Chapter 6

HOSTS OF THE INFECTION

RODENTS AND LAGOMORPHA

Reviewing in 1928 the then rather limited knowledge available concerning the occurrence and importance of plague in rodents other than the common rats and mice, Jorge [89] felt justified in drawing a clear-cut distinction between the pandemic type of plague introduced into human settlements and houses all over the world by the " domestic " rats and mice, and " peste selvatique ", which is dangerous for man only when he invades the remote endemic foci populated by wild rodents.

Although Jorge's concept was accepted, some discussion arose regarding the appropriateness of the term " peste selvatique " or, as Stallybrass [192] and Wu Lien-teh [215] translated it, " selvatic plague ". It was pointed out by Meyer [128] that, on etymological grounds, the name " sylvatic plague " would be preferable, and this term was widely used until Pozzo [158] and Hoekenga [74] doubted, and Girard [64] denied, its adequacy on the grounds that the word " sylvatic " implied that the rodents concerned lived in forests, whereas that was rarely the case. Girard therefore advocated the reversion to the expression " wild-rodent plague " which was used before the publication of Jorge's study—a proposal it has seemed advisable to accept for this monograph.[a]

Much more important than the difficulty of adopting an adequate nomenclature is that of distinguishing between rat and wild-rodent plague—a distinction which is no longer as clear-cut as Jorge was entitled to assume. Since 1928, the list of rodents other than the commensal rats and mice known to suffer from natural plague has grown incessantly so that, instead of little more than a score, almost 200 species or subspecies are now known to be implicated. Besides varying in their etiological importance, they

[a] It is important to note, however, that—as has been pointed out by an erudite reviewer of the above passage [205]—" the Latin name *sylvaticus*, when applied to animals, actually means wild rather than forestal and the names derived from it in Spanish, Italian and Portuguese convey the same meaning so that Jorge's nomenclature is fully justified on etymological grounds, though as it is likely to mislead English and French readers, it may be preferable to use the name wild-rodent plague ".

show marked differences in their ecology; many live near to man rather than in the remote haunts of sylvatic plague, and thus become apt to take part in the perpetuation and spread of the infection in settlements and even houses. It follows that it is no longer possible to deal merely with wild-rodent and rat plague as was done in the past; attention must also be paid to those species other than the commensal rats and mice which, on account of their peri-domestic or semi-domestic habits, take an intermediate position and are, in part, responsible for a transition of the infection from the wild to the commensal rodents, or vice versa.

Wild Rodents [b]

When trying to trace the gradual evolution of present-day knowledge on the existence and importance of wild-rodent plague, it is not surprising to find the earliest evidence in Central Asia where the infection probably originated and where it has certainly existed since time immemorial.

As first stated by laymen such as Tsherkasoff—in his *Memories of a hunter in Siberia* (1856-63)—in the case of Transbaikalia and Outer Mongolia, and Prjevalski [160] for the Ordos country of Inner Mongolia, and later confirmed by medical men, e.g., Rudenko,[172] Skchivan,[187] and Barykin,[14] the inhabitants of these areas had been aware for generations of the periodical occurrence of a fatal, infectious disease in the tarabagans (Siberian marmots) which was apt to spread to man, and had taken surprisingly adequate measures to protect themselves against this danger. The mysterious illness of the marmots was not only the subject of numerous legends, but was also referred to in the old Tibetan sacred books. In the opinion of the local practitioners, small worms invisible to the naked eye caused this and other infectious diseases, a claim curiously similar to that arrived at in 1658 by Athanasius Kircher [94] with regard to human plague.

The infection prevailing among the tarabagans, and among the human beings who had come in contact with them, seems to have been first identified with plague in 1895 by Bjeliavski & Rjeshetnikoff,[17] probably because their attention had been attracted to this disease by its spectacular appearance at Canton and Hong Kong in 1894. However, the evidence brought forward by these two observers was not supported by laboratory examinations and it was not until 1905 that bacteriological proof of the existence of human plague in Transbaikalia was obtained.[215] Two years later, a tarabagan dissected by Barykin [14] was also found positive, but it was only in 1921 that the existence of widespread epizootics among these animals was confirmed (Sukneff and others, quoted by Wu Lien-teh [215]).

These records leave little room for doubt that the foci of wild-rodent plague in Central Asia belong to the category of " natural " foci defined

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b As discussed later, species belonging to the orders of Lagomorpha and Rodentia are involved in what for the sake of brevity, is commonly called " wild-rodent plague ".

by Pavlovski [150] as arising and continuing to exist independently of factors connected with the presence of man.

Whether any other foci of wild-rodent plague fall into the same category, or became so established long ago, is a difficult question to answer. The foci in south-east Russia and in Kurdistan deserve consideration in this respect, but too little is known of their early history to permit of any definite conclusion. It should be noted in this connexion that, though wild rodents were occasionally suspected of playing a part in the spread of plague in south-east Russia during the first decade of the present century, or, perhaps, even since the Vetlianka outbreak in 1878-9, it was not until 1912 and 1913 that the existence of natural plague in the sisels (susliks) was bacteriologically confirmed by Deminski [41] and by Berdnikov,[15] respectively. Proof of the existence of the infection among the gerbils (*Meriones*) of Iranian Kurdistan has been obtained quite recently by Baltazard et al.[4]

As mentioned in chapter 1, claims have been made that the origin of wild-rodent plague in the western parts of the USA was due not to a recent importation of the infection by the sea-route, but to an early immigration from Central Asia of certain of the species involved; however, as was pointed out, the evidence supporting the latter assumption is not convincing. There is no doubt that the foci of wild-rodent plague existing in South Africa, and in parts of South America, became established through a spread of the infection from the commensal to the free-living species during the present century.

It is of historical interest to add that, as far as can be established, the first authentic record of natural plague in wild rodents was made by Simond [185] who, in 1898, reported positive findings in Indian palm-squirrels (*Funambulus palmarum*) at Karachi. As recorded by Bruce Low [110] in the following year, several porcupines, together with some monkeys, died of plague during an outbreak at Mysore. However, since these animals had been kept in a zoological garden and no other records on porcupines are available, one cannot justifiably include them in the lists of rodents, other than the commensal rats and mice, in which the existence of natural plague has been confirmed or suspected.

Species involved

In order to show the occurrence of natural plague in rodent species or subspecies other than the commensal rats and mice, and in Lagomorpha, lists enumerating (*a*) the animals in respect of which positive proof of the infection has been obtained, and (*b*) the suspected animals, are contained in Annex 1, tables I and II (pp. 623 and 633).

Particular attention has been paid to make Annex 1 not only as accurate as possible, by closely following the standard nomenclature of Ellerman [45] and of Ellerman & Morrison-Scott,[46] respectively, but also as complete as possible, though it is realized that entire success has not been obtained in

the latter respect. Indeed, it seems doubtful whether it will ever be possible to compile a really satisfactory list of plague-affected rodents and Lagomorpha other than the commensal rats and mice because it is most probable that, in addition to the known foci, unrecognized foci of sylvatic plague exist, and because, even in the areas where the presence of this type of the infection has been ascertained, by no means all the species or subspecies involved have been detected. In chapter 1 attention was drawn to the statement of Davis that over 100 rodents or other small animals are at risk of infection in South Africa, and it is also noteworthy that Pozzo [157] believed that 70 rodent species were involved in Argentina.

TABLE XIV. FAMILIES AND SUBFAMILIES OF RODENTIA AND LAGOMORPHA IN WHICH THE PRESENCE OF NATURAL PLAGUE HAS BEEN CONFIRMED

Family	Subfamily	Number of species or subspecies found infected	Area
Bathyergidae	—	1	Angola
Caviidae	Caviinae	9	Argentina, Brazil, Ecuador, Peru
Chinchillidae	—	1	Argentina
Dipodidae	Dipodinae	5	Iranian Kurdistan, south-east Russia, Transbaikalia
Echimyidae	Echimyinae	1	Brazil
Geomyidae	—	2	USA (western States)
Heteromyidae	Dipodomyinae	3	USA (western States)
	Heteromyinae	1	Venezuela
Muridae	Cricetinae	48	Argentina, Bolivia, Brazil, Ecuador, Louisiana (USA), Peru, south-east Russia, South Africa, USA (western States), Venezuela
	Dendromyinae	4	Belgian Congo, South Africa
	Gerbillinae	12	Belgian Congo, India, Iranian Kurdistan, south-east Russia, Russian Turkestan, South Africa, Transcaspia
	Microtinae	12	Iranian Kurdistan, south-east Russia, Transbaikalia, USA (western States)
	Murinae	30	Belgian Congo, Burma, Ceylon, East Africa, Egypt, Gold Coast, India, Kenya, Senegal, South Africa
	Otomyinae	6	Belgian Congo, East Africa, Kenya, South Africa
Pedetidae	—	1	South Africa
Sciuridae	—	49	Canada, Ceylon, Ecuador, India, Manchuria, Mongolia, Peru, south-east Russia, Russian Turkestan, Senegal, South Africa, USA (western States), Transbaikalia
Leporidae	—	14	Argentina, Bolivia, Brazil, England, Ecuador, Peru, South Africa, Transcaspia, USA (western States)
Total		199	

Natural plague has been confirmed in the families and subfamilies of the orders of Rodentia and Lagomorpha listed in table XIV.

In order to deal adequately with wild-rodent plague in the strict sense, table XV has been prepared to show : (*a*) the reservoir hosts in the definitely established foci where this type of the infection is known to be, or to have been, active ; (*b*) other families or subfamilies, species or subspecies of which have also been found infected, and which are therefore apt to play a subsidiary role and, if living near man, are able to act as intermediaries between the strictly wild and the commensal rodents.

The data of table XV well bear out the statement of Meyer [130] that

" as a rule, a principal species belonging to the family [of] Sciuridae or [to the subfamily of] Gerbillinae, living in subterranean colonies, in families or singly, presides over the exchanges of the plague bacillus... "

A study of the relative distribution of these two main groups of reservoir hosts in the wild-rodent foci in or near Asia clearly shows that three zones may be distinguished :

TABLE XV. LIST OF DEFINITELY ESTABLISHED WILD-RODENT PLAGUE FOCI SHOWING RESERVOIR HOSTS AND OTHER FAMILIES OR SUBFAMILIES FOUND NATURALLY INFECTED

Plague focus	Main reservoir	Also found infected
Argentina	Caviinae (*Cavia*, etc.)	Chinchillidae, Leporidae
Iranian Kurdistan	Gerbillinae (*Meriones*)	Dipodinae, Microtinae
Manchuria, Mongolia, and Transbaikalia	Sciuridae (*Marmota*)	Dipodinae, Microtinae
Peru	Cricetinae (*Graomys*) Cricetinae (*Akodon*)	Caviinae Leporidae
Peru/Ecuador border region	Sciuridae (*Sciurus*)	Cricetinae
Russian Turkestan	Sciuridae (*Marmola*)	Gerbillinae
South Africa	Gerbillinae (*Tatera*)	Cricetinae, Dendromyinae, Leporidae, Murinae, Otomyinae, Pedetidae, Sciuridae
South-east Russia		
northern foci	Sciuridae (*Citellus*)	Dipodinae Microtinae
southern foci	Gerbillinae (*Meriones*)	
Transcaspia	Gerbillinae (*Meriones*)	Leporidae
USA : western States		
Arizona and New Mexico	Sciuridae (*Cynomys*)	
coastal regions and northern part of intermountain plateau	Sciuridae (*Citellus*)	Dipodomyinae, Geomyidae
southern deserts	Cricetinae (*Neotoma*)	Leporidae, Sciuridae
Washington	Microtinae (*Lagurus* *)	Cricetinae, Dipodomyinae, Sciuridae
Venezuela	Heteromyinae (*Heteromys* Cricetinae (*Sigmodon*	—

* See Link [105]

(1) Central Asia, where large Sciuridae (marmots) form the plague reservoir (Manchuria, Mongolia, Russian Turkestan, and Transbaikalia, and perhaps other adjacent areas mentioned in Annex 1, table II (p. 633));

(2) South-east Russia, northern part, where small Sciuridae (sisels) are of prime importance ;

(3) South-east Russia, southern part, Iranian Kurdistan, and Transcaspia where Gerbillinae (*Meriones*) are the reservoir hosts.

This separation into a north-eastern area, where Sciuridae are the fons et origo mali, and a south-eastern area, where a corresponding role is played by the *Meriones*, is a matter of great interest.

Sciuridae (ground-squirrels and prairie-dogs) also form reservoirs in some of the western States of the USA and in the focus situated at the border between Peru and Ecuador, while Gerbillinae are of paramount importance in South Africa.

The Cricetinae form reservoirs in three wild-rodent foci—namely, Argentina, the Huancabamba area of the Peruvian Andes (Macchiavello [119, 120]), and the southern deserts of some western States of the USA—while the Caviinae, Heteromyinae, and Murinae each act correspondingly in only one area. The comparatively inconspicuous part played in wild-rodent plague by the Murinae, to which subfamily the commensal rats and mice also belong, seems striking when the ominous role played by the latter species in the pandemic type of plague is given consideration. However, as will be discussed later, the observations made in respect of the multimammate rat, *Rattus natalensis*, prove that this rule is by no means without exceptions.

Characteristics of principally involved species

A short description of the species mainly involved in wild-rodent plague, and of those of their habits which are of importance in the perpetuation and spread of the infection, may be given thus :

1. The marmots (tarabagans) of Central Asia and the adjacent plague areas are big animals attaining a length of about half a metre and a weight of 4 to 7.5 kg. They dig deep burrows in firm ground and live in settlements of varying size, each family inhabiting a separate burrow. Following hibernation, which lasts from about October to April, they mate in spring. After a pregnancy lasting 6 weeks, the female gives birth to 2 to 7 young.[215] As far as can be ascertained, the young continue to stay in the maternal burrow even after they have become independent, while the mother seeks new quarters for herself.

2. The small sisels or susliks (*Citellus pygmaeus*) of south-east Russia look like miniature tarabagans and have a body-weight of 60 to 200 g. They use two kinds of burrows : shallow ones during the summer and deeper ones in the winter. Their hibernation lasts longer than that of the

tarabagan, the adults beginning to sleep in July, the young animals in August or September. The mating period begins immediately after the end of the hibernation period in early spring. After a pregnancy lasting probably not longer than three weeks, litters of 8 to 10 young are born.

The young animals leave their mothers about a month after birth, each settling down in an individual burrow, preferably a previously used one. The sisels are therefore most active :

(*a*) in early spring, when the animals, which otherwise lead an individualistic life, meet to mate, and when the females prepare the burrows for their litters ;

(*b*) in early or late June, according to the weather, when the young disperse, sometimes travelling as far as 2 to 5 km before settling down ;

(*c*) a month later, when the winter burrows are prepared and occupied (Wu Lien-teh ; [215] Kalabuchov & Raevsky [91]).

3. The *Meriones meridianus* of south-east Russia are non-hibernating animals which leave their burrows mainly at night. They are ratlike in appearance, but their hind-legs are longer and stronger. Their burrows are shallow and have several hidden entrances in addition to the principal one. The latter is kept closed with sand by the female, the sand being accumulated by several strokes of the hind-legs. The female discontinues blocking up the entrance when her young mature, in order to let them go out on the surface. The mating season of these fertile animals extends from May to October ; pregnancy lasts from 25 to 28 days. Though up to three litters may be born to one female per year, 85% have only one litter annually. The young leave the maternal burrows after 25 to 30 days (Rall [162]).

As stated by Baltazard et al.,[4] the *Meriones* of the Iranian Kurdistan keep away from man and dig their burrows in non-cultivated areas far from settlements. They are of sedentary and peaceful habits, frequently visiting the burrows of their neighbours, particularly during the mating season. Though accumulating food reserves for the winter, they leave their burrows even during the coldest weather.

