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## THE INCIDENCE AND FUTURE EXPECTANCY OF MENTAL DISEASE ${ }^{1}$

By Harold F. Dorn, Statistician, United States Public Health Service

It is commonly believed that the proportion of the population suffering from mental disease is increasing. An impressive array of statistics supports this belief. Quite aside from future trends, the present problem occasioned by mental disorders is one of major proportions. It is estimated that the annual cost of hospitalized patients alone is between $\$ 150,000,000$ and $\$ 200,000,000$ and that these patients occupy 47 percent of the total number of hospital beds.

For the country as a whole the number of persons hospitalized for mental disease increased more than 40 percent from 1926 to 1936 (table 1). In New York State, where fairly adequate hospital facilities are available, the number of first admissions per 100,000 population has consistently increased for the past 30 years, although similar data for Massachusetts show no perceptible upward trend (fig. 1).

One of the most common explanations of the apparent increase in the incidence of mental disease is the complexity and strain of modern life. According to this theory, the human race developed in a simple rural environment, and the physical and mental characteristics of the race evolved as an adaptation to that environment. However, the development of modern civilization has led to the concentration of an increasing proportion of the population in cities. It is thought that, biologically, man is not adapted to modern city life, with its attendant stresses and strains, and that the multiplicity of stimuli, which constantly affect each person, hastens mental maladjustment.

It is important to investigate carefully the truth of this theory; for, if true, the situation is a severe indictment of our culture and is a harbinger of eventual racial decay. Even though the achievements of a culture be great, if they are accomplished at the expense of the biological heritage of the race, eventual doom is almost certain.

Owing to the fact that there is no sharp line of demarcation between normality and abnormality, it is practically impossible to decide

[^0]Table 1.-Number of patients present at the beginning of each year and number of first admissions per 100,000 population-State hospitals, United States, 1926-86

| Year | Patients present on January 1 |  | First admissions |  | Year | Patients present on January 1 |  | First admissions |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\underset{\text { ber }}{\text { Num- }}$ | $\begin{gathered} \text { Rate } \\ \text { per } \\ 100,000 \end{gathered}$ | $\underset{\text { ber }}{\text { Num }}$ | $\begin{gathered} \text { Rate } \\ \text { per } \\ 100,000 \end{gathered}$ |  | $\underset{\text { ber }}{\text { Num- }}$ | $\begin{gathered} \text { Rate } \\ \text { per } \\ 100,000 \end{gathered}$ | $\underset{\text { ber }}{\text { Num- }}$ | $\begin{gathered} \text { Rate } \\ \text { per } \\ 100,000 \end{gathered}$ |
| 1926 | 246, 48§ | 217 | 52,793 | 46 | 1932 | 305, 031 | 245 | 67, 083 |  |
| 1927 | 256, 858 | 219 | 56, 144 | 47 | 1933. | 321, 824 | 257 | 69, 368 | 55 |
| 1928 | 264, 511 | 222 | 59, 417 | 50 | 1934 | 332, 004 | 262 | 69, 934 | 55 |
| 1929. | 272, 252 | 226 | 60, 500 | 50 | 1935. | 342, 167 | 269 | 72,438 | 57 |
| 1930 | 280, 252 | 229 | 62, 738 | 51 54 | 1836. | 353, 305 | 276 |  |  |
| 1831. | 292, 284 | 236 | 67, 152 | 54 |  |  |  |  |  |



Figure 1.-Number of first admissions to mental hospitals per $\mathbf{1 0 0 , 0 0 0}$ population, New York and Massachusetts, 1909-1935
whether or not the relative number of persons with mental disease is increasing. Fundamentally, even though there is a definite physical basis for many mental disorders, mental disease, or insanity as it is popularly called, is a cultural concept and varies from one group to another. In some situations the mentally deranged have become soothsayers, medicine men, prophets, or group leaders; in other situations the same persons would be incarcerated.

Only a few hundred years ago the violently insane were thought to be obsessed by demons and were frequently killed or forced to leave the community. With some modification, mainly the substitution of incarceration as the method of treatment, this idea of "insanity," as it was called, prevailed even later, and indeed still prevails in some places; but, with increased knowledge of the functioning of the human body, the concept of mental ill health has gradually expanded until today it includes many conditions formerly considered normal. The recognition that mental ill health is more prevalent than hospital admissions reveal has led to the establishment of child guidance clinics, the employment of psychiatric social workers, the addition of psychiatrists to the staffs of hospitals, prisons, and similar institutions, to devoting special attention to problem children in school and to various adult education programs.

It is gradully becoming accepted that mental aberration is simply a form of illness which may be cured or alleviated by suitable care and treatment. This attitude is clearly reflected by the fact that the "insane asylums" of 50 years ago have become "mental hospitals" today. Commitment to a hospital is no longer universally regarded with the same horror as formerly. This change in the public attitude toward hospitalization, which has been concomitant with a changing cultural conception of mental disease, has undoubtedly increased the frequency with which cases of mild mental disorder are hospitalized.

The increasing proportion of the population living in cities also tends to increase the use of hospital facilities. Many persons, who would be regarded as merely "queer" in a rural community, undoubtedly experience difficulty in adjusting themselves to an urban environment. Moreover, due to inadequate housing, low income, and the uncertainty of continuous employment, home care of nonviolent cases of mental disorder, especially those associated with senility, is more difficult in the city than on the farm. The two principal exogenous factors which are thought to effect the prevalence of mental disease, alcoholism and syphilis, are also more common in urban communities. For these reasons some increase in the proportion of the population thought to be mentally ill is to be expected, even if there has been no real increase in the incidence of mental disorder.

However, it is impossible to determine whether or not this is true, because there is no practical method of determining the incidence of mental disease, either at the present time or in the past. Regardless of the amount of mental illness in the population, we are forced to measure it by the number of mental cases recognized and committed to an institution. This method of measurement is not so unsatisfactory as might at first appear, since cases of mental disease become a public burden only after they are recognized. And so, instead of
speaking of the number of persons with some form of mental disease, a number which is unknown, it will be necessary to restrict this discussion to the number of persons who are committed to a mental disease hospital. This does not, of course, include all persons with a mental disorder, but merely those who have been recognized as needing institutional care and for whom hospital facilities are available.

This measure of mental illness will not lead to an unequivocal answer to the question of whether or not the proportion of the population with mental disease is increasing. An increase in the proportion of the population committed to a mental hospital does not necessarily mean that an increasing proportion of the population has some form of mental disease. It may arise solely from the operation of the factors previously mentioned. However, if there is no real increase in the proportion of the population committed to a mental hospital, then it is probably true that there has been no increase in the incidence of mental disease.

This measure of mental disease may be expressed in two ways-one, the total number of patients under care during a given period, the other, the number of first admissions during a corresponding period. The former is a measure of the total public burden of mental disease; the latter is a measure of the proportion of the population which eventually is committed at least once to a mental hospital.

Since the present discussion is concerned solely with the latter problem, it is based upon first admissions only. It should be noted, however, that the number of first admissions is not an accurate index of the total number of persons with mental disease at any time, since it does not take into consideration the duration of the disease nor the number of readmissions. Consequently, the number of persons with mental disease is always greater than the number of first admissions.

There are several ways of expressing the incidence of mental disease as just defined. One frequently used index is the number of first admissions per 100,000 population, but this rate is subject to the same criticism as a crude death rate; namely, that it is affected by the composition of the population to which it refers. If the population contains a large proportion of old persons, the rate will be high, since the elderly are more subject to mental disease than the youthful.

Age specific commitment rates for mental disease can be computed in the same way as age specific mortality rates, but it is difficult to summarize concisely a large number of such rates.

A simple modification of the method of computing a life table leads to a measure of mental disease analogous to the expectation of life of the life table. This measure may be called the expectation of mental disease, which is a simplified expression of the longer phrase, expectation of commitment to a mental hospital. This is calculated by applying the mortality and first commitment rates of a given population at
a given time to a hypothetical number of infants, usually 1,000 or 100,000 , and determining the number who would be alive and sane at each age if they were continually subject to the assumed mortality and mental disease rates. The expectation of mental disease is simply the ratio of the number who live to become insane to the original 1,000 infants. This is also referred to as the probability or chance of developing mental disease. For example, it may be said that 50 out of every 1,000 infants will be committed to a mental hospital before death or that the probability or chance of insanity is 50 per 1,000 or 5 per 100.

To a certain extent the increase in the number of first admissions to mental hospitals merely reflects an increase in hospital facilities. In order to eliminate this factor insofar as it is possible, the data used will be the number of first admissions to mental hospitals in Massachusetts, New York, and Illinois, where hospital facilities have been fairly adequate for several years. These States had a combined population of more than $24,000,000$ in 1930.

Fifty-seven out of every 1,000 male infants and 53 out of every 1,000 female infants would live to be committed to a mental hospital if subject throughout their lifetime to the mortality and first admission for mental disease rates prevailing in Massachusetts during 1929-31. These represent increases of 3.6 and 8.2 percent, respectively, since 1920, when 55 male and 49 female babies could expect to live to be committed to a mental hospital (table 2 and fig. 2).

