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## THE RELATIVE AMOUNT OF ILL-HEALTH IN RURAL AND URBAN COMMUNITIES *

By HAROLD F. DORN, *Statistician, United States Public Health Service*

Prior to the date of modern discoveries in preventive and therapeutic medicine and the almost universal adoption of modern principles of sanitation, the wholesale annihilation of a large proportion of the population of large cities during the often-repeated epidemics gave rise to the belief that the open country and small towns were more healthful than cities as places of residence. The annals of medicine and hygiene contain many accounts of high death tolls from the "Black Death" and plague in the large cities of Europe.

Early writers on mortality and political arithmetic referred to cities as the graves of humanity. About 1775 Süssmilch estimated that the annual mortality in the country was from 10 to 30 per 1,000 population, that in small cities it was about 40 per 1,000 population, while in large cities it was at least 50 per 1,000 population (*1*). Sir William Petty stated that, if all the inhabitants of the world were living in cities the size of London and subject to the same mortality conditions prevailing there, the human race would become extinct in a century or two (*2*).

Even as recently as 1874, John Stockton-Hough wrote that the mortality in cities in the United States and Europe was more than twice that of country districts (*3*). The classic example of attempts to state the relative mortality of rural and urban residents in the form of a natural law is the statement of William Farr, the eminent English vital statistician, that the rate of mortality increased as the eighth root of the density of population (*4*).

During the past two generations, however, developments in medicine, sanitation, and public health have achieved remarkable success in lowering the mortality rate from many causes of death. For example, yellow fever, typhus, cholera, and plague have now practically disappeared as causes of death in the United States, although they still are only partially checked in many parts of the world.

* Revision of a paper presented at the Congress of the International Union for the Scientific Study of Population Problems, Paris, France, July 1937.

It is too often forgotten that the successful control of these diseases has occurred within the memory of people still living. Even as recently as 1890, the death rate from all causes in New York City was more than 25 per 1,000 population, as compared with the present rate of about 11 per 1,000 population (fig. 1).

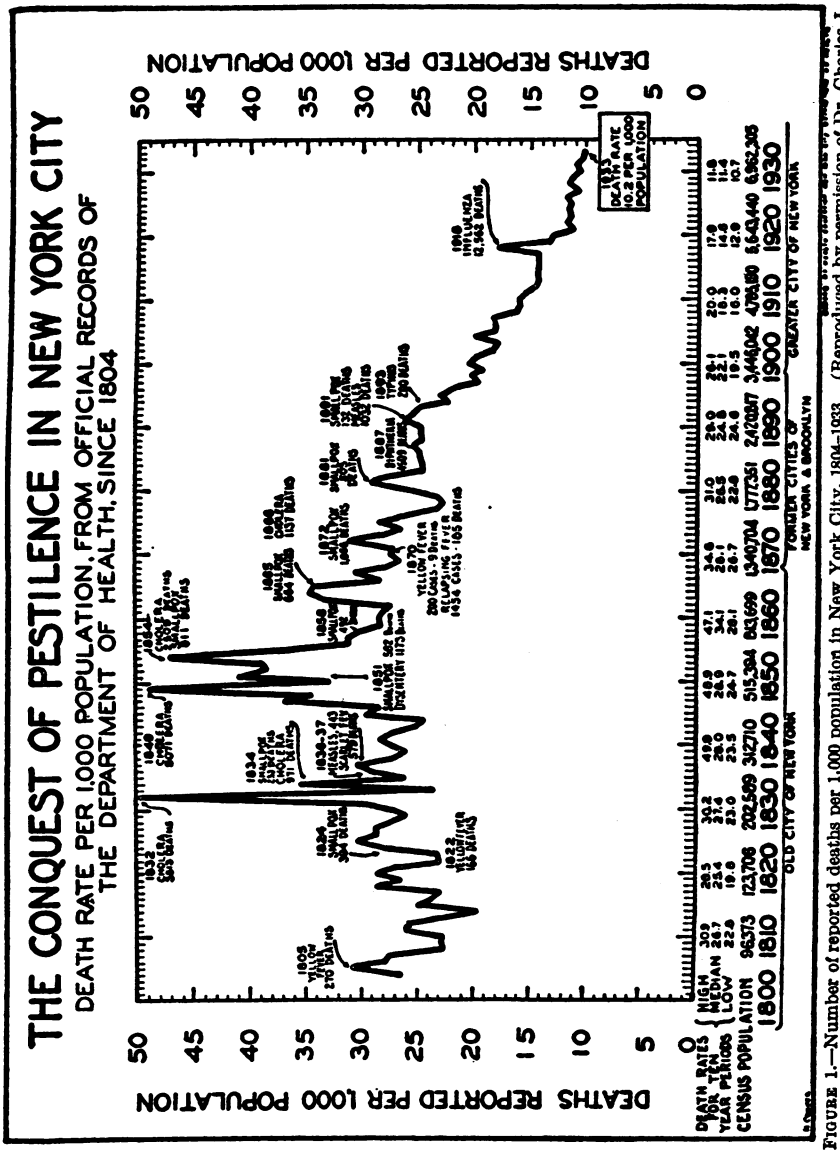


FIGURE 1.—Number of reported deaths per 1,000 population in New York City, 1804–1933. (Reproduced by permission of Dr. Charles L. Bolduan, New York City Department of Health.)

The benefits of these developments have accrued especially to urban dwellers since physicians, nurses, dentists, and hospitals have concentrated in cities. The problems of congestion forced urban communities to adopt modern methods of sewage disposal, water puri-

fication, inspection of milk and other foods, and to provide full-time health service. Similar services have been extended very slowly to rural areas. At the beginning of 1937 only 42 percent of the rural population was served by a local health unit under the direction of a full-time health officer, and in 10 States no service of this kind was available to any part of the rural population (*5*). It would not be surprising if mortality conditions in cities were relatively better than those in the country, and indeed such is the opinion of many people.

One writer, noticing the remarkable success achieved in controlling communicable diseases in cities, has predicted that the expectation of life at birth of urban residents would be about 100 years by 2000 A. D. (*6*). In view of such statements it is desirable to examine the available evidence in order to determine whether or not people living in cities are now more healthy than persons living in the open country.

Ample evidence indicates that the unequal distribution of the benefits of modern sanitation and medicine is altering the relative healthfulness of city and country residents when measured by mortality rates. Between 1900 and 1930 the death rate from typhoid fever decreased about 90 percent in urban areas, but only 75 percent in rural areas. At the present time this disease takes relatively more than twice as many lives in rural areas as in cities. Infant mortality decreased 44 percent in urban areas and 34 percent in rural areas from 1915 to 1934. An infant is more likely to die before completing the first year of life if born to parents living in rural areas, although this varies widely throughout the country. However, mortality from the diseases of adult life is still appreciably lower among rural than among urban residents.

The increase in expectation of life at birth since 1900 has been about 60 percent greater among persons living in urban than among persons living in rural communities (fig. 2).[1] At all ages except 10 to 20 among females, mortality decreased more rapidly in the urban than in the rural population (*7*). The greater occupational risks of urban males, however, are revealed by the fact that the advantage of rural residents in expectation of life is from one and one-half to two times greater for males than for females. At the present time white infants born in the country may expect to live about 5 years longer than white infants born in the city if they are boys and about 4 years longer if they are girls (fig. 3). In spite of the more rapid decline in mortality in urban communities since 1900, rural males subject to the mortality conditions of 1900–1902 had a greater life expectancy at all ages over 1 year than did urban males 30 years later. In other words, the remarkable gains in the preservation of life during the past

[1] In 1930 all places of 10,000 or more inhabitants were classed as urban. In 1900–1902, all places of 8,000 or more inhabitants were classed as urban. The data for 1900–1902 are for the original registration States, while those for 1930 are for the entire United States exclusive of Texas.

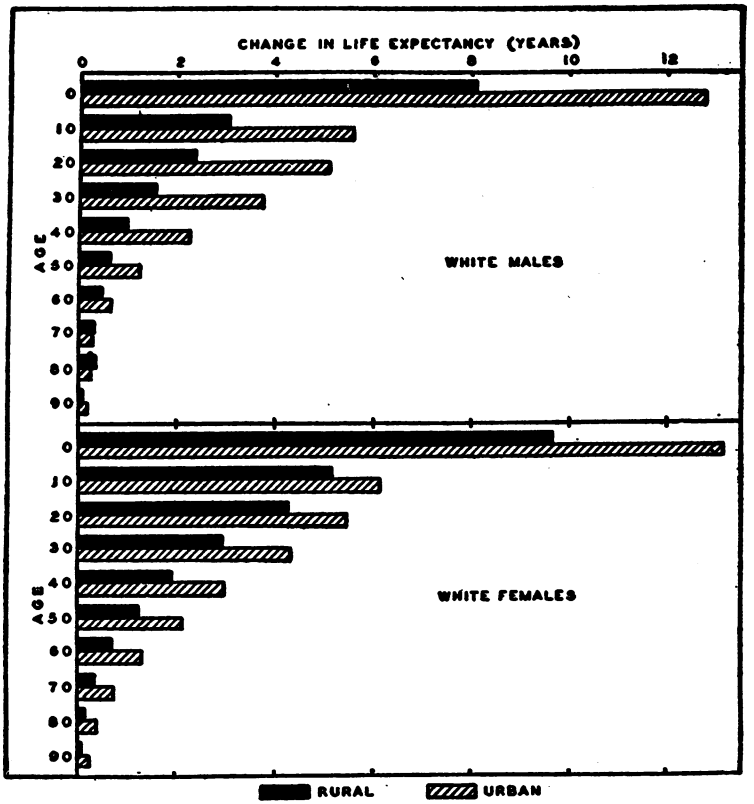


FIGURE 2.—Number of years change in expectation of life at selected ages, by sex, among white persons in rural and urban communities, 1900–1902 to 1930. (Data for 1930 are from Dublin and Lotka (7).)

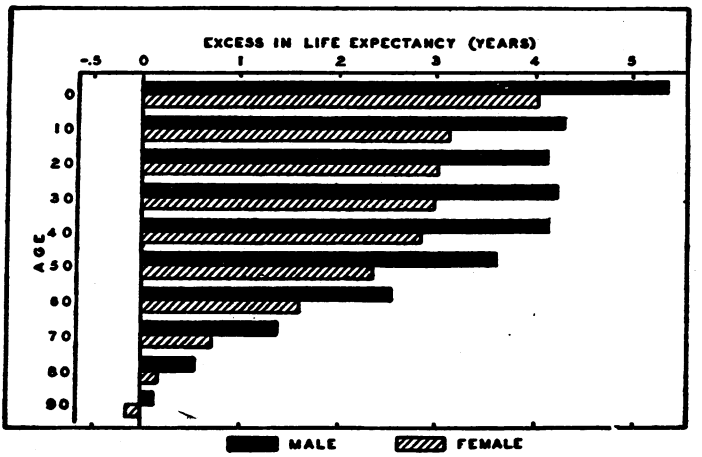


FIGURE 3.—Number of years excess in expectation of life of rural over urban residents at selected ages, white population, male and female, United States, 1930. (Data are from Dublin and Lotka (7).)

generation have merely advanced the urban population to the level of life expectancy attained by the rural population at the beginning of the century. The difference between females is less than that between males; but even so, white women between 30 and 80 years of age living in urban communities in 1930 could not expect to live as many years as rural women of the same ages in 1900.

These differences in mortality between urban and rural residents are somewhat overstated, since deaths were not allocated to the place of residence of the deceased.[2] In general, urban mortality rates are increased and rural mortality rates are decreased by this procedure, but the magnitude of the error varies widely from place to place. For two States, Ohio and New York, with a combined population of 19,000,000 persons in 1930, mortality statistics tabulated according to the usual residence of the deceased are available for different sized communities (tables 1 and 2).

TABLE 1.—*Number of deaths per 1,000 total population in different sized communities, New York State, 1929–31*

| Size of community | Crude rate | Standardized rate [1] | Ratio to rural rate [2] |
|---|---|---|---|
| Rural [3] | 13.9 | 11.0 | 100 |
| Urban [3] | 12.1 | 11.4 | 104 |
| 250,000 and over | 11.3 | 12.7 | 115 |
| 100,000–250,000 | 11.5 | 11.1 | 101 |
| 50,000–100,000 | 11.1 | 11.0 | 100 |
| 25,000–50,000 | 12.6 | 11.1 | 101 |
| 10,000–25,000 | 12.4 | 11.3 | 103 |
| 2,500–10,000 | 13.8 | 11.9 | 108 |

[1] Standardized on total population of New York State in 1930.
[2] Standardized rate.
[3] Exclusive of deaths in institutional districts.

TABLE 2.—*Number of deaths per 1,000 native-white population by sex in different sized communities, Ohio, 1930*

| Size of community | Crude rate | | Standardized rate [1] | | Ratio to rural rate [2] | |
|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female |
| Rural [3] | 11.0 | 10.5 | 8.7 | 8.3 | 100 | 100 |
| Urban [3] | 9.9 | 8.8 | 10.4 | 8.6 | 120 | 104 |
| 100,000 and over | 9.3 | 8.1 | 10.7 | 8.7 | 123 | 105 |
| 10,000–100,000 | 10.3 | 9.2 | 10.3 | 8.5 | 118 | 102 |
| 2,500–10,000 | 11.7 | 10.7 | 10.0 | 8.4 | 115 | 101 |

[1] Standardized on the population of England and Wales 1901.
[2] Standardized rates.
[3] Exclusive of deaths in institutions.

[2] The number of recorded and resident deaths in 1935 for cities of 100,000 or more population in 1930 is given in Vital Statistics–Special Reports, Vol. 3, No. 44, of 1937. Similar data for 1935 and 1936 for the rural area of each county and for each city of 10,000 or more population in 1930 are contained in Vital Statistics–Special Reports, Vol. 4.

The possibility of less complete registration of deaths in rural than in urban areas is another source of error that should not be disregarded.

In both States, mortality rates are definitely higher in urban than in rural areas and are highest of all in the large cities. In Ohio, the standardized death rate increases regularly with the size of the community. In the urban population of New York State, mortality rates are highest in the small towns of less than 10,000 population and in the two largest cities, New York and Buffalo. The rates in the medium sized cities are only slightly larger than the rural rates.

But mortality rates measure only the amount of fatal illness in the population. Many enervating and enfeebling diseases, such as pellagra, malaria, and hookworm, are relatively unimportant as causes of death. Improper nutrition is reflected in listlessness and lack of energy rather than in deaths. A considerable proportion of ill-health which is manifested in discomfort, in decreased vigor and efficiency, and even in sickness and suffering is not revealed by mortality statistics.

Almost no extensive data concerning the incidence of non-fatal illness in rural and urban areas are available. Special studies have been made in a few localities based on reports of diseases treated by physicians; but these give no indication of the number of illnesses that are unattended by physicians, or of the proportion of the population which is ill during any period of time (*8*) (*9*). A few surveys have been made in rural areas, but these have usually obtained only a small proportion of the total number of illnesses (*10*) (*11*).

Rather extensive surveys have been carried out to determine the prevalence on a given day of various causes of illness in families of insured persons (*12*). Nearly all of these were in urban communities.

The first study of the incidence of illness over a period of time was the Hagerstown Morbidity Study, which involved a series of visits to about 1,800 families during a 28-month period (*13*). These families, however, all resided in an urban community. From 1928–31, the Committee on the Costs of Medical Care observed about 9,000 families for a 12-month period; these families resided in different sized communities in 18 States (*14*). The data from this study furnish the most comprehensive record of the incidence of illness over a period of time in the general population. Other papers have summarized the amount and the specific kind of sickness for the entire population studied (*15*). Some of the variations in the incidence of illness in different sized communities will be summarized here.

Since the study was conducted in cooperation with State and local health officials, the localities selected for study were those where the local health departments would give a part of the time of one or more nurses to collect the information. Although the nurses were permitted to select the families to be studied, they were instructed not to

include families regularly visited, but to choose a new group without respect to the presence or absence of illness in the household at the time of the initial visit.

The data were collected by a series of visits to the home of each family at intervals of from two to four months during the course of a year. A few families were visited less frequently; but most families were visited five or six times during the year. Occasional households received as few as four visits and others as many as eight, in addition to calls to check up incomplete records. A record of all cases of illness occurring since the last visit was obtained by the investigator, together with the cause, date of onset, duration, severity, and detailed facts about the nature of the medical care received.

The present study is based on data for 47,575 individuals most of whom were under observation for the entire 12 months. Those observed for only part of the period include births, deaths, and persons who because of marriage, separation, or other reasons left or entered a family during the year of study. The experience of the entire group is equivalent to 42,780 full-time person-years of life.

Although each family was observed for 12 consecutive months, the date of observation varied from February 1928 to June 1931. The families resided in 130 localities scattered throughout the country. The Northeast and the South were somewhat under-represented and the Pacific coast was somewhat over-represented. When compared with the residence of all families of the United States, the percentage of surveyed families was larger in cities of 100,000 population and over and in towns under 5,000 inhabitants but considerably smaller in the open country. The families also had more children, fewer old persons, and more females than the total population of the country.[3]

An illness was defined as any disorder which persisted for one or more days. In practice, the illnesses reported to the nurse necessarily depended upon the informant's (usually the housewife's) conception of an illness and upon her memory. Since repeated attacks of the same disease were considered as separate illnesses, the data refer to illnesses rather than to cases of disease. However, the number of second attacks of the same diagnosis was practically negligible.

A continuous period of sickness was considered as one illness, even though more than one diagnosis was reported. However, in order to have a complete record of all attacks when studying specific diagnosis, a supplementary record was made of all contributory causes.

[3] Additional details concerning the composition and characteristics are given in Causes of Illness in 9,000 Families Based on Nation-Wide Periodic Canvasses, 1928-31. By Selwyn D. Collins. Public Health Reports, 48: 12 (March 24, 1933).

Since the information was secured from the family, the cause of illness represented the informant's diagnosis. Reports on cases attended by a physician were submitted to him for verification or correction. Replies were received for 64 percent of such cases, which represented about 51 percent of all the cases of illness.

Causes of illness were classified according to the International List of the Causes of Sickness and Death (1920 revision) with subdivisions of many categories. More than one diagnosis was reported for about 4 percent of the illnesses. In such cases, one diagnosis was selected as the primary cause of illness in accordance with the following rules:

1. The first cause in order of occurrence applies largely to acute conditions with common complications, such as influenza and pneumonia, measles and otitis media, scarlet fever and nephritis.

2. Acute conditions ordinarily were given preference over an attack of some chronic condition; thus, in a case of grippe and chronic rheumatism, grippe was considered primary.

3. The condition or disease most specifically associated with the period of sickness was preferred over a minor condition which preceded or accompanied it. For example, in a case of illness due to a tooth abscess and rheumatism, the latter was selected as the primary cause of illness. In doubtful cases, the more serious condition was chosen.

4. The more specific diagnosis was selected in preference to a symptom.

5. When none of these rules could be applied, and the record gave no basis for decision, the condition mentioned first by the informant was made primary.

It is certain that an appreciable proportion of mild respiratory, digestive, and other ailments were forgotten and consequently were not reported by the family; but the reports of disabling illnesses, that is, illnesses serious enough to cause loss of one or more days from school or the usual occupation are probably reasonably complete.

The proper measure of morbidity is not so easily defined as the proper measure of mortality. Mortality is commonly measured by the ratio of the number of persons dying in a specified length of time to the number exposed to the risk of death during the same interval. An illness rate formed in the same way has many obvious disadvantages, although it is useful for certain purposes. According to this rate, two populations would have the same illness rate if every person in the first population were sick but once during a year with each illness lasting for a period of 6 months and every person in the second population were sick just once during the year with each illness lasting only 1 week on the average. The illness rate in each population would be 1,000 per 1,000 persons observed, but few people would say that this was an adequate expression of the *amount* of illness in the two populations.

