

## CHAPTER 2

# THE MEDICAL IMPORTANCE OF FLEAS

### BUBONIC PLAGUE IN NORTH AMERICA

The Middle Age scourge of Europe "Black Death," known today as Bubonic Plague, first made its appearance on this continent in man in the year 1900. On March 6 of that year a Chinese man was discovered to have died of the disease in the Chinese section of the big west coast port of San Francisco. This was the beginning of a series of cases in the Chinese and Japanese quarters of this city, which extended through February of 1904. During this period 121 cases were found, of which 113 terminated fatally.

It will probably never be known when plague made its way into the rat population of the Bay area or how long it had been in these rodents before it appeared in man. It is supposed that rats infested with the Oriental rat flea (*Xenopsylla cheopis*) escaped off ships from the Orient into the city of San Francisco to spread the infection through uninfected rats and later to the ground squirrels. Neither the so-called domestic rats, Norwegian, Alexander, Black, nor the house mouse, nor their 3 common fleas—the Oriental rat flea (*Xenopsylla cheopis*), the European rat flea (*Nosopsyllus fasciatus*), nor the mouse flea (*Leptopsyllus segnis*) are native of this country. All seem to have been introduced through shipping channels. These rats and mice are very susceptible to plague; their 3 fleas are known to be vectors (transmitters) of this disease.

The history of the spread of plague in man in North America is interesting.

CALIFORNIA: After the first epidemic in North America had closed in San Francisco in February, 1904, there was no recurrence of the disease until May, 1907. During this month in San Francisco a sailor died of the disease. This was the beginning of the second epidemic. By June 30, 1908 when the last case of this series was reported, there had been 159 cases with 77 deaths. In this second epidemic the cases were scattered all over the city, not confined to the Chinese section. Nor was San Francisco the only locality touched during this period. Oakland reported 12 cases during 1907, Berkeley 1, and Contra Costa County 3. During 1908 Oakland reported 1 case, Contra Costa County 2, and Los Angeles 1. From 1909 through 1915 isolated cases appeared each year, the period covering 14 cases with 6 deaths; the infection spread to San Benito and San Joaquin Counties. Human cases

were not again reported in California until August-September of 1919 when in Oakland a man who had been hunting ground squirrels in an infected area became ill 2 days after the hunt, and developed pneumonic plague. Twelve other cases developed, all terminating fatally. During 1920 a case terminated fatally in Alameda County. Three cases developed in 1921 with 1 death in San Benito County; 1922: 1 case each with no deaths in Oakland and Santa Cruz County; 1923: a non-fatal case in San Francisco.

The year 1924 stands out as an epidemic year. Between November 1, 1924, and January 5, 1925, 33 cases of pneumonic plague resulted in 31 deaths, and 8 cases of bubonic plague resulted in 3 deaths, all in Los Angeles. Authorities seemed unable to trace the infection in this epidemic to rats or ground squirrels, although at the time to the north in San Luis Obispo County there had been a virulent outbreak of plague in ground squirrels.

From 1925 to 1937, 8 California counties reported 10 cases of human plague with 5 deaths. The counties in which these cases occurred were: Los Angeles 1925 and 1933, Contra Costa 1927, Santa Cruz 1928, Monterey 1928 and 1936, Santa Barbara 1928, Tulare 1934, Sonoma 1936, Placer 1936, and Fresno 1937. During 1938, 1939, and 1940, there were no cases reported from the state. Far to the north of any previous record, plague appeared in 1941 in Siskiyou County. Two children, 5-year-old and a 10-year-old boy, died of the infection at Montague and Mt. Shasta City. Infected ground squirrels were subsequently found in the area. At Yreka, Siskiyou County, on November 9, 1942 a case appeared in a small 2½-year-old girl. The tot had been helping her father haul hay from a squirrel infected area. On January 10, 1943, the case terminated fatally.

To date, then, California has had 395 cases of plague in man, 268 of which terminated fatally.

OREGON: But one case of plague in man has come to the attention of authorities in Oregon. In the spring of 1934 a sheepherder made camp on Guano Creek Plateau, Lake County, north of Lakeview. The camp was in the shelter of rim rocks which afforded home sites for many marmots (woodchucks). The sheepherder became ill and died of what was diagnosed as bubonic plague. Suspicion was at once directed toward the marmots in the rocks but it was not until May 8, 1942, that these animals were found plague infested at Abert Lake to the north.

