,466	7,616	63,757	82,558	69,341	209,217	2,031	1,161

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935
New England States:								
Maine	0	0	14	22	0	0	0	0
New Hampshire	1	0	13	2	0	0	0	0
Vermont	0	0	20	17	0	0	0	0
Massachusetts	0	0	289	208	0	0	0	1
Rhode Island	0	0	9	15	0	0	0	0
Connecticut	0	0	126	70	0	0	0	0
Middle Atlantic States:								
New York	1	3	1,420	952	0	0	10	4
New Jersey	1	0	617	166	0	0	2	0
Pennsylvania	1	0	611	675	0	0	6	2
East North Central States:								
Ohio [2]	1	1	454	1,083	1	0	1	3
Indiana	0	0	267	237	3	1	3	0
Illinois	0	2	996	1,046	22	1	5	6
Michigan	0	0	334	423	1	0	3	2
Wisconsin	1	0	604	508	9	22	2	1
West North Central States:								
Minnesota	1	1	362	150	9	7	0	0
Iowa	0	1	155	67	13	1	3	1
Missouri	1	0	195	66	6	2	0	5
North Dakota	0	0	68	211	1	3	0	0
South Dakota	0	0	40	11	15	1	5	0
Nebraska	0	0	202	37	21	17	0	1
Kansas	2	0	266	94	26	32	1	0
South Atlantic States:								
Delaware	0	0	7	29	0	0	0	0
Maryland [3]	0	0	99	109	0	0	2	2
District of Columbia	0	0	34	65	0	0	0	0
Virginia	1	0	56	38	0	0	2	2
West Virginia	0	0	49	158	0	1	1	2
North Carolina [4]	0	0	43	41	1	0	3	1
South Carolina [5]	0	0	4	5	0	0	0	3
Georgia [5]	0	0	12	11	1	0	1	1
Florida	1	0	2	8	0	12	1	0

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Mar. 7, 1936, and Mar. 9, 1935—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935	Week ended Mar. 7, 1936	Week ended Mar. 9, 1935
East South Central States:								
Kentucky	0	0	72	54	1	1	2	4
Tennessee	1	0	34	30	2	0	1	3
Alabama [5]	1	0	18	12	2	0	1	1
Mississippi [3]	0	0	8	13	0	1	0	2
West South Central States:								
Arkansas	0	0	14	5	0	2	0	1
Louisiana	0	0	17	14	2	2	7	5
Oklahoma [6]	1	0	22	16	0	2	3	1
Texas [5]	0	1	87	121	9	30	1	14
Mountain States:								
Montana	0	0	111	19	7	0	0	1
Idaho	0	0	75	2	4	0	0	1
Wyoming	0	0	152	49	12	14	0	0
Colorado	0	1	136	354	13	1	0	0
New Mexico	0	0	88	16	0	0	2	8
Arizona	0	0	20	24	0	0	0	0
Utah [3]	0	0	103	102	1	0	0	0
Pacific States:								
Washington	0	1	100	72	14	29	1	3
Oregon	1	0	59	54	2	0	0	0
California	1	13	387	266	4	3	3	4
Total	17	24	8,871	7,747	202	185	72	85
First 10 weeks of year	204	273	75,040	67,815	2,230	1,944	1,045	1,346

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended Mar. 7, 1936, Ohio, 1 case.
[3] Week ended earlier than Saturday.
[4] Dengue, week ended Mar. 7, 1936, North Carolina, 1 case.
[5] Typhus fever, week ended Mar. 7, 1936, 15 cases, as follows: South Carolina, 1; Georgia, 7; Alabama, 2; Texas, 5.
[6] Exclusive of Oklahoma City and Tulsa.

