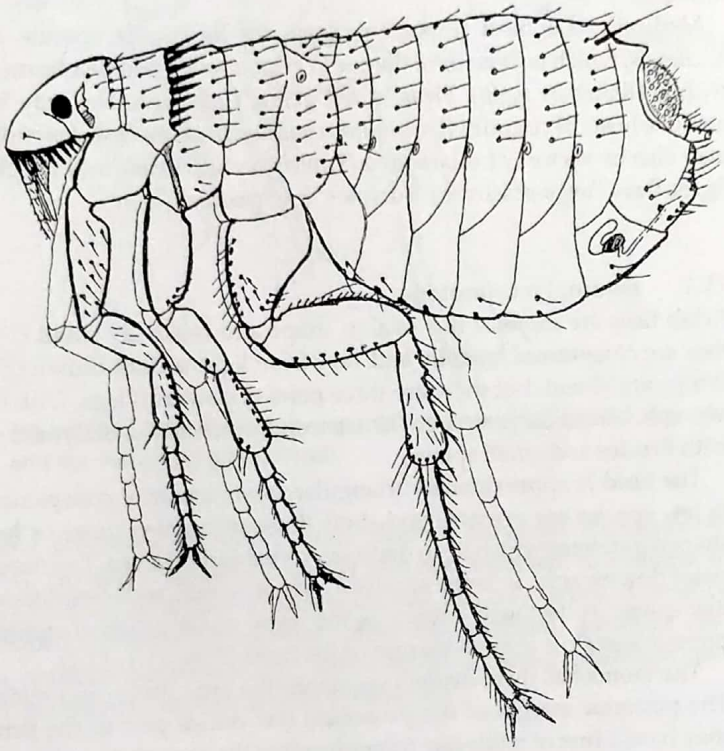
11

Fleas (Siphonaptera)



There are about 2500 species and subspecies of fleas in about 220 genera, but only relatively few are important pests of humans. About 94% of species bite mammals, while the remainder are parasitic on birds. Fleas occur almost worldwide, but many have a more restricted distribution; for example the genus *Xenopsylla*, which contains important plague vectors, is confined to the tropics and warmer parts of some temperate countries.

Medically the most important fleas are *Xenopsylla* species, such as *X. cheopis*, which is a vector of plague (*Yersinia pestis*) and flea-borne murine typhus (*Rickettsia typhi*). Fleas in the genus *Ctenocephalides* may be intermediate hosts of cestodes (*Dipylidium caninum*, *Hymenolepis diminuta*). Fleas may also be vectors of tularaemia (*Francisella tularensis*), and the chigoe or jigger flea (*Tunga penetrans*) 'burrows' into people's feet.

11.1 External morphology

Adult fleas are more or less oval in shape and relatively small (1–6 mm); they are ***compressed*** laterally and vary from light to dark brown (Plate 19). Wings are absent, but there are three pairs of powerful legs, with the hind legs specialized for jumping. The legs, and much of the body, are covered with bristles and small spines.

The head is approximately triangular, bears a pair of conspicuous eyes (a few species are eyeless), and short three-segmented more or less club-shaped ***antennae*** which lie in depressions behind the eyes. The mouthparts point downwards. In some species a row of coarse, well-developed tooth-like spines, collectively known as the ***genal comb*** or genal ctenidium, is present along the bottom margin of the head (Figs. 11.1, 11.2).

The thorax has three distinct segments: the pro-, meso- and metathorax. The posterior margin of the pronotum (i.e. dorsal part of the prothorax) may have a row of tooth-like spines forming the ***pronotal comb*** or pronotal ctenidium (Fig. 11.1). Fleas in some genera lack both pronotal and genal combs and are referred to as ***combless*** fleas (Fig. 11.2). In some genera fleas have both combs, while in other species the pronotal comb is present and the genal comb absent, but never the reverse (Fig. 11.2). A sternite called the mesopleuron is located above the middle pair of legs. In several genera, including *Xenopsylla*, which contains important plague vectors, this sternite is clearly divided into two parts by a thick vertical rod-like structure called the ***meral rod***, pleural rod, mesopleural suture or just rod. The presence of this rod, combined with the absence of both genal and pronotal combs, indicates the genus *Xenopsylla* (Fig. 11.2). However, it must be stressed that the presence of a meral rod by itself does not identify fleas as *Xenopsylla* species, because fleas in several other genera have combs and a meral rod.