4. The *Tatera brantsi*, which, together with the Namaqua gerbil, *Desmodillus auricularis*, form the main plague reservoir in South Africa, are active, non-hibernating animals of strictly nocturnal habits. Their size is approximately that of rats. Their burrows have many entrances and are arranged in colonies, the distribution and size of which vary according to the nature of the soil and the available food-supply. There are two main breeding seasons—one after midwinter towards spring, and one after midsummer towards autumn. Three to five young are born per litter; they leave the parent burrows when maturing and may range over a wide area before settling down to breed at the age of three months (Wu Lien-teh; [215] Davis [39]).

According to Powell (quoted by Schulz [175]), the Namaqua gerbils are less active and less sociable animals than the *Tatera* species. They live in families of 2 to 8 members, inhabiting warrens which have many entrances. They reach sexual maturity at the age of three months, and have four litters annually of 4 to 6 young per litter.

As stated in chapter 1, the coucha rat, *Rattus natalensis*, a fertile, mouselike animal of nocturnal habits, plays an ominous role as a link in the transition of plague from the gerbils to man. Being rather inactive, these rats prefer occupying burrows deserted by gerbils or other rodents to providing their own shelters. Since they prefer to frequent, rather than shelter permanently in, human settlements and houses, and may even live away from human habitations, these animals seem to deserve a place among the wild rodents (Davis; [37] Fourie [53, 54]).

5. The ground-squirrels of the western States of the USA fall, according to Eskey & Hass,[48] into three size-groups :

(i) large animals comparable in size to the grey tree-squirrels, e.g., *Citellus beecheyi*, *C. columbianus*, and *C. variegatus grammurus* ;

(ii) medium-sized animals, somewhat more than half the size of the larger species, including *C. armatus*, *C. beldingi*, *C. richardsoni*, and *C. washingtoni*;

(iii) small ground-squirrels including, among others, *C. spilosoma*, *C. townsendi*, and *C. tridecemlineatus*.

The prairie-dogs (*Cynomys*) are comparable in size to the large ground-squirrels and may be divided, according to Eskey & Haas,[48] into two groups—the white-tails, comprising *Cynomys leucurus*, *C. gunnisoni*, and *C. parvidens*, inhabiting the Rocky Mountains, and the black-tails, *C. ludovicianus*, which range over the Great Plains. The former live in a manner similar to that of the ground-squirrels and often on common ground with them; the latter live in peculiar circumscribed colonies or " towns ", marked by prominent mounds and the absence of all low vegetation in the immediate vicinity.

Eskey & Haas state that the wood- or pack-rats (*Neotoma*) also fall into two groups : bushy-tailed species, such as *Neotoma cinerea*, living in the more forested country of the colder zones and found infected in California; and round-tailed species, found in the southern deserts, among which, according to these authors, *N. desertorum* plays the most important role. It should be noted that these rats, like most rodent species found in the southern deserts, are of strictly nocturnal habits.

The *Peromyscus*, suspected by Mohr [138] to be of etiological importance, are white-footed meadow-mice, apparently apt to frequent the outbuildings of farms (Meyer & Holdenried [134]). The *Lagurus curtatus*, recently incriminated as an important plague reservoir in Washington State by Link,[105] is a vole inhabiting sage-brush areas.

Dealing generally with the ecology of the rodent species found involved in the western States of the USA, Eskey & Haas [48] emphasized that

" human habitation and agriculture produce conditions attractive to many wild rodents. In irrigated valleys, around orchards, or in camps and resorts the various species often congregate, easily adapting themselves to an association with humans and enjoying the artificially enhanced food supply ".

While such species are apt to shelter in haystacks, empty spaces beneath buildings, lofts, and other man-made structures, the rodents which stay away from human settlements generally live in tunnels, the location and architecture of which varies with the species. Most of these rodents congregate in colonies, though in a degree differing according to the species. However, the observations of Brown [21] in Alberta, Canada, prove that there are exceptions to this rule. Brown found that *C. richardsoni* did not live together but were widely scattered and moved extensively over the prairie, whereas *C. columbianus*, common in the mountains and foothills, lived in colonies and moved within a restricted range.

Generally speaking, the wild rodents in the western States of the USA appear to produce a single litter each year, the number of offspring varying from four to a dozen. The young remain in the nests for the first few weeks of their life, after which they build tunnels of their own and forage for themselves (Eskey & Haas [48]).

It is of great importance that many of the species undergo hibernation or aestivation, or a combination of both. It appears, however, that such periods of inactivity do not occur as regularly and universally as in the case of the tarabagans and of the susliks in south-east Russia. Meyer [130] noted in this connexion that the young ground-squirrels did not hibernate or aestivate, while Evans & Holdenried,[49] working at the Calaveras dam in California, found that

" there was evidence of both aestivation and hibernation for varying periods but not all of the squirrels were inactive at the same time ".

Meyer [128] laid some stress on the cannibalistic tendencies of the rodents found plague-affected in the western States of the USA, and pointed out that " involvements of the lymph-nodes adjacent to the upper and lower gastrointestinal tube as a sequel of cannibalism are by no means infrequent ". Though these findings deserve attention, one cannot conceive that this mode of infection could have played a really important role.

6. *Heteromys anomalus*, incriminated together with *Sigmodon hirsutus* as a primary plague-reservoir in Venezuela, is stated to frequent cultivated fields, granaries, and even houses in search of food, whereas the latter rodent lives in forests, rarely visiting fields and never entering houses (Isaac Riaz [86]).

7. The " cuis " (Caviinae), which play a principal role in the wild-rodent plague foci in Argentina, generally live underground but may make

tunnels between plants in locations where the vegetation is sufficiently abundant for this purpose (Barrera [7]). These animals do not penetrate into occupied buildings and, in general, seem to have little contact with man (Macchiavello [119]). However, exceptions to this rule seem to exist; Barrera,[7] for instance, noted that, in the north of the country, the Indians used the " cuis " as food. The breeding season of these species is usually in spring.

Graomys griseoflavus (which probably also serves as a reservoir host in Argentina), although originally an arboreal rodent, may nest in various locations, even under the roofs of houses. As stated by Barrera [9] when dealing with the manifestations of wild-rodent plague in Mendoza Province, these rodents were hunted for their furs, as well as for culinary purposes.

8. Discussing the plague focus in Huancabamba, Peru, Macchiavello [119] stated that the *Akodon* (Cricetinae) which, together with an *Oryzomys* species, formed the primary reservoir of the infection, were attracted to the farms at harvest times. In the Peru-Ecuador border regions, humans contracted the infection in the fields where the secondarily involved Cricetinae lived. *Sciurus stramineus nebouxi*, which formed the primary reservoir, was an arboreal rodent.

Movement-range

The movements of free-living animals like the wild rodents fall into two categories :

(1) a " normal dispersal " (Meyer [129]) caused by physiological needs such as the daily requirements for food, the search of the maturing young for shelter, and the periodical movements of groups to the vicinity of fields or even settlements at harvest-times ;

(2) migrations due to abnormal causes, e.g., food scarcity, or catastrophes such as floods, which bereave the animals of their shelters as well as of their food-supplies.

It is impossible to make a clear-cut distinction between these two categories as far as the range of the movements is concerned. As already noted, the young, when in search of homes, and even the adults (especially the males) of some species, may roam quite far. Still more important, certain species which play a subsidiary role in wild-rodent plague may normally cover wide distances. This is true in particular for the Lagomorpha; the South African hares, for instance, travel far in the course of a single night.

That large-scale true migrations of wild rodents do take place, is well confirmed by the observations made of the lemmings in Scandinavia (Elton [47]). Evidence is also available that migrations undertaken on a minor scale have helped the spread of wild-rodent plague. For instance,

Barrera [8] stated that the *Graomys*, driven by hunger to human habitations, were responsible for a plague outbreak in northern Argentina. Similarly, multimammate mice were considered to have played an important role in the 1944-5 plague epidemic in Ngamiland, Bechuanaland Protectorate. At the height of the gerbil epizootic, the multimammate-mouse population became unusually large; a flood forced them to leave their burrows in the swamps and they invaded the villages, bringing with them the infection contracted from the gerbils.

It would appear, however, that such instances of a spread of wild-rodent plague through true migrations are exceptional.

Susceptibility and resistance

As stated earlier, laboratory tests have proved that different wild-rodent species may vary markedly in their susceptibility to infection with *Pasteurella pestis*. Several observers have claimed that the differences found to exist in this respect are not merely of academic interest, but are of great actual importance. Tikhomirova [199] denied that the highly susceptible sisels (susliks) could be the preliminary plague-reservoirs in south-east Russia, and incriminated the rather resistant *Meriones meridianus* as the fons et origo mali. Similarly, Krumbiegel [95] stated that, compared with the suslik, *Citellus pygmaeus*, *C. fulvus* was of no great importance in the perpetuation of plague in south-east Russia because it succumbed to a fulminant type of the infection which could therefore not persist long in this species or spread far. On the other hand, Davis [40] noted in South Africa that epizootics in the Namaqua gerbil, *Desmodillus auricularis* (a species found by Pirie [153] to be rather resistant to plague), took a protracted course, which no doubt favoured a perpetuation of the infection.

In a recently published paper, Baltazard et al.[4] not only stated that the *Meriones* subspecies—which were somewhat resistant to infection with *P. pestis*—were an important reservoir of the disease in the Iranian Kurdistan, but also maintained that, in general, slightly susceptible rather than highly sensitive species were instrumental in the perpetuation of wild-rodent plague.

The occurrence and importance of seasonal variations in the susceptibility to the infection within any one species have been claimed by observers in south-east Russia as well as in the western States of the USA.

In this connexion mention may be made of the investigations made in south-east Russia by Nikanoroff [144] to study the influence of the seasons on experimental plague in the sisels (susliks). Starting his experiments in the middle of June, he successively tested four batches of susliks, each consisting of 30 animals, at fortnightly intervals by subcutaneous injection with uniformly virulent cultures. While most of the animals in the first batch quickly succumbed to acute plague, the disease displayed an increasingly slow evolution in the three subsequent batches. Thirty days after

infection there were no survivors in the first group, as compared with 3 in the second, 5 in the third, and 18 in the fourth.

In 1934, Tinker & Kalabuchov [202] found that young ground-squirrels born in that year were most susceptible to plague, adult females less so, and adult males least. The two workers assumed that these differences were related to deteriorations in the physiological condition of the animals, found to evolve in the females during gestation, and in the young animals during the period of dispersal.

Carrying out laboratory tests with *Meriones meridianus*, Lobanov & Fedorov [108] noted that the gerbils infected during a period from April to July showed a localized and apparently resolving type of plague, characterized by the presence of minute abscesses which became encapsulated and were eventually replaced by scar tissue ; cultivation and animal experiments gave negative results in such instances. The majority of the animals infected during a period from July to October showed an acute and generalized form of the disease.

The presence of a most acute form of plague among the young ground-squirrels in California was claimed by Harrison [70] and was confirmed by Meyer,[130] who stated that laboratory tests had proved that the young animals were highly susceptible to the infection. He also maintained, in contrast to Tinker & Kalabuchov,[202] that the adult males, and not the adult females, came second in order of experimental susceptibility.

Although the evidence quoted above is noteworthy, it is the present writer's feeling that the higher susceptibility of young wild rodents might sometimes have been more apparent than real. Their chances of infection are particularly great because they may roam far during the period of their dispersal, and also because they may settle down in burrows containing infected fleas, the former inhabitants having succumbed to the disease. The outcome of the laboratory tests might have been the result not of an increased susceptibility of the young animals, but of a decreased susceptibility of the adults due to aestivation or approaching hibernation. This may have been particularly true in the case of the Californian squirrels, the young of which, in contrast to the adults, neither aestivate nor hibernate.

Role of hibernation

The importance of hibernation in the epizootiology of wild-rodent plague has been proved by several observers.

1. *Siberian marmots (tarabagans).* The idea that plague infection might remain quiescent in the bodies of hibernating tarabagans and that it might kill the animals after they awaken in the spring, seems to have been first put forward by Le Dantec.[103] Experimental support for this hypothesis was obtained by Dujardin-Beaumetz & Mosny [42] who found in two alpine

marmots (*Marmota marmota*) which had been infected while hibernating and which died after 61 and 115 days, respectively, foci of chronic pneumonia teeming with plague bacilli.

These results were fully confirmed by further experiments recorded by Wu Lien-teh,[212, 214] Wu Lien-teh & Pollitzer,[216] and Gaiski[58] which definitely proved that :

(*a*) Siberian marmots infected with *P. pestis* while hibernating may continue to sleep and not succumb to a generalized infection until after awakening in spring; in the instances recorded by Wu Lien-teh,[214] the longest periods of survival were 88 and 130 days, respectively;

(*b*) animals infected percutaneously or subcutaneously during hibernation and killed at various intervals during further sleep may show signs of resolving plague or evidence of a peculiar form of " latent " plague in which virulent bacilli persist at the site of inoculation and/or the regional lymph-nodes.

No doubt can exist that the persistence of such a localized infection in the hibernating animals was responsible for the appearance of generalized plague after they awakened in spring.

Gaiski[58] assumed that the peculiar evolution of plague in the hibernating tarabagans " was in causal connexion with the process of bacteriophagy ". However, though he had been able to demonstrate the presence of plague phages in some of his experimental animals, this assumption cannot be considered as generally valid. The extensive work carried out by Wu Lien-teh and Pollitzer with both hibernating and non-hibernating tarabagans produced no evidence of the presence of plague phages.

A most interesting point established by Gaiski[58] was that three tarabagans, which had been infected while hibernating, rapidly succumbed to an acute type of the disease when re-infected with *P. pestis* soon after they awakened. In all three animals, localized signs of the past infection were noted at autopsy. As Gaiski stated, these observations explained " why in spring animals which had been infected during hibernation and had carried plague bacilli to the moment of awakening, may succumb ".

It is curious to note in this connexion that, according to the observations of Rudneff,[173] hibernating sisels (*C. pygmaeus*) showed, during hibernation, a leucopenia with a specially marked diminution of the neutrophiles.

2. *Susliks.* Some observations on the evolution of plague in hibernating susliks seem to have been made by early investigators in south-east Russia, for an editorial appearing in the *Lancet*[99] in November 1913 stated that " it has been shown by experiment that infection of this animal [suslik] may be greatly prolonged, especially during the hibernating season ".

Churilina[28] also reported that hibernating susliks, when plague-infected, were apt to survive up to five months while the controls died in two to seven days.

Further evidence was obtained by Gaiski[56] who studied, during the course of a year, the seasonal changes in the susceptibility of the susliks

to plague. He used for this purpose 242 animals, grouped in 27 batches, infecting each succeeding group with a plague strain isolated from the preceding one. Seasonal differences were noted in two directions :

(i) the mean length of illness varied, showing a minimum (3 days) in June and a maximum (25 days) in winter;

(ii) in June and July, 100% of the animals had bacteraemia, as compared with 60% in winter, and 40% in March. The other animals either had a localized form of plague, with bacilli confined to the site of infection, or harboured the causative organisms in their organs, but not in their blood. The incidence of the localized type of the disease was highest in winter (30%).

Of the 30 susliks infected during hibernation :

21 awoke and succumbed after 2 to 22 days (the average was 8 days) ;
3 were killed after infection (2 after 15 days, and 1 after 35 days) ;
6 succumbed after 45 to 138 days.

Of the three animals killed, two (one killed after 15 days, the other after 35 days) showed latent plague with positive findings at the site of the infection only ; the third animal showed bacteraemia.

Three animals died after 96, 120, and 138 days, respectively, at the end of hibernation. Two of these showed abscesses at the site of infection while, in the third animal, plague bacilli were present in the internal organs as well as at the site of inoculation.

Some field observations made in south-east Russia seem to lend support to the experimental evidence furnished by Gaiski.[56] Thus, Tumansky [206] noted the existence of two periods when plague assumed epizootic proportions among the susliks—a major one, commencing at the time of the dispersal of the young and lasting until the onset of hibernation, and a minor one in early spring at the time of mating, soon after the end of the winter sleep.

