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PLAGUE

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R. POLLITZER, M.D.

formerly of the

Division of Epidemiological and Health Statistical Services,
World Health Organization

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Preface

In reviewing the history of plague research from the discovery of *Pasteurella pestis*, in 1894, to the present day, one cannot fail to perceive that, although work in this field of applied science was carried on assiduously the whole time, the greatest progress was made at the beginning and at the end of this sixty-year period. During the twenty years immediately following the isolation of the causative organism, the work of such pioneers as Yersin, Simond, Albrecht, and Ghon, together with the comprehensive studies of the Plague Research Commission in India, laid a secure foundation for future investigations; and it was also in this period that plague vaccination was introduced by Haffkine and, thus, the first milestone on the road to effective plague-control was reached. But it is only within the last decade that treatment with sulfonamides and antibiotics, on the one hand, and the application of potent insecticides—particularly DDT—on the other, have rendered plague both a normally curable and a thoroughly controllable disease.

The last publication in the English language to deal comprehensively with the plague problem was a manual compiled in 1936 by Wu Lien-teh, J. W. H. Chun, R. Pollitzer, and C. Y. Wu. Appearing as it did before the spectacular advances in the treatment and control of the disease had begun, this work, while still appreciated as a reference-book, has become rather outmoded. For this reason, and because he was the only one of the four authors who had remained active in plague research, the present writer was repeatedly urged to bring out a second edition. However, quite apart from the fact that the writer's work on plague and cholera control occupied the major part of his time both during and immediately after the second World War, several considerations militated against a re-edition of the 1936 manual. In the first place, the book was written primarily for workers in China, and contains some sections of little interest for the general reader. Secondly, as already indicated, the parts concerned with treatment and with the control of rats and fleas have become so obsolete as to be of historical rather than practical importance.

Hence, it became clear that it would be far better to publish an entirely new monograph, dealing with the plague problem as it exists today, than to attempt a revision of the 1936 manual.

It was therefore decided that, initially, a number of studies, dealing successively with all aspects of plague, should be published in the *Bulletin of the World Health Organization*, and that, eventually, these individual studies should be grouped and reprinted in book form. The first study was published in November 1951, the last in September 1953. Before publication in the present monograph, the studies were carefully revised and brought up to date. It is hoped that the final result will be of real assistance to workers engaged in plague research.

Chapter 1

HISTORY AND PRESENT DISTRIBUTION OF PLAGUE

HISTORICAL SUMMARY

Dealing with the historical aspects of the plague problem in 1936, Wu Lien-teh [160] maintained that the disease had been present since time immemorial in the areas within or near the Central Asiatic plateau which he considered as the original home of the infection. He noted that some authors were inclined to place this in Central Africa but, though the focus existing there was undoubtedly of very old standing, Wu Lien-teh was in agreement with the statement of Payne [111] that " possibly, if we could follow the history far enough back, we might find that the African was a colony of the Asiatic plague ".

Basing his statement upon the authority of Sticker,[141] Wu Lien-teh maintained that the first plague epidemic on actual record was the outbreak among the Philistines in 1320 B.C. which, as described in the Bible (1 Samuel, v and vi), was characterized by the appearance of " emerods in their secret parts ".

As claimed by some recent writers, this interpretation of the biblical text has become untenable. Dealing with this subject in 1942, Neustätter [106] pointed out the interesting fact that the identity of the emerods with plague boils had been mentioned already in a marginal note to the revised version of the Bible appearing in 1885, that is, at a time when people in Europe paid little, if any, attention to the subject of plague. It seemed, however, that the 1885 compilers had followed the lead of the Bible commentator Thenius who had asserted the plague nature of the Philistine outbreak about 50 years earlier. Thenius in his turn had probably been influenced by the writings of J. J. Scheuchzer (1672-1733), doctor of medicine and

professor of mathematics and physics at Zürich, Switzerland, referring to the 1720 epidemic in Marseilles, France.

While not definitely committing himself as to the real nature of the biblical outbreak, Neustätter concluded " that the version hemorrhoids, untenable as it is, comes nearer to . . . the truth than plague-boils ". Shrewsbury [136] and Girard [37] felt certain that the emerods were really haemorrhoids because they considered that the disease decimating the Philistines was bacillary dysentery. However, this assumption was vigorously opposed by MacArthur,[80] who asserted the plague nature of the Philistine outbreak on historio-epidemiological and philological as well as on clinical grounds and, in a further note,[81] also adduced evidence putting " the great antiquity of the rat [*Rattus rattus*] in Palestine beyond question ".

A further, generally accepted, record testifying to the existence of plague in the West during the pre-Christian era is contained in a fragment from the writings of Rufus, physician at Ephesus about A.D. 100, who noted the occurrence of fatal bubonic plague in Libya, Egypt, and Syria during and before his time, apparently as far back as about the end of the third century B.C. (Wu Lien-teh [160]).

Whether this scanty information refers to occasional manifestations of the disease which remained localized, or whether some of these outbreaks were episodes of an early pandemic, it is impossible to decide. It is certain that the first really satisfactory evidence regarding the prevalence of plague concerns a pandemic commencing in the fifteenth year of the Emperor Justinian's reign (A.D. 542), which was voluminously dealt with by contemporaneous writers. In the opinion of most of these chroniclers, the pandemic had started at Pelusium in Lower Egypt, but probably this port served merely as the distributing centre of an infection derived from an endemic focus. The contention of Evagrius that the plague had come from Ethiopia might suggest a Central African origin of the pandemic during Justinian's reign (Wu Lien-teh [158]).

Lasting for a period of fifty to sixty years, the pandemic gradually spread, as one of the chroniclers put it, " to the ends of the habitable world ". Usually seaports were invaded first, the infection then progressing inland and eventually involving even the most sequestered localities (Procopius, quoted by Gibbon [33]). It was estimated at the time that the number of victims might have reached a total of one hundred million and Gibbon, when scrutinizing the records of the contemporaneous writers, considered this figure as " not wholly inadmissible ". Certainly the outbreak of plague in Justinian's time which, as deplored by the chronicler Warnefried, " depopulated towns, turned the country into a desert, and made the habitations of men to become the haunts of wild beasts " was one of the worst calamities that ever befell mankind. As Hirsch [54] points out, it is possible that the simultaneous presence of other epidemic diseases partly accounted for this death-toll, but the contemporaneous records leave no

doubt that bubonic plague took the foremost part. The question of whether or not commensal rodents played an important role in the causation of these bubonic epidemics is much debated. In the opinion of many writers, because rats (*R. rattus*) were not imported into Europe before the 12th century through the ships of the returning crusaders, they were still absent at the time of the pandemic during Justinian's reign. It is generally admitted that commensal mice were already abundant in the West at that period, but, as stressed by Shrewsbury [136] and Girard,[37] usually these rodents, though apt to become secondarily involved in the course of rat epizootics, do not play an independent role in the causation of human outbreaks. The same view was strenuously advocated by Tricot-Royer [148] who came to the conclusion that

" interhuman transmission formerly played a considerable if not predominant role in the spread of epidemics, and was often even the only factor concerned ".[a]

However, it deserves attention that this opinion was not shared by MacArthur,[80] who adduced evidence pointing to the early presence of rats in Europe.

Though it is likely that plague outbreaks, due to fresh importation of the infection if not to its local persistence, continued to occur in the West as well as in the East, the information actually available is usually unsatisfactory until once more the evil culminated in a pandemic which, as Hirsch [54] put it,

" arrested the attention of the chroniclers, poets, and physicians of these days ; and that interest was awakened by the enormous diffusion that it reached over the whole of the then known world, by its victims reckoned in millions, and by the shock to the framework of society which it brought with it and left behind it ".

While it is uncertain whether the pneumonic form of the disease played an important part in the plague outbreaks in Justinian's reign, this type figured prominently in the pandemic of the 14th century which, for not fully elucidated reasons (see Wu Lien-teh [160]), became known under the name of the " Black Death ". However, rats being without doubt implicated in the perpetuation of the infection, the bubonic form was also frequent, or even preponderant, as for instance in rural England (Greenwood [41]).

As mentioned above, it is possible that in the case of the pandemic in Justinian's time the infection had been derived from the Central African plague focus. No doubt can exist that the Black Death originated in Central Asia. Circumstantial evidence for this assumption was brought forward by Wu Lien-teh [158] who showed that this pandemic was not restricted to Europe and the Near East but was rampant in India and China as well : a spread of the infection from its original home in inner Asia southwards and eastwards as well as towards the west was therefore likely.

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[a] " dans l'épidémisation ancienne, la convection interhumaine jouait un rôle considérable, sinon prédominant, et souvent même exclusif ".

Through a fortunate accident the present writer was able to obtain confirmation for this contention. In a book entitled *The Nestorian Missionary Enterprise* by Stewart [140] a reference was found to the work of the Russian archeologist Chwolson near Issyk Kul Lake in the Semirechinsk district, an area now known to lie within the Central Asiatic plague focus. Chwolson found in old Nestorian graveyards three memorial stones dating back to the years 1338-9, the inscriptions on which showed that the persons referred to had succumbed to plague. It was, moreover, evident that during the period in question an exceptionally large number of burials had been made. It is certain, therefore, that plague was conspicuous in Central Asia a few years before the Crimean ports became infected (1346) and the disease was carried from there by ship to Europe.

In Great Britain, from half to two-thirds of the people are believed to have been killed by the Black Death. Hecker's [50] estimate that 25 millions or one-fourth of the population fell victim to the pandemic in Europe was therefore perhaps conservative. Millions succumbed in Asia as well where, according to Gabriel de Mussis, an eyewitness of the outbreak in the Crimea,

" so great was the mortality that Arabs, Saracens and Greeks throughout the whole of the East gave themselves up to clamour ".

It was inevitable that in many places the infection introduced during the pandemic became firmly entrenched among the rats. As a result, many countries in Europe continued to have frequent or even perennial epidemics during the centuries following the Black Death. However, as described by Wu Lien-teh,[160] in the course of the 17th century a decline set in, leading to the gradual disappearance of the disease first from western and then from eastern Europe until in 1841 Turkey, the last stronghold of the pest, became free.

The reasons for the cessation of plague in Europe have been the subject of much debate. Great stress was laid by some authors upon the change in the rat population by which *R. rattus* became largely superseded by the sewer-rat (*R. norvegicus*). However, as shown in a classical study by Jorge,[65] the invasion of the latter rodent, taking place during the first half of the 18th century, occurred long after the retrogression of plague from the western part of the continent. Some writers considered the progress of civilization which led to higher standards of cleanliness, housing, and sanitation a possible cause for the disappearance of the infection. However, as pointed out by Wu Lien-teh,[160] a lull during the period under consideration was conspicuous not only in Europe but in the Near East, India, and China as well. It seemed likely, therefore, that a natural decline of plague was responsible for the cessation of the outbreaks, rather than extrinsic factors which at best could have been of auxiliary importance only and became fully operative well after the disappearance of the evil from Europe.

Though, as discussed above, autochthonous plague ceased to exist in Europe and many parts of the East, occasional outbreaks due to an importation of the infection from still active foci continued to occur. Thus, while France had become generally free by 1668, an epidemic, believed to have been due to importation from Syria, and sweeping away 50,000 people (Gibbon [33]), took place at Marseilles, France, in 1720; the infection even spread over a great part of Provence but disappeared in 1722. Likewise, while most parts of India seem to have become plague-free at the end of the 17th century, outbreaks, supposed by some to be due to importation from Persia, occurred during the period 1812-21 at Cutch, Gujarat, and Kattyawar, and in 1836-8 at Pali in Rajputana.

Far more important than these outbursts was that, with its deep-rooted tendency to become latent rather than to disappear altogether, plague continued to linger in quite a considerable number of endemic foci. As stated by Wu Lien-teh,[158] the most important of these were situated in and around the Central Asiatic plateau in Russian Turkistan, Semirechinsk, Chinese Turkistan, Inner Mongolia, Outer Mongolia, and Transbaikalia ; in the foothills of the Himalayas in northern India ; in Kurdistan as well as in Central Africa and possibly also in parts of North Africa.

It would seem that by the end of the 18th century plague outbreaks had become frequent in the north-east of Burma and that inroads of the infection into the adjacent part of Yun-nan Province in China took place. As discussed by Wu Lien-teh,[160] during the first half of the 19th century the disease appears to have become established in the extreme west of Yun-nan without, however, prevailing epidemically.

Quite possibly under normal conditions the infection would have continued to smoulder in west Yun-nan without spreading farther afield. Most unfortunately, however, the equilibrium was upset by a rebellion of the Mohammedans commencing in 1855, for the suppression of which troops had to be sent in. Their operations and possibly also movements of refugees provided suitable means for a propagation of the disease which was to prove disastrous not only for China but for many other parts of the world.

Progressing in general by slow stages, plague reached the provincial capital Yun-nan-fu (now Kunming) in 1866 and it took 28 years more before Canton and Hong Kong were reached in 1894. Rather surprisingly, however, the disease appeared at Pak-hoi in Southern Kwang-tung in 1867 already. Serious doubts were entertained as to whether the invasion of this port could have been due to a long-distance sprint of the infection from Yun-nan. However, since it is difficult to see by what other route Pak-hoi could have been reached, one has to accept the explanation of Simpson : [138]

" An epidemic of plague occurs in Yunnanfu in 1866, which decimates the population while they are in the midst of war, and in 1867 Pakhoi, one of the homes of returning troops from Yunnan, is attacked."

It is depressing to reflect that the situation confronting the world when Hong Kong was invaded was in some respects even worse than that at the onset of the pandemic of Justinian's time, in Pelusium. For while in A.D. 542 the means for transporting the infection were slow and comparatively inadequate, and the orbit within which it could spread was limited, in 1894 steamships and railways had replaced the small sailing-craft and caravans of Justinian's day and the new as well as the old world were open to the inroads of plague. It is true that progress in civilization and public health had made it impossible for the infection to gain a firm foothold in modern Europe, but in many other parts of the world ample fuel was available for its perpetuation and spread.

To draw even in broad lines an adequate picture of the progress of plague since 1894 would be a task far beyond the scope of the present survey. All that can be done, and at the same time all that is really needed for the purposes of this monograph, is to come to a general appreciation of the situation in those areas where plague continues to exist or was recently present.

In compiling the present survey, advantage has been taken of the reports on the prevalence of plague in recent years published by Stowman [142] in 1945 and by Kaul [69] in 1949, and for most African plague foci also of the results of an inquiry instituted by the WHO Expert Committee on Plague at its first session (1950). Valuable information on some of the plague foci in East Asia was found in a series of reports by Wilcocks,[156] while in the case of the Americas the studies of Moll & O'Leary [103] served as a useful guide.

