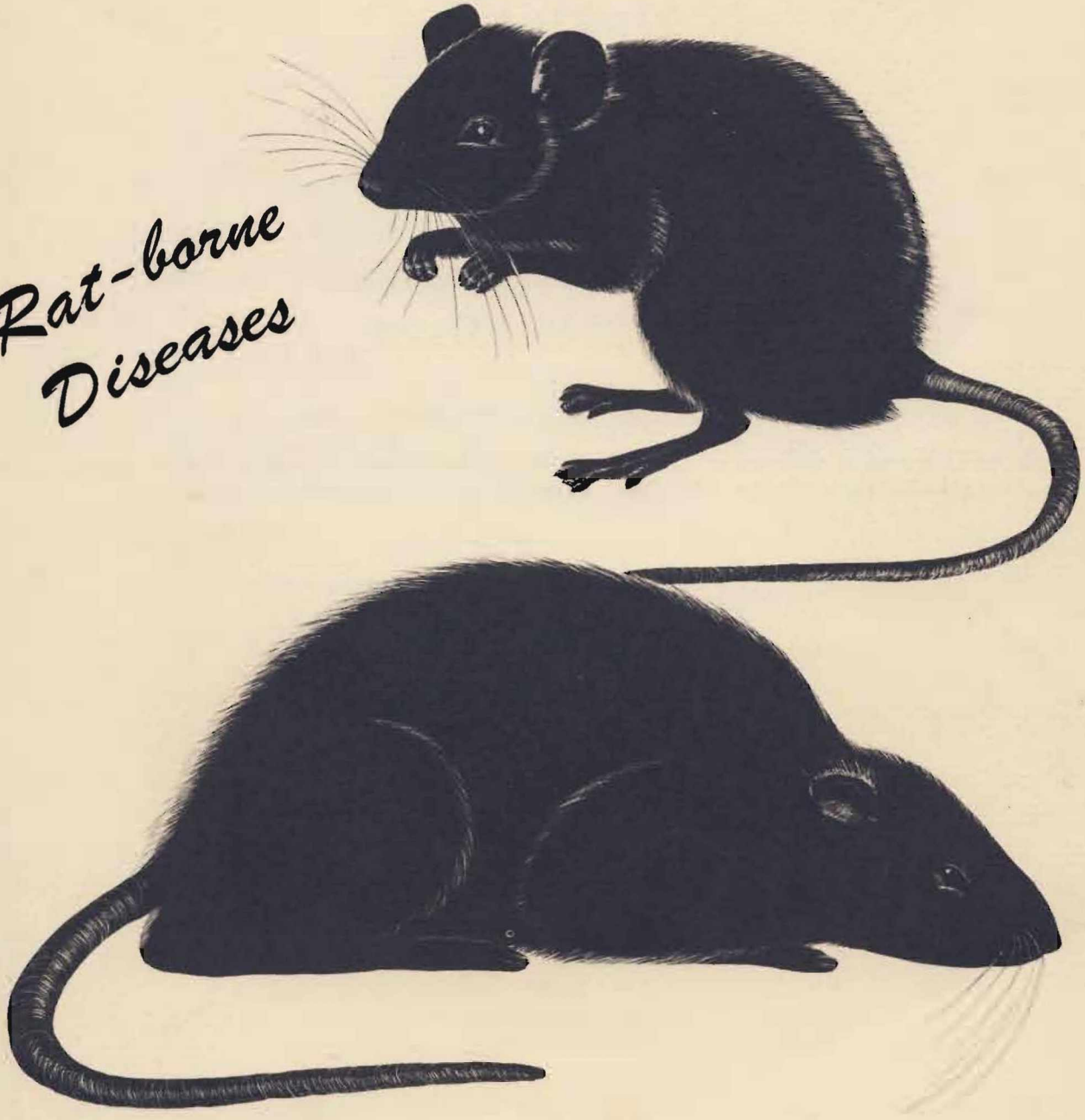
# CDC BULLETIN

AUGUST - 1950

## *Rat-borne Diseases*



**FEDERAL SECURITY AGENCY**
**Public Health Service**
**Communicable Disease Center**
**Atlanta, Ga.**

**COVER:** Depicted on the cover of this issue of the CDC Bulletin are the roof rat (*Rattus rattus*), upper, and the Norway rat (*Rattus norvegicus*) – two principal disease-bearing species.

CDC BULLETIN
Vol. IX. No. 8 AUGUST-1950

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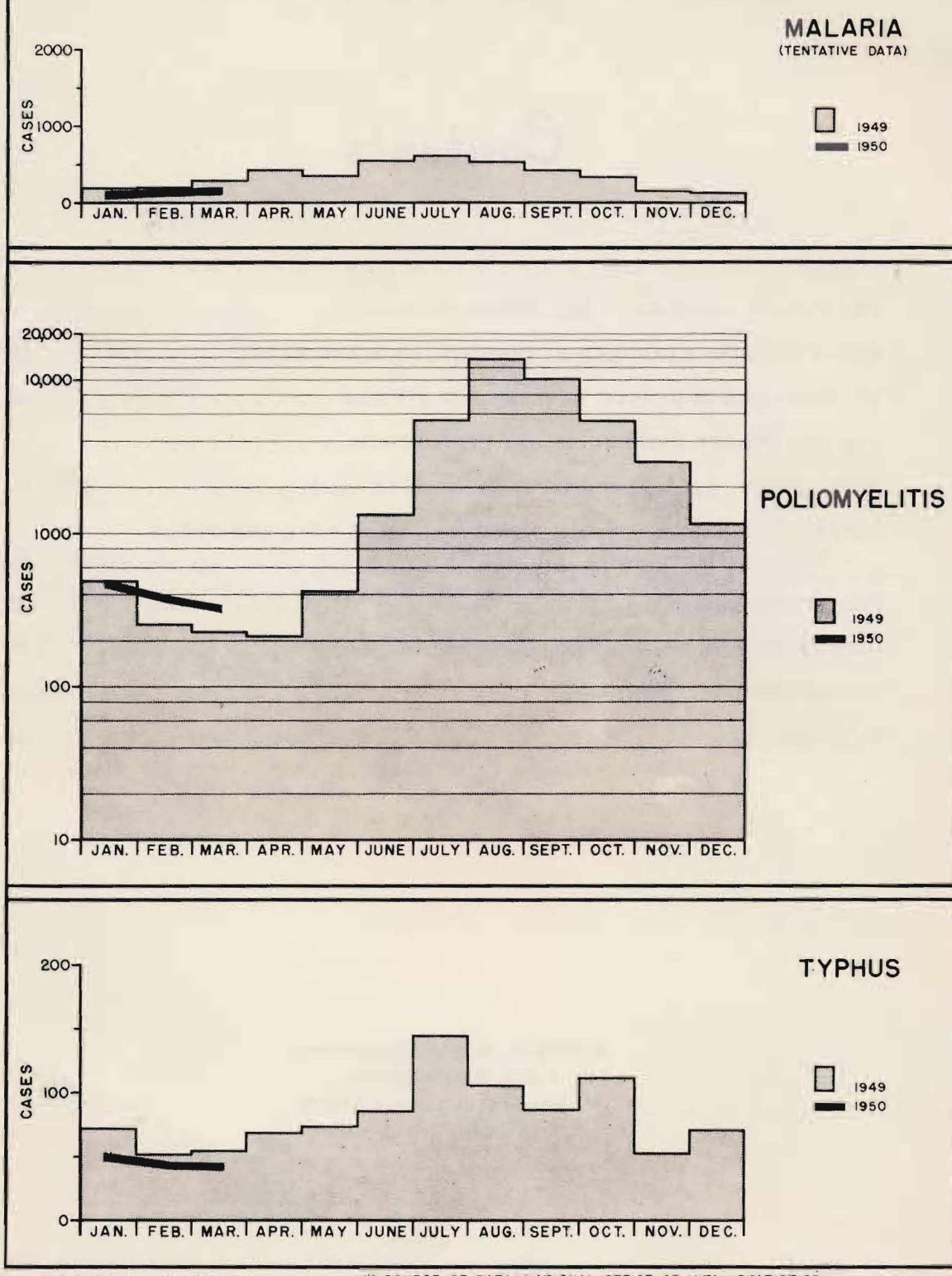
**FEDERAL SECURITY AGENCY**
**Public Health Service**
**Communicable Disease Center**
**Atlanta, Georgia**

The printing of this publication has been approved by the Director of the Bureau of the Budget, January 19, 1950.

# MORBIDITY TOTALS FOR THE UNITED STATES *
# MALARIA, POLIOMYELITIS, TYPHUS

1949 - COMPLETE 1950 - AS REPORTED




FSA-PHS-CDC ATLANTA, GA.

* SOURCE OF DATA: NATIONAL OFFICE OF VITAL STATISTICS

# Plague

VERNON B. LINK, Senior Surgeon*

Plague always has been one of mankind's worst enemies. As a pestilential disease, it can be traced back uninterruptedly to the third century before the Christian era when Dionysius spoke of it as a fatal disease in Libya, Egypt, and Syria. Prior to that time, Homer described plague among the Greeks at the siege of Troy in 1184 B.C., ascribing it to the wrath of Apollo who was angered at an insult to his high priest Chryses, and also to the god's malevolence and disgust at the filth lying about the camp. Probably the earliest known reference to plague occurs in the First Book of Samuel, Chapters 5 and 6, where mention is made of the disease having broken out in Canaan during military operations against the Israelites centuries before the Christian era. It is stated that the inhabitants of several cities were attacked with emerods, and that the pestilence caused a deadly destruction. In Bethschemesch, over 50,000 persons died. It also is recorded that in order that the plague might be stayed, the Philistines made propitiatory offerings of golden images of their tumors and of the mice that marred the land to the God of Israel. This appears to be the earliest reference to an epizootic among mice in connection with the disease.

Rufus of Ephesus, about 100 A.D., probably gave the first description of plague which has been preserved. The first recorded plague epidemic in the world's history occurred in the reign of Marcus Aurelius 164-180 A.D., the second, in Egypt in 542. The Great Plague of Justinian in the sixth century is said to have carried off half the population of the Roman Empire.

In the fourteenth century, a new European epidemic began which became known as the Black Death. It is estimated that it killed one-fourth of the population of Europe, or about 25 million persons. In some countries, however, the total deaths approximated 70 percent of the inhabitants. Plague continued to hold sway in Europe during the fifteenth, sixteenth, seventeenth, and eighteenth centuries, but seems to have disappeared by the middle of the nineteenth century.

The present pandemic, which began in the Orient in 1894, has had the most widespread distribution of any of the known epidemics. It has invaded every continent of the world, and has been reported from nearly every country. India has suffered the most with about 12 million deaths reported in the last 54 years. Although the mortality rate per world population does not approach that of the Black Death of the fourteenth century, the present pandemic ranks alongside the most important previous ones as far as the total number of cases is concerned; and far exceeds all others in regard to its widespread distribution (table 1).

The New World was invaded by plague at Asuncion, Paraguay, in April 1899. Since that time there have been about 60,000 cases reported in North and South America. About 98 percent of these have occurred in South America. Although nearly 900 cases have been reported in Mexico, these occurred in two sharp outbreaks in 1902-3 and 1920-22, and there is no reason to believe that any endemic foci exist in either domestic or wild rodents.

While the United States is second in North America from the point of view of total cases reported, it is first in importance from the over-all viewpoint because of the existence of a tremendous endemic reservoir of infection in its wild rodent populations (table 2).

Quarantine officials began to be concerned about the importation of human plague into this country about the time that the present pandemic started. Their fears appeared to be justified when cases of plague were found on three occasions in 1899 and 1900, on board ships arriving at San Francisco, Calif.; New York, N.Y.; and Port Townsend, Wash. Although maritime quarantine was apparently successful in preventing the importation of human plague, little then was known of the role which the rat and its flea played in transmission, and it was inevitable that rodent plague would be imported. There is no available evidence to show when this occurred; but at some time during the last years of the nineteenth cen-

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Table 1

WORLD DISTRIBUTION OF PRESENT PLAGUE PANDEMIC

| Year First Reported | Location |
|---|---|
| 1893 | Yunnan, China |
| 1894 | Canton, China |
| 1896 | Formosa |
| | Bombay, India |
| | Basra, Turkey |
| 1897 | Jiddah, Arabia |
| 1898 | Osaka, Japan |
| | Maritius, Madagascar |
| | Samarkand, Russia |
| | Vienna, Austria |
| | London, England |
| 1899 | Alexandria, Egypt |
| | Santos, Brazil |
| | French Ivory Coast |
| | Lorenzo Marquez, P.E. Africa |
| | Lisbon, Portugal |
| | Bushire, Persia |
| | Reunion Island |
| | Straits Settlements |
| | Paraguay |
| | Kolobooka, Astrakhan, Russia |
| | Rosario, Argentina |
| | Honolulu, Hawaii |
| | Cape Town, British South Africa |
| | Numea, New Caledonia |
| | Barcelona, Spain |
| | Beirut, Syria |
| | Zanzibar |
| 1900 | Sydney, Australia |
| | Auckland, New Zealand |
| | Persian Kuidistan |
| | Manila, Philippines |
| | Glasgow, Scotland |
| | San Francisco, U.S.A. |
| | Hamburg, Germany |
| | Marseille, France |
| | Cardiff, Wales |
| | Cape Verde Islands |
| 1901 | Naples, Italy |
| | Montevideo, Uruguay |
| | Siam |
| 1902 | Mazatlan, Mexico |
| | French Indo-China |
| | Nairobe, Kenya |
| 1903 | Pisco, Peru |
| | Iquique, Chile |
| | Algeria |
| 1905 | Sumatra |
| | Panama |
| 1906 | Liberia |
| | Canary Islands |
| | Tenerife |
| 1907 | Tunisia |
| | Trinidad |
| 1908 | Azores |
| | Venezuela |
| | Ecuador |
| | Virgin Islands |
| 1910 | Morocco, Casablanca |
| 1911 | Java |
| 1912 | Cuba |
| | Puerto Rico |
| | Granada |
| 1915 | Greece |
| 1921 | Bolivia |
| 1939 | Canada |

tury, infected rats must have left ships in San Francisco harbor and spread plague among the rats of that city. Recognized human cases did not appear until March 1900, although there is some reason to believe that unsuspected cases were occurring as early as 1898. The first San Francisco epidemic lasted for nearly 4 years and accounted for 127 cases, few of which recovered. This epidemic was notable for the fact that there was violent disagreement between the factions who claimed that it was and was not plague. Subsequent epidemics occurred in San Francisco in 1907; in Seattle in 1907; in New Orleans in 1914 and 1919; in Oakland in 1919; in Pensacola, Galveston, and Beaumont in 1920; and in Los Angeles in 1924 (figure 1, table 3). All of these, with the exception of the 1919 outbreak in Oakland which started when a squirrel hunter developed secondary pneumonic plague, took place at a time when domestic rats were involved in an active

Table 2

PLAGUE CASES IN THE NEW WORLD

| Country | Cases | Percent of Total |
|---|---|---|
| Peru | 21,664 | 36.5% |
| Ecuador | 11,770 | 19.8% |
| Brazil | 7,986 | 13.5% |
| Argentina | 6,889 | 11.6% |
| Chile | 5,125 | 8.6% |
| Bolivia | 3,348 | 5.6% |
| Mexico | 868 | 1.4% |
| Venezuela | 565 | 0.9% |
| United States | 503 | 0.8% |
| Paraguay | 500 | 0.8% |
| Puerto Rico | 88 | |
| Cuba | 68 | |
| Trinidad | 13 | 0.5% |
| Panama | 2 | |
| Granada | 1 | |
| Canada | 1 | |
| **Total** | 59,391 | 100.0% |

Adapted from Moll and O'Leary: Plague in the Americas: An Historical and Quasi-Epidemiological Survey.

plague epizootic. The only time in our history when it was known that rats were involved in such epizootics and no human cases arose, was during the period from 1942-44 when the rats of Tacoma, Wash., were involved and over 100 were proved to be infected. At the present time, there is no city in the country where domestic rodents are known to be infected with plague, and active efforts constantly are being made to prevent this from happening.