It is of interest to note in this connexion that, according to the inhabitants of Mongolia and Transbaikalia, spring outbreaks of plague did occur among the tarabagans. However, these statements ought to be received with caution because instances of a high mortality in spring due to other causes have been observed in these animals.[215]

3. *North American ground-squirrels.* Two series of experiments undertaken by Prince & Wayson [159] gave the following results :

(*a*) two hibernating ground-squirrels (*C. richardsoni*) were inoculated with plague. One succumbed to an acute form of the infection after two weeks. The second—and two further animals which had been infested with plague-infected fleas—did not contract the disease.

(*b*) out of four ground-squirrels (*C. townsendi*) which survived four months in hibernation after intracutaneous infection with *P. pestis*, one sickened seven days after awakening and died on the eighth day, showing an acute inflammatory reaction at the site of inoculation and bacteraemia. The plague nature of the process was

fully confirmed. The three other animals remained well and were killed 15 days after awakening. They appeared normal at autopsy.

A point of great interest is the relation between the latent type of plague characterized above and the " inapparent " form of the infection referred to in chapter 4. As stated, it is so far not justifiable to identify these two types. Up to the present, it has merely been claimed that inapparent plague may lead to a generalized infection while, in the case of latent plague, positive proof of such an evolution has been obtained.

Some evidence is available which shows that, as has been fully proved in the case of the commensal rats, in wild rodents also, the prolonged existence of plague may lead to a progressive increase in the number of animals which are resistant to the infection.

Noting that in plague-stricken localities of California the ground-squirrels were rather resistant to laboratory infection with *P. pestis* while those captured in plague-free districts were uniformly susceptible, McCoy [126] expressed the view that the preponderance of insusceptible animals in the foci

" may mean a gradual extinction of the disease or it may indicate that this partially resistant race of rodents will, if not vigorously attacked, perpetuate the disease for many years ".

The early findings of McCoy were fully confirmed by Meyer [130] by the examination of approximately 450 healthy ground-squirrels collected partly from a known plague-focus and partly from an area where the disease had not been demonstrated. Meyer did not feel sure that the insusceptibility to infection with *P. pestis* found in the rodent populations of plague foci was solely the result of a process of " natural selection " by which the receptive strains of the animals were gradually wiped out while the resistant strains survived. He suggested that an immunity induced in individual animals by previous plague attacks might also be of some importance. It also seemed significant that, according to the observations of Bychkov,[25] guinea-pigs which had not become manifestly ill when bitten by plague-infected fleas had proved resistant to subsequent challenge-infection with *P. pestis.*

Evaluating the actual importance of the insusceptibility becoming increasingly manifest in the rodent population of plague foci, Meyer [130] aptly summed up the situation as follows :

" There is every reason to believe that the interplay between infection and immunity may be influenced by an infinite variety of ecological factors which accelerate or retard it. For example, even a coalescing epidemic spread of the plague infection in a rodent population may fail to reach every colony. Thus, at the apparent termination of the epizootics, both susceptible and resistant rats or squirrels may survive and interbreed, furnishing sufficient hosts to maintain the infection in a smoldering non-readily recognizable state."

Effect of density of wild-rodent populations

It is obvious that :

(*a*) if plague spreads to or reappears in a locality populated by wild rodents, the situation is far more likely to become serious if the rodent population-level is high than if the rodents are scarce ;

(*b*) in the case of wild rodents living in colonies, an intercolonial spread of the infection will be facilitated if these colonies are close together and impeded if the colonies are widely separated ;

(*c*) severe epizootics in a highly populated area may reduce the number of wild rodents to a level comparable with that of sparsely populated districts and thus curb the further progress of the infection.

It is important to note in this connexion that the density of the rodent population in any given group of burrows and colonies is not uniformly high throughout the year, but reaches a maximum during the period between the birth and dispersal of the young. Thus, according to Kalabuchov & Raevski,[92] the population level in the sisel colonies of south-east Russia was 3.2 to 3.6 times higher during this period than during the rest of the year. These authors maintained that, if plague was pre-existent or was introduced at that time, the temporary overcrowding of the burrows considerably aggravated the situation and was at least partly responsible for the high mortality (85%) of the susliks during their first year of life.

As has been established by investigation of the epizootics or has been suggested by studies on the occurrence of human outbreaks, the incidence of plague in the wild-rodent foci often showed periodic fluctuations, the peaks of which generally coincided with a maximal abundance of the rodents concerned. The length of these cycles was usually 3 to 5 years, but was sometimes more extended (Wu Lien-teh ; [213, 215] Fourie ; [54] Meyer ; [130] Davis ; [39] Macchiavello [119]).

These observations attracted much attention since extensive studies on the population fluctuations of wild mammals, particularly of the lemmings, had proved that periodical epizootics were of paramount importance in regulating the numbers of these animals. As summarized by Elton : [47]

" the method by which most rodents regulate their numbers is as follows : increase in numbers over several years up to a point at which an epidemic of some sort occurs, which kills off a large proportion of the population. Increase then takes place again, and is followed by another epidemic, and so on indefinitely."

The question naturally arose as to whether plague was one of the epidemic or, one should rather say, epizootic diseases apt to act as a population regulator. While this was considered likely by some workers, e.g., Wu Lien-teh, [213, 215] Barrera, [8] Meyer, [131] and Evans & Holdenried, [49] Davis [38] definitely spoke of " the establishment of so potent a regulatory factor as plague " in the South African wild-rodent foci.

Seasonal incidence of plague

That wild-rodent plague often shows a seasonal incidence, being active during certain months only, and more or less quiescent during the rest of the year, has been proved by many observations, some of which have shown that in addition to the rodents themselves, their fleas may play an important role in this respect.

As far as the part taken by the rodents is concerned, stress has already been laid upon seasonal changes in the susceptibility to infection with *P. pestis* found to exist in some species. Mention has also been made of two extrinsic factors which are apt temporarily to exacerbate plague incidence—namely,

(1) the overcrowding of the burrows in late spring and early summer due to the presence of the young as well as the adult animals;

(2) the dispersal of the young which, for the reasons previously enumerated, greatly enhances their chances of infection.

It is clear that, in hibernating species, plague can remain manifest during the warm season only. However, as discussed earlier, the infection is apt to persist in the hibernating animals in a latent form and also, as will be discussed in a future chapter, in the rodent fleas.

Since, in the western States of the USA, only the adult squirrels hibernate and/or aestivate (the young animals and the non-hibernating species involved in plague outbreaks remain active throughout the year), the seasonal incidence of plague in these areas was less clear-cut than in the regions where the tarabagans or susliks were the reservoir hosts. Indeed, Meyer [130] stated that plague was sometimes found to be active among ground-squirrels in December and January. Generally speaking, however, summer epizootics prevailed and " the lesions observed in squirrels shot in winter are those of subacute and resolving plague ".

In the wild-rodent foci of Argentina, epizootics prevailed in winter but, as pointed out by Barrera,[10] this seasonal incidence was mainly due to a low flea-index prevailing in summer. However, a decrease in the rodent population, caused by epizootics during the preceding winters, was also apt to reduce the incidence of the disease during the warm seasons.

In regard to South Africa, it was stated by Davis [35] that

" man is at risk of infection during the summer 'plague season', whereas rodent epizootics, whether in wild or domestic species, do not show any marked seasonal incidence".

Trend of epizootics

Reporting on the 1935 anti-plague campaign in the Bechuanaland Protectorate, Gerber [61] stated that it usually took 8 to 12 months for an epizootic to burn itself out. About 90% of the wild rodents (mainly gerbils)

were killed, and about three years elapsed before the remainder increased to the previous density. Epizootics then reappeared.

An excellent description of the " epizootic cycle " in *Tatera brantsi* foci was given by Davis [39] thus :

" The development of epizootic conditions is preceded by sporadic outbreaks of epizootic plague in a few smouldering foci. The gradual diffusion of *P. pestis* from these initial foci involves more and more colonies, until the majority are in various stages of decline. It takes from 4 to 6 months for an isolated colony to die out, but as cross-infection from one colony to another is erratic, it may take 12 or 15 months for a major epizootic to run its course. Major epizootics recur on the average every 5 or 6 years. Man is at risk of infection before, during and after this major epizootic, which, in effect, means for at least 3 in every 6 years."

A more detailed description of the evolution, course, and decline of the epizootics in the South African gerbils has recently been given by Schulz [175] who stated that, after a major epizootic, the gerbil population was restored to normal density in 3 to 5 years.

Investigating ground-squirrel epizootics in Kern County, California, Evans et al.[50] found that these exacerbations of the plague situation " coincided fairly well with the period of dispersal of the young squirrels ". The epizootics ran a separate course in each of the localities involved, the active phase apparently lasting for 2 to 3 weeks only.

Persistence of infection

It may be gathered from the preceding discussion that the persistence of wild-rodent plague depends upon the establishment of a kind of equilibrium between the various factors favouring or counteracting the establishment of the causative organism in the host herds.

No doubt can exist that, in some of the species involved, a reduced susceptibility of the animals to infection with *P. pestis* is instrumental in creating such an equilibrium. This mechanism is at work in the case of species such as the Namaqua gerbils of South Africa, the *Meriones meridianus* of south-east Russia, and also, as recently claimed by Baltazard et al.,[4] in case of the *Meriones* forming the plague reservoir in Iranian Kurdistan.

While fully admitting the great importance of this mechanism for the perpetuation of plague in some of the wild-rodent species involved, the present writer cannot agree with the thesis of Baltazard and his colleagues that the presence of such a reduced susceptibility of the host herds is a sine qua non for the continuation of the infection. For, as shown below, a persistence of the infection, even in fully susceptible species, is rendered possible in various ways.

A most important stabilizing influence is exerted in some of the fully susceptible wild-rodent herds by the hibernation period which, while cutting short the decimation of the species through acute or subacute plague,

at the same time permits a carry-over of the infection in the form of latent plague.

Although this regulating mechanism is absent in the case of non-hibernating species, several other factors exert a similar influence. As has been noted, even in the non-hibernating species plague often shows a seasonal incidence, i.e., periods during which epizootics are apt to occur alternate with seasons during which the infection, assuming an enzootic character, becomes far less fatal.

The decrease in the rodent populations during severe epizootics and the erratic progress of the latter, which often spares individual colonies within the affected areas, form important means of preventing a wholesale extinction of the hosts.

On the other hand, during the off-seasons, when the continued existence of the invaders is threatened, there will usually be a sufficient number of rodents which, because suffering from acute or subacute plague with bacteraemia, are capable of serving as links in the perpetuation of the infection. Moreover, even if this mechanism should fail, fleas which continue to harbour *P. pestis* are apt to carry over the infection.

Wild-rodent species other than the primary-reservoir hosts may also take part in the perpetuation of the infection. Their involvement is bound to become more serious at the same rate as the intensity of plague increases among the primarily affected species. Consequently, if the numbers of the latter are reduced to such a degree as to lessen the perpetuation of the disease, the subsidiary species are apt to be sufficiently involved to carry over the infection until the primary hosts become numerous once more.

As is suggested by an interesting observation of Meyer,[132] species which formerly played a subsidiary role in a focus of wild-rodent plague may, under exceptional circumstances, become the primary reservoirs of the infection. He recorded in this connexion that, in a focus originally maintained by *Citellus beecheyi*,

> " under the influence of a control program which was directed solely against the squirrels, *Pasteurella pestis* had transferred its activities to the mice (mainly *Microtus californicus*) and thus had protected its persistence and perpetuation... "

Discussing the perpetuation of wild-rodent plague in south-east Russia, Nikanoroff[146] and Gaiski[57] laid stress upon the existence of a " transitional " zone between the northern steppes, where sisels (susliks) were the primary hosts, and the southern, sandy stretches where, in their opinion, mice were principally involved. They maintained that, in this border region, summer epizootics among the susliks were regularly followed by winter outbreaks among the mice. Thus, a vicious circle favouring the perpetuation of the infection seemed to exist.

Tikhomirova,[199] while confirming that the epizootics in *Meriones meridianus*, which she considered as the reservoir host in the southern

regions, took place in autumn and early winter, did not share this belief in the existence of a transitional zone, but maintained that a spread of plague northwards from the *Meriones* foci led to secondary infection of the susliks.

In a note entitled " Persistence of sylvatic plague ", Meyer & Eddie [133] recorded an observation in San Mateo County, California, according to which

" two of the locations in the County where infected [ground-squirrel] fleas were found in 1936 were recognized as the same colony or series of burrows proven to harbor diseased squirrels in the summer of 1916 ".

They added that " these and similar observations indicate that sylvatic plague persists probably indefinitely in an area once invaded... ".

Though this is undoubtedly the case, it does not seem very likely that the same burrows or colonies remain continually infected throughout prolonged periods.

Spread of infection

Though, as previously discussed, a spread of wild-rodent plague at distance may be effected through the agency of migrations or of animals like hares which normally travel far, these and similar means of disseminating the infection per saltum are of comparatively little importance, or become operative only under exceptional circumstances as holds true of the migrations.

Ordinarily, the spread of wild-rodent plague is by contiguity, due probably to " an accumulation of small movements among rodents " (Meyer [130]).

Discussing this problem, Davis [39] drew an apt distinction between an intracolonial and an intercolonial spread of the infection, stating that

" the movement of individuals brings each warren a contact by relay movement during a night's activity. In consequence, the spread of plague throughout the warrens of a colony is not hindered, but the spread from one colony to another, mainly by adult males and by the maturing young at rather rare intervals, is sporadic ".

That wild rodents may be carried to settlements by rail is suggested by an observation in the port of Quequen, Argentina,[19] where cuis (*Galea leucoblephara*) were found in the grain supplies imported from the hinterland. It was assumed that the transportation of grain by rail was responsible for the rat epizootic present in the port at the time.

As stated in chapter 1, the epizootic prevailing among the commensal rats of Tacoma, Washington, in 1942-3 was suspected to have been due to an importation of plague from wild-rodent foci in the hinterland, but it is noteworthy that Hundley & Nasi [82] spoke in this connexion of the findings of rats and mice in the grain-cars. That, however, possibilities of a transport-

ation of plague-infected wild rodents do exist in the western parts of the USA, is shown by recent observations of Ecke & Johnson [43] in Colorado. As pointed out by these workers, ranchers in the southern part of the State, prompted by the desire to control the prairie-dogs on their property, were prone to pay attention to epizootics among these rodents in New Mexico and to fetch diseased animals from there in order to release them on their land. Ecke & Johnson obtained definite knowledge of three instances of this kind, in one of which the rancher in question drove 250 miles from his home to get diseased prairie-dogs. According to them two workers

" reliable sources state that control by the above method was very good, as may be expected. It is evident, however, that the ranchers are unaware of the dangers to which they are exposing themselves by practicing this form of prairie dog control. If this practice is as common as is believed, it probably is the explanation for some of the ' abnormal jumps ' of plague epizootics over great distances ".

Ecke & Johnson also referred to a rancher's boy, " reported to be catching prairie dogs from an epizootic zone and transporting them to a nearby town to be sold as souvenirs to tourists ", but considered this practice as very uncommon.

In South Africa, great stress was laid on preventing the transportation of infected rodents by rail or even trucks (Thornton [197]), but one must wonder whether the transportation of wild rodents was frequent. According to Fourie,[53] coucha rats (*R. natalensis*) were seldom carried in farm produce. That field rodents may be transported in this manner is proved by an observation in the Cumbum Valley of India where four such animals were detected among rice-bags on bullock-carts,[83] but this was apparently an exceptional occurrence.

In a study on " The focality of rodent plague in the light of ecologo-geographical ideas ", Rall [163] distinguished between " mechanical " barriers against the spread of infection, such as rivers and mountain ranges, and " biological " barriers, created through the presence of " sterile zones " where susceptible rodent-hosts, if present at all, occurred either in small numbers or were patchily distributed.

The importance of such biological barriers is illustrated by the observations made by Fedorov et al.[51] of a sandy stretch on the left bank of the Ural River. Plague did not become established in this locality because the latter was sparsely populated by susliks and mice and also, as Fedorov and his co-workers believed, because the seasons during which the various rodent species became comparatively numerous did not coincide.

However, observations in South Africa, particularly,[136, 137] have shown that zones which were ecologically unsuitable for the establishment of plague, or belts which had been artificially freed from rodents, do not absolutely bar the spread of the infection because they may be overrun or outflanked by waves of severe epizootics.