PRESENT DISTRIBUTION OF PLAGUE

Asia

(1) *China*

When trying to assess the present plague situation in China it is necessary to consider :

(*a*) The incidence of the disease at Pak-hoi since 1867, and at Canton and Hong Kong since 1894 and the spread of infection into Kwang-tung Province of which Canton is the capital and to which Hong Kong belongs geographically;

(*b*) The invasion of other provinces from Canton and Hong Kong by the sea-route ;

(*c*) Outbreaks due to recent entry of the infection by inland routes ;

(*d*) The reappearance of plague in Yun-nan Province.

(*a*) *Kwang-tung Province.* Epidemics at Pak-hoi, the first port which had been reached by plague, continued to be frequent up to the year 1902

and again from 1910 to 1915, the apparent lull during the period 1903-9 being presumably due to mere lack of information.

Perennial outbreaks of varying intensity continued to occur at Hong Kong until 1923 ; after that year sporadic cases were seen in 1928 and 1929. As maintained by Uttley [150] in a retrospective study on plague in Hong Kong, the decline of the infection could not be ascribed to the sanitary improvements made, because even before 1923 the severity of the outbreaks had diminished elsewhere in south China. In fact, as far as one is permitted to judge from incomplete figures, epidemics had ceased to be perennial in Canton as early as 1916 with subsequent outbreaks in 1923 and 1925 (Wu Lien-teh [160]).

The invasion of Canton in 1894 led almost immediately to a contiguous spread of the infection into Kwang-tung Province, most parts of which became successively affected. This period of expansion was followed by a gradual decline becoming manifest about the end of the first World War. At present only some districts situated at or near the Lui-chow Peninsula in the south of the province and east of Pak-hoi continue to be involved, but it would seem that the situation there has become worse recently, 627 cases having been reported during the period January-September 1950 as against 186 cases in 1949. Presumably, plague also continues on the island of Hainan which was originally invaded in 1900 (Landauer [72]).

(*b*) *Spread of the infection from Kwang-tung to other coastal provinces.* Amoy, the principal port of south Fu-kien, became affected in 1894 soon after the appearance of the disease at Canton and Hong Kong. In 1899 the infection made a long-distance sprint to the port of Newchwang in south Manchuria whereas Foochow, the capital of Fu-kien, was invaded in 1901.

The disease continued to reappear at Newchwang and in its immediate hinterland for some years only. The invasion of Amoy and Foochow on the contrary had serious and lasting consequences. Plague not only continued to appear perennially in these two cities for a number of years but, owing principally to the dense water-borne traffic to smaller seaports as well as up the rivers, most of the counties of Fu-kien Province became successively affected. Though the frequency and extent of the infection seemed to be subject to variation, the recorded incidence of plague being low in certain years, the situation during the decade from 1937 to 1946 was, in general, rather serious. 4,764 cases in 35 counties were recorded in 1943, 7,157 cases in 42 counties in 1946 ; in both years serious epidemics occurred in Foochow city. From 1947 to 1949 there was a gradual decrease in the case incidence, but the situation seems to have become worse again in 1950 when up to the end of September 988 cases were reported as against a total of 368 cases during 1949.

Amoy is now quite free from infection. Rat epizootics continue to occur at Foochow, but it has become possible to prevent the appearance

of human plague through the systematic use of DDT and measures aiming at a reduction of the rodent population.

Plague in Fu-kien Province has become increasingly rural in character. Though pneumonic manifestations are occasionally met with, the bubonic form of the disease is preponderant. Commensal rats alone appear to be responsible for the perpetuation of the infection with *Xenopsylla cheopis* as the sole practically important vector.

It has to be added that owing to intensified traffic over hitherto little-used routes during the second World War the two provinces of Che-kiang and Kiang-si, formerly quite plague-free, became affected in 1940 and 1941 respectively. While the latter province seems to have been free from the end of 1949, a slight incidence of the disease continued during 1950 in Che-kiang where Wenchow remains the only major port on the China coast still suffering from human plague.

(*c*) *Outbreaks due to recent entry of the infection by inland routes.* Since China may be said to have some of the regions composing the Central Asiatic focus at her back door, it is not surprising to find that the country remained open to inroads of plague from these endemic areas.

Mention must first be made in this connexion of north Manchuria which, becoming infected by human agency from the wild rodent foci in Outer Mongolia and Transbaikalia, suffered from pneumonic plague epidemics in 1910-1 and again in 1920-1. Though exerting a grievous toll in deaths, these outbreaks, because they did not involve the local rat population, disappeared as rapidly as they had come once the climatic conditions ceased to favour spread of the infection from man to man.

In 1917-8 pneumonic plague, apparently derived from a wild-rodent focus in the Ordos country of Inner Mongolia (now Suiyuan), invaded Chahar and Shan-si and even spread to a slight extent into Chihli, Shan-tung, An-whei, and Kiang-su Provinces, the number of victims amounting to about 16,000. As described by Wu Lien-teh,[160] further outbreaks, some of them mainly or solely bubonic in character, continued to occur in the north-west, infection being derived either from the Ordos Country or from endemic foci which had become established in Shen-si. In 1931 there was an ominous spread of the infection in Shan-si and Shen-si where, it was claimed, at least 20,000 people succumbed to mainly bubonic affections. To judge from incomplete information, a decline seems to have set in since. A mainly pneumonic outbreak with a case incidence of 485 started at the end of 1941 in Suiyuan and spread in 1942 also to Ningsia, Shen-si, and Shan-si (Fan [26]). The presence of an epidemic which, originally bubonic in character, later assumed pneumonic features was reported in 1949 from Chahar where 69 cases with 66 deaths occurred from July to November in 10 villages. However, since the northern part of the province was stated to have been involved, it is possible that the infection was derived from the foci in south Manchuria or Jehol dealt with below.

To trace the origin of the plague outbreaks proved to exist since 1927 in south-west Manchuria is difficult. Weighing the scanty available evidence, Wu Lien-teh [160] assumed that the infection, the history of which could be traced back to about 1917, originated from a wild-rodent focus in Inner Mongolia, possibly in Chahar which, as noted above, had been involved in the 1917-8 outbreak. However, no doubt can exist that about the time when the presence of plague was confirmed in south-west Manchuria epizootics existed among the commensal rats of the affected localities, especially among *R. norvegicus* (Hsiao [57]), and *X. cheopis* served as vector.

The affected area, at first restricted to the Tungliao region in the west, gradually extended. Even north Manchuria, which had been free from plague since 1921, became involved in the 1946 outbreak, while in 1947, the last year for which information is available, 200 fatal cases were reported from Kirin Province in the east of south Manchuria.

Jehol, where an endemic focus existed at the close of the 19th century, again reported an outbreak in 1933 which was probably due to a fresh importation from south Manchuria. To judge from the scanty information recently available, the plague situation in Jehol became once more serious in 1946, when 15,000 cases seem to have been recorded. The case incidence in 1947, when an energetic anti-plague campaign was conducted, seems to have amounted to about 2,000. Only a few dozen cases were recorded in 1948.

While the epidemics in south Manchuria were preponderantly bubonic in character, occasional pneumonic manifestations were met with. An outbreak of this type (39 cases with 3 recoveries) in 1946 was described by Tieh et al. [146]

(*d*) *Reappearance of plague in Yun-nan Province.* As in the case of the historic plague invasion of Yun-nan dealt with above, it is impossible to state exactly when the recent re-entry of the infection into the province commenced. Presumably now as then the manifest appearance of the disease had been preceded by unnoticed invasions of villages just across the Burma border. It is certain that as the result of a serious outbreak at Nam Kham, Burma, in 1939 the infection not only reappeared early in 1940 in that area, but also spread across the Shweli River into Chinese territory (UNRRA Report [148]), where, however, the Mungmao district alone became involved.

While no information is available for 1941 and 1942, plague was again observed in the autumn of 1943 in a locality about two days' walk from the Burma border. In 1944 the disease not only assumed epidemic proportions in that area, but also appeared in three other districts of Yun-nan, a total case incidence of 542 with 247 deaths being recorded.

A further extension of the affected area took place in 1946 when the infection, having crossed the Salween River, reached Paoshan, the principal city of west Yun-nan. The situation was not much changed in 1947 and

there seems to have been less plague in 1948. However, according to information received in 1949, the infection, having crossed the Me-kong River, appeared in Ta-li. At the same time plague was reported from three hitherto-unaffected counties and a traveller from Paoshan was found to be suffering from the disease upon arrival at Kunming. The total plague incidence in 1949 was 283 cases with 92 deaths as against 168 cases with 36 deaths in 1948.

As will be gathered from the account given above, rat-caused plague is entrenched in parts of south China as well as in the north-west of the country and recently perennial outbreaks of the same type occurred in Yun-nan as well. On the other hand, plague has been either altogether absent from the central provinces or the infection, if imported upon rare occasions, has failed to establish itself.

It should be noted in the latter connexion that plague was introduced into Shanghai in 1908 but, though infected rats continued to be found every year until 1916, no widespread epizootics resulted and human cases never became numerous. From 1917 onwards rat falls were few and far between or altogether absent and Shanghai has been quite free from plague since 1927.

Likewise, when plague, believed to have been introduced by bacterial warfare, appeared at Chang-teh, Hu-nan Province, in 1941 (King,[70] Fan [26]), no permanent harm resulted even though in the following year a rat epizootic was rampant in that town and almost 100 human cases occurred. The infection disappeared early in 1943 and since then Hu-nan has been as free from plague as it was before the end of 1941.

The absence of plague from central China or its failure to establish itself cannot be ascribed to a paucity of commensal rats or of *X. cheopis*, both of which abound everywhere. Likewise, as confirmed by the observations in Hu-nan, there is no reason to assume that plague-resistant rat strains prevail in central China. Probably, however, peculiarities of the seasonal incidence of *X. cheopis* go a long way to explain the freedom of central China. As pointed out by Wu,[157] the incidence of that flea in Shanghai was low during the months of the plague season in south China, which commences in early spring. He admitted that the autumnal incidence of the disease in the north exhibited a dangerous coincidence with the *cheopis* season in Shanghai, but pointed out that under the existing conditions the southern plague foci alone were potentially dangerous for an importation of the infection into Shanghai.

(2) *Burma*

Though, as discussed above, the historic as well as the recent invasion of Yun-nan could be traced back to Burma, it would be rash to assert

was not alarming. However, though the case incidence reported for the period 1945-7 was low, incomplete figures for 1948 and 1949 indicated a disquieting increase of the infection-rate (3,422 cases with 3,365 deaths and 874 cases with 844 deaths respectively), the residency of Jogjakarta in central Java being most heavily affected.

The recent incidence of plague in Java is shown below :

	1950	*1951*		*1952* *	
	Cases	*Cases*	*Deaths*	*Cases*	*Deaths*
Eastern Java	8	310	221	52	13
Middle Java	3,450	4,292	1,993	1,524	820
Western Java	129	549	52	155	6

* Incomplete

It will be noted that the plague situation in eastern Java was not serious. In fact, this part of the island seemed to be free from the infection during the second half of 1952. On the contrary, middle Java, particularly the residencies of Jogjakarta and Surakarta were seriously involved. Plague caused less havoc in western Java, but there can be no doubt that there as well as in middle Java endemic foci exist.

As pointed out by Park,[110] in Java, where the temperature remains fairly uniform throughout the year, no pronounced seasonal incidence of the disease was to be expected. Still, the plague mortality showed a tendency to increase during the third quarter of each year, reaching its maximum in December and January ; then a decrease set in which lasted until July.

Human plague in Java was mainly bubonic, but cases with pneumonic features accounted for 6%-8% of the total incidence of the disease and outbreaks of pneumonic plague, usually claiming 2-10 victims only, were met with (Wu Lien-teh,[159] Wilcocks[156]).

As summarized by Wilcocks,[155] the brown Malayan house-rat, *R. rattus diardi*, *R. concolor* inland, and to a very small extent *R. norvegicus* were implicated in the plague outbreaks in Java, the first-mentioned rodent being considered as the chief culprit. *X. cheopis* was the principal vector.

(6) *India*

In a discussion on the endemicity of plague in India, Sharif[135] postulated that the infection is at present entrenched in the following foci :

(*a*) Three endemic centres situated near the foot of the Himalayas which, though occasionally appearing to have been independent of one another, possibly form part of a big sub-Himalayan focus. These centres were responsible for plague outbreaks in east Punjab, the United Provinces, and districts of Bihar north of the Ganges.

(*b*) One focus in central India comprising the watersheds of the Vindhya, Bhanrer, and Maikal ranges, and the Mahadeo hills.

(*c*) Three centres in southern India situated respectively in the watersheds of the Western Ghats in Bombay State and Mysore ; in watersheds located in the districts of Salem, Coimbatore, Nilgiri, and Madura ; in the Hyderabad State. All three foci have been very active lately.

There can be no doubt that the endemic centres in southern India became established after the city of Bombay had become infected in 1896. As stated by Sharif,[135] the first of them (Bombay and Mysore States) had been reached by plague in 1898. Nilgiri became involved in 1903 (George & Timothy [32]). The infection spread in 1898 from the then Bombay Presidency into the adjacent parts of Hyderabad State, but the situation there became serious in 1911 only when Hyderabad city became affected (Rao [120]).

It seems possible on the other hand that the endemic foci in the Himalayas are partly of long standing. Endemicity has been known to exist in the districts of Gharwal and Kumaon since 1823, but, as assumed by Gill,[36] the infection persisting there was perhaps " a relic of the great pestilence in the 17th century ". Though these areas, which are situated within the region involved at present, are known to have been responsible for plague outbreaks up to the year 1877 only, latent infection might have continued to persist in some part of the Himalayan foothills to become active again early in the present century.