In female fleas the tip of the abdomen is more rounded than in males and is not upturned as in males. Internally in about the sixth to eighth

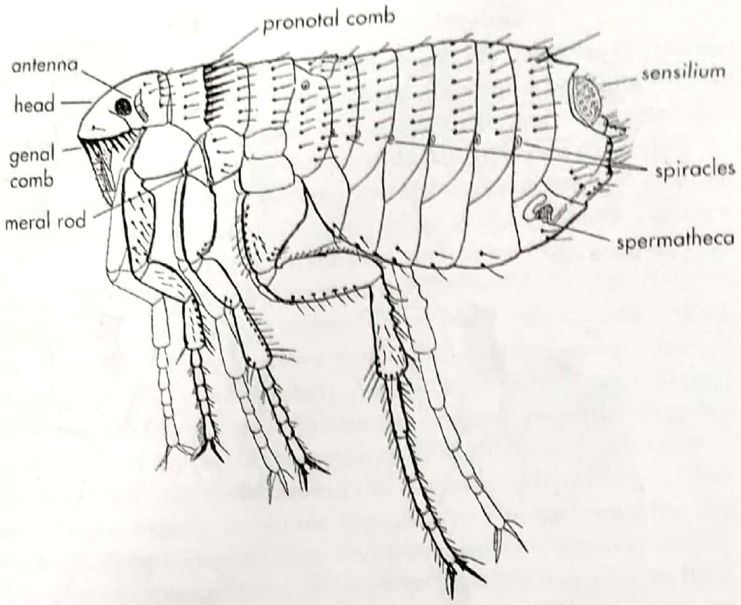


Figure 11.1 Lateral view of an adult flea, showing position of combs and the meral rod (pleural rod).

abdominal segments are one or two distinct brownish spermathecae (Fig. 11.1). However, epidemiologically it is not important to distinguish the sexes because ***both*** take blood-meals and can be vectors. A sensory dome-shaped structure having setae, called the sensilium, is present dorsally on apparent segment 8 and aids fleas in detecting vibrations and temperature changes as well as in host detection.

11.1.1 The alimentary tract of adult fleas

To understand the role fleas have in transmitting plague it is necessary to describe the alimentary tract and the method of blood-feeding.

Saliva, which contains anticoagulants, is injected into the host during feeding. Blood is sucked up through the pharynx and oesophagus into the bulbous ***proventriculus*** (Fig. 11.3), which is provided internally with numerous (250–450) backward-projecting spines. It was previously accepted that these spines prevented the regurgitation of the blood-meal into the oesophagus. However, recent experiments seem to show that regurgitation has little or no effect on disease transmission.

Finally, the blood-meal enters a relatively large stomach (mid-gut), where it is digested. The hind-gut is continuous with a small dilated rectum, which has rectal glands that extract water so that the faeces pass out in a dry state.