At some time about the beginning of this century, plague spread from domestic rats in the San Francisco Bay area to the California ground squirrel. Again there is no evidence as to when this might have occurred. The first indication that it was a possibility was in 1903 when human cases were reported in rural Contra Costa County in an area where there were no rats. At that time, it was suspected that the source of the cases might have been from infected wild rodents, and a search was made but none were found. It was not until during the second San Francisco epidemic, in 1908, that infected ground squirrels were found on a ranch in Contra Costa County, where a human

Figure 1

COUNTIES WHERE HUMAN PLAGUE HAS OCCURRED

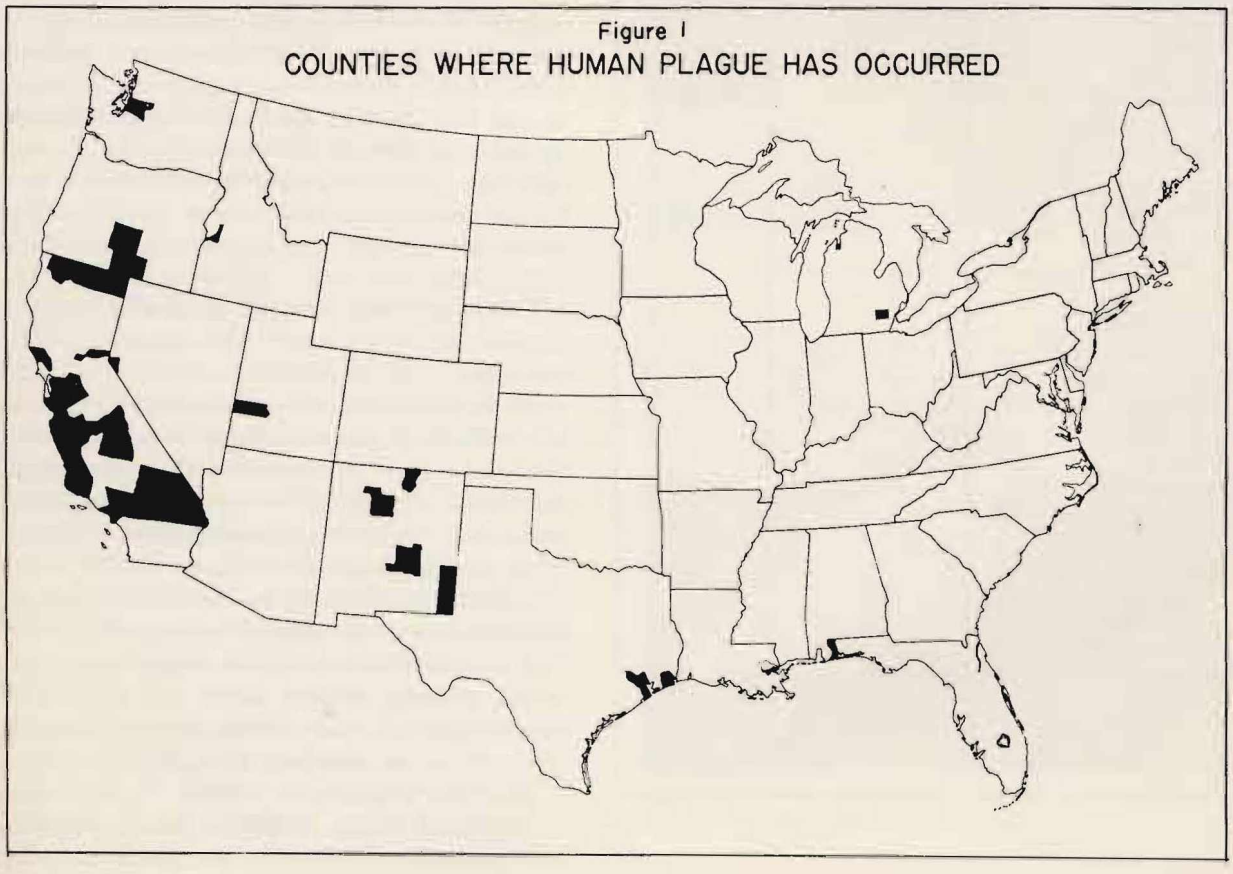


Table 3

COUNTIES IN THE UNITED STATES IN WHICH HUMAN PLAGUE HAS OCCURRED, 1900-1950.

| STATE AND COUNTY | CASES | DEATHS |
|---|---|---|
| **California** | | |
| Alameda | 41 | 28 |
| Contra Costa | 15 | 13 |
| Fresno | 1 | 1 |
| Los Angeles | 42 | 36 |
| Modoc | 1 | 1 |
| Monterey | 2 | 1 |
| Placer | 1 | 0 |
| San Benito | 4 | 3 |
| San Bernardino | 1 | 0 |
| San Francisco | 289 | 196 |
| San Joaquin | 1 | 0 |
| San Luis Obispo | 1 | 0 |
| Santa Barbara | 1 | 1 |
| Santa Clara | 1 | 0 |
| Santa Cruz | 2 | 0 |
| Siskiyou | 4 | 3 |
| Sonoma | 1 | 0 |
| Stanislaus | 2 | 0 |
| Tulare | 1 | 1 |
| **Florida** | | |
| Escambia | 10 | 7 |
| **Idaho** | | |
| Gem | 1 | 1 |
| **Louisiana** | | |
| Orleans | 51 | 18 |
| **Michigan** | | |
| Washtenaw | 1 | 0 |
| **Nevada** | | |
| Douglas | 1 | 0 |
| **New Mexico** | | |
| Taos | 1 | 0 |
| Lincoln | 1 | 1 |
| Lea | 1 | 0 |
| Sandoval | 1 | 0 |
| **Oregon** | | |
| Lake | 1 | 1 |
| **Texas** | | |
| Galveston | 16 | 11 |
| Harris | 1 | 0 |
| Jefferson | 14 | 7 |
| **Utah** | | |
| Beaver | 1 | 0 |
| **Washington** | | |
| King | 8 | 8 |
| **Total:** 11 States, 34 Counties | 520 | 338 |

case had died. At about the same time, a boy in Los Angeles was bitten by a squirrel and developed plague. A search for infected squirrels revealed one in the same general area of the city. Because of these findings, a general survey of California ground squirrels was started in April 1909. The disease was found so widespread in the squirrels of Contra Costa and Alameda that the survey was extended to other California counties, and by the middle of 1910 plague foci had been found in 10 of the 25 counties which represented all but the extreme northeastern and eastern range of the California ground squirrel.

In 1911, infected squirrels had been found in so many counties that fears began to be expressed that plague might already have spread to other States, and reconnaissance surveys were made in the counties of Oregon, Nevada, and Arizona which bordered on California. No infected squirrels were found outside of California at that time.

It then appeared that (1) in order to prevent the reinfection of domestic rodents from wild rodent sources and (2) in order to prevent the establishment of endemic foci of plague which would be a menace not only to California cities but to the rest of the country, it was desirable to eradicate plague in the California ground squirrel. The eradication proposal was divided into two major parts: first, a squirrel-free zone would be created around the cities of the San Francisco Bay area in order to prevent the reintroduction of plague into the domestic rodent populations of those cities; and second, rural plague was to be eradicated by the use of every tool known to be effective.

The program was launched as a fairly long-range proposition which might take several years to accomplish. Federal, State, and county funds were provided to carry on these activities. Literally millions of ground squirrels were killed during the years that millions of dollars were poured into this activity. The Public Health Service maintained an active interest in the eradication of plague in ground squirrels until 1936 when it turned over its share of the work to the California State Department of Health. The State of California has continued some type of plague control ever since, although it long since has given up the idea of being able to eradicate plague in the ground squirrel, which never was accomplished.

A second phase in the history of plague among wild rodents began in 1934 when a sheepherder came down with the disease in Lake County, Oreg.

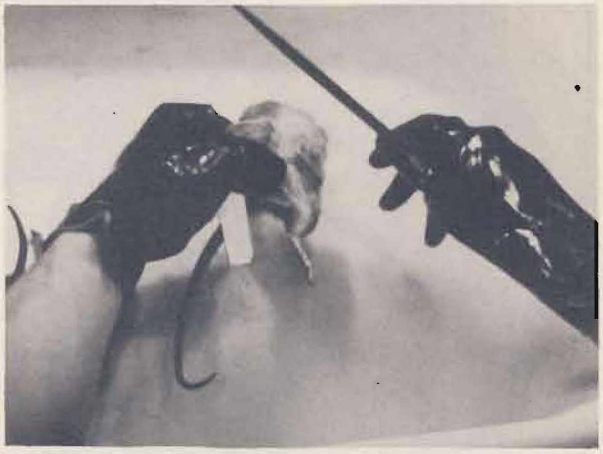
The following year, a search was made for infected rodents in the same area and plague-infected squirrels were found. This was the first evidence that the wild rodents of any State outside of California were involved. These findings led the Service to concentrate its efforts on determining just how widespread plague actually was in the West.

During the past 14 years, from 4 to 14 mobile survey units have been sent out into the field during the spring, summer, and fall months. These units are field laboratory trucks equipped for hunting and trapping. A two-man crew traps and shoots wild rodents, performs autopsies, and removes ectoparasites from the animals obtained. If the animal's organs appear infected, samples are expressed to the San Francisco laboratory in iced thermos jugs. Here the tissue is macerated and injected into or inoculated onto the skin of a guinea pig. If plague is present, the guinea pig generally dies in about 5 days and a presumptive diagnosis can be made at autopsy. This diagnosis then is confirmed by bacteriological procedures and serological tests. All ectoparasites removed from wild rodents are placed in 2 percent saline in glass vials and shipped to the laboratory in mailing tubes. The ectoparasites are counted, identified as to species, macerated in mortars, emulsified in normal saline, and injected into or inoculated onto the skin of guinea pigs.

Beyond the earlier, historical findings of plague which followed introduction in Texas, Louisiana, and Florida, surveys now have been made in 17 of the westernmost States, and nearly every county west of the 100th meridian has been surveyed one or more times. Over 1½ million wild rodents and their ectoparasites have been examined, and nearly 4,000 found to be plague-infected. Plague foci have been found in 131 counties of 15 of these Western States. In addition to California and Oregon, plague first was found in the following States in the years given: Montana, 1935; Idaho, Nevada, Utah, and Wyoming, 1936; Arizona, New Mexico, and Washington, 1938; Colorado and North Dakota, 1941; Oklahoma, 1944; Kansas, 1945; and Texas, 1946 (figure 2). Human cases resulting from contact with infected wild rodents have occurred in California, Nevada, Oregon, Idaho, Utah, and New Mexico (figure 1). (The single case in Washtenaw County, Mich., is a laboratory infection.)