The mortality from plague in India from 1898 to 1948 may be summarized as in table I :

TABLE I. MORTALITY FROM PLAGUE IN INDIA DURING THE PERIOD 1898-1948 [a]

Period	Total deaths from plague	Annual average	Deaths during each period expressed as percentage of total deaths 1898-1948
1898-1908	6,032,693	548,427	47.88
1909-1918	4,221,528	422,153	33.51
1919-1928	1,702,718	170,272	13.52
1929-1938	422,880	42,288	3.36
1939-1948	217,970	21,797	1.73
Total 1898-1948	12,597,789	247,015	100.00

[a] After Bhore,[61] Kaul [69]

It will be noted that almost half of the total plague deaths in India occurred within the period 1898-1908 and more than three-quarters up to 1919. Nevertheless, as shown by the annual figures for the period 1939-52, set forth in table II, the recent plague situation in India was not as reassuring as it might seem at first glance. The condition was quite favourable in 1942

TABLE II. ANNUAL MORTALITY FROM PLAGUE IN INDIA DURING THE PERIOD 1939-52

Year	Total plague mortality	Bihar	Bombay State	Central Provinces (Madhya Pradesh)	Madras	Punjab	United Provinces (Uttar Pradesh)	Other areas	Hyderabad State [a]	Mysore State [a]
1939	26,257	1,938	1,472	852	324	—	21,682	9	6,758	2,352
1940	19,799	1,040	5,573	283	1,169	—	11,725	9	7,500	2,593
1941	11,984	129	5,311	761	1,726	—	4,035	22	2,713	5,417
1942	10,577	108	680	129	701	—	8,953	6	657	3,776
1943	13,578	266	715	144	4,885	1	7,556	11	1,498	3,686
1944	21,525	834	2,514	910	1,738	61	15,454	14	5,233	5,357
1945	29,751	1,523	11,779	575	1,644	203	14,024	3	6,631	8,016
1946	32,997	8,689	3,405	189	2,254	245	18,206	9	4,026	2,694
1947	41,745	6,258	2,129	2,442	3,078	1,704	26,126	8	1,791	1,502
1948	9,757	902	855	2,426	978	196	4,384	16	811	1,128
1949	7,587	647	722	2,860	151	227	2,904	76	2,103	982
1950	6,881	557	93	3,804	42	3[b]	2,073	204	719	265
1951	1,841	8	7	475	60	—	1,276	20	98	542
1952	1,007	0	1	457	9	—	504	36	1	272
Totals	235,286	22,894	35,261	16,407	18,759	2,640	136,882	443	40,569	38,972

[a] Not included in totals [b] East Punjab only

but afterwards became worse once more, the annual incidence-rates for 1945, 1946, and 1947 being increasingly in excess of that recorded in 1939. Kaul [69] assumed that the war with Japan was at least partly responsible for this recrudescence because other health data available in India at the time also showed a turn for the worse. However, the acute food scarcity existing in 1946 and 1947 in certain parts of the country, which necessitated large-scale movements of grain from central collecting stations to various provinces, might have facilitated the dissemination of plague, and a similar influence was probably exerted by the movements of large groups of people in connexion with the partition of India in August 1947. Since these unfavourable conditions were of a temporary nature, it would be tempting to ascribe the considerably reduced incidence of the disease in 1948 and 1949 to an improvement of the general situation. It is, however, disquieting to note that the 1950 plague mortality in the Central Provinces (now Madhya Pradesh) was markedly in excess of the figures reported for the three previous years. Still, the plague mortality there, as well as in India in general, was considerably lower in 1951 and 1952.

Generally speaking, in India as well as in China plague is now a rural rather than an urban problem. It should be noted in this connexion that Bombay city, where plague had been rampant up to 1923 and perennial manifestations had continued to exist until 1934, is now almost free. In Calcutta, which apparently became affected in 1895 when suspicious cases were noted among a group of soldiers recently arrived from Hong Kong (Rao [119]), and where the infection persisted until 1925, considerable outbreaks, due possibly to an importation from Bihar (Greval [44]), took place in 1948 and 1949. Only two autochthonous cases were recorded in 1950 but their number rose to 16 in 1951, and to 54 in 1952.

Dealing with the seasonal incidence of the disease in his classical study " Twenty years of plague in India ", White [155] maintained that

" epidemics normally attain their maximum severity in Bombay in October ; in the Central Provinces in February ; in the United Provinces and Bihar in March ; and in the Punjab in April. In the remaining provinces, taken together, March is the month of maximum mortality ".

While the bubonic type of the disease is by far the most frequent in India, small outbreaks of pneumonic plague have been observed occasionally, recently by Seal [133] and Seal & Prasad [134] at Calcutta and Gaya (Bihar).

So far no evidence has been found that wild rodents played a role in the causation of plague outbreaks in India, the common species of commensal rats forming the usual reservoir of the infection. However, as will be discussed later, the bandicoots which are apt to come or even live near man, also deserve some attention. The " Indian " plague flea *X. cheopis* is the typical vector of the infection.

(7) *Western Asia (Iran, Iraq, Turkey in Asia, and Syria)*

A joint consideration of the plague regions in Western Asia seems justified in so far as according to Tholozan [144] and Payne [112] the mountains of Kurdistan were the chief endemic centre for a large area comprising Persian Kurdistan and adjacent parts of Persia, Turkish Kurdistan, and parts of Mesopotamia. Payne added that from this area plague extended

" to Northern Persia on the shores of the Caspian (Resht) in 1877, to Baku on the western and Astrakhan on the northern shore of that sea ; and up the Volga to the village of Vetlianka and its neighbourhood in 1877-79 ".

Presumably the persistence of the infection in the interior of western Asia was in part responsible for past plague invasions of Syria and Palestine, but it must be noted that these countries were open to inroads of the pest by the sea-route as well.

Findings made by Baltazard et al. [2] after two limited outbreaks of pneumonic plague had been observed in the south of Iranian Kurdistan in October-November 1947 have added much to the scanty information hitherto available in regard to the western-Asiatic focus. Baltazard and

The incidence of the disease in that province since its appearance in 1937 was as follows :

Year	*Month*	*Cases*	*Year*	*Month*	*Cases*
1937	January	9	1944	February	2
1938-9	—	0	1945-6	—	0
1940	February	4	1947	December	1
1941	—	0	1948	January-February	7
1942	October, December	14	1949	March	2
1943	December	5	1950	March	2

It will be noted that the plague season fell into the period from October to February when during the midsummer rains day and night temperatures were high (Davis [20]). Occasional pneumonic cases were noted.

Though so far no direct proof seems to have been obtained, it is most probable that in Northern Rhodesia, as elsewhere in South Africa, gerbils are the primary reservoir of plague, and that the multimammate mouse is instrumental in bringing the infection to man. Possibly swamp-rats (*Otomys, Dasymys, Pelomys*) form an accessory reservoir.

The gerbils were found to harbour *X. philoxera*, *X. hipponax*, and occasionally *Dinopsyllus ellobius*, which was the only flea infesting the swamp-rats. It is interesting that *X. philoxera*, a notorious vector, was with one exception found solely in those areas of Barotseland where plague was enzootic. However, since *X. hipponax*, which abounded in the regions free from *philoxera*, might be a vector, no undue stress ought to be laid upon the absence of the latter flea.

R. natalensis was infested by the wild-rodent fleas mentioned above and by *X. brasiliensis* which no doubt was of main importance in conveying the infection to man (Davis [20]).

North America

(1) *United States of America*

The incidence of human plague in the USA from 1900 to 1950 may be summarized as follows :

(*a*) *Rat-caused epidemics* (Mohr [102])

Locality	*Year*	*Cases*	*Deaths*	*Remarks*
San Francisco, Calif.	1900-4	120	114	Plague existed probably before 1900 (see below).
	1907-8	186	92	
Seattle, Wash.	1907	3	3	2 of these patients and probably also 3 suspected victims had pneumonic plague. Rat infection persisted until 1917 (Fricks [30]).

Locality	*Year*	*Cases*	*Deaths*	*Remarks*
New Orleans, La.	1914-5	31	10	
	1919-21	25	11	
Pensacola, Fla.	1920	10	4	
Galveston, Tex.	1920	18	12	
Beaumont, Tex.	1920	14	6	
Los Angeles, Calif.	1924	41	34	33 of the patients had lung features, but these were in part secondary in nature (Wu Lien-teh [159]).
Totals		448	286	

(*b*) *Infections contracted from wild rodents or through their fleas* (after Link [76, 77])

	Year of detection of:			
State	*Wild-rodent plague*	*Human plague*	*Cases*	*Deaths*
California	1908	1908	80	52
Oregon	1935	1934	1	1
Utah	1936	1936	1	0
Nevada	1936	1937	1	0
Idaho	1936	1940	1	1
New Mexico	1938	1949	6	3
Arizona	1938	1950	1	0
Totals	1908-51		91	57

Remarks: While most of these cases occurred singly, one pneumonic outbreak of wild-rodent origin took place at Oakland, California, in 1919 and claimed 13 victims.

Taken at their face value, these statistics suggest that in the USA, as well as in South Africa and some countries of South America, an initial " murine " phase of plague eventually led to wild-rodent infection. However, according to some observers, the appearance of the disease in North America was due not to its recent introduction by the sea-route, but to an early importation through wild rodents which, coming overland across the Bering Strait from Central Asia, brought *Pasteurella pestis* with them as a population regulator (Jacobsen,[64] Meyer [96]). Meyer [96] pointed out in this connexion that it was difficult to believe in as rapid a spread of wild-rodent plague as ought to have taken place had the infection been recently introduced. Likewise he maintained that as a rule wild-rodent plague in North America

" remained confined to foci and no convincing evidence has been produced that some wide-ranging animal has started new epizootics ".

While it would be out of place to discuss this point at the present juncture, it should be noted here that it is difficult to believe in the fascinating hypothesis set forth above for the following reasons : First, should plague have come overland from Central Asia, it should have appeared in Canada earlier than in the USA. Actually, however, it appeared in that Dominion much later than in the USA and was no doubt imported from there. Further, as will be discussed in chapter 2, the plague strains isolated in the USA

are biochemically different from those in Central Asia. Finally, it is by no means certain that the usually given date of entry of the infection into the USA was the actual one. Kinyoun [71] felt sure that plague had been present in San Francisco in 1898 already and quoted a report claiming that " in all probability, the disease had existed on the Pacific Coast since 1896 ". Seeing that plague had reached the coast of China as early as 1867, one might even wonder if it had not been carried to California long before 1896, so that there was far more time for a spread of the infection to the hinterland than is usually allowed for.

As will be gathered from the first of the statistics inserted above, rat-caused plague, though repeatedly appearing in the USA, has never shown marked tendencies to spread or persist. This is all the more remarkable because, with the exception of Washington State, the commensal rats in the plague-affected localities showed a considerable infestation with *X. cheopis*. *R. norvegicus* was the most preponderant rat species.

Wild-rodent plague was present not only in the States where it led to human infection, but also in others, as shown by the following (Link [76]) :

State	*Year of detection*	*State*	*Year of detection*
California	1908 *	Arizona	1938
Montana	1935	New Mexico	1938
Oregon	1935	Colorado	1941
Idaho	1936	North Dakota	1941
Nevada	1936	Oklahoma	1944
Utah	1936	Kansas	1945
Wyoming	1936	Texas	1946
Washington	1937		

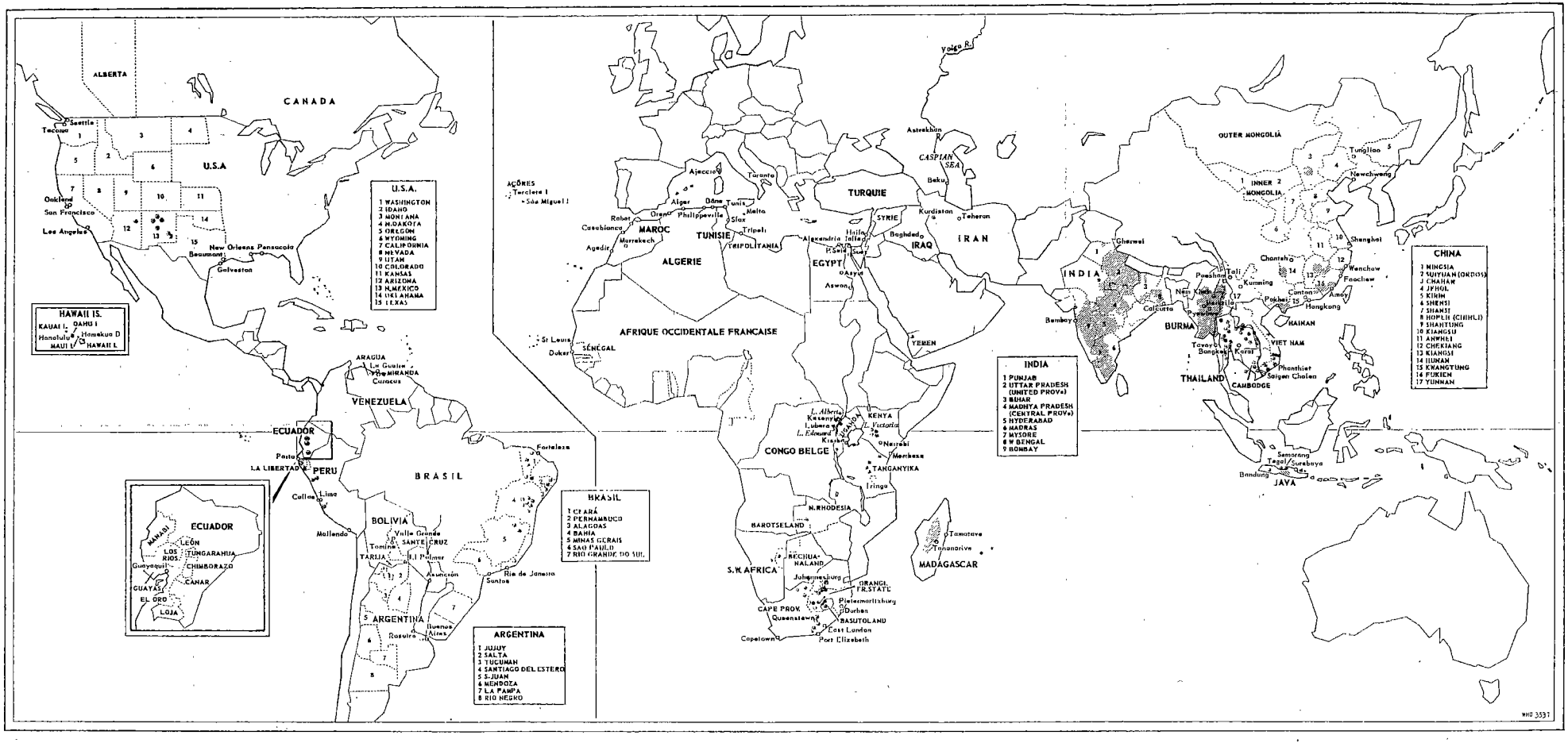
* Wild-rodent plague had evidently existed in the Contra Costa county of California since 1903 at least.

The low incidence of human infections derived directly from the wild rodents or through their fleas is in striking contrast to the large area, comprising 131 counties in 15 States, where evidence of plague among these animals has been found. Discussing this problem, Meyer [95] stated that the disease

" occurs among wild rodents in wooded or rural districts uninhabited or only sparsely inhabited by man, but human contact with the infective agent probably is established in exceptional instances ".

Eskey & Haas,[25] in their classical study on " Plague in the western part of the United States ", came to the conclusion that at least three groups of rodents constituted the great primary reservoirs of the infection —the ground-squirrels, which were widely involved in the coastal regions and in the northern part of the Intermountain Plateau ; the wood-rats, which formed the plague reservoir in the southern deserts ; and finally the prairie-dogs, which harboured the infection in the plateau region of Arizona and New Mexico. Mohr [102] endorsed this opinion but also incriminated sage-brush voles and certain meadow mice.

FIG. 3. GEOGRAPHICAL DISTRIBUTION OF CASES OF HUMAN PLAGUE REPORTED FROM 1948 TO 1952



Map prepared by WHO on the basis of official data made available by governments.

The dots and hatching represent areas where human cases were reported from 1948 to 1952.