SUMMARY OF MONTHLY REPORTS FROM STATES

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

State	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
January 1936										
Virginia	15	101	1,291	-------	92	4	1	221	0	17
February 1936										
Arkansas	7	30	596	39	6	20	1	58	1	4
Connecticut	7	5	34	-------	430	-------	1	320	0	3
Delaware	1	-------	-------	-------	289	-------	0	28	0	1
District of Columbia	19	95	15	-------	61	-------	1	105	0	2
Indiana	12	127	158	-------	92	-------	0	1,453	3	13
Iowa	20	40	21	-------	38	-------	0	627	59	15
North Carolina	17	83	1,107	-------	195	40	1	119	2	12
South Carolina	-------	84	5,900	162	61	62	3	21	1	1

Summary of monthly reports from States—Continued

January 1936

	Cases
Virginia:	
Chicken pox	264
Diarrhea and dysentery	28
Mumps	232
Septic sore throat	3
Tularaemia	16
Undulant fever	4
Whooping cough	108

February 1936

	Cases
Anthrax:	
Delaware	1
Chicken pox:	
Arkansas	74
Connecticut	513
Delaware	44
District of Columbia	72
Indiana	304
Iowa	203
North Carolina	562
South Carolina	110
Conjunctivitis, infectious:	
Connecticut	63
Diarrhea:	
South Carolina	213
Dysentery:	
Connecticut (amoebic)	1
Connecticut (bacillary)	1

February, 1936—Continued

	Cases
Epidemic encephalitis:	
Connecticut	1
Indiana	2
South Carolina	4
German measles:	
Connecticut	718
Delaware	1
North Carolina	380
South Carolina	25
Hookworm disease:	
South Carolina	43
Mumps:	
Arkansas	177
Connecticut	320
Delaware	81
Indiana	303
Iowa	905
South Carolina	179
Ophthalmia neonatorum:	
North Carolina	1
South Carolina	3
Paratyphoid fever:	
Connecticut	3
Rabies in animals:	
Indiana	46
South Carolina	32

February, 1936—Continued

	Cases
Septic sore throat:	
Connecticut	12
Iowa	3
North Carolina	2
Tetanus:	
Connecticut	3
South Carolina	6
Trachoma:	
Arkansas	3
Trichinosis:	
Connecticut	1
Tularaemia:	
North Carolina	2
Typhus fever:	
Connecticut	1
North Carolina	2
South Carolina	4
Undulant fever:	
Connecticut	7
Iowa	6
North Carolina	3
Whooping cough:	
Arkansas	34
Connecticut	288
Delaware	61
District of Columbia	33
Indiana	137
Iowa	38
North Carolina	109
South Carolina	60

WEEKLY REPORT FROM CITIES

City reports for week ended Feb. 29, 1936

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	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Maine:											
Portland	0	2	0	0	4	2	0	1	0	10	27
New Hampshire:											
Concord	0	------	0	0	0	0	0	0	0	0	13
Manchester	0	------	1	0	0	3	0	1	0	0	18
Nashua	0	------	------	2	------	0	0	------	0	0	------
Vermont:											
Barre	------	------	------	------	------	------	------	------	------	------	------
Burlington	0	------	0	0	0	0	0	0	0	0	8
Rutland	0	------	1	6	2	6	0	0	0	0	10
Massachusetts:											
Boston	1	------	1	287	36	65	0	11	1	9	243
Fall River	1	------	2	2	8	3	0	0	0	0	43
Springfield	0	------	0	0	2	5	0	1	0	9	39
Worcester	0	------	0	1	8	15	0	0	0	3	64
Rhode Island:											
Pawtucket	0	------	0	0	0	1	0	0	0	0	24
Providence	0	------	1	25	19	12	0	3	0	0	86
Connecticut:											
Bridgeport	0	3	1	4	0	3	0	0	0	6	31
Hartford	------	------	------	------	------	------	------	------	------	------	------
New Haven	0	1	0	1	4	0	0	2	1	36	44
New York:											
Buffalo	0	------	0	35	29	65	0	14	0	10	194
New York	39	78	16	1,489	286	600	0	98	2	88	1,892
Rochester	0	2	0	0	10	4	0	2	0	2	98
Syracuse	0	------	0	56	4	3	0	2	1	10	57
New Jersey:											
Camden	2	3	1	0	1	6	0	0	0	1	33
Newark	0	28	0	2	17	245	0	6	1	22	129
Trenton	0	------	0	2	5	5	0	2	0	16	32
Pennsylvania:											
Philadelphia	5	19	9	395	68	68	0	35	0	54	635
Pittsburgh	10	9	4	44	51	105	0	6	0	20	229
Reading	0	------	0	3	1	5	0	1	0	0	26
Scranton	0	------	------	81	------	6	0	------	0	2	------