The treatment of human plague has been improved greatly in recent years. Formerly, bubonic

**HANDLING OF ECTOPARASITES**



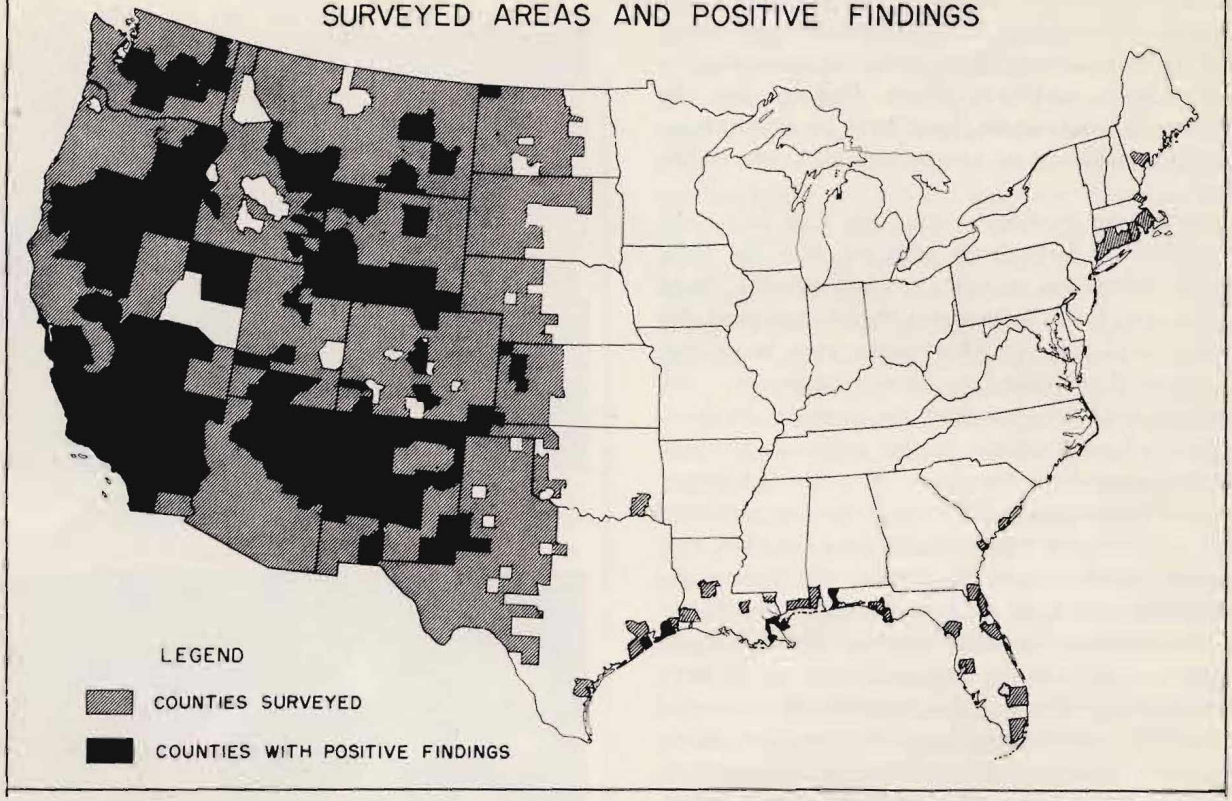
Removing ectoparasites from wild rodents.

Counting and identifying ectoparasites.

Injecting ectoparasites into skin of guinea pig.

Figure 2

SURVEYED AREAS AND POSITIVE FINDINGS

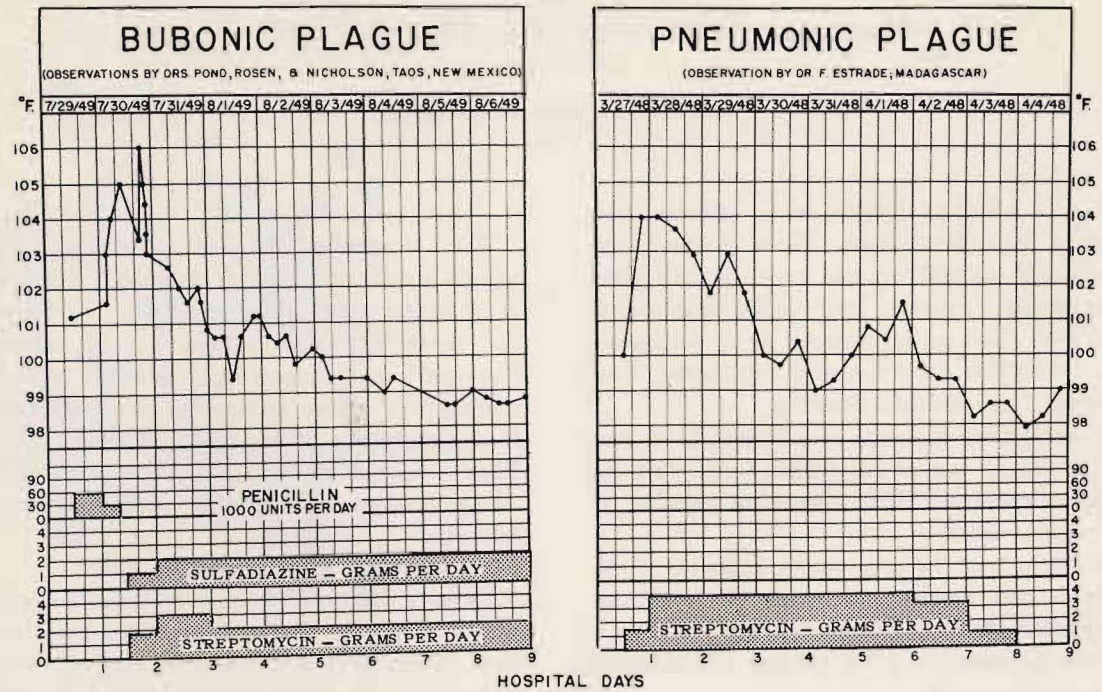



plague killed about two-thirds while pneumonic plague was almost invariably fatal. While many types of therapy have been tried, there were but two which were at all effective: serum with which to treat cases, and vaccines to prevent the disease. Serum was fairly effective if given early in large doses. Vaccines have been used on a large scale in various parts of the world, but are not 100 percent effective, and the period of protection is limited to several months. In the late thirties, sulfa drugs were found to be of value in the treatment as well as in the prophylaxis of plague, and sulfadiazine is probably the most efficacious. Streptomycin is an even better drug. A combination of streptomycin and sulfadiazine, if given early enough and in adequate doses, is capable of curing even pneumonic plague (figure 3).

The development of almost specific methods of treating plague has taken away some of the fears which were so justly warranted when this disease struck a community. However, there are still ample reasons why vigilance cannot be relaxed against plague at this time, merely because good methods of treatment now are possessed. Plague still demands respect because of the fact that in 15 of our Western States there is perhaps the largest focus of wild-rodent plague in the world and many species of wild rodents and ectoparasites are known to be involved in the broad spectrum of this disease. The vastness of the area involved makes it impractical even to think about the eradication of plague in these rodents even if one could forget the illuminating story connected with attempts to eradicate plague in the California ground squirrel.

While it appears biologically and economically impractical to eradicate wild-rodent plague, there is no reason why good preventive measures should be neglected. Experience with plague in this country has demonstrated that epidemics usually begin after domestic rodents have become infected and wild-rodent plague becomes important then for two reasons: first, it is a constant potential source for the infection of domestic rodents in urban communities which are located in the vicinity of plague foci; second, it will continue to cause isolated, single, widely scattered cases of human plague. The infection of domestic rodents can lead quickly to human epidemics. The

Figure 3




occurrence of human cases infected by wild rodents does not lead necessarily to epidemics unless the patient develops a secondary plague pneumonia and starts a pneumonic epidemic.

The single cases of human plague of wild-rodent origin are handled best by educating physicians to recognize the disease when it occurs and start treatment early. This enhances the possibility of recovery and prevents the development of pneumonic signs.

The prevention of the transmission of wild-rodent plague to domestic rodents can be accomplished best, not by efforts directed against wild rodent foci, but by programs of domestic-rodent control. By combating domestic rodents with every possible means, their numbers can be kept down to a point at which they will not be capable of supporting an epizootic of plague. There are effective means of eliminating such epizootics that may occur in domestic rodents: DDT, compound 1080, ratproofing, garbage disposal methods, and good community housekeeping methods should be able to wipe out a plague epizootic in short order should one chance to gain a foothold. However, an opportunity will be missed if efforts are not made to eliminate the possibility of such epizootics by establishing permanent domestic-rodent control programs in all Western cities which are anywhere near known plague foci.

The future program of the Western Communicable Disease Center Laboratory follows these general principles: (1) Surveillance over the eastward spread of plague; (2) Promotion of domestic rodent control programs; (3) Education of the medical profession in early recognition, early diagnosis, and early treatment methods; (4) Continuation of plague research.

At the present time, surveillance over the eastward spread of plague is being limited to the eastern boundary of the known infected territory; that is, to those States which lie between North Dakota and Texas inclusive. Domestic-rodent control is being promoted actively in Washington, Oregon, Idaho, Montana, Colorado, Utah, New Mexico, and Wyoming. It is hoped that the scope of this activity will be extended in the future until there is effective coverage in all States with known plague foci. The education of the public and of the medical profession is being promoted extensively so that knowledge of the epidemiology, diagnosis, and treatment of plague will become common knowledge. Studies on all phases of plague prevention and control will be continued in the hope that more effective methods will result.

Plague may have been one of mankind's worst enemies, but the knowledge and the tools now possessed should make it a far less formidable opponent in the future than it has been in the past.

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# The Plague Problem in the United States

CARL O. MOHR, Scientist (R)

Plague is a rodent disease which affects man, causing a high death rate. Three hundred and thirty-eight of the 520 reported cases in the United States through January 1950 have resulted in death. The disease is caused by a bacterium called *Pasturella pestis* and is contracted commonly from three distinct sources:

a. Directly from domestic rats (figure 1) or from the bite of one of their fleas (especially of the oriental rat flea, a species peculiar to domestic rats).
b. Directly from certain native rodents of the fields and woods or from the bites of their peculiar species of fleas.
c. From persons in whom plague has affected the respiratory tract and is thus transmissible by sputum droplets.

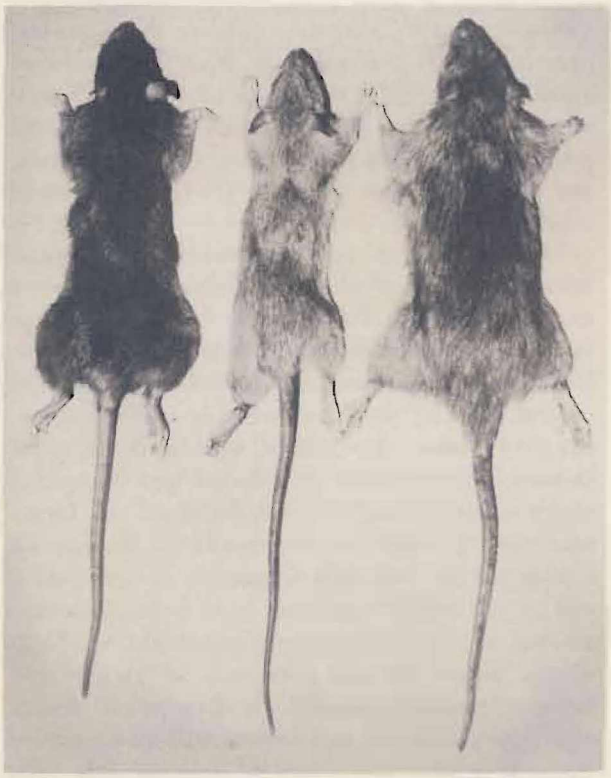


**Figure 1.** Domestic rats: Chief source of human cases of plague in the United States. From left to right: black Norway rat, brown climbing rat, brown Norway rat. Note that the climbing rat, although smaller, has a tail relatively longer than the Norway rats.

When contracted by way of sputum droplets, plague is referred to as "pneumonic." When present in domestic rats, it is referred to as "murine," and when present in native wild rodents, as "sylvan" or "campestral"* plague. It is transmitted from native rodents to domestic rats and from these rats to the native rodents by fleas. When acquired by human beings from murine or campestral sources, it may result in pneumonic infection.

**PROGRESS OF SPREAD OF PLAGUE**

Experts disagree to some extent concerning the origin of plague in North America. Some believe that, since it is a disease of similar rodents in Asia, it must have existed among native wild rodents in North America for many centuries. They point to the fact that, though investigators believe that plague drifted slowly eastward from a point of introduction in California about 1900, those investigators did not conduct their surveys in such a way as to prove their belief.

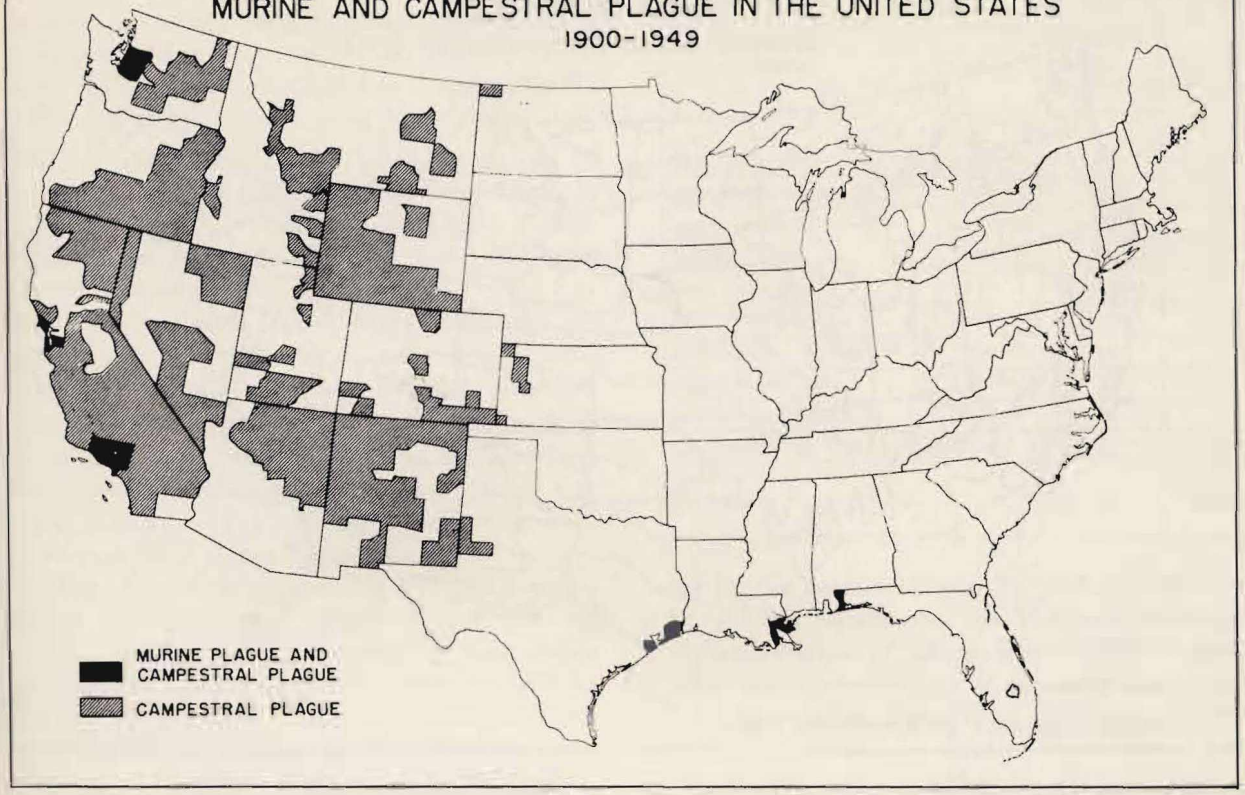
Others believe that plague was introduced from the Orient by way of infected rats brought in on ships. They point to the fact that it was so introduced into Hawaii and the Gulf Coast States, as well as into other parts of the world, and that it affects certain native rodents in a way which would be expected only of a disease completely new to them.

