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PLAGUE STUDIES *

1. A Summary of the History and a Survey of the Present Distribution of the Disease

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HISTORICAL SUMMARY

Dealing with the historical aspects of the plague problem in 1936, Wu Lien-teh [148] 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 [102] 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,[129] 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 ".

According to some recent publications it would seem that this interpretation of the biblical text has become untenable. Dealing with this subject in 1942, Neustätter [97] 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, referring to the 1720 epidemic in Marseilles.

While not definitely committing himself as to the real nature of the biblical outbreak, Neustätter concluded " that the version hemorrhoids,

* This is the first of a series of studies which, when complete, will form a manual on plague and which will be published in separate editions in English and in French in the Monograph Series of the World Health Organization. — ED.

untenable as it is, comes nearer to . . . the truth than plague-boils ". Shrewsbury [124] and Girard [35] felt certain that the emerods were really haemorrhoids because they considered that the disease decimating the Philistines was bacillary dysentery.

It would thus seem that the only record testifying to the existence of plague in the West during the pre-Christian area is contained in a fragment from the writings of Rufus, physician at Ephesus about 100 A.D., 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 [148]).

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 (542 A.D.), 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 [146]).

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 [31]). 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 [51] 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. This is a fact of particular interest if it is realized that at least in Europe and north-western Africa rats were still absent at the time, the generally accepted opinion being that the ships of the returning crusaders were responsible for the importation of these pests (*Rattus rattus*) in the 12th century.

Domestic mice were already abundant in the West at the time of the pandemic in Justinian's reign, but, as recently stressed by Shrewsbury [124]

and Girard,[35] 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 [136] who came to the conclusion that

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

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 [51] 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 outbreak 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 [148]), became known under the name of the ' Black Death '. However, rats being without doubt implicated in the perpetuation of the infection, the bubonic form too was frequent, or even preponderant, as for instance in rural England (Greenwood [39]).

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 [146] 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.

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 [128] 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 off by the Black Death. Hecker's [48] estimate that

25 millions or one-fourth of the population fell victims 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,[148] 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,[61] 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,[148] 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 [31]), took place at Marseilles 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,[146] 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-west 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,[148] 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 : [126]

" 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 melancholy 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 542 A.D. 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 the present studies, 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 [130] in 1945 and by Kaul [65] in 1949, and for most African plague foci also of the results of an inquiry instituted by the WHO Expert Committee on Plague (1950). Valuable information on some of the plague foci in East Asia was found in a series of reports by Wilcocks,[144] while in the case of the Americas the studies of Moll & O'Leary[94] 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, 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 [138] 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 [148]).

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 [68]).

(*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.