City reports for week ended Feb. 29, 1936—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Ohio:											
Cincinnati	4	------	7	14	26	28	0	12	0	2	181
Cleveland	0	48	4	72	25	52	0	18	0	61	223
Columbus	7	6	6	5	12	15	0	6	0	2	107
Toledo	0	1	1	37	8	5	0	8	0	3	78
Indiana:											
Anderson	1	------	0	0	3	3	0	0	0	2	10
Fort Wayne	3	------	0	0	4	12	0	1	0	0	27
Indianapolis	0	------	0	1	31	27	0	2	0	23	130
Muncie	0	------	0	1	1	4	0	0	0	0	12
South Bend	0	------	0	0	5	5	0	0	0	7	17
Terre Haute	0	------	0	0	1	4	0	0	0	0	37
Illinois:											
Alton	0	------	1	0	4	1	0	0	0	3	19
Chicago	14	14	10	11	69	259	1	44	1	212	802
Elgin	0	1	1	0	0	2	0	1	0	0	12
Moline	0	------	1	0	6	55	0	1	0	2	15
Springfield	0	------	0	0	7	25	0	0	0	3	32
Michigan:											
Detroit	5	6	3	17	52	133	0	15	0	135	293
Flint	1	------	0	2	5	13	0	0	0	22	33
Grand Rapids	0	------	0	5	1	11	0	0	0	6	34
Wisconsin:											
Kenosha	0	------	0	1	2	3	0	0	0	5	10
Milwaukee	0	3	1	5	9	121	0	7	0	52	112
Racine	0	------	0	0	1	26	0	0	0	5	19
Superior	0	------	0	0	0	5	0	0	0	0	7
Minnesota:											
Duluth	0	------	0	1	2	3	0	2	0	6	22
Minneapolis	2	------	2	107	9	139	0	0	0	10	103
St. Paul	0	2	2	129	13	33	0	0	0	6	68
Iowa:											
Cedar Rapids	0	------	--------	1	-------	1	0	-------	0	3	--------
Davenport	1	------	--------	0	-------	6	0	-------	0	1	--------
Des Moines	1	------	--------	0	10	5	0	2	0	0	49
Sioux City	0	------	--------	0	-------	13	11	-------	0	0	--------
Waterloo	1	------	--------	0	-------	5	0	-------	0	0	--------
Missouri:											
Kansas City	2	19	1	3	18	61	0	6	0	0	152
St. Joseph	0	------	5	0	23	5	0	3	0	0	99
St. Louis	8	3	0	3	26	88	0	10	0	5	297
North Dakota:											
Fargo	0	------	0	0	0	11	0	0	0	0	4
Grand Forks	0	------	--------	0	-------	0	5	-------	0	0	--------
Minot	0	------	0	0	0	15	0	0	0	0	2
South Dakota:											
Aberdeen	0	------	--------	1	-------	0	0	-------	0	0	--------
Sioux Falls	0	------	0	0	0	8	3	0	0	0	9
Nebraska:											
Omaha	0	------	0	1	19	131	0	1	0	1	80
Kansas:											
Lawrence	0	------	0	0	5	1	1	0	0	0	13
Topeka	0	------	0	1	5	54	0	2	0	1	35
Wichita	1	2	2	0	13	21	1	2	0	4	45
Delaware:											
Wilmington	0	------	0	2	5	1	0	1	0	3	39
Maryland:											
Baltimore	5	29	8	23	29	36	0	14	0	21	295
Cumberland	0	------	0	1	2	6	0	0	0	0	18
Frederick	0	1	1	0	0	0	0	0	0	0	3
District of Col.:											
Washington	22	5	4	25	32	30	0	22	1	7	226
Virginia:											
Lynchburg	0	------	1	0	6	0	0	0	0	8	16
Norfolk	1	------	2	0	5	6	0	2	0	0	30
Richmond	1	10	6	0	9	21	0	4	0	3	80
Roanoke	2	------	0	1	8	4	0	1	0	0	23
West Virginia:											
Charleston	2	6	0	0	3	0	0	0	0	0	20
Huntington	0	------	0	0	0	0	0	0	0	0	0
Wheeling	1	------	0	2	3	1	0	0	0	0	18
North Carolina:											
Gastonia	0	4	0	1	3	0	0	0	0	0	--------
Raleigh	0	------	0	0	6	0	0	0	0	2	27
Wilmington	0	9	0	0	3	1	0	1	0	0	13
Winston-Salem	0	4	1	117	6	3	0	1	0	0	21

