SOME POINTS OF GENERAL INTEREST ON THE SUBJECT OF PLAGUE

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The endeavour in this paper is to place before members some points of general interest on the subject of plague.

The history of plague will first be briefly traced, then the root-cause of the disease, viz. *Bacillus pestis*, will be dealt with by tracing it through the laboratory and noting its vitality both inside and outside the living organisms of various animals, with particular reference to the principal host—the rat.

The next step will be to examine the life history, habits, and morphology of the connecting link between the rat and human beings, viz. the flea.

The final stage is to consider plague in its relation to man.

To summarise, the various steps are: the *Bacillus pestis*, the rat, the flea, and man.

Definition.—Let us first define our subject. Plague is an acute, infective, febrile disease, accompanied by inflammation of the lymphatic glands, and caused by a micro-organism, the *Bacillus pestis*.

History

In thinking of plague, some of us are apt to regard it as a comparatively new disease, or at any rate our thoughts do not carry us farther back than the Great Plague of London, in the year 1665. But plague has been with us from time immemorial. Thus we find a reference to it in the writings of a physician, Rufus of Ephesus, about A.D. 100, and he was referring to an outbreak in the third century before Christ. The Greeks also wrote of 'pestilential buboes,' and we meet it again in the Great Plague of Justinian, which started from Egypt in A.D. 542, and spread over a large part of Europe. At the end of the seventh century, bubonic plague was recorded

in Italy and in England. These epidemics were traceable for the most part to Egypt; but in the fourteenth century a new epidemic invaded Europe from Asia by way of the Crimea and the Black Sea, its origin being referred to Cathay or China. This terrible pestilence appeared in Southern Italy in 1346, and made its way over the whole of Europe. It affected England in 1348, and Scotland and Ireland did not escape. A second epidemic occurred in 1361, and a third in 1368. Many historians have dwelt on this great calamity, and Hecker calculates that 25,000,000 persons—i.e. about one-fourth of the population of Europe at that time—died of this disease. The first epidemic, known as the Black Death, and to which I have referred as starting in Southern Italy in 1346, was evidently one of what is now called pneumonic plague.

Successive epidemics occurred in Great Britain through the fifteenth, sixteenth, and seventeenth centuries. Finally they culminated in the Great Plague of London in 1665, in which about 70,000 persons died, and which extended widely over the country. Those of you who remember your history dates by the aid of rhymes will recall the couplet:

> In sixteen hundred and sixty-five,
> There was scarcely a man in London alive.

Soon after that date the disease vanished, helped no doubt by the cleansing action of the Great Fire of London, when

> In sixteen hundred and sixty-six,
> The Fire of London burnt the bricks,

though of course the fire was merely a local factor.

It did not revisit Britain till a localised epidemic occurred in 1900 in Glasgow.

During the first half of the nineteenth century, plague prevailed in Turkey, and made occasional advances into the countries round about the Danube. It finally left Europe in 1841, receding to the regions round about Persia, Kurdistan, and Mesopotamia.

Let us now try to trace its history to India. There were epidemics of plague in India from the eleventh to the end of the seventeenth century. In 1615 a great epidemic, which

lasted for eight years, affected the Punjab and Mohammedan India generally. From 1684 to 1707 an epidemic ravaged Bombay, Surat, and a great part of Western India. In 1836 an epidemic broke out in Rajputana, known as the 'Pali Plague,' from the name of the town in which it first occurred; and 'Maha Murrie,' which is undoubtedly bubonic plague, has recurred several times in the district of Gharwal in the south-west of the Himalayas since 1823.

In 1896 plague was introduced into Bombay from China, and has ravaged the country since that time. During the first ten years of the present outbreak in India, it accounted for a mortality of over 4,000,000 people.

China has probably had plague since time was. The first definitely known epidemic was in Yunnan in 1860; and from there by way of Pakhoi and Canton it went in 1894 to Hong-Kong, and from this great seaport it was carried by sea traffic to India, Australia, Japan, Europe, Africa, and America—North and South. In 1899 the plague crossed the Equator and appeared at Tamatavi in Madagascar, probably imported from Bombay. From Tamatavi it went across to Lorenzo Marques, and in the same year appeared at Fort Louis in Mauritius. In 1900 it appeared in Capetown, having been imported, in spite of quarantine precautions, from Rosario in South America. It spread to the city and to Port Elizabeth.

In 1902 it was recorded for the first time in Nairobi in this Protectorate. There were sixty-five cases and twenty-one deaths. Where this came from is not accurately known. Possible sources were Bombay, South Africa, or Uganda, which last is held to be an endemic centre of plague.

Bacteriology

Now let me introduce you to the *Bacillus pestis*. It was first discovered in the year 1894 by two independent investigators, viz.: Kitasako, who had been sent by the Japanese Government to investigate the plague outbreak at Hong-Kong, and Yersin, sent by the French Government for the same purpose.