Insurance companies have frequently used a rate based upon the ratio of the number of weeks of sickness to the number of persons alive at the beginning of the period of observation. In a population of constant size, with no entries or withdrawals, this rate is a fairly satisfactory measure of the amount of illness. In practice, however, these conditions are rarely obtained, since births, deaths, and migration, and other losses of families constantly change the size of a population under observation for a period of time such as one year. This difficulty is easily avoided by computing the sickness rate from the ratio of the total number of days or weeks ill to the total person days or weeks observed. In other words, this rate is the percentage of the time which a given population is ill.

Actually it is almost impossible to collect accurate data for the computation of such a rate. For instance, when does a person become ill and when does he recover? For the majority of illnesses this is almost impossible for the average person to determine. Even if the definition of illness is restricted to include only cases necessitating absence from work or school, the data are still likely to be unsatisfactory, especially for housewives. Furthermore, absence from the usual activity is conditioned by many factors besides the severity of the illness.

Even though the reported cases of illness are not strictly accurate, because of forgetfulness, they undoubtedly are more satisfactory than data concerning duration. The measure of illness used hereafter will be the number of reported cases of illness per 1,000 person-years of observation. As such it may properly be termed the "case rate of illness."

In the entire population, there were 856 cases of illness per 1,000 persons under observation for the entire 12 months [4] (table 3). The lowest rate, 787, was reported in the open country and the highest rate, 937, in the villages and towns of less than 5,000 inhabitants. Excluding persons living in the open country, the illness case rate increases as the size of the community decreases, being lowest in the large cities of 100,000 or more inhabitants and highest in the towns of less than 5,000 inhabitants. The difference between the rates in the large cities and the open country is too small to be significant. The same relative rank exists after differences in age distribution are eliminated by the process of standardization.

[4] This is slightly different from the figure given in previous papers, 850, since cases among families observed for only part of a year, which were excluded in previous papers, have been included.

TABLE 3.—*Number of cases of illness per 1,000 person-years exposure by sex in different sized communities* [1]

| Size of community | Both sexes | | Male | | Female | |
|---|---|---|---|---|---|---|
| | All cases | Disabling cases [2] | All cases | Disabling cases | All cases | Disabling cases |
| | Crude rates | | | | | |
| All communities | 856 | 532 | 776 | 485 | 935 | 578 |
| Rural (open country) | 787 | 474 | 731 | 443 | 849 | 508 |
| Less than 5,000 population | 937 | 574 | 859 | 527 | 1,011 | 620 |
| 5,000–99,999 population | 906 | 574 | 828 | 528 | 983 | 620 |
| 100,000 or more population | 809 | 507 | 713 | 451 | 899 | 558 |
| | Standardized rates [3] | | | | | |
| All communities | 856 | 532 | 776 | 485 | 935 | 528 |
| Rural (open country) | 771 | 467 | 715 | 435 | 834 | 504 |
| Less than 5,000 population | 937 | 578 | 864 | 530 | 1,004 | 620 |
| 5,000–99,999 population | 909 | 578 | 833 | 531 | 967 | 624 |
| 100,000 or more population | 803 | 502 | 708 | 446 | 888 | 552 |

[1] Sole or primary cases only.
[2] Causing loss of 1 or more days from school or occupation whether or not gainfully employed.
[3] Standardized by the indirect method, using the age specific case rates of illness for all communities combined.

TABLE 4.—*Number of cases of illness by sex and size of community*

| Size of community | Both sexes | | Male | | Female | |
|---|---|---|---|---|---|---|
| | All cases | Disabling cases | All cases | Disabling cases | All cases | Disabling cases |
| All communities | 36,627 | 22,762 | 16,256 | 10,162 | 20,371 | 12,597 |
| Rural (open country) | 6,156 | 3,711 | 2,897 | 1,757 | 3,259 | 1,954 |
| Less than 5,000 population | 8,250 | 5,059 | 3,665 | 2,249 | 4,585 | 2,810 |
| 5,000–99,999 population | 9,915 | 6,285 | 4,465 | 2,848 | 5,450 | 3,437 |
| 100,000 or more population | 12,306 | 7,704 | 5,229 | 3,308 | 7,077 | 4,396 |

A somewhat more accurate comparison can be obtained by using cases of disabling illness. Although there is some individual variability in reaction to illnesses severe enough to be considered disabling, it undoubtedly is considerably less than the variability for nondisabling illness. About 60 percent of total cases of illness in each community were disabling.

The elimination of cases of nondisabling illness does not appreciably alter the relative rank of the different communities except for the fact that the cities of 5,000 to 100,000 inhabitants and the towns of less than 5,000 inhabitants have identical rates. This rate, however, is higher than the corresponding rate in the large cities and in the open country. The standardized rates present substantially the same picture.

The high case rate of illness in the small towns is contrary to prevailing opinion concerning the relative healthfulness of different sized communities. The idea that cities are unhealthful and that the larger a city the more unhealthful it is, still exists and influences discussions of the subject. It is possible that the difference in observed rates are due entirely to the particular communities studied and do not correctly represent the situation in the entire country. On the other hand, they agree, in general, with the death rates in New York State. There is some reason for believing that, owing to lack of adequate medical and health facilities and service, small towns may have higher morbidity and mortality rates than larger cities. The available data are not sufficient definitely to establish this fact, but they do suggest that it is possibly true.

The case rates of illness for females are of the same general order in the different sized places as the case rates for the total population. For males, the relative position of the open country and large cities of 100,000 or more inhabitants is interchanged; the rate for the latter being slightly but not significantly lower than that for males living in the open country.

The case rate for both total and disabling illnesses is higher for females than for males, varying from an excess of about 20 percent in the open country to an excess of about 33 percent in the large cities. It is interesting that this is directly contrary to mortality rates, which are almost uniformly higher for males than for females. Of course, part of this difference in illness rates may arise from the fact that the housewife was usually the informant and tended to remember more of her minor illnesses than of her husband's. However, the same differences exist when cases of nondisabling illness are eliminated. Moreover, the records of industrial concerns show that women are absent more frequently than men (*16*). Records of a number of industrial sick-benefit associations and company relief departments reporting to the Public Health Service indicate that sickness and nonindustrial injuries are from 60 to 70 percent more frequent among women than among men (*17*). The available evidence indicates that, although the case rate of illness is higher among women, the case fatality is higher among men.

Mortality and morbidity statistics are but two of the many possible measures of ill-health in different communities. Another closely related measure is the relative amount of physical defects and impairments. Such data are inadequate at present and will not be discussed here.

For similar reasons, the adequacy of nutrition in rural and urban communities will not be discussed. Although there is some evidence that nutrition as measured by various indices such as growth rate, height-weight index, and diet is at least as adequate or even more so in urban than in rural communities, a detailed discussion should be

delayed until more comprehensive studies now in progress are completed.

Although ill-health is frequently used only in reference to physical well-being, mental disease should be included in any consideration of the general health of a population. The relative amount of mental disease in a population must necessarily be measured by the rate of admission to hospitals until other methods of detecting mental ill-health are generally available. The rate of admission, however, depends on the availability of hospital facilities, family attitudes, facilities for home care, and the general community environment. It is fairly obvious that persons with mental ailments can be cared for in their own homes more easily in rural than in urban communities. Furthermore, many persons who are regarded as merely "queer" in a rural community undoubtedly would be unable to adjust themselves to the more complex city environment. For these, and other reasons, rates of mental illness would be expected to be higher in urban than in rural communities, even if there were no real difference.

Reports of the Bureau of the Census show that not only is the rate of first admissions to hospitals for the insane greater for urban than for rural residents, but also that the rate increases with the size of the community (*18*). Since part of these differences may be due to variability in the availability of hospital facilities, it is desirable to restrict the comparison to areas where such facilities have been fairly adequate for several years. The data for New York State satisfy these requirements.

In general, the relative ranking of various sized communities in New York State confirms the report for the entire country prepared by the Bureau of the Census (*19*). The rate of first admission is about 66 percent larger for urban than for rural residents. Although there are some irregularities, the rate is also larger in cities of more than 100,000 inhabitants than it is in the smaller cities (table 5).

TABLE 5.—*Standardized rates of first admission for mental disease per 100,000 population to civil State hospitals in different sized communities of New York State during the three years ending June 30, 1931* [1]

| Size of community | Rate | | | Ratio (rural=100) | | |
|---|---|---|---|---|---|---|
| | Total | Male | Female | Total | Male | Female |
| New York State | 95 | 104 | 84 | 153 | 160 | 145 |
| Cities 200,000 and over: | | | | | | |
| New York | 105 | 115 | 94 | 169 | 177 | 162 |
| Buffalo | 93 | 104 | 80 | 150 | 160 | 138 |
| Rochester | 100 | 106 | 91 | 161 | 163 | 157 |
| Syracuse | 101 | 119 | 83 | 163 | 183 | 143 |
| Cities 100,000 to 200,000 | 107 | 113 | 99 | 173 | 174 | 171 |
| Cities 25,000 to 100,000 | 98 | 119 | 84 | 158 | 183 | 145 |
| Cities 10,000 to 25,000 | 88 | 98 | 77 | 142 | 151 | 133 |
| Cities 2,500 to 10,000 | 88 | 100 | 74 | 142 | 154 | 128 |
| Total urban | 103 | 114 | 90 | 166 | 175 | 155 |
| Total rural | 62 | 65 | 58 | 100 | 100 | 100 |

[1] Population of New York State age 15 years and over on April 1, 1930, taken as standard.
Data from reference (*19*).

The differences are uniformly greater among males than among females, the urban rate exceeding the rural rate by 75 percent for males and by 55 percent for females. Even allowing for differences in utilization of hospital facilities, it is apparent that the rate of first admissions is greater in urban than in rural communities and is greater in the large than in the small cities.

Rates of mental disease are commonly supposed to be higher among foreign-born than among native-born persons. Consequently it might be supposed that the higher rates of mental disease reported in urban areas result from the fact that foreign-born persons make up an appreciable proportion of the population. If the rates of first admissions for mental disease of native- and foreign-born persons are standardized for differences in age distribution, it is seen that the higher rate among the foreign-born is due largely to the fact that a large proportion of the population is in the older age groups where the incidence of mental disease is high. Although the crude rate of first admissions for mental disease is twice as high for foreign-born as for native whites, the standardized rates are less than 10 percent higher (*20*).

Regardless of the manner in which ill-health is measured, rural residents possess definite advantages over urban residents. For a few diseases, the superior medical and health facilities and services available to city dwellers have partially compensated for unhealthful environmental conditions. If equivalent care and attention were as readily available to rural residents, it is very probable that the existing differences would be increased.

## SUMMARY

The widespread adoption of modern principles of sanitation and the application of recent discoveries in preventive and therapeutic medicine have given rise to the belief that the health of urban residents is now as good as or even better than the health of persons living in the country. This is contrary to the former belief that cities were "the graves of humanity."

Although mortality rates have decreased more rapidly in urban than in rural areas since 1900, rural males subject to the mortality conditions of 1900 had a greater expectation of life at all ages over 1 year than did urban males in 1930. The difference between females is less, but even so, white women between 30 and 80 years of age living in urban communities in 1930 could not expect to live as many years as rural women of the same ages in 1900.

A comparison of case rates of nonfatal illness in different sized communities shows that the lowest rates occur among people living in the open country and in the large cities of 100,000 or more population. The rates for both total and disabling cases of illness are slightly

but not significantly lower among persons living in the open country than among persons living in the large cities.

Case rates are appreciably higher among people living in cities of less than 100,000 population and in small towns and villages than among people living in the open country and in large cities.

The distribution of the incidence of mental illness is, in general, similar to that of physical illness. In New York State, where hospital facilities for mental disease are fairly adequate, the rate of first admissions is about 66 percent higher for urban than for rural residents. Although there are some irregularities, the rate is higher in the large than in small cities.

Regardless of the way in which ill-health is measured, rural residents possess definite advantages over urban residents. For a few diseases, the superior medical and health facilities and services available to city dwellers have partially compensated for unhealthful environmental conditions. This is especially true for the communicable diseases and those causing infant deaths. However, for most causes of illness, especially fatal illness, rural residents still have definitely lower rates than urban residents in spite of the superior medical facilities available to the latter.

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## THE PREVALENCE OF UNDULANT FEVER (BRUCELLOSIS) IN THE UNITED STATES

About the middle of the last century, during the Crimean War, British medical officers were baffled by an obscure fever which afflicted troops quartered in the Mediterranean area. After long investigation it was discovered that this fever was caused by an organism present in raw goats' milk. The name of Malta, or Mediterranean, fever was given to the disease.

A few years later a Danish veterinarian named Bangs discovered that an organism which he called *Bacillus abortus* was responsible for the disease of cattle known as contagious abortion. It was not until 1918, however, that Miss Alice Evans of the United States Public Health Service showed that these two organisms were closely related. As soon as this was known, it became apparent that there undoubtedly were many cases of undulant fever, as the disease had come to be called, in the United States.

Shortly after the return of the United States Army from the Philippines, a few cases of undulant fever were reported; but except for occasional cases arising in the goat-raising areas of Texas, Arizona, and New Mexico, the disease was practically unknown in the United States. In 1922 an epidemic of undulant fever broke out in Phoenix, Ariz.; and since that date, an increasing number of cases has been reported annually in the United States.

It is not surprising that undulant fever, or brucellosis, as it is now called, escaped attention for so many years. The manifestations of the disease are manifold, and even acute cases are often not diagnosed correctly.

Brucellosis is caused by infection with a specific bacterium of the genus *Brucella*. These bacteria may infect various kinds of domestic animals as well as man. In this country cattle, hogs, and goats are of importance in transmitting the infection to man.

The disease is prevalent in cattle everywhere, though it is more common in some localities than in others. The incidence varies from 10 to 30 percent, with an average of about 14 percent.

On the other hand, the danger of contracting brucellosis from hogs and goats varies greatly in different sections of the country. The disease in hogs is not evenly distributed. In the middle western States, it is common, whereas in certain other sections of the country the hogs are almost or quite free from it. The danger of contracting the disease from goats is greatest in the Southwest, because there the goat-raising industry is extensive. In certain other sections of the country, there are few goats.

The farmers and veterinarians who handle infected animals, and the slaughterhouse workers who handle infected carcasses, may become infected through the skin, particularly through abraded skin. According to our present knowledge, the only way to prevent infections in these occupational groups is by the control of the disease in domestic animals.

There are several varieties of the infecting organism. In this country there are three important varieties, each with a special affinity for one of the three species of animals concerned in transmitting the disease to man. All three varieties may infect man, but the variety which most commonly infects cattle is less virulent for man than are the varieties which infect goats and hogs. The situation is complicated, however, because in localities where cattle are associated with infected goats or hogs, they may become infected with the caprine or porcine variety of the causative organism, and transmit it to man with all its original virulence.

The nature and duration of brucellosis in man are extremely variable—from a mild ambulatory disease to severe illness, and from a few days to many years. A mild attack may be mistaken for influenza, or pass without any attempt at diagnosis. If the disease is more severe, the original diagnosis may be tuberculosis, typhoid fever, malaria, or rheumatism, according to which of those diseases it resembles most closely, for the manifestations of *Brucella* infection are protean, and correct diagnosis is difficult, even in acute cases. If the illness is prolonged, eventually other diseases are ruled out, and the correct diagnosis may be arrived at with the aid of serological tests, a skin test, or best of all by the cultivation of the organism from the blood or excretions.

In chronic cases the laboratory tests may fail to show evidence of disease through long periods of ill health. A characteristic feature of chronic brucellosis is the great variety of aches and pains that beset the patient, although he may appear healthy. In many such cases a diagnosis of neurasthenia may be given; for the symptoms of chronic brucellosis may agree with the textbook definitions of neurasthenia—

exhaustion, insomnia, irritability, and complaints of aches and pains for which no objective signs can be found.

TABLE 1.—*Number of reported cases of undulant fever, by States, 1930–37* [1]

| Division and State | 1937 | 1936 | 1935 | 1934 | 1933 | 1932 | 1931 | 1930 |
|---|---|---|---|---|---|---|---|---|
| New England: | | | | | | | | |
| Maine | 27 | 13 | 20 | 24 | 25 | 15 | 5 | 10 |
| New Hampshire | 4 | ------- | 1 | 9 | 9 | 4 | 1 | 2 |
| Vermont | 43 | 24 | 27 | 25 | 14 | 13 | 17 | 19 |
| Massachusetts | 44 | 55 | 42 | 15 | 11 | 15 | 15 | 12 |
| Rhode Island | 17 | 12 | 15 | 9 | 11 | ------- | 0 | 2 |
| Connecticut | 74 | 99 | 59 | 55 | 36 | 34 | 20 | 21 |
| Middle Atlantic: | | | | | | | | |
| New York | 190 | 212 | 230 | 301 | 255 | 224 | 171 | 165 |
| New Jersey | 60 | 64 | 34 | 27 | 28 | 36 | 49 | 17 |
| Pennsylvania | 87 | 86 | 68 | 80 | 54 | 47 | 55 | 33 |
| East North Central: | | | | | | | | |
| Ohio | 60 | 79 | 69 | 77 | 78 | 88 | 115 | 129 |
| Indiana | 20 | 15 | 15 | 34 | 15 | 30 | 27 | 31 |
| Illinois | 107 | 84 | 144 | 101 | 93 | 75 | 124 | 63 |
| Michigan | 79 | 89 | 73 | 102 | 81 | 45 | 19 | 15 |
| Wisconsin | 65 | 106 | 75 | 86 | 104 | 27 | 71 | 82 |
| West North Central: | | | | | | | | |
| Minnesota | 76 | 84 | 115 | 104 | 80 | 65 | 72 | 65 |
| Iowa | 137 | 113 | 112 | 177 | 151 | 88 | 48 | 145 |
| Missouri | 34 | 36 | 69 | 53 | 79 | 179 | 162 | 137 |
| North Dakota | 1 | 2 | 2 | 0 | 4 | 1 | 42 | 10 |
| South Dakota | 3 | 4 | 3 | 6 | 3 | 3 | 6 | 4 |
| Nebraska | 2 | 1 | 2 | 4 | 5 | 2 | 5 | 8 |
| Kansas | 82 | 97 | 98 | 99 | 86 | 79 | 64 | 100 |
| South Atlantic: | | | | | | | | |
| Delaware | 0 | 0 | 9 | 10 | 6 | 2 | 2 | 7 |
| Maryland | 40 | 41 | 37 | 49 | 39 | 49 | 47 | 32 |
| District of Columbia | 1 | 1 | 2 | 1 | 1 | ------- | 3 | 1 |
| Virginia | 30 | 33 | 39 | 34 | 44 | 37 | 20 | 25 |
| West Virginia | 7 | 0 | 3 | 2 | 2 | 1 | 5 | 1 |
| North Carolina | 26 | 23 | 31 | 19 | 15 | 4 | 0 | 2 |
| South Carolina | 6 | 9 | 11 | 18 | 10 | 3 | 3 | 7 |
| Georgia | 58 | 60 | 62 | 63 | 39 | 35 | 38 | 28 |
| Florida | 21 | 16 | 68 | 9 | 6 | 2 | 3 | 4 |
| East South Central: | | | | | | | | |
| Kentucky | 30 | 46 | 36 | 8 | 8 | 7 | 20 | 8 |
| Tennessee | 10 | 26 | 9 | 4 | 12 | ------- | 7 | 11 |
| Alabama | 48 | 43 | 54 | 45 | 12 | 17 | 20 | 23 |
| Mississippi | 22 | 21 | 14 | 3 | 13 | 11 | 3 | 7 |
| West South Central: | | | | | | | | |
| Arkansas | 31 | 35 | 16 | 11 | 10 | 12 | 12 | ------- |
| Louisiana | 55 | 37 | 49 | 54 | 28 | 35 | 59 | 20 |
| Oklahoma | 431 | 96 | 10 | 4 | 15 | 5 | 13 | 2 |
| Texas | 167 | 43 | 46 | 21 | 43 | 33 | 11 | 3 |
| Mountain: | | | | | | | | |
| Montana | 9 | 4 | 11 | 15 | 17 | 11 | 4 | 4 |
| Idaho | 14 | 4 | 0 | 2 | 18 | ------- | 23 | 2 |
| Wyoming | 1 | 2 | 1 | 2 | 1 | ------- | 1 | 2 |
| Colorado | 5 | 3 | 1 | 7 | 2 | 6 | 7 | 2 |
| New Mexico | 7 | 11 | 12 | 5 | 7 | 3 | 5 | 2 |
| Arizona | 29 | 35 | 4 | 18 | 27 | 11 | 12 | 16 |
| Utah | 5 | 3 | 5 | 8 | 9 | ------- | 11 | ------- |
| Nevada | 7 | 2 | 1 | 0 | 1 | 1 | 0 | ------- |
| Pacific: | | | | | | | | |
| Washington | 27 | 33 | 35 | 25 | 24 | 18 | 34 | 33 |
| Oregon | 9 | 21 | 19 | 35 | 25 | 22 | 24 | 26 |
| California | 189 | 172 | 150 | 157 | 132 | 107 | 103 | 115 |
| Total cases | 2,497 | 2,095 | 2,008 | 2,017 | 1,788 | 1,502 | 1,578 | 1,453 |

[1] Zero (0) indicates that the State health department reported that no cases occurred. Leaders (-----) indicate that no report was received from the State health department.

