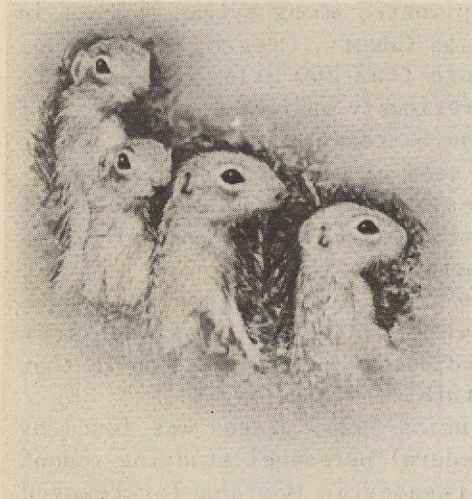


DOMESTIC RATS, FLEAS and

NATIVE RODENTS

In Relation To Plague

In The United States



By Entomologist Carl O. Mohr**

INTRODUCTION

Bubonic plague is a rodent and rodent-flea disease caused by the plague bacillus *Pasturella pestis* which is transmitted from animal to animal and thence to man by fleas. It is highly fatal. At least half of the human cases result in death without modern medication. (Table I — last two columns). Because of their close association with man, domestic rats* and their fleas, especially the oriental rat flea *Xenopsylla cheopis*, are responsible for most human epidemics. Only occasional cases are caused by bites of other fleas or by direct infection from handling rodents.

Infection due to bites of fleas or due to direct contact commonly results in swollen lymph glands, called *buboes*, hence the name *bubonic* plague. Infection may progress to the blood stream causing septicemic plague,

and finally to the lungs causing pneumonic plague.

Pneumonic plague is extremely fatal and highly infectious when sputum droplets pass direct from person to person. The death rate due to it is practically 100 percent.

Plague is dreaded particularly where living conditions are such as to bring human beings into close contact with large oriental-rat-flea populations, and where crowded conditions permit rapid pneumonic transmission from man to man.

ANCIENT AMERICAN DISEASE OR RECENT INTRODUCTION

Two widely different views exist concerning the arrival of plague in North America. The prevalent view is that it was introduced from the Orient into North America at San Francisco through ship-

* *Rattus rattus* and *Rattus norvegicus*.

**Dr. Mohr is Chief of the Ectoparasite Branch, Entomology Division, Communicable Disease Center, Atlanta, Georgia.

borne rats and their fleas about 1900. Facts supporting this view:

1. The first cases were recognized in the Chinese section of San Francisco region during that year.
2. Succeeding human cases were recognized, first near San Francisco, and, as the years went by, at increasingly greater distances as plague presumably spread northward and inland.
3. Results of plague surveys in wild rodents revealed the presence of plague in an ever-widening area after its original recognition in California. For example, in 1935, after it was learned that animals in other western states were infected, examinations were extended to all of the Rocky Mountain states as far as the great plains. No infection was found in the plains states until after a lapse of several years.
4. Plague occurred in Pensacola, Florida; in New Orleans; and at Beaumont, Port Arthur and Galveston from 1914 to 1921, apparently having been introduced by shipping, thus giving support to the theory that the San Francisco outbreak had occurred in the same way.

The other view, perhaps not so widely held, is that plague is a characteristic disease of colonial rodents in North America, for it is also found in very closely-related species in Asia. Therefore, it must have been present on this continent in those rodents and their fleas thousands of years before modern shipping. Facts supporting this view:

1. It is a common occurrence for diseases as well as parasites to accompany their hosts into new territory. Mammalogists generally agree that ground squirrels and meadowmice are Asiatic in origin. Since plague is a common disease of these animals in Asia it may therefore have arrived with them during prehistoric times.
2. A considerable number of single human cases or epizootics have occurred in areas far in advance of those in which plague had been suspected. Plague,

therefore, may have been present there from ancient times. The following instances may be cited:

- a. In 1908 a case in Los Angeles County, California was the first warning of its existence there.
- b. The death of a sheepherder and an epizootic among sylvan rodents in Lake County, Oregon, in 1934 was more than 200 miles north of the previously suspected area south of the Sacramento River.
- c. A case of bubonic plague in southwest Utah in 1935 was the first indication of the presence of plague there.
- d. In 1937 a human case near Lake Tahoe, in Nevada, was the first indication of its presence in that state.
- e. During 1935, plague was found by federal personnel studying rodent diseases in Montana far removed from any areas in which plague was previously suspected.

HISTORY OF HUMAN CASES

Epidemics of plague have struck in North America in the past and can strike again if protection is relaxed in present danger zones or is not extended promptly to new danger spots as they develop. America's recent record has been good in this respect. During the 21 years between 1925 and 1946, only 21 reported human cases occurred in the vast western half of this continent (see back cover) where plague was known to be present among wild rodents. (Table I). No human cases have been reported in the eastern states since 1921. This fine rapport has not always existed; our earlier plague history illustrates how outbreaks can take place if watchfulness is relaxed and protection not provided in danger areas. Its manner of striking in the future may resemble that of the past.

As previously stated, the disease was first recognized in North America during 1900 (Hampton 1945) in the San Francisco area. From that date until 1924, it struck repeatedly and disastrously in California

TABLE I

CASES OF, AND DEATHS FROM, PLAGUE IN THE UNITED STATES (HAMPTON, 1945)

| YEAR | CASES | | | | | | | | | TOTALS | |
|-----------|--------|-------|-----|------|------|--------|------|------|-------|--------|--------|
| | CALIF. | WASH. | LA. | FLA. | TEX. | OREGON | UTAH | NEV. | IDAHO | CASES | DEATHS |
| 1900 | 22 | | | | | | | | | 22 | 22 |
| 1901 | 30 | | | | | | | | | 30 | 26 |
| 1902 | 41 | | | | | | | | | 41 | 41 |
| 1903 | 17 | | | | | | | | | 17 | 17 |
| 1904 | 10 | | | | | | | | | 10 | 8 |
| 1905-06 | | | | | | | | | | 0 | 0 |
| 1907 | 178 | 3 | | | | | | | | 181 | 90 |
| 1908 | 8 | | | | | | | | | 8 | 5 |
| 1909 | 3 | | | | | | | | | 3 | 1 |
| 1910 | 3 | | | | | | | | | 3 | 1 |
| 1911 | 4 | | | | | | | | | 4 | 1 |
| 1912 | | | | | | | | | | 0 | 0 |
| 1913 | 2 | | | | | | | | | 2 | 2 |
| 1914 | 1 | | 30 | | | | | | | 31 | 10 |
| 1915 | 1 | | 1 | | | | | | | 2 | 1 |
| 1916-18 | | | | | | | | | | 0 | 0 |
| 1919 | 13 | | 15 | | | | | | | 28 | 18 |
| 1920 | 1 | | 7 | 10 | 33 | | | | | 51 | 27 |
| 1921 | 3 | | 3 | | | | | | | 6 | 4 |
| 1922 | 2 | | | | | | | | | 2 | 0 |
| 1923 | 1 | | | | | | | | | 1 | 0 |
| 1924 | 41 | | | | | | | | | 41 | 34 |
| 1925 | 1 | | | | | | | | | 1 | 0 |
| 1926 | | | | | | | | | | 0 | 0 |
| 1927 | 1 | | | | | | | | | 1 | 1 |
| 1928 | 3 | | | | | | | | | 3 | 2 |
| 1929-32 | | | | | | | | | | 0 | 0 |
| 1933 | 1 | | | | | | | | | 1 | 1 |
| 1934 | 1 | | | | | 1 | | | | 2 | 1 |
| 1935 | | | | | | | | | | 0 | 0 |
| 1936 | 3 | | | | | | 1 | | | 4 | 0 |
| 1937 | 1 | | | | | | | 1 | | 2 | 1 |
| 1938 | | | | | | | | | | 0 | 0 |
| 1939 | | | | | | | 1 | | | 1 | 0 |
| 1940 | | | | | | | | | 1 | 1 | 1 |
| 1941 | 2 | | | | | | | | | 2 | 2 |
| 1942 | 1 | | | | | | | | | 1 | 0 |
| 1943 | 1* | | | | | | | | | 1 | 1 |
| 1944 | 1 | | | | | | | | | 1 | 0 |
| 1945-46** | | | | | | | | | | 0 | 0 |
| TOTAL | 397 | 3 | 56 | 10 | 33 | 1 | 2 | 1 | 1 | 504 | 318 |

* Case acquired in laboratory

** 1945-1946 data from Public Health Reports for 1945, 1946, and 1947. One case occurred in California in 1947



Roof rat. Domestic rats, being closely associated with man and bearing at least one highly plague-susceptible flea, are largely responsible for most epidemics.

until the general public and public officials became sufficiently aware of the hazard to support protective measures. From 10 to 41 cases occurred annually in San Francisco up to 1904. Then for two years none was recognized and the city was declared to be free of detectable plague. During 1907, however, plague struck that city again. Total cases numbered 178, of which 87 were fatal. Eight cases were reported from San Francisco in 1908; since then, none has been detected in that city*. However a few human plague cases were reported from California nearly every year until 1919 when 13 recorded cases of pneumonic plague occurred in Oakland. All patients died. A 41-case epidemic, 34 fatal, occurred in Los Angeles during 1924. From that date onward, control and good fortune have held the rate down, as noted above.