City reports for week ended Feb. 29, 1936—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
South Carolina:											
Charleston	1	394	4	0	11	3	0	1	0	0	38
Columbia											
Florence	0		0	0	5	0	0	0	0	0	13
Greenville	0		0	7	2	0	0	0	0	0	6
Georgia:											
Atlanta	4	502	18	1	33	13	0	5	0	1	132
Brunswick	0		0	1	5	0	0	0	0	0	7
Savannah	0	339	5	0	3	2	0	1	0	0	29
Florida:											
Miami	2		0	2	1	0	0	1	3	4	40
Tampa	0	3	1	0	4	0	0	4	1	0	38
Kentucky:											
Ashland	1	4		0		0	0		0	1	
Covington	0		0	4	4	0	0	0	0	3	38
Lexington	0		0	0	5	2	0	0	0	0	25
Louisville	2	6	2	6	19	22	0	3	0	11	
Tennessee:											
Knoxville	3	9	2	51	6	3	0	1	0	0	42
Memphis	4		5	2	27	8	0	8	0	2	122
Nashville	1		4	0	10	2	0	3	0	0	70
Alabama:											
Birmingham	3	223	5	0	11	4	0	2	0	0	84
Mobile	1	95	2	2	2	1	0	1	0	0	25
Montgomery	1	57		0		2	0		0	0	
Arkansas:											
Fort Smith											
Little Rock	2	2	0	0	2	3	0	1	0	0	3
Louisiana:											
New Orleans	11	20	6	22	29	15	0	17	0	8	203
Shreveport	2		0	17	13	0	0	3	0	0	41
Oklahoma:											
Oklahoma City	1	20	0	0	20	7	0	0	0	0	55
Texas:											
Dallas	4	5	3	112	12	2	0	7	0	1	79
Fort Worth	3		6	2	13	13	0	3	0	1	66
Galveston	1		0	7	7	0	0	4	0	0	30
Houston	11		2	18	20	1	0	12	0	0	102
San Antonio	1		9	0	9	3	0	9	0	0	92
Montana:											
Billings	0		0	0	2	10	0	0	0	3	9
Great Falls	0		0	0	1	11	0	0	0	1	8
Helena	0		0	0	0	1	0	0	0	0	4
Missoula	0		0	0	0	11	0	0	0	0	1
Idaho:											
Boise	0		0	3	2	14	0	0	0	0	8
Colorado:											
Colorado Springs	1		0	5	0	6	0	2	0	4	18
Denver	4		3	6	21	34	0	2	2	13	108
Pueblo	0		0	0	2	16	0	0	0	3	9
New Mexico:											
Albuquerque	0		1	0	3	26	0	2	0	4	12
Utah:											
Salt Lake City	1		0	0	2	112	0	1	0	9	
Washington:											
Seattle	0		6	71	15	20	0	14	0	5	131
Spokane	0	5	5	5	3	11	0	1	0	3	40
Tacoma	0		0	85	11	2	0	0	0	0	40
Oregon:											
Portland	0	27	2	366	13	8	0	0	0	3	104
Salem	0	4		1		2	0		0	0	
California:											
Los Angeles	7	203	1	575	31	122	0	18	1	15	350
Sacramento	2	80	2	21	4	6	0	5	0	4	48
San Francisco	0	18	5	421	9	71	0	11	1	13	230