Plague belongs to the group of haemorrhagic septicaemias,

which are diseases due to microbes which invade the blood stream acutely, and are found in large numbers over the whole body. The plague bacillus, as seen under the microscope in plague lesions, shows a considerable polymorphism. Three forms predominate: short oval rods, long rods, and large oval, pear-shaped or round involution or degenerate forms, which only take on stains faintly. In pure cultures all these forms are also met with. In staining the *B. pestis*, we find a peculiarity which separates it from most other bacilli, though not from all, and that is the phenomenon of what is known as ' polar staining.' The micro-organism appears stained at the ends, but not at all, or only slightly so, in the middle. This, for diagnostic purposes, is a striking and important point.

Now let us see how the bacteriologist would proceed to grow this bacillus. He would employ several methods. First he would take a little material from, say, the bubo, and streak it on a gelatinous substance made from seaweed, known as agar. If he added 2 to 3 per cent. of salt to this agar, he would find that the characteristic involution forms I have just described would be readily obtained ; this feature distinguishing it from other bacilli. He would next proceed to grow the bacillus in broth, especially in broth on which oil-drops were floating. After a time, he would find a growth as a powdery thread, in regular stalactite forms, hanging from the under surfaces of the oil-drops. This, again, to his mind, would form a characteristic and diagnostic feature. (I shall remind you later on of these stalactite forms when I am speaking on plague vaccines.)

You will wonder why the bacteriologist takes all this trouble. Are there, then, other bacilli so like plague in their forms, and in the way they grow on these cultures, as to be mistaken for it ? There are several. For instance, the bacilli that causes swine plague ; but in particular, one with a name far longer than itself, the *Bacillus pseudotuberculosis rodentium*, which is moderately pathogenic to rats, but not to human beings. This so closely resembles *B. pestis*, not only in microscopical but in cultural tests, that we have to resort to the cutaneous inoculation of *white* rats to differentiate it. We

find, then, that where *B. pestis* kills, the *B. pseudotuberculosis rodentium* is harmless to these animals.

Vitality of the Plague Bacillus outside the Body.—I have described to you now the form of the bacillus and how it is grown. Let us turn to the important point as to the degree of resistance to destruction which it manifests when outside the living body. In general the resistance is slight; but pure cultures, kept in the dark and prevented from drying, can live for months or even years, depending a good deal on the temperature at which they are kept. Otto was able to demonstrate that the plague bacillus taken from putrid cadavers of plague rats were alive up to sixty-one days, provided they were kept at as low a temperature as 6° C. In bubonic pus it has been found to survive for twenty days, and also for that length of time in fresh water, and in sea water for forty-seven days. In cow-dung it has been found to live for months when kept moist. You may perhaps be aware that the common custom in India is to plaster the sides of the walls and the floors of the houses with moist cow-dung, which is then allowed to dry. On such floors plague bacilli were found to remain infective for forty-eight hours.

The resistance of *B. pestis* to *drying* is not great. Thus, plague matter dried on wool, silk, linen, glass, wood, &c., died within six days. In dust it rapidly dies out; and in Hankin's experiments it was found to die out within thirteen days in grain and meal. Exposure to *light* causes rapid sterilisation of plague cultures—three to four hours sufficing. Dry heat at 160° C. kills in a minute. Cold, however, has very little effect on plague bacillus. It was found alive after forty days, after the severe treatment of freezing and thawing it daily. Chemical disinfectants rapidly and easily kill it. Thus, 1 per cent. carbolic acid or lysol will kill in ten minutes.

Pathogenic Action of B. pestis on Animals.—What happens when this bacillus gets inoculated into animals? And which are susceptible to its action? The most susceptible are all kinds of rats, also guinea-pigs, mice, monkeys, mongooses, squirrels, bats, jerboas, and marmots. Dogs, jackals, and hyenas are mostly unsusceptible. Cats are not highly so, and the bird family is immune.

All varieties of rats can be infected with the greatest ease, and Albrecht and Ghon, of the Austrian Plague Commission, have shown that by smearing plague material upon the intact shaven skin of a guinea-pig or rat, infection occurs. This is an important crucial test.

Rats can become infected when fed on plague cultures or on plague cadavers, and the German Commission gave it as their opinion that, under natural conditions, rats frequently infect themselves by gnawing the infected cadavers of other rats, and that they can also become infected through the unbroken nasal or conjunctival mucous membrane. Workers in India have also been able to infect rats by feeding them on plague material or on the cadavers of plague rats. It would be natural to conclude that, as the German Commission stated, rats in nature often become infected in this manner. But this is probably not the case, for the common site in naturally infected plague rats of the primary bubo is in the neck, where the fleas usually are most numerous ; whereas in experimentally infected animals, fed on the afore-mentioned material, buboes in the intestinal or mesenteric site are in the great majority. Thus out of 5000 post mortems on naturally infected plague rats not a single mesenteric bubo was seen.