While California — chiefly in San Francisco and surrounding cities — was having these difficulties, New Orleans, Pensacola, Port Arthur, Beaumont, and Galveston also had outbreaks. Thirty cases of plague occurred in New Orleans during 1914. After years of survey and control measures, the last reported case occurred during 1921. Florida and Texas cities had a few cases during 1920, but having been forewarned by the experience of San Francisco and New Orleans, they quickly instituted sufficient control measures and eliminated plague from their borders.

* With the exception of one case acquired in an experimental laboratory.

SOURCES OF PLAGUE OUTBREAKS

DIRECTLY FROM NATIVE RODENTS OR FLEAS Plague among rodents of fields and woods is known as sylvatic plague. Sixty-six human infections in rural western areas (with a 63 percent death rate) have been traced to contact with rodents or to bites from their ectoparasites (Meyer 1947). Many of these cases were traced to non-domestic rodents. From 1934 through 1943, cases were reported only in California, Oregon, Idaho, Utah, and Nevada.

One outbreak due to contact with ground squirrels is described as occurring in Oakland in 1919. The first patient in the series is thought to have acquired plague from squirrels which he had shot and prepared for eating. The disease developed pneumonic phases. Thirteen of the persons who visited or attended him acquired the pneumonic infection from him, became ill with the disease, and died. Deaths included two doctors and two nurses.

Most of the sylvan-acquired plague has not, however, spread beyond the first contact, (principally because the patients are more or less isolated) and pneumonic plague has not resulted.

DIRECTLY FROM DOMESTIC RATS OR THEIR FLEAS. Domestic rats dying from plague are the most common sources of infection. Their blocked, plague-infected fleas feed

on man as opportunity is offered and thus pass the disease from their rat hosts to him.

An incident is described by Kellogg (1920) in which the first case was a Mexican woman in Los Angeles who set up a train of pneumonic cases following her infection. Forty-one deaths resulted. These included the patient's husband, an ambulance driver, nurses, a priest, and many of the patient's visitors.

The rats implicated in this chain of deaths are believed to have acquired infection from native rodents.

Cases in Pensacola, New Orleans, Beaumont, Port Arthur, and Galveston were apparently urban-acquired from domestic rat fleas. Most of the cases in San Francisco were also urban-acquired. Some might have originated in ground squirrels handled as food in San Francisco.

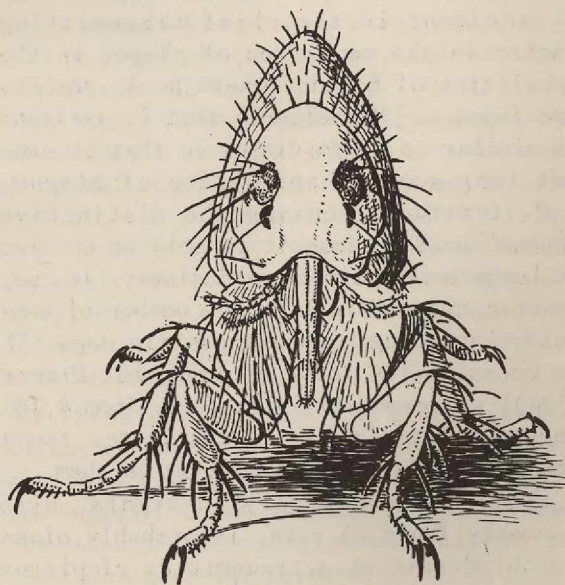
RELATIVE IMPORTANCE OF DOMESTIC RAT FLEAS AS PLAGUE VECTORS

The degree of potential infection in any community usually depends upon the kind and abundance of rats and fleas present; the amount of contact between diseased animals and man; and, if pneumonic phases develop, upon the amount of contact between diseased persons and susceptible persons. Plague among human beings is generally uncommon where suitable domestic rat fleas are uncommon. With the exception of the Oakland outbreak, in which the basic case was apparently acquired by direct contact with native rodents, plague outbreaks have been most disastrous where domestic rats and certain species of their fleas are abundant. These fleas will be considered separately.

ORIENTAL RAT FLEA. It is generally conceded that the oriental rat flea, *Xenopsylla cheopis*, is a most efficient carrier. It is prevalent on domestic rats throughout most of the world plague belt; experiments on its ability as a vector put it high on the list of vectors; and development of plague organisms within its digestive tract is great. According to Eskey (1938) it causes sharp but short

epidemics in any one community due to rapid infectibility, quick transmission, and early deaths of the fleas which become infected.

NORTHERN RAT FLEA. *Nosopsyllus fasciatus*, is generally regarded as a second-rate carrier. Of it, Eskey wrote in 1938: "insofar as is known, plague epidemics have never occurred in communities where *N. fasciatus* existed alone and were not associated with *X. cheopis*." He adds that if *N. fasciatus* were as efficient a vector of this disease as *X. cheopis*, California outbreaks would have been much more severe than they were, and that these outbreaks would have been comparable with a Guayaquil, Ecuador, outbreak where: (1) *X. cheopis* alone of rat fleas was present; (2) Its index on rats was about twice that of *X. cheopis* in San Francisco; (3) Many more human cases occurred than in San Francisco.



A "head on" view of the oriental rat flea, *Xenopsylla cheopis*, the most dangerous plague transmitter of domestic rat fleas. (Mohr).

He believed, however, (1938) that *N. fasciatus* was important in prolonging epizootics of plague among rats in more northern climates in the presence of *X. cheopis* even though relatively few human cases would result. He cited as an example

the Seattle plague epizootic during 1907. Plague smouldered slowly among rats for at least 10 years with only three human cases officially reported. The recent discovery of plague among rats in Tacoma (1942) without human cases is probably due to similar conditions. It may have been a continuation of the 1907 epizootic in the Puget Sound area.

Experiments conducted by Eskey showed *N. fasciatus* to be only slightly susceptible to infection. When infected, however, this species may carry the infection for two or more months in contrast with *X. cheopis*, which is much more easily infected and which dies in a matter of only a week or two.

HUMAN FLEA. *Pulex irritans* is also regarded as a second-rate carrier in spite of the fact that it commonly lives in close contact with man. Eskey (1930) concluded that, "By the process of elimination, one is compelled to believe that *P. irritans* is the chief transmitting factor in the causation of plague in the localities of Ecuador where no *X. cheopis* are found..." He believes that *P. irritans* is similar to *N. fasciatus* in that it cannot long support epizootics of plague.

P. irritans received the distinctive "human" name because it is able to survive on human beings almost exclusively. It can, however, maintain itself on a number of mammals, including hogs and prairie dogs. It is occasionally found on rats, but Prince (1943) who examined 5,785 fleas from 4,188 domestic rats in 13 western states found none, attesting to its scarcity on them.

MOUSE FLEA. *Leptopsylla segnis*, also commonly found on rats, is probably close to third-rate as a transmitter of plague to human beings.

STICKTIGHT FLEA. *Echidnophaga gallinacea*, is abundant on rats in certain sections of North America. It probably is a third-rate carrier of plague, although it has been found naturally infected with a virulent strain of this disease (Wheeler, Douglas and Evans, 1941). *E. gallinacea* acquires and transmits plague readily under experimental conditions (Burroughs, 1947). Unlike the oriental rat flea which feeds

upon a large number of individual hosts during the course of its life, the sticktight is inclined to be monophagous. Illingworth (1916) found that adults attach themselves firmly to their hosts and engorge with blood. He found that the males move around during the night and may be found attached in different positions each morning. Females, as a rule, do not change host or their position of attachment. Even the more restless male makes few host-changes. Parman (1923) found that adults were inactive during the first few days after emergence and usually did not attach themselves to hosts until the fifth or eighth day. The females became engorged and oviposition began in from six to 10 days after attachment. Practically all oviposition observed took place while fleas were attached to the host. Even copulation was observed to take place on the host with copulating fleas attached to the head of the hosts about a body-length apart. They remained attached to the host in the same place from four to 19 days.

Upon the death of their original host, or upon occasional transfer or other removal from the original host, the sticktight flea may transfer to another host and possibly infect it. However, possibility of exchange to plague-susceptible hosts appears to be slight. Burroughs found this flea to have a tick-like habit of feeding to repletion on only one individual.

The sticktight flea may accumulate in large numbers in the nests of predators where their infected feces and bodies may infect rodent contacts. This does not seem to be an important means of transmittal, however, because it is relatively difficult to inoculate animals without considerable abrasion of the skin.