Beginning in 1927, the United States Public Health Service has received reports of cases of brucellosis from the various States. Only 112 cases were reported in 1927, but by 1937 the number had increased to 2,497. The total number of cases actually occurring is undoubtedly much larger than that reported.

TABLE 2.—*Number of reported deaths from undulant fever, by States, 1930–1937* [1] [2]

| Division and State | 1937 | 1936 | 1935 | 1934 | 1933 | 1932 | 1931 | 1930 |
|---|---|---|---|---|---|---|---|---|
| New England: | | | | | | | | |
| Maine | -- | 0 | 2 | 1 | 1 | 1 | 0 | 0 |
| New Hampshire | -- | 3 | 1 | 0 | 1 | 0 | 0 | 0 |
| Vermont | -- | 0 | 1 | 0 | 0 | 0 | 1 | 0 |
| Massachusetts | -- | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| Rhode Island | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 |
| Connecticut | 1 | 1 | 1 | 0 | 1 | 0 | 1 | 0 |
| Middle Atlantic: | | | | | | | | |
| New York | 3 | 6 | 4 | 4 | 6 | 4 | 3 | 4 |
| New Jersey | -- | 0 | 1 | 0 | 2 | 1 | 2 | 1 |
| Pennsylvania | -- | 12 | 3 | 2 | 3 | 1 | 4 | 3 |
| East North Central: | | | | | | | | |
| Ohio | -- | 4 | 8 | 3 | 8 | 10 | 4 | 5 |
| Indiana | 3 | 7 | 4 | 7 | 2 | 2 | 0 | 3 |
| Illinois | 2 | 3 | 3 | 4 | 1 | 3 | 5 | 3 |
| Michigan | 0 | 2 | 1 | 1 | 3 | 3 | 1 | 6 |
| Wisconsin | 4 | 5 | 2 | 5 | 2 | 0 | 2 | 0 |
| West North Central: | | | | | | | | |
| Minnesota | 5 | 3 | 3 | 1 | 0 | 2 | 1 | 0 |
| Iowa | 1 | 4 | 5 | 4 | 4 | 2 | 3 | 2 |
| Missouri | 4 | 11 | 6 | 3 | 8 | 5 | 2 | 1 |
| North Dakota | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 1 |
| South Dakota | 1 | 2 | 0 | 0 | 1 | 0 | 0 | 0 |
| Nebraska | -- | 0 | 0 | 0 | 1 | 0 | 2 | 0 |
| Kansas | 8 | 4 | 1 | 3 | 3 | 3 | 3 | 2 |
| South Atlantic: | | | | | | | | |
| Delaware | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 |
| Maryland | 2 | 2 | 4 | 1 | 0 | 3 | 0 | 0 |
| Dist. of Columbia | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 |
| Virginia | 2 | 2 | 2 | 3 | 0 | 2 | 2 | 0 |
| West Virginia | -- | 0 | 1 | 0 | 0 | 1 | 0 | 0 |
| North Carolina | 3 | 1 | 0 | 2 | 3 | 2 | 2 | 1 |
| South Carolina | -- | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Georgia | -- | 0 | 1 | 1 | 2 | 2 | 0 | 2 |
| Florida | -- | 2 | 4 | 1 | 0 | 0 | 1 | 1 |
| East South Central: | | | | | | | | |
| Kentucky | 3 | 2 | 3 | 2 | 0 | 1 | 4 | 1 |
| Tennessee | -- | 2 | 4 | 1 | 2 | 0 | 0 | 0 |
| Alabama | -- | 2 | 6 | 2 | 0 | 0 | 5 | 1 |
| Mississippi | -- | 0 | 0 | 0 | 1 | 0 | 0 | 2 |
| West South Central: | | | | | | | | |
| Arkansas | -- | 0 | 2 | 1 | 0 | 0 | 0 | 3 |
| Louisiana | -- | 4 | 5 | 4 | 1 | 3 | 4 | 2 |
| Oklahoma | -- | 1 | 1 | 2 | 2 | 1 | 2 | 0 |
| Texas | 13 | 0 | 8 | 3 | 6 | 11 | 5 | 2 |
| Mountain: | | | | | | | | |
| Montana | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Idaho | 1 | 2 | 0 | 0 | 2 | 2 | 4 | 0 |
| Wyoming | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Colorado | -- | 0 | 1 | 0 | 0 | 0 | 1 | 0 |
| New Mexico | -- | 1 | 0 | 0 | 0 | 1 | 1 | 1 |
| Arizona | -- | 2 | 1 | 0 | 1 | 1 | 0 | 0 |
| Utah | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 |
| Nevada | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Pacific: | | | | | | | | |
| Washington | -- | 0 | 2 | 0 | 1 | 1 | 0 | 2 |
| Oregon | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 1 |
| California | -- | 1 | 2 | 3 | 1 | 2 | 3 | 5 |
| Total deaths | 58 | 107 | 98 | 65 | 72 | 72 | 70 | 55 |

[1] The number of deaths is from the Annual Reports on Mortality of the Bureau of the Census except for Texas, 1930–1932, and for all States for 1937, which are from reports forwarded to the United States Public Health Service by the respective State departments of health.

[2] Zero (0) indicates that the State health department reported that no cases occurred. Leaders (----) indicate that no report was received from the State health department.

About one-sixth of the cases reported during 1937 were in Oklahoma (fig. 1). Prior to 1936 very few cases were recognized in Oklahoma; but during 1936, 96 cases were reported, followed by 431 cases in 1937. Preliminary reports for 1938 indicate that the number of cases will probably be larger than in 1937.

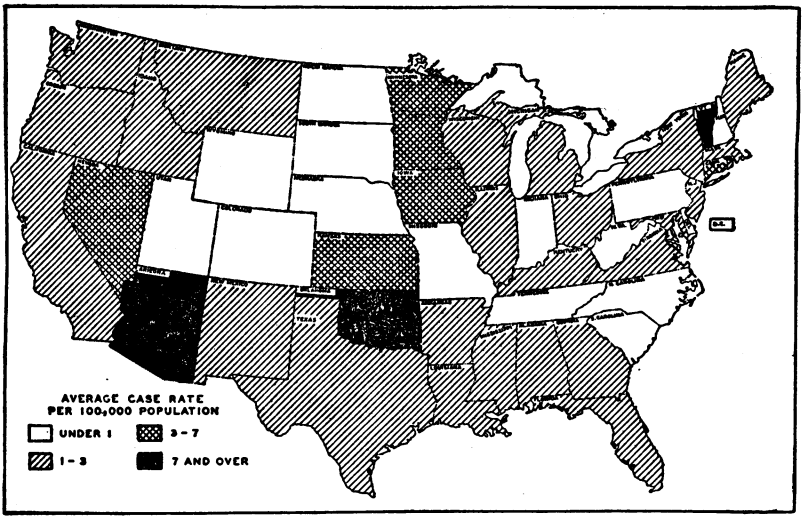


FIGURE 1.—Average case rates of undulant fever per 100,000 population, 1936-37.

The majority of people have no direct contact with *Brucella*-infected animals; for them brucellosis is a preventable disease. If all milk were efficiently pasteurized or boiled before being consumed, there would be no brucellosis excepting in those occupational groups whose work brings them into contact with infected animals or infected carcasses.

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## STUDIES ON THE FATE OF SELENIUM IN THE ORGANISM*

By M. I. SMITH, *Principal Pharmacologist*, B. B. WESTFALL, *Assistant Chemist*, and E. F. STOHLMAN, *Junior Pharmacologist, Division of Pharmacology, National Institute of Health, United States Public Health Service*

We have previously shown (*1*) that in chronic poisoning in cats with inorganic selenium administered as sodium selenite from 50 to 80 percent of the total daily intake is usually excreted in the urine, and that the selenium which is stored in the tissues is found in highest concentration in the liver, kidney, spleen, pancreas, heart, and lungs.

*Presented before the Pharmacological Society at its annual meeting in Baltimore, March 30–April 2, 1938.

The present paper contains data on (*a*) the elimination from the blood stream and tissue fixation of intravenously injected sodium selenite; (*b*) the excretion and retention of naturally occurring organic food selenium as compared with inorganic selenium; and (*c*) some exploratory experiments aimed to shed light on the state of the selenium stored and excreted in animals chronically poisoned with organic food selenium as compared with acute or subacute poisoning with inorganic selenium.

## MATERIALS AND METHODS

Cats and rabbits were used in this work. In the experiments with inorganic selenium, sodium selenite, or selenate, was injected intravenously in the acute experiments, and given orally, subcutaneously, or intravenously in the subacute and chronic experiments. In the experiments on the rabbits inorganic selenium was administered orally by delivering an accurately measured amount of a solution of sodium selenite or selenate from a tuberculin syringe connected with a blunt needle placed well back of the tongue. In the cats the sodium selenite or selenate was administered in a small amount of milk. Cats will usually take in this manner up to 0.25 mg per kilo of selenium daily with little difficulty. Cats will take doses of 0.5 mg per kilo voluntarily for only a limited period, usually not over a week or two, when they begin to refuse all food. In such instances the administration of selenium was continued by the subcutaneous route.

Organic selenium[1] was administered to cats in the form of gluten prepared in this laboratory from selenium-bearing wheat. The gluten was found upon analysis to contain 50 p. p. m. selenium and it was administered as a daily supplement mixed in with chopped raw lean beef. At first the gluten was autoclaved at 15 pounds for 2 hours, but experience soon showed that the untreated gluten was utilized equally well and the practice of autoclaving was discontinued. From 75 to 150 grams of meat were allowed per day, according to the size of the animal, with a supplement of gluten varying from about 5 to 25 grams per day depending on the weight of the animal and the dose of selenium. When the ration was consumed, 50 to 100 cc of milk were allowed. Fresh water was accessible at all times. The animals were kept in metabolism cages suitable for collecting urine and feces as needed.

The source of organic selenium for rabbits was selenium-bearing oats administered with a minimum of loss in specially designed feeders so that the average daily intake of selenium per kilogram of body weight could be computed. Fresh cabbage supplemented the oats to provide water, salts, and vitamins. The selenium content of the oats used was 10 p. p. m. The selenium-bearing grains used in this work were secured from farms in southern South Dakota.

[1] The term "organic selenium" as used in this paper applies to the naturally occurring food selenium which is known to be in organic combination (*2*, *3*), though its precise chemical nature is unknown.

No serious difficulties were encountered in getting the animals to consume sufficient organic selenium for the purposes of the present experiments. Some difficulty was encountered when the doses of selenium allowed approached the toxic limits; but this, together with the toxicological aspects of organic selenium, will be dealt with in another paper.

The analyses of tissues and body fluids for total selenium were carried out by methods previously outlined (*1*, *4*). Modifications thereof which were developed in the course of this work for the purpose of distinguishing between different types of selenium will be described below in connection with the experimental work. The results will be presented under several headings.

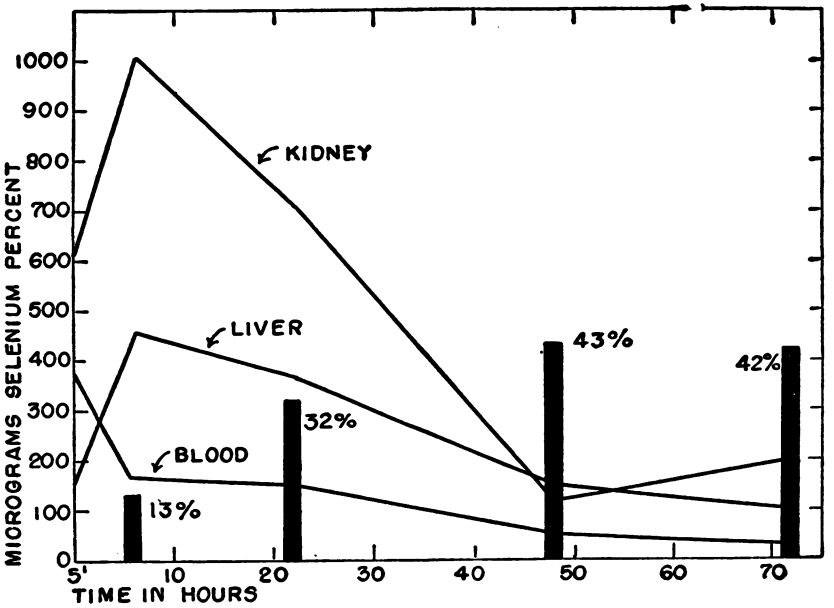


FIGURE 1.—Elimination, from the blood stream, of intravenously injected inorganic selenium, its fixation in the tissues, and its excretion in the urine. Vertical bars show the urinary excretion as percent of the total amount injected.

## RESULTS

### 1. THE ELIMINATION OF INTRAVENOUSLY INJECTED SELENIUM FROM THE BLOOD STREAM AND ITS FIXATION IN THE TISSUES

One milligram per kilogram of body weight of selenium as sodium selenite was injected intravenously in a series of rabbits weighing 2 to 2.5 kilograms each. This is approximately two-thirds of the minimum lethal dose. After intervals of from 5 minutes to 72 hours the animals were exsanguinated from the carotid artery under ether anesthesia, and the blood, liver, kidneys, and urine were analyzed for selenium. Two or more animals were used for each experiment, and the average results expressed as micrograms percent selenium are plotted in figure 1. It will be noted that within 5 minutes of the injection the selenium con-

centration in the kidney is considerably higher than that of the blood. The selenium concentration of the liver and kidney mounts during the first 6 hours, while that of the blood falls rather rapidly. Following this, the blood selenium falls slowly, that of the kidney and liver more rapidly, so that by the end of 48 hours the values reach a low retention level with little change during the following 24 hours. Examination of the urinary selenium indicates that the fall in tissue selenium during the first 42 hours following its preliminary rise is accounted for chiefly by the relatively rapid excretion by the kidney; for, on the average, 43 percent of the total selenium injected is recoverable in the urine at the end of 48 hours. Table 1 shows in detail the selenium excreted in the urine at various intervals in relation to the amount injected. It may be concluded, therefore, that selenium leaves the blood stream slowly to be absorbed selectively by certain tissues, thence to be eliminated chiefly in the urine. Relatively little selenium is found in the bile of such animals.

TABLE 1.—*Urinary excretion of selenium after a single intravenous injection of sodium selenite in rabbits*

| Experiment No. | Dose, mg Se per kilo | Number of hours after injection | Urinary selenium | | Experiment No. | Dose, mg Se per kilo | Number of hours after injection | Urinary selenium | |
|---|---|---|---|---|---|---|---|---|---|
| | | | Micrograms per cent | Percent of amount injected | | | | Micrograms per cent | Percent of amount injected |
| 3 | 1.0 | 6 | 3,500 | 10 | 20 | 0.8 | 24 | 2,260 | 61 |
| 4 | 1.0 | 6 | 6,400 | 17 | 9 | 1.0 | 48 | | 50 |
| 5 | 1.0 | 24 | 1,000 | 9 | 12 | 0.8 | 48 | 510 | 36 |
| 6 | 1.0 | 24 | 1,530 | 38 | 7 | 1.0 | 72 | 935 | 59 |
| 10 | 1.0 | 24 | 530 | 16 | 8 | 1.0 | 72 | 840 | 48 |
| 16 | 0.8 | 24 | 360 | 29 | 11 | 1.0 | 72 | | 35 |
| 17 | 0.8 | 24 | 400 | 30 | 13 | 1.0 | 72 | 390 | 28 |
| 19 | 0.8 | 24 | 2,160 | 40 | | | | | |

### 2. THE EXCRETION OF SELENIUM IN CHRONIC POISONING

There is a considerable difference in the excretion of selenium in chronic poisoning with the element in organic combination as compared with inorganic. Though the chief path of excretion in both cases is by way of the kidneys, relatively less selenium is eliminated in the urine of animals receiving organic than in those receiving inorganic selenium. This is not due to any marked difference in availability, but rather to greater retention of organic selenium. We have already called attention to the fact that the daily urinary excretion of selenium in cats chronically poisoned with daily doses of from 0.02 to 0.25 mg of selenium per kilo in the form of sodium selenite is usually from 50 to 80 percent of the total intake. The fecal excretion of selenium is not over 20 percent of the intake, and is usually less (*1*). In a series of parallel experiments with organic selenium (selenium-bearing gluten) carried out on cats over a period of from one to nearly

five months, the average fecal excretion was about the same as in the inorganic group, while the urinary excretion of selenium was in the neighborhood of only about 40 percent of the total intake, as shown in table 2.

TABLE 2.—*The selenium excretion in cats chronically poisoned with selenium-bearing wheat gluten*

| Cat No. | Sex and weight | Daily dose selenium, mg per kilo | Number of days | Urinary selenium | | Fecal selenium | |
|---|---|---|---|---|---|---|---|
| | | | | Average concentration, micrograms, per cent | Output, percent of intake | Average concentration, micrograms, per cent | Output, percent of intake |
| 42 | M 3.2 | 0.1 | 134 | 129 | 41 | 344 | 8 |
| 46 | F 2.6 | 0.1 | 138 | 136 | 47 | 685 | 12 |
| 40 | M 2.4 | 0.2 | 133 | 223 | 43 | 700 | 18 |
| 47 | F 1.7 | 0.2 | 140 | 177 | 41 | 650 | 25 |
| 52 | F 1.8 | 0.5 | 25 | 445 | 42 | ---------- | ---------- |
| 58 | F 2.5 | 0.5 | 96 | 435 | [1] 32 | ---------- | ---------- |

[1] Intermittent anorexia, hence amount ingested uncertain.