Whatever the case, plague among wild rodents now is distributed widely in the United States (figure 2). Various species of native wild rodents and their fleas are known to be infected. However, only a few of these rodent-flea combinations or "teams" are capable of supporting plague. Perhaps the best known of the native wild rodent-flea combinations capable of harboring plague are those of prairie dogs, California ground squirrels, and certain species of wood rats or pack rats. Sagebrush voles and certain species of meadow mice are probably equally important. In the Hawaiian

*The term "campestral" is probably more appropriate than "sylvan" inasmuch as rodents of the plains, prairies, and fields are more commonly infected than those of woods.

Figure 2

MURINE AND CAMPESTRAL PLAGUE IN THE UNITED STATES
1900-1949




Islands, a semidomestic species, the Hawaiian rat, and its peculiar fleas constitute a primary reservoir.

**CAMPESTRAL PLAGUE CONTROL**

Since plague was first recognized in 1900, only about eighty cases have been reported as having been acquired through handling of native wild rodents or bites of their fleas.

It is impractical to control plague among native wild rodents or their fleas sufficiently to prevent occasional cases, although dusting with DDT to control fleas and poisoning of wild rodents is feasible in limited, heavily used areas like school yards and parks.

Generally the best way to avoid infection is to avoid handling native wild rodents.

**MURINE PLAGUE CONTROL**

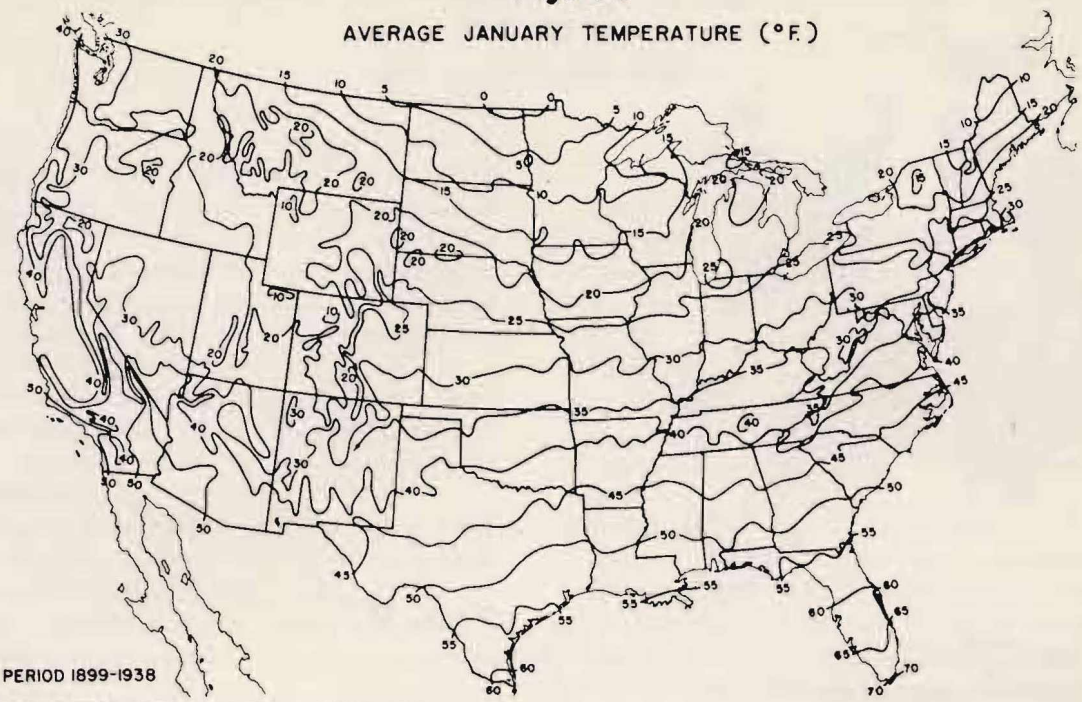
Over eight hundred human cases have been acquired from domestic rats, mainly through bites of the oriental rat fleas which parasitized them since plague first was recognized in the United States. About half of these occurred on the mainland and half in Hawaii.

The danger of acquiring plague from domestic rats depends partly on the degree of association of persons with rats, and partly on the size and distribution of oriental rat flea populations inhabiting these rodents. A general abundance in any given locality of at least one oriental rat flea per rat usually is regarded as the minimum required to support plague among domestic rats and thus to cause considerable danger to humans. In New Orleans, for example, the general midwinter level of oriental rat flea population was about one and a half per rat during the plague years. During the summer, the level was over four.

In the United States, no plague outbreaks or cases due to bites of the oriental rat flea have occurred where the midwinter temperature is lower than 45° F. (compare figure 2 with figure 3) or where the mean relative humidity at noon in July is less than about 60 percent (compare figure 2 with figure 4). Nevertheless, in climates where in summer the rat flea population exceeds one per rat despite a lower winter population, some danger conceivably can exist and must be guarded against. A study of oriental flea populations on domestic rats indicates that the danger may be considerable

**Figure 3**

AVERAGE JANUARY TEMPERATURE (°F.)

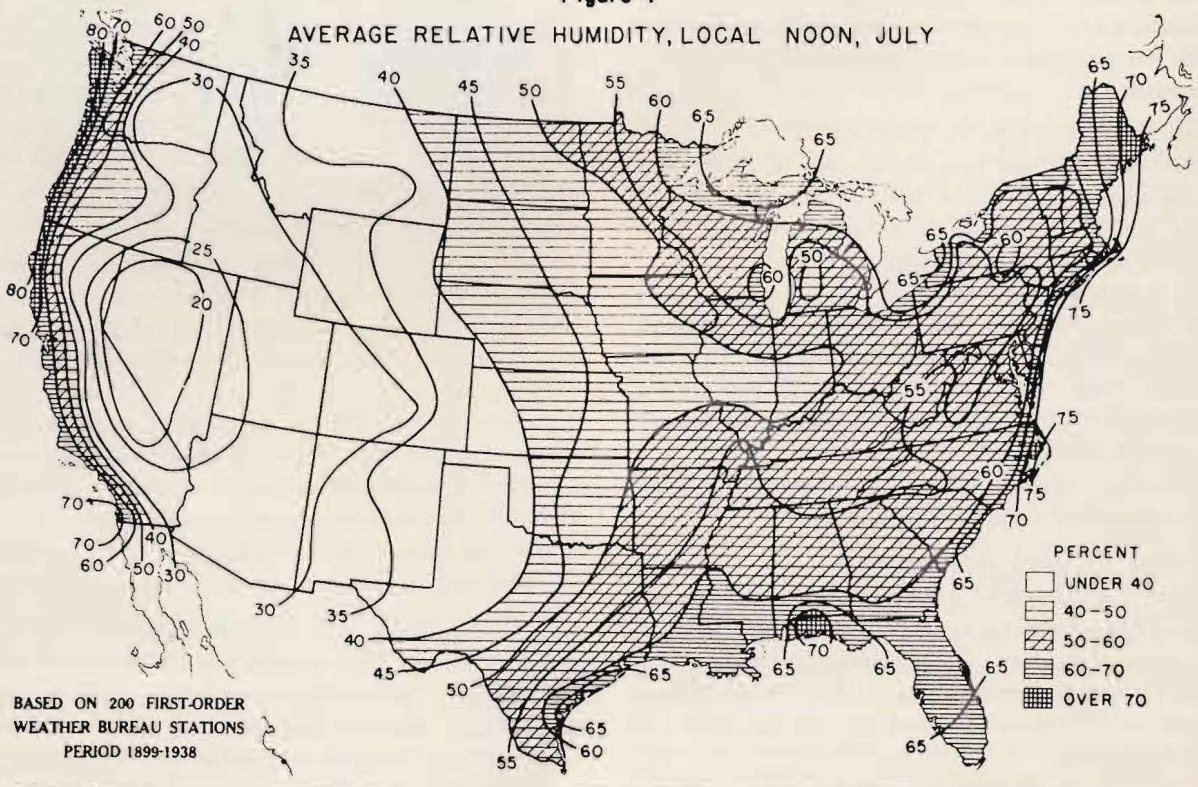


PERIOD 1899-1938

FROM: *Yearbook of Agriculture, 1941*

**Figure 4**

AVERAGE RELATIVE HUMIDITY, LOCAL NOON, JULY




BASED ON 200 FIRST-ORDER WEATHER BUREAU STATIONS
PERIOD 1899-1938

where the relative humidity is above 35 percent.

The flea population, and the danger as well, is greatest in moist, warm climates; rat-flea cases have occurred in Honolulu, T. H.; Seattle, Wash.; San Francisco, Berkeley, and Los Angeles, Calif.; Galveston and Beaumont, Tex.; New Orleans, La., and Pensacola, Fla., and in Puerto Rico (table 1). Infected domestic rats also have been found in Tacoma, Wash.; in Ventura, Marin, and Contra Costa Counties, Calif., and in Kaui and Maui Counties in Hawaii.

In Pacific Coast States, plague has been reduced to a minimum among domestic rats by anti-rat sanitation and poisoning. By such reduction of rat and rat-flea populations there, the possibility of infected rats being introduced to east-bound commerce is decreased. Nine Western States now have cooperative State-Federal programs to prevent local plague outbreaks.

There is now in progress in the Southeastern States a murine typhus control program with the purpose of keeping down domestic rat and rat-flea populations for the ultimate object of reducing the number of cases of murine typhus. This program involves application of DDT dust for control of fleas, back-lot and alley sanitation, ratproofing, food-establishment sanitation, and finally poisoning for the control of rats. By such reductions of rat and flea populations, the likelihood is reduced of implantation of plague from westward areas even should infected rats be transported eastward.

By keeping the domestic rat and rat-flea populations at a minimum locally, transmission from native wild rodents to human beings by way of domestic rat-fleas can be held to a minimum.

Table 1

HUMAN PLAGUE OUTBREAKS IN THE UNITED STATES

| City | Date | No. of Cases Reported | No. of Deaths |
|---|---|---|---|
| San Francisco, Calif. | 1900-04 | 120 | 114 |
| San Francisco, Calif. | 1907-08 | 186 | 92 |
| Seattle, Wash. | 1907-08 | 3 | 3 |
| New Orleans, La. | 1914-15 | 31 | 10 |
| Oakland, Calif. | 1919 | 13* | 13 |
| New Orleans, La. | 1919-21 | 25 | 11 |
| Pensacola, Fla. | 1920 | 10 | 4 |
| Galveston, Tex. | 1920 | 18 | 12 |
| Beaumont, Texas. | 1920 | 14 | 6 |
| Los Angeles, Calif. | 1924 | 41 | 34 |
| **Total** | | 461 | 299 |

# Human Diseases Harbored by Domestic Mice and Rats

**CARL O. MOHR, Scientist (R)**

Rodents and their ectoparasites are hosts of a number of disease-producing viruses, rickettsiae, and bacteria which affect man, generally in proportion to the degree to which the rodent hosts associate with man. Some of these disease organisms are transmitted directly by the rodent through contamination of man's food, water, and quarters through infected urine and feces, and some by biting. Many are transmitted from rodent to man by their ectoparasites. In some cases, rodents simply appear to be the host of an ectoparasite which is the reservoir of the disease.

Domestic rats and mice* bring ectoparasites and disease-producing organisms closer to man more constantly than do the rodents of the fields and woods, and therefore are responsible for a large number of such illnesses.

*Rattus norvegicus, Rattus rattus, and Mus musculus*

## FLEA-BORNE DISEASES

Plague and murine typhus are two of the best known diseases of domestic rodents transmissible to human beings, partly because of the high death rate caused by the first and because of the long illness induced by the second. House mice, although found infected, appear to be unimportant as hosts to these diseases.

Before adequate rat control measures were practiced in the United States and possessions, outbreaks of plague occurred in Seattle, Wash.; San Francisco, Oakland, and Los Angeles, Calif.; Galveston and Beaumont, Tex.; New Orleans, La.; Pensacola, Fla.; and Hawaii and Puerto Rico. The total number of reported cases in cities on the continent was 460 odd and the number of resulting deaths was 299. In Hawaii over 400 cases of plague were reported through 1949.

During 1945, the number of reported murine typhus cases totaled 5,179 on the mainland and 106 in Hawaii. Many cases probably were unrecognized.

The deaths and illnesses caused by these diseases have prompted extensive control measures which are responsible for the low incidence of these diseases today: fewer than a thousand cases of murine typhus and only one murine-based case of plague in Hawaii in 1949.

There are a number of other diseases, taking a less violent toll in sickness and death, that are not so well known, partly because rodents are not the sole reservoirs and partly because of the milder nature of the disease. In toto, however, they cause considerable illness.

High on the list of these is the group of alimentary-tract diseases of both domestic rats and mice. These include the feces-borne diarrheas, caused by various bacteria of the genus *Salmonella*; tapeworm infections; and the urine-borne leptospiral jaundice or Weil's disease.

## SALMONELLOSIS

*Salmonella enteritidis* and *typhi-murium** are two bacteria which occur commonly in mice in all parts of the world. No animal, according to Cameron (2) has more favorable opportunity of infecting human food with its droppings than the house mouse (figure 1). Numerous outbreaks of acute gastroenteritis (food poisoning) in human beings have been attributed to the contamination of food by mouse droppings.