MEANS OF POSSIBLE FUTURE SPREAD OF PLAGUE IN THE UNITED STATES

BY EPIZOOTIC WAVES AND MIGRATIONS. If plague was introduced into North America at San Francisco by oriental shipping about 1900, it has progressed inland, as the crow flies, for at least 1,125 to 1,175 miles in 47 years. This progression is at



Norway rat burrows in bluegrass. Meadowmice and ground squirrels are common in such habitats. Glove shows location of burrow.

a rate of about 25 miles per year. There can be little doubt that natural movements of infected mammals and their fleas could be partly responsible for such spread and that their transportation by predators might play a part. Transportation of infected man or other mammals by common carrier over land or water could cause plague, as it has in the past, to show up in unexpected places. Transportation by common carrier is perhaps the quickest method of transmission.

However, movement by most species of small plague-infected field rodents is not great; although young of the year do go forth annually to seek new and unoccupied territories, they generally move only a few hundred yards away from their points of origin.

In most cases, nature confines mammal populations to expansion within suitable habitats. This expansion, generally, is slow although some species and varieties of mammals are known to have extended their range. Norway rats, for example, have appeared in western states after a slow expansion of range, apparently from the west coast. The rate of this expansion, however, was long ago outstripped by plague if the disease was not originally present.

Domestic rats migrate locally from time to time. Zinsser (1935) reports as follows:

"Dr. Lantz tells us that in 1903 hordes of rats migrated over several counties in western Illinois, suddenly appearing where for several years no abnormal numbers had been seen." But the waves of these migrations break themselves up on resistance offered by unsuitable habitats and territories already occupied by members of the same species. They are not repeated year after year. Such movements could hardly have served as dispersal factors in the spread of plague at the rate it appears to have spread.

In Canada, in the absence of an already-established rat population, Norway rats apparently did spread rapidly. Professor V. W. Jackson of the University of Manitoba states:

"The Norway rat entered our boundary at Emerson in 1903. In two years (it) had reached Winnipeg and two years later Portage La Prairie. By 1911 the rat had reached Brandon - a steady spread of 25 miles per year in all settled directions. By 1920 (it) was half way across Saskatchewan on a fairly even western front and reached Alberta about 1925."

The rate of this reported spread of rats in Canada was somewhat similar to the rate of spread of plague in the United States. However, it seems unlikely that plague could have been carried inland by rats from the west coast because its passage outstripped that of rats into Montana - (if plague were not present there as a native American disease).

BY PREDATORY MAMMALS AND BIRDS Wide-ranging predators, such as coyotes, hawks, owls, and eagles might transport plague more rapidly. It is no great feat for these creatures to carry large quantities of flea-laden prey for several miles in order to reach their young. It is conceivable that in 47 years plague might have been relayed the 1,175 miles from San Francisco to North Dakota and Kansas by this means, superimposed on epizootics at each new location.

Predators, particularly the mammals, commonly accumulate a variety of fleas from the prey which they eat or carry during the breeding season. Randolph and

Eads (1946) found seven species of fleas on 16 grey foxes; three species on four Texas red wolves; and six species each on 13 raccoons and 27 possums. With the possible exception of some of the human and sticktight fleas, which have catholic tastes, all were foreign: that is, belonging properly to such prey species as rabbits and tree squirrels. Coyotes are often heavily infested. They have been known to carry food a distance of eight miles to their young in the den. (Young, 1937).

Jellison (1939) gave some attention to the matter of plague-dispersal by scavenger and predatory birds. These birds, like the predatory mammals, appear quite resistant to plague. He found that scavenger birds such as crows and magpies seldom carry entire carcasses to their nests. He concluded that they are probably of slight importance in its dispersion by that means. Jellison found, by examination of 12 nests,



Cotton rats are a numerous, inconspicuous species of small field rodent, common in the southeastern part of the United States. Fleas from western members have been found infected with plague. (Courtesy Typhus Investigations Laboratory, Thomasville, Ga.)

only one rodent flea on 54 of these birds. However, the predatory birds such as owls, hawks, and eagles carry a considerable number of carcasses to their nests. Thus, they can scatter fleas along the entire route. Jellison found seven species of ro-

dent fleas in the nests of these birds. In the nest of one burrowing owl, he found 109 rodent fleas, including six species. He quotes a Major Bendire as stating that burrowing owl nests invariably swarm with fleas. Movements of the burrowing owl are, however, decidedly local.

Experiments with casts and droppings of scavenger and predatory birds indicated that these commonly were infected. Though Jellison was unable to transfer infection to experimental animals, results of his experiment are not necessarily conclusive.

Wayson (1947) reported finding infected fleas on weasels and badgers. Since no predators have been found plague-infected to date and since obligatory predatory-fleas are uncommon, it is likely that these infected fleas were acquired from the prey of weasels and badgers* in question. The species of fleas concerned were not identified.

There can be little doubt that some of the fleas carried to other areas by predatory mammals and birds find new rodent hosts. If infected themselves, they pass that infection along to these hosts.

Wayson, who concluded that plague had spread eastward in the past, (1947) thinks that the rapidity of dissemination of plague which has occurred cannot be estimated. He asserts that further advancement eastward will be slow in terms of years. Eskey and Haas (1940) stated that it

might at least progress into that section of this country occupied by ground squirrels. Wayson, however, indicates that meadowmouse, rice rat, and cotton rat populations of the eastern half of the United States may also become infected.

* The finding of two plague-infected ticks on a badger (Anon 1943) is an indication that this carnivore might be susceptible, since ticks do not frequently change hosts. African workers have found some viverrine carnivorous mammals susceptible to plague.

MAMMALIAN RESERVOIRS

The genera of mammals, in addition to domestic rats, from which plague has been recovered in tissue cultures are:

| GENUS | COMMON NAME |
|---------------------|---------------------|
| <i>Oryzomys</i> | Rice rats* |
| <i>Microtus</i> | Meadowmice |
| <i>Peromyscus</i> | Deer mice |
| <i>Onychomys</i> | Grasshopper mice |
| <i>Mus</i> | Housemice |
| <i>Neotoma</i> | Woodrats, pack rats |
| <i>Citellus</i> | Ground-squirrels |
| <i>Marmota</i> | Marmots |
| <i>Cynomys</i> | Prairie dogs |
| <i>Eutamias</i> | Western chipmunks |
| <i>Tamiasciurus</i> | Pine squirrels |
| <i>Dipodomys</i> | Kangaroo rats |
| <i>Sylvilagus</i> | Cottontail rabbits |
| <i>Lepus</i> | Jackrabbits |

Plague was also reported from fleas only, in the following mammalian genera, though this does not necessarily incriminate the hosts as plague reservoirs:

| | |
|------------------------|--------------|
| <i>Reithrodontomys</i> | Harvest mice |
| <i>Sigmodon</i> | Cotton rats |
| <i>Taxidea</i> | Badgers |
| <i>Mustela</i> | Weasels |

Eskey and Haas (1940) also report plague as present in fleas or tissues of flying squirrels (*Glaucomys*). Either fleas or



White-footed mice are commonly found plague-infected. Species of the genus *Peromyscus* are found nearly everywhere in North America. (Courtesy U. S. Soil Conservation Service).

tissues of harvest mice have been found to be plague-positive (Volume 62 of Public Health Reports).

Plague is not recorded from the following mammals or in fleas from them. Only a small, but unstated, number were examined:

| GENUS | COMMON NAME |
|--|------------------|
| <i>Evotomys</i> | Red-backed mice |
| <i>Synaptomys</i> | Lemming mice |
| <i>Phenacomys</i> | Rufous Tree mice |
| <i>Zapus</i> | Jumping mice |
| <i>Ondatra</i> | Muskrats |
| <i>Castor</i> | Beavers |
| <i>Aplodontia</i> | Mountain beavers |
| <i>Erethizon</i> | Porcupines |
| <i>Blarina, Cryptotis, Sorex</i> | Shrews |
| <i>Scapanus, Neurotrichius</i> | Moles |
| <i>Didelphis</i> | Possums |
| Most predatory mammals (<i>Carnivora</i>). | |

In popular literature it is common, when discussing ground-squirrels, to drop the prefix and refer simply to "squirrel." From a practical control point-of-view, it would be erroneous to assume that tree squirrels are largely involved in plague transmission simply on the basis of the popular misuse of the term "squirrel."

Few data are available for evaluating the relative importance of the many species of mammals as plague reservoirs, although a knowledge of this subject is patently important. Ground-squirrels were first suspected in America because they were more commonly shot and handled than were smaller or more nocturnal field rodents. While investigating the origin of a case of fatal plague contracted by a blacksmith in Contra Costa County, Rupert Blue suspected that ground-squirrels, which the patient had hunted, were the cause. By 1908, proof was obtained that conspicuous mortalities among this large, diurnal species were, in fact, often due to plague. Survey and control programs were accordingly concentrated upon them.

An indication of the amount of detectable plague that exists among ground-squirrels may be had from a report by the Surgeon

* From the Annual Report of the Surgeon General, 1920.



Young prairie dogs. Prairie dogs are one of the more conspicuous plague-susceptible mammals, being diurnal, easily infected, and about as large as young cats. Numerous small wild rodents apparently transmit plague from one prairie dog colony to another. (Courtesy J. W. Jackson).