Post Mortem Appearances.—Supposing we found a dead rat and suspected it of having died of plague. Apart from the microscope, what post mortem appearances would we expect to find ? Before examining, we would of course take precautions to rid the animal of fleas, if indeed these had not already deserted its cold carcass.

In dissecting the rat, we would find dark-red, subcutaneous injection of the flaps of the abdominal walls. There would be fluid in the pleural cavity, haemorrhagic swelling in the outer layers of the glands, and swelling of the neck-glands, and, in particular, a creamy mottled appearance of the liver. The spleen also would be found swollen, congested, and granular in appearance.

Kinds of Rats.—The kinds of rats that concern us mainly are those which we might describe as the domestic breed as opposed to the field variety. These are *Mus rattus*, the common house-rat, and *Mus decumanus*, otherwise known as

Mus norvegicus, the sewer-rat. The distinguishing features between these two are that in the sewer-rat, *M. norvegicus*, we have the ears barely reaching the eyes when laid forward, and the tail rather shorter than the length of the head and body together; whereas with *M. rattus*, the house-rat, the extended ear covers or reaches beyond the middle of the eye, it has a sharper nose, thinner ears, and a tail which is longer than the length of the head and body combined by 25 per cent. *Mus alexandrinus* is a variety of *M. rattus*, and the common mouse is known as *M. musculus*. In the United States the ground-squirrel, *Cytellus beechyi*, acts as a reservoir of plague, and has as its flea *Hoplopsyllus anomalus*.

Insects which have been implicated in the Spread of Plague.—The insects which have been specially studied in connection with plague are flies, bugs, mosquitos, pediculi, ants, cockroaches, and fleas.

They are of more or less importance in this connection in relation to their habits of sucking blood and of transferring themselves from one host to another, as, for example, the flea, biting first the rat and, later, man. Great interest also attaches to the problem as to what becomes of the plague bacillus in the alimentary canal of such insects, and especially whether it is capable of multiplying there, and either increasing or decreasing in virulence.

Flies have been proved to be capable of conveying the bacilli on their legs and thus infecting food material. Plague bacilli may remain virulent in the intestines of a fly for forty-eight hours.

Bugs were found to have plague bacilli in their intestines after sucking the blood of rats suffering from plague; but it was found impossible to transmit plague through the agency of these insects.

Mosquitos also have not been implicated.

Pediculi have been shown by the experiments of de Raadt, in 1915, to be capable of transmitting plague infection. Both the body-louse and the head-louse have been implicated.

Ants of a certain species (*Monomorium vastator*) can transmit plague, and they often succumb after feeding on plague rats. This is on the authority of Dr. Hankin.

Cockroaches may harbour plague bacilli and carry them for a considerable distance in their intestines. Dr. Hunter was able to recover cultures from faeces of these insects.

Fleas, however, are the most important of them all, and on them we shall dwell at somewhat greater length.

Life History of Fleas.—These undergo a complete metamorphosis. The adult female lays eggs, about the size of a small pin's head, on the ground. These are not attached to the fur of the host. One to five eggs are laid at a time, and they hatch in about two days in such a climate as that of Bombay, and it would probably be about the same time here. A larva issues from the egg, and in this stage the insect is very vulnerable. It has chewing or biting mouth-parts, and lives on any kind of animal or vegetable refuse. These larvae are remarkably long in the case of *Pulex cheopis*, the common rat flea, and comparatively short in *P. irritans*, the human flea. After about a week, the larva becomes sluggish, ceases to eat, and spins a cocoon of white silk-like fibres, and, enclosing itself in this, turns into a pupa. The surface of the cocoon is frequently covered over with dust or other small particles of rubbish which adhere to the fibres. In from seven to fourteen days the pupa turns into the perfect flea—the imago, and escapes from the cocoon. The mouth-parts of the flea are so constructed that it can only live on liquid food. Young fleas, which have just escaped from the cocoon, can live without food for one to two weeks, whereas, after having taken their first meal, older fleas die within a week if deprived of food: at least, this was the case in the Bombay experiments, and I will just pause here to warn you that we must be cautious in our generalisations. Things which occur in one country, and under a certain set of conditions, will not necessarily be the same in another land where the conditions are different.

The time taken from the egg to the imago or perfect flea is about twenty-one to twenty-two days, under favourable circumstances; but under unfavourable ones this period may be greatly prolonged. Climatic conditions affect the development of some species of fleas. Dampness in the surroundings of a breeding-place hinders the development, and adult fleas

dislike wetness. The breeding-places differ with the habits of the host. Thus, in the case of *Mus decumanus*, they are found in the burrows, while in the case of *M. rattus* in all sorts of situations which afford a shelter to these animals. Thus, on grain or gunny-bags. This, as a source of distribution of the flea, should be kept in mind.