*This bacterium also is known as *Salmonella aertycke*

**Figure 1.** House mouse (*Mus musculus*), bearer of Salmonella (food infection) organisms and lymphocytic choriomeningitis. — Courtesy Earnest P. Walker

It is, unfortunately, difficult to determine what percentage of cases are caused by contamination of food by rats and mice, since contamination of food is caused also by flies and unwashed hands and by flesh of unhealthy animals.

Edwards and co-workers (3) record *S. typhi-murium* and *S. enteritidis* as common in domestic rats and mice. *S. enteritidis* is the more common. It is also common among turkeys, from which humans also may acquire it.

While culturing flea feces to determine the presence of plague infection, Eskey (7) discovered that a number of rat fleas were excreting *S. enteritidis* which they apparently had acquired from blood streams of mice infested with this organism. Although other species of fleas had previously been infected by feeding them on *enteritidis*-infected mice, the laboratory workers had not been able to infect healthy mice with the infected fleas. Eskey and his co-workers were able to transmit *Salmonella* organisms by the bite of infected oriental rat fleas and Northern rat fleas. In one instance, transmission was obtained by feeding fleas on a mouse which itself was infected by the bites of infected fleas, demonstrating that the infection can be transmitted from mouse to mouse by fleas.

Although house mice in nature seldom have fleas in any great number, the same mode of transmission may occur among rats which do have great numbers in the milder climates with high humidities.

Ilajna (8) found *Salmonella* in cultures from 47 specimens of human blood and concluded that there was reason to believe that certain of these organisms may cause septicemia (blood poisoning). He found *S. typhi-murium* present in two "rats" and in 31 "mice," probably domestic species, but does not state how many of each of these he

examined. He also found *S. enteritidis* and *S. anatum* present in one "rat" each.

Eskey concluded that the two common fleas found on rats in the United States may play an important part in the dissemination of *S. enteritidis* among rodents and that human infection might be contracted directly from the bite of the fleas or from infected flea feces contaminating food.

Prevention of infection, according to Hull (12), consists in the use of meat products and eggs from healthy animals and birds and in the protection of food supplies from contamination by rats, mice, and flies. It is possible that bites of fleas also should be avoided.

Innumerable single cases due to transmission by mice and rats probably occur in the United States, since most food-handling establishments that have not been ratproofed are infested by one or both of these domestic rodents during a part of the year. The great majority are infested all of the year.

**LEPTOSPIRAL JAUNDICE**

Human infections of leptospiral jaundice are caused by contamination of food and water by urine of infected animals. This disease, also called Weil's disease, is reported as seemingly becoming more common in North America (2). Mice and roof rats apparently are not as much infected as are Norway rats, which commonly bear the organism. Of 197 Norway rats examined from various districts in Honolulu, 9 were found infected, the spirochaetes being present in large numbers in the kidneys and excreted in the urine (1). Studies in various parts of the world have indicated that about 10 percent of domestic rats harbor the causative organism (12) but that adult rats living in sewers have been found 45 percent infected. In Chicago, to cite a summary by Hull, from 3 to 52 percent of the rats examined were found infected; in New York, N. Y., from 17 to 22 percent; in Nashville, Tenn., 10 percent; in Albany, N. Y., 40 percent; in Baltimore, Md., 7 percent; in Washington, D. C., 24 percent; in San Francisco, Calif., 33 percent; in Rochester, N. Y., 38 percent; and in Detroit, Mich., 16 percent. Although these percentages vary greatly due to location and time of year and with variable sizes of samples, they do show widespread and rather high rates of infection. Persons working in wet or damp premises are infected most commonly. Of the 73 cases tabulated by Hull, there were 8 sewer workers, 6 fish cutters, 10 swimmers, 3 abattoir workers, 2 eating-place operators, 1 butcher, 1 salesman in a meat and vegetable market, and 1 who lived in a rat-infested home.

**AMEBIASIS AND TAPEWORM INFECTIONS**

The several animal parasites that affect rats are not of great epidemiological importance (12). Food contaminated by rat droppings nevertheless may contain eggs of the rat tapeworms and dwarf tapeworms.

**LYMPHOCYTIC CHORIOMENINGITIS**

In five of six homes of patients ill due to lymphocytic choriomeningitis, investigated by Dr. Armstrong of the Public Health Service, according to Hull (12), house mice also were found infected, suggesting that possibly the patients had acquired the disease from the mice. In the sixth home, only two mice, both uninfected, were found, but the home previously had been overrun by mice.

Suspecting that house mice might be primary reservoirs, one worker, according to Hull, obtained 369 of these mice from 78 scattered homes in Washington, D. C., in order to determine to what degree they were infected. One mouse of every five examined was a carrier of the choriomeningitis virus. At least 45 percent of the homes in which the mice were taken had infected mice. In Boston, other workers found only 8 percent of 108 mice were carriers.

Other less extensive studies also have shown that the disease is most common among persons who have had direct and indirect contact with house mice prior to illness (12).

According to a memorandum from the CDC Epidemiologic Services, lymphocytic choriomeningitis is not a nationally notifiable disease, i. e., the Division of Public Health Methods and the National Office of Vital Statistics do not request the States to submit figures on the incidence of this disease. However, some States in some years have transmitted such figures to Washington, and they have been included in footnotes to either the quarterly or annual provisional summaries of incidence of notifiable diseases. In the final summary of incidence of notifiable diseases, published annually in a supplement to Public Health Reports, figures on lymphocytic choriomeningitis have been omitted. Table 1 shows the figures submitted by those States which gather data on the disease and which chose to transmit them to Washington.

Although the number of reported cases in the

Table 1

REPORTED INCIDENCE - LYMPHOCYTIC CHORIOMENINGITIS

| States | 1949* | 1948** | 1947** | 1946** | 1945** | Total |
|---|---|---|---|---|---|---|
| Ill. | - | - | - | - | 2 | 2 |
| Ind. | 2 | 1 | - | - | - | 3 |
| Iowa | 1 | - | - | - | - | 1 |
| Maine | - | 1 | - | - | - | 1 |
| Md. | - | - | - | - | 6 | 6 |
| Mass. | 2 | 19 | 6 | 4 | 4 | 35 |
| Minn. | 5 | 1 | 5 | - | 2 | 13 |
| Mont. | 1 | - | - | - | - | 1 |
| R. I. | - | 5 | - | - | - | 5 |
| Tenn.+ | 18 | 11 | 13 | 21 | 31 | 94 |
| Utah | - | - | - | - | 1 | 1 |
| TOTAL | 29 | 38 | 24 | 25 | 46 | 162 |

* Source of data: Quarterly summaries of notifiable diseases, Public Health Reports.

** Source of data: Preliminary annual summaries of notifiable diseases, Public Health Reports.

+ Includes choriomeningitis undefined.

United States is small, the desirability of keeping premises mouse-free is indicated.

Although infection via the respiratory tract is suggested by early symptoms, other possible routes of infection may be contaminated food or contamination through the skin or membranes. Present recommended means of prevention are construction of homes with a view to making them mouseproof and reduction or elimination of mouse infestation in quarters frequented by man.

**HISTOPLASMOSIS**

A fungus organism called *Histoplasma capsulatum* also has been found in Norway rats in Loundoun County, Va., (4,5,6) and in southwest Georgia (6). Although this disease affects human beings, the significance of the rat in the epidemiology of human cases is not yet apparent, according to Emmons. The disease is so poorly known in human beings that it is difficult to estimate the number of cases occurring annually. It is possible that rats and human beings both may acquire infection from some common source or that the rat, being closely associated with human beings, may heighten the chances of human infection by disseminating the infecting bodies of the *Histoplasma* organism. Dogs, however, also bear the disease organism.

**RICKETTSIALPOX**

During July 1946, a peculiar disease, later found to be a mouse disease transmitted by house-mouse mites (*Allodermanyssus sanguineus*) occurred in a middle-class housing development in New York City. An investigation of 80 cases during the succeeding 10 weeks showed that the disease resembled chickenpox and that it is mild (9,10,11). This disease is known only from New York City but the house-mouse mite is known to be distributed spottily over the United States, having been found in Salt Lake City, Utah; Tucson, Ariz.; Urbana, Ill.; Philadephia, Pa.; Indianapolis, Ind.; Boston, Mass.; and Washington, D. C. (15).

The mite is troublesome as a biter even though it may not be infected. It has been found on both domestic rats and mice, but it appears to be primarily a house-mouse parasite.

A related species *(Liponyssus bacoti)*, the tropical rat mite, is also a troublesome biter when it becomes common. It has been found capable of transmitting rickettsialpox from mouse to mouse in the laboratory (14), but appears to be a poor vector and is not known to transmit rickettsialpox other than under ideal laboratory conditions. It is much more common on domestic rats than on domestic mice. Apparently, this mite is not important in transmitting murine typhus from rat to rat or from rat to man. Ilowever, it causes most severe dermatitis when it bites.

**RAT-BITE FEVER AND HAVERHILL FEVER**

Occasionally the bites of rats are followed by a fever, due either to infection caused by a *Spirillum (minus)* or a *Bacillus (Streptobacillus moniliformis)*. Recently, a case of rat-bite fever in Montana was noted under circumstances indicating that house mice were responsible (13). A child, bitten by a house mouse, became ill with the disease in an area where she had no contact with rats. A high percentage of the house mice in the buildings in which she was bitten were infected with the spirilla.

The bites themselves are, of course, painful, particularly those of rats. In poor housing sections of cities, children and helpless adults frequently are bitten severly and sometimes killed.

**SUMMARY**

Domestic rats and mice are known to harbor a considerable number of disease-producing organisms or infected ectoparasites, bringing them into the homes of man. Few studies have been made to determine what percentages of such diseases as salmonellosis, lymphocytic choriomeningitis, toxoplasmosis, and others are transmitted by domestic rats and mice; but the fact that they do transmit them, along with the known vectorship of plague and murine typhus by rat fleas, adds up to the fact that these rodents are too dangerous to be allowed in the homes of man.

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# Methods Used in Plague Transmission Studies

**VERNON B. LINK, Senior Surgeon, and FRANK M. PRINCE, Medical Entomologist***

In addition to its field work on plague, the Western Communicable Disease Center Laboratory carries on plague research activities. One of the more interesting types of research is concerned with transmission studies in which the vector efficiency of fleas is studied under varying conditions.

Ogata in 1897 (1) brought forth the theory that fleas were involved in the spread of plague among rats. Simond in 1898 (2) supported this theory experimentally, but assumed that flea feces were responsible for the transfer of the organism from

* Western Communicable Disease Center Laboratory, San Francisco, Calif.

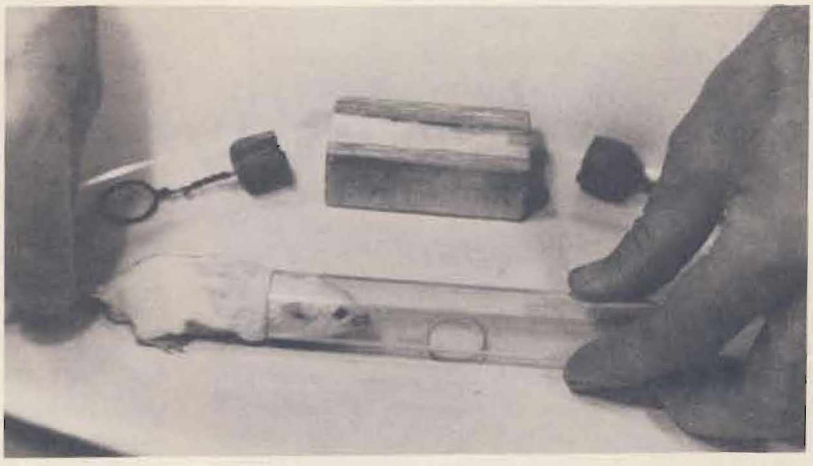
diseased to healthy rats. The Commission for the Investigation of Plague in India, working from 1905 to 1916 (3), proved beyond a doubt that the rat and its fleas were the common reservoir and vector in plague epidemics. However, they did attach undue importance to the role played by deposition of flea feces during the process of biting and subsequent rubbing into the skin by the victims' scratching efforts. Bacot and Martin in 1914 (4) demonstrated the ability of infected fleas to transmit *Pasteurella pestis* by feeding them on the shaved abdomens of rats. They first observed the "blockage" of fleas by masses of plague organisms.

In flea transmission research at this laboratory, unique methods have been developed for the study of the flea in determining its efficiency as a vector of plague. Laboratory albino mice, used as the host animal, are bred in the animal house in quantities sufficient to provide several thousand every winter. After the young mice have reached an age of approximately 4 weeks, they are separated according to sex and placed in stock cages. When mice are used in experiments, they are transferred to an individual quart jar which is provided with sawdust, drinking water, food pellets, and an identification record.

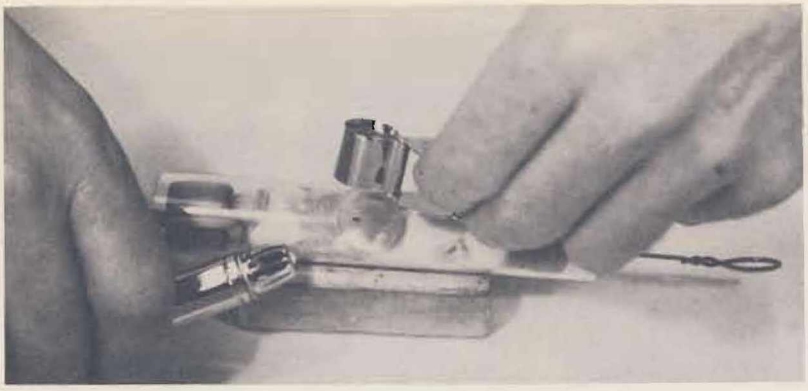
Fleas to be used in experiments either are raised in the laboratory or are obtained in the field from wild rodent sources, brought to the laboratory, and placed in numbered test tubes, one flea to each tube. Fleas subsist on animal blood and exhibit a peculiar preference for blood from a particular host. This result of host adaptation is so specific that the entomologist usually can tell from what host the flea was taken when the flea is identified. Certain fleas will feed on almost any available warm-blooded host, whereas others have been known to starve to death before they would feed on man or laboratory animals.