WASHINGTON: The first cases of plague in Washington appeared in Seattle during October of 1907. The records are not exactly clear as to the number, but 7 cases with 7 deaths is the largest figure. This large port town was heavily infested with rats which were found infected with plague that year and continued to be plague positive to 1917. No human cases developed after the 7 cases and no infected rats were found after 1917.

LOUISIANA: In 1912 and 1914 plague infected rats were found in

New Orleans and between June 21 and September 8, 1914, 30 cases of human plague were recorded, of which 10 terminated fatally. A mild case of plague was found during 1915 and during 1919-1920, 22 cases occurred with 8 deaths. In 1921 this city reported 3 cases which terminated fatally. No further cases have been reported from Louisiana.

FLORIDA: In Pensacola between May 31 and August 31, 1920, 10 cases of plague in man were reported, 3 of them ending in death.

TEXAS: From June 16 to November 14, 1920, 18 cases of plague in man were reported from Galveston. There were 12 deaths. Plague infested rats failed to show in trappings after May 29, 1922. From June 19 to August 23, 1920, Beaumont reported 14 cases with 6 deaths. Port Arthur reported 1 case during 1920. It terminated fatally.

UTAH: No fatal cases of plague have been reported in man from this state, but during 1936 a mild case was reported and during 1939 another. In the latter case the 29-year-old patient was skinning coyotes and other animals prior to the onset December 4. The case was in Millard County.

NEVADA: In 1937, a non-fatal case was reported from this state.

CANADA: Rodent plague surveys carried on in British Columbia have failed to uncover plague infection in that territory; human cases have not so far been reported from there.

During 1939, in the province of Alberta, a farmer died from what was diagnosed as acute septicemia; but other circumstances led to the belief that the death was caused by plague. The victim, a 35-year-old mink raiser had been feeding his mink on the ground squirrel *Citellus columbianus* collected in the vicinity. Some of the mink became ill and died. The farmer, in skinning these, scratched his hand and apparently received the fatal infection. Shortly after the death of this individual, an epizootic was noted in the ground squirrels of this part of south-eastern Alberta, and an investigation proved by tissue and by flea pools that these squirrels, *Citellus columbianus*, were plague positive. Since that time, plague positive ground squirrels of the same variety have been found in the regions of Stanmore, Suffield, Sunnybrook, Youngstown and Hanna.

#### SUMMARY

From its inception into man in North America in 1900, plague has been found in 506 persons, 321 of whom died of the infection. Eight states have reported human cases, the outbreaks having occurred in the following chronological order: California 1900, Washington 1907, Louisiana 1914, Florida 1920, Texas 1920, Oregon 1934, Utah 1936, Nevada 1937.

#### A WORD OF WARNING

Because of the small amount of plague in humans in North America in recent years, the American peoples should not be lulled into a sense of security, for any such security would surely prove a false one. Plague

all through history has had periods of quiescence and recrudescence. No one can tell when the disease will again strike or where. Every state in the Rocky Mountains and west to the Pacific Ocean has its centers of infection in the wild rodents. The infection is there. It has only to spring forth.

#### HOW PLAGUE WORKS

Plague is a bacterial disease caused by the organism *Pasteurella pestis*. This bacillus was first isolated during 1894. Yersin and Kitasato working independently discovered it about the same time. The bacillus is a short, plump oval rod measuring from 0.3 to 1.25  $\mu$  in length. It can be found singly or in pairs, but long chains are rare. There is no characteristic arrangement. The bacilli are encapsulated, non-motile. Involution forms are common and may appear as coccus forms, large rods or swollen bodies. The plague bacillus does not produce soluble toxic substances. In general, the life of this bacteria outside the animal body is precarious. It seems to disappear speedily from soil, water, and buried bodies.

The opinion has been expressed that plague caused by this organism is primarily a disease of wild rodents, and that man is only an incidental victim. But in spite of this opinion it is said that about one-quarter of the population of Europe was carried off by this disease during the "great mortality" of Black Death of the fourteenth century. A civilization, or lack of it, in which peoples are allowed to live in filth and poverty, making them bedfellows with rats and mice, fosters outbreaks of human plague. In North America, besides the imported rats and mice, a whole series of native rodents, particularly ground squirrels, are known to harbor plague.