General (1927). Of 558,706 rodents examined between 1908 and 1927, chiefly ground-squirrels from California, 2,069 (0.4%) were plague-infected. This relatively high incidence was due, in part, to the selection of areas where plague was known to be present, or likely to be present. Therefore, it cannot be compared with the rate among ground-squirrels or other rodents from areas chosen at greater random. Much higher rates are recorded from more restricted areas and for shorter survey periods.

Again, from 505,097 rodents of all kinds and a "small number of other species" collected from many western states (1936-1945) 153 pools* of tissues were found infected (Wayson, 1947). Most of these rodents - exact number unstated - were

ground-squirrels. Since the investigations were more random in nature and organs from several animals were frequently pooled, the percentage of infection was naturally lower (0.03). Thirty-seven pools of tissues from 85,414 prairie dogs taken during the last five years of the period were found infected. This is a rate of 0.04%. The difference is probably not significant but only indicative of the general rate of infection.

Investigators gradually became aware that other less conspicuous, but often more numerous, species were also plague-infected. During the period from 1941 through 1945, greater emphasis was shifted to such species. Of 188,815 rodents examined, 38,277 were kangaroo rats, 16,876 were grasshopper mice, 16,493 were meadowmice,

* A pool of tissue is a portion of the tissues of each of one or more animals of the same species, collected at one hunting area on the same day.

and 4,465 were marmots. Although 85,414 prairie dogs, 2,411 variegated ground-squirrels, and other species were included, the rate was low. Only twelve pools of tissues (0.006%) were found infected (Wayson, 1947).

From 1,186,777 fleas taken from the 505,097 rodents during the entire ten year period, 308 pools were found infected. Ground-squirrels were still commonly regarded as primary reservoirs of plague from which stemmed the epizootics that destroyed their colonies and neighboring species of mammals.

From the time of the earliest surveys wood rats were occasionally caught by investigators. Possibly because of their obvious relationship to domestic rats, their tissues — and finally their fleas — were tested for plague. Some were found to be infected. In 1940 the desert wood rat, *Neotoma desertorum*, was cited as a primary reservoir of plague in certain areas. Plague had been found among desert wood rats where there were almost no ground-squirrels and where other native rodents had consistently failed to show infection (Eskey and Haas, 1940).

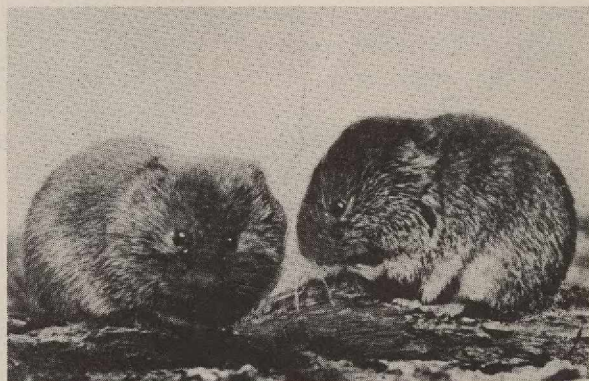
The role of prairie dogs is in doubt. Although plague epizootics sweep through prairie dog colonies, almost, if not entirely, wiping many out, the plague quite obviously might have been acquired from one or more of the mice or ground-squirrels which form a bridge from one colony to another. It is probable that prairie dogs, like other conspicuous diurnal species, were over-suspected.

Meadowmice are frequently infected and loaded with fleas. They are commonly found to be plague-infected along with rats. According to Meyer (1947) they have been found to harbor plague in the absence of ground-squirrels. Like ground-squirrels, these mice attain large absolute numbers, varying from a few per acre, where present at all, to over 800. However, their usual number is in tens or scores of individuals to the acre (Mohr, 1947).

Meyer informed the writer that plague is generally not self-maintaining in ground-squirrel populations (as few as five to

the acre) in the absence of other capable reservoirs. Therefore, wherever meadowmice are present, they must be considered as an important factor because their numbers are generally high and their habits colonial. A control aimed specifically at them may be called for in a large number of cases if any sylvan control is deemed necessary. Mice of other genera are also involved and often more numerous than ground-squirrels.

Wayson found a pool of infected fleas on cotton rats which may or may not incriminate the animal itself. Cotton rats, like meadowmice, attain large populations and are therefore suspect where large populations of susceptible species are an indicator of danger. Also, cotton rats frequently inhabit outbuildings and therefore can serve as a considerable source of contact with domestic rats. Wayson (1947) minimizes the possible role of population-size as having any relation to persistence of a plague focus. He states: "Neither the persistence of a focus from year to year nor its primary discovery has shown any correlation with the total population of rodents..." Here he is probably referring to extensive areas at least several miles across rather than to smaller units. He stresses the role of colonial rodents as common reservoirs of plague.



Meadowmice (*Microtus*) are frequently found infected and may be primary plague-reservoirs in many localities. They are abundant and widely distributed in western and northeastern states. Note that the ears do not show, being characteristically hidden by fur. *Microtus* is an extremely abundant genus of mammals, very susceptible to plague and frequently found infected on the outskirts of towns where rats are infected. (Courtesy U. S. Fish and Wildlife Service).

IRREGULAR OCCURRENCE OF PLAGUE EPIZOOTICS

Plague epizootics are extremely spotty among rodent populations. The period during which plague will persist in rodents of a given area has not been determined by systematic investigations under properly controlled circumstances (Wayson, 1947). Wayson claims plague has been found in specimens collected from one locality during each of four successive "animal seasons." On the other hand, plague was not found again during four other successive seasons (after having been detected in a given area) in spite of collections made from the same area where it had previously been found.

Byington (1940) recorded similar experience; no plague was observed among native rodents during two years of close observation of an area where plague had decimated the rodents during one season. Years of negative results do not necessarily mean that plague is not, or has not, been present.

Meyer (1942) gave a description of local spottiness of detectable plague areas.

According to him, preliminary field studies on the course of plague epizootics were made in Kern County (California) during 1934 and plague was found present (Figure 1). Annual surveys thereafter gave negative results until 1941 when plague was found again among ground-squirrels and their fleas. It was present in at least five separate and distinct localities, each at least 15 to 20 miles distant from any other, located within the foothill area. Meyer concluded that focal occurrence and discontinuous distribution is apparently one of the characteristics of sylvatic plague.

Meyer also discussed seasonal distribution of detected plague. The first specimens of ground-squirrels positive for plague were collected on April 24. The last specimens were taken on July 17. Subsequent surveys yielded neither infected fleas nor rodents. The duration of detectable active disease in ground-squirrel fleas was therefore short. In the ground-squirrels themselves, the duration was even shorter, though on one ranch the disease had involved as much as 90% of the ground-

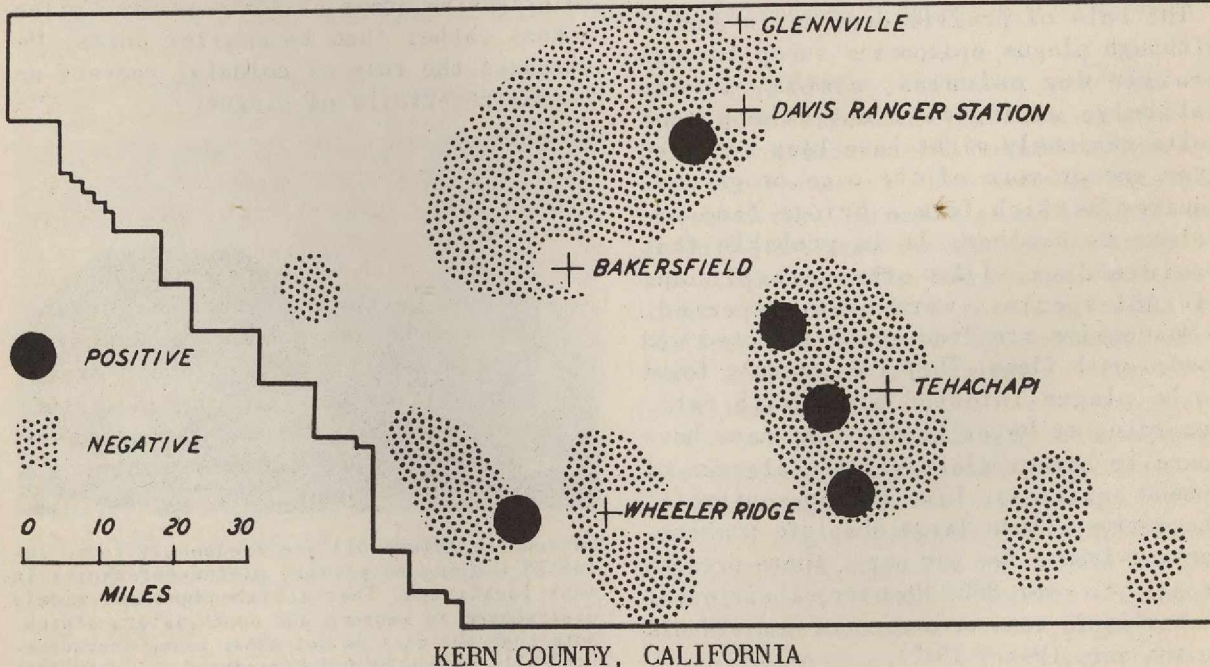


FIGURE I. Plague was found present among ground squirrels in five areas but not among those from areas shown in gray in 1941. None had been discovered in this area during seven years previously although some surveys, most of them intensive, were made there annually.

squirrel population. It may be latent in native rodents in a form difficult to detect by our present methods; it may carry over in a relatively few fleas; or it may be carried over in some rodents not heretofore examined thoroughly during inter-epizootic periods.