For females the chance of eventually being committed to a mental hospital increased at each age throughout the entire span of life. In general, the largest increases were at the older ages. Since there are very few commitments before age 15 , the differences in the probabilities of commitment for the younger ages are determined by differences in mortality rates at those ages.

In contrast to the females, the probability of developing mental disease among males decreased from ages 5 to 55 during the period 1920 to 1930. As will be shown later, this difference in the expectancy on the basis of sex results from different rates of change in the mortality rates of the two sexes.

In New York State the chances of eventual commitment for mental disease are slightly less than in Massachusetts, being 53 per 1,000 for male infants and 48 per 1,000 for female infants. With the exception of a few ages for women, the chances increased appreciably at all ages for both sexes. The increase for males was considerably greater than in Massachusetts (table 3 and figs. 3 and 4).

The exact meaning of the data in tables 2 and 3 should be kept clearly in mind. They apply to a hypothetical population unaffected by emigration or immigration and subject throughout its lifetime to the mortality rate and first commitment rate for mental disease existing in 1930. Moreover, the figure for any age is affected by the rates at

Table 2.-Chances per 1,000 of being committed to a hospital for mental disease, by age and sex, total population, State of Massachusetts, 1919-21, and 1929-s1

| Exact age | Males |  |  | Females |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Chance per 1,000 |  | $\begin{gathered} \text { Percent } \\ \text { change } \\ 1919-21 \text { to } \\ 1929-31 \end{gathered}$ | Chance per 1,000 |  | Percent change 1919-21 to 1929-31 |
|  | 1919-21 | 1929-31 |  | 1919-21 | 1920-31 |  |
| 0........ | 6564656564 | 5762636361 | $\begin{aligned} & +3.6 \\ & -3.1 \\ & -3.1 \\ & -3.1 \\ & -4.7 \end{aligned}$ | $\begin{aligned} & 49 \\ & 55 \\ & 56 \\ & 56 \\ & 55 \end{aligned}$ | 5357585856 | $\begin{aligned} & +8.2 \\ & +3.6 \\ & +3.6 \\ & +3.6 \\ & +1.8 \end{aligned}$ |
| 5. |  |  |  |  |  |  |
| 10. |  |  |  |  |  |  |
| 15... |  |  |  |  |  |  |
| 20..-- |  |  |  |  |  |  |
| 25-... | 6158545046 | 5955524845 | $\begin{aligned} & -3.3 \\ & -5.2 \\ & -3.7 \\ & -4.0 \\ & -2.2 \end{aligned}$ | $\begin{aligned} & 53 \\ & 51 \\ & 47 \\ & 44 \\ & 41 \end{aligned}$ | $\begin{aligned} & 54 \\ & 52 \\ & 49 \\ & 46 \\ & 43 \end{aligned}$ | $\begin{array}{r} +1.9 \\ +2.0 \\ +4.3 \\ +4.5 \\ +4.9 \end{array}$ |
| 30-.... |  |  |  |  |  |  |
| 35-. |  |  |  |  |  |  |
| 40 |  |  |  |  |  |  |
| 45... |  |  |  |  |  |  |
| 50 | 434148383634 | $\begin{aligned} & 42 \\ & 40 \\ & 38 \\ & 37 \\ & 35 \end{aligned}$ | $\begin{array}{r} -2.3 \\ -2.4 \\ 0.0 \\ +2.8 \\ +2.9 \end{array}$ | $\begin{aligned} & 37 \\ & 34 \\ & 32 \\ & 31 \\ & 30 \end{aligned}$ | 4036343332 | +8.1+5.9+6.3+6.5+6.7 |
| 55.... |  |  |  |  |  |  |
| 60 |  |  |  |  |  |  |
| 65 |  |  |  |  |  |  |
| 70 |  |  |  |  |  |  |
| 75. | $\begin{aligned} & 30 \\ & 27 \end{aligned}$ | 3330 | $\begin{aligned} & +10.0 \\ & +11.1 \end{aligned}$ | 2725 | 3028 | $\begin{aligned} & +11.1 \\ & +12.0 \end{aligned}$ |
| 80... |  |  |  |  |  |  |



Figure 2.-Chances per 1,000 of eventually being committed to a mental hospital, by sex and age, total population, Massachusetts, 1919-21 and 1929-31.
each older age. For example, the fact that 57 out of 1,000 male infants will eventually be admitted to a hospital for mental disease results from the mortality and mental disease rates affecting these infants throughout their lifetime. If they should all die before age 20 , very few would develop mental disease; if all lived until age 80 , a large number would develop mental disease. It is obvious, then,

Table 3.-Chances per 1,000 of being committed to a hospital for mental disease, by age and sex, total population, New York State, 1919-21 ${ }^{1}$ and 1929-31

| Exact age | Males |  |  | Females |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Chance per 1,000 |  | $\begin{gathered} \text { Percent } \\ \text { change } \\ 1919-21 \text { to } \\ 1929-31 \end{gathered}$ | Chance per 1,000 |  | Percent change 1919-21 to 1029-31 |
|  | 1919-21 | 1929-81 |  | 1919-21 | 1929-31 |  |
| 0----------------1. | 47 | 53 | 12.8 | 44 | 48 | 9.1 |
| 5. | 54 | 58 | 7.4 | 50 | 51 | 2.0 |
| 10-- | 54 | 59 | 9.3 | 51 | 52 | 2.0 |
| 15--- | 54 | 59 | 9.3 | 51 | 52 | 2.0 |
| 20-.- | 53 | 58 | 9.4 | 50 | 51 | 2.0 |
| 25...-- | 50 | 55 | 10.0 | 49 | 49 | 0.0 |
| 30-. | 47 | 52 | 10.6 | 46 | 47 | 2.2 |
| 35 | 44 | 48 | 9. 1 | 44 | 44 | 0.0 |
| 40 | 40 | 45 | 12.5 | 41 | 41 | 0.0 |
| 45---- | 37 | 41 | 10.8 | 38 | 38 | 0.0 |
| 50-..- | 34 | 38 | 11.8 | 34 | 35 | 2.9 |
| 55-- | 31 | 36 | 16.1 | 31 | 32 | 3.2 |
| 60 | 28 | 34 | 21.4 | 29 | 30 | 3.4 |
| 65 | 26 | 32 | 23.1 | 26 | 29 | 11.5 |
| 70-...- | 23 | 31 | 34.8 | 24 | 28 | 16.7 |
| 75-- | 20 | 28 | 40.0 | 22 | 26 | 18.2 |
| 80...-- | 18 | 24 | 33.3 | 20 | 24 | 20.0 |

${ }^{1}$ Data for 1919-21 are from H. M. Pollock and Benjamin Malzberg, "Expectation of Mental Disease," Psychiatric Quarterly, October 1928.


Tigure 3.-Chances per 1,000 of males eventually being committed to a mental hospital, by age, total population, New York State, 1919-21 and 1929-31.
that the data in tables 2 and 3 reflect changes both in mortality and mental disease rates.

To compare changes in the incidence of mental disease at any age independently of changes in mortality rates or of changes in mental disease rates at other ages, it is necessary to consider commitment rates for each age separately as given in tables 4 and 5. These


Figure 4.-Chances per 1,000 of females eventually being committed to a mental hospital, by age, total population, New York State, 1919-21 and 1929-31.

Table 4.-Number of first admissions to hospitals for mental diseases per 100,000 total population, by age and sex, State of Massachusetts, 1919-21 and 1929-81

| Exact age | Males |  |  | Females |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Admissions per 100,000 population |  | $\begin{gathered} \text { Percent } \\ \text { change } \\ 1919-21 \text { to } \\ 1929-31 \end{gathered}$ | Admissions per 100,000 population |  | $\begin{gathered} \text { Percent } \\ \text { change } \\ 1919-21 \text { to } \\ 1929-31 \end{gathered}$ |
|  | 1919-21 | 1929-31 |  | 1919-21 | 1929-31 |  |
| 15-.-- | 33 | 24 | -27.3 | 25 | 21 | -16.0 |
| 20... | 76 | 64 | -15.8 | 57 | 49 | -14.0 |
| 25 | 94 | 82 | -12.8 | 76 | 62 | -18.4 |
| 30. | 103 | 87 | -15.5 | 93 | 70 | -24.7 |
| 35-.-- | 116 | 96 | -17.2 | 96 | 82 | -14.6 |
| 40. | 119 | 98 | -17.6 | 92 | 85 | -7.6 |
| 45 | 110 | 102 | $-7.3$ | 106 | 92 | -13.2 |
| 50. | 104 | 110 | +5.8 | 111 | 104 | -6.3 |
| 55. | 122 | 116 | -4.9 | 102 | 106 | +3.9 |
| 60. | 147 | 131 | -10.9 | 108 | 102 | -3.8 |
| 65. | 192 | 165 | -14.1 | 133 | 129 | -3.0 |
| 70. | 264 | 243 | -8.0 | 199 | 195 | -2.0 |
| 75. | 331 | 355 | +7.3 | 261 | 267 | +2.3 |
| 80. | 399 | 434 | +8.8 | 314 | 341 | +8.6 |

tables present the number who will be committed to a mental hospital within one year, out of 100,000 persons alive and sane at the beginning of each age. For example, the figure 24 opposite males aged 15 for 1929-31 in table 4 means that out of 100,000 males alive and sane on their fifteenth birthday 24 will be committed to a mental hospital before they become 16 vears of age.