Plague in man is generally found to be of two types.

**BUBONIC PLAGUE:** This type of plague develops in a man only after he has been bitten by a flea which has become infective through sucking blood from a plague infected animal. In this type of plague diagnosis on clinical grounds is said to be relatively simple. An inflammatory swelling of the lymphatic glands occurs. These are called buboes. From these, bacilli may pass over into the blood. It is from these buboes that the disease received its name bubonic plague and from the point of attack the name of glandular plague. When the bacilli pass into the blood, they multiply extensively. Septicemia may occur and at times subcutaneous hemorrhages. These hemorrhages were far more common during the Middle Ages than now. The black patches due to the hemorrhages gave the medieval name of "Black Death." The case fatality of bubonic plague is 60 to 90 per cent.

In bubonic plague the role played by the flea is of vital importance. It is the go-between or vector. Under certain conditions the flea may transmit plague between rodent and man. These conditions are as fol-

lows: First, the rodent must be infected and have the plague bacilli invading the blood stream. Second, the flea must feed upon the infected rodent and suck into its mouth parts and swallow into its proventriculus and stomach the blood containing the plague bacilli. Third, in the proventriculus of the flea the bacteria of plague reproduce rapidly to form an obstruction. This obstruction or dam is called a "block" and fleas so affected are said to be "blocked." Sometimes this block may occur in the esophagus. It is not possible for the flea to swallow beyond the block. Fourth, the flea is now infectious. It becomes hungry, wishes to feed. If it bites a man or an uninfected rodent it sucks the blood into its mouth parts but is unable to swallow it because of the block. As the new blood washes across the block, the plague bacilli become mixed with it. Because the flea cannot swallow, it vomits or regurgitates the now infected blood back into the bite and so the bacilli make their way into the new animal and infection begins. Blocked fleas are unusually dangerous because, being unable to satisfy their hunger, they repeatedly bite and try to feed, thus spreading the infection as they go.

Since plague bacilli can be found in the feces of fleas it has been thought that the scratching of this fecal matter into the skin could cause plague infection, but recent research tends to discredit this theory.

**PNEUMONIC PLAGUE:** This type of plague is also known as plague pneumonia. It occurs secondary to bubonic plague. It seems that the bubonic plague infection settles in the lungs to give a pneumonia-like condition there. In this type of the disease in man no flea is necessary for the spread of the infection. In patients suffering from this type, huge numbers of the plague bacilli are found in the sputum and the infection is spread from person to person through the cough or sneeze droplets. Because of this direct spread, pneumonic plague is very much more dangerous than the bubonic type. It is also much more fatal, the case fatality being almost 100 per cent. The outbreak in Oakland, California during 1919 was of this type. In Manchuria during the years 1910-1912, 60,000 fatal cases were attributed to this lung infection.

**IMMUNITY:** Suspensions of attenuated or killed plague bacilli used as a vaccine have proved successful in immunizing experimental animals. Haffkine in 1897 developed a method of protective inoculation consisting of injecting heat-killed bacilli from old cultures. This method has been used extensively in India and has proved successful in some cases. Antisera may be prepared for the disease by immunization of horses and other animals, but their efficacy as therapeutic agents is doubtful. Research laboratories are constantly searching for a successful treatment of human plague.

#### TYPHUS IN NORTH AMERICA

Only recently has it become generally known that fleas in North America have become vectors of typhus. This disease is one of the seri-

ous scourges of the Old World. The late Dr. Hans Zinsser of Harvard University states that it has killed more human beings than any other disease.

According to the statements of Dr. J. D. Ratcliff, Chief of the United States Public Health Service, Washington, D. C., the chief health officer of Baltimore called for the Service to examine people stricken with a high fever that ran for about two weeks, the persons having a reddish rash on their chests and abdomens. Dr. Rumreich, sent to investigate, found that the persons were suffering from typhus, the ancient killer of Eastern Europe and Asia. This disease is known to have caused Napoleon to withdraw his army from Russia. It is known to have killed 3 million Russians during World War I. But the disease in Baltimore did not possess this killing power.