AMOUNT OF EFFECTIVE CONTACT BETWEEN DOMESTIC RATS AND NATIVE RODENTS

The amount of effective plague-exchanging contact between domestic and native rodents can be measured roughly by the rate of exchange of fleas between them. Other means of exchanging plague, say pneumonically, by direct contact of mammals (with living or dead individuals) or contact with infected feces, nesting material, or other matter, seems unlikely.

Several faunistic lists record sylvan-rodent fleas on domestic rats. The records of Prince (1943) however, are especially informative because they are based on 5,785 fleas from 4,188 rats caught in 13 states. In addition to the usual rat fleas, Prince recorded the following fleas which are normal parasites of the species of mammals shown below:

| FLEA | MAMMAL |
|------------------------------|--|
| <i>Megabothris abantis</i> | Meadowmice* |
| <i>Megabothris lucifer</i> | Meadowmice* |
| <i>Malareus telchinum</i> | Deer mice* |
| <i>Monopsyllus wagneri</i> | Deer mice |
| <i>Orchopeas nepos</i> | Tree squirrels |
| <i>Orchopeas sexdentatus</i> | Pack rats |
| <i>Amoniopsyllus spp.</i> | Pack rats |
| <i>Diamanus montanus</i> | Ground-squirrels |
| <i>Hoplopsyllus anomalus</i> | Ground-squirrels |
| <i>Opisocrostis lavis</i> | Ground-squirrels |
| <i>Thrassis petiolatus</i> | Ground-squirrels |
| <i>Oropsylla rupestris</i> | Probably ground-squirrels |
| <i>Thrassis fatus</i> | Probably ground-squirrels or native mice |
| <i>Thrassis howelli</i> | Marmots |
| <i>Foxella ignotus</i> | Pocket gophers |

* And possibly other native rodents.

Eskey (1938) found 48 *Malareus telchinum* on 3,027 Norway rats.

Ground-squirrel fleas, pack rat fleas, and meadowmouse fleas are most common of the "foreign" fleas on rats. Rarely is more than one of these found at a time on domestic rats. Normal prairie dog fleas, including *Pulex irritans*, are absent from the list.

FLEA - ECOLOGY SURVEYS

INLAND WESTERN AND PACIFIC-COAST STATE SURVEYS. A few surveys of domestic rat fleas have been made in inland western states and in three Pacific-coast states. These surveys show the numbers of sylvan-rodent fleas as well as the number of rat fleas found on domestic rats. Data on the domestic-rat fleas are recorded by states (Table IIA&B). Although it is acknowledged that conditions in each state may vary so widely that this method is too crude to serve maximum usefulness, it will indicate regional tendencies. Furthermore, this is about all we have in the way of data. Where a breakdown appears to be desirable the publication by Prince (1943) should be consulted.

The conditions under which these surveys were made were described as follows: Norway and roof rats were collected by trapping and shooting in rural areas and cities during a six-year period from 1935 through 1941. Surveys were made chiefly during the summer months.

Although data show *cheopis* present from Washington to California, this flea appears to be relatively uncommon in the northernmost section of the Pacific coast area. The 23 localities examined in Washington and Oregon show only two in which *cheopis* is present. The 21 farther south, in California, show ten with *cheopis*. This may account in part for the fact that relatively fewer cases (only three from Seattle) of plague have been reported from these northern states in contrast with the record in California. The data also show that *cheopis* was found in relatively few localities of the dry northern and southern states. Of 28 localities from Idaho to

TABLE IIA
NUMBER OF INLAND LOCALITIES SURVEYED
AND NUMBER WITH FLEAS (PRINCE, 1943)

| STATE | NUMBER OF LOCALITIES: | | | | |
|--------|-----------------------|------------------------|--------------------------|---------------------------|-----------------------|
| | SURVEYED | WITH <i>CHEOPIS</i> | WITH <i>FASCIATUS</i> | WITH <i>GALLINACIA</i> | WITH <i>SEGNIS</i> |
| Wash. | 8 | 2 | 5 | 0 | 1 |
| Idaho | 2 | 0 | 1 | 0 | 0 |
| Mont. | 8 | 0 | 2 | 0 | 0 |
| Ore. | 15 | 0 | 7 | 0 | 1 |
| Wyo. | 3 | 0 | 1 | 0 | 0 |
| Calif. | 21 | 10 | 10 | 7 | 9 |
| Nev. | 2 | 0 | 2 | 0 | 0 |
| Utah | 3 | 1* | 2 | 0 | 0 |
| Colo. | 7 | 1** | 4 | 0 | 0 |
| Nebr. | 3 | 0 | 3 | 0 | 0 |
| Ariz. | 4 | 2 | 0 | 0 | 0 |
| N.Mex. | 8 | 2 | 3 | 3 | 1 |
| Texas | 3 | 0 | 2 | 1 | 0 |

TABLE IIB
NUMBER OF RATS EXAMINED
AND NUMBER OF FLEAS CLASSIFIED

| STATE | NUMBER OF RATS EXAMINED | NUMBER OF FLEAS ⁺ | | | |
|--------|-------------------------------|------------------------------|------------------|-------------------|---------------|
| | | <i>CHEOPIS</i> | <i>FASCIATUS</i> | <i>GALLINACIA</i> | <i>SEGNIS</i> |
| Wash. | 503 | 76 | 305 | 6 | 6 |
| Idaho | 246 | 0 | 62 | 0 | 0 |
| Mont. | 321 | 0 | 16 | 0 | 0 |
| Ore. | 171 | 0 | 40 | 0 | 5 |
| Wyo. | 110 | 0 | 1 | 0 | 0 |
| Calif. | 411 | 735 ⁺⁺ | 42 | 139 | 39 |
| Nev. | 40 | 0 | 15 | 0 | 0 |
| Utah | 371 | 475* | 18 | 0 | 0 |
| Colo. | 910 | 45** | 133 | 0 | 0 |
| Nebr. | 272 | 0 | 128 | 0 | 0 |
| Ariz. | 147 | 10 | 0 | 0 | 0 |
| N.Mex. | 411 | 30 | 27 | 35 | 3 |
| Texas | 128 | 0 | 10 | 1 | 0 |

⁺ Calculated from author's figures. These figures are not usable for calculating indices but only to compare relative abundance of fleas.

⁺⁺ 350 of these from San Diego.

* Salt Lake City.

** Denver.

Nebraska and Nevada to Wyoming, only two were found to have *cheopis*. Jellison, Kohls, and Mills (1943) found *fasciatus* but no *cheopis* in Montana. Of 15 localities in New Mexico, Arizona, and in the drier parts of Texas, only four showed *cheopis*. The writer's experience in the arid Brownfield, Texas, area was similar. Of several hundred Norway rats observed from December 1946 through August 1947, only two or three bore *cheopis*, none bore *fasciatus*, but many bore *gallinacea*.

Ewing and Fox (1943), summarizing data from other authors, record *cheopis* from Indianapolis, Indiana; St. Paul, Minnesota; Ames and Des Moines, Iowa; from Illinois and Ohio; Nashville, Tennessee; and Washington, D. C. It is probably distributed over a somewhat wider range; the localities named above are in or near university areas where collecting is fairly concentrated. It seems likely that this flea accompanied domestic rats and that its numbers in any area where rats are present depend considerably on climate and housing. *X. cheopis* populations can build up to large proportions under highly artificial conditions in cities and garbage dumps of areas otherwise apparently uncongenial. This is shown by records from Salt Lake City where 475 *cheopis* were found on 295 rats caught in dairies, markets, garbage dumps, and stockyards. That rats are visited by fleas from native rodents is shown by Prince. He recorded 18

of such species as having been found on domestic rats during the course of his survey. However, past records show that danger from plague outbreaks in cities in such areas has been small. Not a single case occurred in dry inland cities and few in northern cities anywhere.

The rat flea *N. fasciatus*, another possible vector of plague, has a numerical distribution somewhat different from that of *cheopis*. This species was found in about half of the 23 localities in Washington and Oregon and in all of the 21 California localities. It was found in about half of the dry northern-state localities and occurred less frequently in the dry portions of the southern areas. Its position as a possible plague-vector has already been discussed. It is reluctant to bite man and difficult to infect.