Temperature has a decided effect on the breeding of fleas. A high mean temperature of about 90° F. not only seems to restrain the imago from depositing eggs, but is deleterious to the development of the eggs into larvae. There seems to be an optimum temperature at which breeding takes place more vigorously than at other temperatures. In Bombay this appeared to be from 75° to 80° F.

A French worker in Annam states that *P. cheopis* eggs do not hatch there under 55° F., and at 90° F. 75 per cent. of them remain sterile. He states that for the active larvae a certain amount of moisture and a still atmosphere are necessary. Badly ventilated houses, therefore, favour their development. He also states that the adult flea can live thirty-eight days without food, and that it lives for preference on the body of its host rather than in its bed, which makes it a far more dangerous agent in plague transaction.

Fleas have Particular Hosts.—Different kinds of fleas have their own hosts in particular. Thus *P. irritans* is natural to man as *P. cheopis* is to the rat. This does not mean that fleas will not attack other animals than their normal hosts: for instance, *P. felis* has been found on the dog, cat, tiger, panther, goat, horse, rat, hedgehog, kangaroo, deer, guinea-pig, rabbit, monkey, and on man. But, nevertheless, they prefer their natural hosts.

P. irritans, the human flea, does not thrive on the rat, and, when artificially placed on such a host, rapidly dies off. Yet it can be infective for at least four days after feeding on a plague rat. In some experiments, however, which were carried out in Bombay, it was found that the guinea-pig is as readily chosen by *P. cheopis* as its true host the rat. When many rat fleas are present, some of them will attack man, even when a rat is available for their food supply. Conversely, when the number is small, they will not desert their true host

for man. Rat fleas, when starved, will readily attack all animals, not then being particular in their choice of host. Such fleas, deprived of food from seventy-two to ninety-six hours, attack and feed on man more readily than at other times; but even so, they prefer their true host to man. A point to be remembered is that rat fleas may be attracted to man, jump on him, but take some time to feed; and thus a man may carry fleas from one place to another which when brought near a rat will attack it in preference to the man.

Mode of Dispersal of Fleas.—I have already indicated one way in which fleas may be carried from one vicinity to another. Other modes are:

(*a*) By means of the host in its natural wanderings. The flea does not stay continually on its host, but often drops off; thus rats are continually shedding fleas wherever they go. Also remember that sick rats harbour a greater number of fleas than healthy ones.

(*b*) When the rat is carried about in merchandise, fleas accompany him.

(*c*) But in the absence of the rat, fleas themselves may travel in merchandise, grain, or clothing. However, it must be remembered that the adult flea, in the absence of any host, will rapidly die, generally in about four or five days. Still, the larvae, which can feed on any kind of organic rubbish, and the pupae, which require no food, can be carried considerable distances in merchandise, i.e. for periods as long as one or two months.

In whatever way transported, they will select, when reaching their new surroundings, their true host or the next best available animal.

In numbers rat fleas seem to have a distinct seasonal variation. Moreover, this appears to coincide with the height of the plague epidemic more or less closely. Also during the epidemic season of plague, fleas remain infective for a period of from ten to fifteen days; whereas during the non-epidemic season they are only infective for seven days, and then not so highly.

The Collection and Examination of Fleas.—How are we to

catch our fleas for examination ? The simplest way is by means of chloroform or Keating's powder. If collecting from a rat, put the rat into a large glass bottle, on the bottom of which a little chloroform has been dropped. The fleas become anaesthetised and fall to the bottom of the jar, or remain entangled in the hairs. As soon as the rat becomes anaesthetised it may be removed from the bottle and the fleas picked off it and placed in a test-tube. They rapidly recover from the anaesthesia, and can be used for experimental purposes. I would like to warn you here, if you wish to collect fleas from a caged rat, not to expose the cage to sunlight; for fleas dislike the sun intensely, and will leave their hosts to seek the shade. This is a point also which I would call to the notice of those who are catching rats on a large scale in their endeavours to prevent the spread of plague. If these cages are carried for any distance in the sunlight through a town, they will inevitably be shedding their fleas on the way. *Verbum sapienti.*

Another method is by the use of animal traps. Thus, should we desire to catch *P. cheopis* in a room, a guinea-pig or man would serve as a suitable trap. The guinea-pig is allowed to wander about the floor of the room and soon gathers up all the fleas present. If the experiment takes place at night in a rat-infested room, the bag will be a large one.

Still another method might be by the use of fly-paper; the ordinary Tanglefoot paper is very suitable. Put a suitable animal in a cage, and all around it for a depth of at least 6 inches, place Tanglefoot paper. The flea's maximum hop is about $4\frac{1}{2}$ inches. Attracted by the animal, the flea will endeavour to reach it, and be caught in the process. For examination, pick it off and wash in alcohol.