Dr. Rumreich finally found in the basement of a drug store the focal center of the disease. The cellar was alive with rats. He trapped these and sent them back to Washington. In the laboratory the well-trained Dr. R. E. Dyer watched and examined the rats. The Pasteur Institute had found the louse responsible for human typhus in Europe, but the rats at hand were infested heavily with *Xenopsylla cheopis*—the dreaded vector of bubonic plague. Could this flea also spread typhus? Dr. Dyer found it could, by himself becoming an accidental guinea pig. One evening while preparing a salt solution of macerated remains of fleas for injection into guinea pigs, a few, quite accidentally, escaped and settled upon his body. Two weeks later the doctor had his first chill, then a temperature of 105 degrees. The doctor recovered; his disease was typhus. To quote Dr. Ratcliff, "He (Dr. Dyer) had taken the disease into the laboratory where research men could get it, and had proved the flea as the vector—the agent which passed the disease from animal to animal and from animal to man. Fleas could do the job all right: from rat to rat; from rat to guinea pig; from rat to man."

During 1941 it was reported that two pools of fleas taken from Norwegian rats in Georgia during 1939 were found to be typhus positive; these finds definitely condemned the sticktight hen flea and the European mouse flea as vectors of murine typhus. During May of 1939 on a certain farm in Georgia, where the farmer lay ill with a case of endemic typhus fever, several rats were trapped. From these were removed 135 1.\* *Echidnophaga gallinacea* Westwood 1875, the sticktight hen flea; five 9.\* *Xenopsylla cheopis* Rothschild 1903, the oriental rat flea; and seven 213.\* *Leptopsylla segnis* Schonherr 1811, the European mouse flea, all of which proved typhus positive as did the brains of the rats. The strain of typhus was proved in these fleas by being passed through 14 generations of guinea pigs. The great majority of these developed clinical endemic typhus with necrotic scrotal reactions.

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\* See page 43 for explanation of reference numbers.

So typhus is definitely known to be spread in America by rat fleas, the mouse flea, and the sticktight hen flea.

For some time typhus in the Old World has been considered different from New World typhus. The former was considered to have a much higher death rate than the latter. The two types of the disease were considered to be caused by a slightly different organism.

The causative agent of the disease is the organism *Rickettsia prowazekii*, which is stated by some authorities to lie midway between bacteria and filterable virus. Not all investigators hold that there are two varieties of this organism but it is generally felt that *Rickettsia prowazekii* causes European or epidemic typhus and *R. p. mooser* causes New World endemic or murine typhus.

One of the chief differences found in the two varieties of typhus organisms is that *R. p. mooser* will produce a necrotic scrotal reaction in guinea pigs while *R. p. prowazekii* will not. These organisms characteristically occur in the cytoplasm but not in the nucleus of the invaded cells.

In both types of the disease the incubation period in man is from 5 to 18 days. Violent headache, with fever and chills are characteristic. A macular eruption occurs after the fourth day. The crisis occurs in 12 days with recovery in about another 2 weeks. There are various complications. Case fatality may be from 2 per cent in North America to as high as 70 per cent in the Old World.

The disease is almost always associated with filth and overcrowding, with body lice in Europe and with fleas in North America. In Europe it is not known what maintains the reservoirs of typhus but in North America the reservoirs are in the domestic rats and many wild mice, thus the name murine typhus.

Typhus is not an uncommon disease in the United States. The reported cases for 1934 are over 1,308, the majority of which occur in the southern states. The disease is common in Mexico.

Clinically, it is stated that European typhus is not appreciably different from North American typhus, but it is known that the vector of the disease in Europe is the human body louse and possibly the human head louse, while the vectors of murine or North American typhus are various fleas and probably the rat louse *Polyplax spinulosa*.

Recovery from either type of typhus is said to result in a solid and lasting immunity to both. Vaccinations administered for murine typhus give complete protection against murine typhus only and give only incomplete protection against Old World typhus.

Since it has been a definitely established fact that the reservoirs of murine typhus are in the domestic rats and that both the rat flea and rat louse may act as vectors of the disease, it seems that strict control over rats is a very desirable method of controlling the disease at its source.

## TULAREMIA IN NORTH AMERICA

The disease tularemia is primarily one of rodents but when expressed in man it is known by a series of common names such as rabbit fever, deer fly fever, the tired disease. Early in the history of plague investigations in the state of California, tularemia came to light. McCoy working on plague in the California ground squirrel reported the discovery of the organism causing the disease in 1912 and with Chapin named the organism *Bacterium tularense*. It is generally understood that the organism bears the name of the California county in which it was first found, that is Tulare County.