The same difference is noted when the selenium concentration in the urine is compared in the two sets of experiments. Though uncontrollable factors affecting diuresis may at times alter the general trend, it is nevertheless clear that equivalent doses of selenium chronically administered in organic combination result in relatively lower concentration of selenium in the urine than when administered in inorganic form. This is illustrated graphically in figure 2. In the

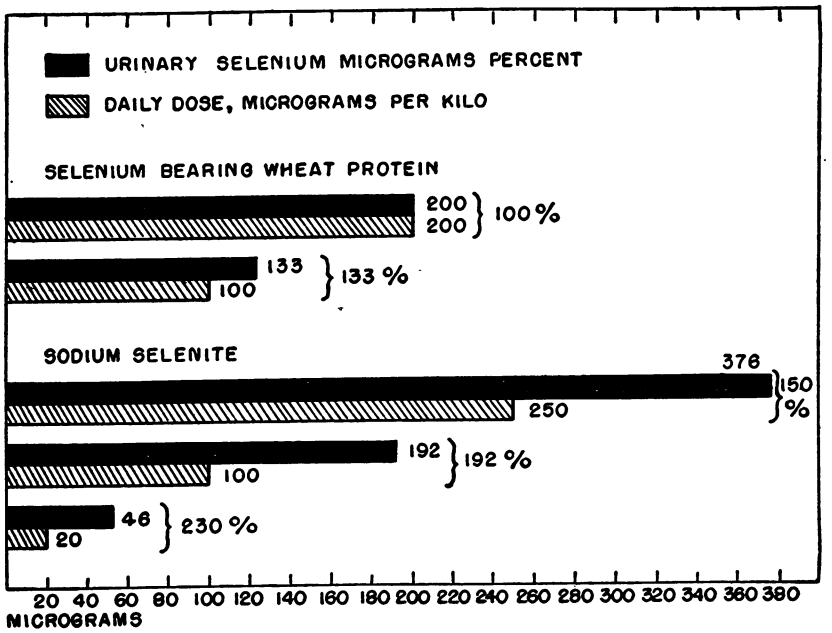


FIGURE 2.—Relation of excretion level of selenium in the urine to the daily intake in chronic selenium poisoning in cats. Shaded bars represent daily intake micrograms per kilo and solid bars represent the selenium excreted as micrograms per 100 cc.

group of animals chronically poisoned with selenium-bearing wheat protein, the urinary selenium concentration in micrograms percent is usually close to the daily dose ingested as micrograms per kilo. In cats chronically poisoned with inorganic selenium the average urinary concentration varies from 150 to 230 percent of the daily intake reckoned as micrograms selenium per kilo. Similar differences have been noted in rabbits receiving sodium selenite on the one hand and organic selenium in the form of selenium-bearing oats on the other, as may be seen from figures 4 and 6.

### 3. THE RETENTION OF SELENIUM IN CHRONIC POISONING

The retention of selenium in the tissues of animals fed naturally occurring organic selenium is far greater than its retention when

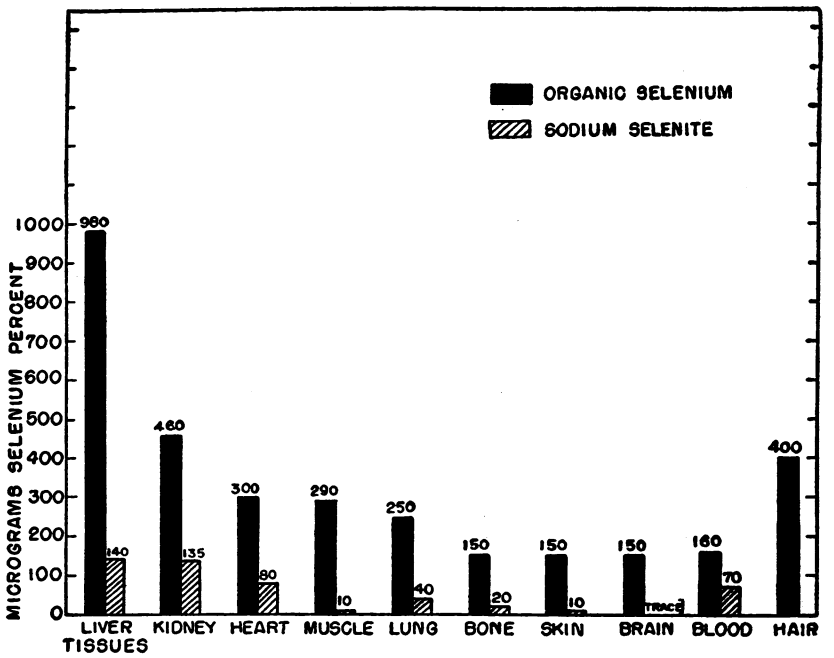


FIGURE 3.—The relative storage of selenium in the tissues in chronic poisoning with organic and inorganic selenium. Solid bars represent average values in animals fed selenium-bearing oats, shaded bars in animals treated with sodium selenite. All figures are in micrograms per 100 grams wet weight.

administered as sodium selenite. Indeed, in some tissues the difference may be 100 fold or more. In chronic poisoning with organic selenium there is fixation of the element in appreciable amounts in all the tissues and organs of the body with the exception of body fat. The differences in retention of the two types of selenium is most apparent in those tissues in which inorganic selenium is retained to a minor degree. Inspection of figure 3 will reveal marked differences in the brain, muscle, skin, and bone, and somewhat lesser differences

in other tissues. In the organic selenium group represented by solid bars in the figure there were five rabbits that had received an average of from 0.12 to 0.22 mg of selenium per kilo per day for a period of 54 to 135 days in the form of selenium-bearing oats. The selenium content in the tissues was uniformly high. In the inorganic group, represented by the shaded bars, there were nine rabbits receiving daily doses of from 0.15 to 0.3 mg per kilo either intravenously or orally over a period of from 56 to 115 days and five cats that had received orally daily doses of 0.25 mg per kilo of the element over approximately the same length of time, both as sodium selenite. The selenium content of the tissues in this group was uniformly low, with but little variation from the averages, as indicated in the figure. The maxima and minima for the different tissues in the two groups are given in table 3. The capacity of tissues to store selenium when fed in inorganic combination in the doses indicated seems to be limited, for its concentration is not materially affected by the length of the feeding period. In the organic group there is a progressive increase in the tissue selenium with the lengthening of the feeding period. We have recently analyzed the tissues of some rabbits that have been similarly fed selenium-bearing oats for a period of about 8 to 9 months, and we have found as much as 3,000 micrograms percent in the liver, 1,800 micrograms percent in the kidney, and 690 micrograms percent in muscle.[2]

TABLE 3.—*Selenium content of tissues in chronic poisoning with inorganic and organic selenium*

(Averages shown in fig. 3)

| Tissue | Micrograms selenium percent | | | |
|---|---|---|---|---|
| | Inorganic Se poisoning | | Organic Se poisoning | |
| | Minimum | Maximum | Minimum | Maximum |
| Liver | 66 | 292 | 680 | 1,690 |
| Kidney | 70 | 200 | 232 | 750 |
| Heart | Trace | 171 | 272 | 346 |
| Muscle | 3 | 25 | 197 | 342 |
| Lung | Trace | 72 | 232 | 287 |
| Bone | 17 | 24 | 92 | 189 |
| Skin | Trace | 10 | 140 | 157 |
| Brain | ---- | Trace | ---- | 152 |
| Blood | 51 | 72 | 152 | 173 |

The differences in retention of the two types of selenium is further brought out by the relative persistence of selenium in the urine and some of the tissues after the administration of the element is discontinued. The urines of 3 rabbits that had received 101 daily intra-

[2] See, for example, table 5. We have previously reported as much as 800 micrograms percent selenium in lean beef, pork, and chicken muscle of animals slaughtered for human consumption on farms located within selenium endemic regions (*5*). We have also found selenium in pericardial and peritoneal effusions, usually in amounts of about 150 to 200 micrograms percent. The selenium content of nails of rabbits has usually paralleled that of the hair.

venous injections of 0.15 mg per kilo of selenium as sodium selenite were analyzed at intervals of from 3 to 7 days until the selenium fell to a negligible trace (2–3 micrograms percent). For a week before the injections were discontinued these rabbits were excreting on an average 168, 212, and 224 micrograms percent, respectively. The subsequent course is shown in figure 4, in which the concentration of

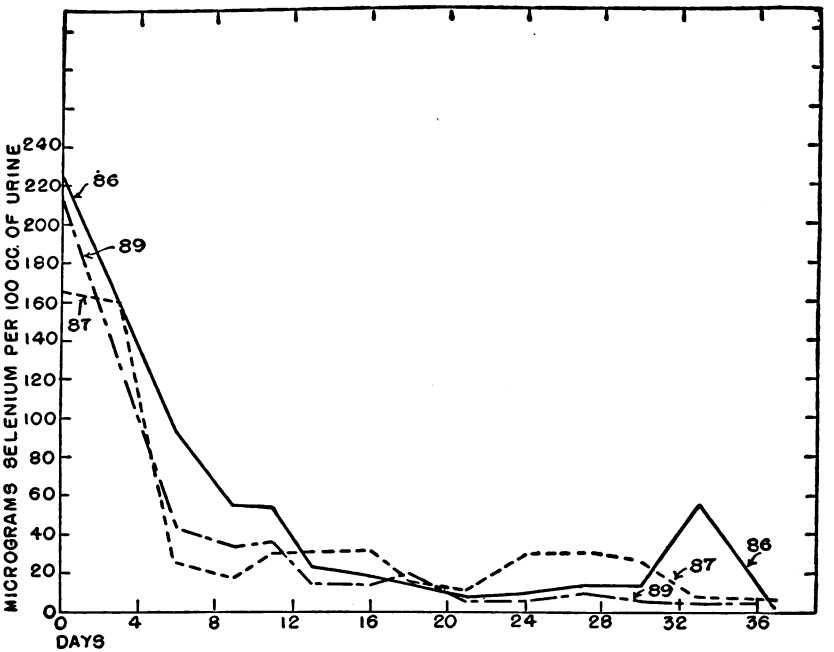


FIGURE 4.—The urinary excretion of selenium stored in the tissues of rabbits chronically poisoned with sodium selenite. The rabbits had received 101 daily doses of 0.15 mg selenium per kilo. Before discontinuing the injections they were excreting 168, 212, and 224 micrograms percent selenium, respectively. All values expressed in micrograms percent.

urinary selenium is plotted against the time in days. It will be noted that within 6 to 8 days the urinary selenium dropped to a low level of about 20 to 50 micrograms percent, but persisted in small amounts for nearly a month. Figure 5 shows the urinary excretion of selenium in these animals computed in micrograms per day.

A comparable experiment was carried out on a group of 3 rabbits chronically poisoned with organic selenium. These animals had received an average of from 0.15 to 0.19 mg of selenium per kilogram per day in the form of selenium-bearing oats over a period of 144 days. Toward the end of the feeding period they were excreting an average of 55, 101, and 108 micrograms percent of selenium, respectively. The administration of selenium was discontinued and the urines were analyzed at 3 to 7 day intervals for a period of 140 days as shown in figure 6. While the excretion level of selenium tapered off considerably within 15 to 20 days, appreciable amounts continued to be

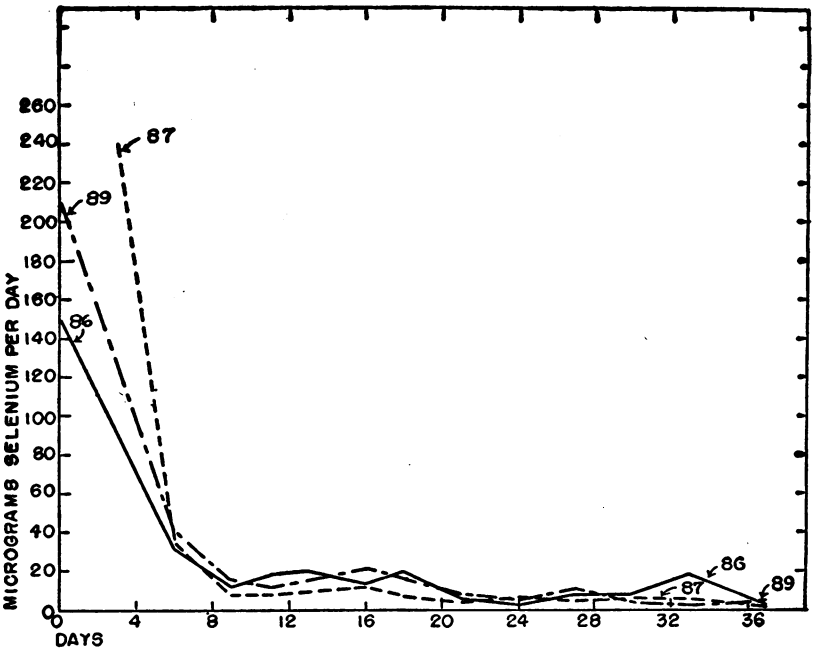


FIGURE 5.—Same as figure 4; values expressed in micrograms per day.

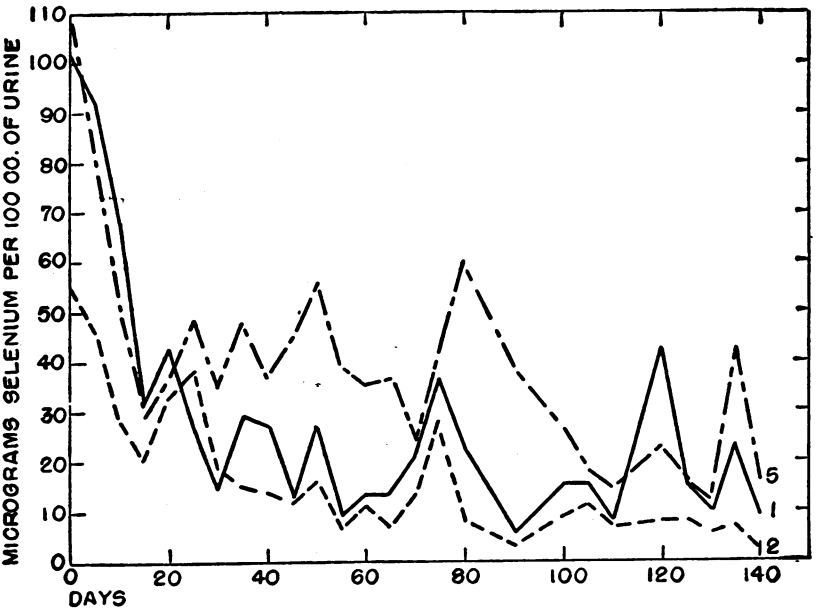


FIGURE 6.—The urinary excretion of selenium stored in the tissues of rabbits chronically poisoned with organic selenium. The rabbits had received an average of from 0.15 to 0.19 mg selenium per kilo per day for 144 days in selenium-bearing oats. Before the selenium was discontinued, they were eliminating 55, 101, and 108 micrograms percent selenium, respectively. All values expressed in micrograms percent.

excreted throughout the observation period. One month later, that is approximately 6 months after the administration of selenium had been discontinued, these animals still had 8, 13, and 25 micrograms percent, respectively, in the urine, 92, 147, and 214 micrograms percent, respectively, in the liver, and 12 to 29 micrograms percent in the blood.

The excretion level of selenium in the urine during this period computed in micrograms per day is shown in figure 7. Simple calcula-

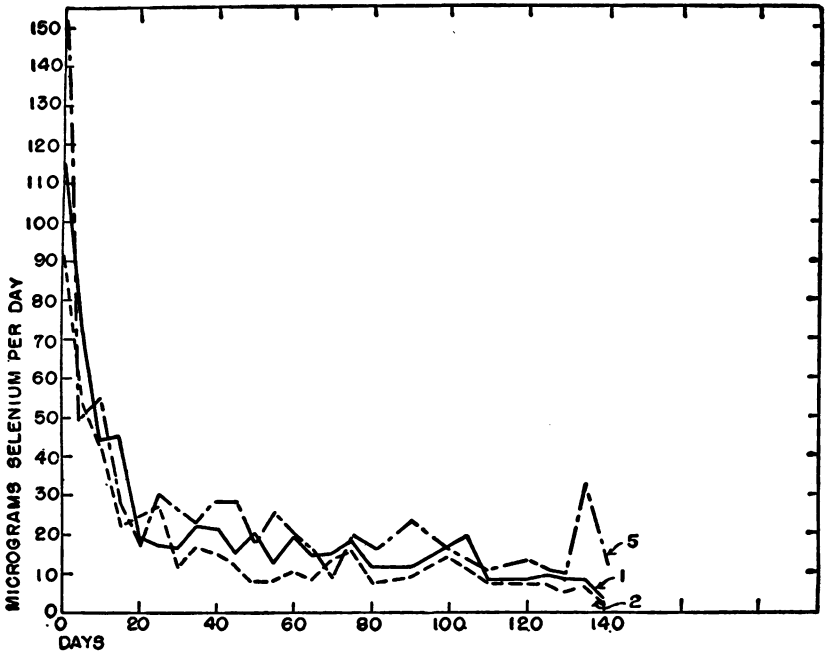


FIGURE 7.—Same as figure 6. All values expressed in micrograms per day.

tion showed that rabbit No. 5, for instance, in figure 7, eliminated 3.1 mg of selenium during the observation period of 140 days. The estimated amount of selenium stored in the body of this animal could not have exceeded 5 mg and probably was not over 4 mg. This is further evidence that the urine is the chief pathway for the elimination of selenium from the body.

The marked difference in the excretion level of selenium in the urine and its retention in chronic poisoning with the two types of selenium is further illustrated in figure 8.

#### 4. THE CHEMICAL NATURE OF SELENIUM IN THE TISSUES AND BODY FLUIDS

The differences in the fixation, storage, and retention of inorganic and organic selenium already noted suggested the possibility of differences in chemical behavior of the element in the course of its trans-

port in the body. It seemed likely that selenium introduced as sodium selenite or selenate might undergo a different fate from that of the selenium introduced into the body as the naturally occurring organic selenium. When biological material is analyzed for selenium as described here and in previous publications, the method involves complete destruction of organic matter by wet ashing and the result gives the total selenium content. It appeared of interest to inquire what part of the selenium, if any, is in non-protein combination, what part, if any, is in inorganic combination, and lastly, whether there is any selenium, especially in the urine, that is of a volatile nature.

Nonprotein selenium could obviously be determined in protein-free tissue extracts. It was indeed found that added sodium selenite or

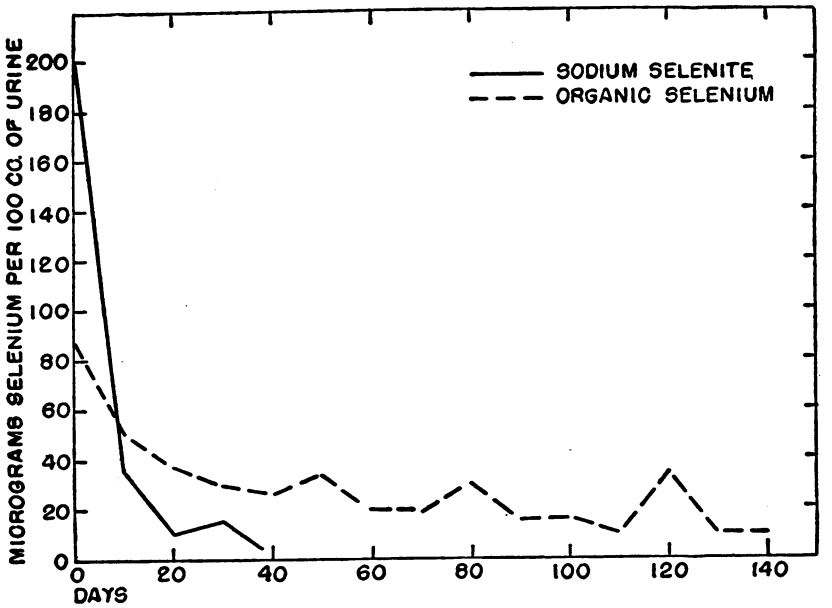


FIGURE 8.—Comparison of urinary excretion level and rate of elimination of stored selenium in chronic poisoning with inorganic and organic selenium in rabbits. The curves represent the average values for the three rabbits in figures 4 and 6, respectively.

selenate to a trichloracetic acid filtrate of a tissue extract, such as liver or kidney, could be recovered quantitatively by distillation with bromine-hydrobromic acid mixture and subsequent precipitation by reduction in the usual manner. However, when sodium selenite was added to a tissue such as liver and then extracted with trichloracetic acid none of the selenium could be recovered in the filtrate unless the tissue was extracted simultaneously with bromine-hydrobromic acid mixture and trichloracetic acid. It seemed probable that the selenite may have been reduced by contact with tissue to elementary selenium. This seemed all the more likely; for, when

the less easily reducible sodium selenate was added to tissue such as liver and then extracted with trichloracetic acid, it was recoverable by distillation of the protein-free filtrate with bromine-hydrobromic acid. To insure, therefore, complete recovery of all nonprotein selenium, including that which may exist in the tissues as elementary selenium, it was decided upon extraction of ground-up tissues with bromine-hydrobromic acid-trichloracetic acid mixtures, centrifugation or filtration of the extracts, and estimation of the selenium in a suitable aliquot by distillation and subsequent precipitation of the distillate by reduction with $SO_2$ and hydroxylamine hydrochloride.