Referring to the likelihood that wild-rodent plague will not spread much further in the Union of South Africa, Davis [39] made the important statement that

"the geographical distribution of *X. eridos* and *X. piriei* is a useful biological indicator of the conditions in which *P. pestis* can be perpetuated, but it does not explain them".

One might venture to suggest that these two fleas are efficient components of rodent-vector combinations or "teams", which, according to the concept of Mohr,[138] are of fundamental importance in the genesis and perpetuation of plague manifestations.

Interrelation between wild-rodent and rat plague

Since the interrelations existing between wild-rodent and rat plague differ most markedly in the various areas concerned, it is necessary to consider each area separately instead of dealing with the subject in a comprehensive manner.

India. Though India, where no convincing evidence for the independent existence of wild-rodent plague has so far been found, does not seem to fall within the compass of the present discussion, some observations made in that country in regard to the bandicoots deserve mention because they throw an interesting sidelight on the relations between wild and commensal rodents.

Since plague workers have used various Latin names to designate the bandicoots, it is necessary to note first the classification of these animals recently adopted by Ellerman : [46]

Species	*Subspecies*
Bandicota bengalensis Lesser bandicoot-rat or "Indian mole-rat"	*B. bengalensis bengalensis*
	B. bengalensis kok
	B. bengalensis gracilis
	B. bengalensis varius
	B. bengalensis wardi
Bandicota indica Large bandicoot-rat	*B. indica indica* [c]
	B. indica nemorivaga
	B. indica savilei
	B. indica siamensis
	B. indica jabouillei

According to this classification, *Bandicota malabarica* Shaw, previously classed as a separate species by Ellerman,[45] is now being considered as falling into the subspecies *B. bandicota indica.* Similarly, *B. bengalensis kok* (often called "*Gunomys kok*" in plague publications) and *B. bengalensis gracilis* no longer appear as species, but as subspecies in the new list.

[c] Ellerman states that "many specimens of this form have been examined, and the conclusion reached [is] that there is only one (individually variable) subspecies in the area just listed" (i.e., India and Ceylon).

The first available record indicating that the bandicoots might be implicated in plague outbreaks is a statement made in 1906 by Hossack [79] according to which " *Nesokia bengalensis* " (the small bandicoot-rat) seemed to be intimately concerned in the spread of the infection in Calcutta.[d]

As reported by the Plague Research Commission in 1907 and 1910,[154, 155] in Belgaum and other places of Bombay Presidency (now Bombay State), the large bandicoots (*B. indica*), which had formerly visited the houses, had completely disappeared with the advent of plague. Since no reliable evidence of migration could be obtained, it seemed highly probable that these animals, which had been found susceptible to experimental infection with *P. pestis*, had been wiped out by epizootics.

It should be noted in this connexion that, according to ecological studies recently carried out by Sharif & Narasimham [179, 180] in the Barsi, Belgaum, and Dharwar Districts of Bombay State, *Bandicota malabarica* (scilicet *indica*), a rodent of domestic habits, " was very common in localities situated on soft ground ".[180] *Gunomys kok* (fig. 26 (p. 281)) was found in the open fields but was rarely discovered near human habitations. In Sharif & Narasimham's examinations no instance of natural plague was found in either of these two species.

Observations made in respect of the bandicoot rats in Bombay City are of great interest. Referring to these animals in the 1937 report of the Haffkine Institute, Sokhey & Chitre [189] stated that :

" during 1907 when the plague epidemic was still active in Bombay, the Indian Plague Commission noted that *Nesokia bengalensis* (*Gunomys varius*) [e] was not a common rodent in Bombay City and that it formed only 1 per cent. of the rat population of the City and that the rest of the rat population was made up almost entirely of *Mus rattus* (*Rattus rattus*) and *Mus decumanus* (*Rattus norvegicus*); *Mus rattus* forming 66.2 per cent. of the rat population. During 1937, June to December, we have classified 164,787 rats collected in the City of Bombay ... and find that *Gunomys varius* now (1937) forms over 30 per cent. of the rat population while *Rattus rattus* forms only about 25,7 per cent. Some of the difference between the observations carried out in 1907 and 1937 may well be due to personal factor in classification and possibly methods of trapping. The Indian Plague Commission also found, working in 1907, that both *Rattus rattus* and *Rattus norvegicus* were equally and highly susceptible to experimental infection. They found that 45 per cent. of the rats tested succumbed to infection. While, in 1937, [we] find that both *Rattus rattus* and *Rattus norvegicus* are highly resistent to plague (10 per cent. susceptible), and have in course of time been replaced to a considerable extent by *Gunomys varius* which is highly susceptible (about 70 per cent. susceptible). This is an interesting observation and may have some bearing on the mechanism of pandemics, how they come to an end and how they start again after a lapse of time".

As far as the figures of table XVI are comparable, they show that, from 1941 onwards, the incidence of *Gunomys kok* continuously increased to

[d] According to recent information, lesser bandicoots (" *Gunomys varius* ") were frequent in Calcutta showing an incidence of about 26% (Rao [175]) and 68.8% (Lal & Seal [97]). *B. indica* were rarely found but were also more conspicuous in the material of Lal & Seal (4.5%) than in that of Rao (under 1%). Of the 19 rodents examined and found plague-infected by Lal & Seal in 1949, 5 were " *Gunomys* ".

[e] As stated in the report of the Haffkine Institute for 1940-1, a reclassification of the Bombay rodents established that " *Gunomys varius* " was identical with *Gunomys kok;* " *Bandicota indica* " with *Bandicota malabarica ;* " *Rattus rattus* " with *Rattus rattus rufescens ;* " *Mus musculus* " with *Mus dubius* and the insectivore " *Crocidura caerulea* " with *Suncus caeruleus.*

reach a maximum of 52% in 1947, and then gradually declined. It will also be noted that, in 1948, 42 of these rodents were found infected as against 2 *Rattus rattus rufescens*. However, in 1949, only a few specimens of each species proved positive and no instances of rodent plague were found in 1950 and 1951.

TABLE XVI. PERCENTAGE INCIDENCE OF RAT SPECIES IN BOMBAY MUNICIPAL AREA DURING THE PERIOD 1938-51 *

Year	*R. rattus rufescens*	*R. norvegicus*	*Gunomys kok*	*Bandicota malabarica*	Incidence of rodent plague
1938	24.7	25.4	27.3	1.2	—
1939	23.8	24.4	35.5	1.2	—
1940	24.9	23.9	34.8	1.0	—
1941	24.2	23.0	37.1	0.8	—
1942	20.9	22.5	45.7	0.8	—
1943	20.3	22.7	46.3	1.1	—
1944	21.9	22.1	47.0	1.1	—
1945	21.0	21.6	49.2	1.8	—
1946	19.6	23.7	49.0	1.1	—
1947	18.9	23.0	52.0	0.6	—
1948	23.7	18.3	47.4	0.8	2 *R. r. rufescens* and 42 *Gunomys kok* found infected
1949	23.3	20.0	42.2	0.4	4 *R. r. rufescens* and 2 *Gunomys kok* found infected
1950	23.9	17.6	39.3	0.7	—
1951	21.1	16.5	36.7	0.8	—

* The figures for the period 1938-48 are taken from the reports of the Haffkine Institute, Bombay, while those for 1949-51 were kindly supplied by Dr. P. M. Wagle, Director of the Institute. The rodents were reported to have been trapped alive only.

Some further observations on bandicoots were made in South India during the course of plague inquiries instituted by the Indian Research Fund Association (now the Indian Council of Medical Research).

Dealing with the results of this work in the Cumbum Valley, Madras Presidency (now Madras State), George & Webster [60] noted that bandicoots were occasionally found dead from plague in the villages and actually proved the presence of the infection in two *Gunomys kok* found near settlements. Another report on this work even stated that

" it is usual in several villages of the Cumbum Valley for the epizootics to commence in bandicoots. The infection then passes to house rats and mice ". [83]

According to Wu Lien-teh,[215] a statement to the same effect was made at the 1934 Conference of Medical Research Workers in Calcutta.

George & Timothy,[59] reporting on a preliminary study of plague at a hill-station in the Nilgiris, also viewed the mole-rats (*Gunomys kok*) and *Bandicota malabarica* with suspicion. They stated that, in that locality, the mole-rats lived around human habitations and sought shelter in the houses during rainy seasons or when food was scarce outside. *Bandicota malabarica*, on account of its semi-domestic habits, seemed apt to act as " the intermediary in the extension of plague from house rats to wild rodents ". However, though conditions seemed suitable for the establishment of a sylvatic-plague focus, the two workers failed to find infected wild-rodents.

Later work at Coonoor in the Nilgiris led to the suspicion that, besides *R. rattus* and some mouse species, bandicoots (*Bandicota malabarica*) and mole-rats (*Gunomys kok*) were also " concerned with the spread of plague ". It was stated that the latter animals, which were found in large numbers in the potato-fields and compounds of dwelling-houses, migrated into the houses when the potato season was over. Their fleas were mostly *Stivalius*, but occasionally other fleas, including *Xenopsylla cheopis* and *X. brasiliensis*, were also found on them.[85]

While no convincing evidence has so far been brought forward that the bandicoots of India play a role in the establishment and maintenance of sylvatic-plague foci, the findings made in Bombay City are of importance since they prove that plague-susceptible rodents (other than the commensal rats and mice) which are able to accommodate themselves to a life near man may become dangerous not only because they are apt to act as intermediaries in the spread of the infection, but also because they may take the place of less-susceptible rat-strains in the houses.

Mongolia, north Manchuria, and Transbaikalia. As noted in chapter 1, plague, evidently spreading from a wild-rodent focus in Inner Mongolia, became entrenched among the commensal rats of south Manchuria. A similar evolution was presumably responsible for the frequent appearance of bubonic epidemics in Shan-si and Shen-si. On the contrary, in north Manchuria, Outer Mongolia, and Transbaikalia, such a transition of the infection from the wild to the commensal species seems never to have been observed. However, Wu Lien-teh [215] assumed that the absence of rat plague in Outer Mongolia and Transbaikalia was due to a scarcity of these animals. Conditions for their involvement would have been more suitable in north Manchuria, but the occasional appearance of the disease there was due to an importation through human agency resulting in a spread of pneumonic plague directly from man to man without involvement of the rats.

South-east Russia. In south-east Russia also, the commensal rats did not seem to have become involved in the wild-rodent epizootics.[215] However, in the southern part of this area, an important role was played by commensal mice, *Mus musculus musculus* and *Mus musculus wagneri*.

The latter, living mainly in the open during summer but retiring, at the approach of cold weather, to haystacks and human dwellings (Tikhomirova & Zagorskaya [201]) played an important role in bringing the infection to man, particularly as they were in the habit of visiting the burrows of wild rodents (Obolenski [148]).

Central Africa. The interesting relationship existing in Central Africa between *R. coucha ugandae* auctt. (*R. natalensis*) and *R. rattus* seems to merit attention.

As stated in chapter 1, *R. rattus* formed the sole reservoir of the infection in Kenya and Tanganyika, as well as in those parts of Uganda where these rodents, having arrived at a comparatively recent date, were able to replace the native *R. coucha ugandae*. It was stated [101] that *R. rattus* not only sheltered in the thatch of roofs, in the walls, and under the floors of the houses, but also sometimes lived, during the dry seasons, in sweet-potato fields near the settlements. The common field-rat, *Arvicanthis abyssinicus*, was sometimes seen to seek shelter in houses from which *R. rattus* was absent, and was then apt to harbour *X. cheopis* in addition to its usual flea, *X. brasiliensis*.

R. coucha ugandae—the rodent by far the most frequently found in human habitations—was the plague reservoir in the focus at Lake Albert, Belgian Congo, but, in the Lake Edward focus, seems to have shared this causative role with *R. rattus alexandrinus*. *Arvicanthis abyssinicus* was also regularly trapped indoors but, although more conspicuous in the huts of the Lake Edward focus, appears, in that area, to have remained free from plague so far.

South Africa. Both *R. natalensis* and *R. rattus* act as intermediaries between the primary gerbil-reservoir and man. The former—a rather inactive animal which prefers settling down in a deserted gerbil-burrow to digging a new one, and, at the same time, has a strong tendency to enter human habitations or even to seek shelter in them—is particularly apt to act as an intermediary.

The problem of the comparative importance of these two species is curious. In his 1948 study on sylvatic plague in South Africa,[38] Davis stated that

> " the semi-domestic multimammate mouse (*Mastomys coucha*), [scilicet *R. natalensis*] is the intermediary between the primary gerbil reservoir and man. The house-rat (*Rattus rattus*) brings infection into even closer contact with man than the multimammate mouse. The risk of plague to man is greatest in areas where one or both of these species commonly frequent farm buildings, especially when they are abundant and in close contact with wild-rodent colonies during an epizootic ".

Referring to this subject again in 1950, Davis,[40] while once more emphasizing the importance of *R. natalensis* as a link between the wild-rodent reservoir and the commensal rodents and man, stated that

" in South Africa *R. rattus* stands at the end of the chain of infection from the sylvatic reservoir and is now largely responsible for human infections, especially in the hyper-enzootic areas ".

At the same time Davis stressed that, in South Africa, *R. rattus*

" does not however act as a *permanent* reservoir of plague in rural areas nor in urban areas where plague has from time to time shown itself... It appears therefore that the climatic conditions are against the permanent circulation of *P. pestis* in *R. rattus* populations in spite of the fact that they are heavily parasitised with well known vectors... ".

Referring to the plague manifestations in the Cape midlands during the period 1925-31, Davis [38] pointed out that both *R. rattus* and *R. natalensis* were absent from the central and western karroo, *Mus musculus* being the only commensal rodent present. Consequently, conditions for a spread of the infection to man were not as suitable as in the Orange Free State. Human infection was mostly spread by means of a direct contact with wild rodents or their fleas.

USA : western States. In their fundamental study on plague in the western areas of the USA, Eskey & Haas [45] stated that the commensal rat species, which were not indigenous to the western hemisphere, had been introduced into the Pacific regions more than 100 years ago. As a result, the rats became " well established from the coast to the western ramparts of the Sierra Nevada-Cascades, enjoying both rural and urban distribution " but, they added, " most of the vast region lying between the Sierra Nevada-Cascades and the Rocky Mountains—the great inter-mountain plateau—is entirely rat-free ".

On the eastern side of the Rocky Mountains, commensal rats were numerous in all States except Montana. In New Mexico, the valley of the Rio Grande was well populated by these rodents, as was the country east of this valley.

It would seem that, thus far, no instance of rat plague has been recorded in the interior of the western States of the USA. That this is not solely due to an absence of these animals, is suggested by a recent observation of Link.[104] Investigating an epizootic among the cotton-tail rabbits of Lea County, New Mexico, he found commensal rats to be present but free from plague ; none of the 32 specimens trapped had fleas.

In the coastal areas of the western States of the USA, a spread of plague not only from the commensal rats to wild rodents, but also from the latter back to the " domestic " species, seems to have been observed. As noted in chapter 1, it is usually assumed that an importation of plague into California by the sea-route led first to rat plague, the infection then spreading to the wild rodents. However, attention was drawn to the belief of some workers that pre-existing sylvatic plague had secondarily involved the San Francisco rats. It was also stated that a transition of the infection from the wild to the commensal species had probably been responsible for the 1924 Los Angeles epidemic and for the rat epizootics present

in 1942-3 at Tacoma, Wash. During the former outbreak, in which mainly *R. norvegicus* were involved, a few infected ground-squirrels were found but it was assumed that these animals might have become secondarily infected from the rats.[215]

Attention was also drawn in chapter 1 to the fact that, on several occasions, wild-rodent fleas had been detected on rats and that, in one of these instances, Meyer & Holdenried,[134] working in California, succeeded in establishing the presence of plague in *R. rattus rattus* and *R. norvegicus* some of which were infested with ground-squirrel fleas. These authors stressed the potential danger of a spread of the infection from wild rodents living away from human habitations to species like *Peromyscus* and *Microtus* or to the commensal rodents, all of which had, to some extent, close contact with man. It should be noted, however, that, so far, instances of a transition of plague to the commensal rodents have been fairly infrequent, and that much attention is being paid to rodent and flea control in and around human settlements.