Today a whole series of rodents and other animals are known to carry tularemia. In them it is a plague-like disease. It has been found in rabbits, meadow mice, ground squirrels, marmots, tree squirrels, prairie dogs, muskrats, beaver and other wild animals.

As early as 1922 Dr. Edward Francis stated that the California ground squirrel flea *Ceratophyllus acutus* (69. *Diamanus montanus* Baker 1895) had transmitted the disease between ground squirrels in one experiment. During 1933 in Minnesota an epizootic of tularemia among cottontail rabbits revealed tularemia infection in two pools of fleas from these rabbits. During 1934 another pool of fleas off these cottontail rabbits proved tularemia positive. During 1935 tularemia positive fleas were taken off snowshoe rabbits in Minnesota. A survey made on snowshoe rabbits between June 9 and July 28, 1937, in south central Alaska, north to Fairbanks and Circle, which involved the examination of 172 animals, proved these varying hares to be tularemia positive. During this survey both ticks and fleas were found very scarce. Some of the ticks recovered were found tularemia positive.

During 1938 tularemia positive Sage Rats (*Citellus townsendi vigilis*) were determined by tissue pool from animals taken at Ontario, Malheur County, Oregon.

Twenty-four prairie-dogs secured for examination during June, 1941, in Carbon County, Wyoming, supplied a pool of 43 fleas which proved tularemia positive. These fleas produced tularemia in guinea pigs in the Plague Suppressive Measures Laboratory at San Francisco. While no specific mention was made of the fleas involved, it is known that the chief flea carried by prairie-dogs in this part of the country is 76. *Opisocrostis hirsutus* Baker 1895.

During 1939 and 1940 spontaneous tularemia was reported from southern Montana and northern Wyoming in the beaver populations. Muskrats taken in Klamath Lake, Lake County, Oregon, during April, 1942, were found tularemia positive. In the beaver and muskrat epizootics mentioned, fleas were not involved, since these animals seem not to carry them.

A disease commonly referred to as "muskrat fever" by local physicians at Ruby Valley, Nevada, may prove to be tularemia. In Ruby

Valley lies Ruby Lake, a large swamp 60 miles south of Wells, Elko County, Nevada. From it each year the catch of muskrat and beaver is large. During the trapping season the trappers suffer from the disease which consists of a painful ulcer on the hand or forearm. The ulcer may become multiple. Fever appears with the first ulcer and persists until recovery. No fatal cases have been reported. It is thought the disease is contracted from the muskrats while the trappers are skinning them. The disease came to the attention of health authorities during 1939 and 1940.

An Illinois natural history survey during 1939-1940 revealed tularemia in the rabbits of this state and the total of 485 human cases to that date.

On August 18, 1942, a tularemia-positive flea was found 40 miles south of Suffield, Alberta, Canada, the flea presumably off a ground squirrel.

Tularemia-positive ticks were reported from several localities in Alberta, Canada, during 1942; and a very interesting case cited for this year was of a band of 850 sheep in southern Alberta in which 24 died and many were ill of this disease. The sheep herder who tended these sheep skinned the dead animals and contracted the disease. Ticks removed from the infected animals proved tularemia positive in 1 case.

During September, 1942, a dead meadow mouse (*Microtus*) picked up at Newell, South Dakota, was found to be tularemia positive.

Man can contract tularemia while skinning any animal suffering from the disease if the animal's blood comes in contact with an abrasion on his person. Man can also contract this disease by being bitten by a vector of the disease. To date it is thought that certain fleas, certain ticks, and the deer fly, *Chrysops discalis*, can carry the disease from animal to man. The disease is widely spread in the United States. By 1936 it had been reported from 46 of our 48 states. It is far more prevalent than plague. There are thousands of cases on record.

The causative agent of the disease *Pasteurella tularensis* is a minute rod 0.2  $\mu$  in breadth and from 0.3 to 0.7  $\mu$  in length. In young cultures the organism is rod-shaped, in old cultures, coccoid. These bacteria are non-motile, non-spore forming and apparently encapsulated.

In man four clinical types of tularemia are recognized. The more common type is the glandular or ulceroglandular variety, another type is the so-called "typhoidal" variety.