While, as noted above, the danger of a spread of plague to man through direct contact with wild rodents or through their fleas is slight, secondary involvement of the rats or other rodent species living near man might greatly enhance the chances for human infection.

The possibility of such a transition had been claimed by the advocates of an overland introduction of the disease from Central Asia who pointed out that up to 1908 large numbers of ground-squirrels had been imported into San Francisco for culinary purposes (Meyer [94]). It was also suspected that an infection recently derived from wild rodents had been responsible for the epizootic causing the 1924 Los Angeles epidemic (Wu Lien-teh [159]), and that the presence of plague among the rats of Tacoma (Washington State) in 1942 and 1943 had been due to an importation of the infection from the hinterland through grain transports (Hundley & Nasi [59]). Wild-rodent fleas had been found upon several occasions on commensal rats and isolated instances of rat plague had been detected in urban centres as well as in rural areas of California (Meyer & Holdenried,[97] Mohr [102]).

Working recently on a Californian ranch round which wild-rodent plague was present, Meyer & Holdenried [97] were able to prove the presence of the infection in rats (*R. rattus rattus* and *R. norvegicus*) which were in part infested with ground-squirrel fleas. *L. segnis* was the preponderant specific parasite on these rats, while *Nosopsyllus fasciatus* was rare and *X. cheopis* absent. However, in the opinion of the authors, the wild-rodent fleas might have been able to maintain the infection which they had introduced. Meyer & Holdenried came to the important conclusion that :

" on numerous occasions, rural plague has swept with destructive force through native rodent populations with little if any danger to human beings, but when domestic rats (*Rattus*) or mice (*Peromyscus*, *Microtus*, or *Mus*), which may live in man's immediate environment and the fleas of which will attack and thus infect him, are involved, a much more serious problem arises ".

Fortunately, this potential danger is fully realized, due attention being paid to rodent and flea control in and round human settlements.

(2) *United States of America — Hawaii*

Plague became manifest in Honolulu in November 1899, a few months after patients suffering from the disease had been found on two ships arriving from Hong Kong. The infection not only persisted for about 10 years in Honolulu, but spread to adjacent districts as well as to other islands of the archipelago :

Locality	*Period*	*Cases*
Oahu Island :		
Honolulu	1899-1910	187
Other places	1902-7	41
Kauai	1901-6	15
Maui	1900	9
	1930-2	6
	1938	1

Locality	*Period*	*Cases*
Hawaii Island :		
Hilo sector	1900-12	43
North Hilo	1918	2
Hamakua district	1910-45	111
	1949	1
	Total cases 1899-1949 =	416

As will be noted, within recent years human plague was present in the Hamakua district of Hawaii Island only, where after a clear interval a case was recorded in 1949 and where rodent infection persists to date. Positive results were also obtained in 1949 with two batches of pooled tissues from Maui rats.

The district of Hamakua contained many sugar plantations and, as stated by Eskey,[24] human-plague infection was contracted in the cane-fields rather than in the houses. *R. rattus alexandrinus* formed half of the rat population in the district, but *R. norvegicus* and *R. hawaiiensis*, a small mouse-like animal rarely entering houses, were also met with. *X. cheopis* constituted 70% of the rat fleas.

The mortality from human plague was high since, as stated by Hampton,[46] 360 of the 397 patients seen from 1899-1933 and all 17 observed from then up to 1944 died.[e]

(3) *Canada*

An importation of plague from the USA being anticipated, a large-scale wild-rodent survey was made in Western Canada in 1938. No positive results were obtained (Gibbons [34]).

However, in 1939 the prevalence of an epizootic among the ground-squirrels of Alberta was noted, and bacteriological findings proving the presence of plague were made in one of these animals taken at Stanmore, a locality about 180 miles (290 km) north of the boundary separating Canada from Montana, USA. Since, as noted above, wild-rodent plague had been found to exist in that State since 1935, there can be no doubt that the infection had spread from there into Canada.

Further evidence of plague in the Alberta ground-squirrels was found in 1939-42, and again in 1945. Spread of the infection from Alberta to an adjacent region of Saskatchewan was noted in 1946 (Humphreys & Campbell [58]). Pools of ground-squirrel fleas were found positive for plague in 1947 both there and in Alberta.

The ground-squirrels involved in Canada belonged to the species *Citellus richardsoni richardsoni*. They harboured *Opisocrostis labis*, *O. tuber-*

[e] Besides in the Hawaii archipelago, plague has been reported within recent years in another Pacific island, New Caledonia, where early in 1941 a village near the seaport of Goro, situated about 30 miles east of Noumea, the capital, became affected. Up to March 1, nine cases with six deaths were recorded, eight of which were stated to have occurred among pupils of a native school. Prior to this epidemic, which was bacteriologically confirmed, an unusual mortality among the " brush or coconut-tree " rats in the Goro region had been noted. It was also stated that plague was " latently endemic in small areas of the colony ".

The entire population of the plague-affected area was vaccinated. Plague serum and sulfathiazole were used for treatment.[101]

culatus, and *Oropsylla rupestris*, three species of fleas found able by Eskey & Haas [25] to convey plague under experimental conditions.

It is reassuring that according to Humphreys & Campbell [58] commensal rats seemed not to have colonized to any extent in Alberta. However, they infested the large municipalities of Saskatchewan.

Thus far the existence of human plague has not been proven in Canada, but it is possible that one fatal case occurred in Alberta before the existence of wild-rodent infection had been established. The man in question was breeding minks which he fed with ground-squirrels from a locality later found to be plague-affected. Several of the minks died and their breeder got scratched whilst skinning one of these animals (Gibbons & Humphreys [35]).

South America

(1) *Venezuela*

Plague, believed to have been imported from Trinidad, appeared in 1908 in the port of La Guaira and soon spread from there to Caracas, the capital of Venezuela, where it continued to occur until 1919, leading to a total of 204 cases with 99 deaths.

Even before this " urban " phase had come to an end, the disease had spread in 1910 to rural areas of Miranda State, in one district of which it continues to exist. An adjacent region of Aragua State, reached by the infection in 1939, also remained involved. As stated by Isaac Riaz,[63] the two affected districts cover an area of 1,000 km² (386 square miles)—1 % of Venezuelan territory.

The plague incidence in the two affected regions from 1910 to 1951 was :

Miranda State

Year	*Cases*	*Year*	*Cases*
1910	35	1928	10
1911	18	1932	10
1914	16	1933	7
1919	110	1950	5
		1951	7

Total cases 1910-51 = 218

Aragua State

Year	*Cases*	*Deaths*
1939	11	8
1943	17	5
1948	7	3
1949	2	1
1939-49	37	17

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

[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]).

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

on the outskirts which bordered on fields. It was the predominant species on the rats of farm-houses, and also in premises infested by bandicoots.

Wild-Rodent Fleas

Annex 1, table V (see page 638) shows that, in contrast to the commensal rodents which are infested by a few, but, as a rule, widely distributed, flea-species, the groups of wild rodents found in the various plague-areas are usually parasitized by diverse flea-species which show, almost invariably, a local, or at most a limited, distribution. It is, however, important to note that, as far as the individual wild-rodent species are concerned, marked differences exist in this respect, some of the animals having only one or two specific fleas, while others have several. In this connexion, Eskey & Haas [54] recorded the interesting observation that the rodents parasitized by only one or two flea-species usually showed a higher degree of infestation than those harbouring several kinds of fleas. It is impossible to decide to what extent this is due to the presence of an antagonism between the various flea-species — as is supposed to exist in the case of *X. cheopis* and *X. astia*—but it seems more likely that the difference is due to the advantages derived by the fleas from close adaptation to one host or a few closely-related hosts.

Eskey & Haas further noted that the degree of flea infestation depended also on the size of the rodents concerned. These findings, which seem to have been confirmed by the field observations of Humphreys et al.,[112] are in accord with those made in case of the commensal rats.

As far as the incomplete information available goes, it would appear that, generally speaking, in the case of the wild-rodent fleas, no sharp line of demarcation can be drawn between host-dwelling and nest-dwelling species, as even those fleas which are known to have a tendency to stay on their hosts also occur in considerable numbers in the burrows of the latter. This situation is well illustrated by the case of the tarabagan flea, *Oropsylla silantievi*. Observations made in Manchuria and Transbaikalia [281] suggested that this flea had a marked tendency to stick to the carcasses of its hosts and to the pelts removed from them. Yet, Golov & Ioff,[84] who had opportunities for more thorough observations in south-east Russia, found that, even though *O. silantievi* had a propensity for staying on its hosts, the majority of these fleas nevertheless dwelt in the burrows.

However, in the case of some flea species, more clear-cut differences seem to exist in this respect. Thus, Kusenkov [141] found that, in the North Caucasus area, *Citellophilus tesquorum* was found principally on the susliks, while *N. setosa* was found mainly in the suslik-burrows. The seasonal incidence of these two predominant suslik-fleas was also different. *N. setosa* was most frequent in spring, diminished in numbers in summer, and became frequent once more in autumn. The host-dwelling *C. tesquorum*, on the contrary, became apparently more numerous pari passu with the increase

of the suslik population due to breeding, showed its maximal incidence in July, and then began to decrease.

As shown by the observations of Nikanoroff & Gaiski [186] and of Borzenkov et al.,[22] wild-rodent fleas are not merely capable of subsisting during the hibernation period of their hosts, but may even remain numerous throughout the winter. However, as has been established by most observers, they do not survive or stay long in uninhabited burrows, even under favourable extrinsic conditions.

Eskey & Haas [54] stated in this connexion that

"... the assumption has been advanced at times that fleas may continue to survive in abandoned nests and burrows for many months and, if plague infected, such fleas are believed to be capable of transmitting the infection over long periods. Field observations, however, did not support this view. Practically no nests yielded fleas that did not show evidence of recent occupation. Even nests which were excavated within a month after ground squirrels had abandoned them were devoid of fleas".

The opinion that wild-rodent fleas do not survive long in burrows deserted by their hosts was shared by Kolpakova & Lippert [139] and by Fedina,[61] who found that, under such circumstances, the fleas sooner or later emigrated.

Tiflov & Potapov,[256] who also noted that the suslik fleas left empty burrows, stated that *N. setosa*, while capable of surviving well on the ground outside the burrows for 24 hours, rarely survived there for 48 hours or more. No doubt can exist, however, that wild-rodent fleas may be present in large numbers on the ground near the openings of inhabited burrows. Eskey & Haas stated in this connexion that, by simply drawing a white cloth over the ground, they were able to collect in such a location over 200 ground-squirrel fleas in a few hours.

It is of interest to note that, owing no doubt to the abundance of free-living fleas not only in the nests of the burrows, but also in their runways and round their openings, wild rodents which were released after they had been captured, marked, and de-fleaed, were found to be heavily re-infested when caught again.[106, 173, 241] Holdenried and his co-workers were thus able to take 1,835 fleas in 37 collections from a single squirrel.

Probably, wild-rodent fleas, although capable of staying away from their hosts in or near the burrows, are not well adapted to a free existence in more remote locations. This is suggested by the observation of Vincke & Devignat [265] that, in contrast to the rat-*Xenopsylla* and *Ctenocephalides*, two wild-rodent species—namely, *Dinopsyllus* and *Chiastopsylla*—were incapable of subsisting in debris on the surface of the ground.

Interchange of Fleas between Commensal and Wild Rodents

Observations in some plague areas have shown that, in locations where commensal and wild rodents could meet, an interchange of their fleas was

apt to take place. As pointed out with much reason by Hopkins,[109] the relative frequency of such a flea exchange indicates the degree to which contact between the two types of rodents takes place.

In the western areas of the USA, the occurrence of ground-squirrel fleas on rats was recorded by some early observers, e.g., Doane,[47] Fox,[66] and McCoy & Mitzmain.[167] Identical findings were made in the course of a recent extensive survey by Prince.[208]

However, Eskey & Haas,[54] who examined a considerable number of ground-squirrels and rats obtained from locations where these rodents intermingled, found an interchange of their fleas exceedingly rare. In their experience " many more fleas normal to native rats and mice than fleas of ground squirrels were removed from domestic rats ".

Findings made during a survey undertaken on a limited scale in a rural area of California by Meyer & Holdenried [178] did not confirm the rarity of ground-squirrel fleas on commensal rats because about a quarter of the species collected from these animals consisted of such fleas, mainly *D. montanus*. On the contrary, 53 ground-squirrels taken from dumps and ranches heavily infested with rats yielded no rat fleas. Meyer & Holdenried assumed, therefore, that " the transfer of rat fleas to squirrels is less frequent than the reverse ".

As established in the course of a recent study by Holdenried,[105] in the southern and southwestern areas of the USA, *Polygenis gwyni*, an efficient plague-vector, regularly infested Norway rats as well as the cotton-rat, *Sigmodon hispidus*.

Less detailed evidence indicates that an interchange of wild-rodent and commensal-rat fleas also occurred in other plague-areas.

Thus, to judge from a record by Hecht,[91] *Rhopalopsyllus* subspecies were predominant on the field-inhabiting rats, as well as on the wild rodents, examined during a survey in Aragua State, Venezuela. Jordan [132] examined collections of fleas from Peru, and noted the occasional presence of *X. cheopis* and *L. segnis*, respectively, on some wild-rodent species; Ramos Díaz [211] made a similar observation of *X. cheopis* on guinea-pigs in the Ecuadorian Sierra.

As previously noted, *X. brasiliensis* was found on both wild and commensal rodents in South Africa and Uganda. In Uganda [109] and in the Belgian Congo,[44] this flea, as well as *X. cheopis*, was found on the semi-domestic *Arvicanthis abyssinicus*. According to Heisch,[92] " *X. cheopis* and *D. lypusus* were the predominant wild rodent fleas " in the Rongai area of Kenya, where *X. brasiliensis* appeared to be rare.

X. astia was sometimes found to be abundant on semi-domestic rodents in India. *X. cheopis* and *X. brasiliensis* were far more rarely recovered from such animals or their nests.[70, 71, 122, 124, 231]

It will be gathered, therefore, that an interchange of fleas between commensal and wild rodents is by no means uncommon. Fortunately,

as has been discussed in chapter 6, instances where such fleas served as vectors of *P. pestis* were far less frequent.

Role of the Rat Flea in Plague

It is often overlooked that the credit for the discovery of the role played by the rat fleas in the transmission of plague belongs to Ogata [190] and to Simond,[233] who was obviously unaware of Ogata's publication when starting his own investigations in 1897.

Working in Formosa, Ogata reached, on epidemiological grounds, the assumption that plague was an insect-borne infection and suspected the rat fleas in particular, stating that " one should pay attention to insects like fleas, for as the rat becomes cold after death, they leave their host and may transmit the plague virus direct to man ".

Obtaining positive results in mice which had been inoculated with a suspension of triturated fleas collected from plague rats, he was able to prove that these insects had actually ingested *P. pestis*.