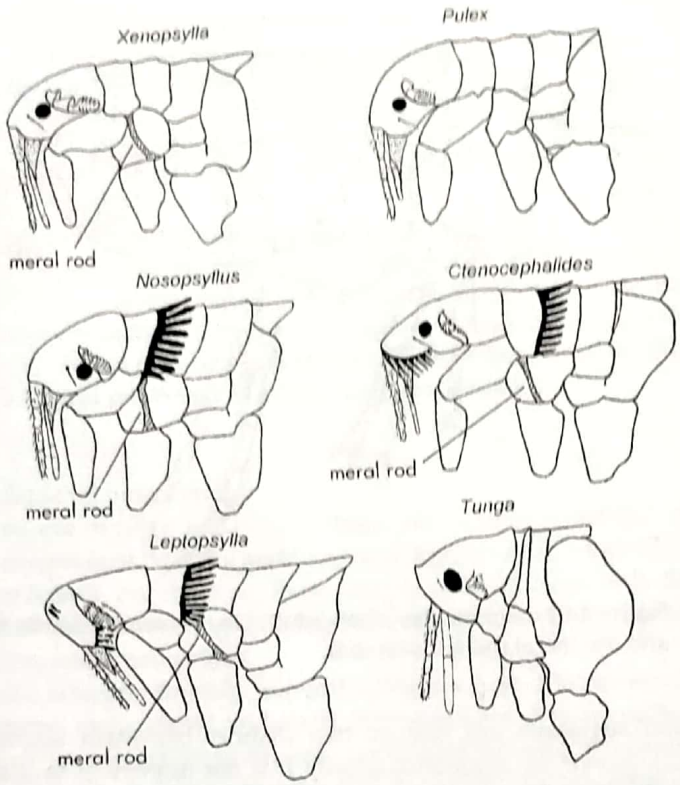


Figure 11.2 Diagrams of the head and thoracic segments of adult fleas, showing how the presence or absence of combs and the meral rod can distinguish six medically important genera.

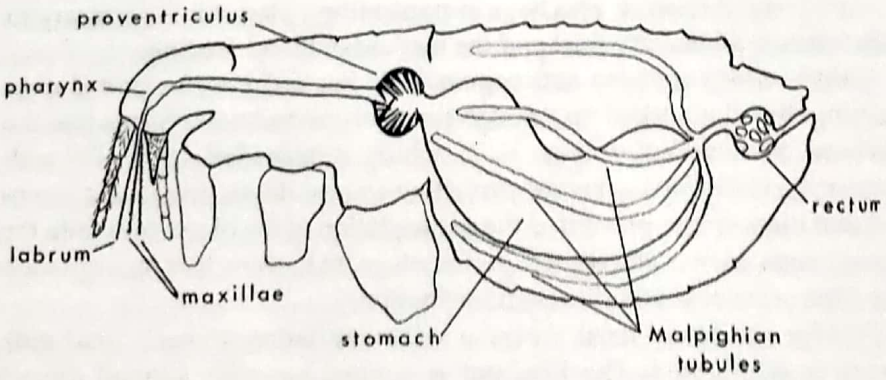


Figure 11.3 Diagrammatic representation of the alimentary canal of an adult flea, showing backward-projecting spines in the proventriculus.

11.2 Life cycle

Both sexes take blood-meals and are therefore equally important as disease vectors. The following account is a generalized description of the life cycle of fleas that feed on humans or animals, such as dogs, cats and commensal rats. The life cycle of the chigoe flea (*Tunga penetrans*) is described separately.

A gravid female rodent flea leaves the host and deposits her eggs in debris which accumulates in the host's dwelling place, such as rodent burrows. Fleas that bite humans or their domestic pets, such as cat fleas, lay their eggs while they are still on the host but because they are not sticky they soon fall off the host and are mainly found in areas where hosts, such as cats or dogs, spend the most time. Eggs are very small (0.1–0.5 mm), oval, white or yellowish and lack any visible pattern. Adults commonly live for 10 days to 6 weeks, but sometimes for 6–12 months or even longer. During her lifetime a female may lay 300–1000 eggs, mostly in batches of 3–25 a day.

Eggs usually hatch after 2–5 days, but this depends on the species of flea, temperature and humidity. A minute legless larva emerges from the egg (Fig. 11.4). It has a small brownish head with a pair of very small antennae, followed by 13 pale brown, distinct and more or less similar segments. Each ***segment*** has a circle of setae near the posterior border. The last segment ends in a pair of finger-like ventral processes termed ***anal struts***. The presence of these struts and setae on the body distinguish larval fleas from other insects of medical importance.