In the flea transmission studies, fleas first are given an opportunity to feed on a normal albino mouse. The abdomen of the mouse is shaved with electric clippers. The mouse then is placed in a white enamel pan and induced to enter a plastic tube which has a hole cut in the center. When the mouse is in the tube with his shaven abdomen over the hole, a rubber stopper is placed in each end of the tube to keep the mouse immobilized. The tube then is placed upside down on a grooved wooden block with the mouse's abdomen exposed

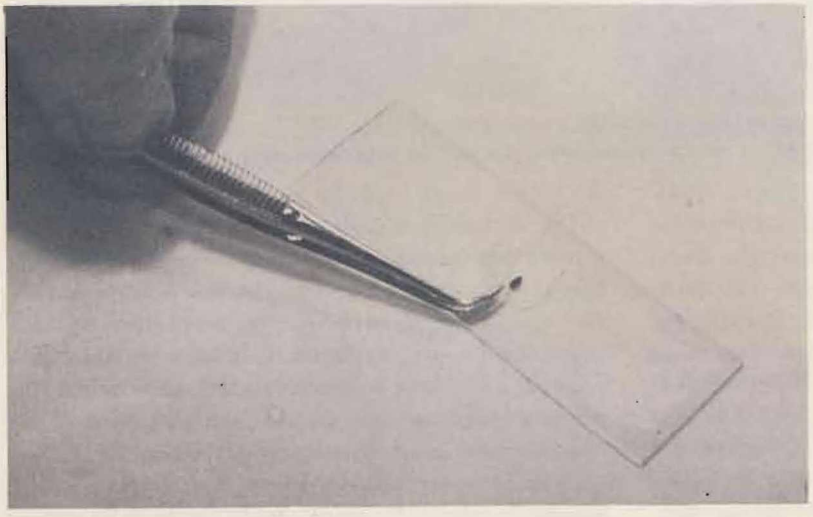
Mouse being shaved.



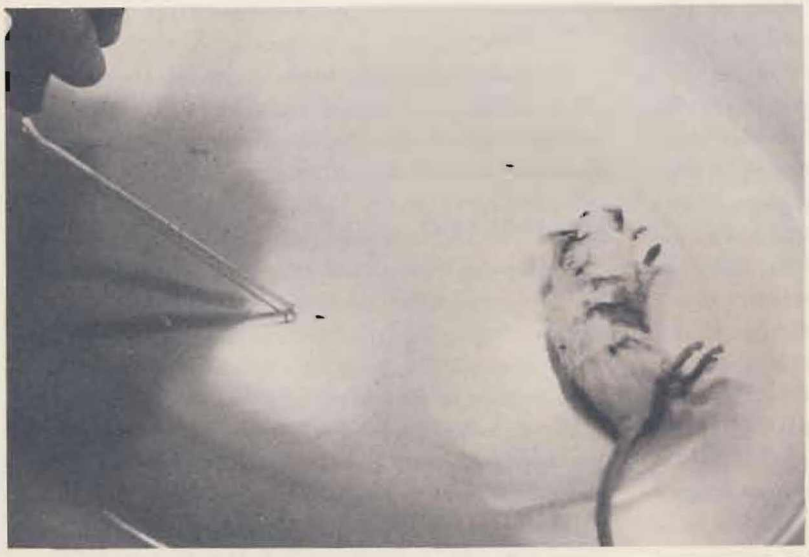
Mouse entering plastic tube.



Placing flea on mouse in stoppered tube.



Flea on slide.



Fleas being removed from jar.

through the hole in the tube. The flea then is taken from its test tube by removing the cotton plug and up-ending the tube onto the mouse. The flea ordinarily will settle down and feed on the mouse. By the use of a hand lens and a pocket flashlight. the attendant can see whether the feeding flea is taking blood into his stomach. The record of details of the feeding are kept on a card prepared for each individual flea. After fleas have fed, selected specimens are removed from the mouse, placed in a drop of water on a microscopic slide, and covered with a cover slip. The flea then can be examined under the microscope to check on the quantity of blood taken and for the purpose of making serial photographs.

Albino mice to be used as reservoir hosts for the infection of fleas, are inoculated by the subcutaneous injection of 0.1 cubic centimeter of a 24-hour tryptose broth culture of *P. pestis*. In 24 to 48 hours after injection, most of the mice will be moribund and have a terminal septicemia. This is checked by snipping off the tip of the tail and making a blood smear which is stained and examined for organisms. Frequently, there are as many organisms as red blood cells. If the smear shows 10 or more bacilli per oil-immersion field, the mouse is satisfactory for use as a host. Fleas are placed *en masse* on that mouse in a jar. After they have fed, the fleas are removed from the jar containing the mouse by means of a suction tube and placed in their individual test tubes.

On succeeding days, each flea's feces are cultured in order to determine whether or not the flea

Sanitation, had autopsied four of the rats found dead, made smears of the enlarged glands and spleens, and found organisms identical to those seen on the slides from the human buboes. Animal inoculations had been made with both human and rodent material for final confirmation of the diagnosis.

This presumptive confirmation of the diagnosis of human and rodent plague was sufficient to warrant the full implementation of "Operations Pest" and a telephone call was made to the Western CDC Laboratory to carry out the other procedures of the plan.

**PROCEDURE NO. 2**

**Mobilizing Manpower and Equipment.** During the afternoon of May 18 the staff of the Western CDC Laboratory had been getting together the equipment which would be taken to Bay City, and estimating the supplies and manpower necessary to dust the entire city, set up a laboratory, initiate rat trapping, carry on a poisoning program, inaugurate permanent ratproofing, and institute antirat sanitation. A liberal estimate of 50,000 buildings in this city of 150,000 inhabitants (including 40,000 residences and 10,000 business establishments) was used as a basis for calculating the quantities of equipment, supplies, and manpower to be required. Based on the antityphus program in Southern States, it was estimated that an average of 0.5 man-hours would be required to treat each building with 3.5 pounds of 10 percent DDT dust. It was calculated that 448 men divided into 14 groups of 32 men each, under the direction of 14 supervisors, could dust the entire city with 175,000 pounds of DDT in 1 week. These crews would need two trucks each (28 trucks with drivers) to transport men, equipment, and DDT.

A telephone call to a wholesale supplier in Utopia's largest city resulted in a promise of immediate shipment of one carload (40,000 pounds) of dust which could be expected on the afternoon of May 19. Additional carloads would be forwarded until the fifth and last was delivered in Bay City not later than May 21.

It was decided that since large numbers of dusting cans could not be obtained on short notice, DDT dust would be distributed by hand, each duster carrying a 10-quart bucket of dust. A telephone call to a factory in Utopia resulted in the acquisition of 500 galvanized pails which were to be delivered in Bay City by truck on the afternoon of May 19.

The Medical Officer then called the Commanding Officer of nearby Fort Manson, explained the epidemic situation, and made arrangements for the Army to provide 28 trucks, with drivers, and the services of 448 enlisted men for a period of 7 days in order to dust Bay City with DDT. The Commanding Officer agreed to have the detail in Bay City at 8 a.m. on the morning of May 20.

The following personnel of the Western CDC Laboratory were flown by an Air Forces plane to Bay City on the evening of May 18, together with equipment to set up a plague laboratory: medical entomologist, assistant medical entomologist, medical bacteriologist, medical technician, sanitary engineer, and administrative officer.

In addition to the above, 14 Western CDC rodent control specialists on duty in nine Western States were ordered by telegram to fly to Bay City on May 19. These men, especially trained in domestic rodent control methods, were to carry out direct supervision of the 14 dusting crews.

Finally, telegrams were sent to the four Western CDC mobile laboratory units conducting plague surveys of wild rodents in Oklahoma and Kansas. The eight men were instructed to depart for Bay City on May 19 with their trucks and equipment and to arrive on May 22. These experienced hunters and trappers of both domestic and wild rodents were to be used to supplement the regular Bay City Department of Health rat trapping force.

**PROCEDURE NO. 3**

**Informing the Public.** At 10 p.m. on May 18, the Utopia State Health Officer called in representatives of the local newspapers and the national news associations for a press conference. He stated that he had requested epidemic aid from the Public Health Service because of the occurrence of six cases of suspected human plague. He said that a medical officer of the Western CDC Laboratory had flown to Bay City and accompanied him and the Bay City Health Officer to see the six patients. The tentative clinical diagnosis now had been confirmed. In addition, a presumptive diagnosis of rodent plague had been confirmed on four rats found dead in the neighborhood where the six patients resided. As a result of these confirmations, "Operations Pest," the Public Health Service's emergency antiplague program had been put into effect to assist the health authorities of Bay City and the State of Utopia. He told the reporters that the entire city was to be dusted with DDT starting the morning of May 20, for which the Army generously had provided the manpower and trucks

required. He stated that about 30 additional Public Health Service experts had been ordered to proceed to Bay City at once to assist in supervising the operations. About 20 of these would arrive by plane by noon of May 19 and the remainder would arrive a few days later, because they were bringing field-laboratory trucks. He said that during the next few days full-scale trapping and poisoning activities would be instituted, and eventually the present permanent ratproofing program would be expanded. He said bubonic plague caused swollen glands in the armpit and the groin and occasionally plague pneumonia. All persons with such swollen glands should report to a physician immediately. Physicians should suspect any pneumonia until it was proven not to be plague. Since fleas spread bubonic plague, the city was being dusted with DDT to kill any infected fleas and prevent more persons from becoming infected. After the flea populations were reduced, the rats would be eliminated wherever possible by trapping and poisoning. He said that the public should not become unduly alarmed, that more cases probably would occur before control measures were effective, but that modern treatment of plague was excellent. The six known patients already were being treated with sulfadiazine and streptomycin, and all had good chances for recovery. He expressed a direct appeal to the public for cooperation with the antiplague forces, especially in the dusting operations. Finally, he stated he was appointing the CDC Medical Officer as his representative in charge of all operations.

**PROCEDURE NO. 4**

**Acquisition of Quarters for Offices, Laboratory, and Operations.** By the time that the five other persons arrived from San Francisco, arrangements had been made for office and laboratory space in the Bay City Department of Health and for the temporary use of a large, privately owned warehouse to function as a base of operations, storage of supplies, and garaging vehicles.

**PROCEDURE NO. 5**

**Establishment of the Plague Field Laboratory.** A laboratory for entomological and bacteriological examinations was set up May 19 in the space provided at the Bay City Department of Health.

The entomological activities were to include identification and classification of all ectoparasites removed from the rats obtained; determination of flea indices (both the percentage of rats with fleas and the average number of fleas per rat) after dusting operations. Indices before dusting were to be obtained from the previous Bay City Department of Health rat trapping records. These activities were to carried out by the CDC entomologists.

The bacteriological activities were to consist of confirmation of the diagnosis of human and rodent plague. Rats were to be combed free of all ectoparasites which would be identified and then inoculated into guinea pigs. All rats then would be autopsied for gross and microscopic examination of tissues. These activities were to be carried out by the CDC medical bacteriologist and technician.

**PROCEDURE NO. 6**

**Organization of the Dusting Program.** Bay City already was divided into three districts totaling 28 sections for purposes of rodent control. These arbitrary divisions were utilized by assigning one crew of 32 men, two trucks with drivers, and one supervisor to each two sections. Large maps were made of each section, showing block outlines, and were distributed to each supervisor. Each morning the crews were to assemble at the warehouse, travel to their respective sections on the trucks, and proceed with the dusting operations. Each man was to carry a 10-quart pail of DDT which was to be refilled at the truck whenever necessary. DDT was to be sprinkled liberally by hand over the floor space of every building. In selected buildings with evidence of rat runs, DDT was to be distributed freely wherever rats might be expected to travel. Each supervisor was to be required to keep an accurate record of all buildings dusted by recording the address in the corresponding block on his section map. At the end of each day, this information was to be turned in at headquarters and recorded on the master map of the city. All dusting operations were to be under the direction of a CDC sanitary engineer.

**PROCEDURE NO. 7**

**Organization of Trapping Activities.** The regular trappers of the Bay City Department of Health were to continue trapping all sections of the city as intensively as possible. Their efforts were to by supplemented by the eight Public Health Service trappers who not only would help trap in the city, but would make a sample survey later of the wild rodents in the adjacent rural areas surrounding Bay City. The rat trapping was to be under the

direction of the Bay City Department of Health's Sanitary Engineer.

**PROCEDURE NO. 8**

**Organization of Poisoning Campaign.** The 10 pest control companies in Bay City were asked to provide seven men each to be used for a period of 1 week in a mass rat poisoning campaign. These experienced men were to work under the direction of the crew supervisors in distributing poison after DDT dusting had been completed in each block. Red squill was the poison of choice for the residential areas, while compound 1080 was to be used throughout the business districts and wherever the hazard to human beings could be carefully controlled.

**PROCEDURE NO. 9**

**Organization of Antirat Sanitation Activities.** Suggestions were made to the Department of Public Works for improvement of garbage collection methods. Garbage collections were increased from once a week in the residential areas to twice a week. In the downtown business district, collections were increased from three times weekly to once a day.

The Department of Public Works also took over the responsibility of organizing a demolition squad which was available upon call from the section supervisors to tear down and destroy any suspected outside rat harborage. However, many rat harborages were removed by property owners themselves following suggestions made by the section supervisors.

**PROCEDURE NO. 10**

**Maritime and Railroad Control Measures.** The Foreign Quarantine Division of the Public Health Service was requested to assign an officer and crew for the purpose of HCN (hydrocyanic gas) fumigating and dusting DDT on every ship which sailed from Bay City. One experienced rodent-control operator was assigned to work with the railroad and trucking companies. He assisted in inspection of railroad cars and trucks leaving the city and, with crews provided by the various companies concerned, directed the DDT dusting of all warehouses, freight depots, and departing cars and trucks.

**PROGRESS OF THE EPIDEMIC**

On May 19, six more cases of bubonic plague were reported. Three of these were cases which had read the newspaper accounts that morning and had gone to physicians because of swollen glands in the armpit or groin.