City reports for week ended Feb. 29, 1936—Continued

State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths	
Massachusetts:			
Boston	8	4	1
Worcester	1	2	0
Rhode Island:			
Providence	1	0	0
New York:			
New York	24	4	1
New Jersey:			
Newark	2	1	0
Pennsylvania:			
Philadelphia	2	1	0
Pittsburgh	2	1	0
Ohio:			
Cincinnati	3	1	0
Cleveland	0	0	2
Illinois:			
Chicago	6	4	0
Mighigan:			
Detroit	0	1	0
Grand Rapids	1	0	0
Minnesota:			
Minneapolis	1	0	0
Iowa:			
Sioux City	0	1	0
Missouri:			
Kansas City	2	1	0
St. Joseph	6	4	0
St. Louis	1	0	0
Delaware:			
Wilmington	1	1	0
Maryland:			
Baltimore	11	7	0
District of Columbia:			
Washington	7	4	0
Virginia:			
Norfolk	1	0	0
Richmond	5	2	0
South Carolina:			
Charleston	17	0	0
Georgia:			
Atlanta	7	4	0
Savannah	1	0	0
Florida:			
Miami	1	1	0
Kentucky:			
Ashland	1	1	0
Louisville	2	1	0
Tennessee:			
Knoxville	2	0	0
Memphis	1	2	0
Alabama:			
Birmingham	0	1	0
Louisiana:			
New Orleans	2	2	0
Shreveport	0	4	0
Oklahoma:			
Oklahoma City	2	1	0
Texas:			
Houston	5	2	0
Colorado:			
Colorado Springs	0	1	0
Denver	1	0	0
Utah:			
Salt Lake City	1	0	0
Washington:			
Seattle	1	0	0
Oregon:			
Portland	0	0	1
California:			
Los Angeles	5	1	6
Sacramento	0	1	0
San Francisco	2	0	0

Epidemic encephalitis.—Cases: Newark, 1; Baltimore, 1.
Pellagra.—Atlanta, 1; Montgomery, 1; Dallas, 1; Albuqerque, 1.
Typhus fever.—Cases: Philadelphia, 1.

FOREIGN AND INSULAR

BRAZIL

Malaria.—According to information dated February 15, 1936, an outbreak presumed to be pernicious malaria has been reported on Lago Grande located between Santa Rem and Obidos, about 500 miles from Para, Brazil.

BRITISH WEST INDIES

Island of Dominica—Vital statistics—1935.—Following are vital statistics for the Island of Dominica, British West Indies, for the year 1935:

Birth rate per 1,000 population	31.05
Stillbirths per 100 live births	4.88
Infant mortality rate per 1,000 live births	97.60
Deaths per 1,000 population	14.58
Deaths from:	
All causes	683
Diarrhea and enteritis (under 2 years)	28
Diarrhea and enteritis (2 years and over)	17
Dysentery (amoebic)	2
Deaths from—Continued.	
Dysentery (bacillary or undefined)	13
Influenza	42
Malaria	47
Tetanus	2
Tuberculosis (all forms)	71
Typhoid fever	2
Yaws	1

CANADA

Communicable diseases—2 weeks ended February 22, 1936.—During the 2 weeks ended February 22, 1936, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada, as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				2	3	1		1		7
Chicken pox		21	2	334	547	49	34	47	185	1,219
Diphtheria		12	4	20	7	9	5	3	1	61
Dysentery				2	2					4
Erysipelas				11	8	2		3	2	26
Influenza		4	1		266	20			50	341
Lethargic encephalitis					1					1
Measles	10	58	33	2,579	4,413	970	1,275	225	745	10,308
Mumps		20			1,069	94	310	71	350	1,914
Paratyphoid fever	1				3					4
Pneumonia	2	2			55				20	79
Poliomyelitis					1					1
Scarlet fever		19	8	239	677	136	31	90	61	1,261
Trachoma									1	1
Tuberculosis	4	3	6	116	84	31	18	3	28	293
Typhoid fever			1	29	8	5		4	2	49
Undulant fever				1				1		2
Whooping cough		15	6	164	413	21	56	19	98	792