As dramatically described in a paper published by Simond in 1936,[234] his attention was attracted in 1897 to a possible role played by the fleas in the transmission of plague through observations on the presence of plague pustules (" phlyctènes précoces ") in about 5% of the patients. These pustules, often found on the legs of the sufferers and invariably on sound, unexcoriated parts of the skin, seemed to represent the portal of entry of a flea-borne infection, the more so as their appearance sometimes suggested that they were due to flea-bites.

On the other hand, Simond established that, under natural conditions, infection by feeding could not play an important role in the spread of plague from rat to rat because, as shown by laboratory tests, enormous numbers of *P. pestis* had to be ingested to produce an infection per os and, moreover, the pathological findings in animals infected by this route were altogether different from those found in naturally infected, or cutaneously inoculated, rats.

He obtained direct proof of the role of fleas in the transmission of plague by :

(1) demonstrating the presence of *P. pestis* in smears prepared from fleas which had been collected from plague rats and the absence of these bacilli in specimens from healthy rats;

(2) obtaining positive results with the technique used by Ogata;

(3) successfully infecting healthy rats which had been confined in a wire cage and suspended above a plague-infected rat in a glass jar, and failing to get positive results when conducting such experiments with rats which had been freed from ectoparasites.

Finding that fleas which had ingested plague bacilli could continue to harbour them for prolonged periods, Simond correctly assumed that these insects were apt to play a role in the perpetuation, as well as in the transmission, of the infection. In fact, the only one of his postulations which was not confirmed by further research was that the conveyance of plague by the fleas was invariably due to an entry of their infected faeces into the bite-wounds.

Though the findings of Ogata and Simond found the early support of some investigators, such as Tidswell [252, 253] and Gauthier & Raybaud,[68, 69] they were, in general, ignored or even ridiculed. Lowe,[153] who in 1942 published an enthusiastic appreciation of Simond's work, noted in this connexion that an editorial published in the *Indian Medical Gazette* in 1902 had spoken of the " worthlessness " of Simond's findings, the validity of which had been " pretty completely demolished " by the work of some other observers.

However, further evidence of the importance of fleas in the transmission of plague was furnished by other workers, e.g., Verjbitzki [264] and Liston,[149] and from 1905 onwards a systematic investigation of this problem formed one of the principal objects and, at the same time, one of the monumental achievements of the Plague Research Commission. As aptly stated by Wu : [281]

" . . . these classical researches . . . still constitute the foundation of our knowledge of the transmission of plague by the rat-flea. Later workers have added considerably to our conception of the mechanism of infection, the respective roles of *X. cheopis* and *X. astia*, the problem of ' preservers ' and of the ' infective ' and ' infected ' flea, the significance of the zoological and climatic factors, and so on, but their observations, epoch-making and conclusive though these are, serve to round out the picture rather than to alter the perspective in any important particular."

At the 1909 Bombay Medical Congress, Lamb read a paper on " The etiology and epidemiology of plague " [143] which summarized a more elaborate report on the early work of the Plague Research Commission published by him in 1908,[142] and quoted the following evidence in support of the role of the rat fleas in the spread of plague.

1. Attempts to convey plague from rat to rat by direct contact, aerial transmission, or soil infection, or by means of contaminated food, had shown that these modes of infection either played no role at all or did not play a role under natural conditions.

2. An enormous number of successful transmission-experiments had been carried out with rat fleas, all other means of infection having been rigorously excluded.

3. Experiments carried out, mainly with guinea-pigs but also with rats and monkeys, in specially constructed warehouses had proved that :

(*a*) close and continuous contact with plague-infected animals (including contact with faeces and urine of the infected animals and

the eating of food contaminated with such faeces and urine) did not give rise to epizootics among the healthy animals as long as fleas were excluded;

(*b*) when fleas were present, epizootics were apt to start which varied in severity and rate of progress according to the season of the year and the number of fleas present;

(*c*) the seasons in which such experimental epizootics were readily produced and spread rapidly, coincided with the seasons during which plague was epidemic;

(*d*) epizootics could occur without direct contact between the originally infected and the healthy animals, the infection being effective in proportion to the access the fleas had to the healthy animals.

4. The site of the primary buboes in animals found plague-affected in nature and those infected in the laboratory by means of fleas was identical.

5. Work carried out in houses where plague had occurred showed that :

(*a*) guinea-pigs allowed to run about in such houses contracted the infection in 21% of the houses, regardless of whether or not these had been disinfected with a " strong solution of acid perchloride of mercury "; [143]

(*b*) animals which had been placed in cages to which fleas had access in plague-infected houses often contracted the infection, while those exposed in flea-proof cages remained healthy;

(*c*) more than 30% of the fleas trapped in plague-infected houses contained abundant living and virulent plague-bacilli in their gastro-intestinal tracts;

(*d*) fleas derived from the carcasses of plague rats found in the houses, or recovered from test animals kept in them, were capable of infecting healthy animals in the laboratory;

(*e*) houses which were definitely proved to be plague-infected contained, on an average, nearly three times as many rat fleas as houses which were only presumably plague-infected, and twelve times as many as houses which were free from suspicion.

6. Observations on the pathogenesis of human plague and on the time relationship between epizootics and epidemics were consistent with the assumption that the rat fleas, which were found to bite man readily when hungry, were responsible not only for the transmission of plague from rat to rat, but also for the conveyance of the infection from the rats to man.

These results are all the more striking when it is considered that the members of the Commission, though believing that they worked with

X. cheopis only, probably also experimented to some extent with *X. astia* —a then unidentified, less-efficient plague-vector.

Though both Simond [233] and the Plague Research Commission [202] had obtained some evidence that natural plague occurred in wild as well as in commensal rodents, the findings in the wild rodents were not sufficiently impressive to lead to investigations on the role played by their fleas. However, this gap was soon filled by McCoy,[165, 166] who showed that plague could be transmitted from ground-squirrels (*Citellus beecheyi*) to guinea-pigs, as well as between ground-squirrels, through one of their fleas, now called *Diamanus montanus*.

Infectibility of fleas with plague

The only way in which fleas can take up plague bacilli is by obtaining a blood-meal from an animal or from a patient suffering from plague septicaemia. Fleas which ingest *P. pestis* in this manner do not contract infection in the true meaning of this term, but merely become capable of harbouring this organism in their gastro-intestinal tracts. Nevertheless, it is customary to consider such pestiferous fleas as being " infected " with plague. As discussed later (see page 350), a great multiplication of the ingested plague bacilli may take place in a flea's stomach and/or proventriculus, and may result in a mechanical obstruction or " blockage " which sooner or later may prove fatal to the insect.

Observations by numerous workers have shown that in order to "infect" fleas with plague under laboratory conditions, they must be exposed on experimental animals in which bacteraemia has reached a high degree. This necessity is well illustrated by the following data supplied by Eskey & Haas : [54]

Degree of septicaemia in the hosts (guinea-pigs)	*Number of fleas fed*	*Percentage of fleas plague-infected*
10 or more organisms per field in smear	439	32
2-10 organisms per field in smear	457	29
1 or less organisms per field in smear	546	25
Smears negative, culture strongly positive	469	17
Smears negative, culture slowly positive	344	10

It will be noted that, even under optimal conditions, only about one-third of the fleas used in these experiments became infected. It was found, however, that marked differences existed in this respect between the different flea-species. In a series of fleas previously examined by Eskey,[52] it had been established that the infection in *X. cheopis* was 66.0% as compared with about 25% in the case of *N. fasciatus*, *D. montanus*, and *Hoplopsyllus anomalus*, and 10% in the case of *L. segnis*.

In another paper published at the same time, Eskey [51] maintained, with much reason, that the different infection-rates observed in the various flea-species might depend on differences in feeding habits, those fleas which

fed at long intervals being less likely to obtain a meal during the time their hosts had numerous plague bacilli in their blood. Possibly, therefore, the high incidence of infection in *X. cheopis* was due to its voracious feeding-habits.

Findings similar to those of Eskey were recorded by Eskey & Haas [54] who, feeding 2,031 fleas of different species 3,521 times on plague-infected guinea-pigs, produced infection in 894 fleas. They noted that practically all the 28 rat and wild-rodent flea-species tested could be infected to a greater or lesser extent regardless of differences in sex or size. Rabbit fleas (*Hoplopsyllus glacialis affinis*) and cat fleas (*Ct. felis felis*) were found much less liable to contract infection than the rodent fleas.

It is difficult to decide to what extent fleas may become plague-infected under natural conditions. As summarized by Petrie,[198] the Plague Research Commission established in this connexion that the average stomach content of a flea was about 0.5 mm^3 and that, therefore, a flea imbibing rat-blood containing 10,000 or more plague bacilli per ml would take at least 5 bacilli into its stomach. It was estimated that 66% of the plague rats in Bombay had more than 10,000 bacilli per ml of their blood either before or immediately after their death.

The Commission also established that approximately 50% of fleas collected from the carcasses of these rats harboured *P. pestis*.

Eskey & Haas [54] considered it as entirely problematical whether fleas were more readily plague infected under natural than under experimental conditions. Since, in the laboratory, the fleas were fed to capacity, their chances of ingesting *P. pestis* seemed as great as when they would have fed on their infected natural hosts. It seemed permissible, therefore, to assume that

"... the average plague infection of fleas in nature rarely exceeds one-third of those feeding on their plague-infected normal hosts, as this was the proportion of flea infection from exposure to guinea-pigs having over ten organisms per field in smears of their blood, indicating about as great a bacteremia as would likely occur in any animal".

Disappearance of the bacilli

As summarized by Petrie,[198] the Plague Research Commission [203] found that some of the fleas which had ingested *P. pestis* were able to rid themselves of the bacilli, and that the clearing mechanism was probably a phagocytic one. Experiments showed that a rise of temperature of about 8°C (14°F) between the limits 24°C and 32°C (75°F and 90°F) doubled the rate at which the bacilli disappeared from the gastro-intestinal tracts of the fleas. This seemed explained through the observation of Ledingham [145] that, for a rise of 10°C (18°F), the rate of phagocytosis increased about 2.2 times.

That fleas were able to get free from plague bacilli was also observed by other workers—more recently by Douglas & Wheeler,[48] Eskey & Haas,[54] Burroughs,[25] and Holdenried.[105]

Experimenting with *D. montanus* and *X. cheopis*, Douglas & Wheeler noted that 62% of the *D. montanus* were free from plague bacilli 24 hours after one infective meal, while only 2% of *X. cheopis* became free after an interval of more than 48 hours.

Eskey & Haas [54] tabulated their observations as follows :

Disappearance of plague infection from fleas after existence of infection had been demonstrated by one or two inoculations of faeces

Flea species	*Number originally infected*	*Percentage that became free*
Xenopsylla cheopis	140	4.0
Thrassis pandorae	58	7.0
Nosopsyllus fasciatus	51	8.0
Anomiopsyllus nudatus	9	11.0
Opisocrostis labis	178	12.0
Orchopeas sexdentatus	81	13.0
Thrassis acamantis	8	13.0
Malaraeus telchinum	74	16.0
Megarthroglossus divisus	6	17.0
Thrassis petiolatus	6	17.0
Thrassis arizonensis	58	19.0

Burroughs,[25] who carried out transmission studies with nine flea-species, established that the percentages of fleas which had become free from plague bacilli at the time of their death were as follows :

Species	*Percentage*
Xenopsylla cheopis	28.0
Orchopeas sexdentatus	30.0
Opisodasys nesiotus	46.0
Nosopsyllus fasciatus	58.0
Megabothris abantis	60.0
Pulex irritans	69.0
Diamanus montanus	74.3
Malaraeus telchinum	83.0
Oropsylla idahoensis	93.0

Burroughs noted that the *X. cheopis* which were found free from plague at death had survived much longer than those in which *P. pestis* had become established.

Holdenried [105] worked with *D. montanus* and *X. cheopis* which, after an infective meal, were kept in jars at a temperature of 68°-73°F (20°-23°C) and 60%-90% relative humidity, and were given opportunities to feed on normal mice. In the *D. montanus* group, 25% were found to be free from *P. pestis* as early as three days after an infective meal; the last flea of this species which retained viable plague-bacilli died on the 35th day. In the *cheopis* group, the presence of infected fleas was demonstrated up to 52 days, and only 5 out of a total of 76 fleas became free from the bacilli.

Examining fleas which had been killed at intervals of 22-31 days after infection in the course of transmission experiments, Holdenried found that

80% *Polygenis gwyni* and 68% *X. cheopis* contained plague bacilli, as compared with 13% *D. montanus.*

Holdenried came to the conclusion that the disappearance of *P. pestis* from infected fleas probably did not depend on phagocytosis which ought to have exerted an identical action in the case of all flea species. He also disbelieved that the mechanical flushing-out of the alimentary tract with blood ingested from normal rodents after an infective meal offered an explanation, as had been adduced. He stressed in this connexion that *P. gwyni* was observed to defaecate profusely after the first few feedings, yet, as noted previously, 80% of these fleas continued to harbour plague bacilli until their death. In his opinion, therefore, the mechanism by which fleas became plague-free was still unknown.

As shown by these observations, no doubt can exist that, at least under experimental conditions, fleas which have ingested plague bacilli can become free from them. It is of importance to note in this connexion that the percentage of such a clearance was markedly lower in the case of highly efficient vectors, such as *X. cheopis* and *P. gwyni*, than in the case of less-efficient vectors.

Virulence of the bacilli harboured by fleas

It was suggested by some workers, e.g., Bacot & Martin,[8] Otten,[193, 194] Meyer,[175] Blanc,[16] and Macchiavello,[156, 162] that plague bacilli harboured by fleas may undergo a loss in virulence. As assumed by Otten, such weakly virulent bacilli, instead of producing infection, might be immunizing agents.

It would be unwise to disregard the possibility that plague bacilli might become less virulent in the fleas—the more so, because bacteriophage, the presence of which in *X. cheopis* has been demonstrated by Girard,[73] might exert an influence in this respect. However, it must be emphasized that, in the experience of most observers, prolonged survival in the fleas did not lead to an abatement of the virulence of *P. pestis.* As has been shown by some workers, particularly Blanc,[16] and Blanc & Baltazard,[20] the virulence of these organisms is apt to remain unimpaired for prolonged periods even in dried-up carcasses of plague fleas.

Mechanism of Plague Transmission by Fleas

As previously noted, Simond,[233] when investigating the role of fleas in plague, was led to believe that the entry of infected flea-faeces into bite-wounds, rather than the flea-bites themselves, was responsible for the transmission of the infection. The Plague Research Commission [202] also suspected a role of the flea-faeces, and suggested that a transmission of plague by fleas might be effected as well by :

(*a*) the rodents eating infected fleas ;

it sometimes is to find plague-infected rats or their fleas during an outbreak, little credence can be given to these and similar contentions.

It is significant that the workers who studied the problem of plague in the bed-bugs thoroughly were rather averse to the idea that these parasites played a dangerous role.