The procedure for extraction which was finally adopted consisted in thoroughly grinding a weighed amount of tissue in a glass mortar with a little pure sand and one volume of distilled water, then with one volume of a mixture of 10 percent bromine in 48 percent hydrobromic acid, and finally with two volumes of 40 percent aqueous trichloracetic acid solution. The extract was at once filtered or centrifugated. The possibility that some of the nonprotein selenium was in organic combination requiring hydrolysis for the liberation of the element was also considered, and for this reason the practice was adopted of hydrolyzing the protein-free filtrate in the distillation flask with sulfuric acid for about 10 minutes before the actual distillation of the volatile bromide was begun. The distillation mixture thus consisted of 0.5 percent bromine, 19 percent hydrobromic acid, and 25 percent sulfuric acid.[3]

TABLE 4.—*Relation of nonprotein to total selenium in acute and subacute poisoning with sodium selenite*

| Experiment | Dose, mg Se per kilo | Interval | Selenium, micrograms percent | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Blood | | | Liver | | | Kidney | | |
| | | | Total | Nonprotein | Nonprotein, percent of total | Total | Nonprotein | Nonprotein, percent of total | Total | Nonprotein | Nonprotein, percent of total |
| Rabbit 4 | 1.0 i. v.[1] | 6 hours | 160 | 156 | 98 | | | | | | |
| Rabbit 6 | 1.0 i. v | 24 hours | 140 | 136 | 97 | | | | 760 | 650 | 87 |
| Rabbit 10 | 1.0 i. v | 24 hours | 200 | 171 | 86 | 260 | 220 | 85 | | | |
| Rabbit 5 | 1.0 i. v | 24 hours | | | | 384 | 356 | 93 | | | |
| Rabbit 15 | 1.0 i. v | 24 hours | | | | 240 | 139 | 58 | | | |
| Rabbit 12A | 1.0 i. v | 48 hours | | | | 200 | 200 | 100 | | | |
| Rabbit 7 | 1.0 i. v | 72 hours | 44 | 43 | 98 | | | | 225 | 216 | 96 |
| Rabbit 8 | 1.0 i. v | 72 hours | | | | | | | 175 | 200 | 114 |
| Rabbit 11 | 1.0 i. v | 72 hours | | | | 100 | 66 | 66 | | | |
| Cat 60 | 0.5 [2] | 4 days | | | | 580 | 334 | 58 | 840 | 762 | 91 |
| Cat 61 | 0.5 [2] | 4 days | | | | 120 | 77 | 62 | 320 | 338 | 106 |
| Cat 62 | 0.5 [2] | 10 days | | | | 228 | 110 | 49 | 290 | 326 | 112 |

[1] Intravenously.

[2] Eight daily doses per os and 3 to 15 daily doses subcutaneously with an interval of from 4 to 10 days between the last administration and autopsy.

[3] Percentages of bromine, sulfuric, and trichloracetic acids by volume; that of hydrobromic acid by weight.

TABLE 5.—*Relation of non-protein to total selenium in chronic poisoning with naturally occurring organic selenium (selenium-bearing oats)*

| Experiment | Average daily dose Se, mg per kilo | Days | Selenium, micrograms percent | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Blood | | | Liver | | | Kidney | | |
| | | | Total | Non-protein | Non-protein, percent of total | Total | Non-protein | Non-protein, percent of total | Total | Non-protein | Non-protein, percent of total |
| Rabbit 13 | 0.19 | 258 | 415 | 86 | 21 | 2,230 | 81 | 3 | 1,680 | 640 | 38 |
| Rabbit 18 | 0.20 | 255 | 265 | 31 | 12 | 1,450 | 51 | 3 | 830 | 275 | 33 |
| Rabbit 12 | 0.20 | 253 | ------ | ------ | ------ | 1,110 | 83 | 8 | ------ | ------ | ------ |
| Rabbit 20 | 0.20 | 260 | 315 | 142 | 45 | 3,160 | 55 | 1 | 1,820 | 140 | 8 |
| Rabbit 33 | 0.21 | 122 | ------ | ------ | ------ | 1,210 | Trace | Trace | 735 | 240 | 33 |
| Rabbit 25 | 0.24 | 192 | ------ | ------ | ------ | 1,480 | 438 | 30 | ------ | ------ | ------ |
| Rabbit 34 | 0.25 | 150 | 190 | 124 | 65 | 1,100 | 180 | 16 | 1,650 | 830 | 50 |
| Rabbit 23 | 0.23 | 295 | 180 | 90 | 50 | 1,550 | 350 | 22 | 797 | 205 | 25 |

With this method of analysis for nonprotein selenium a series of determinations was made to ascertain the relation of nonprotein to total selenium under various experimental conditions of selenium poisoning. The results of these experiments are presented in tables 4 and 5. The values for total and nonprotein selenium in blood, liver, and kidney of a series of rabbits and cats treated with sodium selenite are given in table 4. Similar values for a series of rabbits chronically poisoned with organic selenium are presented in table 5. The experiments indicate that in acute poisoning with sodium selenite most of the selenium in the circulating blood and in the kidneys is nonprotein. In the liver the greater part of the selenium is nonprotein in acute poisoning and apparently a considerable amount of it is in protein combination in subacute poisoning. In chronic poisoning with organic selenium the greater part of the element occurs in protein combination in all the tissues analyzed, namely, blood, liver, and kidney. Especially in the liver, usually very little of the selenium can be removed by extraction with trichloracetic-bromine-hydrobromic acid mixture as used here. We have also made similar observations on muscle of cats chronically poisoned with selenium-bearing gluten as well as rabbits chronically poisoned with selenium-bearing oats. In four such experiments on cat's muscle the total selenium varied from 115 to 172 micrograms percent with none or a trace of nonprotein selenium. In an experiment on rabbit's muscle the nonprotein selenium was 18 percent of the total, the total selenium in this case having been 690 micrograms percent. These results seem to indicate clearly that the tissue selenium in chronic poisoning with naturally occurring organic food selenium is for the most part in some firm protein combination whereas only a relatively small part of it is nonprotein, and this probably the end product or products of metabolism. The possibility of inorganic selenium being built up into a

protein-selenium complex is indicated from the data on the relative nonprotein and total selenium in the livers in the experiments on cats detailed in table 4. More work on this point appeared desirable.

At this juncture attention was directed to the chemical behavior of selenium in the protein derived from selenium-bearing wheat. Among other things, which will be discussed in detail elsewhere, it was observed that all the selenium can be split off by extracting the protein for 24 hours with trichloracetic-bromine-hydrobromic acid mixture at room temperature. There is apparently little, if any, dissolution of the protein under these conditions. This was surprising in view of the failure by previous investigators to remove the selenium from wheat protein by any procedure short of complete hydrolysis. Thus Franke and Painter (*6*) were unable to remove it except by complete hydrolysis of the protein with sulfuric acid; and Jones and associates (*3*, *7*) were unable to remove any selenium by heating the protein to boiling with 35 percent $H_2SO_4$ or 20 percent NaOH, and they concluded that the selenium was an integral part of the protein and could be split off only in the same manner as the amino acids by hydrolysis. Our observation, on the contrary, would seem to indicate that the protein-bound selenium, in wheat at any rate, is rather labile. This raised the question whether some of the tissue selenium might not be of a similar nature, and whether some of the selenium separated from the tissue by extraction with trichloracetic-bromine-hydrobromic acid mixture which we have designated nonprotein might not, in effect, represent loosely bound protein selenium.

Another series of experiments was therefore carried out in which the distribution of tissue selenium was determined successively in (1) the trichloracetic acid fraction, (2) the bromine-hydrobromic acid fraction, and (3) the protein fraction. After extracting the tissue with four to five volumes of 20 percent trichloracetic acid and centrifugation, the protein residue was stirred for a few minutes with 3 to 5 volumes of a mixture of 0.25 percent bromine,[4] 12 percent hydrobromic acid, and 16 percent trichloracetic acid. This was centrifugated and the protein residue washed by centrifugation with 16 percent trichloracetic acid.

[4] In some of the experiments 2.5 percent bromine was used with apparently little difference in result.

TABLE 6.—*Distribution of tissue selenium in the trichloracetic acid, bromine-hydrobromic acid, and protein fractions in rabbits*

| Experiment | Nature of poisoning | Tissue | Trichloracetic acid fraction | | Bromine-hydrobromic acid fraction | | Protein fraction | |
|---|---|---|---|---|---|---|---|---|
| | | | Micrograms, percent | Percent of total | Micrograms, percent | Percent of total | Micrograms, percent | Percent of total |
| 22 | Se-bearing oats, 0.2 mg Se per kilo per day, 288 days. | Liver | 40 | 5 | 92 | 12 | 600 | 83 |
| | | Kidney | 137 | 14 | 110 | 11 | 730 | 75 |
| 23 | Se-bearing oats, 0.25 mg Se per kilo per day, 294 days. | Blood | 0 | 0 | 90 | 50 | 90 | 50 |
| | | Liver | 0 | 0 | 350 | 22 | 1,200 | 78 |
| | | Kidney | 0 | 0 | 205 | 25 | 592 | 75 |
| | | Muscle | | | 123 | 18 | 567 | 82 |
| 30 | Se-bearing oats, 0.23 mg Se per kilo per day, 310 days. | Blood | 30 | 12 | 45 | 18 | 180 | 70 |
| | | Liver | Trace | 0 | 75 | 8 | 835 | 92 |
| | | Kidney | 115 | 21 | 84 | 15 | 350 | 64 |
| 22A | 14 daily doses of 0.5 mg Se per kilo per os as $Na_2SeO_4$. Killed 24 hours after last dose. | Blood | 16 | 34 | 0 | 0 | 31 | 66 |
| | | Liver | 51 | 33 | 0 | 0 | 100 | 67 |
| | | Kidney | 70 | 23 | 0 | 0 | 230 | 77 |
| 21A | 11 daily doses of 0.5 mg Se per kilo per os and 1.0 mg per kilo i. v., all as $Na_2SeO_4$. Killed 30 minutes after last injection. | Blood | 200 | 40 | 48 | 9 | 246 | 51 |
| | | Liver | Trace | 0 | 50 | 6 | 730 | 94 |
| | | Kidney | 520 | 36 | 147 | 10 | 790 | 54 |
| 16A | 0.8 mg Se per kilo i. v. as $Na_2SeO_3$. Killed after 24 hours. | Liver | 25 | 17 | 80 | 53 | 45 | 30 |
| | | Kidney | 73 | 21 | 165 | 47 | 110 | 32 |

The results of these experiments are summarized in table 6. In three of the experiments, tissues of animals chronically poisoned with selenium-bearing oats were used, and in three, those of animals with acute and subacute poisoning with inorganic selenium. Selenium was found in the protein fractions in every instance, the concentration varying from 30 to 94 percent of the total. The trichloracetic and bromine-hydrobromic acid fractions contained much less selenium than the protein fractions except in animals acutely poisoned with massive doses of inorganic selenium. The liver protein fractions especially exhibited a relatively high concentration of selenium. Thus from 30 to 94 percent of the total selenium was found in the liver protein fractions in animals poisoned with inorganic selenium and from 78 to 92 percent in animals poisoned with organic selenium. It is assumed here that the bromine-hydrobromic acid fraction merely represents metallic or possibly some other form of nonprotein selenium. It is not impossible, however, that this fraction may also represent in part some of the more labile organic selenium more or less intimately associated with the proteins.[5] It may be safely concluded, therefore, that much of the selenium ingested in its naturally occurring organic form enters into tissue proteins, and a considerable amount of the inorganic selenium ingested continuously in sublethal doses similarly enters into protein combination in some of the tissue, especially the liver. It may also be inferred from these results that there can be but little metallic selenium in the tissues in chronic selenium poisoning; and, consequently, reduction of selenium in the

[5] Protein sulfur has been shown to be partially oxidized to sulfate by bromine water (*8*).

tissues to the relatively nontoxic metallic form is probably not an important factor in its detoxification.

The foregoing findings suggested the possibility of differences in the urinary selenium in the two types of poisoning. Since the nonprotein selenium in the blood and tissues probably represents the end products of its metabolism, and since it appears to be a relatively labile compound, it seemed probable that it might constitute the major part of the urinary selenium in animals receiving the element in its naturally occurring form. In animals receiving inorganic selenium it seemed probable that, in addition to this, part of the selenium found in the urine might also be inorganic. In any event it appeared probable that it might be possible to recover the selenium in the urine in both cases by simple distillation with bromine-hydrobromic acid mixture after preliminary short hydrolysis with $H_2SO_4$ to insure the liberation of the selenium from its organic combination. A series of experiments upon urines of animals poisoned with organic as well as inorganic selenium showed that the selenium recoverable by distillation with bromine-hydrobromic-sulfuric acid mixture and referred to as "nonprotein" in table 7 is quantitatively equal to the total selenium in urine as obtained by the more time-consuming oxidation and wet-ashing process. For an estimation by this technique, proportions used are as follows: urine, 1 volume; bromine, enough to make its concentration about 0.5 percent in the final mixture; hydrobromic acid, 48 percent, 1 volume; sulfuric acid, 96 percent, 1 volume. The reagents are slowly added to the urine in the assembled distillation apparatus in the order given. A distillate of about 1.5 volumes is needed to ensure complete recovery of the selenium. Fifty to 100 cc of urine are sufficient for urines containing 10 or more micrograms percent selenium.

TABLE 7.—*Relation of "nonprotein" to total selenium in the urine in acute poisoning with inorganic and chronic poisoning with organic selenium*

| Inorganic selenium poisoning ($Na_2SeO_3$) | | | | Organic selenium poisoning (selenium-bearing oats) | | | |
|---|---|---|---|---|---|---|---|
| Experiment | Selenium, micrograms percent | | | Experiment | Selenium, micrograms percent | | |
| | Total | Nonprotein | Nonprotein percent of total | | Total | Nonprotein | Nonprotein, percent of total |
| Rabbit 9 | 460 | 460 | 100 | Rabbit 15 | 96 | 95 | 99 |
| Rabbit 10 | 510 | 530 | 104 | Rabbit 35 | 167 | 172 | 103 |
| Rabbit 12A | 485 | 510 | 105 | Rabbit 30 | 233 | 241 | 103 |
| Rabbit 13 | 380 | 390 | 102 | Rabbit 35A | 337 | 300 | 90 |
| Rabbit 16 | 360 | 316 | 88 | Rabbit 26 | 427 | 433 | 101 |
| Rabbit 17 | 400 | 394 | 99 | Rabbit 37 | 512 | 506 | 99 |
| | | | | Rabbit 32 | 454 | 434 | 96 |
| | | | | Rabbit 36 | 413 | 398 | 96 |

To determine the presence of volatile selenium in the urine we have distilled some of the urines in table 7 after acidifying with acetic acid and tested the distillate for selenium either directly with bromine-hydrobromic acid followed by reduction, or after a preliminary hydrolysis of the distillate with sulfuric acid. No volatile selenium could be demonstrated in any of the urines in this manner except for possible traces. Finally the presence of inorganic selenium was tested in a series of urines by direct precipitation of the bromine-hydrobromic acid-treated urine by reduction with $SO_2$ and hydroxylamine hydrochloride. The precipitate so obtained was then washed with distilled water, dissolved in bromine-hydrobromic acid, distilled and reprecipitated with $SO_2$ and hydroxylamine hydrochloride. The precipitate was finally estimated for selenium in the usual manner. Inorganic selenium added as sodium selenate to normal urine was recovered quantitatively by this procedure. The results showed that in animals injected intravenously with 0.8 to 1.0 mg per kilo of sodium selenate or selenite, as much as 50 percent of the urinary selenium may be recoverable in this manner within the next 24 hours. In animals chronically poisoned with organic selenium, usually little or none of the element could be recovered by direct precipitation as just outlined. In subacute poisoning with inorganic selenium administered orally in doses of 0.5 mg per kilo over a period of 8 to 15 days, the results were variable, from traces to as much as 20 percent of the total selenium having been found in this form.

## SUMMARY

Studies on the fate of selenium in the body lead to the following conclusions:

1. Intravenously injected inorganic selenium leaves the blood stream slowly to be selectively absorbed in certain tissues, mostly the liver and kidney, thence to be excreted chiefly by way of the kidney.

2. The excretion level of selenium in the urine is higher in animals chronically poisoned with inorganic than when similarly poisoned with naturally occurring food selenium.

3. There is far greater retention of selenium in the tissues of animals chronically poisoned with naturally occurring organic selenium than with inorganic selenium.

4. After a 3- or 4-month period of poisoning with small doses of inorganic selenium, the element continues to be excreted in the urine for about a month. After a similar period of poisoning with like doses of naturally occurring organic selenium the element continues to be excreted in the urine for at least 6 months, and probably longer.

5. The selenium stored in the tissues in the course of chronic poisoning with organic selenium is, for the most part, in protein

combination. A small amount of it is nonprotein, and it probably represents the end product or products of its metabolism. Most of the selenium in the blood and kidney in acute poisoning with relatively large doses of inorganic selenium is nonprotein, while some of the liver selenium appears to be in protein combination. In subacute poisoning with inorganic selenium there is evidence of a protein-selenium complex in all the tissues examined, especially in the liver.

6. The urinary selenium in all cases is recoverable quantitatively by simple distillation with bromine-hydrobromic acid-sulfuric acid mixture. Little, if any, of the urinary selenium is in volatile form. It appears to consist of a relatively labile organic compound or compounds. Little, if any, inorganic selenium is demonstrable in animals chronically poisoned with organic selenium. In animals acutely poisoned by intravenous injection of inorganic selenium up to 50 percent of the urinary selenium is recoverable by direct precipitation and is therefore regarded to be inorganic. In subacute poisoning with inorganic selenium by oral administration some inorganic selenium is demonstrable in the urine, but not uniformly.

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## TWO NEW SPECIES OF *MERINGIS* JORDAN* (SIPHONAPTERA)

By GLEN M. KOHLS, *Assistant Entomologist, Rocky Mountain Laboratory, United States Public Health Service*

Two new species of fleas collected incident to field studies of Rocky Mountain spotted fever and sylvatic plague are described in this

*Contribution from the Division of Infectious Diseases, National Institute of Health, Rocky Mountain Laboratory, Hamilton, Mont.

paper. Additional material of one of these species from the collection of Prof. C. Andreson Hubbard, Pacific University, Forest Grove, Oreg., has also been studied.

*Meringis hubbardi* n. sp.

(Figures 1, 2, and 3)

This species is allied to *M. shannoni* (Jordan) 1929 and *M. parkeri* Jordan 1937, but the males may be distinguished from these species by the posterior segments. The males agree with *M. shannoni* and differ from *M. parkeri* in having only 2 antepygidial bristles. The females agree with *M. parkeri* and differ from *M. shannoni* in having 3 antepygidial bristles. The shape of the eighth tergite of the female differs in all three species.

*Holotype* ♂ was collected from *Sylvilagus* sp. (accidental host) Mayfield, Idaho, June 25, 1932; *allotype* ♀ was collected from *Dipodomys* sp., Elko County, Nev., September 1936; both are deposited in the collection of the Rocky Mountain Laboratory.