Larvae are very active. They avoid light, and shelter in cracks and crevices and amongst debris on floors of houses, or in nests or animal burrows. Occasionally, however, larvae are found on people who wear dirt-laden clothes, and sometimes in beds. Larvae feed on almost any organic debris, but to successfully achieve adulthood it seems that larvae

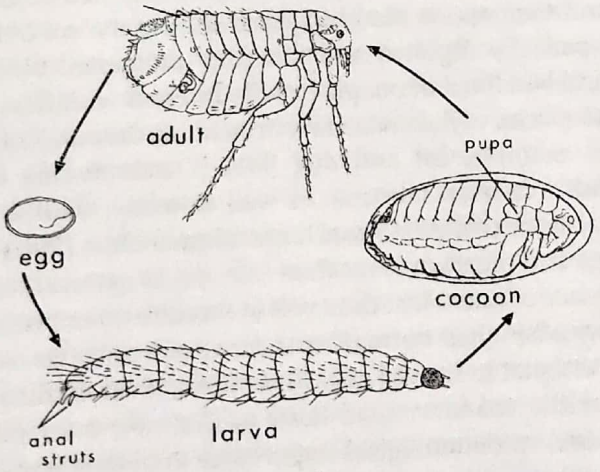


Figure 11.4 Life cycle of a flea.

of many species must consume partly digested blood evacuated from the alimentary canal of adult fleas (i.e. adult flea faeces). There are usually three larval instars, but in a few species there are only two instars (e.g. *Tunga penetrans*, page 185). The larval period is commonly 2–3 weeks, but varies according to species, and may be prolonged to 200 days or more by unfavourable conditions such as limited food supply and low temperatures. Mature larvae are 4–10 mm long. Unlike adult fleas, larvae die if relative humidities are either too low or too high.

At the end of the larval period the larva spins a whitish ***cocoon*** from silk produced by its salivary glands. Being sticky, it becomes covered with fine particles of dust and debris including sand picked up from the floor of the host's home. Consequently cocoons are difficult to distinguish from their surroundings. About 2–3 days after having spun a cocoon around itself the larva pupates within the cocoon. Adults are ready to emerge from the pupa after about 5–14 days, although this period depends on ambient temperature and also a stimulus. This is usually vibrations generated by a host's movements within its home, burrow or nest. If, however, animal shelters or houses are vacated adult fleas remain in their cocoons until their dwelling places are reoccupied. In some species, carbon dioxide emitted from hosts or a seasonal increase in humidity stimulates emergence. Adults may remain alive in their cocoons for ***4–12 months*** or sometimes for over a year. This explains why people moving into buildings which have been vacated for many months may suddenly be attacked by large numbers of newly emerged very bloodthirsty fleas seeking their first blood-meal.

The life cycle from egg to adult emergence may be as short as 2–3 weeks for certain species under optimum conditions, but frequently the life cycle is considerably longer, taking many months.

Fleas avoid light and are usually found sheltering amongst the hairs or feathers of their hosts, or on people under their clothing or even in beds. During feeding fleas eject faeces composed of semi-digested blood of the previous meal and then excess blood ingested during the act of feeding. This mixture of partially digested and virtually undigested blood often marks clothing and bed linen of people heavily infested with fleas.

Although most species of fleas have favourite hosts, they are not entirely host-specific. For example, cat and dog fleas (*Ctenocephalides felis* and *C. canis*) will readily feed on humans, as well as many wildlife species, especially in the absence of their normal hosts. Human fleas (*Pulex irritans*) often feed on dogs and pigs, while rat fleas (*Xenopsylla* species) will attack people in the absence of rats. Most fleas will in fact bite other hosts in their immediate vicinity when their normal hosts are absent or scarce. Although feeding on less acceptable hosts keeps fleas alive, their fertility can be reduced. Fleas rapidly abandon dead hosts to find new ones, behaviour which is of profound epidemiological importance in plague transmission. Some fleas can withstand both considerable ***desiccation*** and prolonged

periods of ***starvation***, for example six months or more, when no suitable hosts are present. However, cat and dog fleas die within 10 days away from their hosts. On a host, fleas move by rapidly crawling, whereas off the host they jump more than crawl in their search for new hosts. Some flea species can jump about 20 cm vertically and 30 cm or more horizontally. Such remarkable feats are achieved through a rubber-like protein called ***resilin***. This is very elastic and can become highly compressed; rapid expansion of the compressed state gives the power for jumping.