Verjbitzki [264] maintained in this connexion that " bugs which have sucked their full complement of blood do not as a rule bite again for a considerable interval ".

He showed, however, that bed-bugs which had been interrupted during the act of feeding were inclined to feed again, and were thus able to convey infection from an infected to a healthy host. He also found that " the crushing of infected bugs *in situ* during the process of biting, occasioned in the majority of cases the infection of the healthy animal with plague ".

Bacot,[7] while in accord with Verjbitzki's findings, pointed out that :

(1) A transmission of plague through the faeces of infected bugs could be ruled out because these insects do not defaecate during the act of feeding and " hurry after their meal to some nook or cranny to digest at leisure ".

(2) " The development of *B.* [*P.*] *pestis* within the crop of bugs differs generally from that which takes place in the stomach of the flea in respect of its slower and looser growth, this limitation of activity being accompanied by and possibly due to the preservation of the structural character of the blood for many days after its ingestion into the crop."

(3) " The absence of any definite valve between the pump and the crop, together with the looser nature of the growth within the bug, preclude the idea of such mechanical blockage as causes regurgitation and mouth infection by fleas."

Cornwall & Menon [38] were also convinced that plague-infected bed-bugs could not regurgitate their stomach-contents during the act of feeding. They were able to find plague bacilli in the proboscides of bed-bugs up to 46 hours after feeding and therefore considered it possible that infection of a healthy host might result from the washing-out of these bacilli from the proboscis of a bed-bug whose feeding on an infected host had been interrupted. Nevertheless, Cornwall & Menon maintained that plague was not likely to result from the bites of infected bed-bugs.

While this contention deserves attention, it cannot be denied that bed-bugs are capable of transmitting plague. However, there is no reason to assume that they play more than an occasional, or, at most, an adjuvant, role in the conveyance of the infection.

Lice of rodents and other animals

As was established by Simond,[233] and confirmed by many subsequent workers, the lice of plague rats are apt to harbour *P. pestis*. Identical observations were made in the case of *Neohaematopinus columbianus*—the louse

of the Californian ground-squirrel—by McCoy,[166] who found that these parasites remained infective for two weeks and were capable of conveying plague to experimental animals.

The presence of *P. pestis* in a louse occurring on tarabagans (*Marmota bobac*) and identified as *Neohaematopinus citelli* (" *Linognathoides* " *citelli*) was demonstrated by Sukneff,[244] who obtained positive results when inoculating guinea-pigs with triturates of such lice collected from either naturally affected or artificially infected Siberian marmots.[245]

A thorough study of the tarabagan louse was later made by Jettmar.[127] He established that *P. pestis* rapidly multiplied in the stomachs of these lice, which invariably died 2-3 days after an infective meal. Plague bacilli were also plentiful in the faeces of the infected lice and persisted in their carcasses for about two weeks. Exposure of 40 live specimens of this louse which had been collected from an artificially infected tarabagan produced acute plague in a sisel (*Citellus undulatus*). The tarabagan louse was found capable of biting man, but was apparently unable to survive long when separated from its specific host.

These findings deserve attention because there can be no doubt that the rodent lice play an adjuvant role in the transmission of plague from animal to animal.

Blanc & Baltazard [20] were able to demonstrate the presence of virulent plague bacilli in *Pedicinus albidus* which had been collected from artificially infected specimens of its specific host—the Barbary ape, *Macaca sylvanus*. They found the louse of the pig (*Haematopinus suis*) not only capable of harbouring *P. pestis*, but also of conveying the infection to guinea-pigs.

Mites

It was maintained by Russo [222, 223] that free-living mites of the family of Tyroglyphidae, which were apt to feed on the carcasses of plague-affected rodents, could harbour and transmit *P. pestis*. He incriminated, in particular, *Glyciphagus domesticus* and *Tyroglyphus siro*.

Yamada (quoted by Mitsuhori [180]) established that *Liponyssus nagoyoi* (family of Dermanyssidae), a parasite of the commensal rats—particularly of *R. rattus*—in Japan, could transmit plague from animal to animal. As Mitsuhori added, this mite, which was possibly identical with *Lyponyssus bacoti*, could attack man.

According to a statement made by Jorge,[135] Bacot & Stanley Hirst succeeded in conveying plague from rat to rat with *Laelaps echidninus*, a mite infesting *R. norvegicus* in many parts of the world.

Ticks

Paying attention to " the ticks infesting rats suffering from plague ", Skinner [236] was able to prove that these parasites could harbour *P. pestis*.

Findings made in the case of other tick species may be summarized as follows.

Marmot-ticks. Skorodumoff [237] was able to prove, with the aid of animal experiments, that ticks (? *Rhipicephalus haemaphysaloides*) collected from naturally plague-affected tarabagans (Siberian marmots) contained virulent *P. pestis.* Identical findings were made by Tikhomirova & Nikanoroff [257] and by Borzenkov & Donskov [21] in the case of *Ixodes autumnalis*, a tick infesting the marmots of south-east Russia.

Suslik-ticks. As summarized by Wu,[281] the suslik (*Citellus*) ticks were incriminated by several plague-workers in south-east Russia, particularly by Tikhomirova & Nikanoroff,[257] who proved the presence of *P. pestis* in *Rhipicephalus schulzei* with the aid of culture tests and animal experiments, and by Golov & Kniazevski,[85] who established that the larvae and nymphs, as well as the adults, of this tick could harbour the infection.

Argas persicus. Faddeeva [58] proved, by cultivation and animal experiments, that *Argas persicus* (a free-living tick), when fed upon guinea-pigs in the septicaemic stage of plague, could remain infected for periods of up to 110 days.

Dermacentor silvarum. Sassuchin [225] demonstrated that virulent plague bacilli could persist for up to 35 days in the nymphs of *Dermacentor silvarum*, which during its larval and nymph stages infested susliks or other wild rodents and, when adult, fed on camels, horses, or man. As was afterwards shown by Sassuchin & Tikhomirova,[226] the larvae and nymphs, but not the adults, of this tick could harbour *P. pestis*, even though over 60% of them died when given an infective meal. The organisms persisted in the surviving larvae for 2-10 days and in the nymphs for 2-6 days, but invariably disappeared during the moulting stages.

Hyalomma volgense. Borzenkov & Donskov [21] proved that the larvae and nymphs as well as the adults of the cattle-tick, *Hyalomma volgense*, were apt to harbour *P. pestis* in their gastro-intestinal tracts after they had been fed on plague-infected guinea-pigs, jerboas, or marmots, the organisms remaining viable in the larvae for seven days, in the nymphs for three days, and in the adults for at least 11 days. Borzenkov & Donskov succeeded in conveying plague to healthy rodents through the bites of these ticks, and also demonstrated the presence of virulent *P. pestis* in the faeces of infected specimens.

Rhipicephalus sanguineus. Blanc & Baltazard,[20] summarizing observations they had made of *Rhipicephalus sanguineus*, a tick principally infesting dogs, stated that " under the conditions of our experiments, it did not transmit plague mechanically when taking its first meal on an infected guinea-pig and continuing to feed after several days on a healthy guinea-pig.

Since, on the other hand, the plague bacilli could not resist the profound changes taking place during the moulting stages of these ticks, one can conclude that, as far as the available evidence goes, *Rhipicephalus sanguineus does not seem to play any role in the natural transmission and preservation of plague* ".[i]

Opinions regarding the role played by the ticks in the transmission of plague were sharply divided. Sassuchin & Tikhomirova,[226] like Blanc & Baltazard, came to the conclusion that the species they worked with were of no importance in this respect. On the other hand, as summarized by Wu,[281] Tikhomirova & Nikanoroff [257] asserted that the suslik-ticks, particularly *Rhipicephalus schulzei*, on account of their great prevalence, eagerness to change hosts, capacity for transmitting and preserving other infections, blood-sucking faculty, and hardiness, might, under equal conditions, prove more efficient vectors than other insects, e.g., fleas and lice, and might, on account of their aptitude for changing hosts, be responsible for an extension of the enzootic foci.

In evaluating these divergent opinions, one is led to believe that different tick-species might play different roles in plague so that no hard-and-fast general rule can be laid down. However, while admitting that species like the suslik-ticks are of some importance, one cannot share the opinion of Tikhomirova & Nikanoroff and some other workers in south-east Russia that the ticks play not an adjuvant, but the principal, role in the transmission of plague. No other arthropod vector surpasses or even equals the rodent fleas in this respect.

Flies

Observations on the presence of *P. pestis* on or in houseflies (*Musca domestica*) were made by some early workers, e.g., Yersin,[285, 286] Albrecht & Ghon,[2] and Nuttall.[189] While Albrecht & Ghon merely established that flies which had come in touch with plague material were apt to become externally contaminated, Yersin and Nuttall found that *P. pestis* was present in the gastro-intestinal tracts and faeces of these insects.

Nuttall came to the conclusion that plague infection was fatal for the flies, death occurring more rapidly the higher the temperature rose, e.g., after 3 days at 23°-31°C. This opinion was also held by Gosio,[86] who further claimed that fly larvae, which had been fed on plague organs, could develop normally while continuing to harbour *P. pestis*, but that the adult flies developing from them succumbed to the infection in 15-24 hours. However, Hunter [113] and Jettmar [128] were unable to confirm that plague-infected houseflies showed any unusual mortality.

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[i] " ... n'a pas dans les conditions de nos expériences, transmis la peste mécaniquement en prenant la première moitié de son repas sur cobaye infecté, et en l'achevant en plusieurs jours sur cobaye neuf. Comme, d'autre part, le virus ne résiste pas à l'ensemble de modifications profondes qu'est la mue chez ces acariens, on peut conclure de ces brèves expériences que *Rhipicephalus sanguineus ne doit jouer aucun rôle dans la vection et le maintien naturel de la peste* ".

Though, in view of the evidence set forth above, there can be no doubt that houseflies may become carriers of *P. pestis*, it is not at all likely that they then become apt to convey plague.

Wayson,[271] working with the stable-fly, *Stomoxys calcitrans*, found that

" when . . . applied to an animal (guinea pig) acutely ill of this . . . disease, or of plague bacteremia, eight bites by one or more flies (not over two have been used simultaneously) will effectively transmit the disease to a healthy animal, if the application is made within an hour after the flies have bitten the affected pig ".

In view of this experimental evidence, one cannot exclude the possibility of a mechanical transmission of plague through the stable-fly. Certainly, however, actual instances of such a conveyance of the infection, if occurring at all, are exceptional.

Gosio [86] reported that, when fed on the organs of animals dead from plague, the larvae of necrophilous flies harboured numerous plague bacilli and that these organisms were passed on to the pupae and adults in diminishing numbers. Similar claims were also made by Russo.[223] However, interesting as these findings are, they are of no practical importance as far as the transmission of plague is concerned.

Mosquitos

The evidence incriminating the mosquitos, and allied species of blood-sucking insects, as potential vectors of plague may be summarized as follows :

(1) Bonnardière & Xanthopoulides (quoted by Dieudonné & Otto [46]) found plague bacilli in the proboscis and stomach of an insect (" Stechmuecke ") which had just bitten a plague patient.

(2) Flu [63, 64] was able to prove that specimens of *Mansonia*, *Culex pipiens*, *Anopheles rossi*, and *Aëdes aegypti*, which had been fed on plague-affected laboratory animals, could harbour virulent plague bacilli for some days, but did not transmit the infection through their bites.

(3) Blanc & Baltazard [20] found that *Aëdes aegypti* which had fed on plague-infected guinea-pigs harboured *P. pestis* for ten days after the last infective meal, but were incapable of transmitting the infection.

Many observations made in China during the second World War proved that mosquitos, even though they may become infected, are unable to transmit plague. Although the patients in the emergency hospitals were often covered with mosquito-bites, not a single case of plague occurred which could be ascribed to such bites. With the exception of one medical officer, who was infected during the day through a flea-bite, no instances of bubonic plague were noted among the staff of the hospitals. These findings are in accord with results obtained by Hunter,[113] who failed to detect plague bacilli in a group of 20-30 mosquitos which had been caught inside the nets covering plague patients in the Hong Kong Infectious Diseases Hospital.

Triatoma

Mertens [171] explored the possibility that *Triatoma rubrofasciata*, an insect normally feeding on the rats of Java, but occasionally attacking man, might convey plague. He found that guinea-pigs inoculated subcutaneously with specimens of this insect which had been fed on plague-affected animals became fatally infected, and that the insects, if kept starving after an infective meal, could harbour *P. pestis* for at least one month. However, the faeces of infected *Triatoma* were free from plague bacilli. Carrying out transmission experiments with this insect, Mertens obtained only one positive result when using a *Triatoma* which bit a healthy guinea-pig immediately after its meal on an infected animal had been interrupted. *Triatoma rubrofasciata* is, therefore, certainly not a dangerous plague-vector.

Ants

Hankin [90] was able to demonstrate *P. pestis* in ants which had fed on the carcasses of plague rats, and to infect laboratory animals (rats and mice) with triturates of these insects.

In contrast to these findings, Russo [223] maintained that the virulence of plague bacilli harboured by ants was apt to become lost.

Beetles

Early experiments by Cao [33] showed that beetles which had been fed on plague material were apt to excrete virulent *P. pestis.*

Skorodumoff,[237] working in Transbaikalia, was able to produce plague in a laboratory animal inoculated with a triturate of *Necrophorus dauricus* found on a naturally infected tarabagan. However, experiments performed with other specimens of this beetle species, and with *Silpha perforata* collected from infected tarabagans, gave negative results.

Russo [223] found that two species of necrophilous beetles—namely, *Sitophilus* and *Tenebrio molitor*—could harbour *P. pestis* when fed on plague material.

Notwithstanding these positive findings, it is altogether unlikely that beetles can play a role in the transmission of plague.

Cockroaches

Early observations on the presence of *P. pestis* in cockroaches were made by : (*a*) Cao [33] and Kuester (quoted by Dieudonné & Otto [46]), who found that these insects, when fed on plague material, could void virulent plague bacilli in their faeces; (*b*) Pound,[206] who ascribed an instance of laboratory infection in a healthy guinea-pig to young cockroaches which had had previous contact with plague-infected animals, and was able to prove this contention by bringing a second guinea-pig into contact with such cockroaches; and (*c*) Hunter,[113] who demonstrated the presence of *P. pestis* in cockroaches (*Blatta orientalis*) collected in plague houses.

However, further observations indicated that the ability of cockroaches to harbour *P. pestis* was not great. Barber,[10] who injected these organisms into the body-cavities of 61 cockroaches (*Periplaneta americana* and *Rhyparobia maderae*), obtained only one positive result when making animal experiments with these insects. Jettmar [128] found that : (*a*) as proved by guinea-pig experiments, plague bacilli perished or soon lost their virulence in the alimentary tracts of cockroaches (*Blatta germanica*); (*b*) it was not possible to infect guinea-pigs with fresh excrement from cockroaches fed on plague material; and (*c*) inoculation of *P. pestis* into the coxa-stumps of an amputated leg did not produce plague in cockroaches.