Examination of Fleas.—Now, having caught our fleas, we proceed to examine them. Living fleas may best be examined while under the influence of chloroform, and $\frac{1}{4}$-inch objective of an ordinary microscope is of sufficient power for identification. For examining dead specimens, dissection is employed, and, to facilitate this, fleas may be boiled for a short time in caustic soda or potash, or allowed to soak in water at 80° F. for twenty-four to forty-eight hours. The soft parts of the

body disintegrate, allowing the harder shell, which is composed of chitin, to be separated into segments. Microscopical specimens can be prepared by boiling the fleas in glycerine and mounting them in that medium, or they may be boiled in alcohol, cleared in cedar-wood oil, and mounted in Canada balsam.

The Process by which the Flea transfers Infection.—Now let us introduce the *Bacillus pestis* into the flea—or rather he will do that himself, given the chance. The average capacity of a rat flea's stomach is about 0·5 c.mm., and on this basis a flea may take in as many as 5000 plague germs into his stomach after imbibing the blood of a plague rat. Moreover, multiplication of plague bacilli takes place in the stomach of the rat flea. This multiplication varies with the season of the year. In the epidemic season it is six times greater than in the non-epidemic. Plague bacilli are present in the rectum and faeces of fleas taken from plague rats, and such faeces are infective to guinea-pigs, both by the cutaneous and subcutaneous methods of inoculation. Plague bacilli have been found in the oesophagus of the flea, but never in any other region of the body, such as the body cavity or salivary glands. A single flea *may* transmit the disease, and both male and female fleas can do so.

Two modes of transmission have been proved. (1) The flea defaecates while he is yet sucking blood, and the rubbing of this material into the puncture brings about infection. (2) Regurgitation, as the result of masses of plague bacilli in the oesophagus of the flea, causes injection of plague bacilli into the rat or man in the act of biting, and this is the more important mode.

Experiments showing that the Flea is the Transmitting Agent of Infection.—1. Certain experiments which were carried out in godowns in Bombay went to prove that close and continuous contact of plague-infected animals with healthy animals, if fleas are excluded, does not give rise to an epizootic among the latter. As the godowns were never cleaned out during the experiments, close contact included contact with faeces and urine, and the eating of food contaminated with these materials.

2. When fleas are present, the epizootic, if it does start, varies in severity and rate of progress according to the season, and this season corresponds to that of the plague epidemic.

3. An epizootic of plague may occur in a godown containing infected fleas without direct contact of healthy animals and infected animals.

4. In an infected godown, the infection is effective in proportion as the test animals are accessible to fleas.

5. Infection can take place without any contact with contaminated soil.

6. The experiments exclude aerial infection.

7. The general conclusion was that fleas, and fleas alone, were the transmitting agents of infection.

Construction of Godowns.—These were six in number, and were built in a row. The walls were of brick and mortar and the floor of cement, and so were rat proof. The rooms were of the same size, and the essential difference was in the construction of the roofs.

In the case of rooms Nos. 1 and 2, the roof was of ordinary, semi-cylindrical country tiles, which afford a very good harbourage to rats.

Immediately underneath this roof was a wire netting, carried on a wooden framework, which was let into the walls with cement; the idea being to allow a free passage to the rat flea, but not to the rat.

The roofs of rooms Nos. 3 and 4 were similar, but built with the flat Mangalore tiles. These, though they afford harbourage to rats, do not do so to anything like the same extent as the country tiles.

The third set of two rooms had corrugated iron roofing, which should have been rat and flea proof. As a matter of fact they had to make these last rooms with concrete, as they found that somehow or other fleas did actually find their way through the corrugated iron roof—probably by way of cracks in the cement.

The difference in the construction of the roofs is of such a nature that the natural supply of fleas, depending as it does on the number of rats in the roofs, varies in the different

go-downs. In Nos. 1 and 2 they are abundant and regular, in Nos. 3 and 4 they are scanty, whereas in Nos. 5 and 6 there should be none at all.

Differentiation of Fleas.—The fleas which are of main interest to us are those which are met with in association with *P. cheopis* on rats, and from which we might require to differentiate them. These are :—

1. *Pulex irritans*, the human flea.
2. *Ceratophyllus fasciatus*, the European rat flea.

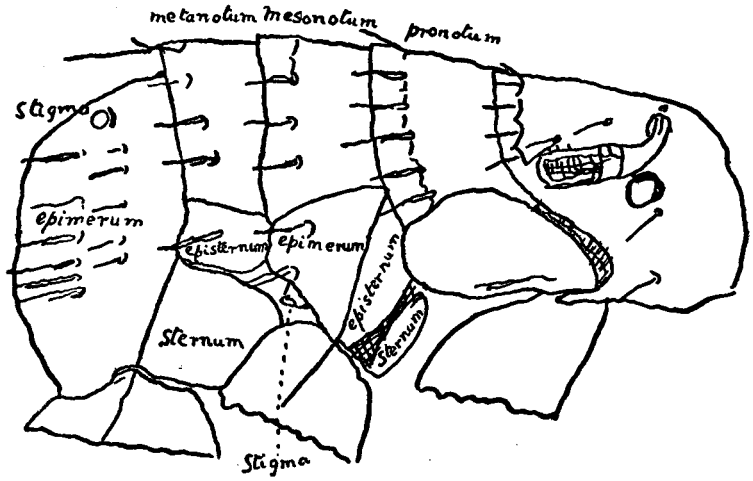

FIG. 1.—*Pariodontis riggenbachi* ♂.