In Massachusetts, from 1920 to 1930, with two exceptions, these rates decreased at each age for both males and females until age 75. The decreases were larger for ages under 40 and were generally slightly greater for males than for females (fig. 5).


Figure 5.-Percentage change in the number of first admissions to mental hospitals per 100,000 total population, by age and sex, Massachusetts, 1919-21 to 1929-31.

Table 5.-Number of first admissions to hospitals for mental disease per 100,000 total population, by age and sex, New York State, 1919-21 ${ }^{1}$ and 1929-31

| Exact age | Males |  |  | Females |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Admissions per 100,060 population |  | $\begin{gathered} \text { Percent } \\ \text { change } \\ \text { 1919-21 to } \\ 1929-31 \end{gathered}$ | Admissions per 100,000 population |  | $\begin{gathered} \text { Percent } \\ \text { change } \\ \text { 1919-21 to } \\ 1929-31 \end{gathered}$ |
|  | 1919-21 | 1829-31 |  | 1919-21 | 1929-31 |  |
| 15----- | 35 | 27 | -22.9 | 24 | 19 | -20.8 |
| 20 | 78 | 73 | -6.4 | 48 | 48 | 0.0 |
| 25-.. | 95 | 89 | -6.3 | 70 | 64 | -8.6 |
| 30... | 102 | 91 | -10.8 | 87 | 75 | -13.8 |
| 35-...-- | 107 | 102 | -4.7 | 91 | 84 | -7.7 |
| 40.- | 108 | 111 | +2.8 | 94 | 94 | 0.0 |
| 45. | 102 | 116 | +13.7 | 100 | 97 | -3.0 |
| 50 | 99 | 123 | +24.2 | 108 | 100 | -5.7 |
| 55.-. | 106 | 131 | +23.6 | 108 | 105 | -2.8 |
| 60-.----- | 122 | 149 | +22.1 | 111 | 109 | -1.8 |
| 65.-. | 149 | 182 | $+22.1$ | 135 | 133 | -1.5 |
| 70- | 190 | 257 | +35.3 | 167 | 186 | +11.4 |
| 75. | 220 | 355 | +614 | 214 | 265 | +23.8 |
| 80 | 261 | 390 | +49.4 | 280 | 333 | +18.9 |

[^1]Much the same holds true for females in New York State, except that the percentage decrease was smaller; but for males, beginning at age 40 the rates in New York State increased rapidly at each age from 1920 to 1930 (fig. 6). The reason for this is not readily apparent. Examination of the commitment rates for individual psychoses reveals that this increase results largely from an increase in the manic depressive and cerebral arteriosclerotic psychoses.

The seeming contradiction of tables 2 and 3 and tables 4 and 5 is more apparent than real. Owing to the fact that the decrease in mortality rates between 1920 and 1930 permits a larger proportion of the popula-


Figure 6.-Percentage change in the number of first admissions to mental hospitals per 100,000 total population, by age and sex, New York State, 1919-21 to 1820-31.
tion to live to the ages when mental disease most frequently develops, it is inevitable that there should be an increase in the proportion of the total population which eventually is subject to some mental disorder, even though the probability of mental disease at each age remains unchanged. The increase in the expectation of life means that an increased number of persons now live to develop mental disease. Similarly, an increase in mortality rates would result in fewer persons living to develop mental disease, so that the expectation of mental illness at birth would decrease.

The rates of first admissions to mental hospitals in Illinois shown in table 6 are ungraduated average rates for the respective age groups. The rates standardized for age decreased 3.1 percent for males and 0.3

Table 6.-Number of first admissions to mental hospitals per 100,000 population, by age and sex, Illinois, 1922-24 and 1929-31

| Age | Male |  |  | Femsle |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1922-24 | 1929-31 | Percentage change | 1922-24 | 1929-31 | Percantage change |
| 10-14-2. | 2.2 | 0.2 | -90.9 | 2.2 | 1.0 | -54.5 |
| 15-19.... | 35.8 | 28.6 | -20.1 | 20.8 | 20.0 | -3.8 |
| 20-24. | 77.2 | 65.1 | -15. 7 | 42.4 | 37.8 | -10.8 |
| 25-29. | 96.4 | 81.1 | -15.9 | 59.2 | 52.3 | -11.7 |
| 30-34---- | 120.3 | 100.5 | -16.5 | 76.7 | 63.9 | -16.7 |
| 35-39--- | 139.1 | 116.2 | -16.5 | 76.9 | 71.4 | -7.2 |
| 40-44 | 145.9 | 123.8 | -15.1 | 85.4 | 80.4 | -5.9 |
| 45-49 | 125.1 | 123.0 | $-1.7$ | 87.7 | 85.5 | -2. 5 |
| 50-54 | 128.3 | 133.9 | +6. 0 | 89.0 | 79.1 | -11.1 |
| 55-59 | 116.5 | 146.8 | +26.0 | 73.8 | 84.2 | +14.1 |
| 60-64 | 149.0 | 165.4 | +11.0 | 93.0 | 83.5 | -10.2 |
| 65-69 | 175.9 | 216.1 | $+22.9$ | 96.7 | 109.8 | +13.5 |
| 70-74. | 238.0 | 270.3 | +13.6 | 160.4 | 140.8 | -122 |
| 75-79 | 260.8 | 399.5 | +53.2 | 197.3 | 208.3 | +5.6 |
| 80 and over. | 306.6 | 463.8 | +51.3 | 203.3 | 290.4 | +42.8 |
| Total | 83.6 | 83.9 | +0.4 | 51.2 | 50.5 | -1.4 |
| Standardized rate ${ }^{1}$ | 86.6 | 33.9 | -3.1 | 53.3 | 50.5 | -5.3 |

1 Standardized on the basis of the 1930 population of Illinois.


FIGURE 7.-Percentage change in the number of first admissions to mental hospitals per 100,000 total population, by age and sex, Ilinois, 1922-24 to 1929-31.
percent for females during the 7-year period 1922-24 to 1929-31. Substantial decreases occurred until the age group 75-79 for females. The rates for males decreased for all age groups under 50-54 but increased markedly at the older age groups (fig. 7).

From the data in tables 7 and 8 an appreciation can be obtained of the influence of changes in mortality rates upon the incidence of mental disease. The expectation of mental disease based upon first commitment rates for 1929-31 and mortality rates for 1919-21, which assumes that mortality rates remained unchanged during the decade, is shown in these tables. For Massachusetts these figures are about 10 percent less than those computed from the mortality rates of 1929-31 (table 7, column 5). In other words, if mortality rates had remained unchanged during the two periods, the probability of developing mental disease during a lifetime would have decreased 10 percent instead of increasing as it did. The increase in average length of life accounted for the entire increase in the incidence of mental disease.

Table 7.-Chances per 1,000 at birth of being committed to a hospital for mental disease, 1919-21 and 1929-31, compared with corresponding chances based upon the mortality rate of 1919-21 and the commitment rate of 1929-31, by sex, total population, Massachusetts

| Sex | Chances per 1,000 of being committed to a hospital for mental disease, based on- |  |  | Ratio of column 2 to column 1 | Ratio of column 3 to column 1 |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | 1919-21 commitments 1919-21 mortality | 1929-31 commitments 1929-31 mortality | 1929-31 commitments 1919-21 mortality |  |  |
| Male | 55 49 | 57 53 | 49 | 1.04 1.08 | 0.89 0.90 |

Table 8.-Chances per 1,000 at birth of being committed to a hospital for mental disease, 1919-21 and 1929-31, compared with corresponding chances based upon 1919-21 mortality statistics and 1929-31 first admissions, by sex, total population, New York State

| Sex | Chances per 1,000 of being committed to a hospital for mental disease, based on- |  |  | Ratio of column 2 to column 1 | Ratio of column 3 to column 1 |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\begin{aligned} & \text { 1919-21 com- } \\ & \text { mitments } \\ & \text { 191-21 mor- } \\ & \text { tality } \end{aligned}$ | $\begin{aligned} & \text { 1929-31 com- } \\ & \text { mitments } \\ & \text { 1929-31 mor- } \\ & \text { tality } \end{aligned}$ | $\begin{aligned} & \text { 1929-31 com- } \\ & \text { mitments } \\ & \text { 1919-21 mor- } \\ & \text { tality } \end{aligned}$ |  |  |
| Male Female $\qquad$ | 47 44 | 53 48 | 54 46 | 1.13 1.09 | 1. 1.55 1.05 |

In New York State the expectation of life decreased after about age 40 for males and age 60 for females. Consequently, some decrease in the expectation of mental disease would be expected, especially among males, if there had been no increase in the incidence of mental disease. The decrease in mortality rates for females accounted for about one-half of the increase in the expectation of mental disease. The remaining increase resulted from an increase in the age specific commitment rates at the older ages. Since mortality rates actually
increased for males at the ages when the incidence of mental disease is greatest, the entire increase in mental disease arose from increases in the age specific commitment rates.