DENMARK

Communicable diseases—October–December 1935.—During the months of October, November, and December 1935, cases of certain communicable diseases were reported in Denmark as follows:

Disease	October	November	December	Disease	October	November	December
Cerebrospinal meningitis	2	2	2	Paratyphoid fever	12	11	5
Chicken pox	19	55	75	Poliomyelitis	36	13	6
Diphtheria and croup	345	365	334	Puerperal fever	21	13	18
Epidemic encephalitis	4	4	2	Scabies	1,321	1,174	963
Erysipelas	343	335	302	Scarlet fever	990	812	632
German measles	6	9	52	Syphilis	77	68	73
Gonorrhea	1,053	894	728	Tetanus, neonatorum	2	4	1
Influenza	4,168	5,057	5,138	Tetanus, traumatic			3
Malaria	5	12	8	Typhoid fever	2	4	3
Measles	1,085	639	680	Undulant fever (Bact. abort. Bang)	47	45	32
Mumps	469	616	762	Whooping cough	3,321	3,582	2,941
Paradysentery	42	23	96				

JAMAICA

Communicable diseases—4 weeks ended February 22, 1936.—During the 4 weeks ended February 22, 1936, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Cerebrospinal meningitis		2	Poliomyelitis		1
Chicken pox	4	21	Scarlet fever		1
Dysentery	14	4	Tuberculosis	54	105
Leprosy		4	Typhoid fever	25	144

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

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for February 28, 1936, pages 227–240. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued March 27, 1936, and thereafter, at least for the time being, in the issue published on the last Friday of each month.

Plague

Argentina—Buenos Aires Province—Ingeniero White.—A report dated March 9, 1936, stated that 2 cases of plague with 2 deaths were reported at Ingeniero White, Buenos Aires Province, Argentina.

Brazil—Ceara State—Crato.—During the month of February 1936 7 cases of plague with 2 deaths were reported at Crato, Ceara State, Brazil.

Egypt—Minya Province.—During the week ended February 29, 1936, 2 cases of plague were reported in Minya Province, Egypt.

Smallpox

Algeria—Department of Algiers.—During the week ended February 15, 1936, 1 case of smallpox was reported in the Department of Algiers, Algeria.

Argentina.—A report dated March 9, 1936, states that smallpox has been reported in Argentina, as follows: Buenos Aires Province, Puerto Belgrano, 4 cases; Entre Rios Province, Lucas Norte, 5 cases, 2 deaths.

Egypt.—During the week ended February 15, 1936, 2 cases of smallpox were reported in Egypt.

Poland—Department of Lublin.—During the week ended February 8, 1936, 1 case of smallpox was reported in the Department of Lublin, Poland.

Typhus fever

Mexico—San Luis Potosi.—During the week ended February 29, 1936, 1 death from typhus fever was reported at San Luis Potosi, Mexico.

Yellow fever

Brazil.—Yellow fever has been reported in Brazil as follows: Minas Geraes State: Dores Campo, Formoso, February 9, 1936, 1 case, 1 death; Sao Thomaz de Aquino, February 4, 1936, 1 case, 1 death; Parana State, Londrina, February 17, 1936, 1 case, 1 death; Sao Paulo State, Araraquara, February 9, 1936, 1 case, 1 death; Santo Antonio Alegria, February 9, 1936, 1 case, 1 death.

Gold Coast—Kumasi.—During the week ended February 29, 1936, 1 fatal case of yellow fever was reported at Kumasi, Gold Coast.

Ivory Coast—Indenie Circle—Correction.—The fatal case of suspected yellow fever during the last 10 days of December 1935, in Indenie Circle, Ivory Coast, and published in the PUBLIC HEALTH REPORTS on pages 107, 137, and 240, has been rediagnosed as not due to yellow fever.