3. *Pulex felis*, the cat and dog flea.
4. *Ctenopsylla musculi*, found on mice and rats.
5. *Sarcopsylla gallinacea*, a common bird flea.

The varieties of fleas are very numerous. Thus, *Loemopsylla*, to which genus *Pulex cheopis* belongs, has alone twenty-four known varieties. Still, as *P. cheopis* comprises 98 per cent. of the flea population on rats in India, and as it was found to comprise 66 per cent. of them during the epidemic of 1909, in the Muanza district, other kinds of fleas on rats, though possibly capable of transmitting plague, are not of such practical importance.

In Europe, the common rat-flea is the *Ceratophyllus fasciatus*, which occupies in point of numbers a similar position to *P. cheopis* in tropical climates.

Now to point out the differences between those I have mentioned. They have all well-developed eyes, with the exception of *Ctenopsylla musculi*. Of the remainder, *Ceratophyllus fasciatus* and *P. felis* have a prothoracic comb of bristles and the latter also a perioral comb, which are absent in *P. cheopis*, *P. irritans*, and *Sarcopsylla gallinacea*. The

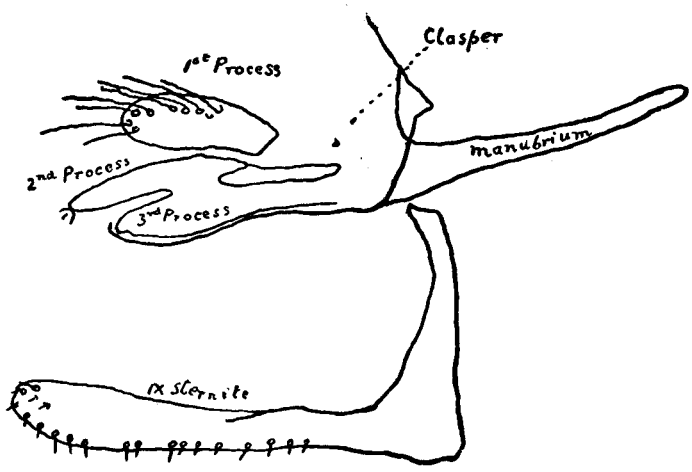

FIG. 2.—*Loemopsylla cheopis.*

last named can readily be distinguished from the other two by its angular fish-shaped head and its largely developed mandibles. We are left now with the two important ones, *P. cheopis* and *P. irritans*.

The plague flea has been given various names by workers who thought they were dealing with new varieties. Thus Taraboschi in Italy called it *P. murinus*, Tidswell in Sydney *P. pallidus*, and Herzog in Manila *P. philippinensis*. Rothschild identified all these as *P. cheopis* and *P. irritans*.

These may be differentiated from each other as follows:—

1. *P. cheopis* is small and more yellow than brown in colour when compared with *P. irritans*. It was this feature that led Tidswell in Sydney to call it *P. pallidus*, thinking he was dealing with a new variety.

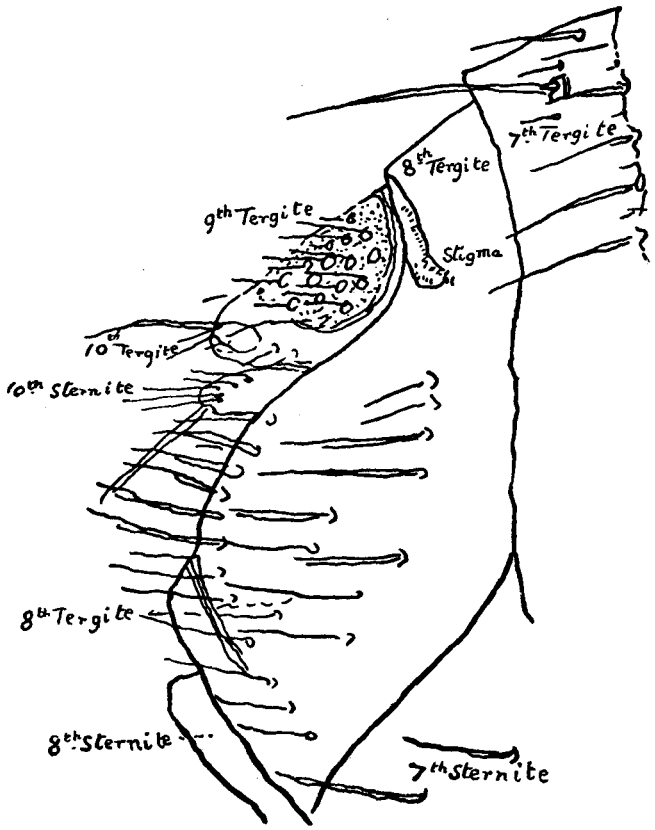

FIG. 3.—*Loemopsylla cheopis* ♀.