11.3 Medical importance

11.3.1 Flea nuisance

Although fleas can be important vectors of disease, the most widespread complaint is their troublesome bites, which may result in considerable discomfort and irritation. The most common nuisance flea is the cat flea (*Ctenocephalides felis*), which has a worldwide distribution. Females often lay up to 25 eggs a day for about a month. Of lesser importance as a pest is the dog flea (*Ctenocephalides canis*) and more rarely the human flea (*Pulex irritans*). The cat flea has become the most common flea on dogs.

Fleas frequently bite people on the ankles and legs, but at night a sleeping person may be bitten on any part of the body. People who become hypersensitive to flea bites can suffer from dermatitis, and inhalation of flea faeces can cause allergies. Children under 10 years tend to experience greater discomfort from flea bites than older people.

Because fleas are difficult to catch this increases the annoyance they cause. People attacked by fleas frequently spend sleepless nights alternately scratching themselves and trying to catch the fleas.

11.3.2 Plague

There are three main types of plague, bubonic, septicemic and pneumonic, all caused by the bacterium *Yersinia pestis*. Medically the most important is bubonic plague, of which there are worldwide about 1000–3000 cases annually in parts of Asia, northwestern and southern Africa, South America and western North America. Bubonic plague is a ***zoonosis***, being primarily a disease of wild animals, especially rodents. About 200 rodent species and 14 lagomorphs (e.g. hares and rabbits) have been shown to harbour plague bacilli. The transmission cycle of plague between wild rodents, such as gerbils, marmots, voles, chipmunks and ground squirrels, is termed ***sylvatic***, campestral, rural or enzootic plague. Many different species of fleas bite rodents and maintain plague transmission amongst them. When people such as fur trappers and hunters handle these wild animals there is the risk that they will get bitten by rodent fleas and become infected with plague.

An important form of plague is ***urban plague***. This describes the situation when plague circulating among wild rodents has been transmitted to commensal rats (e.g. the black rat, *Rattus rattus* and brown (Norwegian) rat, *R. norvegicus*). It is maintained in the rat population by fleas such as *Xenopsylla cheopis* (Europe, Asia, Africa and the Americas), *X. astia* (Southeast Asia) and *X. brasiliensis* (Africa, South America and India). When rats are living in close association with people, such as in rat-infested slums, fleas normally feeding on rats may bite humans. This commonly happens when rats are infected with plague and rapidly develop an acute and fatal septicaemia. On their death infected fleas leave the rats and feed on humans. In this way bubonic plague is spread by rat fleas to human populations.

The most important vector is *Xenopsylla cheopis*, but other fleas such as *X. astia* and *X. brasiliensis* are also plague vectors in some areas. More rarely *Nosopsyllus fasciatus* and *Leptopsylla aethiopica* can be vectors, but these two flea species are reluctant to feed on people and so rarely transmit plague to humans. In addition to humans becoming infected by the bites of fleas that previously fed on infected rats, plague can also be spread from person to person by fleas, such as *Xenopsylla* species and *Pulex irritans*, feeding on a plague victim then on another person. This latter method, however, appears to play a minor role in transmission. *Pulex irritans* may play a more important role in transmission than previously considered, especially in areas not having *X. cheopis*, but it seems that transmission is mainly mechanical, that is through contaminated flea mouthparts.

It is important to understand the methods by which fleas transmit plague. Plague bacilli sucked up by male and female fleas during blood-feeding are passed to the stomach, where they multiply greatly and extend forwards to invade the ***proventriculus***. In some species, especially those in the genus *Xenopsylla*, further multiplication in the proventriculus may result in it becoming partially, or more or less completely, ***blocked*** (Fig. 11.5). In a partially blocked flea, because the proventriculus is not

Figure 11.5 Development of *Yersinia pestis* in *Xenopsylla cheopis*: (a) early stage with proventriculus functioning normally; (b) partial blocking, with proventriculus failing to form an efficient valve mechanism; (c) completely blocked flea, with blood not being able to enter the stomach. (Courtesy of Miss M. A. Johnson, and Blackwell Publishing, Oxford, publishers of *Entomology for Students of Medicine* (1962) by R. M. Gordon and M. M. J. Lavoipierre.)

functioning normally, some of the blood that has been sucked into the stomach is regurgitated along with plague bacilli into the host. With completely blocked fleas, blood is sucked up from a host with considerable difficulty about as far as the proventriculus, where it mixes with plague bacilli and is then regurgitated back into the host. Blocked fleas soon become starved and repeatedly bite in attempts to get a blood-meal, and therefore are epidemiologically potentially very dangerous.