*Paratypes.*—Collections were as follows: One ♂ and 2 ♀ ♀ from *Dipodomys* sp., Hampton, Oreg., July 18, 1937; 1 ♂ from *Dipodomys* sp., Crane, Oreg., July 16, 1937; 2 ♂ ♂ from *Dipodomys* sp., Adel, Oreg., June 21, 1937; 1 ♂, 1 ♀ from *Dipodomys* sp. (nest) Adel, Oreg., June 23, 1937; 2 ♂ ♂ from *Dipodomys* sp., Crane, Oreg., July 16, 1937; 1 ♂ from *Peromyscus* sp., Crane, Oreg., July 17, 1937; 1 ♀ from *Peromyscus* sp., Willowdale, Oreg., June 14, 1937; 1 ♂, 3 ♀ ♀ from *Peromyscus* sp., Crane, Oreg., July 16, 1937; 1 ♂, 1 ♀ from *Peromyscus crinitus crinitus*, Narrows, Oreg., July 18, 1937; 2 ♂ ♂ from *Peromyscus* sp., Coleman's ranch, Nevada (P. O., Adel, Oreg.), June 27, 1937; 1 ♂ from *Microtus* sp., Burns, Oreg., September 1, 1932; 2 ♂ ♂, 2 ♀ ♀ from *Dipodomys* sp., Elko County, Nev., September 1936; 1 ♂, 3 ♀ ♀ from *Microtus* sp., Elko County, Nev., September 1936. Holotype and the paratype from Burns, Oreg., were collected by a field party from the Rocky Mountain Laboratory. Allotype and paratypes from Elko County, Nev., were collected by the field crew of the San Francisco Plague Laboratory, United States Public Health Service. All other type material was collected by Professor Hubbard. Paratypes have been deposited at the United States National Museum, the Rocky Mountain Laboratory, the San Francisco Plague Laboratory, and in the collection of Professor Hubbard.

*Male.*—Inner antepygidial bristle one-fourth to one-third the length of the outer one. Process (P) of the clasper broadly rounded at apex as in *M. parkeri*, differing in this respect from *M. shannoni*, in which this process is cone-shaped. Exopodite (F) nearly as in *M. parkeri*, but posterior margin more broadly rounded; anterior margin straight. Apical portion of sternite IX as in *M. parkeri*, but having, however,

six or seven bristles instead of five along the ventral margin posterior to the ventral apical spiniform. The two stout bristles immediately posterior to this spiniform in *M. shannoni* are absent. The proximal ventral lobe (P. V. L.) of sternite IX has a stout conical spiniform; margin of lobe immediately posterior to this spiniform dilated ventrally and smoothly rounded. A row of four bristles on margin of ventral portion of sternite IX anterior to proximal ventral lobe. Posteroventral margin of lateral lobe (L. P.) S shaped; apex pointed and upturned.

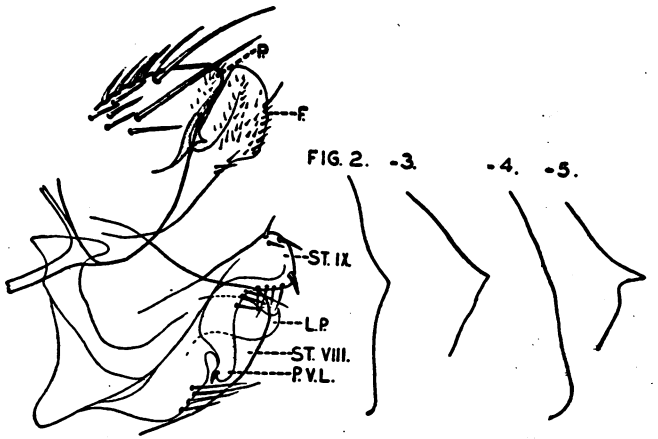


FIG. 1.




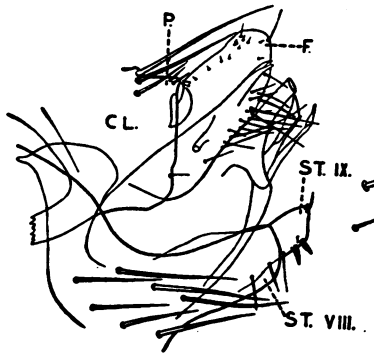


FIG. 6.

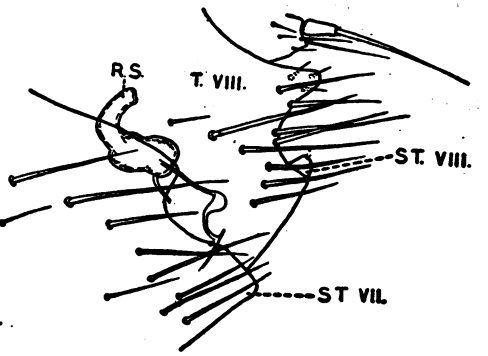


FIG. 7.

Explanation of figures

1. Genitalia of *M. hubbardi* n. sp., male.
2. *M. hubbardi*, seventh sternite, female.
3. *M. hubbardi*, eighth tergite, female.
4. *M. parkeri*, seventh sternite, female.
5. *M. parkeri*, eighth tergite, female.
6. Genitalia of *M. dipodomys* n. sp., male.
7. Genitalia of *M. dipodomys*, female.

*Abbreviations:* CL., clasper; P, process of clasper; F, exopodite of clasper; St. IX, ninth sternite; St. VIII, eighth sternite; St. VII, seventh sternite; T. VIII, eighth tergite; L. P., lateral process of ninth sternite; P. V. L., proximal ventral lobe of ninth sternite; R. S., spermatheca.

*Female.*—Length of upper antepygidial bristle two-sevenths, and lower bristle five-sevenths that of the middle one. Apical angle of tergite VIII (fig. 3) not so sharply pointed as in *M. parkeri* (fig. 5) and the margin below the apex is not so strongly incurved. Posterior margin of sternite VII with a distinct lobe (fig. 2); in *M. parkeri* the posterior margin (fig. 4) is nearly straight. Spermatheca as in *M. shannoni* and *M. parkeri.*

*Variation.*—In one paratype male the margin of the proximal ventral lobe of sternite IX posterior to the spiniform is as in *M. parkeri*, i. e., not dilated ventrally.

In the female paratype series there are specimens showing variation in the posterior margin of the seventh sternite. Certain specimens have the lobe more pointed and the margin immediately below it more concave than in the type.

*Acknowledgments.*—The writer wishes to thank Professor Hubbard and Senior Surg. C. R. Eskey, in charge of the Plague Laboratory, San Francisco, Calif., for furnishing specimens for study, and to acknowledge his indebtedness to Mr. Benjamin J. Collins, formerly of the National Institute of Health, for comparing the holotype with the types of *Meringis cummingi* (Fox) 1926, *M. arachis* (Jordan) 1929 and *M. shannoni* (Jordan) 1929.

## *Meringis dipodomys* n. sp.

(Figures 6 and 7)

This species is allied to *Meringis arachis* (Jordan) 1929, collected from near Tucson, Ariz., but is distinguished by the modified abdominal segments.

*Holotype* ♂ and *allotype* ♀ were collected from *Dipodomys* sp., Imperial County, Calif., December 1935; deposited at the Rocky Mountain Laboratory. *Paratypes*: 3 ♂♂ and 5 ♀♀ from *Dipodomys* sp., Imperial County, Calif., December 1935; 1 ♂ from *Dipodomys* sp., Inyo County, Calif., November 25, 1936; 1 ♀ from *Neotoma* sp., Inyo County, Calif., November 25, 1936. Paratypes have been deposited at the United States National Museum, the San Francisco Plague Laboratory, and the Rocky Mountain Laboratory.

The above mentioned materials were collected by representatives of the San Francisco Plague Laboratory and were forwarded to the Rocky Mountain Laboratory for identification by Senior Surg. C. R. Eskey.

*Male.*—Three antepygidial bristles, lower bristle about one-half and upper bristle about one-third the length of the middle one. Posterior margin of clasper (CL.) nearly straight. Process (P) very short in relation to exopodite (F), which is long, much broader than in *M. arachis*, and with the apex broadly rounded. Apical portion of

sternite IX similar to *M. arachis;* a very small, pale, cone-shaped spine and a hair situated at the apex. Two spiniforms, the more distal one of which is the smaller, on the ventral apical margin of this sternite. Ventral posterior portion of sternite VIII produced into a long, terminally rounded process.

*Female.*—Agrees with the description of *M. arachis* (figure not given) except for the shape of the apex of tergite VIII. The apex of this tergite is truncate (fig. 7), while in *M. arachis* it is described as pointed. Three antepygidial bristles, lower bristle two-thirds and upper bristle one-third of the length of the middle one. A portion of duct above bursa sclerified; a small hammer head shaped sclerification below the bursa.

*Variation.*—One paratype male with but one spiniform on the ventral apical margin of sternite IX.

The female paratypes exhibit considerable variation in the contour of the apex of tergite VIII, ranging from definitely truncate to bluntly pointed.

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Jordan, K.: Notes on North American fleas. Novit. Zool., 35: 28–39 (1929).
Jordan, K.: On some North American Siphonaptera. Novit. Zool., 40: 262–271 (1937).

## DEATHS DURING WEEK ENDED JUNE 25, 1938

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

| | Week ended June 25, 1938 | Corresponding week, 1937 |
|---|---|---|
| Data from 87 large cities of the United States: | | |
| Total deaths | 7,425 | [1] 7,612 |
| Average for 3 prior years | 7,688 | |
| Total deaths, first 25 weeks of year | 213,672 | 234,774 |
| Deaths under 1 year of age | 455 | [1] 524 |
| Average for 3 prior years | 512 | |
| Deaths under 1 year of age, first 25 weeks of year | 13,267 | 14,534 |
| Data from industrial insurance companies: | | |
| Policies in force | 69,280,198 | 69,933,379 |
| Number of death claims | 11,718 | 12,242 |
| Death claims per 1,000 policies in force, annual rate | 8.8 | 9.1 |
| Death claims per 1,000 policies, first 25 weeks of year, annual rate | 9.7 | 10.7 |

[1] Data for 86 cities.

# PREVALENCE OF DISEASE

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

# UNITED STATES

## CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

In these and the following tables, a zero (0) indicates a positive report and has the same significance as any other figure, while leaders (------) represent no report, with the implication that cases or deaths may have occurred but were not reported to the State health officer.

*Cases of certain diseases reported by telegraph by State health officers for the week ended July 2, 1938, rates per 100,000 population (annual basis), and comparison with 1937 and 5-year median*

| Division and State | Diphtheria | | | | Influenza | | | | Measles | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Week ended— | | | | Week ended— | | | | Week ended— | | | |
| | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median |
| New England: | | | | | | | | | | | | |
| Maine | 0 | 0 | 0 | 0 | ------ | ------ | 2 | 1 | 493 | 81 | 19 | 19 |
| New Hampshire | 0 | 0 | 0 | 0 | ------ | ------ | ------ | ------ | 174 | 17 | 30 | 26 |
| Vermont | 0 | 0 | 1 | 1 | ------ | ------ | ------ | ------ | 762 | 56 | 2 | 35 |
| Massachusetts | 0 | 0 | 5 | 10 | ------ | ------ | ------ | ------ | 542 | 460 | 288 | 440 |
| Rhode Island | 15 | 2 | 1 | 1 | ------ | ------ | ------ | ------ | 8 | 1 | 37 | 20 |
| Connecticut | 0 | 0 | 4 | 4 | 15 | 5 | 1 | 1 | 78 | 26 | 45 | 99 |
| Middle Atlantic: | | | | | | | | | | | | |
| New York | 7 | 17 | 31 | 34 | [1] 1.4 | [1] 2 | [1] 5 | [1] 3 | 799 | 1,986 | 894 | 894 |
| New Jersey | 5 | 4 | 10 | 12 | 2 | 2 | 2 | 3 | 279 | 232 | 393 | 393 |
| Pennsylvania | 11 | 22 | 19 | 31 | ------ | ------ | ------ | ------ | 518 | 1,010 | 927 | 988 |
| East North Central: | | | | | | | | | | | | |
| Ohio | 18 | 23 | 20 | 20 | ------ | ------ | 9 | 14 | 418 | 540 | 1,342 | 971 |
| Indiana | 9 | 6 | 10 | 10 | ------ | ------ | 2 | 9 | 66 | 44 | 271 | 69 |
| Illinois | 13 | 20 | 21 | 37 | 5 | 7 | 8 | 14 | 120 | 182 | 490 | 490 |
| Michigan [2] | 5 | 5 | 17 | 12 | ------ | ------ | ------ | ------ | 1,086 | 1,006 | 218 | 214 |
| Wisconsin | 4 | 2 | 2 | 3 | 36 | 20 | 14 | 11 | 2,219 | 1,245 | 53 | 159 |
| West North Central: | | | | | | | | | | | | |
| Minnesota | 6 | 3 | 0 | 5 | ------ | ------ | ------ | 1 | 293 | 149 | 2 | 63 |
| Iowa [3] | 0 | 0 | 2 | 4 | ------ | ------ | ------ | ------ | 337 | 165 | 15 | 25 |
| Missouri | 1.3 | 1 | 6 | 16 | ------ | ------ | 18 | 8 | 24 | 18 | 80 | 87 |
| North Dakota | 37 | 5 | 0 | 1 | 59 | 8 | ------ | ------ | 283 | 39 | 1 | 10 |
| South Dakota | 8 | 1 | 3 | 1 | ------ | ------ | ------ | ------ | 0 | 0 | ------ | 4 |
| Nebraska | 4 | 1 | 1 | 2 | ------ | ------ | ------ | ------ | 134 | 35 | 6 | 21 |
| Kansas | 3 | 1 | 11 | 5 | ------ | ------ | 1 | 1 | 187 | 67 | 15 | 88 |
| South Atlantic: | | | | | | | | | | | | |
| Delaware | 0 | 0 | 0 | 1 | ------ | ------ | ------ | ------ | 80 | 4 | 3 | 9 |
| Maryland [2] [3] | 19 | 6 | 7 | 4 | ------ | ------ | ------ | 1 | 121 | 39 | 79 | 79 |
| District of Columbia | 17 | 2 | 5 | 5 | ------ | ------ | ------ | ------ | 158 | 19 | 42 | 42 |
| Virginia | 6 | 3 | 8 | 6 | ------ | ------ | ------ | ------ | 243 | 126 | 64 | 97 |
| West Virginia | 11 | 4 | 7 | 7 | 17 | 6 | 25 | 11 | 243 | 87 | 30 | 30 |
| North Carolina [3] [4] | 22 | 15 | 5 | 5 | 7 | 5 | ------ | 1 | 493 | 330 | 134 | 134 |
| South Carolina | 6 | 2 | 0 | 4 | 231 | 83 | 28 | 41 | 206 | 74 | 18 | 18 |
| Georgia [4] | 7 | 4 | 4 | 7 | ------ | ------ | ------ | ------ | 0 | 0 | ------ | ------ |
| Florida [4] | 22 | 7 | 1 | 2 | ------ | ------ | 1 | 1 | 150 | 48 | ------ | 7 |

See footnotes at end of table.

*Cases of certain diseases reported by telegraph by State health officers for the week ended July 2, 1938, rates per 100,000 population (annual basis), and comparison with 1937 and 5-year median*—Continued

| Division and State | Diphtheria | | | | Influenza | | | | Measles | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Week ended— | | | | Week ended— | | | | Week ended— | | | |
| | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median |
| East South Central: | | | | | | | | | | | | |
| Kentucky | 5 | 3 | 1 | 3 | 16 | 9 | ------- | 1 | 127 | 71 | 98 | 25 |
| Tennessee | 16 | 9 | 3 | 3 | 7 | 4 | 1 | 8 | 58 | 32 | 71 | 71 |
| Alabama [4] | 5 | 3 | 5 | 8 | 29 | 16 | 5 | 7 | 112 | 62 | 20 | 27 |
| Mississippi [2] | 10 | 4 | 3 | 5 | ------- | ------- | ------- | ------- | ------- | ------- | ------- | ------- |
| West South Central: | | | | | | | | | | | | |
| Arkansas | 8 | 3 | 5 | 4 | 28 | 11 | 2 | 2 | 143 | 56 | 7 | 8 |
| Louisiana [4] | 12 | 5 | 6 | 9 | 17 | 7 | 13 | 8 | 27 | 11 | 3 | 5 |
| Oklahoma | 14 | 7 | 1 | 4 | 49 | 24 | 5 | 9 | 61 | 30 | 23 | 14 |
| Texas [4] | 14 | 16 | 20 | 26 | 95 | 113 | 76 | 59 | 41 | 49 | 171 | 147 |
| Mountain: | | | | | | | | | | | | |
| Montana | 10 | 1 | 1 | 2 | ------- | ------- | ------- | 2 | 474 | 49 | 4 | 4 |
| Idaho [3] | 0 | 0 | 0 | 0 | 21 | 2 | 2 | ----- | 21 | 2 | 8 | 4 |
| Wyoming [3] [5] | 0 | 0 | 0 | 0 | ------- | ------- | ------- | ------- | 44 | 2 | ------- | 4 |
| Colorado [5] | 63 | 13 | 10 | 9 | ------- | ------- | ------- | ------- | 234 | 48 | 49 | 49 |
| New Mexico | 12 | 1 | 1 | 1 | 49 | 4 | ------- | ------- | 222 | 18 | 23 | 23 |
| Arizona | 51 | 4 | 3 | 0 | 114 | 9 | 15 | ------- | 152 | 12 | 14 | 14 |
| Utah [2] [3] | 0 | 0 | 0 | 0 | ------- | ------- | ------- | ------- | 1,808 | 180 | 58 | 6 |
| Pacific: | | | | | | | | | | | | |
| Washington [3] | 0 | 0 | 1 | 0 | ------- | ------- | ------- | ------- | 47 | 15 | 72 | 124 |
| Oregon [3] | 20 | 4 | 4 | 0 | 51 | 10 | 10 | 10 | 203 | 40 | 9 | 15 |
| California | 14 | 16 | 25 | 31 | 9 | 11 | 14 | 17 | 400 | 472 | 97 | 534 |
| Total | 10 | 245 | 290 | 420 | 18 | 358 | 259 | 262 | 379 | 9,235 | 6,225 | 6,968 |
| First 26 weeks | 191 | 2,185 | 11,649 | 15,531 | 84 | 43,690 | 272,835 | 138,583 | 1,166 | 739,432 | 224,733 | 327,352 |

| Division and State | Meningitis, meningococcus | | | | Poliomyelitis | | | | Scarlet fever | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Week ended— | | | | Week ended— | | | | Week ended— | | | |
| | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median |
| New England: | | | | | | | | | | | | |
| Maine | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 55 | 9 | 11 | 11 |
| New Hampshire | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 6 | 5 |
| Vermont | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 41 | 3 | 5 | 4 |
| Massachusetts | 0 | 0 | 2 | 2 | 0 | 0 | 2 | 2 | 153 | 130 | 122 | 143 |
| Rhode Island | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 38 | 5 | 10 | 10 |
| Connecticut | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 72 | 24 | 37 | 33 |
| Middle Atlantic: | | | | | | | | | | | | |
| New York | 2 | 6 | 4 | 4 | 0.8 | 2 | 2 | 3 | 91 | 226 | 235 | 292 |
| New Jersey | 0 | 0 | 0 | 1 | 0 | 0 | 2 | 2 | 35 | 29 | 57 | 75 |
| Pennsylvania | 2 | 4 | 8 | 5 | 0 | 0 | 1 | 1 | 147 | 286 | 284 | 253 |
| East North Central: | | | | | | | | | | | | |
| Ohio | 0.7 | 1 | 2 | 2 | 0.7 | 1 | 2 | 1 | 90 | 116 | 152 | 204 |
| Indiana | 3 | 2 | 1 | 1 | 0 | 0 | 0 | 0 | 26 | 17 | 42 | 41 |
| Illinois | 0.6 | 1 | 1 | 7 | 3 | 4 | 2 | 3 | 102 | 154 | 183 | 209 |
| Michigan [2] | 0 | 0 | 2 | 2 | 0 | 0 | 1 | 1 | 136 | 126 | 337 | 196 |
| Wisconsin | 4 | 2 | 0 | 0 | 0 | 0 | 0 | 1 | 107 | 60 | 113 | 173 |
| West North Central: | | | | | | | | | | | | |
| Minnesota | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 53 | 27 | 49 | 49 |
| Iowa [3] | 0 | 0 | 3 | 1 | 2 | 1 | 0 | 0 | 31 | 15 | 52 | 31 |
| Missouri | 4 | 3 | 0 | 1 | 0 | 0 | 1 | 0 | 21 | 16 | 35 | 25 |
| North Dakota | 0 | 0 | 0 | 0 | 7 | 1 | 0 | 0 | 15 | 2 | 5 | 5 |
| South Dakota | 0 | 0 | 0 | 0 | 8 | 1 | 0 | 0 | 38 | 5 | 11 | 8 |
| Nebraska | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 61 | 16 | 8 | 10 |
| Kansas | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 56 | 20 | 46 | 23 |

See footnotes at end of table.