On succeeding days the following numbers of cases tentatively were diagnosed: May 20, 8; May 21, 10; May 22, 6; May 23, 12; May 24, 10; May 25, 11; May 26, 7; May 27, 15; May 28, 9; May 29, 7; May 30, 1. One of the cases reported on May 25 prove to be pneumonic plague. At first, it was thought that the patient had primary pneumonic plague because no buboes were evident. At autopsy, it was discovered that he had enlargement of the sacral and iliac lymph glands which could not be felt clinically but which had led to a fatal secondary plague pneumonia. Because the man was not seriously ill at first, he had not been hospitalized for the pneumonia. He eventually transmitted pneumonic plague to three other members of his family, to two visitors, and to his family physician. These six secondary cases accounted for nearly half of the number of cases reported on May 27. This unrecognized bubonic plague case was the only fatality of the epidemic. No cases were reported after May 30. Because the majority of cases were bubonic plague transmitted by fleas, the blanketing of the city with DDT eventually created a situation where no fleas were left to transmit the disease.

The DDT dusting of the city proceeded according to plan. A total of 171,410 pounds of 10 percent DDT in pyrophyllite was distributed in 48,964 buildings in 6½ days. The following figures show the buildings dusted by days:

| Days | Buildings | Lb. DDT |
|---|---|---|
| May 20 | 6,241 | 21,943 |
| May 21 | 7,350 | 25,725 |
| May 22 | 7,214 | 25,249 |
| May 23 | 8,872 | 31,052 |
| May 24 | 8,540 | 29,890 |
| May 25 | 8,312 | 29,029 |
| May 26 | 2,435 | 8,522 |
| **Total** | 48,964 | 171,410 |

An additional 9,332 pounds of DDT were used during the period from May 20 to June 19 inclusive in dusting DDT in outgoing ships, railroad cars, and freight trucks.

**TRAPPING RESULTS AND FLEA INDICES**

From the records of the Bay City Department of Health, previous flea indices for May and June

showed an average of 3.2 and 4.6 respectively per rat. In previous years, rats had been about 83 percent Norway rats (*Rattus norvegicus*), and 17 percent climbing rats as follows: 11 percent *Rattus rattus rattus*, and 6 percent *Rattus rattus alexandrinus*. These proportions remained about the same during the control campaign. The flea populations usually had been about 95 percent Northern rat fleas (*Nosopsyllus fasciatus*); less than 1 percent were oriental rat fleas (*Xenopsylla cheopis*). However, during the 6 months prior to the epidemic, a consistent rise in *X. cheopis* had been noted until it represented about 25 percent of all fleas recovered. During and immediately following the epidemic, *X. cheopis* predominated in the area where most of the cases were reported. Bay City, therefore, possessed two flea species known to be good vectors of plague, with the most efficient vectors of plague known predominating in the infected area.

Rats had been trapped in Bay City at a rate of about 10 per day prior to the epidemic. This work was expanded so that between May 19 and June 19 inclusive, the numbers in table 1 were obtained.

The DDT dusting exhibited a marked effect on reduction of the numbers of fleas per rat and eventually was responsible for the dramatic reduction of positive rats found as well as in numbers of positive flea pools. These reductions roughly paralleled the reductions in human cases.

The big increase in rats obtained starting on May 23 represented the additional trapping efforts of the eight Service trappers who arrived on May 22, plus the results of the poisoning campaign which was begun on May 21. In addition to the rats listed above, more than 300 mice were obtained, none of which were plague-infected.

During the last week of antiplague activities, from June 12 to 16 inclusive, the four Service mobile field crews made a survey of the rural areas around Bay City and shot or trapped 342 wild animals, most of which were rodents. Of these, 13 ground squirrels (*Citellus columbianus*), 12 white-footed deer mice (*Peromyscus maniculatus*), and 4 chipmunks (*Eutamias minimus*) were found to be plague-infected or harbor plague-infected fleas. The presence of plague in this rural area indicates that the disease in the domestic rodents of Bay City might have originated from

**Table 1**

**RATS TRAPPED AT BAY CITY**

| Date | Rats Obtained | Rats Found Dead | Flea Index (Av. Per Rat) | Plague-positive Tissue | Plague-positive Flea Pools* |
|---|---|---|---|---|---|
| May 19 | 15 | 6 | 5.2 | 7 | 4 |
| 20 | 20 | 7 | 4.9 | 8 | 12 |
| 21 | 22 | 8 | 6.4 | 10 | 14 |
| 22 | 24 | 4 | 5.7 | 6 | 15 |
| 23 | 102 | 3 | 5.6 | 5 | 20 |
| 24 | 100 | 2 | 5.4 | 4 | 16 |
| 25 | 118 | 1 | 4.6 | 2 | 12 |
| 26 | 115 | 0 | 2.2 | 0 | 10 |
| 27 | 119 | 0 | 1.4 | 0 | 8 |
| 28 | 48 | 0 | 0.6 | 0 | 6 |
| 29 | 57 | 0 | 0.3 | 0 | 2 |
| 30 | 63 | 0 | 0.2 | 0 | 0 |
| 31 | 42 | 0 | 0.1 | 0 | 0 |
| June 1 | 51 | 0 | 0.1 | 0 | 0 |
| 2 | 59 | 0 | 0.1 | 0 | 0 |
| 3 | 54 | 0 | 0.0 | 0 | 0 |
| 4 | 52 | 0 | 0.1 | 0 | 0 |
| 5 | 47 | 0 | 0.0 | 0 | 0 |
| 6 | 41 | 0 | 0.1 | 0 | 0 |
| 7 | 48 | 0 | 0.0 | 0 | 0 |
| 8 | 39 | 0 | 0.0 | 0 | 0 |
| 9 | 42 | 0 | 0.0 | 0 | 0 |
| 10 | 32 | 0 | 0.0 | 0 | 0 |
| 11 | 29 | 0 | 0.0 | 0 | 0 |
| 12 | 31 | 0 | 0.0 | 0 | 0 |
| 13 | 25 | 0 | 0.0 | 0 | 0 |
| 14 | 27 | 0 | 0.0 | 0 | 0 |
| 15 | 26 | 0 | 0.0 | 0 | 0 |
| 16 | 20 | 0 | 0.0 | 0 | 0 |
| 17 | 19 | 0 | 0.0 | 0 | 0 |
| 18 | 18 | 0 | 0.0 | 0 | 0 |
| 19 | 12 | 0 | 0.0 | 0 | 0 |
| **Total** | 1,517 | 31 | | 42 | 119 |

*Fleas from individual rats were inoculated separately.

the wild rodents in the territory around the city.

On June 20, Bay City was declared to be free of plague and all Public Health Service activities were completed, leaving the long-range ratproofing program to be carried on by local effort.

**SUMMARY**

A plague epidemic of 109 human cases has been described. Of these, 102 were bubonic, 1 was bubonic with a secondary plague pneumonia, and 6 were pneumonic. All but the bubo-pneumonic case survived because of early diagnosis and prompt treatment with streptomycin and sulfadiazine.

Antiplague efforts consisted in the use of 10 percent DDT dust in pyrophyllite as a city-wide application to all buildings, trapping, poisoning, and antirat sanitation measures. All outgoing ships were fumigated with HCN, and all outgoing ships, railroad cars, and trucks were dusted with DDT. The number of human cases, rats found dead, positive rodents, and the flea index all showed a dramatic drop beginning about the time that the DDT city-wide dusting was completed.

**CONCLUSION**

Modern antiplague control measures have demonstrated that a plague epidemic can be terminated in short order. Modern methods of treatment were effective in saving the lives of over 99 percent of those who were afflicted with the disease.

* * *

*(This purely fictional account of how a plague epidemic would be handled today represents the planning efforts of the Western CDC Laboratory in anticipation that the above situation may occur in the future in some Western city.)*


# Rat-borne Disease Prevention and Control

*Communicable Disease Center, Public Health Service, Federal Security Agency, February 1949, pp. 293 + xiv.*

This manual, a work which has been needed for some time, appears to have accomplished two objectives. The first of these is the bringing together in one volume the wealth of information on rat-borne disease prevention and control accumulated over the years by public health workers. The second is the presentation of this information in such a way that it could be used readily for training of public health workers in this field. The manual is not the work of any one individual but is the cooperative effort of a number of experts and illustrates the desirability of this method of approach in dealing with the controversial aspects of the subject. Also, before publication the manual was submitted for review to prominent authorities including members of Federal and State agencies, as well as to certain interested representatives of national commercial organizations, and constructive suggestions from these sources were integrated into the text.

A most practical and logical approach has been made in the presentation of the subject matter with a view toward making it as useful as possible to health workers. The manual is divided into eight integral parts or sections, each of which is written so that it may be studied independently;

however, various sections may be combined as necessary for training purposes. This has resulted in some repetition of material which, however, is necessary in a manual of this type.

The value of the manual is enhanced greatly by the illustrative material which appears on almost every page and includes photographs, line drawings, charts, maps, tables, diagrams, and pictorial identification keys. The antics of "Roscoe, the Rat-Ridder," help break the monotony of scientific presentation often common to such manuals; yet, at the same time, they emphasize visually to the reader certain important principles which otherwise might be missed. Well selected references are found at the end of each section for those individuals who may have the need or opportunity to delve more deeply into each subject.

The first two sections of the manual present basic biological information. In the first section the epidemiology of rat-borne diseases is discussed. Murine typhus fever and plague are covered in detail as to etiology, diagnostic procedures, treatment, source of infection, mode of transmission, immunization, prevalence, and distribution. Those diseases of lesser importance – salmonellosis, hemorrhagic jaundice or Weil's disease, rat-bite fever, and trichinosis – also are given due consideration. Following this is a section on rat habits and characteristics which is especially inclusive. Both species of rats, the Norway and roof rat, are compared as to specific identification and as to distribution; habits such as food preferences, mating, burrowing, climbing, and migration; and characteristics such as life cycle, reproduction, behavior patterns, senses, and agility. This section is concluded with a discussion of the factors which influence the degree of rat infestation; the principal factor emphasized is the availability of food.

Section III covers the principal steps to be taken in the organization of local programs on rat-borne disease control. The first step consists of determining disease prevalence and distribution. The relationship of the program to other health activities is pointed out. The size and type of program, whether it is aimed at rat or ectoparasite control, or both, training of personnel, public relations, and a recommended rat-control ordinance are the chief subjects covered in this section.

Sections IV through VII are concerned primarily with control measures aimed directly at the rat. The importance of sanitation to rat control through the proper storage, collection, and disposal of refuse is stressed. The types and sizes of containers for storage, the proper method and frequency of collection, and adequate refuse disposal by incineration, sanitary land fill, and garbage grinding are discussed. The two sections dealing with the ratproofing of buildings, both existing and new structures, have been handled admirably. Many detailed illustrations are contained in these sections, and one does not have to qualify as an expert to follow the directions given. The subject of cost estimation which is certainly not a simple or easy task for the uninitiated is dealt with very adequately. Rat eradication and poisoning logically follow the section on ratproofing, and the interrelationships of the two programs are pointed out. The various methods used in rat eradication such as fumigation, poisoning, and trapping, together with the advantages, disadvantages, and precautions, are taken up in detail.

The last section of the manual covers rat ectoparasites and their control. The more common fleas, lice, mites, and ticks found on rats are described and their biologies and habits are discussed. Very useful pictorial keys are given for the identification of the fleas, lice, and mites. The greater portion of this section deals with the control of rat ectoparasites by means of DDT dust, the planning of dusting operations, application of the dust, equipment used, and the keeping of adequate records. The section is concluded by a discussion of evaluation of a DDT dusting program in terms of the changes occurring in the incidence of human typhus, rat typhus, and rat ectoparasites, together with the proper methods to be used in such determinations.

This work is more than just a manual; it is a sourcebook, as well as an operational guide for all those who are concerned with the subject of rat-borne diseases, their prevention, and their control.

*F. Earle Lyman, Scientist (R)*

# Recent Manuscripts by CDC Personnel on Plague and Other Rat-borne Diseases

**MANUSCRIPTS PUBLISHED**

Dent, J. E., Morlan, H. B., and Hill, E. L.: Effects of DDT dusting on domestic rats under colony and field conditions. Pub. Health Rep. 64(21): 666-671 (1949).

Eskey, C. R., Prince, F. M., and Fuller, F. B.: Transmission of *Salmonella enteritidis* by the rat fleas *Xenopsylla cheopis* and *Nosopsylla fasciatus*. Pub. Health Rep. 64(30):933-941 (1949).

Hayes, W. J., Jr., and Simmons, S. W.: The benefits and hazards of insecticides to public health. Advances in Chem. Series. No. 1: 56-60 (1950).

Morlan, H. B., Hill, E. L., and Schubert, J. H.: Serological survey for murine typhus infection in southwest Georgia animals. Pub. Health Rep. 65(2): 57-63 (1950).

Skaliy, Peter, and Hayes, W. J., Jr.: The biology of *Liponyssus bacoti* (Hirst; 1913) (Acarina, Liponyssidae), Am. J. Trop. Med. 29(5): 759-772 (1949).

**MANUSCRIPTS CLEARED FOR PRESENTATION AND/OR PUBLICATION**

Andrews, J. M.: How the Communicable Disease Center can serve the western states.

Douthirt, C. H., and Link, V. B.: Plague – I. Epidemiology.

Ecke, Dean H., and Johnson, Clifford W.: Sylvatic plague in Park County, Colorado.

Engler, Hershel: Methods of municipal waste disposal in relation to rat control.

Gaines, T. B., Sumerford, W. T., and Hayes, W. J., Jr.: The non-toxicity of urine from rats poisoned with 1080.

Hess, A. D.: Some recent developments in the control of animal reservoirs and vectors of disease.

Link, Vernon B.: Plague in North America.

Link, Vernon B.: Plague in the United States.

Mohr, C. O., Tiship, Victor, and Good, N. E.: Results of the DDT dusting program for the control of murine typhus fever in the southeastern states.

Nicholson, H. P., and Vetter, M. H.: A lethal trap for capturing small mammals with their ectoparasites.