In general, the data for Massachusetts, New York, and Illinois do not support the contention that the incidence of mental disorders has been rapidly increasing. This does not mean that the public burden of caring for persons with mental disease has not been increasing. The average number of patients in State hospitals increased from 248,852 in 1926 to 347,620 in 1935 . Rather it means that most of the increase may be attributed to increase in the expectation of life, an increasing proportion of old people in the population, increasing urbanization, and similar environmental factors. As the standards of care and treatment are raised, the total cost will increase, although there is no increase in the incidence of the disease.

Even if mental aberration apparently is not increasing appreciably, its importance should not be minimized. Of the $2,144,800$ children born in 1936, from 110,000 to 120,000 will probably be committed to a hospital for mental disease. The loss in future productive power as well as the cost of caring for this number of patients emphasizes the importance of developing a thorough mental hygiene program.

This is all the more important, since the total burden of caring for the mentally ill is almost certain to increase, even though there is no increase in the incidence of mental disease at each age. Changes now underway in the population of the nation presage an increase in the number of mentally ill in the total population because a larger proportion of the population will be in the age groups when mental disease is most frequent (table 9).

Table 9.-Percentage age distribution, total population, United States, 1900, 1930, $1960^{1}$


[^2]An idea of the effect of population changes upon the problem of mental disease can be obtained by applying the age specific first commitment rates of New York State, 1929-31, to the estimated population of the United States in 1960. In an estimated population of about $150,000,000$ in 1960, there would be nearly 135,000 persons committed to mental hospitals for the first time. The number is nearly twice the present number of annual first admissions. An
appreciable increase in the cost of caring for the mentally ill must be expected, even if there is no increase in the incidence of such diseases.

## SUMMARY

It is commonly believed that there has been an alarming increase in mental disease in recent years and that this is in large part the direct result of the stress and strain of a complex urban environment.

Analysis of the number of first admissions to mental hospitals in Massachusetts, New York, and Illinois does not bear out this belief. The number of first admissions per 100,000 population decreased among women under 70 years of age in each State. In Massachusetts the same was true for men. In New York and Illinois commitment rates decreased at the younger ages, but after age 45 or 50 some increase occurred.

Even though the incidence of mental disease has not been increasing as rapidly as commonly supposed, nevertheless mental diseases constitute an important health problem. Unless there is a decrease in the first admission rates to mental hospitals, from 110,000 to 120,000 of the $2,144,800$ infants born during 1936 will eventually be committed to a hospital for mental disease. This number includes only the most severe cases of mental illness and excludes the large number of emotionally unstable and unbalanced persons who should be included in a comprehensive mental hygiene program.

Owing to the increasing proportion of the population in the older age groups, an increase in the number of admissions to mental hospitals is to be expected in the future, since rates of mental disease are highest at the older ages. A continued increase in expectation of life will also tend to increase the number of mentally ill, since more persons will live to the ages when mental disease is most frequent. Assuming that the commitment rates for mental disease for the total United States are no greater in 1960 than they were in New York State during the 3 -year period 1929-31, it is estimated that about 135,000 persons annually will be committed to a mental hospital for the first time. This is nearly twice the present number of first commitments.

## STUDIES ON THE MECHANISM OF EXPERIMENTAL INTRANASAL INFECTION IN MICE*

By Charles Armstrong, Senior Surgeon, United States Public Health Service
The causes which determine that certain common respiratory ailments tend to be at their minimum prevalence during the hot summer and fall months and to increase with the advent of cool weather, while with poliomyelitis and encephalitis of the St. Louis and Japanese types the reverse is true, are largely matters of conjecture.

[^3]Previously reported pathological studies (1) indicate that the intensity of the brain reactions in mice experimentally infected with the virus of St . Louis encephalitis tend to be at a minimum during the warm season of the year or when artificially high environmental temperatures are maintained, and that they tend to intensify at lower temperatures. Notwithstanding these pathological findings, the incidence of death following intranasal inoculation with a uniform dose of St. Louis encephalitis virus "D" did not appear to be significantly different in groups of mice held at temperatures of approximately $95^{\circ}, 70^{\circ}$, and $42^{\circ} \mathrm{F}$., although deaths did tend to occur somewhat earlier at the higher environmental temperatures (table 1). These results, which have been verified many times during our studies, suggest that temperature per se has but little effect upon the susceptibility of groups of mice to intranasally inoculated encephalitis virus. Nevertheless, in man the pathogenic agents of diphtheria (the disease or carrier states), scarlet fever, catarrhal conditions, and the like, apparently do tend to multiply or spread at an increased rate with the advent of cool weather. It further appears reasonable to assume that the factors accountable for the increase in these agents probably exert a similar stimulating influence upon the so-called normal bacterial flora of the nose and throat as well.

With this conception in mind, the writer was led to investigate the possibility that an alteration of the nasal flora, whatever its cause, might exert an influence upon the susceptibility of experimental animals to intranasal infection with certain neurotropic viruses.

## PROCEDURE OF STUDY

The nares of several normal white mice from clean stock were washed out with sterile saline and the pooled washings were cultured in plain broth and on blood agar slants. After 24 to 48 hours, the growth was removed from the agar slants by means of a loop and suspended in saline to a turbidity approximating 500 parts of silica per million. The broth cultures were employed either directly or diluted with equal parts of saline.

These suspensions of nasal bacteria (types undetermined) were given 2 to 4 times intranasally to groups of white mice ${ }^{1}$ at intervals of 2 to 7 days; each mouse was lightly etherized, and 0.03 cc of the suspension was dropped into its nostrils. The mice usually showed no ill effects from the treatment. One to 2 days following the last bacterial instillation, the mice so treated, together with the nonprepared controls, were inoculated intranasally with 0.03 cc of a suspension of encephalitis virus (St. Louis type). One mouse was inoculated from each cage in succession so that the period of time between the begin-

[^4]ning and completion of inoculation of all cages was approximately the same. The identity of the cages was unknown to the administrator of the virus. The animals were observed for 14 days and the date of all deaths was recorded.

By reference to table 1, it may be noted that the various environmental temperatures apparently exerted no noteworthy effect upon the total number of deaths. The prepared groups of mice maintained at temperatures of approximately $95^{\circ}, 70^{\circ}$, and $42^{\circ} \mathrm{F}$. in each instance, however, resisted the infection better than the controls, 63.8 percent of the total prepared animals surviving as compared to 24.7 percent for the controls. Surviving animals of all groups tended to be immune to subsequent intracerebral inoculation of the virus.

Table 1.-Effect of temperature and intranasal instillation on intranasal inoculation of mice with encephalitis virus

${ }_{1}$ Excluded; death too early to be due to virus.

## MECHANISM OF PROTECTION

Cultures from the nostrils of several groups of apparently normal mice have been employed in various tests and the results have been uniformly consistent, with one exception. In this instance, a pathogenic agent was present which killed, with a hemorrhagic pneumonia, many of the mice receiving the culture. It therefore appeared probable that no specific type of organism was involved. However, to test this belief further, a mixed culture, No. 824, was plated and four
distinctly different colonies showing microscopically distinct organisms were cultured in broth. After 24 hours, the various broth cultures were administered intranasally to separate groups of mice, administration being repeated three times at intervals of 3 to 4 days. The virus was given intranasally on the day following the last bacterial instillation. The four cultures and also a fifth, one of Park 8 diphtheria bacilli conveyed a definite degree of protection as shown by a comparison with the control group (table 2). It was likewise found that both bacterial suspensions killed by heating at $158^{\circ}$ to $167^{\circ} \mathrm{F}$. for 45 minutes and the sterile Berkefeld filtrate from broth cultures were effective.

Table 2.-Protective effect of intranasal administration of 5 different broth cultures

| Broth culture used | Broth culture, 0.03 cc intranasally |  |  | $\begin{gathered} 0.03 \mathrm{cc} \\ 1: 1050 \\ \text { dil, } \\ \text { dD } \\ \text { virus, } \\ \text { irtra- } \\ \text { nal } \\ \text { sally } \end{gathered}$ | Deaths, by dates, from encephalitis virus administered following pure strain of broth cultures of organisms |  |  |  |  |  |  |  |  |  |  |  | Num ber sur viving | Percent sur-viving |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Number of mice |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Date-.----.-.-.-.-.-.-.-.-.-- | 5/9 | 12 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 23 | 27 | 28 | 29 |  |  |
| Colony 1 (cocel) .-.-.-...... | 35 | 35 | 35 | 35 |  |  |  | 1 |  |  | 4 | 9 | 1 | 8 |  |  | 17 | 50.0 |
| Colony 2 (becilii) | 35 | 35 | 34 | 34 |  |  |  |  |  |  | 3 | 4 | 5 | 1 |  | 1 | 18 | 52.9 |
| Colony 3 (bacilli) --.--..- | 35 | 34 | 33 | 33 |  | 1 |  | -- | -- |  | 4 | 3 | 4. | 1 |  |  | 20 | 62.5 |
| Colony 4 (cocei and bacilli). | 35 | 34 | 32 | 32 |  |  |  |  | - |  | 4 | 7 | 3 | 1 |  |  | 17 | 53.1 |
| Park 8 culture (broth) | 35 | 35 | 31 | 31 |  |  | - |  |  | 1 | 5 | 4 | 4 |  |  |  | 17 | 54.8 |
| Nonprepared controls.....- | 35 | 34 | 32 | 32 |  | - |  | -- | --- | 4 | 11 | 7 | 6 | 1 |  |  | 4 | 12.5 |

That the presence of bacteria per se in the nasal cavities was not the immediate cause of the protective effect against the encephalitis infection was further clearly shown by the addition of bacteria to the intranasally inoculated virus, when it was found that their presence exerted no protection but, as employed, actually tended to lessen the protection, especially in the non-prepared control groups (table 3).