PORT-CITY SURVEYS. Results of domestic-rat flea surveys made by other authorities in cities in coastal areas are summarized in Tables III, IV, and V. The more useful summaries stress the percentages of rats found infested.

Data for western cities in these tables conform with the general pattern indicated by Prince's data; only 50% of the rats examined in Seattle had fleas, and the *cheopis* index was only 0.7. In Tacoma,

during the winter months, only nine *cheopis* were found on 2,648 rats. Farther south, at San Francisco, 64% of the rats had fleas. Rats averaged seven fleas each and 20% were *cheopis*. At Los Angeles the *Cheopis* index was 0.9 during a survey extending from December through August. (The authors had examined only 331 rats for fleas and did not record the percentage of rats with *cheopis*).

At Galveston, *cheopis* was the most prevalent rat flea during the plague outbreak during the 1920's. It was also the most prevalent at Mobile and at Jacksonville, 67 to 72% of the rats bearing them. At New Orleans, where the percentage of rats with *cheopis* was not recorded, the *cheopis* indices were fairly high (2.7 for the year). Farther north, Philadelphia had a small average number of *cheopis* per rat (0.03 to 4.2).

A number of these surveys are inadequate by present standards and needs. This is partly due to irregular distribution of samples during the season and partly because percentages of rats infested by each species of flea are not recorded. They do show, however, that cities which suffer from real plague outbreaks had a high proportion of rats infested with *cheopis*.

TABLE III
PERCENT OF RATS WITH FLEAS (YEAR)

| CITY | ALL FLEAS | *CHEOPIS | FASCIATUS | SEGNIS | GALLINACEA |
|-------------------------|-----------|----------|-----------|--------|------------|
| Seattle | 50 | — | — | — | — |
| San Francisco | 64 | — | — | — | — |
| <i>norvegicus</i> | — | 27 | 58 | 28 | 0 |
| <i>rattus</i> | — | 34 | 11 | 15 | 0 |
| Berkeley and Oakland | 77 | — | — | — | — |
| New Orleans | 87 | — | — | — | — |
| Mobile | — | 67* | 14 | 14 | 10 |
| Jacksonville | — | 72** | 6 | 27 | 19 |
| Norfolk | 57 | — | — | — | — |
| New York | 35 | — | — | — | — |
| Providence | 57 | — | — | — | — |
| Boston | 43 | — | — | — | — |

* Varied from 26 to 94

** Varied from 16 to 92

TABLE IV

DURATION, DATA AND DESCRIPTION OF CITY SURVEYS

| CITY | AUTHORITY | NO. OF RATS EXAMINED | SPECIES COMPOSITION: % <i>Norvegicus</i> | DURATION OF SURVEY |
|----------------------|---------------------------|----------------------|---|---------------------|
| Seattle | Fricks, 1936 | 261 | -- | Apr. '27 — Aug. '29 |
| Tacoma | Hundley & Nasi, 1945 | 2,048 | 72 | Nov. '42 — Mar. '43 |
| San Francisco | Eskey, 1938 | 3,027 | 75+ | 1936 — 1937 |
| Berkeley and Oakland | Mitzmain, 1910 | 4,916 | -- | Jan. — Dec. 1909 |
| Los Angeles | Trimble & Sherrod, 1935 | 231 | 97 | Dec. '31 — Aug. '32 |
| Galveston | Boyd & Kemmerer, 1920 | 46,623** | -- | June — Dec. 1920 |
| Galveston | Surgeon General, 1921 | 46 | 23 | July '20 — June '21 |
| Galveston | Surgeon General, 1922 | 56 | -- | July '20 — June '21 |
| New Orleans | Surgeon General, 1916 | 1,268 | 71* | July '15 — June '16 |
| New Orleans | Surgeon General, 1921 | 2,144 | -- | Nov. '22 — June '23 |
| New Orleans | Surgeon General, 1922 | 3,839 | 90+* | July '21 — June '22 |
| New Orleans | Fox and Sullivan, 1925 | 1,661 | 86* | July '20 — June '21 |
| Mobile | Cole & Koeppeke, 1946 | 6,123 | 95 | Jan. — Dec. 1934 |
| Pensacola | Surgeon General, 1921 | 752 | 91 | July '20 — June '21 |
| Jacksonville | Rumreich & Wynn, 1945 | 4,853 | 93 | Jan. — Dec. 1934 |
| Savannah | Fox, 1931 | 387 | -- | Feb. — Mar. 1927 |
| Savannah | Fox, 1931 | 500 | -- | Sept. — Oct. 1927 |
| Norfolk | Hasseltine, 1929 | 1,561 | 99+ | Mar. '27 — Mar. '28 |
| Philadelphia | Vogel & Cadwellader, 1935 | 2,765 | 99+ | May '32 — Dec. '33 |
| New York | Fox & Sullivan, 1925 | 4,756 | 99+ | Apr. '23 — Feb. '25 |
| Providence | Robinson, 1913 | 342 | -- | July — Dec. 1912 |
| Boston | Fox & Sullivan, 1925 | 1,524 | -- | Dec. '22 — Nov. '23 |

* Calculated from author's figures.

** Most of the rats were caught dead in snap traps.

TABLE V

| CITY | SPECIES-COMPOSITION OF FLEA INFESTATIONS (PERCENT) AVERAGE NO FLEAS PER RAT | | | | | |
|-------------------------|--|-----------|--------|------------|-------------|----------|
| | CHEOPIS | FASCIATUS | SEGNIS | GALLINACEA | ALL SPECIES | CHEOPIS |
| Seattle | 22 | 67 | 11 | 0 | 3 | 0.4 |
| Tacoma | ** | 95+ | 0 | 0 | 1.2 | -- |
| San Francisco | 20* | 66* | ** | 0 | 7 | -- |
| Berkeley and Oakland | -- | -- | -- | -- | 2.8 | -- |
| Los Angeles | 31* | ++ | *** | -- | 1.0 | 0.9 |
| Galveston '20 | *** | ** | -- | -- | 25 | -- |
| Galveston '21 | 90 | 7 | 3 | 0 | -- | -- |
| Galveston '22 | *** | 0 | ** | ** | -- | -- |
| New Orleans '16 | 74* | 2* | 30* | ** | 3.5 | 1.0-7.7 |
| New Orleans '21 | -- | -- | -- | -- | 3.3 | -- |
| New Orleans '22 | 37 | 0 | 58 | 0 | 4.8 | 1.7 |
| New Orleans '25 | 72 | 0.5 | 27 | ** | 2.3 | 1.2-3.2 |
| Mobile | 64 | 9 | 9 | 16 | 7 | 2.6-9.4 |
| Pensacola | 54* | 7* | 2* | + | 11 | 5 |
| Jacksonville | 58 | 2 | 19 | 20 | 6 | 1.6-7.5 |
| Savannah | 51 | 20 | 26 | 3 | 4.6 | 2.3-7.2 |
| Norfolk | 82 | 18 | 0 | 0 | | 0.02-8.4 |
| Philadelphia | 60 | 32 | ** | ** | 1.6 | .02-2.3 |
| New York | 30 | 70 | * | 0 | 0.9 | 0-0.8 |
| Providence | 75 | 22 | 3 | -- | 4-10 | |
| Boston | 33 | 64 | * | 0 | 1.0-1.4 | 0.1-1.0 |

+ *Gallinacea* not recorded but *Pulex irritans* recorded as making up about 20%.

++ Some of these surveys were intended chiefly to determine *Cheopsis* indices hence other species of fleas were not recognized or not counted. Percentages are therefore not monobasic but give only general indications. They also vary in accordance with the type of habitat, the species-composition of the rat sample and with the method of interpretation of the seasonal data.

* Calculated from author's total figures.

** Present, but in small numbers

*** Was most prevalent, numerous, or predominant flea.

SOUTHEASTERN STATES SURVEYS

Surveys in connection with murine typhus control by nine southeastern states, co-operating with the Communicable Disease Center, have resulted in the addition of considerable data.

Of these states, Texas is of particular interest because, as in California, the presence of plague in native rodents is close to a dangerously high population of *cheopis* on domestic rats. Analysis of 1946 and 1947 data shows that rats in southeastern Texas (east of the 100° west longitude and somewhat south of the Dallas-Fort Worth level) have a very high incidence of cheopism. Even during winter, when the level is low, more than 20 percent of the rats examined bore this flea. During summer more than 60 percent bore it. Past records indicate that plague will flourish in southeastern Texas if it bridges the few score miles from west Texas. Plague has existed among domestic rats in Galveston, Beaumont, and Port Arthur. Also, plague appears to flourish where murine typhus flourishes. A high incidence during all seasons of the year extends eastward through the typhus belt.

Data from Dallas and Fort Worth are not distributed widely enough through the year for analysis by months. Indications are, however, that the percentage of rats infested is fairly high. Between 27 and 39 percent of 297 rats captured in these two cities bore *cheopis*. In western Texas, however, the percentage of rats with *cheopis* is considerably smaller. Less than 14 percent of some 522 rats taken at all times of the year from El Paso and from panhandle counties were infested during 1946 and 1947.