Another, but less important, method of infection is by the flea's faeces being rubbed into abrasions in the skin or coming into contact with mucous membranes. Plague bacilli can remain infective in flea faeces for as long as three years. Occasionally the tonsils become infected with plague bacilli due to crushing infected fleas between the teeth!

In septicaemic plague the bacilli are in the blood, and in pneumonic plague they invade the lungs. Very occasionally septicaemic plague is transmitted mechanically by fleas.

11.3.3 Murine typhus

Although murine typhus, also known as endemic typhus or Mexican typhus, occurs almost worldwide, the annual number of human cases has fallen from more than 5000 in 1945 and 1946 to presently just 20–80 cases a year.

Murine typhus is caused by the bacterium *Rickettsia typhi*, which is ingested by a flea with its blood-meal. In the gut the rickettsiae multiply, but unlike plague bacilli they do not block the proventriculus. Transmission occurs when infected ***faeces*** are scratched or rubbed into abrasions or come into contact with delicate mucous membranes, and also by the release of rickettsiae from crushed fleas. Faeces remain *infective* for many months to a year or more; under laboratory conditions they have remained infective for 4.5–9 years! Murine typhus is essentially a disease of rodents, particularly rats such as *Rattus rattus* and *R. norvegicus*. It is spread among rats and other rodents by *Xenopsylla* species, especially *X. cheopis*, but also by *Nosopsyllus fasciatus* and *Leptopsylla segnis*. A few ectoparasites which are not fleas are vectors, such as the spined rat louse (*Polyplax spinulosa*) and possibly the cosmotropical rat mite (*Ornithonyssus bacoti*).

People become infected mainly by the faeces of *Xenopsylla cheopis*, but occasionally species such as *Nosopsyllus fasciatus*, *Ctenocephalides canis*, *C. felis* and *Pulex irritans* may be involved in transmission. *Leptopsylla segnis* does not bite humans, but it is possible that murine typhus is sometimes spread to people by an aerosol of this flea's infective faeces.

Rickettsiae of murine typhus can pass to the flea's ovaries and subsequently to the eggs, larvae and adults, that is ***transovarial*** transmission. But whether this is epidemiologically important remains uncertain.

Rickettsia felis was first detected as a human pathogen in 1994, but it is very similar to *R. typhi*; in fact polymerase chain reaction (PCR) techniques

are required to separate these two species, so infections of *R. felis* may have often been missed. *Rickettsia felis* has been isolated in the USA from opossums (*Didelphis* species) and cat fleas (*C. felis*) which feed on them. It seems there is an urban type of murine typhus involving *R. typhi*, rats and rat fleas (described above) and a rural type involving *R. felis*, opossums and cat fleas which can also be transmitted to humans. The infection can be maintained in cat fleas by vertical transmission for at least 12 generations.

11.3.4 Cestodes

Dipylidium caninum is the commonest tapeworm of dogs and cats, and it occasionally occurs in children. It can be transmitted by fleas (*C. felis*, *C. canis* and *P. irritans*) to both pets and humans as follows. Tapeworm proglottids containing eggs excreted by a pet crawl away from the host and dry on exposure to air. Larval fleas feeding on organic debris in host bedding bite into the dried proglottids, releasing the eggs, which they then swallow. Larval worms hatching from the ingested eggs penetrate the gut wall of the larval flea and enter the body cavity (coelom). They remain trapped here before passing to the pupa and finally to the adult flea, where they encapsulate and become cysticercoids (infective larvae). Animals become infected by licking their coats during grooming and swallowing infected adult fleas. Similarly, young children fondling and kissing dogs and cats can become infected by swallowing cat and dog fleas, or by being licked by dogs which have crushed infected fleas in their mouths, thus liberating the infective cysticercoids.

The rat tapeworms *Hymenolepis diminuta* and *H. nana* have similar life cycles.