Table 3.-Effect of adding bacterial suspension to virus


Cook (2) sprayed, simultaneously, cultures of various organisms (strength of suspension not stated) and St. Louis encephalitis virus into the nasal passages of mice and found the pathogenetic quality of the virus unaltered.

It appears, therefore, as though the bacterial suspensions produced their protective action through the effect which they induced in the host and that an interval of time must elapse before the protective effect is apparent. Probably the effect is largely a local one, since groups of animals that have received the bacterial suspensions tend to show but slight protection against intracerebral inoculation of the virus.

## STUDY OF NASAL WASHINGS

Since both living and dead bacteria ( 5,4 ), or their products (5), are known to be chemotactic for leucocytes, an attempt was made to determine the effect of the bacterial instillations upon the number of leucocytes in the nasal washings of control and treated mice.

Each nostril of five prepared and five control mice was washed out each day with 0.5 cc of sterile saline. The recovered washings from each group were pooled and the leucocytes counted, without further dilution, in an ordinary blood counting chamber. The results indicate that the prepared animals respond to the bacterial instillations by a definite outpouring of leucocytes into the nasal cavities. The counts. tend to fall sharply after a couple of days, and then more gradually for several succeeding days (table 4).

Table 4.-Effect of bacterial instillations on leucocytes in nasal washings of control and treated mice

| Preparation | Number of leucocytes, by days, in pooled nasal washings of five test and five control mice |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Interval in days, from the last bacterial instillation to cell count |  |  |  |  |  |
|  | 1 | 2 | 3 | 4 | 5 | 6 |
| Broth culture intranasally. Nonprepared controls. | 1,308 18 | 1,800 52 | 391 18 | 686 25 | 514 20 | 101 49 |

## BACTERIAL INSTILLATION AND TETANUS TOXIN

The effect of the bacterial method of prophylaxis upon the absorption of a fixed dose of tetanus toxin administered intranasally was next investigated, tetanus toxin being incapable of multiplication and not being influenced, so far as known, by the presence of leucocytes. The results indicate that the preliminary intranasal preparations afforded no protection against tetanus toxin; in fact, more of the prepared animals tended to die, and did die earlier than the controls.

The different behavior of tetanus toxin from that noted with encephalitis virus might theoretically be explained by assuming that multiplication of the virus tended to be inhibited in the prepared animals. It would seem to be quite as probable, however, that the results are related to the smaller size, different chemical composition or lack of chemotactic properties of the toxin molecule as compared with the virus particles, or may be explained by: assuming that a different mechanism of absorption applies to the effective portions of the two types of injurious agents.

## BACTERIAL INSTILLATIONS AND INFLUENZA VIRUS

The effect of the bacterial instillations upon the death rate in groups of mice subsequently inoculated intranasally with influenza virus was next investigated.

The earlier trials in which 0.03 cc of the virus, suspension was instilled into the nostrils and the animals jolted while in an inverted position with a view to aiding the spread of the virus, as was our custom with the encephalitis inoculations, indicated that the bacterial treatment had practically no influence upon the death incidence. This did not appear to be peculiar, since influenza in mice invades the lungs. However, when tests were made by instilling the same quantity of virus in 0.03 and 0.01 cc of saline, respectively, into different groups, with the mice being handled gently, it was found that the prepared mice were less affected by the virus than were the controls, especially with the smaller quantity of inoculum (table 5).

Table 5.-Effect of -bacterial instillation on the death rate of mice subsequently intranasally inoculated with influenza virus

| Date.-.----.-...-- | Number of mice given bacterial intranasal prophylaxis |  |  |  | Num- ber of mice given infu- enza virrs intra- nasal- ly | Deaths, by dates. following inoculation with influenza virus intranasally |  |  |  |  |  |  |  |  |  |  |  |  | Total number of death | Percent sur-viving |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | 4/13 | 16 | 19 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 5/1 | 2 | 3 | 4 | 5 | 6 |  |  |
| 0.03 ce 1:2400suspen- sion. | 40 | 40 | 37 | 36 | 36 |  |  |  |  | 3 | 2 | 6 | 3 | 1 |  | 1 | 1 | 1 | 18 | 50.1 |
| 0.01 ec 1:800 suspension. $\qquad$ |  | 39 | 38 | 38 | 35 |  |  |  |  |  |  |  |  |  |  |  |  |  | 1 | 97.1 |
| 0.03 ce 1:2400 suspension (controls) |  |  |  |  | 35 |  |  |  |  | 4 | - | 9 |  |  |  |  |  |  | 29 | 17.1 |
| 0.01 ec 1:800 suspension (controls). |  |  |  |  | 35 |  |  |  |  |  | 1 | 5 | 8 |  |  | 2 |  |  |  | 54.2 |

These results suggest either that there was a tendency for the virus to be prevented from multiplying, for it to be neutralized or destroyed in the nasal passages, or, more probably, in some way for it to be prevented from spreading to the lungs of the prepared mice.

## POSSIBLE INFLUENCE OF LEUCOCYTES AND FIXED TISSUE CELLS

The significance of leucocytes in virus infections is, for obvious reasons, much less understood than is the case in bacterial infections. Observations have, however, been made from time to time which indicate that they may be of some significance in virus ailments. For instance, Landsteiner and Russ (6), in 1906, found the virus of fowl plague present in greater amounts in the cellular elements of the blood than in the plasma, a fact which Todd (7), in 1928, confirmed by showing that the virus was most abundant in the leucocytes. Likewise, Douglas and Smith (8), in 1930, found that leucocytes take up the virus of vaccinia in vitro. By reference to tables 4 and 6 it may be noted that the rather sharp falling off in the percentage of survivors among mice inoculated after the first 48 hours following the last bacterial instillation is paralleled by a simultaneous rapid fall in the leucocytes of the nasal washings, and it is possible that these two occurrences are related. It is conceivable, however, that other elements of the induced exudate may exert a protective influence.

We have attempted to test in vitro mixtures of leucocytes and virus for infectivity, but the results with the methods employed were inconclusive and must be repeated.

There is also the possibility that the fixed cells may be altered by the intranasal instillations in such a way that they also play a part in the increased resistance noted in treated mice. The writer (9, 10) found that the irritation produced in rabbits by means of diphtheria toxin rendered the affected area resistant to vaccine virus and that the insusceptibility was evident after the acute irritation had subsided.

## PATHOLOGY OF THE NARES

In the light of presented facts, the pathology of the nasal cavity of the experimental animals is of interest. A total of 16 mice killed 24 hours after receiving the last of a series of intranasal instillations with a broth culture of mixed organisms and 16 normal controls bave been examined by Surgeon R. D. Lillie. A possible tendency was noted for local cellular infiltration and cellular exudation to be more prevalent in the nostrils of the prepared animals than in the control group. It was not possible, however, for the pathologist to determine whether individual mice in most instances were from the control or test groups.

## DURATION OF PROTECTION

The duration of protection has not been fully explored, but, according to table 6, the protection appeared to be most marked during the first two days following the bacterial treatment, after which it tended
to drop rather sharply and then more slowly, but was still in evidence after the lapse of 5 days.
TABLE 6.-Duration of protection following last intranasal inoculation of bacterial culture

${ }^{1}$ Excluded from calculation as death not due to virus.

## DISCUSSION

The resistance engendered in mice by the intranasal instillation of nonpathogenic bacterial cultures is probably related to the activity of a natural defense mechanism. In the experiments here reported, a huge dose of virus was flooded into the nostrils at one time, and it seems probable that the defense mechanism would be relatively more effective against the small initial infecting dose of virus that conveys a naturally acquired infection. Moreover, if the engendered defense tends to prevent the spread of a virus, as is suggested with influenza, the virus would apparently have a lessened chance of reaching an inaccessible portal of entry, such as the olfactory area of a mouse or man. Thus the individual would tend to be protected from encephalitis infection. Likewise, the opportunity for spread from individual to individual might conceivably be lessened, especially if the virus particles should become fixed to the relatively huge phagocytes.

We must, of course, be careful in reasoning from mice to man, especially since we have failed to engender the protection by natural means, such as changes in the environmental temperature. This failure, however, may be related to the crude method of inoculating the virus, to the tendency of mice to pile up in the cages and thus to protect themselves from temperature changes, or to the fact that the ratio of the mouse's nasal membranes to the cross section of the external nasal openings is tremendous as compared to that in man. However, the observed experimental facts seem to be in harmony with many of the epidemiological peculiarities of certain neurotropic virus infections of man and appear worthy of additional investigation.