It is unlikely, therefore, that cockroaches play any role in the transmission of plague. Certainly, however, it is essential to keep premises where experimental animals are kept free from these pests because, as shown by the observations of Pound and others, young cockroaches have an almost uncanny ability to insinuate themselves into containers which seem impregnable to the inroads of insects.

Conclusions

Dealing with the role of insects other than fleas in the transmission of plague, Wu [281] expressed the opinion that " rodent lice and ticks alone have to be taken into serious consideration ", but that " compared, however, to the rodent fleas, especially *X. cheopis*, the role of even these insects is of little import. Indeed, broadly speaking, the terms ' insect vectors of plague ' and ' rodent fleas ' may be taken as interchangeable."

Though, under special conditions, certain insect species other than the rodent fleas may play a comparatively important adjuvant role, in general, one ought to be in agreement with the dictum of Wu.

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ANNEXES

Annex 1

LIST OF RESERVOIRS AND VECTORS OF PLAGUE

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

Particular attention has been paid to make table I not only as accurate as possible, by closely following the standard nomenclature of Ellerman [30] and of Ellerman & Morrison-Scott,[31] but as complete as possible. For its compilation valuable information has been derived from unpublished working documents generously made available by Macchiavello [99] and Davis.[23]

These two documents as well as published material of Wu [204] and of Chabaud [20] were also used in the compilation of table V.

TABLE I. WILD RODENTS AND LAGOMORPHA PROVED NATURALLY INFECTED OR STRONGLY INCRIMINATED THROUGH POSITIVE FINDINGS IN THEIR ECTOPARASITES

RODENTIA		Locality	Bibliographical reference
family and subfamily	species		
BATHYERGIDAE	*Cryptomys* sp. White-toothed mole-rat	Angola	38
CAVIIDAE			
Caviinae [a]	*Cavia aperea* Restless cavy	Brazil Ecuador	96 32
	Cavia pamparum Pampas cavy or Lund's guinea-pig	Argentina	160
	Cavia tschudii atahualpae Peruvian cavy	Peru	99
	Caviella australis australis	Argentina	9
	Caviella australis joannia	Argentina	6
	Galea musteloides leucoblephara	Argentina	6
	Galea musteloides littoralis	Argentina	9
	Galea spixii " Prea "	Brazil	96
	Kerodon rupestris Brazilian rock-cavy	Brazil	96

[a] The common guinea-pig, *Cavia porcellus* (*Cavia cobaya* auctt.), though repeatedly found plague-infected (Wu Lien-teh[205] and others), has not been included in this annex since it is a domestic rather than a wild rodent. Referring to the findings recorded in Peru, Macchiavello [99] stated that " plague infection in Cavia, has been mentioned by several authors but without accurate taxonomic classification of specimens ".

TABLE I (continued)

RODENTIA		Locality	Bibliographical reference
family and subfamily	species		
MURIDAE			
Murinae (continued)	*Pelomys campanae* [h]	Senegal	92
	Pelomys fallax iridescens	East Africa	95
	Rattus natalensis (*Mastomys coucha* auctt.) Multimammate rat	South Africa Kenya	53, 118 54
	Rhabdomys pumilio Four-striped grass-mouse	South Africa	117
Otomyinae	*Otomys* sp.	East Africa	38
	Otomys angoniensis	Kenya	54
	Otomys irroratus South African water-rat	South Africa	183
	Otomys tropicalis elgonis	Belgian Congo	25, 26
	Otomys unisulcatus [j] (*Myotomys unisulcatus* auctt.) Karroo rat	South Africa	183
	Parotomys brantsi luteolus Eastern Karroo rat or Brants' Otomys	South Africa	119
PEDETIDAE	*Pedetes cafer* South African spring-hare	South Africa	119
SCIURIDAE	*Citellus armatus* Uinta ground-squirrel	USA (western States) (Idaho, Montana, Nevada, Washington, Wyoming, Utah)	21, 103
	Citellus beecheyi beecheyi California ground-squirrel	California, USA	102, 201
	Citellus beecheyi douglasi Douglas ground-squirrel	California, USA	108, 131
	Citellus beecheyi fisheri Fisher's ground-squirrel	California, USA	108, 109
	Citellus beecheyi nudipes	Fleas only: California, USA	60
	Citellus beldingi beldingi Belding's ground-squirrel	California, USA	133
	Citellus beldingi oregonus Oregon ground-squirrel	California, USA; Oregon, USA Nevada, USA	107 33

[h] Garnham [33] claimed that this rodent was afterwards identified as being *Lemniscomys griselda*.

[i] Davis [23] noted that "in Barotseland *P. f. frater* [*Pelomys fallax frater*] is associated with *Otomys* and *Dasymys* and has a similar flea fauna. It may act as a transient reservoir with these species".

In the list of plague-affected wild rodents inserted in the 1936 *Manual* [205] separate mention was made of three subspecies of *Otomys* (*Myotomys* auctt.). To make the present list tally with that of Davis,[23] *Otomys unisulcatus* Cuvier and Geoffroy alone has been embodied.

TABLE I (continued)

RODENTIA		Locality	Bibliographical references
family and subfamily	species		
SCIURIDAE (continued)	*Citellus dauricus dauricus* Dauria sisel	Transbaikalia	170
	Citellus columbianus columbianus Columbian ground-squirrel	Washington, USA Montana, USA	111 148
	Citellus columbianus ruficaudus Blue Mountain ground-squirrel	Oregon, USA	111
	Citellus fulvus Large-toothed suslik	South-east Russia	81
	Citellus idahoensis Idaho ground-squirrel	Fleas only : Idaho, USA	198
	Citellus lateralis chrysodeirus Golden-mantled ground-squirrel	California, USA Colorado, USA	109 28
	Citellus lateralis lateralis (*Callospermophilus lateralis* auctt.) Say's ground-squirrel	Fleas only : Wyoming, USA	137 [k]
	Citellus leucurus leucurus (*Ammospermophilus leucurus* auctt.) Antelope ground-squirrel	Fleas only : Arizona, USA California, USA	33, 133
	Citellus mexicanus	Fleas only : New Mexico, USA	195
	Citellus pygmaeus Little suslik	South-east Russia	24, 10
	Citellus richardsoni elegans Wyoming ground-squirrel	Wyoming, USA Also Colorado, Idaho, Montana, and Utah	18
	Citellus richardsoni nevadensis Nevada ground-squirrel	Nevada, USA	111
	Citellus richardsoni richardsoni Richardson's ground-squirrel	Alberta, Canada Montana, USA Saskatchewan, Canada	115 107 61
	Citellus spilosoma major	Fleas only : New Mexico, USA	141
	Citellus townsendi mollis Piute ground-squirrel	Fleas only : Idaho, USA Nevada and Oregon, USA	132 99
	Citellus tridecemlineatus 13-striped ground-squirrel	Fleas only : New Mexico, USA Texas, USA	135 114
	Citellus variegatus grammurus Say's rock-squirrel	Utah, USA also, fleas in Arizona, Colorado, and New Mexico, USA	108
	Citellus variegatus utah Utah rock-squirrel	Utah, USA	110

[k] Plague was also confirmed in fleas from "*Callospermophilus sp.*" in California,[137] and in fleas and ticks from "*Citellus lateralis*" in Colorado.[143]

TABLE 1 (continued)

RODENTIA			
family and subfamily	species	Locality	Bibliographical references
SCIURIDAE (continued)	*Citellus washingtoni loringi* Loring's ground-squirrel	Washington, USA	109, 112
	Citellus washingtoni washingtoni Washington ground-squirrel	Washington, USA	109, 112
	Cynomys sp.	Fleas only : Texas, USA Colorado, USA	114 28
	Cynomys gunnisoni gunnisoni Gunnison prairie-dog	New Mexico, USA	141
	Cynomys gunnisoni zuniensis Zuni prairie-dog	Arizona, USA : New Mexico, USA	109
	Cynomys leucurus White-tailed prairie-dog	Fleas and lice : Wyoming, USA	109, 33
	Cynomys ludovicianus Black-tailed prairie-dog	USA : Colorado, Kansas, Montana, New Mexico, Texas, Wyoming	134, 150
	Cynomys parvidens Utah prairie-dog	Utah, USA	108
	Funambulus sp. (? *F. pennanti*)	South India	40
	Funambulus palmarum Indian palm-squirrel	Ceylon ; India	55, 167
	Glaucomys sabrinus lascivus Sierra Nevada flying-squirrel	California, USA	109
	Marmota bobak Bobak marmot or tarabagan	Manchuria, Mongolia, and Transbaikalia	1
	Marmota caudata Long-tailed marmot	South-east Russia	76
	Marmota flaviventris subsp. Yellow-bellied marmot	Colorado, USA Fleas and lice : Oregon, USA Fleas : New Mexico, USA Fleas : British Columbia, Canada	130 133 93 62
	Marmota flaviventris avara	Fleas only : Oregon, USA	130
	Marmota flaviventris engelhardti Engelhardt marmot	Montana, Utah, Wyoming, USA	108, 109
	Marmota flaviventris nosophora Golden-mantled marmot	Montana, USA	109
	Marmota marmota baibacina (*Arctomys centralis* auctt.)	Russian Turkestan	185, 75
	Sciurus stramineus nebouxi Neboux' squirrel	Ecuador : Peru	99
	Tamias minimus (*Eutamias minimus* auctt.)	Fleas only : Washington, USA	151

TABLE I (concluded)

RODENTIA		Locality	Bibliographical reference
family and subfamily	species		
SCIURIDAE (continued)	*Tamias quadrivittatus frater* (*Eutamias speciosus frater* auctt.) Tahoe chipmunk	California, Nevada, USA	108, 109
	Tamiasciurus douglasi albolimatus Sierra Nevada chickaree	California, USA	108, 109
	Xerus erythropus Central African side-striped squirrel	Senegal	87
	Xerus inauris inauris (*Geosciurus capensis* auctt.) Bristly ground-squirrel	South Africa	192
LAGOMORPHA			
LEPORIDAE	*Lepus californicus* Black-tailed jack-rabbit	California, USA	133
	Lepus capensis [l] Cape hare	South Africa Argentina	183 8
	Lepus europaeus European hare	England	15, 101
	Lepus saxatilis [l] Karroo hare	South Africa	125
	Lepus timidus Mountain or varying hare	Transcaspia	63
	Lepus zuluensis Zulu hare	South Africa	125
	Oryctolagus cuniculus [m] Rabbit	England	101
	Sylvilagus sp. Cotton-tail rabbit	Bolivia Ecuador Peru	99 99 98
	Sylvilagus andinus	Huancabamba, Peru	98
	Sylvilagus audoboni	New Mexico, USA	139
	Sylvilagus bachmani California-brush rabbit	Fleas only (?): California, USA	133
	Sylvilagus brasiliensis	Brazil	96
	Sylvilagus brasiliensis gibsoni	Argentina	178
	Sylvilagus nuttalli nuttalli Washington cotton-tail rabbit	California, USA	109

[l] Davis [23] stated in regard to these two hares that "it is doubtful whether the records of first infections can be relied on to be zoologically exact. *L. capensis* and *L. saxatilis* occur together over most of South Africa and extend northwards throughout the continent".

[m] Instances of secondary plague manifestations among domesticated rabbits, due to the presence of the infection among rats, have been recorded by several workers, e.g. recently by Buck et al.,[14] who also referred to previous observations made in this respect in Madagascar.

TABLE II. WILD RODENTS AND LAGOMORPHA PRESUMED TO BE NATURALLY INFECTED

RODENTIA		Locality	Reference	Reason for non-inclusion in Table 1[a]
family and subfamily	species			
CAVIIDAE				
Caviinae	*Galea* sp.	Bolivia	99	B
DIPODIDAE				
Dipodinae	Jerboa (? *Jaculus* sp.)	South-east Russia	116	B
	Jerboa	Somaliland	100	B[b]
ECHIMYIDAE				
Echimyinae	*Ctenomys* sp. (? *Ct. mendocinus*) "Tucu-tucu"	Argentina	194, 4	A, B
MURIDAE				
Gerbillinae	*Gerbillus* sp.	Tunis	42	A, B
	Tatera valida liodon	Barotseland	23	A
Microtinae	*Arvicola* sp.	Kenya	130	A, B
	Brachytarsomys albicauda	Madagascar	90, 158	A
	Microtus sp.	Angola	106	A, B
Murinae	*Aethomys chrysophilus*	South Africa	23	A
	Bandicota bengalensis varius (*Gunomys varius* auctt.)	Calcutta, India	35	B
	Saccostomus campestris	Nyasaland	86	
	Cape pouched-rat	South Africa	23	A
	Thallomys nigricauda	South Africa	23	A
SCIURIDAE	*Citellus* sp. (*C. mongolicus umbratus* auctt.)	South Manchuria	1	A, B
	Marmota sp.	Tibet	163	
	(? *M. himalayana robusta*)	Kansu	123	A, B
UNCLASSIFIED	Brush (coconut-tree) rat	New Caledonia	129	A, B
	Field-mouse (mulot)	Azores	127, 128	A, B
		Khorassan	46	B
	Field-rat	Rhodesia	79	B
		Tunis	42	A, B
LAGOMORPHA				
LEPORIDAE	*Lepus* sp.	Brazil	96	B
		French West Africa	88	A, B
		Russian Turkestan	76, 77, 181	A, B

[a] A = incomplete evidence of infection
B = species undetermined or doubtful

[b] The presence of plague in other desert rodents was also suspected.