Venezuela. According to the descriptions given by Isaac Riaz,[86] conditions suitable for an exchange of the infection between the commensal and wild rodents existed in the Aragua State of Venezuela. Among the rats—which appear to have been immediately responsible for the infection of man—*R. rattus rattus* in particular was often of peri-domestic rather than of domestic habits, while *Heteromys anomalus*, one of the two wild rodents involved, frequented the fields and granaries and even entered the houses in search of corn. Guinea-pigs kept in houses (acures domesticos) seem to have been involved in the 1943 outbreak.

Argentina. As stated in chapter 1, the relations between wild-rodent and rat plague were not uniform in the various plague-affected areas of Argentina. Since contact between the two groups of rodents was brought about only by the attraction of the wild rodents to the food-supplies in settlements and houses, secondary involvement of the rats was bound to take place mainly in well-cultivated areas. In sparsely populated and little-cultivated regions, e.g., Mendoza Province, there was hardly any chance for a transition of plague from the wild rodents to the rats.[10, 12]

Barrera[11] pointed out with much reason that the greatest potential danger of the transition of plague to the commensal rats was the possibility of an establishment of the infection in these animals which would lead to successive epizootics. He claimed that such a stage had not been reached, the major rat-epizootics observed so far in Argentina having evolved in the coastal areas where no wild-rodent plague existed.

Quite naturally, human plague showed a sporadic incidence in the non-cultivated areas where man could contract the infection only when entering the haunts of the wild rodents. A secondary involvement of the

rats through contact with wild rodents invading the settlements led, on the contrary, to the infection of groups of people.[11, 109]

Peru. In chapter 1 attention was drawn to a curious observation made by Ramos Díaz [164] during a plague outbreak at Lambayeque. The rats (*R. rattus*) in this mountainous locality lived in the fields, but visited the houses at night and thus came in contact with guinea-pigs kept by the people. No doubt the infection of the latter animals, which was the immediate cause of the epidemic, had been derived from the free-living rats.

In the wild-rodent plague focus in the Peruvian-Ecuador border region commensal rats were altogether absent (Macchiavello [118, 119]). The human victims contracted plague when harvesting corn in fields infested with Cricetidae which thus served as a link for passing the infection originally present in tree-squirrels (*Sciurus stramineus nebouxi*) to man.

Discussing the recently discovered plague-focus in Huancabamba, Peru, Macchiavello [119] stated that there, also, commensal rats and their fleas were absent. An invasion of the houses by the Cricetidae (the primary reservoirs of the infection) after the harvests was responsible for violent outbreaks of human plague.

Ecuador. As mentioned when dealing with the plague situation in Ecuador in chapter 1, in that country also, the guinea-pigs (*Cavia aperea*) kept in the houses, though often the immediate cause of epidemics, were merely instrumental in passing the infection perpetuated among free-living commensal rats to man. While Sáenz Vera [174] stated that the rats came in direct contact with the guinea-pigs and even cohabited with them, Macchiavello [117] came to the interesting conclusion that infection of the latter was effected by an invasion of the houses by the rat-fleas, rather than by the rats themselves.

In 1936, when dealing with the problem now under review, Wu Lien-teh [215] maintained that " generally speaking the danger of a spread of plague from wild to domestic rodents is more apparent than real ".

Bold as this statement seems, no cogent reasons exist to refute it, particularly if it is kept in mind that increasing emphasis is now being laid on reducing and even abolishing this danger by adequately controlling the commensal rodents.

Commensal Rodents

Biology and ecology

Before entering into a discussion of the subject now under review, an explanation is due as to why the term " commensal rodents " has been used in these studies in preference to the usual designation of " domestic rodents ". The reasons for adopting the former name—which has also been used by some other recent writers, e.g., Schwartz [176]—are : (*a*) to indicate that the common rats and mice, though often forced in their struggle for

existence to shelter in human habitations, have really nothing in common with truly domestic animals, and (*b*) to indicate that it is by no means unusual for these rodents to lead a more independent existence instead of living in constant contact with man. Since, even when away from man, they usually depend on food-supplies destined for human consumption, the designation " commensal " seems to remain appropriate.

While, as discussed earlier (see p. 253), several families or subfamilies and numerous species are involved in wild-rodent plague, apart from some local exceptions, the commensal rodents implicated in plague outbreaks belong to three species of Murinae—*Rattus norvegicus*, *Rattus rattus*, and *Mus musculus*. As aptly maintained by Hinton,[73]

" possibly these species are the most highly organized members of their family; but unquestionably they are the most successful of mammals. They are clearly of Asiatic origin; but uninvited, and unfortunately for us, they have linked their fortunes with those of humanity. Human enterprise, in all its phases, and human negligence have disturbed the balance of Nature in favour of these species, have afforded them an unnatural degree of protection from their many enemies, a large and unmerited share of the world's food-stuffs, together with perfect travelling facilities. Small wonder then that these creatures have invaded and colonized all lands...; that they have developed into serious pests, taking a heavy toll from human prosperity, and forming a most deadly menace to public health ".

Description

According to Hinton,[73] these three species of commensal rodents may be described thus :

R. norvegicus is a large species of heavy and rather clumsy build ; muzzle blunt, ears small, densely clad with fine and short hairs, thick and opaque ; tail stout, never as long as the combined length of the head and body (fig. 25).

R. rattus is a smaller and slenderer animal of elegant build ; muzzle sharp, ears large, almost naked and translucent ; tail slender, at least as long as, and often considerably longer than, the combined length of the head and body (fig. 24).

Mus musculus is a small animal, looking like a miniature *R. rattus* ; ears moderately large, clothed almost everywhere with short, fine hairs ; tail about as long as the combined length of the head and body, frequently longer, rarely shorter.

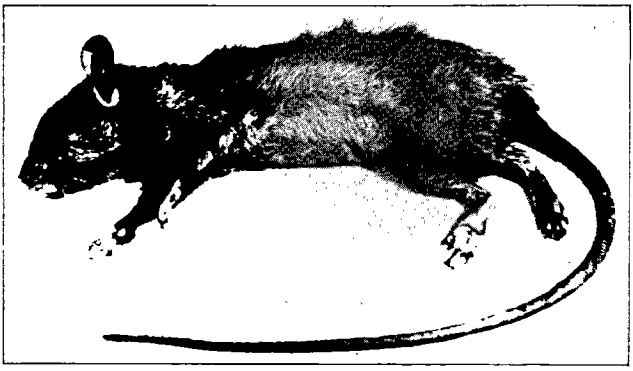
Other easily ascertainable characteristics of the three species are enumerated in table XVII, the measurements quoted referring to adult specimens.[67, 208, 215]

As shown by table XVII, the coloration of the two rat species shows marked variations.

Black varieties of *R. norvegicus* are to be found, thus stultifying the name of " brown rat " which is often given to this species.

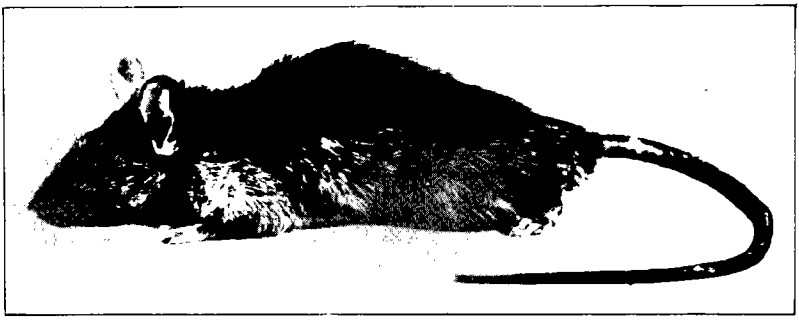
The differences in coloration found in the case of *R. rattus* have been used to distinguish between three subspecies—namely, *R. rattus rattus*, which is black or slate-coloured on both back and belly ; *R. rattus alexandrinus*, which has a tawny back and a greyish-white belly ; and *R. rattus*

FIG. 24. RATTUS RATTUS RATTUS



Size = 35.6-46 cm (including tail)

FIG. 25. RATTUS RATTUS NORVEGICUS



Size = 33-47 cm (including tail)

FIG. 26. BANDICOTA BENGALENSIS KOK (GUNOMYS KOK AUCTT.)

Size = 39.8-47 cm (including tail)

TABLE XVII. CHARACTERISTICS OF ADULT COMMENSAL RODENTS

	R. norvegicus	*R. rattus*	*M. musculus*
Body-weight	10-17 ounces (280.5-484.5 g)	4-12 ounces (114-342 g) Usually not over 8 ounces (228 g)	Less than 1 ounce (28.5 g)
Length of head and body	7-10 inches (180-255 mm)	6½-8 inches (165-205 mm)	2½-3½ inches (65-90 mm)
Colour	Grey-brown, but may be black	Black, grey, brown, or tawny ; may have white belly	Brown-grey
Length of tail	6-8½ inches (150-215 mm)	7½-10 inches (190-255 mm)	3-4 inches (75-100 mm)
Hind-foot (length from heel to tip of longest toe)	Usually over 1½ inches (40 mm). Long, sixth pad on foot	Generally less than 1½ inches (40 mm). Long, sixth pad on foot	Generally less than ¾ inch (20 mm). Round, sixth pad on foot
Mammary-glands (in female)	Normally 12 (3 pairs on chest, 3 pairs towards groin)	Normally 10 (2 pairs on chest, 3 pairs towards groin)	Normally 10 (3 pairs on chest, 2 pairs towards groin)
Droppings	In groups ; spindle-shaped	Scattered ; sausage-shaped	Scattered ; fine spindles

frugivorus, which also has a tawny back but has a white or lemon-coloured belly.[208] However, many intergradations exist between these three subspecies and it is the opinion of some experienced observers, e.g., Jorge [89] and Webster,[210] that, for the practical purposes of plague work at least, these colour distinctions should be disregarded. It is interesting to see that this opinion has been endorsed by Ellerman [46] who now considers *R. r. alexandrinus* and *R. r. frugivorus* not as true subspecies, but merely as forms or colour phases of a typical *rattus* race.

Distribution

General agreement seems to exist that the three rodent species now under review are of Asiatic descent, the original home of *R. norvegicus* and of *M. musculus* having been Inner Asia, and that of *R. rattus*, Burma and India.[73] It is in agreement with this concept that *R. norvegicus* is the predominant species in north China, where it presumably arrived long ago by a land-route, while *R. rattus*, which is restricted to the coastal districts, appears to be a comparative newcomer. In central and south China on the other hand, both rat species usually coexist in varying proportions.

In contrast to what has been found in north China, *R. rattus* alone is found in the inland districts of India, *R. norvegicus* being present in seaports only (Webster ; [210] Taylor ; [195] George & Webster ; [60] George & Timothy [59]). Referring to *R. rattus*, Ellerman [46] stated that

" in India wild (whitish-bellied) and commensal (dark-bellied) races occur together extensively. There is doubtless much interbreeding between the two ".

In India, as in China, commensal mice (*M. musculus*), belonging to various and, in part, not well-identified subspecies or races, are widely distributed.

Regarding the time of arrival of the three species of commensal rodents in Europe, it seems to be universally agreed that *M. musculus* came from Asia " with a people not older than the Neolithic " (Hinton [73]). The question as to when the rats penetrated into Europe is, on the other hand, still debated.

Dealing with this problem in 1936, Wu Lien-teh [215] felt entitled to refer to the " generally accepted opinion ... that the ships of the returning crusaders were responsible for the importation of these pests (*Rattus rattus*) in the 12th century "—an opinion endorsed by several subsequent writers, e.g., Holsendorf,[76] Morgan et al.,[141] Shrewsbury,[181] and Tricot-Royer.[204] However, as has been mentioned in chapter 1, in a recently published note, MacArthur [114] quoted evidence to show that the rats have been known in Europe since ancient times.

While the possibility that *R. rattus* have been present in Europe longer than has usually been assumed should be given attention, it is difficult to believe that *R. norvegicus* became widespread there at an early date. Voicing the opinion of most authorities, Hinton [73] stated in this respect that

" the people of western Europe had no knowledge of the species [*R. norvegicus*] until 1716, when it was introduced to Copenhagen as the result of a visit by the Russian fleet. In the year 1727, a ' mouse year ' in the Caspian region, vast hordes of these rats, according to Pallas, moved westwards after an earthquake (but probably in search of food) ; they swam across the Volga and swarmed into the houses of Astrakan. Thence they spread across Russia into western Europe ".

As a result of this mass immigration, *R. norvegicus* became the predominant rat-species in Europe. As maintained by Liston,[107] this replacement of *R. rattus* by the Norway rat was due not so much to an actual struggle between the two species, but to the fact that the former rat could adapt itself less well to changes in the habits of man which led to the expulsion of the rats from human dwellings.

The validity of Liston's contention was proved by further observations which showed that *R. rattus* was gradually apt to regain more and more of its lost territory. In this connexion, Jorge [89] pointed not only to the importance of an incessant importation of this species by vessels, but also to the environmental changes produced by a further progress of civilization which put the Norway rats at a disadvantage. Thus, in Great Britain, it was found that structural improvements such as impermeable floors, ferro-concrete construction, and separate sewerage-systems were inimical for *R. norvegicus*, while *R. rattus* profited not only by the handicaps of its competitor, but also by the availability of fixtures like telephone-wires which facilitated its progress from building to building.

It has to be noted, however, that the conditions arising during the second World War were once more apt to favour the Norway rats. Morgan [140] stated in this connexion that

" as the result of enemy action sewers have been opened up and other barriers against the brown rat have been destroyed. It is anticipated therefore that the black rat [*R. rattus*] will have a tough fight to maintain his hold against an invader whose tastes are more catholic and who will live and breed under conditions that are not acceptable to his brother ".

With regard to Africa, reference was made in chapter 1 to the statement of Jorge [90] that *R. norvegicus* was preponderant in the Mediterranean part of the continent, while *R. rattus* reigned supreme in " Greater Africa ". Attention was also drawn to interesting observations in Central Africa according to which *R. rattus*, though now forming the principal plague-reservoir in Uganda, seemed not to have arrived there before the beginning of the present century. Considering the rather conflicting views as to how this introduction took place, Hopkins [78] reached the tentative conclusion

" that *R. rattus* was brought into the inland areas of Kenya and into Uganda by the railway, which was begun at Mombasa in 1896 and reached Kisumu at the end of 1901".

As shown by a recent invasion of *R. rattus* into the Kasenyi region of the Belgian Congo, the infiltration of this species into Central Africa is still continuing.

The distribution of the two rat species in South Africa seems similar to that noted in India, i.e., *R. norvegicus* are found in the ports and their immediate vicinity, while *R. rattus* are widely distributed in the interior (Davis).[40] *Mus musculus* apparently shows an even wider distribution; as noted earlier, it was the only commensal rodent in parts of the Cape midlands.

Dealing with the rat problem in the USA, Holsendorf [76] stated that

" the ' black rat ', *Rattus rattus rattus*, otherwise known as the English rat or ship rat, was introduced to Europe in the twelfth century and was transferred to America about 4 centuries later. This antedated the arrival of the brown [Norway] rat by at least 200 years ".

As noted earlier, Eskey & Haas [48] were of the opinion that the importation of the two species into the coastal areas of the west took place at a time " well in excess of 100 years ".

Most interesting data on the present distribution of the commensal rats in the USA is given in a handbook entitled *Rat-borne disease : prevention and control*,[208] published by the Communicable Disease Center of the US Public Health Service.

Contrary to formerly-held beliefs, it is stated in the handbook that, like the common mice, *R. norvegicus* have become established in nearly

all parts of the USA. It was noted in particular that, though more than 99% of the rats on ships arriving in the USA from abroad were *R. rattus*, this species predominated in only some of the South Atlantic and Gulf Coast ports; in many seaports of these areas Norway rats were preponderant.