*Cases of certain diseases reported by telegraph by State health officers for the week ended July 2, 1938, rates per 100,000 population (annual basis), and comparison with 1937 and 5-year median*—Continued

| Division and State | Meningitis, meningococcus | | | | Poliomyelitis | | | | Scarlet fever | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Week ended— | | | | Week ended— | | | | Week ended— | | | |
| | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median |
| South Atlantic: | | | | | | | | | | | | |
| Delaware | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 60 | 3 | 2 | 2 |
| Maryland [2 3] | 3 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 59 | 19 | 14 | 22 |
| Dist. of Columbia | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 67 | 8 | 4 | 6 |
| Virginia | 10 | 5 | 4 | 4 | 4 | 2 | 1 | 1 | 8 | 4 | 1 | 12 |
| West Virginia | 3 | 1 | 4 | 2 | 0 | 0 | 1 | 1 | 36 | 13 | 18 | 18 |
| North Carolina [3 4] | 0 | 0 | 3 | 3 | 3 | 2 | 7 | 1 | 24 | 16 | 14 | 14 |
| South Carolina | 3 | 1 | 0 | 0 | 0 | 0 | 1 | 1 | 3 | 1 | 1 | 1 |
| Georgia [4] | 0 | 0 | 0 | 0 | 5 | 3 | 4 | 0 | 8 | 5 | 10 | 4 |
| Florida [4] | 0 | 0 | 0 | 0 | 3 | 1 | 0 | 0 | 0 | 0 | 7 | 2 |
| East South Central: | | | | | | | | | | | | |
| Kentucky | 11 | 6 | 1 | 1 | 2 | 1 | 2 | 0 | 14 | 8 | 17 | 12 |
| Tennessee | 0 | 0 | 2 | 2 | 2 | 1 | 19 | 1 | 11 | 6 | 1 | 5 |
| Alabama [4] | 7 | 4 | 4 | 1 | 9 | 5 | 2 | 2 | 13 | 7 | 4 | 4 |
| Mississippi [3] | 0 | 0 | 1 | 0 | 8 | 3 | 30 | 0 | 5 | 2 | 5 | 5 |
| West South Central: | | | | | | | | | | | | |
| Arkansas | 0 | 0 | 3 | 0 | 0 | 0 | 26 | 0 | 5 | 2 | 9 | 1 |
| Louisiana [4] | 2 | 1 | 0 | 1 | 7 | 3 | 7 | 1 | 15 | 6 | 8 | 8 |
| Oklahoma | 2 | 1 | 0 | 0 | 0 | 0 | 7 | 0 | 14 | 7 | 7 | 7 |
| Texas [4] | 0 | 0 | 3 | 3 | 0.8 | 1 | 23 | 2 | 35 | 42 | 40 | 32 |
| Mountain: | | | | | | | | | | | | |
| Montana | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 39 | 4 | 9 | 10 |
| Idaho [3] | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 11 | 2 |
| Wyoming [3 5] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 155 | 7 | 12 | 3 |
| Colorado [5] | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 83 | 17 | 10 | 13 |
| New Mexico | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 74 | 6 | 5 | 5 |
| Arizona | 13 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 38 | 3 | 1 | 7 |
| Utah [3] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 171 | 17 | 11 | 6 |
| Pacific: | | | | | | | | | | | | |
| Washington [3] | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 1 | 35 | 11 | 13 | 19 |
| Oregon [3] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 102 | 20 | 12 | 15 |
| California | 0.8 | 1 | 6 | 5 | 0.8 | 1 | 7 | 7 | 82 | 97 | 93 | 113 |
| Total | 2 | 41 | 61 | 61 | 1.3 | 33 | 158 | 158 | 65 | 1,617 | 2,139 | 2,228 |
| First 26 weeks | 3 | 1,898 | 3,709 | 3,630 | 0.8 | 547 | 815 | 815 | 202 | 130,360 | 157,273 | 157,273 |

| Division and State | Smallpox | | | | Typhoid and paratyphoid fever | | | | Whooping cough | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Week ended— | | | | Week ended— | | | | Week ended— | |
| | July 2, 1938, rate | July 2 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases |
| New England: | | | | | | | | | | |
| Maine | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 2 | 280 | 46 |
| New Hampshire | 0 | 0 | 0 | 0 | 10 | 1 | 1 | 0 | ------ | ------ |
| Vermont | 0 | 0 | 0 | 0 | 14 | 1 | 0 | 0 | 354 | 26 |
| Massachusetts | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 93 | 79 |
| Rhode Island | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 100 | 13 |
| Connecticut | 0 | 0 | 0 | 0 | 6 | 2 | 0 | 1 | 302 | 101 |
| Middle Atlantic: | | | | | | | | | | |
| New York | 0 | 0 | 0 | 0 | 2 | 5 | 19 | 13 | 190 | 472 |
| New Jersey | 0 | 0 | 0 | 0 | 5 | 4 | 1 | 3 | 193 | 161 |
| Pennsylvania | 0 | 0 | 0 | 0 | 13 | 25 | 17 | 17 | 170 | 332 |

See footnotes at end of table.

*Cases of certain diseases reported by telegraph by State health officers for the week ended July 2, 1938, rates per 100,000 population (annual basis), and comparison with 1937 and 5-year median*—Continued

| Division and State | Smallpox | | | | Typhoid and paratyphoid fever | | | | Whooping cough | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Week ended— | | | | Week ended— | | | | Week ended— | |
| | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases | July 3, 1937, cases | 1933–1937 median | July 2, 1938, rate | July 2, 1938, cases |
| East North Central: | | | | | | | | | | |
| Ohio | 0.7 | 1 | 8 | 0 | 6 | 8 | 8 | 12 | 165 | 213 |
| Indiana | 29 | 19 | 9 | 2 | 3 | 2 | 3 | 5 | 20 | 13 |
| Illinois | 14 | 21 | 28 | 1 | 11 | 16 | 8 | 21 | 162 | 245 |
| Michigan [2] | 0 | 0 | 0 | 0 | 3 | 3 | 4 | 4 | 310 | 287 |
| Wisconsin | 4 | 2 | 1 | 6 | 0 | 0 | 0 | 1 | 369 | 207 |
| West North Central: | | | | | | | | | | |
| Minnesota | 45 | 23 | 7 | 3 | 0 | 0 | 0 | 0 | 49 | 25 |
| Iowa [3] | 25 | 12 | 20 | 12 | 0 | 0 | 3 | 1 | 39 | 19 |
| Missouri | 22 | 17 | 10 | 4 | 4 | 3 | 31 | 21 | 39 | 30 |
| North Dakota | 22 | 3 | 12 | 2 | 0 | 0 | 0 | 0 | 140 | 19 |
| South Dakota | 30 | 4 | 4 | 3 | 0 | 0 | 0 | 0 | 45 | 6 |
| Nebraska | 0 | 0 | 2 | 4 | 0 | 0 | 0 | 0 | 34 | 9 |
| Kansas | 20 | 7 | 6 | 6 | 0 | 0 | 6 | 6 | 308 | 110 |
| South Atlantic: | | | | | | | | | | |
| Delaware | 0 | 0 | 0 | 0 | 60 | 3 | 2 | 1 | 220 | 11 |
| Maryland [2 3] | 0 | 0 | 0 | 0 | 9 | 3 | 9 | 9 | 174 | 56 |
| District of Columbia | 0 | 0 | 0 | 0 | 0 | 0 | 7 | 0 | 42 | 5 |
| Virginia | 0 | 0 | 0 | 0 | 13 | 7 | 6 | 18 | 104 | 54 |
| West Virginia | 0 | 0 | 0 | 0 | 17 | 6 | 7 | 7 | 215 | 77 |
| North Carolina [3 4] | 0 | 0 | 0 | 0 | 30 | 20 | 30 | 30 | 532 | 356 |
| South Carolina | 0 | 0 | 0 | 0 | 61 | 22 | 20 | 20 | 234 | 84 |
| Georgia [4] | 0 | 0 | 0 | 0 | 42 | 25 | 50 | 38 | 74 | 44 |
| Florida [4] | 0 | 0 | 0 | 0 | 6 | 2 | 4 | 4 | 53 | 17 |
| East South Central: | | | | | | | | | | |
| Kentucky | 0 | 0 | 0 | 0 | 32 | 18 | 13 | 18 | 82 | 46 |
| Tennessee | 2 | 1 | 1 | 0 | 20 | 11 | 32 | 31 | 81 | 45 |
| Alabama [4] | 0 | 0 | 0 | 0 | 25 | 14 | 10 | 20 | 92 | 51 |
| Mississippi [2] | 8 | 3 | 0 | 0 | 39 | 15 | 17 | 17 | ------- | ------- |
| West South Central: | | | | | | | | | | |
| Arkansas | 3 | 1 | 0 | 0 | 43 | 17 | 29 | 17 | 94 | 37 |
| Louisiana [4] | 0 | 0 | 0 | 0 | 51 | 21 | 19 | 23 | 105 | 43 |
| Oklahoma | 16 | 8 | 3 | 1 | 20 | 10 | 7 | 9 | 51 | 25 |
| Texas [4] | 11 | 13 | 1 | 2 | 45 | 53 | 35 | 35 | 218 | 258 |
| Mountain: | | | | | | | | | | |
| Montana | 10 | 1 | 11 | 2 | 10 | 1 | 1 | 1 | 503 | 52 |
| Idaho [3] | 137 | 13 | 7 | 3 | 32 | 3 | 2 | 1 | 53 | 5 |
| Wyoming [3 5] | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 355 | 16 |
| Colorado [5] | 0 | 0 | 2 | 1 | 10 | 2 | 2 | 2 | 156 | 32 |
| New Mexico | 49 | 4 | 2 | 0 | 111 | 9 | 2 | 6 | 185 | 15 |
| Arizona | 25 | 2 | 0 | 0 | 114 | 9 | 4 | 3 | ------- | ------- |
| Utah [2 3] | 0 | 0 | 1 | 0 | 10 | 1 | 0 | 0 | 964 | 96 |
| Pacific: | | | | | | | | | | |
| Washington [3] | 9 | 3 | 2 | 6 | 9 | 3 | 1 | 2 | 208 | 66 |
| Oregon [3] | 36 | 7 | 5 | 5 | 20 | 4 | 2 | 2 | 132 | 26 |
| California | 19 | 22 | 9 | 2 | 8 | 10 | 5 | 8 | 175 | 206 |
| Total | 8 | 187 | 151 | 151 | 14 | 359 | 421 | 495 | 170 | 4,136 |
| First 26 weeks | 19 | 11,937 | 7,370 | 4,852 | 7 | 4,299 | 3,791 | 4,583 | 176 | 111,737 |

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Rocky Mountain spotted fever, week ended July 2, 1938, 17 cases as follows: Iowa, 1; Maryland, 2; North Carolina, 4; Montana, 1; Idaho, 2; Wyoming, 1; Utah, 4; Washington, 1; Oregon, 1.
[4] Typhus fever, week ended July 2, 1938, 32 cases as follows: North Carolina, 1; Georgia, 10; Florida, 3; Alabama, 9; Louisiana, 1; Texas, 8.
[5] Colorado tick fever, week ended July 2, 1938, 7 cases as follows: Wyoming, 4; Colorado, 3.

## 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 | Meningitis, meningococcus | Diphtheria | Influenza | Malaria | Measles | Pellagra | Poliomyelitis | Scarlet fever | Smallpox | Typhoid fever |
|---|---|---|---|---|---|---|---|---|---|---|
| *April 1938* | | | | | | | | | | |
| South Carolina | ---- | 58 | 848 | 450 | 1,321 | 147 | 2 | 17 | 0 | 8 |
| *May 1938* | | | | | | | | | | |
| Hawaii Territory | 1 | 15 | 14 | ---- | 54 | ---- | 0 | 2 | 0 | 5 |
| Massachusetts | 4 | 8 | ---- | ---- | 1,350 | 1 | 0 | 1,525 | 0 | 1 |
| Montana | 1 | 5 | 58 | ---- | 290 | ---- | 0 | 59 | 37 | 3 |
| Oregon | 2 | 12 | 140 | ---- | 167 | ---- | 1 | 125 | 55 | 8 |
| Puerto Rico | ---- | 36 | 455 | 2,294 | 15 | ---- | 0 | ---- | 0 | 53 |
| South Carolina | ---- | 54 | 308 | 517 | 563 | 193 | 0 | 8 | 0 | 17 |
| Washington | 1 | 2 | 9 | ---- | 150 | ---- | 0 | 91 | 81 | 5 |

*April 1938*

| | Cases |
|---|---|
| South Carolina: | |
| Chickenpox | 146 |
| Dengue | 2 |
| Diarrhea | 627 |
| Hookworm disease | 90 |
| Mumps | 101 |
| Ophthalmia neonatorum | 9 |
| Rabies in animals | 38 |
| Tularaemia | 1 |
| Typhus fever | 2 |
| Undulant fever | 1 |
| Whooping cough | 358 |

*May 1938*

| | Cases |
|---|---|
| Actinomycosis: | |
| Montana | 1 |
| Anthrax: | |
| Massachusetts | 1 |
| Washington | 1 |
| Chickenpox: | |
| Hawaii Territory | 58 |
| Massachusetts | 1,129 |
| Montana | 146 |
| Oregon | 320 |
| Puerto Rico | 139 |
| South Carolina | 35 |
| Washington | 773 |
| Conjunctivitis: | |
| Washington | 1 |
| Conjunctivitis (follicular): | |
| Hawaii Territory | 1 |
| Diarrhea: | |
| South Carolina | 927 |
| Dysentery: | |
| Hawaii Territory (amoebic) | 2 |
| Massachusetts (bacillary) | 20 |
| Oregon (amoebic) | 1 |
| Puerto Rico | 22 |
| South Carolina | 8 |
| Encephalitis, epidemic or lethargic: | |
| Hawaii Territory | 3 |
| Massachusets | 1 |

*May 1938*—Continued

| | Cases |
|---|---|
| Encephalitis, epidemic or lethargic—Continued. | |
| Montana | 3 |
| South Carolina | 1 |
| Washington | 2 |
| Filariasis: | |
| Puerto Rico | 2 |
| German measles: | |
| Massachusetts | 74 |
| Montana | 3 |
| South Carolina | 1 |
| Washington | 19 |
| Hookworm disease: | |
| Hawaii Territory | 1 |
| South Carolina | 81 |
| Impetigo contagiosa: | |
| Hawaii Territory | 8 |
| Montana | 4 |
| Oregon | 41 |
| Jaundice (infectious): | |
| Oregon | 3 |
| Leprosy: | |
| Hawaii Territory | 3 |
| Montana | 1 |
| Mumps: | |
| Hawaii Territory | 53 |
| Massachusetts | 977 |
| Montana | 86 |
| Oregon | 75 |
| Puerto Rico | 1 |
| South Carolina | 53 |
| Washington | 696 |
| Ophthalmia neonatorum: | |
| Massachusetts | 64 |
| Puerto Rico | 7 |
| South Carolina | 3 |
| Paratyphoid fever: | |
| Massachusetts | 20 |
| South Carolina | 1 |
| Puerperal fever: | |
| Puerto Rico | 9 |
| Washington | 3 |
| Rabies in animals: | |
| Massachusetts | 6 |
| Oregon | 7 |
| South Carolina | 22 |
| Washington | 16 |

*May 1938*—Continued

| | Cases |
|---|---|
| Rocky Mountain spotted fever: | |
| Montana | 5 |
| Oregon | 12 |
| Scabies: | |
| Montana | 3 |
| Oregon | 50 |
| Septic sore throat: | |
| Hawaii Territory | 2 |
| Massachusetts | 22 |
| Montana | 9 |
| Oregon | 6 |
| Washington | 17 |
| Tetanus: | |
| Hawaii Territory | 1 |
| Massachusetts | 2 |
| Puerto Rico | 9 |
| South Carolina | 1 |
| Tetanus (infantile): | |
| Puerto Rico | 1 |
| Trachoma: | |
| Hawaii Territory | 4 |
| Montana | 9 |
| Trichinosis: | |
| Massachusetts | 1 |
| Tularaemia: | |
| Montana | 2 |
| Oregon | 1 |
| Typhus fever: | |
| Hawaii Territory | 3 |
| Undulant fever: | |
| Massachusetts | 2 |
| Montana | 1 |
| Oregon | 2 |
| South Carolina | 1 |
| Washington | 2 |
| Vincent's infection: | |
| Montana | 1 |
| Oregon | 11 |
| Whooping cough: | |
| Hawaii Territory | 143 |
| Massachusetts | 478 |
| Montana | 209 |
| Oregon | 85 |
| Puerto Rico | 149 |
| South Carolina | 288 |
| Washington | 674 |