11.3.5 Less important diseases

Cat-scratch disease (CSD) caused by *Bartonella henselae* is transmitted among cats by cat fleas. It seems that cats' claws are contaminated with *Bartonella*-infected flea faeces and that transmission to humans is mainly by cat scratches. Tularaemia (*Francisella tularensis*) may occasionally be transmitted to humans by flea bites. Ticks, however, are the main vectors, and it must be stressed that the role of fleas as vectors of these two infections is minimal.

11.4 *Tunga penetrans*

Tunga penetrans is found in the Caribbean, Central and South America and sub-Saharan Africa including Madagascar. It is increasingly found in travellers returning home from tropical countries. *Tunga penetrans* is sometimes referred to as the chigoe, jigger flea or sand flea. *Tunga penetrans* does not transmit any disease but is a nuisance because females become imbedded in the skin.

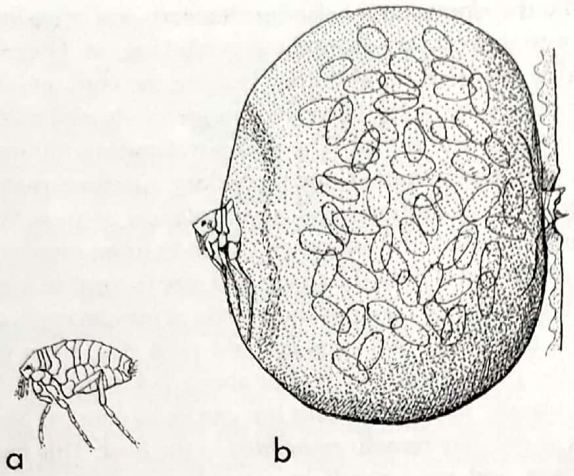


Figure 11.6 Adults of *Tunga penetrans* (chigoe flea): (a) non-gravid female; (b) gravid female with enormously swollen abdomen full of eggs which is embedded in the skin of a host. Note (on right) that the tip of the abdomen projects from the host's skin to the exterior.

11.4.1 External morphology

Adults of both sexes are exceedingly small, only about 1 mm long (Fig. 11.6a, Plate 20). They have ***neither*** genal nor pronotal combs and are easily separated from other fleas of medical importance by their very ***compressed*** first three (thoracic) segments, and by the paucity of spines and bristles on the body.

11.4.2 Life cycle and medical importance

Eggs of *Tunga penetrans* are dropped onto the floor of houses or on the ground outside, and hatch in about 3–4 days. Larvae inhabit dirty and dusty floors or dry sandy soils, especially in areas frequented by hosts of the adult fleas. There are two instars, not three as in most flea species, and under favourable conditions larval development is completed within about 10–14 days; the pupal period lasts about 5–14 days.

Newly emerged adults jump and crawl on the ground until they locate a host, usually a person or pig. ***Both sexes*** feed on blood, but whereas the male soon leaves the host after taking a blood-meal, the female, after being fertilized, 'burrows' into the skin where it is soft, such as between the toes or under toenails. Strictly speaking she does not burrow, but the host's skin envelops her. Other areas of the foot, including the sole, may also be invaded. In people habitually sitting on the ground, such as beggars or infants, the buttocks may be infested, and particularly large infestations have been recorded from leprosy patients. In heavily infested individuals

the arms, especially the elbows, may also be attacked, and occasionally the females 'burrow' into the soft skin around the genital region. The entire flea, with the exception of the tip of the abdomen bearing the anus, genital opening and large respiratory spiracles, becomes ***completely buried*** in the host's skin, and here she continues to feed. The area surrounding the embedded flea becomes very itchy and inflamed, and secondary infections may become established, resulting in ulcerations and accumulation of pus. While the blood-meal is being digested, the abdomen distends to an enormous size, and after 8–10 days the flea is both the shape and size (6 mm) of a small pea (Fig. 11.6b). The abdomen now contains thousands of minute eggs, and over the next 7–14 days about 150–200 eggs are laid each day, most of which eventually fall to the ground and hatch after about 3–4 days. The life cycle from egg to adult usually takes 4–6 weeks but can be as short as 18 days.