It appears on the other hand that, recently, *R. rattus*, probably because of its inability to compete with the Norway rats in the southern seaports,

" has moved inland more and more each year and now predominates in a number of inland cities. . . How far it will spread is uncertain, but it is not likely to become widely distributed in the Northern States as the temperature may limit its spread in that direction ".[208]

In the USA, as elsewhere, changes in local conditions were found apt to bring about shifts in local dominance from one rat species to another.

In Canada, commensal rats (mainly *R. norvegicus*) have been known to exist " since the earliest times " on the west coast of British Colombia.[81] A few places in the interior of this province also became infested, e.g., Nelson, where *R. rattus* were found. The prairie regions seem to have remained free until about 1900 when rats appeared at the international boundary of North Dakota and Manitoba. A gradual invasion of Manitoba and Saskatchewan followed so that rats are now established in all the larger municipalities of these two provinces. So far, however, rats do not seem to have colonized in Alberta.[81] Brown [23] stated in this connexion that they were repeatedly imported into this province by the railways, but were always found and destroyed.

The distribution and comparative importance of the two species of commensal rats in the South American plague-areas has been dealt with in chapter 1. Generally speaking, *R. norvegicus* appears to be the predominant species in the various Latin American countries (Moll & O'Leary [139]).

General habits of rats and mice

From Hinton's description [73] of the general habits of the commensal rats it may be gathered that :

(*a*) both *R. norvegicus* and *R. rattus* are normally nocturnal in habits, spending the day in their nests;

(*b*) while establishing their shelters as near as possible to their food-supplies, they may go further away in search of food. When regularly undertaking such foraging trips, they usually establish temporary hiding-places en route which, if convenient, may eventually be turned into the permanent dwellings;

(*c*) The rats invariably follow definite runs when leaving their shelters, proving so conservative in this respect that they may be caught by unbaited traps set in their path.

The general habits of the commensal mice are similar to those of the commensal rats. As a rule, however, they move about only in the immediate vicinity of their shelters, going, as maintained by Harrison,[69] not further than 10 feet (approximately 3 m) from them. Another important point is that, in contrast to the commensal rats, particularly the Norway rats, *M. musculus* do not display shyness to alterations in their environment (" new-object reaction " quoted by Barnett [5]).

Gnawing. Like all rodents, the commensal rats do not gnaw only to get access to food-supplies, but they must also do so in order to keep their rapidly growing incisors short enough for use.

Describing the gnawing abilities of the rats, the handbook on rat-borne disease [208] states that these animals " will gnaw through any material with a gnawing edge and with a degree of hardness less than the hardness of the enamel of their teeth " including many synthetic building-materials, unhardened concrete, and even lead-pipes.

Burrowing. While *R. rattus* are more proficient in gnawing than the Norway rats, the latter show outstanding prowess in burrowing. However, in India *R. rattus* were seen to dig in soft materials like earthen floors and walls,[215] and in the USA were also found able to burrow underground shelters (Milmore; [135] Perolio [151]).

It is important to note that although burrows for shelter and nesting rarely exceed 18 inches (approximately 45 cm) in depth, rats will dig much deeper and further to gain access to food-supplies.[208]

Gaining entrance. Rats have been found able to gain entrance through openings admitting a cylinder with a diameter of half an inch (1.27 cm).[142]

Reaching and jumping. As stated in the above-mentioned handbook,[208]

" rats have been observed reaching successfully from one point of vantage to another almost as far as their own length along smooth vertical walls. For this reason the figure of 18 inches [approximately 45 cm], the maximum reach of a large rat plus a small allowance for safety, represents the distance which must be completely clear of possible holding points. "

It was also noted that

" a rat can be expected to do a standing high jump of nearly 2 feet [approximately 60 cm]. With a running start, and with a trick of bouncing against the vertical surface two-thirds of the way up to gain momentum, the rat can jump 3 feet [approximately 90 cm] and occasionally a little more than 3 feet. Jumping out and down from a standstill, a rat can cover a horizontal distance of about 8 feet [approximately 2.5 m] while dropping less than 15 feet [approximately 4.5 m], and it can do even better with a running start ".

Rats have been observed to drop 50 feet (approximately 15 m) without being killed.[142] A roof rat swinging under a rafter is shown in fig. 27.

Climbing. While *R. rattus* is undoubtedly more able and prone to climb than the Norway rat, the latter will climb almost as well as the

former when necessary. In fact, as recently stressed by Jany,[86a] *R. norvegicus* by adopting tree-climbing habits may become dangerous, not only by depredating fruit-trees, but also by entering the upper storeys of houses from trees planted too close to the buildings.

FIG. 27. ROOF RAT SWINGING UNDER A RAFTER

Occasionally, Norway rats will also walk along telephone-wires almost as well as *R. rattus* do habitually.[208]

Rats are able to climb any vertical surface on which they can get a toenail-hold, but do not do so regularly in order not to rub their foot-pads raw.[208] However, they can easily climb vertical wires which are not rat-guarded, and vertical pipes if the diameter of the latter does not exceed 3 inches (approximately 7.5 cm); they can also climb on the outside of any vertical pipe which is within 3 inches of a wall or other continuous support. Likewise, they can climb on the inside of vertical pipes, which are not rat-guarded, with a diameter ranging from 1½ to 4 inches (approximately 4 to 10 cm).[142]

Within the limits imposed by their size, the commensal mice climb well. It may be added that these agile animals are also proficient in jumping and burrowing.

Swimming. As shown by many observations, the Norway rats are excellent swimmers and divers. They are able to " swim through the water-seals in toilets and floor-drains without fear or hesitation " [208] and habitually infest sewers and drains, the more so because—being by no means dainty in feeding habits—they find there food and drinking-water.[215] Likewise, they may establish their shelters in the banks of surface-waters in order to prey on fish-food or even young fish (Cottam [30]).

R. rattus and *M. musculus* do not enter water voluntarily. However, resembling the proverbial sailor who, though spending his life at sea, has never learnt to swim, *R. rattus* is the species of commensal rat usually found on ships.

Feeding habits and food requirements

As has been generally observed, and has been confirmed by baiting tests with Norway rats,[27] owing to their nocturnal habits the commensal rats and mice usually feed, even in a quiet environment, at night. However, if their food-supply is short, they may snatch some nourishment at any time of the day, and may then devour the food available at the place of discovery instead of carrying it back to their shelters for consumption or storage, as they usually do.

Recent observations by Calhoun [26] seem to suggest that rats differ in social standing within their colonies, each colony having a domineering bully which partakes first of the best food available and lays in no stores, in contrast to the weaker rats which try to do so whenever they have a chance.

As previously noted, *R. rattus* and also the commensal mice, being dainty feeders, are rather dependent on food-supplies destined for human consumption, while the Norway rats may subsist on any nourishment available to them on garbage heaps, in sewers, and the like.

No doubt can exist that, if given a choice, commensal rodents will show marked preferences for certain kinds of food, but they can adapt themselves to the staple foods available, in particular, often living mainly on cereals. It is interesting to note in this connexion that, Davis,[33] on the basis of studies at Baltimore, Md., maintained that

" Norway rats must have a substantial amount of grain (usually available in the form of bread) in their diet. Rats will starve in the midst of plenty of raw or cooked vegetables".

While little credence can be given to many of the statements made in regard to the amount of food a commensal rodent can consume in a given period of time, some reliable figures have recently become available.

Thus, Chitty & Shorten [27] estimated that, on an average, a Norway rat consumed 30 g of wheat per day (0.35 g to 0.50 g per gulp). An estimate made in India [84] was that *R. rattus* consumed about 3,908 g of rice per year or 10.7 g per day—a figure which tallies well with the 11 g of wheat found by McDougall [127] to represent the daily food-consumption of *R. conatus*.

Water requirements

Though *R. norvegicus* has been found able to subsist under laboratory conditions for quite considerable periods without water, in general, water is indispensable to these rats as they are used to drinking freely. If necessary, they may climb the roofs of houses to get to water-tanks.

R. rattus appears to be far less in need of drinking-water supplies than the Norway rat. Still, according to the above-mentioned observations in India,[84] *R. rattus* consumed 143.5 ounces (approximately 4 kg) of water per year, or 0.4 ounce (approximately 11 g) per day. It was found that this species could subsist on dry rice without water up to 24 days, and on water alone for 10 days, while controls given neither food nor water subsisted up to 6 days.

Harbourage

As emphasized in the handbook on rat-borne disease,[205] the possibilities for the harbourage of commensal rodents are almost endless in variety. The animals may prepare shelters by burrowing in embankments, near or under buildings, or may settle in the latter where, as stated by Holsendorf,[75, 76] three types of harbourage are available :

(1) *structural*—offered by permanently protected spaces between double walls, under double floors, above false ceilings, behind enclosed stairways, raised platforms, or boxed-in pipes, etc.;

(2) *incidental*—offered by furniture and equipment;

(3) *temporary*—offered by

" mass storage of material or merchandise, rubbish heaps, old furniture, odds and ends piled in cellars, attics, and closets, and similar accumulations which, if left undisturbed for periods of several weeks, can and will be used by rats for homes and breeding places ".[75]

Commensal rodents are apt to harbour not only in or near premises inhabited by man, but also in those parts of houses or in outhouses where domestic animals are kept, in buildings where foodstuffs are processed or stored, and in establishments such as slaughterhouses.

Morgan et al.[111] found colonies of *R. norvegicus* and *M. musculus* flourishing in cold-storage plants where these animals had adapted themselves to an existence at 17°F (−8.3°C) by growing slightly thicker furs and layers of fat underneath. The ability of *M. musculus* to live and breed in cold-storage plants was confirmed by Laurie.[100]

As indicated by the common names often given to them, the " sewer rat ", *R. norvegicus*, as a rule prefers to live underground, whereas the " roof rat ", *R. rattus*, harbours in or under the roofs of buildings, or in the upper storeys.

The Norway rat is well suited for an underground existence, not only because it is much more hardy and far less dainty in habits and food requirements than *R. rattus*, but also because, though not keen to climb as a rule, it is outstandingly proficient in burrowing shelters. One must also suppose that, in hot climates, this rat prefers the comparative coolness of its burrow to an existence above ground.

The reasons why *R. rattus*, an excellent climber, prefers to shelter in the upper parts of buildings, appear to be threefold : it is less able to withstand hardships, particularly a cold environment, than the Norway rat; it is much cleaner in habits and feeding than *R. norvegicus*; by harbouring high up, it is able to keep out of the way of any Norway rats living in the same premises underground, or on the ground floors.

The importance of the last-mentioned factor is proved by observations which have shown that, in the absence of Norway rats, *R. rattus* may live on the ground floors or even underground. For instance, according to Girard,[63] in Madagascar, where Norway rats are absent, *R. rattus* inhabits the sewers in their place.

If the two rat species coexist in a locality, their relative distribution may be irregular. Thus, Milmore [135] noted that, under such circumstances, sometimes buildings or whole city-blocks were infested by *R. rattus* and *M. musculus* only, whereas adjacent houses or blocks also harboured *R. norvegicus*.

Breeding habits

The commensal rats and mice are extremely fertile. They reach sexual maturity long before they have completed their growth. The classical investigations undertaken in this respect by the Plague Research Commission in Bombay (quoted by Hinton [73]) yielded the following results :

	Minimum weight of sexually mature animals		*Average weight of adult animals*	
	(g)	*(ounces)*	*(g)*	*(ounces)*
R. norvegicus	100	about 3.5	250	about 9
R. rattus	70	about 2.5	140	about 5

As confirmed by the recent investigations of Davis & Hall,[36] male rats remain fertile throughout the year. According to Hinton,[73] the females have a sexual season extending for any one female for 9 months of the year, during which time they come " on heat " at intervals of about 10 days. These periods last for a few hours only and if no copulation takes place during that time, a female cannot be impregnated until her next heat. During

the sexual season some evidence of the regular occurrence of heat is shown even by a pregnant female; parturition during the season is immediately followed by heat so that a female may be impregnated a few hours after the birth of a litter.

The period of gestation is usually about 21 days, but may be prolonged for a further 10 days in a female nursing her young.

According to figures supplied by the Plague Research Commission (quoted by Wu Lien-teh [215]), the average and maximum numbers of young rats per litter were :

	R. norvegicus	*R. rattus*
average	8.1 [f]	5.2
maximum	14.0 [g]	9.0

The menopause usually appears at the age of 15 to 18 months, but particularly good environmental conditions tend to delay its appearance.[73]

Dealing with the breeding habits of *M. musculus*, Hinton [73] stated that these animals

" attain sexual maturity when three months old. The sexual season of the females is a very long one. The period of gestation is normally from nineteen to twenty-one days ; it may in certain circumstances be shortened to twelve or thirteen days. ' Heat ', not lasting longer than twelve hours, rapidly succeeds parturition. Many litters are born throughout the year, but fewer in the cold months. The number of young per litter is between five and six, but it may be as many as nine or as few as two."

Scrutinizing the evidence available in regard to annual pregnancy- and lactation-rates, both from cities in the USA and other places, e.g., Bombay and Dakar, Davis [33] arrived at the following figures for *R. norvegicus* :

pregnancies per year	4.3
lactation periods per year	3.8

However, since there was a great loss of sucklings, Davis reached the conclusion that " an adult female weans rats at the rate of about 10 rats per year ".

It is important to note that this figure is rather below the estimates given by earlier plague-workers in regard to the number of young rats produced per year.

Though rat-breeding continues throughout the year, particularly under optimal environmental-conditions, it has been observed that, as a rule, there are one or two seasons during which the number of pregnant and nursing females is maximal. Studying this problem, Buxton [24] concluded that, in temperate and subtemperate climates, the main breeding-periods coincided with the warm season. That great heat exerts an unfavourable

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f 8.7, according to Davis,[33]

g According to Holsendorf,[76] litters of more than 20 have been observed.

influence on the breeding-rate seems to be suggested by the observation of Raynal [167] that, at Shanghai, which has an excessively hot summer, the breeding season of the rats coincided with the end of winter and the beginning of spring.

Buxton [24] was of the opinion that the degree to which the rats breed is mainly determined by the state of their nutrition. Their breeding-rate might, therefore, be influenced by an abundant or poor harvest which, in turn, is governed by climatic conditions.

It is quite likely, therefore, that in the case of the rats, as in that of the wild rodents, a mechanism regulating their numbers according to Elton's concept [47] might be at work. There can be no doubt, however, that in the case of the rats, many extrinsic influences would be bound to interfere with the operation of this " natural " regulatory mechanism.

Density of rat populations

One cannot doubt that a relation exists between the density of the rat population and that of the human population. Thus, it was recently pointed out by Gracie [66] that in Great Britain the greatest concentrations of rats in sewers were present beneath the most densely-populated precincts.

At the same time, however, one should give no credence to the time-worn statement that the number of rats in a given locality invariably equals that of the human population—an estimate which, as pointed out by Davis & Fales,[35] was originally derived from statements regarding the rat incidence on English farms. Davis & Fales announced as the result of their observations in Baltimore, Md., that, in 1947, the estimated total number of rats in Baltimore was 200,000, or not more than 1 rat per 5 human inhabitants. They added, however, that this figure was much lower than that of 400,000 rats estimated to have been present in Baltimore in 1944 and ascribed this reduction in the rodent population on the one hand to rat-control measures and general sanitary improvements, and to an influence of the cold, wet spring of 1947 on the other.

Obviously, therefore, it is impossible to set standards for the density of rat populations in different places, or at different times in any one place. On the contrary, it is necessary to ascertain in each instance the number of rats present through a preliminary survey, and then to watch the trend of the rodent population with the aid of the methods which will be described in a future chapter.

As far as one is entitled to draw conclusions from the scanty information available, it would seem likely that, in other well-sanitated cities, as in Baltimore, less than one rat per head of human population would be found. There is reason to assume that in less well-sanitated cities, and in smaller settlements in general, the number of rats equals or even exceeds the number of people. This is presumably the rule on farms (Tice [198]).

Population dynamics

In a profound study on the characteristics of global rat populations, Davis [33] drew a distinction between two types of changes in rat populations :

(1) an increase up to the capacity of the area in question to support the rats, and

(2) fluctuations due to changes in this capacity.