TABLE III. RODENTS AND LAGOMORPHA NOT FOUND NATURALLY INFECTED BUT PROVED SUSCEPTIBLE TO ARTIFICIAL INFECTION

RODENTIA		Locality	Bibliographical reference
family and subfamily	species		
BATHYERGIDAE	*Bathyergus suillus*	South Africa	23
	Cryptomys hottentotus	South Africa	23, 125
DASYPROCTIDAE	*Dasyprocta aguti* Golden aguti (" Cotia ")	Brazil	165
	Dasyprocta prymnolopha Hairy-rumped aguti	Brazil	166
DIPODIDAE Dipodinae	Jerboa (? *Jaculus* sp.)	Egypt	189
	Jaculus orientalis Greater Egyptian-jerboa	Tunis	197
ECHIMYIDAE Echimyinae	*Cercomys inermis* " Punaré "	Brazil	166
	Echimys lamarum Rata de espinho	Brazil	166
MURIDAE Cricetinae	*Cricetulus* sp. (? *Cr. barabensis griseus*) Striped hamster	China	59
	Cricetulus barabensis barabensis (*Cr. furunculus* auctt.)	Transbaikalia	69, 176
	Cricetulus migratorius cinerascens (*Cr. migratorius isabellinus* auctt.)	Iranian Kurdistan	2
	Cricetulus (*Mesocricetus*) *eversmanni* Eversmann's hamster	South-east Russia	44
	Mesocricetus auratus [a] Golden hamster	USA Iranian Kurdistan	105 2
	Sigmodon chonensis	Ecuador	99
Gerbillinae	*Gerbillus campestris* subsp.	Tunis	197
	Gerbillus campestris dodsoni	Tunis	197
	Gerbillus pyramidum hirtipes	Tunis	197
	Meriones crassus charon	Iranian Kurdistan	2
	Meriones shawi grandis	Morocco	12
	Meriones shawi shawi	Tunis	197
	Psammomys obesus roudairei Fat sand-rat	Tunis	197

[a] Baltazard et al.[2] worked with *Mesocricetus auratus brandti* and *Mesocricetus auratus raddei* which they found as little susceptible to plague as the subspecies (? *Mesocricetus auratus auratus*) used by McMahon.[105]

TABLE III (continued)

RODENTIA		Locality	Bibliographical reference
family and subfamily	species		
MURIDAE			
Gerbillinae (continued)	*Tatera afra*	South Africa	23
	Tatera vicina	Kenya	157
Microtinae	*Arvicola amphibius* Water-vole	South-east Russia	44
	Apodemus agrarius Striped field-mouse	Formosa	84
	Apodemus sylvaticus Common field-mouse	Europe	122
	Golunda ellioti Indian bush-rat	India	40
	Lemniscomys barbarus (*Arvicanthis barbarus* auctt.) Barbary striped-mouse or zebra-mouse	Tunis	197
	Micromys minutus Harvest-mouse	South-east Russia	44
	Microtus irani Persian vole	Iranian Kurdistan	2
	Thallomys namaquensis [b]	South Africa	23
Tachyoryctinae	*Tachyoryctes daemon* Orange-toothed mole-rat	Central Africa	95
MUSCARDINIDAE			
Graphiurinae	*Graphiurus* sp. Dormouse (lérot)	French West Africa	91
	Dryomys nitedula Forest dormouse	South-east Russia	44
SCIURIDAE	*Atlantoxerus getulus*	Morocco	12
	Citellus dauricus mongolicus Mongolian suslik	Manchuria	207
	Citellus dauricus ramosus	Manchuria	121
	Citellus undulatus (*Citellus eversmanni* auctt.) Long-tailed Siberian suslik	Transbaikalia	70
	Citellus suslicus guttatus Spotted suslik	South-east Russia	163, 164
	Marmota marmota Alpine marmot	Europe	208
	Sciurus stramineus stramineus	Ecuador	99

[b] *P. pestis* was isolated from *X. brasiliensis* found in a deserted nest of *Thallomys namaquensis*.[23]

TABLE III (concluded)

LAGOMORPHA		Locality	Biblio-graphical reference
family and subfamily	species		
LEPORIDAE	*Sylvilagus ecaudatus* " Tapeti "	Ecuador	99
OCHOTONIDAE	*Ochotona dauricus* Daurian pika	Transbaikalia	69, 176

TABLE IV. MAMMALS OTHER THAN RODENTS AND LAGOMORPHA KNOWN OR SUSPECTED TO BE NATURALLY INFECTED*

Order	Species	Locality	Remarks	Biblio-graphical reference
ARTIODACTYLA	*Camelus* sp. Camel	South-east Russia	See chapter 6	
CARNIVORA	*Canis aureus* Asiatic jackal	India	Suspected by the Plague Research Commission	205
		French West Africa	Suspected	74
	Canis familiaris Domestic dog	Manchuria Morocco	See chapter 6	
	Cynictis penicillata Yellow mongoose	South Africa		125
	Felis catus Domestic cat	Most major plague-areas	See chapter 6	
	Fox	Brazil	Suspected	19
	Herpestes sp. Mongoose	India	Found infected	27
		Hawaii	One specimen found infected	47
	Mustela sp. Weasel	Argentina		94
		USA : western States		198
	Mustela altaica Alpine weasel	Iranian Kurdistan		2
	Mustela putorius European polecat	Near Odessa (south-east Russia)		169
	Mustela putorius eversmanni	South-east Russia		66

* No mention has been made of the kangaroo because the solitary specimen found plague-infected by Thompson [182] had been involved in an outbreak among animals kept in the Sydney Zoological Gardens.

TABLE IV (concluded)

Order	Species	Locality	Remarks	Bibliographical reference
CARNIVORA (continued)	*Mustela putorius furo* Ferret	South Africa	Plague was confirmed in ferrets used to catch rats at Port Elizabeth	205
	Mustela sibirica itatsi Siberian weasel	Japan	One specimen found infected	63a
	Mustela vison Mink	Canada	Incriminated	41
	Suricata suricatta Suricat	South Africa	Suspected	125
	Taxidea taxus neglecta Western badger	Oregon, USA	Found infested with plague-infected ticks	133
INSECTIVORA	*Crocidura olivieri* (*Cr. stampflii* auctt.) Egyptian giant-shrew	Senegal		91
	Erinaceus sp. Hedgehog	Senegal	Found infected	74
	Suncus murinus (*Crocidura caerulea* auctt.) House-shrew [a]	India		205
		Formosa		205
		Cambodia		78
		China		Pollitzer (unpublished reports)
	Sylvisorex gemmeus irene	Belgian Congo		26
MARSUPIALIA	*Monodelphis domesticus* (*Peramys domesticus*) Opossum	Brazil		99
	Didelphis aurita	Brazil	Found infested with infected fleas	99
PERISSODACTYLA	*Equus* sp. Donkey [b]	Manchuria		36
PRIMATES	*Presbytis entellus* Langur	India		50, 51
	Macaca radiata Bonnet monkey [c]	India		50, 51

[a] According to Bangxang,[3] shrews also played a role in the plague outbreaks of Thailand.
[b] Strong & Teague,[173, 174] who found donkeys insusceptible to plague infection by inhalation doubted the validity of Fujinami's findings.
[c] According to Sáenz Vera,[153] monkeys were possibly also involved in Ecuador.

TABLE V. WILD-RODENT FLEAS FOUND NATURALLY INFECTED OR PROVED TO BE SUSCEPTIBLE TO EXPERIMENTAL INFECTION

Species	Locality	Usual hosts	Findings *	Bibliographical reference
Amphipsylla rossica	South-east Russia	*Lagurus* *Microtus*	E	67
Anomiopsyllus sp.	New Mexico, USA	*Neotoma*	S	153
Anomiopsyllus hiemalis	Texas, USA	*Neotoma*	S	114
Anomiopsyllus nudatus	USA, western States	*Neotoma*	E	33
Atyphloceras sp.	USA, western States	*Lagurus* *Peromyscus*	S^p	153
Atyphloceras multidentatus	USA, western States	*Neotoma* *Microtus* *Peromyscus*	E, T	33
Catallagia decipiens	Washington, USA	*Lagurus* *Peromyscus*	S^p	153
Catallagia wymani	USA, western States	*Microtus* *Neotoma* *Reithrodontomys*	E	33
Cediopsylla spillmanni	Huancabamba, Peru	*Sylvilagus*	S	99
Chiastopsylla rossi	South Africa	*Otomys* *Rhabdomys* *Rattus*	E, T, X	65
Citellophilus tesquorum	South-east Russia Transbaikalia	*Citellus*	E, T, X	44
Craneopsylla wolffhuegeli	Argentina	*Graomys* and other rodents	S	8
Ctenophthalmus breviatus	South-east Russia	*Citellus* *Microtus*	E	44
Ctenophthalmus cabirus	East Africa Belgian Congo	*Arvicanthis* and other rodents	E, T S, T	179 26
Ctenophthalmus orientalis	South-east Russia	*Citellus* and other rodents	E	44
Ctenophthalmus phyris	Belgian Congo	*Arvicanthis* *Lemniscomys* *Otomys*	S	26
Ctenophthalmus pollex	South-east Russia	*Citellus* *Arvicola*	S	189a
Ctenophthalmus wagneri	South-east Russia	*Cricetus*	E	184
Delostichus (Parapsyllus) talis	Argentina	*Cavia*	S, T, X	4-7, 178
Diamanus montanus	USA, western States	*Citellus*	S, T, X	33, 34 199, 200

* E = experimentally infected
S = found naturally infected
T = transmitted plague in the laboratory
X = known to bite man
S^p = found together with wild-rodent fleas belonging to other species in pools which proved plague positive.

TABLE V (continued)

Species	Locality	Usual hosts	Findings *	Bibliographical reference
Dinopsyllus ellobius ellobius (*D. lypusus* auctt.)	South Africa	*Rhabdomys*, *Tatera*, and other rodents	S, X	65
Dinopsyllus ellobius lypusus	East Africa Belgian Congo	*Arvicanthis* and other wild rodents	S, T, X	25, 54, 156, 157
Foxella ignota	Colorado, USA	*Thomomys*	S	28
Frontopsylla semura	South-east Russia	*Citellus*	E, X	44
Hectopsylla eskeyi	Peru	*Cavia* and rats	S, T, X	99
Hectopsylla suarezi	Ecuador	*Cavia* *Rattus*	S, X	175
Hoplopsyllus andensis	Huancabamba, Peru	*Sylvilagus*	S	99
Hoplopsyllus anomalus	USA, western States	*Citellus*	S, T, X	34, 103
Hoplopsyllus exoticus	Huancabamba, Peru	*Sylvilagus*	S	99
Hoplopsyllus glacialis affinis (*H. affinis* auctt.)	New Mexico, USA	*Sylvilagus*	S	153
Hystrichopsylla dippiei	USA, western States	*Citellus* and other wild rodents	E, T	33, 198
Listropsylla sp.	South Africa		S	172
Malaraeus telchinum	USA, western States	*Microtus* *Peromyscus*	E, T	16, 33
Megabothris abantis	USA, western States	*Microtus* and other wild rodents	E, T	17
Megabothris clantoni	Washington, USA	*Lagurus* *Peromyscus*	Sp	153
Megarthroglossus divisus (*M. longispinus* auctt.)	USA, western States	*Neotoma* *Tamias*	E	33
Meringis shannoni	Washington, USA	*Lagurus* and other wild rodents	Sp	153
Monopsyllus ciliatus	USA, western States	*Tamias* *Tamiasciurus*	E	33
Monopsyllus eumolpi	USA, western States	*Tamias*	S, T	33, 153
Monopsyllus exilis	Texas, USA	*Onychomys*	S	114
Monopsyllus wagneri	USA, western States	*Lagurus*, *Peromyscus*, and other wild rodents	Sp	154
Neopsylla inopina	USA, western States	*Citellus*	E	33
Neopsylla setosa	South-east Russia	*Citellus* and other wild rodents	S, T, X	44
Neotyphloceras rosenbergi	Ecuador	*Didelphis* and wild rodents	S	99

TABLE V (continued)

Species	Locality	Usual hosts	Findings *	Bibliographical reference
Nosopsyllus sp.	Iranian Kurdistan	*Meriones*	S, X	2
Nosopsyllus consimilis	South-east Russia	Mice	E, T, X	44, 184
Nosopsyllus laeviceps	South-east Russia	*Lagurus*	E, X	44
Nosopsyllus mokrzeckyi	South-east Russia	Mice	S, T, X	44, 188
Nosopsyllus nilgiriensis	South India	*Bandicota*	E (suspected as vector)	39, 64
Odontopsyllus sp.	Huancabamba, Peru	*Sylvilagus*	S	99
Opisocrostis bruneri	USA, western States	*Citellus*	E, T	126
Opisocrostis hirsutus	USA, western States	*Cynomys*	S, T	33, 153
Opisocrostis labis	USA, western States	*Citellus*	E, T	33
Opisocrostis tuberculatus	USA, western States	*Citellus* *Cynomys*	E, T	33
Opisodasys nesiotus	California, USA	*Peromyscus*	E, T	17
Orchopeas sexdentatus sexdentatus	USA, western States	*Neotoma*	S, T	33, 153
Oropsylla idahoensis	USA, western States	*Citellus*	E	33
Oropsylla rupestris	USA, western States	*Citellus*	E, T	33
Oropsylla silantiewi	Manchuria Mongolia Transbaikalia	*Marmota*	S, T, X	177, 206
Pleochaetis sp.	Huancabamba, Peru	*Akodon* *Oryzomys*	S, T	99
Plocopsylla hector	Ecuador	*Thomasomys* and other wild rodents	S	99
Polygenis sp.	Ecuador	*Akodon*	S	99
	Raquia, Peru		S	99
	Huancabamba, Peru	*Akodon* *Oryzomys*	S	203
	Venezuela	*Heteromys* *Sigmodon*, rats	? S	68
Polygenis gwyni	USA, southern and southwestern States	*Sigmodon* *R. norvegicus*	E, T	57
Polygenis litargus	Ecuador-Peru border region	*Sciurus* *Oryzomys*	S, T	98, 99
	Huancabamba, Peru	*Akodon* *Oryzomys*	Suspected as vector	98
Polygenis platensis cisandinus	Argentina	*Cavia* and other wild rodents	Suspected as vector	98
Thrassis acamantis acamantis	USA, western States	*Marmota*	E, T	33
	Canada	*Marmota*	? S	62

TABLE V (concluded)

Species	Locality	Usual hosts	Findings *	Bibliographical reference
Thrassis acamantis howelli	USA, western States	*Marmota*	E, T	33
Thrassis arizonensis	USA, western States	*Citellus*	E, T	33
Thrassis bacchi bacchi (= *Thr. gladiolis*)	USA, western States	*Citellus*	S, T	126, 153
Thrassis bacchi johnsoni	USA, western States	*Lagurus* *Peromyscus*	S?	153
Thrassis fotus	USA, western States	*Onychomys*	S, T	153, 198
Thrassis francisi	USA, western States	*Citellus*	E, T	33
Thrassis pandorae	USA, western States	*Citellus*	E, T	33
Thrassis petiolatus	USA, western States	*Citellus*	E	33
Tiamastus cavicola (*Rhopalopsyllus cavicola* auctt.)	Ecuador Peru	*Cavia*	S, T	99
Tritopsylla intermedia copha	Ecuador		S	99
Xenopsylla sp. (*conformis* group)	Iranian Kurdistan	*Meriones*	S, X	2
Xenopsylla sp. (*X. mycerini* auctt.)	South-east Russia	*Meriones*	E, X	44
Xenopsylla eridos (*X. pasiphae*)	South Africa	*Otomys* and other wild rodents	Probably spontaneously infected	23
Xenopsylla hirsuta	South Africa	*Tatera* and other wild rodents	E, T	171
Xenopsylla philoxera (*X. eridos* auctt.)	South Africa	*Tatera* and other wild rodents	S, T	35
Xenopsylla phyllomae	South Africa	*Desmodillus* and other gerbils	S, X	172
Xenopsylla piriei	South Africa	*Desmodillus*	S	172
Xiphiopsylla lippa	Belgian Congo	*Lophuromys* and other wild rodents	S	26

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