After female fleas die they remain ***embedded*** in the host. This frequently causes inflammation and may result in secondary infections, which if ignored can lead to loss of the toes, tetanus, or even gangrene. Male fleas cause no such trouble, as they do not 'burrow' into the skin.

Chigoe fleas are most common in people not wearing shoes, such as children. Up to 100 lesions have been found in the feet of a single child in endemic areas, and there are commonly 40 or more lesions per person. Because these fleas are feeble jumpers, wearing shoes is a simple method of reducing the likelihood of flea infestation.

Females embedded in the skin can be removed with fine needles under aseptic conditions, and the wounds should then be treated with antibiotics and dressed. They are best removed within the first few days of their becoming established, because when they have greatly distended abdomens, containing numerous eggs, they are difficult to extract without rupturing them, and this increases the risk of infections. Surgical extractions, however, are not easy and it may be better to treat lesions topically with lotions of ivermectin or metrifonate (trichlorfon) on two consecutive days to kill fleas in situ.

Pigs, in addition to humans, are commonly infested with *Tunga*, and may provide a local reservoir of infestation. Other animals such as cats, dogs and rats are also readily attacked.

11.5 Control of fleas

Repellents such as DEET or permethrin-impregnated clothing may afford some personal protection against fleas.

Insecticide resistance has been reported in cat fleas, human fleas and *Xenopsylla* species to one or more of the following categories of insecticides: organochlorines, organophosphates, carbamates, pyrethrins and pyrethroids. Nevertheless, insecticides remain the main tool for flea control, although there is increasing reliance on insect growth regulators (IGRs).

11.5.1 Rodent fleas

Both adults and larvae of rodent fleas can be killed by using hand-operated dusters to apply insecticidal dusts such as carbamates, pyrethroids or organophosphates to rodent burrows and their nearby runways. Treatment with carbamates and pyrethroids can remain effective for 2–4 months in dry conditions. Rodent fleas in houses can be controlled by spraying floors with bendiocarb, malathion or pyrethroids, or fogging with permethrin or pirimiphos-methyl.

Controlling fleas in urban outbreaks of plague or murine typhus requires extensive and well-organized insecticidal operations. While insecticides are being applied, rodenticides formulated as baits, such as the anticoagulants warfarin and brodifacoum, can be used to kill rodents. If there is resistance to these rodenticides then the fast-acting anticoagulants such as bromadiolone and chlorophacinone can be used, but it is then ***essential*** to apply these several days after insecticidal applications. Otherwise rodents will be killed before their fleas are killed, and the fleas will then bite other mammals including people, which may result in increasing disease transmission. Where there is resistance to warfarin and other anticoagulants, calciferol, a fast-acting rodenticide, can often be substituted.

Although IGRs appear to have good potential for control of plague fleas, they have received relatively little evaluation.

11.5.2 Cat and dog fleas

Cat and dog fleas (*Ctenocephalides felis* and *C. canis*) can be detected by examining the fur on the neck or stomach of the hosts. Powders, sprays or spot-on concentrates of insecticides such as bendiocarb, pyrethroids and organophosphates and also IGRs can be applied to the animal's fur. Dusts are safer than liquids because they are less likely to be absorbed through the animal's skin and cause unpleasant side reactions. Increasingly IGRs such as pyriproxyfen, methoprene and lufenuron can be applied to the animal's skin or formulated as a pill to be given orally, or by injection. With IGRs fleas will still lay eggs but either they will fail to hatch or the emerging larvae will fail to develop. Such treatments may be effective for 3–6 weeks. Flea collars impregnated with insecticides or IGRs are not very effective.

However, an important consideration is that most fleas are found away from the host, not on it. Typically there may be only about 25 adult fleas on a cat, but on the floor and bedding, apart from a few adult fleas, there may be 500 cocoons and as many as 3000 larvae and 1000 eggs. Clearly, control measures should also be applied to beds, kennels and other places where pets sleep. These items should either be treated with insecticidal powders or lightly sprayed with malathion, chlorpyrifos, one of the pyrethoids or an IGR. Cocoons, however, will not be killed, although the emerging adults will be. Duration of effective control depends on the types of materials