2. The number of bristles on the head is greater in *P. cheopis* than in *P. irritans*, and the ocular bristle in *cheopis* is situated nearly on a level with the upper border of the eye; while in *P. irritans* it arises nearer to the lower margin.

3. The antipygidial bristle in *P. cheopis* is longer than that of *P. irritans*.

4. The claws in *P. cheopis* are small in comparison with those of *P. irritans*.

5. Two mandibles slenderer and longer than in *P. irritans*.

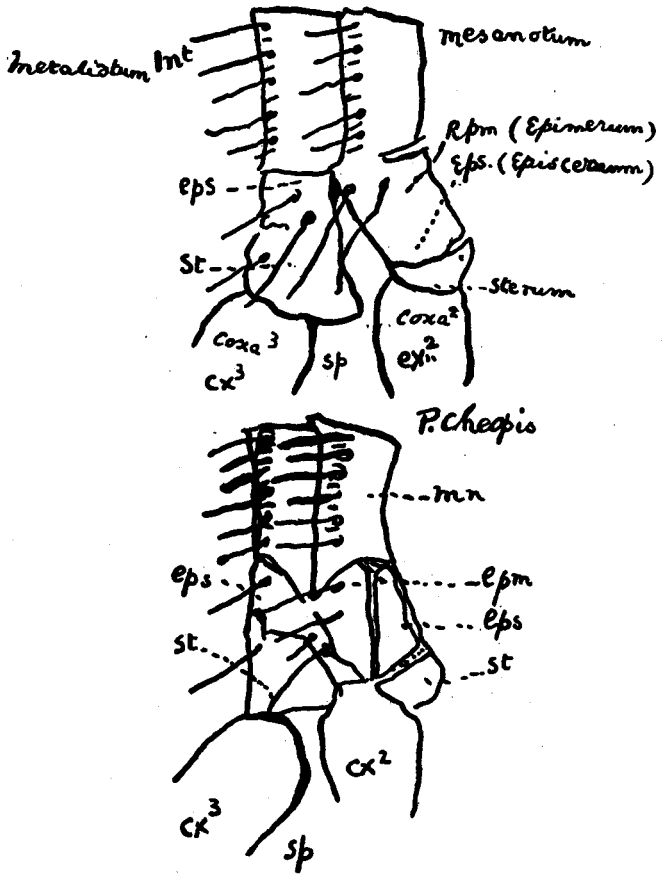

FIG. 4.—Mesa- and metathorax of *P. irritans*.

Another flea which might be noticed in passing is the *Pygiosylla ahalae*, which is the outdoor flea, commonly found on field-rats. It is said to be unimportant in regard to the spread of plague from one native dwelling to another; but as the house- and field-rat come into fairly intimate connection

in the neighbourhood of dwellings, this statement needs confirmation.

Distinguishing Characters of Genus Loemopsylla

1. Four segmented labial palps.
2. Closed antennal groove.
3. Anteriorly solid antennal club.
4. Position of dorsal apical bristle of the seventh abdominal tergite remote from the edge of the segment.
5. Presence of short spines on the inner surface of the hind coxa.
6. The division of the rodlike incrassation inside the mid coxa taking place near the base.
7. The structure of the modified abdominal segments.
8. Two mandibles slenderer and longer than in *P. irritans*.

Genus Pulex. Species—one only. P. irritans

1. Chief character, greatly reduced thorax. Mesosternite (i.e. sternum) highly specialised, narrow, strongly oblique, and lacks the internal cariniform incrassation found in other fleas in compensation. The anterior ventral portion of the mesosternite is much strengthened inside.

2. Male genitalia like *Sarcopsyllidae*, and not in other species, i.e. second and third processes of the claspers form a kind of claw.

3. Bristle in front of eye, in most other species, is absent in *P. irritans* and is replaced by one below the eye.

4. Hind coxa is distinguished from that of any known flea by bearing a number of hairs on the inner surface of the posterior (meral) portion.

5. Has a small tooth (often absent) at the genal edge of the head slightly behind the lower oral corner. Genal portion —below eye from oral edge to antennal groove.

Human Plague

We will now shortly glance at plague as it affects man. There are two main forms of plague: *Pestis minor*, which is a

mild form of bubonic plague, and *Pestis major*, which is a severe form. The latter is further subdivided into a number of varieties. Where the plague bacilli are found chiefly in the glands, we call it bubonic plague; when in the lungs, pneumonic plague; when as a general septicaemia, septicaemia plague. But it must be remembered that all forms have a common cause—the *Bacillus pestis*.