## SUMMARI AND CONCLOEIONS

1. Cultures or culture products of nonpathogenic organisms from the nostrils of mice when given intranasally to other groups of mice tend to render them resistant to subsequent intranasal inoculation with St. Louis strain of encephalitis virus.
2. The protection is apparently the result of a nonspecific type of local stimulation and is accompanied by an outpouring of leucocytes.
3. The curve of protection roughly parallels the leucocyte count in the nasal washings and is in evidence for at least five days-longer intervals have not yet been tested.
4. The method affords no protection in mice against intranasally instilled tetanus toxin, but does apparently afford some- protection against influenza virus, provided the dosage is given in a small volume of fluid.
5. The experimental results in mice suggest a possible antagonistic relationship between the bacterial flora of the upper respiratory tract and susceptibility to intranasally inoculated encephalitis virus (St. Louis type).

## references

(1) Lillie, R. D., Dyer, R. E., Armstrong, C., and Pasternack J. G.: Seasonal variation in intensity of brain reactions of the St. Louis encephalitis in mice and of endemic typhus in guinea pigs. Pub. Health Rep., 52: 1805 (1937).
(8) Cook, Enid A.: The effect of simultaneous inoculation with various microorganisms on the pathogenesis of St. Louis encephalitis in mice. J. Inf. Dis., 63: 127 (1938).
(3) Leber, T.: Uber die Entstehung der Entzündung und die Wirkung der entzündungserregenden Schädlichkeiten. Fortschr. Med., 6: 460 (1888).
(4) Gabritschevsky, G.: Sur les proprietés chimiolactiques des leucocytes. Ann. Inst. Pasteur, 4: 346 (1890).
(5) Buchner, H.: Die chemische Reizbarkeit der Leukocyten und deren Beziehung zur Entzündung und Eiterung. Berl. klin. Wchnschr., 27: 1084 (1890).
(6) Landsteiner, K., and Russ, V. K.: Beobachtungen über das Virus der Hühnerpest. Z. Bakt. Ref., 38: 540 (1906).
(7) Todd, C.: Experiments on the virus of fowl plague. Brit. J. Exp. Path., 9: 19 (1928).
(8) Douglass, S. R., and Smith, W.: A study of vaccinal immunity in rabbits by means of in vitro methods. Brit. J. Exp. Path., 11: 96 (1930).
(9) Armstrong, Chas.: Modification of the vaccine response in rabbits by the application of diphtheria toxin to the vaccination site. Pub. Health Rep., 48: 1 (1933).
(10) Idem: Effect of experimental local irritation upon susceptibility to vaccine and encephalitis virus. Ibid., 50: 43 (1935).

## DEATHS DURING WEEK ENDED OCTOBER 22, 1938

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

|  | Week ended Oct. 22, 1938 | Corresponding week, 1937 |
| :---: | :---: | :---: |
| Data from 88 large cities of the United States: |  |  |
| Total deaths ---.- | 7,073 | 18,195 |
|  | - $\begin{array}{r}17,842 \\ .-840,672\end{array}$ |  |
|  | $\cdots$ - 840,672 | 365,175 1493 |
| Average for 3 prior years | 1528 |  |
| Deaths under 1 year of age, first 42 weeks of year | 22, 117 | 23, 524 |
| Data from industrial insurance companies: |  |  |
| Policies in force----1-.-- Number of death claims | $68,203,546$ 13,245 | 79, 003, 13,138 |
| Death claims per 1,000 policies in force, annual rate | 10.1 | 9.8 |
| Death claims per 1,000 policies, first 42 weeks of year, apnual rate........- | 9.3 | 9.8 |

[^5]
## 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 herint oficers.
In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other igure, while leaders (......) represent no report, with the triplication that cases or deaths may have occurred but were not reported to the State helith officer.

Cases af certain diseases reported by telegraph by State health officers for the week ended October 29, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1937 and 5-year median


See footnotes at end of table: .-

Cases of cerlain discases reported by telegraph by State heallh officers for the week onded October 29, 1938, rates per 100,000 population (annual basis), and comparison with corresponding week of 1957 and 5 -year median-Continued


Cases of certain diseases reported by telegraph by State health officers for the week ended October 29, 1958, rates per 100,000 population (annual basis), and comparison with corresponding week of 1957 and 5-year median-Continued


See footnotes at end of table.

Cases of certain discases reported by tolegraph by State heallh officers for the week onded October 20, 1988, rates per 100,000 population (annual basis), and comparison with corresponding week of 1957 and $\delta$-year median-Continued


## New York City only

Period ended earlier than Saturday.
Rocky Mountain spotted fever, week ended Oct. 20, 1938, 5 cases as follows: Illincis, 1; North Carolina, 4.
-Typhus fever, weak ended Oct. 29, 1938, 68 cases as follows: North Carolina, 10; South Carolina, 6;
Georgia, 20; Alabama, 12; Teras, 19; California, 1.

## SUMMARY. OF MONTHLY REPORTS PROM STATES

The following summary of ceises reported monthly by states is published weokly and eovers ealy those States from which reports are recaived during the current weok:

| State | $\begin{gathered} \text { Menin- } \\ \text { gitis, } \\ \text { menin- } \\ \text { sococ- } \\ \text { cus } \end{gathered}$ | $\begin{aligned} & \text { Diph } \\ & \text { theria } \end{aligned}$ | Inflaensa | Ma- | $\begin{aligned} & \text { Meer } \\ & \text { Eles } \end{aligned}$ | Pellagra | Polio-myelitis | Searlet fover | Smanpoz | Ty. phoid and paraty phold fever |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| July 1958 |  |  |  |  |  |  |  |  |  |  |
| Colorado Massachusetts | 8 | 83 88 | 82 |  | 110 |  | 0 | 82 832 |  | 28 7 |
| Soptember 1938 | $\because$ |  |  |  |  |  |  |  |  |  |
| Arizona....-......-. |  |  |  |  | 23 |  |  | 16 | $\because 2$ |  |
| Floride-............-. |  | 82 | 2 | 925 | 80 | 17 | $\bigcirc 2$ | 16 | 0 | 19 |
| Hawail Territory--- |  | 2 | 14 |  | ${ }^{9}$ | -.--- | 1 | 2 | 0 | 9 |
| Kansas....-. |  | 28 87 | 9 |  | 16 | ${ }^{17} 17^{\circ}$ | 1 | 114 | ${ }_{6}^{6}$ | 15 54 |
| Maryland.---.-.-.-- |  | 820 | 9 | 78 | 38 |  | 4 | + 44 | 0 | 54 44 |
| Massachusetts...-.-- | 5 | 10 |  | 8 | 138 | 1 | 4 | 139 | 0 | 30 |
| Nobraska.-........- | 2 | 15 |  |  | 11 | - | 0 | 47 | 29 | 4 |
| Oregon-...........- | \% | 189 | $\begin{aligned} & 19 \\ & 248 \end{aligned}$ | $\begin{aligned} & 8 \\ & 20 \end{aligned}$ | 34 12 | $\cdots$ | 1 5 | 79 89 | $\begin{array}{r}29 \\ \hline 0\end{array}$ | 8 59 |



## September 1958

Ghickenpox:
Arizona
16
Florida
Hawail Territory
Kansas
7
Louisiana
Maryland
Massachusetts
Nebraska
Oregon.
Virginia
Dengue:
Florids
Diarrhea:
Maryland $\qquad$ 102
Dysentery:
Arizona -...-.-.-............ 88
Florida (bacillary)
88
2
Kansas (bacillary)
Louisiana (amoebic)
Louisiana (bacillary)
Maryland (bacillary)...... 145
Massachusetts (bacillary)
Virginia (bacillary)

## September 1988-Continued

Encephalitis, epidemic or
lethargic:
Cases Arizona
Hawaii Territory
Kansas
Massachusetts
Nebraska Virginia.
German measles:
Arizona
Kansas.
Maryland.
Massachusetts
Hookworm disease:
Florida
16....................... 161

Hawaii Territory........... 15 Louisiana 15
4
Impetigo contagioss:
Hawaii Territory.......... 17
Maryland.................... 16
Oregon.
110
Jaundice, epidemic:
Kansas.

Oregon.

## Leprosy:

Hawaii Territory
Louisiana
3
3
Mumps:
Arizona
Florida
Hawaii Territory
Kansas
Maryland
-------- 25
-........- 144
Oregon
Virginia
Ophthalmia neonatorum:
I.ouisiana.-.-...............