As pointed out by Davis [33] in the latter connexion, the rat population in a place at any given moment is the result of an interaction of two factors —reproduction and mortality. The effect of a possible third factor—namely, the increase or decrease of the rodent population through movements—is actually of no importance.

With regard to the force of mortality, Davis [32] maintained that, as indicated by data from a farm, only about 5 out of 100 rats lived for 12 months, the average life of a rat in a population being about 6 months.

Dealing in greater detail with the quantitative relations of the various regulatory factors, Davis [33] stated the following :

(*a*) *Environment.* A shortage of either food-supplies or nesting-sites (harbourage) limits the number of rats in a given locality or at a given time. This limitation is " density-independent ", i.e., it is operative regardless of the size of the rat population in question.

(*b*) *Predation.* Davis used the term " predation " to indicate all causes of rat mortality, including that caused by cats, viruses, traps, or poisons. He emphasized that, in contrast to the above-mentioned environmental factors, predation was " density-dependent ", the proportional effect exerted by predators decreasing pari passu with a decrease in the rodent population according to the law of diminishing returns.

(*c*) *Competition.* As is obvious, the effect of competition among the rodents for food, living space, and mates is also " density-dependent ", increasing hand in hand with population increases.

Comparing the practical value of predation with that of sanitation, which increases competition between the rodents for food and living space, Davis came to the important conclusion that, while it is generally feasible to increase competition, it is rarely possible to increase predation to a satisfactorily high degree.

Movements and migrations

As in the case of wild rodents, when dealing with the movements and migrations of the commensal rodents, it is necessary to distinguish between :

(*a*) short-range daily (or, one should rather say, nightly) excursions, necessitated by the search for food and water ;

(*b*) seasonal or, as Macchiavello [119] called them, " reversible " migrations undertaken periodically to fields under cultivation, and return to the settlements after the harvests have been gathered ;

(*c*) true, progressive migrations covering considerable distances.

Recent observations have confirmed that the urban rats and mice spend most of their life within a very limited home-range. Davis,[33] summarizing the evidence available in this respect, maintained that movements of rats even from one street-block to another were rare, and that, if a few rats left their home-range, the chances of their survival were slight.

Commensal rodents living in rural environments may also have a strictly limited home-range [34, 209] but, as noted above, they are able to make seasonal migrations.

Some observers have noted that commensal rats living in rural environments are also apt to undertake progressive migrations covering wide distances. Recently, Macchiavello [119] stated that these animals may move en masse over hundreds of kilometres, possibly in search of vitamin E in grain-germs.

Nevertheless, it would seem that, in most plague-areas, such mass migrations are exceptional. It is true, as discussed earlier, that, even now, commensal rats invade areas previously unoccupied by them, but it is probable that these movements are not mass migrations in the true sense, but rather gradual infiltrations.

Passive transportation

As discussed in chapter 1, a passive transport of rats by ships and railways was of most fateful importance for the spread of plague. Further, there can be no doubt that, in addition to rail-transport, in some plague-areas, motor-vehicle transport—particularly trucks—led to a spread of the infection by the carriage not only of infected fleas, but also of infected rats.

It is less certain whether this holds generally true of primitive means of transport. Summarizing the evidence available in this respect, the annual report for 1937 of the Eastern Bureau of the League of Nations Health Organisation, Singapore, pointed out that observations made in the Cumbum Valley of south India did not support the view that rodents or even fleas were frequently carried in consignments of rice transported on bullock-carts. It was noted, however, that, in the opinion of some observers, even " pack horses are common means in Java of conveying infected rats and their fleas ".[102]

It is curious to note that, according to a report made in 1942,[161] a live mouse was found in the galley of an aeroplane arriving at Miami, Florida, from Puerto Rico. It was stated in this connexion that

" considering the increase in the size of transport planes, the carrying of foodstuffs that are attractive for rats, and the ingenuity of these animals in boarding vessels, seeking

food supplies, establishing nesting places and avoiding man's devices for destroying them, the possibility of rats boarding airplanes is certainly not remote".

Damage caused

Calling the rat "Public Enemy No. 1 among the animal pests", Holsendorf [76] aptly quoted a statement by Creel & Akin [31] who considered this rodent not only as the least useful, but also as one of the most dangerous, of nature's parasitic animals living at man's expense.

A recently published indictment [77] of the commensal rats stated that, apart from their ominous role as the reservoirs of plague and other diseases, they caused tremendous harm by consuming or spoiling grain, merchandise, fruits, vegetables, nuts, and eggs; by killing chicks and other young birds, as well as young pigs and sheep; by damaging sugar and other plantations; and by starting fires by gnawing the insulations on electrical conductors, and in other ways.

Though many authors have tried to assess the monetary equivalent of the depredations caused by the commensal rodents, one must fully agree with Kalmbach's statement that hardly any reliable data is available in this respect.[93] This holds particularly true in the case of foodstuffs because, as has been pointed out with much reason,[142] rats will feed on any edible substance from valueless garbage to expensive packaged-products, which they may damage to an extent far in excess of the value of the amount of food they actually consume.

That the damage done by rats to foodstuffs may far exceed the loss caused by actual consumption, has been clearly demonstrated by the recent experiments of Barnett.[6] Forty rats were divided into 4 groups of 10 and each group was put into a separate room. One ton of wheat, divided into 9 sacks, was placed in each of the 4 rooms, and left for 12, 20, 23, and 28 weeks, respectively. The rats fouled 70.4% of the wheat while causing a weight-loss of only 4.4%. The total monetary loss amounted to 18.23% of the value of the wheat and sacks; most of this was due to the damage done to the sacks.

With regard to the diseases caused by the commensal rodents, it has been recently stated by Mohr [138] that these animals are responsible, or probably responsible, for the following infections or infestations.

Disease	*Reservoir*	*Mode of transmission to man*
Plague and murine typhus	Rats and to a much lesser extent *Mus musculus*	Through rat fleas
Salmonellosis	*M. musculus* and rats	Contamination of foodstuffs by droppings and possibly also through rat fleas

Disease	*Reservoir*	*Mode of transmission to man*
Leptospiral jaundice	Norway rats in particular	Contamination of food and water by urine of infected animals
Rat-bite fever	Rats, and probably also *M. musculus*	Bite by an infected rodent
Rickettsialpox	*M. musculus*	Through mouse-mite (*Allodermanyssus sanguineus*)
Lymphocytic choriomeningitis	Probably *M. musculus*	Possibly through direct contact, or through contaminated food
Amoebiasis and tapeworm infections	Rats	Food contaminated by droppings
Histoplasmosis	Dogs and possibly Norway rats	As stated by Mohr, both rats and human beings might acquire infection from a common source, but rats might take a share in infecting man

Plague in Commensal Rodents

As shown by the studies of Abel,[1] Tiraboschi,[203] and others, the now generally accepted concept that, as a rule, plague epizootics in the commensal rats precede and cause human manifestations of the disease had been arrived at by some early observers. Thus, a sacred, poetical work, the *Bhagavata Purana*, written centuries ago in Sanskrit, warned householders in Hindustan to leave their homes as soon as rats fell from the roofs and died.[107] To judge from a statement recorded by Forbes [52] (quoted by Simpson [186]), this advice was still followed by the population of Marwar during the Pali plague of 1836-8.

A great rat-mortality, preceding and accompanying the human outbreaks, was noted by a British official during the Kumaon outbreak of 1834-5. The same phenomenon was observed and commented on by medical men during several of the later epidemics in that area, and during earlier outbreaks in Garhwal (Simpson [186]).

The Chinese in Yunnan were also well aware of the fact that rat mortality presaged outbreaks of human plague.[215] Thus, during the epidemic at Chaochow in 1792, Shih Tao-nan (quoted by Wu Lien-teh [215]), in a poem entitled " Death of rats " which he wrote a few days before he died of the disease, deplored that :

> " Few days following the death of the rats,
> Men pass away like falling walls ! "

In China also, some medical men appear to have suspected that the rats were of etiological importance in plague. Lowry,[111] noted that, during the 1882 outbreak at Pakhoi, " in nearly every house where the disease broke out, the rats had been coming out of their holes and dying on the

floors " and dissected some of these animals, but found nothing definite microscopically.

Niles [147] recorded that, during the 1894 outbreak in Canton, one of the Chinese officials urged the population to kill rats, and to collect dead ones, offering to pay, out of his own pocket, 10 cash for every carcass brought to him.

Rennie,[168] who examined a considerable number of the rats thus collected, noted that 90% of them showed enlargement of lymph-nodes which, however, was present " in a much less marked degree than in the human subject ", and he raised the question " is the disease in man and animals identical ? "

In marked contrast to the advanced views held in the East, no convincing evidence seems to be available to show that the role played by the rats in the causation of plague outbreaks was realized at an early date in Europe. Though it was commonly believed that animals as well as humans were affected by the plague poison, which was supposed to be fermenting in the ground, no particular attention, or even none at all, was paid in this connexion to the rats (MacArthur [113]). It is also significant that the rats and mice often depicted in illustrated texts and in pictures after A.D. 1250 invariably seem to be healthy (Neustätter [143]).

One of the main reasons why the rats attracted no attention was probably that their dying seemed of no importance in comparison with the havoc caused by simultaneous " pestilences " among domestic animals which, though actually due to other infectious agents, were then thought to be identical with plague.

Far more distressing than this early ignorance is the fact that, even when evidence proving the existence and importance of rat plague became available about sixty years ago, it was not accepted forthwith.

It should be noted in this connexion that Yersin (quoted by Lagrange [96]) obtained positive findings in rats almost immediately after the discovery of the plague bacillus in June 1894, for he noted in his diary under the date of 23 June 1894 : " I search and find the organism in the corpses of dead rats, and there are many throughout the city ".

Yersin amplified this statement in the first elaborate report on his work at Hong Kong,[217] reaching the conclusion that " plague is therefore a contagious and inoculable disease. It is probable that the rats are the principal vector... ". *h*

Convincing as Yersin's evidence was, it did not impress the other workers at Hong Kong. Lowson's official report on the 1894 epidemic,[112] for instance, contained the following statement : "The question of the infection of rats previous to the epidemic being noted in human beings has been made too much of ".

h " la peste est donc une maladie contagieuse et inoculable. Il est probable que les rats en constituent le principal véhicule... ".

Since, as Millot Severn [178] (one of Lowson's successors) put it, the early plague investigators in Hong Kong were " obsessed with the idea that bubonic plague was primarily a gastro-intestinal infection ", it was not until 1901 that rat destruction was started there (Brown [20]), even though this method had been expressly recommended by Yersin [218] as early as 1897.

Similarly, as recently stated by Link [106] in an article entitled " Plague on the high seas ", the importance of the rats in ship-borne plague was not recognized until 1906, even though, between the years 1900 and 1904, infected animals had been found on vessels on nine different occasions.

Nevertheless, Yersin's findings were confirmed by some early plague-workers, among whom Ogata [149] in Formosa (1897), Simond [185] and Hankin [68] in India (1898), Ashburton Thompson [196] in Sydney (1900), and Blackmore in Port Elizabeth (1902) were particularly praised by Liston [107] who, himself, played a worthy part in plague research. In 1905, the British Secretary of State for India, following proposals made by the Lister Institute a year earlier, established a Joint Advisory Committee, appointed jointly by him, the Royal Society, and the Lister Institute. The Committee appointed a Plague Research Commission and, from 1905 onwards, Liston and other workers (notably, Lamb, Petrie, and Rowland) took part in its activities. The findings of this Commission formed an outstanding monument to scientific teamwork and were instrumental in defining the fundamental problems of plague in rats as well as in rat fleas.[215]

As summarized by Lamb,[98] at the time the Plague Research Commission commenced its work, even those who were agreed upon the causal role of the rats differed in their views as to how the infection was spread among these animals. Some observers were still influenced by time-honoured beliefs ascribing importance to an infection through the air, through direct contact, or through contaminated inanimate objects (fomites). Others, laying stress upon the cannibalistic habits attributed to rats, considered that plague might be transmitted through the rats feeding on one another. Through a series of well-planned experiments the Commission was able to refute these beliefs and, at the same time, to establish that plague was principally an insect-borne—and, particularly, a flea-borne—infection.

Relative importance of roles of rats and mice

Rats. In the past, as well as recently, it has been argued by some writers that *R. norvegicus* is less susceptible to infection with *P. pestis* than *R. rattus*, and therefore cannot independently act as a plague reservoir.

However, no convincing evidence has ever been brought forward to support such claims. Reliable laboratory-investigations have proved that both species are equally susceptible to the infection and, as pointed out earlier in this monograph, observations made in the various plague-areas have

shown that each of the species is capable of taking an independent part in the causation and perpetuation of the disease.

At the same time it must be realized that, if coexistent in a plague-affected locality, the two rat species may play different roles. This point was well illustrated by the investigations made by the Plague Research Commission in Bombay.[98]

The Commission's observations showed that, as far as could be judged from statistical evidence, *R. norvegicus*, though considerably less common in India, appeared to be twice as liable to natural plague-infection as *R. rattus*. There seemed no doubt, however, that this higher incidence of the disease in the Norway rats was the result not of a higher susceptibility to the infection, but of their far heavier flea-infestation. Evidently it was mainly due to the same reason that *R. norvegicus* was of prime importance for the carry-over of the infection during the inter-epizootic periods.

It was further found that, at the onset of the plague season, the infection became rampant among this species first. Then, after an interval of about ten days, epizootics appeared among *R. rattus* and these, in turn, were mainly responsible for the appearance of human plague after a further interval of 10 to 14 days. However, it was certain that this evolution was due merely to the fact that *R. rattus* lived in houses and therefore in close contact with man, whereas *R. norvegicus*, though infesting the ground floors of houses, were more numerous in other locations, such as gullies, compounds, stables, warehouses, and shops.

The conclusion reached by the Plague Research Commission that differences in the habitat of the rats and in their flea-infestation, rather than differences in their susceptibility to the infection, determined the role played by these animals in plague, has been endorsed by other workers, recently by Roberts [169, 171] in Kenya. Though *R. rattus* alone were involved in the Kenya outbreaks, two types of plague manifestations could be distinguished—namely, an urban one characterized by the occurrence of epidemics, and a rural one of an endemic type. In the opinion of Roberts, it was of importance in this connexion that, in the urban localities, the rats lived underground so that both they and their fleas were apt to come into close contact with man, whereas in the rural areas there was little, if any, possibility for such a close contact because the rats there lived in the thatch of roofs. In fact, as maintained by Cormack [29] and Roberts,[171] the roof-rats did not enter the interior of human dwellings at all ; during the day they hid in the thatch, and emerged at night on the outside of the huts, and then proceeded to the cereal stores to feed.

Mice. As pointed out by Jorge,[89] *M. musculus* usually lives in close contact with man and would be rather dangerous were its role in plague similar to that of the rat. Actually, however, though often found infected during the course of rat-epizootics, the mouse is usually a victim of the

disease rather than an agent in its perpetuation. The main reasons for this fortunate circumstance are :

(*a*) as established by the Plague Research Commission and confirmed by other workers, these animals were often less susceptible to plague infection than the rats collected in the same localities ;

(*b*) the flea-index of the mice was often considerably lower than that of the rats and, still more important, the specific flea of the mice, *Leptopsylla segnis*, is not an efficient plague-vector and hardly ever attacks man.

However, as shown by several observations, *M. musculus*, if infested by other than its usual fleas, may be of some, or even of considerable, importance in the perpetuation and spread of plague. Mention has already been made of the role ascribed to these animals in south-east Russia, and also of the observation by Herivaux & Toumanoff [72] in Indochina of an epizootic among *M. musculus* infested with *X. cheopis*. Some importance was also ascribed to this rodent species in Brazil.[13] Girard,[63] while stating that the mice usually did not appear to be involved in the plague outbreaks of Madagascar, referred to one instance where, in a rural environment, a considerable epizootic had been found present among these animals as well as among the rats.