East of Texas, the incidence of cheopism is high in southern states, particularly in the typhus belt (see page 65). It is lower in northern areas. Its seasonal peak of abundance varies: in more southern states it occurs in summertime; in more northern states in the fall.

The northern rat flea is quite uncommon in Texas, but the sticktight flea is extremely common. West of the 100th degree

west longitude, the sticktight flea is by far the most abundant rat flea on farms and on outskirts of cities.

TEMPERATURE, LATITUDE, AND PLAGUE

The possible effects of temperature on occurrence of rat-borne and human plague are discussed by Robertson (1923) who generalized as follows:

1) Bubonic plague is essentially a disease of hot climates. Once introduced into tropical countries, it tends to persist indefinitely.

2) Outside of the immediate tropics, this disease is rather definitely limited in the extent to which it will spread.

3) In countries with a mean midwinter temperature of 45° F. or below, bubonic plague is occasional, accidental, and distinctly self-limited. It seems possible for it to occur in the colder regions only for short periods under unusual conditions.

When evaluating these generalizations, it is important to keep in mind that little was known concerning the distribution of *sylvatic* plague when Robertson wrote them. Also, his conclusions applied, and still apply, only to rat-borne and human cases. He found that the area of incidence of such plague was bounded roughly by the thirty-fifth parallel both north and south of the equator. In Europe, however, the 45° north latitude probably described the northern boundary more accurately. This northerly occurrence is due mostly to the tempering effect of the warm waters of the Mediterranean.

Robertson discussed some apparent exceptions to the rule of temperature, which may be of interest in the United States. One of the most impressive was an outbreak of 50,000 cases of pneumonic plague in Manchuria during the winter of 1911. The basic causes of this outbreak are ascribed to contact with a sylvan rodent, the tarbagan. Like our American marmots, the tarbagan harbored plague. Poor Manchurian peasants and fur-hunters who skinned hundreds of thousands of tarbagans, contracted plague. Crowded together in poor

shelters, they passed it on pneumonically to fellow inhabitants, friends, and relatives. Such an extensive outbreak could hardly occur in America under our present eating and living conditions. A limited number of cases have occurred under circumstances very similar to it, however, namely 13 in Berkeley and 41 in Los Angeles, California.

It is Robertson's theory that the chain of plague-transmission from rat to rat is broken in northern latitudes by early onset and long duration of cold weather. Winter weather reduces the numbers of *cheopis* on rats to such a low point that plague dies out. Fricks' (1936) findings in Seattle, and previously-mentioned findings in Tacoma, apparently support this thesis. He states that rat plague existed in Seattle for at least 10 years, the first infected rat having been found there in November 1907, the last in March 1917. Fricks adds that it is interesting to speculate on the length of time plague may have existed prior to its discovery in 1907. His reasons: only one case of flea-borne plague (the other two officially-reported human cases were pneumonic) is known to have occurred in Seattle; and rat plague existed for almost 10 years subsequently without another human case. He points out that of two campaigns to eradicate plague in Seattle, the first, which followed promptly the discovery of the human cases, was discontinued after a few months of activity. The second, which followed a great increase in rat plague discovered in October 1913, was dis-

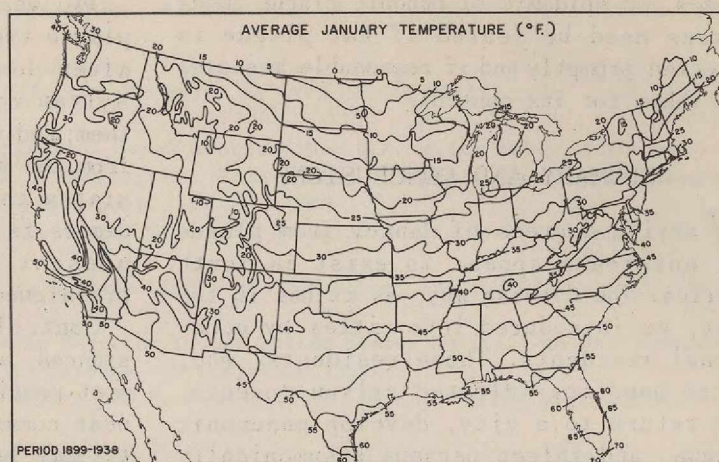


FIGURE 2. Average January temperature in the United States for the period 1899-1938 as shown in Yearbook of Agriculture for 1941. Both plague and murine typhus have attacked man in the more humid, warm regions.

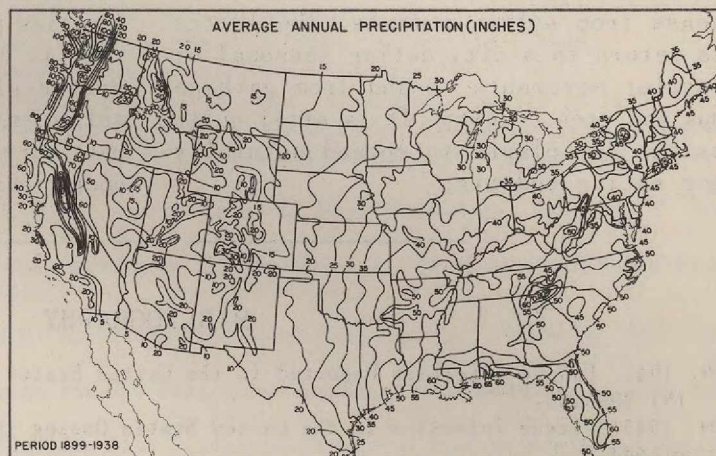


FIGURE 3. Average annual precipitation in the United States for the period 1899-1938 as shown. Severely dry climates suppress the numbers of *cheopis* fleas.

continued after three years. However, surveys continued to June 1933. Fricks concluded that probably no extensive infection of rats would result if plague were again introduced. His premise: the uniformly low flea index observed (and he might have added low flea-infestation-incidence); and the pronounced improvement in sanitary conditions during the past two decades. Fricks states that although Seattle climate favors development of pneumonic

phases, no epidemic of bubonic plague among humans need be feared if rat plague is detected promptly and if reasonable measures are taken for its control.

SUMMARY AND CONCLUSIONS

Varying degrees of danger from plague outbreaks appear to exist in North America. The disease may, as it has in the past, be introduced into cities by occasional residents. These residents, who, after handling infected sylvan rodents, may return to a city, develop pneumonic plague, and infect persons pneumonically by means of droplet infection. Plague may, as it has in the past, be introduced among resident rats in cities by: (1) domestic rats from ships or other common carriers; (2) domestic rats which have acquired the disease from sylvan rodents. The latter rats return to a city during seasonal or irregular movements to and from garbage dumps and open country. It is still quite possible for plague to spread naturally among native rodents.

Two weak links in the entire chain of plague transmittal exist: the stage when, after domestic rats acquire plague from sylvan rodents, it is maintained among them; and when plague is finally transmitted from the rats to human beings. Eskey (1938) states that the intensity of plague epidemics is regulated by the species of flea present. It is also governed by the prevalence of that flea.

Control, except under unique circumstances, will probably be aimed largely and most profitably at domestic rats inside and near communities, though in limited areas it may be extended to certain easily controllable native rodents.

Permanent control of rats and rat fleas in communities where plague would exist among them prevents its gaining a foothold. Permanent control in non-infected communities prevents its spread to them.

It is believed well worth while to conduct ecological surveys of rat fleas in sufficient proportion to measure and delineate varying types of danger areas wherever operations for control are undertaken.

BIBLIOGRAPHY

- ANON. 1941. Plague Infection Reported in the United States in 1940. Public Health Reports, 56 (9) 399-400.
- ANON. 1943. Plague Infection in the United States During 1942. Public Health Reports, 58 (16) p. 641.
- ANON. 1944. Plague Infection in the United States During 1943. Public Health Reports, 59 (28) 911-915.
- ANON. 1945. The Control of Communicable Diseases. American Public Health Association, 1945.
- ANON. 1947. Plague Infection in the United States in 1946. Public Health Reports, 63 (37) 1336-1340.
- ANON. 1947. Fatal Case of Plague in California. Public Health Reports, 62 (29) p. 1068.
- ANON. 1947. Plague Infection Report in the United States in 1945, Public Health Reports, 62 (37) 1336-1339.
- BOYD, MARK F. and T. W. KEMMERER. 1921. Experience with Bubonic Plague (Human and Rodent) in Galveston, 1920. Public Health Reports, 35 (30) 1754-1764.
- BURROUGHS, ALBERT L. 1947. Sylvatic Plague Studies: The Vector Efficiency of Nine Species of Fleas Compared with *Xenopsylla cheopis*. J. Hyg. 4S (3) 371-396.
- BYINGTON, L. B. 1940. Two Epizootics of Plague Infection in Wild Rodents in the Western United States in 1938. Public Health Reports, 55 (33) 1496-1501.
- COLE, LAMONT C. and JEAN A. KOEPKE. 1946. A Study of Rodent Ectoparasites in Mobile, Alabama. Public Health Reports, 61 (41) 1469-1487.
- ESKEY, C. R. 1938. Recent Developments in our Knowledge of Plague Transmission. Public Health Reports, 43 (1) 49-57.
- ESKEY, C. R. 1938. Flea Infestation of Domestic Rats in San Francisco, California, Public Health Reports, 43 (23) 948-951.