Massachusetts_-...........-. 64
Rabies in animals:
Louisiana.
Massachusetts
Oregon
Rocky Mountain spotted fever:
Maryland
Virginia
Scabies:
Kansas
Oregon

September 1958-Continued
Septic sore throat:
Florida
Hawaii Territory..........- 3
Kansas 2
2
Louisiana 10
Maryland............................ 17
Massachusetts.-.--.-...... 6
Nebraska-........-.............. 6
Virginia-....................-.-.-.-. 55
Tetanus:
Florida-...................... 1
Hawaii Territory---...... 1


Maryland-.-.-..............- 3
Massachusetts--.--......-. $\quad 6$
Trachoms:

Hawaii Territory-.-....... 2
Louisiana_-.................... 3
Trichinosis:
Massachusetts_-.......... 1
Tularaemia:
Kansas....................... 2

Typhus fever:
Florida........................ 18
Hawail Territory.--...-.- 6

Undulant fever:
Arizona_-..................... 2

Kansas........................... 15
Louisiana_.................... 9
Maryland.-.......-.-............... 4
Oregon.-....-................. 2
Virginia......-.-.-............ 4
Vincent's infection:
Florida_-.................... 24
Kansas..............-............ 3
Maryland..................... 8
Oregon................................ 7
Whooping cough: $\quad 56$
Florida
Hawail Territory............. 134
Kansas. ....................... 125
Loaisiana.-.-................ 84
Maryland...-...-.............. 107
Massachusetts.............. 323
Nebraska.-.................. 45
Oregon. 45
97


## PLAGUE INFECTION IN GROUND SQUIRREL IN ELDORADO COUNTY, CALIF.

Under date of October 26, 1938, Dr. W. M. Dickie, Director of Public Health of California reported plague infection proved in one Beecheyi squirrel collected October 10, 2 miles southeast of Bay View Resort, Eldorado County, Calif.

WEEKLY REPORTS FROM CITIES
City reports for week ended Oct. 22, 1938
This table summarizes the reports received weekly from a selected list of 140 eities for the purpose of showing a cross section of the current urban incidence of the commnnicsble diseases listed in the table.

| State and city | Diphtheria cases | Infuenva |  | $\underset{\text { Mlos }}{\substack{\text { Mes- }}}$cases | $\begin{aligned} & \text { Pnenen- } \\ & \text { monfa } \\ & \text { deathes } \end{aligned}$ | $\begin{aligned} & \text { Scar- } \\ & \text { foter } \\ & \text { fover } \\ & \text { cases } \end{aligned}$ | $\left\lvert\, \begin{gathered} \text { Small- } \\ \text { pox } \\ \text { cases } \end{gathered}\right.$ | Tuber culosis deaths | Typhoid fever cases | Whoopcoagh cases | $\begin{aligned} & \text { Deaths, } \\ & \text { all } \\ & \text { causes } \end{aligned}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Cases | Deaths |  |  |  |  |  |  |  |  |
| Data for 90 cities: 5-year average.Current week.- | 2236 | 105 80 | 29 | 229 388 | 449 | 880 671 | ${ }^{6}$ | 347 325 | 59 35 | $\begin{array}{r} 811 \\ 1,288 \end{array}$ | --...-- |
| Maine: <br> Portland |  |  |  | 1 | 3 | 1 | 0 | 0 | 0 | 6 |  |
| New Hampshire: ${ }^{\text {- }}$ |  |  |  |  |  |  |  |  |  | - |  |
| Concord.-....-- | 0 |  | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |  |
| Nashua--......-- | 0 |  | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |  |
| Vermont: | 0 |  | 0 | 3 | 0 | 0 | 0 |  | 0 | 8 |  |
| Burlington...- | 0 |  | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |  |
| Rutland...----- | 0 |  | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |  |
| Massachusetts: |  |  |  | 6 | 19 | 22 | 0 | 7 | 1 | 27 | 218 |
| Boston | 0 |  | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 22 |
| Springfield. | 0 |  | 0 | 6 | 1 | 1 | 0 | 0 | 0 | 2 | 34 |
| Worcester... | 0 |  | 0 | 5 | 6 | 0 | 0 | 1 | 0 | 0 | 46 |
| Rhode Island: |  |  |  |  |  |  |  |  |  |  |  |
| Providence.-.--- | 0 | 3 | 1 | 0 | 1 | 5 | 0 | 3 | 0 | 29 | 63 |
| Connecticut: |  |  |  |  |  |  |  |  |  |  |  |
| Bridgeport. | 0 |  | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 24 |
| Hartford | 2 | 1 | 0 | 0 | 0 | 3 | 0 | 5 | 0 | ${ }_{16}^{4}$ | 30 |
| New Haven...- | 0 |  | 0 | 3 | 0 | 1 | 0 |  | 0 | 16 | 35 |
| New York: |  |  |  |  |  |  |  |  |  |  |  |
| Buffalo-..- | 15 | 6 | 1 | 8 17 | 10 | 11 | 0 | ${ }_{71}^{3}$ | 5 | 225 | 136 1,422 |
| Nochester--- | 1 |  | 0 | 3 | 3 | 2 | 0 | 2 | 0 | 0 | 1, 58 |
| Syracuse--------- | 0 |  | 0 | 0 | 6 | 3 | 0 | 0 | 0 | 6 | 53 |
| New Jersey: |  |  |  |  |  |  |  |  |  |  |  |
| Camden----.--- | 0 |  | 0 | 0 | 5 | 4 | 0 | 5 | 1 | 52 | 103 |
| Trenton-----...-- | 0 | 1 | 0 | 0 | 10 | 1 | 0 | 0 | 0 | 0 | 44 |
| Pennsylvania: |  |  |  |  |  |  |  |  |  |  |  |
| Philadelphia-.- | 6 | 11 | 3 | 8 0 | ${ }_{28}^{23}$ | ${ }_{28}^{38}$ | 0 | 32 5 | 1 | ${ }_{20}^{56}$ | 188 |
| Pittsburgh...----- | 11 |  | 3 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 24 |
| Scranton.-.-.-- | 0 |  |  | 0 |  | 1 | 0 |  | 0 | 3 |  |
| Ohio: |  |  |  |  |  |  |  |  |  |  |  |
| Cincinnati----- | 17 | 2 | 0 | 1 |  |  | 0 |  |  | 2 44 | 137 |
| Cleveland.-. | 2 10 | 1 | 0 | 4 | 10 3 | 17 4 | 0 | 10 2 | 0 | 44 0 | ${ }_{82}^{194}$ |
| Toledo-.--.------ | 1 |  | 0 | 1 | 0 | 5 | 0 | 2 | 0 | 0 | 64 |
| Indiana: |  |  |  |  |  |  |  |  |  |  |  |
| Anderson.-- | 0 |  | 0 | 1 | 1 | 0 | 0 | 0 | 0 |  |  |
| Fort Wayne..-- | 0 |  | 0 | 0 | 2 | 6 | 0 | 0 | 0 | 0 | 18 |
| Indianapolis.-.- | 6 |  | 1 | 5 | 11 | 16 | 2 | 3 | 0 | 9 | 82 |
| South Bend.... | 0 |  | 0 | 0 | 0 | $\stackrel{2}{5}$ | 0 | 0 | 0 | 0 | 12 |
| Terre Haute.-.- | 12 |  | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 19 |
| Illinois: |  |  |  | 0 | 0 | 0 | 0 | 0 |  | 0 | 5 |
| Chicago-.-.-....- | 11 | 4 | 2 | 6 | 19 | 88 | 0 | 32 | 5 | 362 | 646 |
| Elgin | 0 |  | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 | 8 |
| Moline. | 0 |  | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 10 |
| Springfield.-. | 0 | --...- | 0 | 0 |  | 1 | 0 |  |  |  |  |