Tularemia is characterized by headache, pains and fever. A papule appears where the bacteria enter the body. This later breaks down and forms an ulcer. Certain glands become painful and swollen and may break down and discharge purulent material. Infection through the eye area may cause ulcers on the eyelids. The disease runs a course of from 2 to 4 weeks. The case fatality is low, being about 5 per cent.

An attack of this disease confers a solid immunity. Second infections, if they occur, produce only a local lesion.

Attempts to actively immunize human beings have been unsuccessful. Antisera seem to have no value.

#### FLEA ALLERGY

It has long been known that certain individuals suffer more severely from the bites of insects than do others. Of late, those people who suffer severely from flea bites are said to have flea allergy. There is no doubt that many adults and most children do seem to be sought out by fleas for attack. Very fair skinned individuals, often those with red hair, and tender skinned youngsters become targets for fleas, the bites starting frequently at the top of the shoes and then ranging all over the body as the flea or fleas feed. In people sensitive to flea bites the wound may be followed by swelling which can develop into large welts. The situation is aggravated by the accompanying itching and scratching. In so far as most of these attacks are from cat fleas, dog fleas and human fleas, the attack is more in the nature of a nuisance raid than one of danger.

In many places people suffering from flea allergy can clear up most of the situation by doing without pet cats and dogs, but in many coast towns and on farms where pigs are raised the numbers of human fleas are so great that control seems almost out of the question. Practically all itch allaying preparations obtainable at drug stores have been tried to sooth the bites of fleas. One investigator informed the writer that a piece of adhesive tape placed over the bite would stop the itching.

After considerable research upon flea allergy, Ely Lilly and Co., well known American drug house, took over the manufacture of a preparation which they named "Flea Antigen." This is prepared by the method of Dr. Albert C. Reed and associates at the Hooper Foundation for Medical Research of the University of California. It is an extract of the fleas of cats, dogs, and human beings in sterile phenolated isotonic saline solution. The usual initial subcutaneous dose for adults is 0.2 cc. This dose is usually increased to 0.4 cc. for 5 subsequent injections with intervals of 2 days between. The preparation number is PA-90. It may be purchased in 5 cc. vials. This preparation is thought to be effective against human, cat, and dog fleas only. It must be administered by a physician.

#### FLEAS AS HOUSEHOLD PESTS

The human flea, the cat flea, and dog flea can become household pests during their season of maximal abundance, which is generally during summer and fall. As sawdust burners become more and more common in basements, people are blaming the sawdust more and more for being the vehicle in which the fleas are delivered to the house. This may be true but it is difficult to see how an insect which lives entirely on blood in the adult stage and is interested mostly in host castings of one type or another in the larval stage can be associated with a pile of

sawdust which has just come from a lumber mill out of a wet log. It is far more probable that the fleas are coming from domestic animals in the house or in the yard, their presence in the sawdust being incidental. If cats and dogs are allowed the liberty of the house, their fleas will drop their eggs everywhere on the premises. The eggs hatch into worm-like larvae which live in cracks and crevices and feed upon accumulations in these places. On farms where pigs are raised, these animals often become heavily infested with the human flea. Under such conditions the farm cats and dogs and the farmers themselves carry large numbers of these fleas into the house. Occasionally the European rat flea and the western chicken flea may contact man and get into houses.

The first point of control of fleas in the home is determining where the fleas are originating. Such determinations can only be made by examining the fleas. It is very difficult at times to make owners of pet cats and dogs realize that their pets may be bringing the fleas into the home, but too often this is the case. These household pets should be periodically powdered or bathed with a flea preparation. Floors and cracks should be cleaned of dust and castings, flea eggs and larvae. Basements should have their floors liberally sprinkled with salt water, or better still, a good application of five pounds or more of naphthalene flakes. After lying in place a day or so the flakes can be swept up and stored away for future application.

#### FLEAS AS FARM PESTS

Three fleas of agricultural importance may at times become startlingly common upon the farm. The western chicken flea, *Ceratophyllus niger niger* C. Fox and the tropical hen or sticktight flea, *Echidnophaga gallinacea* Westwood may seriously menace the poultry on the farm. In the pig pens, the human flea, *Pulex irritans* Linnaeus, may become a serious nuisance. All three fleas can be controlled by liberal applications of kerosene or used engine oil about the premises.