Pestis major.—The incubation period in *Pestis major* is generally from three to five days. It may be more. The onset is sudden, and the patient may suffer from headache, giddiness, and a staggering gait. He soon gets a thick, slow mode of speech. The temperature rapidly rises from 102° to 104° F., and even higher. The pulse also is rapid, from 90 to 130; the normal rate being about 75. The temperature lasts two to five days. The lymphatic glands are enlarged, and in the majority of cases those in the groins are affected. The enlargement of these may indeed be the first symptom noted; but usually they occur after the commencement of the fever. They are very painful, and at first very hard. If they go on to suppuration, it is considered a favourable sign; but if, on the other hand, rapid softening, flattening, or disappearance of the bubo takes place during the height of the fever, it generally means that the patient will succumb. In severe cases haemorrhage from various organs may be observed: thus bleeding from the nose or lungs may be seen; and the latter is characteristic of the pneumonia variety of the disease.

The duration of the attack is usually from three to five days, in fatal cases; but it may be very much shorter than this, even to sudden death.

The mortality in plague varies. During the height of the epidemic it is often 80 to 90 per cent. In India the general case mortality has been 70 to 85 per cent. In Hong-Kong it was 89 to 96 per cent. in Chinese; but was less among the Indians there, 77 per cent., and still less among the Japanese, 60 per cent. In South Africa it was 66 per cent. for the coloured population. Among Europeans it appears to be much less: thus in Hong-Kong it was only 34 per cent., in Bombay 30 to 40 per cent., and in Capetown 33 per cent. The percentage of deaths may tail off at the end of the epidemic season.

The pneumonic and septicaemic varieties of plague are extremely fatal forms. Unlike the bubonic form, pneumonic plague can easily be conveyed from one person to another without the intervention of fleas. In coughing, a pneumonic plague patient is showering his neighbourhood with millions of germs, and an attendant accidentally receiving some of this material on the mucous membrane of the mouth, or the conjunctiva of the eyes, may contract plague.

The treatment of plague, so far, has not produced any specific. Most drugs have been tried, but with disappointing results. Anti-plague serum has also been disappointing in producing curative results.

On the other hand, from a prophylactic point of view, we can produce a very considerable degree of active immunisation by the inoculation of cultures of plague bacilli or its products. The chief of these is Haffkine's Vaccine, of which all of you have personal acquaintance. Plague bacilli are prepared in broth cultures, in the stalactite forms, to which I called your attention in the earlier part of this paper. These are cultivated for five to six weeks, and, when pure cultures are obtained they are killed by raising the temperature to 65° C. for an hour, and, as an additional precaution, a small quantity of lysol is added. Of this preparation, from 0·5 to 4 c.c. are injected, according to the age and size of the individual treated. Susceptibility to plague is reduced by about one-fourth, and of those attacked after previous inoculation the mortality is only about one-fourth of what it is among the non-inoculated.

We have not yet got accurate knowledge as to the length of time this inoculation confers protection: it probably is for a year, and possibly even longer.

Preventive Measures

Now that you have learnt what plague is and how it is spread, you will readily see that preventive measures must be directed against the rat and the rat-flea.

The United States Public Health Service have outlined such measures in a very succinct form.

Scheme based on the Experience of the U.S. Public Health Service

- **General Measures**
  - **Survey**
    - (*a*) Plague laboratory
      - Examination of rodents.
      - Examination of human plague suspects.
    - (*b*) Delineation of infected areas
      - Rodent examination.
      - Epidemiology of human cases.
      - 'Sentinel' guinea-pigs.
  - **Eradicative**
    - Rodent destruction
      - Trapping.
      - Poisoning.
      - Miscellaneous.
    - Rat-proofing
      - Protection of food supply.
      - Elimination of harbourage.
  - **Restrictive**
    - Fumigation SO_2, CO, HCN
      - Ships.
      - Cargo or railway freight.
      - Baggage.
    - Rat proofing
      - Railway carriages.
      - Railway stations and freight warehouses.
- **Special measures (applied to foci)**
  - Evacuation
    - Fumigation HCN, SO_2.
  - Intensive rodent destruction
    - Immediate removal of harbourage.
  - Flea destruction
    - Fumigation.
    - Coal-oil emulsion.
    - General cleanliness.
    - Attention to household pets (cats and dogs).

INSECTS AND THEIR RELATION TO SOME DISEASES OF STOCK

LECTURE TO MEMBERS. BY R. E. MONTGOMERY

When I accepted the invitation of our energetic Secretary to offer for discussion before the Society a paper on Insects and their Relation to some Diseases of Stock, I did not quite realise the enormity of the task imposed.

Many people, when they see or hear of natural history,