City reports for week onded Oct. 28, 1938-Continued

| State and eity | Diph- <br> theria <br> cases | Influenza |  | Mes8les cases | Pnen monia death | Scar-- let fever cases | 8mall pox cases | Tuber: culosis deaths | Ty. phoid , fever cases | $\begin{gathered} \text { Whoop- } \\ \text { ing } \\ \text { corigh } \\ \text { cases } \end{gathered}$ | $\begin{gathered} \text { Deaths, } \\ \text { sll } \\ \text { causes } \end{gathered}$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Cases | Deaths |  |  |  |  |  |  |  |  |
| Michigan: |  |  |  |  |  |  |  |  |  |  |  |
| Flint | 0 | ------ | 1 | 8 | 16 | 75 | 0 | 16 | 1 | 79 | $\cdots \quad 223$ |
| Grand Rapids.- | 1 |  | 0 | 0 | 2 | 18 | 0 | 1 | 0 | 0 | 27 |
| Wisconsin: | 0 |  |  |  |  |  |  |  |  |  |  |
| Kenosha.-.-...-- | 0 | -....- | 0 | 0 | 0 | 4 | 0 | - 0 | 0 | 5 | 6 |
| Madison------ | 0 |  | 0 | 2 | 0 | 1 | 0 | 0 | 0 | $\because 2$ | - 16 |
| Milwaukee....- | 1 | 1 | 1 | 2 | 2 | 32 | 0 | 0 | 0 | 147 | 85 |
| Racine........-- | 0 |  | 0 | 2 | 0 | 0 | 0 | 1 | 0 | 6 | 15 |
| Superior---.---- | 0 |  | 0 | 1 | 2 | 5 | 0 | 0 | 0 | 3 | 7 |
| Minnesota: |  |  |  |  |  |  |  |  |  |  |  |
| Duluth..-.....-- | 0 |  | 0 | 0 | 4 | 2 | 1 | 0 | 0 | 8 | 20 |
| ... Minneapolis-..-- | 2 |  | 0 | 34 | 9 | 9 | 0 | 1 | 0 | 7 | 107 |
| Iows: |  |  |  |  |  |  |  |  |  |  |  |
| Cedar Rapids.- | 1 |  | - | 0 | -- | 1 | 0 | ------ | 0 | 0 |  |
| : Davenport. .-.- | 3 |  |  | 1 |  | 5 | 0 |  | 0 | 0 |  |
| Des!Moines, | 0 |  | 0 | 0 | 0 | 8 | 0 | 0 | 0 | 0 | 27 |
| Sioux City .-.-- | 1 |  |  | 6 |  | 2 | 0 |  | 0 | 1 |  |
| Missouri: |  |  |  |  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Kansas City ${ }_{\text {- }}$ | 1 |  | 0 | 0 | 11 | 6 | 0 | 4 | 0 | 2 | 95 |
| St. Joseph | 0 |  | - 0 | 0 | - 2 | 0 | 0 | 0 | 0 | 0 | 18 |
| St. Louis.....-. | 6 |  | 0 | 1 | 7 | 16 | 0 | 2 | 0 | 7 | 168 |
|  |  |  |  |  |  |  |  |  |  |  |  |
| . Fargo--...--- | 0 |  | 0 | 116 | ; 0 | 4 | 0 | 0 | 0 | 0 | 10 |
| Grand Forks---- | 0 |  | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 5 |
|  |  |  |  |  |  |  |  |  |  |  |  |
| Aberdeen...-.-- | 0 |  |  | 0 |  | 0 | 0 |  | 0 | 0 |  |
| Sioux Falls.:-- | 0 | -- |  | 1 |  | 2 | 0 |  | 0 | 0 |  |
| Nebraska: |  |  |  |  |  |  |  |  |  |  |  |
| : Kincoln....-...- | 1 |  | 1 | 0 |  | 3 | 0 |  | 0 | 3 |  |
| Kansas: $\quad 0.0$ |  |  |  |  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Topeka......-.-- | 0 |  |  | 0 |  | 3 | 0 |  | 0 | 5 |  |
| Wichita_-.-.--- | 0 |  | 0 | 0 | 0 | 4 | 0 | 1 | 0 | 0 | 28 |
|  |  |  |  |  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Maryland: |  |  |  |  |  |  |  |  |  |  |  |
| Baltimore | 1 | 2 | 1 | 16 | 12 | 11 | 0 | 6 | 3 | 6 | 199 |
| Cumberland.--- | 0 |  | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 9 |
| Frederick...---- | 0 |  | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 4 |
| Dist. of Col.: <br> W ashington | 6 |  | 0 | 0 | 6 | 13 | 0 |  | 0 | 7 | 159 |
|  |  |  |  |  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Norfolk.-.-.-.-- | 0 |  | 0 | 0 | 8 | 3 | 0 | 2 | 0 | 0 | 34 |
| Richmond...--- | 7 |  | 0 | 0 | 3 | 5 | 0 | 1 | 0 | 0 | 52 |
| Roanoke.-...-- | 0 |  | 0 | 0 | 0 | 2 | 0 | 1 | 0 | 0 | 14 |
| West Virginia: <br> Charleston |  |  |  |  |  |  |  |  |  |  |  |
| Charleston. <br> Huntington | 1 |  | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 16 |
| Wheeling-.-.--- | 0 |  | 0 | 0 | 2 | 0 | 0 | 1 | 0 | 0 | 18 |
| North Carolina: |  |  |  |  |  |  |  |  |  |  |  |
| Gastonia.....-.- | 0 |  |  | 0 | -- | 0 | 0 |  | 0 | 0 |  |
| - Raleigh | 1 |  | 0 | 0 |  | 0 | 0 | 2 | 0 | 3 | 9 |
| Wilmington.--- | 1 |  | 0 | 0 | 3 | 1 | 0 | 0 | 0 | 0 | 15 |
| Winston-Salem. | 3 |  | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 17 |
| South Carolina: |  |  |  |  |  |  |  |  |  |  |  |
| Charleston....- | 1 | 7 | 0 | 0 | 4 | 0 | 0 | 1 | 1 | 0 | 20 |
| Florence....-.-- | 0 |  | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 8 |
| Greenville. | 7 |  | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 1 | 9 |
| Georgia: |  |  |  |  |  |  |  |  |  |  |  |
| Atlanta,------- | 10 | 8 | 0 | 0 | 9 | 8 | 0 | 3 | 0 | 1 | 87 |
| Brunswick. | 0 |  | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| Savannah....- | 0 | 13 | 2 | 0 | 3 | 0 | 0 | 0 | 0 | 4 | 30 |
| Florida: <br> Miamj | 0 |  | 2 | 0 |  | 0 | 0 | 3 |  |  |  |
| Tampa | 1 |  | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 4 | 37 25 |
| Kentucky: |  |  |  |  |  |  |  |  |  |  |  |
|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Covington....-- | 1 |  | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 9 |
| Lexington....-. | 1 |  | 0 | 0 | 2 | 2 | 0 | 1 | 0 | 0 | 23 |
| Louisville.....- | 0 | 1 | 0 | 0 | 3 | 15 | 0 | 3 | 1 | 1 | 50 |
| Tennessee: |  |  |  |  |  |  |  | 1 |  |  |  |
| Knoxville | 4 |  | 0 | 0 | 2 |  |  | 1 | 0 |  | 63 |
| Memphis...-.--- | 1 |  | 0 | 0 | - 1 | 4. | 0 | 5 | 0 | 7 | 72 |
| Nashville........ | 0 |  | 2 | 0 | 2 | 8 | 0 | 1 | 0 |  | 61 |

City reports for week ended Oct. 28, 1938-Continued


## FOREIGN AND INSULAR

## CZECHOSLOVAKIA

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


## ITALY

Communicable diseases-4 weeks ended August 14, 1938.-During the 4 weeks ended August 14, 1938, cases of certain communicable diseases were reported in Italy as follows:

| Disease | July 18-24 | July 25-31 | Aug. 1-7 | Aug. 8-14 |
| :---: | :---: | :---: | :---: | :---: |
| Anthrax. | 38 | 38 | 60 | 60 |
| Cerebrospinal meningitis | 15 | 12 | 12 | 16 |
| Chickenpox. | 90 | 118 | 128 | 78 |
| Diphtheria | 316 | 328 | 330 | 366 |
| Dysentery . | 43 | 123 | 116 | 95 |
| Hookworm disease. | 49 | 20 | 61 | 39 |
| Lethargic encephalitis | 3 | 1 | 4 |  |
| Measles.. | 1,357 | 988 | 934 | 747 |
| Mamps. | 114 | 133 | 122 | 73 |
| Paratyphoid fever | 198 | 248 | 275 | 218 |
| Pellagra--.... | 16 | 8 | 8 | 7 |
| Poliomyelitis | 67 | 70 | 67 | 49 |
| Puerperal fever | 17 | 30 | 25 | 25 |
| Rabies--..-- |  |  | 1 |  |
| Typhoid fever | 1,098 | 1,448 | 1477 | 135 1,851 |
| Undulant fever | 1,99 | 1,98 | 1,63 | 1,84 |
| Whooping cough | 697 | 642 | 532 | 348 |

## JAMAICA

Communicable diseases-4 weeks ended October 1, 1938.-During the 4 weeks ended October 1, 1938, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

| Disease | $\begin{gathered} \text { Kings- } \\ \text { ton } \end{gathered}$ | Other localities | Disease | Kingston | Other localities |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Chickenpox. | 1 | 5 | Leprosy. | 1 | 5 |
| Diphtheria | 4 | 2 | Puerperal sepsis |  | 1 |
| Dysentery.- | 6 | 1 | Tuberculosis. | 47 | 68 |
| Erysipelas...--.-. |  | 1 | Typhoid fever. | 3 | 35 |

## 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 Healit Reports for October 28, 1938, pages 1946-1959. A similar cumulative table will appear in future issues of the Public Health Reports for the last Friday of each month.

## Cholera

China.-During the week ended October 22, 1938, cases of cholera were reported in China as follows: Hong Kong, 7, Shanghai, 87.

Indochina (French).-During the week ended October 22, 1938, 7 cases of cholera were reported in Annam Province, and 1 case of cholera was reported in Tonkin Province, French Indochina.

## Plague

Hawaii Territory-Island of Hawaii-Hamakua District-Paauhau Sector.-A rat found on October 14, 1938, and another rat found on October 18, in Paauhau Sector, Hamakua District, Island of Hawaii, Hawaii Territory, have been proved positive for plague.

United States-California.-A report of a plague-infected squirrel in Eldorado County, California, appears on page 2019 of this issue of Public Health Reports.
$x$


[^0]:    ${ }^{1}$ Revision of a paper presented at the Round Table of the American Psychopathological Association, AtJantic City, May 4, 1938.

[^1]:    ${ }^{2}{ }^{2}$ Data for 1919-21 are from H. M. Pollock and Benjamin Malzberg, "Expectation of Mental Disease," Paychiatric Quartorly, October 1928.

[^2]:    ${ }^{1}$ Estimated population for 1960 taken from Population Statistics: I. National Data, National Resources Committee, 1937.

[^3]:    ${ }^{\bullet}$ From the National Institute of Health, Washington, D. O.

[^4]:    1 White mice from various producers were employed-the only precaution being that mice for each test be from a single source.

[^5]:    ${ }^{1}$ Data for 86 cities.