- ESKEY, C. R. and V. H. HAAS. 1940. Plague in the Western Part of the United States, Public Health Bulletin, 254, pp. 1-82.
- EVANS, F. C., C. M. WHEELER and J. R. DOUGLAS. 1943. Sylvatic Plague Studies: An Epizootic of Plague Among Ground Squirrels (*Citellus deecheyi*) in Kern County, California. Jour. Infect. Diseases. 72, pp. 68-76.
- EWING, F. C. and IRVING FOX. 1943. Fleas of North America. U.S.D.A. Misc. Publ., 500.
- FOX, CARROL and E. C. SULLIVAN. 1925. A Comparative Study of Rat-Flea Data for Several Seaports of the United States. Public Health Reports, 40 (37) 1909-1934.
- FOX, CARROL. 1931. A Limited Rat Flea Survey of Savannah, Georgia. Public Health Reports, 46 (11) 574-575.
- FRICKS, L. D. 1936. Review of Plague in Seattle (1907) and Subsequent Rat and Flea Survey. Public Health Bulletin, 232, pp. 1-28.
- HAMPTON, BROCK C. 1945. Plague Infection Reported in the United States During 1944 and Summary of Human Cases 1900-44. Public Health Reports, 60 (46) 1361-1365.
- HASSELLTINE, H. E. 1934. Rat-Flea Survey of the Port of Norfolk. (Va.). Public Health Reports 44 (11) 579-589.
- HUBBARD, CLARENCE ANDERSON, 1947. Fleas of Western North America. Iowa State College Press, pp. 1-533.
- HUNDLEY, JAMES H. and KAARLO W. NASI. 1945. Anti-Plague Measures in Tacoma, Washington. Public Health Reports, 59 (33) 1239-1255.
- ILLINGWORTH, J. F. 1916. Notes on the Hen Flea (*Echidnophaga gallinacea* Westw.) Hawaii Ent. Soc., Proc. (1915) 3: 252-254.
- JACKSON, V. W. 1925. Spread of Animals on the Prairie. Country Guide. Winnipeg.
- JELLISON, WILLIAM L. 1939. Sylvatic Plague: Studies of Predatory and Scavenger Birds in Relation to its Epidemiology. Public Health Reports, 54 (19) 792-798.
- KELLOG. W. H. 1920. Epidemic of Pneumonic Plague. Amer. J. Publ. Health, 10 p. 599.
- MEYER, K. F. 1941. The Ecology of Plague. Medicine, 21 (2) pp. 143-174.
- MEYER, K. F. 1942. The Known and Unknown in Plague. Am. Jour. Trop. Med., 22 (1) pp. 9-36.
- MEYER, K. F., R. HOLDENRIED, AL BURROUGHS and E. JAWETZ. 1943. Sylvatic Plague Studies—Inapparent, Latent Sylvatic Plague in Ground Squirrels in Central California. Jour. Infectious Diseases, 73, pp. 144-147.
- MEYER, K. F. 1947. The Prevention of Plague in the Light of Newer Knowledge. Anns. N. Y. Acad. Sci., XLVIII (6) 429-467.
- MITZMAIN, MAURICE B. 1910. General Observations on the Bionomics of the Rodent and Human Fleas. Public Health Bulletin, 38 (1910).
- MOHR, CARL O. 1947. Table of Equivalent Populations of North American Small Mammals. Amer. Midl. Natrl., 37 (1) pp. 223-249.
- PARHAM, D. C. 1923. Biological Notes on the Hen Flea, *Echidnophaga gallinacea*. Jour. Agr. Res., 23, 1007-1009.
- PRINCE, FRANK M. 1943. Species of Fleas on Rats Collected in States West of the 102nd Meridian and Their Relation to the Dissemination of Plague. Public Health Reports, 58 (18) 700-708.
- RANDOLPH, NEAL M. and RICHARD B. EADS. 1946. An Ectoparasitic Survey of Mammals from Lavaca County, Texas. Anns. Ent. Soc. America, 39 (4) 597-600.
- ROBERTSON, H. McG. 1923. A Possible Explanation of the Absence of Bubonic Plague in Cold Countries. Public Health Reports, 38 (27) 1519-1531.
- ROBINSON, GEORGE H. 1913. The Rats of Providence and Their Parasites. Am. Jour. Publ. Health, 3 (8) 773-776.
- RUMREICH and R. S. WYNN. 1945. A Study of the Rodent-Ectoparasite Population of Jacksonville, Florida. Public Health Reports, 60 (3) pp. 885-905.
- SURGEON GENERAL. Annual Reports of the Surgeon General of the Public Health Service of the United States for the Fiscal Years 1916, 1921, and 1922.
- TRIMBLE, H. E. and G. C. SHERRARD. 1935. Rat and Rat-Flea Survey of Los Angeles Harbor, Public Health Reports, 50 (3) 74-79.
- VOGEL, C. W. and CHARLES CADWELLADER. 1935. Rat-Flea Survey of the Port of Philadelphia, Pa. Public Health Reports 50 (30) 952-957.
- WAYSON, N. E. 1947. Plague — Field Surveys in Western United States During Ten Years (1936-1945). Public Health Reports, 62 (22) pp. 780-791.
- WHEELER, C. M., DOUGLAS, J. R. and ECANS, F. C. 1941. The Role of the Burrowing Owl and the Stick-Tight Flea in the Spread of Plague. Science, 94, 560-561.
- YOUNG, STANLEY P. and HAROLD W. DOBYNS. 1937. U. S. Dept. Agr. Leaf. No. 132:1-8.

COUNTIES PRE-APPROVED FOR
TYPHUS CONTROL OPERATIONS — 1948 SEASON

Computed from (1) counties with 10 or more cases of human typhus per year for 5-year period 1943-1947; (2) counties with 10 or more cases for year 1947.

| | | | |
|--------------------|----------------------|---------------|----------------|
| ALABAMA (14 Cos.) | Georgia (Con't) | Texas (Con't) | Texas (Con't) |
| Barbour | Laurens | Dallas | Milam |
| Calhoun | Mitchell | DeWitt | Nueces |
| Coffee | Richmond | Erath | Runnels |
| Covington | Screven | Fayette | Tarrant |
| Crenshaw | Sumter | Galveston | Tom Green |
| Dale | Tattnall | Gonzales | Travis |
| Dallas | Telfair | Guadalupe | Victoria |
| Geneva | Terrell* | Harris | Waller |
| Henry | Thomas | Henderson* | Washington |
| Houston | Tift | Hidalgo | Webb |
| Jefferson | Ware | Howard* | Wharton |
| Mobile | Wayne | Jasper | Willbarger |
| Montgomery | Worth | Jefferson | Wilson* |
| Pike | Berrien | Lampasas | Jim Wells |
| | Irwin | Lavaca | Falls |
| | | Lee | Harrison |
| ARKANSAS (1 Co.) | LOUISIANA (5 Cos.) | McLennan | Liberty |
| Pulaski | Caddo | Madison* | Robertson |
| | Calcasieu* | | |
| CALIFORNIA (1 Co.) | Iberia* | | |
| Los Angeles | Orleans | | |
| | Washington | | |
| | | | |
| FLORIDA (9 Cos.) | MISSISSIPPI (2 Cos.) | | |
| Dade | Harrison | | |
| Duval | Pike | | |
| Escambia | | | |
| Hillsborough | | | |
| Nassau | N. CAROLINA (3 Cos.) | | |
| Orange | Craven | | |
| Pinellas | New Hanover | | |
| Polk | Wilson | | |
| Volusia | | | |
| | | | |
| GEORGIA (30 Cos.) | S. CAROLINA (2 Cos.) | ALABAMA | NORTH CAROLINA |
| Appling | Charleston | Autauga | Harnett |
| Bibb | Orangeburg | | |
| Bulloch | | FLORIDA | TEXAS |
| Chatham | TENNESSEE (1 Co.) | Gadsden | Anderson |
| Coffee | Davidson | | Bosque |
| Colquitt | | GEORGIA | Comanche |
| Crisp | TEXAS (43 Cos.) | Emanuel | Fisher |
| Decatur | Angelina | Evans | Crayson |
| Dodge | Bastrop | Schley | Grimes |
| Dooly | Bexar | Turner | Hunt |
| Dougherty | Caldwell | Randolph | Nolan |
| Fulton | Camerson | | Refugio |
| Glynn | Colorado | LOUISIANA | |
| Grady | Comal | La Fayette | |
| Jenkins | | St. Landry | |

*No cases reported in 1947

COUNTIES REPORTING
10 OR MORE CASES — 1946