Spread of rat epizootics

It is obvious that, when paying attention to the spread of plague not from one individual rat to another, but from one group of these animals to other groups, a distinction has to be made between what Gill [62] adequately called the " intramural " spread of the disease from one house, block, or part of a settlement to adjacent houses, blocks, or precincts, and spread at distance or, as observers in Java have called it, " metastatic " spread.

It is easy to understand how plague, conveyed either by the rats themselves or by their fleas, may creep from one house or group of houses to others, the more so since, as observed by the Plague Research Commission, these rodents may desert infected buildings, particularly when the human inhabitants have died or have left.

Such a spread of the infection may be extremely slow; one instance was recorded where a rat epizootic in India took six weeks to travel 300 feet (approximately 91 m).[215] A further fact of great importance is that the intramural spread of plague often takes place in an irregular manner, leaving the rat population of some houses, blocks, and even whole precincts unaffected.

The question as to whether active movements of the rats are responsible for the long-distance spread of plague, and if so, to what extent, has been the subject of considerable debate. In the opinion of the Plague Research Commission,[98] this factor was of no importance, the spread of the infection

per saltum being effected by the passive transportation of rats or their fleas by human and goods traffic. This opinion has since been shared by most subsequent workers.

Factors limiting the spread of rat plague

It may be maintained that the spread of plague among the commensal rodents is limited mainly by :

(*a*) factors governing the role of the insect vectors, particularly the fleas;

(*b*) a diminution in the number of rats by severe epizootics which reduces the chances of infection among the scattered survivors;

(*c*) the irregular spread of the infection which may by-pass groups of animals living in the vicinity of plague foci;

(*d*) a state of non-susceptibility to the infection developing in individual animals or in rodent-herds which have been exposed to plague.

With regard to the last-mentioned factor—the only one which can be evaluated at present—it should be noted that some writers stressed the importance of an immunity acquired by the commensal rodents in the course of plague epizootics. They claimed that the animals became immune by surviving an attack of the disease, and sometimes even maintained that rats may acquire an active immunity by exposure to subinfective doses of *P. pestis* without passing through a stage of manifest illness. More important still, these writers postulated that the immunity acquired by the commensal rodents through exposure to plague was passed on to their offspring.

While no doubt can exist that commensal rodents, particularly those which have recovered from an attack of plague, may become immune a gainst the infection, it is probable that such an active immunity is maintained for short periods only; Gill [62] was of the opinion that the immunity did not last more than a few months.

The rather unlikely assumption that the acquired immunity of commensal rodents is passed on to their offspring has been definitely disproved by investigations made by Sokhey & Chitre.[188, 189] Experimenting with the offspring of white mice which had survived plague infection owing to having been previously immunized with Haffkine vaccine, these workers demonstrated that the young mice, when inoculated with standard infective doses of *P. pestis* at an age of 3 to 4 months, showed no evidence of immunity.

For these reasons, it would appear that the immunity acquired by some commensal rodents in the course of plague epizootics usually plays no important, and certainly no permanent, role in the limitation of the

epizootics. However, it is of the utmost importance in this respect that, as first shown by the Plague Research Commission [156] and confirmed by other observers, particularly Sokhey & Chitre,[188, 189] the presence of plague in a herd of commensal rodents leads to a gradual extinction of the susceptible strains of the animals, whereas resistant strains survive and produce offspring which are also plague-resistant.

In this connexion the Plague Research Commission [156] established that :

(*a*) a considerable number of the Bombay rats were resistant to laboratory infection with *P. pestis* produced either through flea-bites or through subcutaneous injection of infective doses standardized according to the rather unsatisfactory methods then available;

(*b*) the survivors of experimental epizootics were also highly resistant when challenged with such test doses;

(*c*) rats caught in plague-free localities in India showed a high susceptibility to the infection, amounting to 97% to 100% in the case of Madras City, whereas rats from plague-affected localities were more or less resistant, the degree of their resistance to the test doses generally being proportional to the extent to which the places in question had suffered from the disease.

Sokhey & Chitre [188, 189] repeated these investigations with accurately standardized test-doses. They compared the aggregate human-plague death-rate (calculated per thousand of the population) of numerous Indian cities and towns during the period 1899-1929 with the percentage death-rate from plague in batches of *R. rattus* obtained from the same localities. As a rule, about 50 adult animals were taken from each locality. The results showed that, on the whole, the percentage rates of infection were inversely proportional to the human-plague death rates, i.e., the resistance of the rats to test infection was higher the more the locality in question had suffered from plague.

Confirming some earlier observations, e.g., those of Spencer [191] at Alabama, Sokhey & Chitre also established the important fact that, even in localities which had never suffered from plague, a considerable minority of the rat populations could be resistant to the infection. It is therefore obvious that, normally, both plague-susceptible and plague-resistant rat-strains exist side by side, the occurrence of plague upsetting the balance between the two groups by killing the susceptible animals.

The question of whether this also holds true of the rat populations in rural endemic-areas—only urban settlements were investigated by the above-mentioned workers—seems, thus far, to have received insufficient attention. Rao [166] stated that he had found evidence of resistance to the infection in a group of 22 rats caught in a village of Hyderabad State. However, George & Webster [60] established the reverse in the Cumbum

Valley and, similarly, Roberts [169] reported that he had found no evidence of plague resistance among the rats of a rural endemic-area in Kenya. It would be desirable to confirm these findings through further and more extensive investigations. As far as evidence is available, it would appear that the rat populations in rural endemic-areas may remain susceptible to infection with *P. pestis*.

Considerable time may elapse before the preponderance of resistant rats becomes sufficiently marked to exert an influence on the plague situation in the localities concerned. For, as summarized by Wu Lien-teh,[215] this process of selection

" may show an indefinite variety in different localities and certain influences may tend to retard it. Liston for instance pointed out with reason that an epizootic may leave susceptible rat colonies untouched so that at its termination both resistant and susceptible rats may survive and interbreed. A similar situation may be created if susceptible rodents are imported into a locality where the local rats have become more or less resistant. In fact the Plague Research Commission found that town rats in Bombay showed a higher immunity to plague infection by feeding and by flea transmission than ship-rats from the harbour, which latter had presumably not been exposed to infection ".

No doubt can exist, however, that when reaching a sufficiently high degree, the resistance of the commensal rodents to the infection is apt to exert a profound influence on the plague situation in the areas concerned. Liston [107] maintained in this connexion that

" the evolution of an immune race of rats following on a long series of epidemics may explain the gaps in the continuity of epidemics which are known to have occurred in the history of plague in different countries "

and that this may also

" afford an explanation for the cessation of epidemics of plague in countries in which changes in the social life of the inhabitants fail to supply us with an adequate solution"

A point of great interest and importance is how long the resistance, becoming manifest in the rat populations of infected localities, will persist in the absence of plague. The Plague Research Commission (quoted by Wu Lien-teh [215]) noted, in this respect, that the rats of Vaniyambadi which had suffered severely from plague in 1901-3 were highly susceptible when tested eight years later. Further information on this point was furnished by Sokhey & Menezes [190] (see table XVIII).

Table XVIII shows that apparently the herd susceptibility to plague became nil in the second year during which rat infection was absent from Bombay, but began to rise again in the following year. However, as shown by table XVI (see p. 274), rat plague continued to be practically absent from Bombay.

TABLE XVIII. SUSCEPTIBILITY OF RATS TO PLAGUE IN BOMBAY DURING THE YEARS 1931-9

Year	Epizootic among rats		Susceptibility of rats to plague	
	total number examined at Bombay	number found plague-infected	number infected	percentage mortality
1931	290,316	748	118	9.3
1932	272,018	600	106	8.5
1933	256,900	393	139	7.9
1934	237,854	34	93	6.5
1935	226,289	0	40	5.0
1936	182,727	0	20	0.0
1937	186,987	0	90	7.8
1938	187,276	0	—	—
1939	207,230	0	50	10.0

Seasonal incidence of rat plague

While, as will be discussed later, the seasonal incidence of plague is largely governed by the influence which climatic conditions exert on the flea vectors of the infection, some observers have laid stress also on a role played in this respect by the breeding periods of the rats.

This point seems to have been brought up first by Gotschlich [65] who, finding that the season during which plague became epidemic at Alexandria coincided with the main breeding-period of the rats, postulated that a causal connexion existed between these two events, the young rats furthering a recrudescence of the epizootics.

While Martin [125] expressed disbelief, other observers, e.g., Gill,[62] were of the opinion that, in India also, a seasonal increase of the rat populations caused by peaks in breeding gave impetus to the epizootics. In Lamb's summary of the work of the Plague Research Commission,[98] it was stated in this connexion that

" both in Bombay and in the Punjab . . . breeding of rats goes on all the year round, but that it is especially vigorous during the season between the end of one epizootic and the beginning of the next. During this interval there would, therefore, be added to the rat population a large number of young susceptible individuals, a factor which would evidently influence the rise of the epizootic ".

Considering the knowledge now available on the population dynamics of rats, and bearing in mind the observation of Roberts [170] that, in Kenya, the breeding-rate of *R. rattus* was much higher in the endemic than in the unaffected areas, one might postulate that a vicious circle exists in this

respect, i.e., a decrease of the rat population through an epizootic leads to more frequent births during the off-season which in their turn, help to promote the subsequent epizootic.[i]

OTHER HIGHER ANIMALS

Mammals

As will be seen in Annex 1, table IV (see p. 636), a number of animals belonging to various orders of the class Mammalia, other than rodents and Lagomorpha, have been found naturally plague-infected, or have been suspected of suffering from plague.

Camels

The occurrence of plague in camels was suspected by some of the earlier workers in south-east Russia and strength was lent to their assumptions through the investigations of Nikanoroff,[145] who succeeded in infecting these animals through administration of *P. pestis* by the subcutaneous route, per os, and by inhalation. He pointed out that contamination of the forage of the camels through the faeces of plague-infected rodents might lead to their natural infection, and that such infection might prove dangerous for man because the Kirghese were wont to kill diseased domestic animals and to consume their meat. In the opinion of Nikanoroff, a number of human outbreaks had been recorded which appeared to be due to this mode of infection.

It has to be noted that these claims have not been accepted universally. Petrie,[152] for instance, stated that Nikanoroff's postulations

" conflict so sharply with experience gained elsewhere, under both natural and experimental conditions, that it seems best to await further information".

Dogs

Fujinami [55] claimed to have found a dog suffering from pneumonic plague during the 1910-11 Manchurian epidemic. The validity of this observation was upheld by Strong & Teague,[193, 194] because they found that dogs were susceptible to experimental infection with *P. pestis* by inhalation.

In the course of their work in Morocco, Blanc & Baltazard [18] had the opportunity of examining a dog which had died in a plague-affected household. The spleen of this animal was somewhat enlarged. Smears made from this organ, as well as from the liver, lungs, and nasal mucus, showed

[i] Further reference to the subject of rodent plague will be made when discussing the problems of epidemiology.

numerous suspicious bipolar-stained bacilli. The diagnosis of plague was supported by the positive results obtained through inoculating white rats with the pooled fleas (*Ctenocephalides canis*) of the dog. Fleas (*Ct. felis*) from a cat which had died in the same house soon after the dog, likewise proved positive for plague.

It would therefore seem that, on rare occasions, dogs may contract plague under natural conditions. Certainly, however, instances of this kind are of little importance when contrasted with the far more dangerous role dogs are apt to play by picking up plague-infected rodent-fleas and conveying them to their masters.

Cats

While experience gained in all major plague-areas has confirmed that cats may suffer from plague under natural conditions, it has been much debated whether they can contract infection by feeding, and whether their infection occurs frequently enough to be of importance for the spread of the disease.

Araujo [3] and Henriques,[71] who recently devoted attention to the former problem, confirmed the conclusion previously reached by Dujardin-Beaumetz (quoted by Henriques [71]) that, under natural conditions, cats could contract infection by feeding only if their buccal or intestinal mucosa was traumatized by bone fragments.

In the opinion of several observers, e.g., Henriques,[71] and Silva & Valença,[184] cats were not, in general, very susceptible to experimental infection with *P. pestis*. Uriarte & Morales Villazon [207] found that such infection produced a chronic disease characterized by emaciation and abscess formation, but that the causative organisms could not be recovered at autopsy. As shown by Henriques,[71] the death of experimentally infected cats could be due to the action of the endotoxin of *P. pestis*, and not to a bacteraemia.

According to Moll & O'Leary,[139] plague-workers in Argentina often noted instances where the presence of plague in cats seemed to be responsible for human cases. Pozzo,[157, 158] while admitting that in some parts of the country a high mortality among these animals was observed during plague outbreaks, was not certain whether this was invariably due to infection with *P. pestis*.

In Brazil, the frequency of a rapidly spreading and acutely fatal disease among cats was noted in the plague foci, and sometimes also in the plague-free areas (Silva).[182] Bezerra Coutinho & Macchiavello who studied this problem closely, ascribed this fatal cat-disease, which they called " adeno-myelo-enterosis ", to infection with a filterable virus.[16, 115, 116, 121, 122, 123, 124] This conclusion was questioned by Silva [183] who claimed that the cat mortality was due, in part, to plague.

Be this as it may, generally speaking the incidence of plague among cats was not high enough to play a conspicuous role in the spread of the infection to man. However, the cats, like the dogs, are apt to be dangerous by bringing infected rodent-fleas into the houses.

Shrews

The shrews, which live side by side with the rats and regularly visit houses at night, play a not inconsiderable role in the spread of plague. As shown in Annex 1, table IV (see p. 636), they have been found liable to contract natural plague in several plague-areas. More important still, being infested with rat ectoparasites, they are apt to convey infected fleas to human habitations.

Sharif & Narasimham [179] also stressed that *Suncus murinus*, besides wandering from house to house, could undertake excursions from village to village.

Plague-Insusceptible Beasts and Birds of Prey

Ample evidence exists to show that carnivora and birds of prey may play a role in the spread of plague, even though they are resistant to the infection, because they are apt to pick up, and later to disperse, infected rodent-fleas. Some workers have also stressed the fact that birds of prey regurgitate the indigestible portions of their meals some time after they have fed and have postulated that these " casts ", if consisting of the remnants of plague-infected carcasses, may be a means of conveying the infection to rodents eating such morsels. Egorov,[44] who seems to have been the first to propound this view, found that virulent plague bacilli were present in " casts " regurgitated two days after an infectious meal by an eagle (*Aquila hipalensis*) which had been fed in the laboratory with the carcasses of plague-infected guinea-pigs.

Experiments performed by Araujo [2] did not lend support to Egorov's contention because it was not possible to demonstrate the presence of *P. pestis* in the contents of the gizzards and intestines of buzzards (*Urubus*) which had been fed with plague-infected materials. However, Jellison [87, 88] repeatedly succeeded in isolating plague bacilli from the casts of owls and falcons which had been infected in this manner. In the course of field investigations he was able also to demonstrate several times the presence of *P. pestis* in the remnants of ground-squirrels which had been partly eaten by birds of prey, sometimes after the latter had transported the rodent carcasses for some distance. Believing that the ground-squirrels were often carnivorous, Jellison postulated that they could contract plague by feeding on remnants of infected carcasses.

Even if one is ready to subscribe to such views, it is likely that infections caused by the consumption of casts or remnants of plague carcasses are of

less importance than those due to the transport of infected fleas by birds of prey.

Sergeev,[177] a worker in south-east Russia, noted in the latter connexion that many species of birds were apt to seek temporary refuge in the burrows of wild rodents, and that some nested there permanently. On the other hand, one species (*Oenanthe oenanthe*), which had been proved to harbour wild-rodent fleas known to be plague-vectors, nested in houses, and therefore seemed capable of conveying the infection from the fields to human settlements.

Brown,[22] like Jellison,[88] drew special attention to a North American species, the burrowing-owl (*Speotyto cunicularia*), which lived in close contact with ground-squirrels, often sharing their burrows. The dangerous role of this owl in the spread of plague was confirmed by Wheeler & Douglas [211] through inoculation tests with pools of sticktight fleas (*Echidnophaga gallinacea*), common ectoparasites of the ground-squirrels, which had been collected from one such bird.

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