## WEEKLY REPORTS FROM CITIES

*City reports for week ended June 25, 1938*

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

| State and city | Diphtheria cases | Influenza | | Measles cases | Pneumonia deaths | Scarlet fever cases | Smallpox cases | Tuberculosis deaths | Typhoid fever cases | Whooping cough cases | Deaths, all causes |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Cases | Deaths | | | | | | | | |
| Data for 90 cities: | | | | | | | | | | | |
| 5-year average | 149 | 52 | 19 | 3,282 | 390 | 1,095 | 11 | 388 | 42 | 1,260 | -------- |
| Current week [1] | 110 | 33 | 10 | 2,874 | 279 | 725 | 11 | 356 | 40 | 1,369 | -------- |
| Maine: | | | | | | | | | | | |
| Portland | 0 | ------ | 1 | 16 | 0 | 0 | 0 | 0 | 0 | 4 | 19 |
| New Hampshire: | | | | | | | | | | | |
| Concord | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 15 |
| Manchester | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 13 |
| Nashua | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 7 |
| Vermont: | | | | | | | | | | | |
| Barre | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 3 |
| Burlington | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 9 |
| Rutland | 0 | ------ | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 7 |
| Massachusetts: | | | | | | | | | | | |
| Boston | 1 | ------ | 1 | 127 | 9 | 50 | 0 | 7 | 0 | 13 | 220 |
| Fall River | 0 | ------ | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 29 |
| Springfield | 0 | ------ | 0 | 115 | 2 | 0 | 0 | 1 | 0 | 17 | 35 |
| Worcester | 0 | ------ | 0 | 1 | 4 | 15 | 0 | 0 | 0 | 5 | 47 |
| Rhode Island: | | | | | | | | | | | |
| Pawtucket | 1 | ------ | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 | 13 |
| Providence | 0 | ------ | 0 | 0 | 2 | 11 | 0 | 1 | 1 | 18 | 47 |
| Connecticut: | | | | | | | | | | | |
| Bridgeport | 0 | ------ | 0 | 4 | 0 | 1 | 0 | 1 | 0 | 3 | 28 |
| Hartford | 0 | ------ | 0 | 2 | 2 | 12 | 0 | 0 | 1 | 5 | 27 |
| New Haven | 0 | ------ | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 6 | 46 |
| New York: | | | | | | | | | | | |
| Buffalo | 1 | ------ | 0 | 2 | 2 | 29 | 0 | 10 | 0 | 28 | 115 |
| New York | 25 | 2 | 1 | 1,095 | 52 | 118 | 0 | 80 | 5 | 264 | 1,230 |
| Rochester | 2 | ------ | 0 | 34 | 1 | 10 | 0 | 1 | 2 | 4 | 47 |
| Syracuse | 0 | ------ | 0 | 67 | 4 | 2 | 0 | 1 | 0 | 7 | 48 |
| New Jersey: | | | | | | | | | | | |
| Camden | 0 | ------ | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 3 | 29 |
| Newark | 0 | ------ | 1 | 13 | 2 | 5 | 0 | 1 | 0 | 34 | 77 |
| Trenton | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 6 | 24 |
| Pennsylvania: | | | | | | | | | | | |
| Philadelphia | 1 | ------ | 0 | 161 | 14 | 37 | 0 | 26 | 2 | 32 | 423 |
| Pittsburgh | 4 | ------ | 1 | 12 | 7 | 22 | 0 | 8 | 0 | 53 | 118 |
| Reading | 1 | ------ | 0 | 5 | 2 | 0 | 0 | 2 | 0 | 1 | 18 |
| Scranton | 0 | ------ | -------- | 2 | ------- | 0 | 0 | ------- | 0 | 1 | -------- |
| Ohio: | | | | | | | | | | | |
| Cincinnati | 9 | ------ | 0 | 9 | 2 | 0 | 0 | 6 | 0 | 10 | 122 |
| Cleveland | 4 | 10 | 0 | 103 | 3 | 24 | 0 | 16 | 2 | 52 | 200 |
| Columbus | 1 | ------ | 0 | 3 | 1 | 3 | 0 | 2 | 0 | 0 | 74 |
| Toledo | 0 | ------ | 0 | 33 | 1 | 7 | 0 | 4 | 0 | 32 | 51 |
| Indiana: | | | | | | | | | | | |
| Anderson | 0 | ------ | 0 | 0 | 1 | 1 | 14 | 0 | 0 | 0 | 12 |
| Fort Wayne | 0 | ------ | 0 | 2 | 2 | 0 | 0 | 1 | 0 | 0 | 18 |
| Indianapolis | 2 | ------ | 0 | 29 | 12 | 12 | 3 | 3 | 1 | 4 | 76 |
| Muncie | 0 | ------ | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 11 |
| South Bend | 0 | ------ | 0 | 11 | 2 | 0 | 0 | 0 | 0 | 0 | 14 |
| Terre Haute | 0 | ------ | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 15 |
| Illinois: | | | | | | | | | | | |
| Alton | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 11 |
| Chicago | 17 | ------ | 0 | 82 | 20 | 85 | 0 | 35 | 1 | 141 | 636 |
| Elgin | 0 | ------ | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 1 | 14 |
| Moline | 0 | ------ | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 10 |
| Springfield | 0 | ------ | 0 | 0 | 0 | 6 | 2 | 0 | 0 | 8 | 24 |
| Michigan: | | | | | | | | | | | |
| Detroit | 1 | ------ | 0 | 66 | 5 | 76 | 0 | 14 | 0 | 136 | 232 |
| Flint | 0 | ------ | 0 | 37 | 4 | 8 | 0 | 2 | 0 | 8 | 26 |
| Grand Rapids | 0 | ------ | 0 | 84 | 0 | 5 | 0 | 1 | 0 | 0 | 29 |
| Wisconsin: | | | | | | | | | | | |
| Kenosha | 0 | ------ | 0 | 31 | 0 | 1 | 0 | 0 | 0 | 3 | 9 |
| Madison | 0 | ------ | 0 | 110 | 0 | 2 | 0 | 0 | 0 | 3 | 7 |
| Milwaukee | 0 | ------ | 0 | 10 | 4 | 24 | 0 | 3 | 0 | 102 | 89 |
| Racine | 0 | ------ | 0 | 19 | 0 | 8 | 0 | 0 | 0 | 4 | 16 |
| Superior | 0 | ------ | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 1 | 6 |

[1] Figures for Boise, Idaho, estimated; report not received.

*City reports for week ended June 25, 1938*—Continued

| State and city | Diphtheria cases | Influenza | | Measles cases | Pneumonia deaths | Scarlet fever cases | Smallpox cases | Tuberculosis deaths | Typhoid fever cases | Whooping cough cases | Deaths all causes |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Cases | Deaths | | | | | | | | |
| **Minnesota:** | | | | | | | | | | | |
| Duluth | 0 | ------ | 1 | 52 | 1 | 1 | 0 | 0 | 0 | 18 | 22 |
| Minneapolis | 1 | ------ | 0 | 47 | 1 | 6 | 2 | 1 | 0 | 5 | 83 |
| St. Paul | 0 | ------ | 0 | 6 | 1 | 4 | 0 | 2 | 0 | 11 | ------ |
| **Iowa:** | | | | | | | | | | | |
| Cedar Rapids | 0 | ------ | ------ | 21 | ------ | 1 | 0 | ------ | 0 | 1 | ------ |
| Davenport | 0 | ------ | ------ | 0 | ------ | 0 | 0 | ------ | 1 | 0 | ------ |
| Des Moines | 0 | ------ | 0 | 13 | 0 | 5 | 2 | 0 | 0 | 0 | 28 |
| Sioux City | 0 | ------ | ------ | 55 | ------ | 1 | 0 | ------ | 0 | 1 | ------ |
| Waterloo | 0 | ------ | ------ | 2 | ------ | 3 | 0 | ------ | 0 | 2 | ------ |
| **Missouri:** | | | | | | | | | | | |
| Kansas City | 0 | ------ | 0 | 2 | 4 | 11 | 0 | 6 | 0 | 5 | 101 |
| St. Joseph | 1 | ------ | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 13 |
| St. Louis | 1 | ------ | 0 | 4 | 8 | 11 | 0 | 9 | 1 | 1 | 233 |
| **North Dakota:** | | | | | | | | | | | |
| Fargo | 0 | ------ | 0 | 7 | 0 | 0 | 0 | 0 | 0 | 5 | 6 |
| Grand Forks | 0 | ------ | ------ | 4 | ------ | 0 | 0 | ------ | 0 | 2 | ------ |
| Minot | 0 | ------ | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 1 | 5 |
| **South Dakota:** | | | | | | | | | | | |
| Aberdeen | 0 | ------ | ------ | 1 | ------ | 0 | 1 | ------ | 0 | 0 | ------ |
| Sioux Falls | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 12 |
| **Nebraska:** | | | | | | | | | | | |
| Omaha | 0 | ------ | 0 | 48 | 2 | 2 | 0 | 1 | 0 | 0 | 57 |
| **Kansas:** | | | | | | | | | | | |
| Lawrence | 0 | ------ | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 5 | 3 |
| Topeka | 0 | ------ | 0 | 13 | 0 | 0 | 0 | 0 | 0 | 28 | 13 |
| Wichita | 1 | ------ | 0 | 15 | 1 | 1 | 1 | 0 | 0 | 24 | 30 |
| **Delaware:** | | | | | | | | | | | |
| Wilmington | 0 | ------ | 0 | 0 | 2 | 1 | 0 | 1 | 0 | 7 | 30 |
| **Maryland:** | | | | | | | | | | | |
| Baltimore | 1 | 1 | 0 | 28 | 7 | 34 | 0 | 14 | 0 | 34 | 182 |
| Cumberland | 0 | ------ | 0 | 9 | 0 | 1 | 0 | 0 | 0 | 0 | 7 |
| Frederick | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| **Dist. of Col.:** | | | | | | | | | | | |
| Washington | 8 | ------ | 0 | 22 | 4 | 13 | 0 | 10 | 0 | 8 | 143 |
| **Virginia:** | | | | | | | | | | | |
| Lynchburg | 1 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11 |
| Norfolk | 0 | 2 | 0 | 0 | 2 | 2 | 0 | 0 | 0 | 2 | 19 |
| Richmond | 0 | ------ | 0 | 77 | 2 | 0 | 0 | 3 | 0 | 0 | 49 |
| Roanoke | 0 | ------ | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 2 | 10 |
| **West Virginia:** | | | | | | | | | | | |
| Charleston | 0 | ------ | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 14 |
| Huntington | 0 | ------ | ------ | 0 | ------ | 0 | 0 | ------ | 0 | 0 | ------ |
| Wheeling | 0 | ------ | 0 | 0 | 0 | 2 | 0 | 1 | 0 | 2 | 20 |
| **North Carolina:** | | | | | | | | | | | |
| Gastonia | 0 | ------ | ------ | 4 | ------ | 0 | 0 | ------ | 0 | 5 | ------ |
| Raleigh | 0 | ------ | 0 | 3 | 4 | 0 | 0 | 1 | 0 | 14 | 22 |
| Wilmington | 0 | ------ | 0 | 1 | 0 | 2 | 0 | 1 | 0 | 4 | 9 |
| Winston-Salem | 0 | ------ | 0 | 55 | 0 | 0 | 0 | 2 | 0 | 10 | 12 |
| **South Carolina:** | | | | | | | | | | | |
| Charleston | 0 | 1 | 0 | 0 | 1 | 3 | 0 | 0 | 0 | 0 | 20 |
| Florence | 0 | ------ | 0 | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 14 |
| Greenville | 1 | ------ | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 1 | 5 |
| **Georgia:** | | | | | | | | | | | |
| Atlanta | 1 | 9 | 0 | 0 | 1 | 2 | 0 | 3 | 4 | 24 | 63 |
| Brunswick | 0 | ------ | 0 | 3 | 2 | 0 | 0 | 0 | 0 | 0 | 11 |
| Savannah | 0 | ------ | 0 | 4 | 2 | 0 | 0 | 2 | 0 | 15 | 30 |
| **Florida:** | | | | | | | | | | | |
| Miami | 1 | ------ | 0 | 0 | 6 | 0 | 0 | 1 | 0 | 0 | 22 |
| Tampa | 1 | ------ | 0 | 2 | 1 | 0 | 0 | 1 | 0 | 1 | 19 |
| **Kentucky:** | | | | | | | | | | | |
| Ashland | 0 | ------ | 0 | 0 | 4 | 0 | 0 | 2 | 0 | 3 | 12 |
| Covington | 3 | ------ | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 13 |
| Lexington | 0 | ------ | 0 | 11 | 0 | 2 | 0 | 3 | 1 | 1 | 19 |
| Louisville | 1 | ------ | 0 | 14 | 2 | 5 | 0 | 2 | 0 | 6 | 65 |
| **Tennessee:** | | | | | | | | | | | |
| Knoxville | 0 | ------ | 0 | 3 | 1 | 0 | 0 | 2 | 0 | 8 | 25 |
| Memphis | 1 | ------ | 0 | 1 | 4 | 0 | 0 | 4 | 3 | 7 | 70 |
| Nashville | 0 | ------ | 0 | 6 | 3 | 1 | 0 | 0 | 0 | 4 | 57 |
| **Alabama:** | | | | | | | | | | | |
| Birmingham | 0 | ------ | 0 | 0 | 2 | 0 | 0 | 2 | 3 | 3 | 72 |
| Mobile | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 16 |
| Montgomery | 0 | ------ | ------ | 1 | ------ | 0 | 0 | ------ | 0 | 2 | ------ |

*City reports for week ended June 25, 1938*—Continued

| State and city | Diphtheria cases | Influenza | | Measles cases | Pneumonia deaths | Scarlet fever cases | Smallpox cases | Tuberculosis deaths | Typhoid fever cases | Whooping cough cases | Deaths all causes |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Cases | Deaths | | | | | | | | |
| Arkansas: | | | | | | | | | | | |
| Fort Smith | 0 | ------ | -------- | 3 | ------- | 1 | 0 | ------- | 0 | 0 | -------- |
| Little Rock | 0 | ------ | 0 | 0 | 5 | 0 | 0 | 2 | 0 | 0 | 9 |
| Louisiana: | | | | | | | | | | | |
| Lake Charles | 1 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 3 |
| New Orleans | 4 | 2 | 3 | 9 | 12 | 2 | 0 | 9 | 6 | 37 | 131 |
| Shreveport | 0 | ------ | 0 | 0 | 2 | 0 | 0 | 0 | 2 | 0 | 44 |
| Oklahoma: | | | | | | | | | | | |
| Oklahoma City | 0 | ------ | 0 | 0 | 4 | 2 | 0 | 1 | 0 | 0 | 46 |
| Tulsa | 0 | ------ | -------- | 14 | ------- | 0 | 0 | ------- | 1 | 6 | -------- |
| Texas: | | | | | | | | | | | |
| Dallas | 0 | 1 | 1 | 2 | 3 | 3 | 0 | 2 | 2 | 14 | 55 |
| Fort Worth | 1 | ------ | 0 | 0 | 1 | 0 | 1 | 0 | 1 | 5 | 23 |
| Galveston | 0 | ------ | 0 | 0 | 1 | 1 | 0 | 2 | 0 | 0 | 17 |
| Houston | 1 | ------ | 0 | 1 | 4 | 0 | 0 | 5 | 1 | 0 | 76 |
| San Antonio | 0 | ------ | 0 | 0 | 0 | 2 | 0 | 7 | 0 | 0 | 68 |
| Montana: | | | | | | | | | | | |
| Billings | 0 | ------ | 0 | 0 | 1 | 0 | 0 | 2 | 0 | 1 | 11 |
| Great Falls | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 | 9 |
| Helena | 0 | ------ | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 4 |
| Missoula | 0 | ------ | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 4 |
| Idaho: | | | | | | | | | | | |
| Boise | ------- | ------ | -------- | ------- | ------- | ------- | ------- | ------- | ------- | -------- | -------- |
| Colorado: | | | | | | | | | | | |
| Colorado Springs | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 10 |
| Denver | 4 | ------ | 0 | 5 | 3 | 9 | 0 | 9 | 0 | 4 | 65 |
| Pueblo | 1 | ------ | 0 | 13 | 1 | 1 | 0 | 0 | 0 | 4 | 3 |
| New Mexico: | | | | | | | | | | | |
| Albuquerque | 0 | ------ | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 17 |
| Utah: | | | | | | | | | | | |
| Salt Lake City | 0 | ------ | 0 | 193 | 2 | 5 | 0 | 0 | 0 | 11 | 19 |
| Washington: | | | | | | | | | | | |
| Seattle | 0 | ------ | 0 | 5 | 3 | 3 | 1 | 6 | 0 | 23 | 98 |
| Spokane | 0 | ------ | 0 | 1 | 5 | 0 | 0 | 0 | 0 | 10 | 32 |
| Tacoma | 0 | ------ | 0 | 0 | 1 | 3 | 0 | 0 | 0 | 4 | 21 |
| Oregon: | | | | | | | | | | | |
| Portland | 0 | 1 | 1 | 10 | 1 | 11 | 3 | 3 | 0 | 1 | 71 |
| Salem | 0 | 1 | -------- | 0 | ------- | 0 | 1 | ------- | 0 | 0 | -------- |
| California: | | | | | | | | | | | |
| Los Angeles | 13 | 7 | 0 | 58 | 11 | 27 | 1 | 12 | 0 | 23 | 277 |
| Sacramento | 0 | ------ | 0 | 34 | 3 | 2 | 0 | 3 | 0 | 7 | 39 |
| San Francisco | 1 | ------ | 0 | 3 | 4 | 4 | 0 | 8 | 0 | 22 | 154 |

| State and city | Meningitis, meningococcus | | Poliomyelitis cases | State and city | Meningitis, meningococcus | | Poliomyelitis cases |
|---|---|---|---|---|---|---|---|
| | Cases | Deaths | | | Cases | Deaths | |
| Massachusetts: | | | | Georgia: | | | |
| Boston | 1 | 0 | 0 | Savannah | 0 | 0 | 2 |
| New York: | | | | Kentucky: | | | |
| New York | 3 | 2 | 1 | Louisville | 1 | 0 | 0 |
| Syracuse | 1 | 0 | 0 | Tennessee: | | | |
| Pennsylvania: | | | | Memphis | 0 | 0 | 1 |
| Philadelphia | 1 | 0 | 0 | Alabama: | | | |
| Pittsburgh | 2 | 1 | 0 | Birmingham | 0 | 0 | 1 |
| Illinois: | | | | Louisiana: | | | |
| Chicago | 1 | 0 | 0 | New Orleans | 0 | 0 | 1 |
| Maryland: | | | | California: | | | |
| Baltimore | 1 | 1 | 0 | Los Angeles | 0 | 0 | 1 |
| District of Columbia: | | | | | | | |
| Washington | 1 | 1 | 0 | | | | |
| Virginia: | | | | | | | |
| Lynchburg | 0 | 0 | 1 | | | | |
| Richmond | 0 | 0 | 1 | | | | |

*Encephalitis, epidemic or lethargic.*—Cases: New York, 3; Cleveland, 1; Portland, Oreg., 2.
*Pellagra.*—Cases: Baltimore, 2; Charleston, S. C., 2; Atlanta, 2; Savannah, 1.
*Typhus fever.*—Cases: New York, 1; Florence, 5; Savannah, 1; Miami, 1; Mobile, 1.

# FOREIGN AND INSULAR

## FINLAND

*Communicable diseases—May 1938.*—During the month of May 1938, cases of certain communicable diseases were reported in Finland, as follows:

| Disease | Cases | Disease | Cases |
|---|---|---|---|
| Diphtheria | 183 | Poliomyelitis | 5 |
| Dysentery | 3 | Scarlet fever | 1,249 |
| Influenza | 11,240 | Typhoid fever | 26 |
| Paratyphoid fever | 19 | Undulant fever | 1 |

## GREAT BRITAIN

*England and Wales—Infectious diseases—13 weeks ended April 2, 1938.*—During the 13 weeks ended April 2, 1938, certain infectious diseases were reported in England and Wales, as follows:

| Disease | Cases | Disease | Cases |
|---|---|---|---|
| Diphtheria | 20,750 | Puerperal pyrexia | 2,509 |
| Dysentery | 2,549 | Scarlet fever | 30,444 |
| Ophthalmia neonatorum | 1,394 | Smallpox | 4 |
| Pneumonia | 17,115 | Typhoid fever | 322 |

*England and Wales—Vital statistics—First quarter 1938.*—During the first quarter ended March 31, 1938, 155,269 live births and 137,926 deaths were registered in England and Wales. The following statistics are taken from the Quarterly Return of Births, Deaths, and Marriages, issued by the Registrar General of England and Wales, and are provisional:

*Birth and death rates in England and Wales, quarter ended March 31, 1938*

| | |
|---|---|
| Annual rates per 1,000 population: | |
| Live births | 15.3 |
| Stillbirths | .61 |
| Deaths, all causes | 13.6 |
| Deaths under 1 year of age | [1] 68 |
| Deaths from: | |
| Diarrhea and enteritis (under 2 years of age) | [1] 6.6 |
| Diphtheria | .10 |
| Influenza | .20 |
| Measles | .08 |
| Scarlet fever | .01 |
| Typhoid fever and paratyphoid fever | .01 |
| Violence | .56 |
| Whooping cough | .04 |

[1] Per 1,000 live births.

*England and Wales—Vital statistics—Year 1937.*—Following are vital statistics for England and Wales for the year 1937:

*Birth and death rates in England and Wales, year 1937*

| | |
|---|---|
| Annual rates per 1,000 population: | |
| Live births | 14.9 |
| Stillbirths | .60 |
| Deaths, all causes | 12.4 |
| Deaths under 1 year of age | [1] 58 |
| Deaths from: | |
| Diarrhea and enteritis (under 2 years of age) | [1] 5.5 |
| Diphtheria | .07 |
| Influenza | .45 |
| Measles | .03 |
| Scarlet fever | .01 |
| Typhoid fever and paratyphoid fever | .01 |
| Violence | .56 |
| Whooping cough | .04 |

[1] Per 1,000 live births.

## 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 June 24, 1938, pages 1049–1064. A similar cumulative table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

### Smallpox

*Venezuela.*—During the period May 16–31, 1938, smallpox (alastrim) was reported in Venezuela as follows: Barcelona, Anzoategui State, 1 death; Valencia, Carabobo State, 1 death; San Juan los Morros, Guarico State, 2 deaths; Trujillo, Trujillo State, 1 death; Sanare, Lara State, 1 death.

### Yellow Fever

*Brazil.*—Yellow fever has been reported in Brazil as follows: Minas Geraes State, June 2, 1938, 1 death; Rio de Janeiro State, May 18, 1938, 1 death.

*French Equatorial Africa—Gabon—Koula Moutou.*—On June 27, 1938, 1 suspected case of yellow fever was reported in Koula Moutou, Gabon, French Equatorial Africa.

*Gold Coast—Big Ada.*—On June 24, 1938, 1 suspected case of yellow fever was reported in Big Ada, Gold Coast.

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