Simmons, S. W.: A resume of recent developments on insecticides and rodenticides at the U.S. Public Health Service Laboratory, Savannah, Ga.

# CDC Training Courses

Listed below are training courses, sponsored by Services of the Communicable Disease Center, to be held during the ensuing months of this year. Further information on the courses may be obtained from the *Bulletin of Field Training Programs* issued by the Center.

## TRAINING SERVICES

1. **ENVIRONMENTAL SANITATION FIELD TRAINING**, September 25 to December 16, 1950. Twelve weeks. Amherst, Mass.

2. **FIELD SURVEY AND EVALUATION METHODS FOR MEASURING QUALITY OF HOUSING ENVIRONMENT**, October 9-14 and December 4-9, 1950. One week. Atlanta, Ga.

3. **FIELD SURVEY AND EVALUATION METHODS IN HOUSING SANITATION**, September 18 to October 20 and November 13 to December 15, 1950. Five weeks. Atlanta, Ga.

4. **RAT-BORNE DISEASE PREVENTION AND CONTROL**, October 2-20, 1950. Three weeks. Atlanta, Ga.

5. **ENVIRONMENTAL SANITATION FIELD TRAINING**, September 11 to December 1, 1950. Twelve weeks. Buffalo, N.Y.

6. **ADVANCED TRAINING COURSE FOR STATE SANITARY CHEMISTS PRIMARILY CONCERNED WITH WATER POLLUTION INVESTIGATIONS**, October 2-13, 1950. Two weeks. Cincinnati, Ohio.

7. **ORIENTATION COURSE FOR LABORATORY PERSONNEL IN THE EXAMINATION OF SEWAGE, POLLUTED WATER, AND INDUSTRIAL WASTES**, September 11-29, 1950. Three weeks. Cincinnati, Ohio.

8. **GENERAL SANITARY ENGINEERING FIELD TRAINING** (Special course for newly commissioned engineer officers of the U. S. Public Health Service), September 18 to December 8, 1950. Twelve weeks. Columbus, Ga.

9. **ENVIRONMENTAL SANITATION FIELD TRAINING**, September 18 to December 8, 1950. Twelve weeks. Denver, Colo.

10. **FIELD SURVEY AND EVALUATION METHODS FOR MEASURING QUALITY OF HOUSING ENVIRONMENT**, October 23-28 and December 11-15, 1950. One week. Syracuse, N.Y.

11. **FIELD SURVEY AND EVALUATION METHODS IN HOUSING SANITATION**, October 2 to November 3 and November 20 to December 22, 1950. Five weeks. Syracuse, N.Y.

12. **SPECIAL TRAINING IN RODENT CONTROL**, September 11-22, 1950. Two weeks. Topeka, Kans.

## LABORATORY SERVICES

1. **LABORATORY DIAGNOSIS OF BACTERIAL DISEASES**, Part 1, General Bacteriology. September 11-22, 1950. Two weeks. Atlanta, Ga.

2. **LABORATORY DIAGNOSIS OF BACTERIAL DISEASES**, Part 2, General Bacteriology, September 25 to October 6, 1950. Two weeks. Atlanta, Ga.

3. **LABORATORY DIAGNOSIS OF ENTERIC DISEASES**, Part 1, Introductory Enteric Bacteriology, October 9-13, 1950. One week. Atlanta, Ga.

4. **LABORATORY DIAGNOSIS OF ENTERIC DISEASES**, Part 2, Advanced Enteric Bacteriology, October 16-27, 1950. Two weeks. Atlanta, Ga.

5. **LABORATORY DIAGNOSIS OF PARASITIC DISEASES**, Part 1, Intestinal Parasites, September 18 to October 6, 1950. Three weeks. Atlanta, Ga.

6. **LABORATORY DIAGNOSIS OF PARASITIC DISEASES**, Part 2, Blood Parasites, October 9-27, 1950. Three weeks. Atlanta, Ga.

7. **LABORATORY DIAGNOSIS OF RICKETTSIAL DISEASES**, November 6-10, 1950. One week. Atlanta, Ga.

8. **IDENTIFICATION OF MEDICALLY IMPORTANT ARTHROPODS**, November 13-24, 1950. Two weeks. Atlanta, Ga.

9. **VIRUS ISOLATION AND IDENTIFICATION TECHNIQUES**, November 13-17, 1950. One week. Montgomery, Ala.

10. **LABORATORY DIAGNOSIS OF INFLUENZA**, November 20-24, 1950. One week. Montgomery, Ala.

11. **LABORATORY DIAGNOSIS OF TUBERCULOSIS**, December 4-15, 1950. Two weeks. Atlanta, Ga.

*By Special Arrangement:*

**LABORATORY DIAGNOSIS OF MALARIA**, two weeks, Atlanta, Ga.; **LABORATORY DIAGNOSIS OF VIRUS DISEASES**, two to four weeks, Montgomery, Ala.; and **PHAGE TYPING OF SALMONELLA TYPHOSA**, one week, Atlanta, Ga.

## VETERINARY SERVICES

1. **LABORATORY DIAGNOSIS OF RABIES**, November 27 to December 1, 1950. One week. Montgomery, Ala.


# Present Status of Warfarin As A Rodenticide

WAYLAND J. HAYES, JR., Surgeon*

Warfarin presents a completely new approach to the control of commensal rodents. It was discovered by Link and his students (3,4) who later recognized its rodenticidal potentialities (5). The material is actually 3-(α-acetonylbenzyl)-4-hydroxycoumarin. It was known for some time as compound 42 or W.A.R.F.-42. Under these names it received experimental registration and was tested rather extensively. In June 1950, it was registered for general use, and in the same month a common name, warfarin, was adopted for it. This paper attempts to portray our present knowledge of how this new material fits into the over-all picture of rat control.

Warfarin acts by inhibiting the formulation of prothrombin and by causing capillary damage. Both actions favor the production of hemorrhage, and animals killed by warfarin die from blood loss and shock. Bleeding may occur in any part of the body. The distribution apparently is determined largely by chance, and very minor trauma which under ordinary circumstances would be harmless. In rats, hemorrhages are observed beneath the skin, in muscle septa, within muscles, in the intestines, in the lungs, or retroperitoneally. Any one animal usually shows hemorrhage at a single point or, at most, at a few points. Because of the bleeding, animals poisoned by warfarin gradually develop pallor and weakness. A few of them show external bleeding, but in most of them the actual hemorrhage is not apparent.

Warfarin differs from all previous successful rodenticides in that it must be consumed on several successive days in order to kill, and it produces no acquired bait refusal (bait shyness). These characteristics make it self-prebaiting and adapted to residual control. On the other hand, they make it a slow rodenticide.

Reports already published show that warfarin may be used successfully for the control of mice as well as Norway and roof rats. Early reports by Crabtree (1) and Schein (6) showed that control could be achieved with rather high concentrations of warfarin in bait and suggested baiting practices and schedules which were effective. Hayes and Gaines (2) showed that the material was effective against Norway rats at lower concentrations (0.05 milligrams per gram or 0.005 percent). Thus far, control of roof rats has been achieved with 0.25 milligrams warfarin per gram of bait. Actually, the lowest concentration of poison in bait which will give effective control under actual field conditions has not yet been determined for any of the commensal rodents of public health importance. However, it is possible to obtain control by the concentrations just listed. Further experimental work needs to be done to determine the lower limits of effectiveness. Baits containing lower concentrations of poison would be somewhat cheaper and present slightly less hazard to man and domestic animals. However, the cost of finished bait containing 0.1 milligram of warfarin per gram of corn meal is only $8.25 per hundred pounds** and even with higher concentrations of poison (0.25 milligrams per gram) no actual difficulty with toxicity to man or domestic animals has developed yet in spite of rather extensive field trials. What probably will determine whether warfarin can be used economically as a rodenticide for public health purposes is not the cost of finished bait but the cost of protective bait stations and the cost of labor of those who actually carry out the poison-

*From Technical Development Services, Savannah, Ga.

**Based on $4.05, the present cost of 100 pounds of corn meal to the government, and $2.15, the current cost per pound of 0.5 percent formulations of warfarin in 5-pound lots.

ing program. So far, the experimental work has been done using protective stations for all bait placements that were accessible to children or domestic animals. Even the simplest station costs about $1.30, provided new material is used and labor is estimated at current pay scales. Thus, the cost of materials for initial poisoning for a single establishment has been estimated at about $0.46 for finished bait and $11.23 for bait and stations. Further work will be necessary to determine whether the use of many or all bait stations may be dispensed with. In the meantime, warfarin is effective for the control of commensal rodents and is economically feasible under certain circumstances. On farms and around private residences, satisfactory bait stations may be improvised of scrap materials at essentially no cost. An 8- or 10-inch board 2 or 3 feet long placed on edge and nailed against the wall and floor at about a 45° angle to form a tunnel makes a good station. On ships that do not carry children as passengers, no bait stations are required if the poisoned bait is colored and each placement is marked "POISON." Similarly, in warehouses and certain other business establishments where it is possible to warn all personnel that have access to the building, it may be possible to dispense with bait stations and simply use colored bait and prominent warning signs. Even where it is necessary to use bait stations, it may be feasible economically for rodent control units either of governmental agencies or of commercial pest operators to invest in a stock of bait stations. These stations are, after all, quite durable and, if poisoning should be carried out over a period of months and years, then the initial cost of the stations can be considered a part of the long-time operating cost.

At the present time, then, warfarin offers a form of residual rodent control previously found impossible. This control may now be obtained at a reasonable cost under certain conditions. Further experience is needed greatly to determine whether it is feasible economically to use warfarin under the general conditions existing in public health work.

**REFERENCES**


(1) Crabtree, D. G.: Raticidal potentialities of WARF-42. Soap & Sanit. Chem. 26: 131-137 (1950).

(2) Hayes, W. J., Jr., and Gaines, T. B.: Control of Norway rats with the residual rodenticide warfarin (In press).

(3) Ikawa, M., Stahmann, M. A., and Link, K. P.: 4-Hydroxycoumarins. V. Condensation of $\alpha,\beta$-unsaturated ketones with 4-hydroxycoumarin. J. Amer. Chem. Soc. 66: 902-906 (1944).

(4) Overman, R. S., Stahmann, M. A., Huebner, C. F., Sullivan, W. R., Spero, L., Doherty, D. G., Ikawa, M., Graf, L. H., Roseman, S., and Link, K. P.: Studies on the hemorrhagic sweet clover disease. XIII. Anticoagulant activity and structure in the 4-hydroxycoumarin group. J. Biol. Chem. 153: 5-24 (1944).

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(6) Schein, M. W.: Field test of the efficiency of the rodenticide compound W.A.R.F. 42. Pub. Health Rep. 65: 368-372 (1950).


# International Cooperation

CDC's cooperation with other countries has been pointed up again, with the Western CDC Laboratory at San Francisco, Calif., aiding a Canadian provincial health department in June of this year.

The Saskatchewan Provincial Department of Health, Division of Communicable Disease, sought instruction in Public Health Service field methods for a crew doing survey work on ticks and plague-infected wild rodents.

When a Western CDC Laboratory field unit was operating in Bottineau County, N. Dak., June 18-30, the Saskatchewan field unit spent 2 days studying the methods used by the Service.

Personnel of the Saskatchewan unit – representatives of the University of Saskatchewan Department of Biology – were instructed in hunting, trapping, mass fumigation with cyanide, collecting ectoparasites, animal autopsy, pathological signs of plague, record keeping, and mailing of specimens.


# Ten-Eighty, A Rat Poison for Professional Use

**PRODUCTION NO.:** CDC 5-124, Released 1950

**DATA:** Filmstrip; 35 mm., Sound, Black and White; Length: 73 Frames; Time: 15 Minutes

**GRAPHIC FORM:** Photographs, Drawings

**PURPOSE**

To aid in teaching the properties and use of the powerful but dangerous poison for rodent control: 1080 or sodium fluoroacetate.

**AUDIENCE**

Professional public health and pest control personnel and other groups involved in rodent control (positively restricted to these groups).

**CONTENT**

**I. Introduction:** Effectiveness of 1080. Preparation of 1080 water. Manufacture of 1080. Placing poison cups in buildings.

**II. Properties of compound 1080:** A. Favorable properties: (1) Ready acceptance. (2) Effective overwide range of concentration. (3) No lasting tolerance developed. (4) Acts quickly. B. Properties with both good and bad aspects: (1) Extremely toxic. (2) 1080 is absorbed rapidly in the gastrointestinal tract. (3) It is chemically stable and is not volatile. (4) It is highly soluble in water. C. Unfavorable properties: (1) It resembles certain common foodstuffs in appearance. (2) Its dust floats readily in the air.

**III. Preparation of 1080 water:** Safe storage for stock 1080 is necessary. 1080 poison label should appear on everything contacting 1080. Steps in the preparation of 1080 concentrate are shown. All apparatus must be washed thoroughly after use. The small jars of 1080 concentrate should be carried to the field shop by private carrier or in person.

**IV. Poisoning rats with 1080 water in cups:** The rodent control kit for use with 1080. Preparing 1080 water from concentrate. Crew member arriving at the building where 1080 will be used, after employees leave for the day. Approved types of cups. Choosing locations and placing 1080 cups. Filling the cups. Cups to be placed near fresh rat signs such as tracks, droppings, gnawings, and harborages. DDT to be used if typhus is suspected. Cups not to be placed where food may become contaminated by them. All utensils used in putting out 1080 to be washed thoroughly after the cups are placed and filled. Danger notice to be displayed in conspicuous place. Rats drinking 1080 water. Crew member returning early next morning before employees. Cups and dead rats being picked up. Scenes at the cups showing dead rats. Hunting for dead rats. Disposal of dead rats, used cups, and surplus 1080 in incinerator. Washing up. Structure and use of permanent stations. Checking on remaining infestation.

**COMMENT**

By illustrating the favorable and unfavorable physical and chemical properties of 1080, and by showing approved and safe methods for its use, this filmstrip should aid rodent control personnel both in following the present procedures for killing rats with 1080 and in developing even better techniques in the future.

Gathering rats poisoned by compound 1080.



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