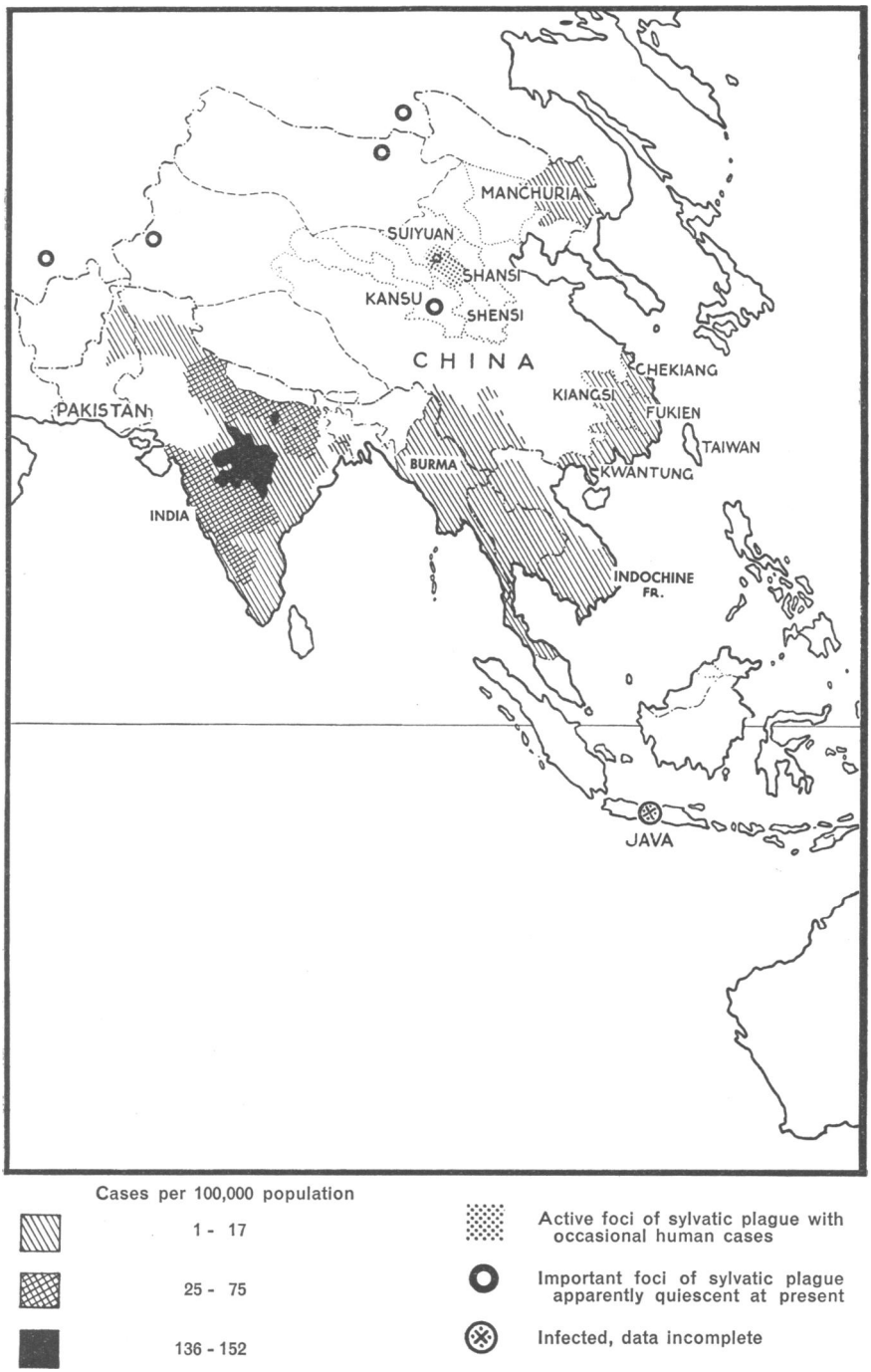
In 1917-8 pneumonic plague, apparently derived from a wild-rodent focus in the Ordos country of Inner Mongolia (now Suiyuan), invaded Chahar and Shan-si and even spread to a slight extent into Chihli, Shan-tung, An-whei, and Kiang-su Provinces, the number of victims amounting to about 16,000. As described by Wu Lien-teh,[148] 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 [24]). 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 [148] 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 [54]), 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

FIG. 1. DISTRIBUTION OF PLAGUE IN ASIA, 1947



importation from south Manchuria. Though detailed information is lacking, it is likely that plague continues to occur in Jehol Province.

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.[134]

(*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 [137]), 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,[66] Fan [24]), 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,[145] 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 that permanent endemic foci, comparable in standing to those in Central Asia, exist in the latter country. On the contrary, it seems more likely that there, as well as in west Yun-nan, prolonged periods during which rat epizootics continued, and epidemics were consequently frequent, alternated with quiescent periods. In conformity with this concept, Kaul [65] considered an importation of plague into Rangoon in 1905 as responsible for the present wave of infection which led to the establishment of endemic foci in Meiktila (especially the town of Mahlaing), Pyawbwe town, and the northern Shan States near the Yun-nan border (Wilcocks [144]). Though country districts as well as towns were involved, according to Wilcocks 70% of the plague deaths were recorded in Rangoon and in the towns on the main lines of communication by river or rail.

The incidence of the disease, which had increased to 3,517 cases with 2,743 deaths in 1946, seems to have decreased since as shown by the following figures :

Year	*Cases*	*Deaths*
1947	1,518	1,192
1948	1,616	1,174
1949	778	615
1950	621	430

The usual plague season in Burma falls into the period November to April, but in Lower Burma there may be a secondary rise in July (Wilcocks [144]).

The bubonic type is preponderant but occasional pneumonic epidemics have been recorded, recently by Wynne-Griffith [149] at Rangoon.

Wilcocks [144] considered *R. concolor* and *R. rattus* of main importance in the perpetuation of plague in Burma ; the former was more common. *R. norvegicus*, dwelling in fields and sewers, seemed less dangerous. Harrison & Woodville [45] found during a recent survey in Rangoon *R. exulans concolor* most frequent (45%), followed by *Bandicota bengalensis* (31%) ; *R. rattus* formed only 8% of the total, *R. norvegicus* 6%. However, plague infection was found only in two *R. rattus* and one bandicoot.

In some parts of Burma *Xenopsylla astia* was more commonly found than *X. cheopis*, but the latter was responsible for most instances of human infection (Wilcocks [144]).

(3) *Indochina*

Médecin-Général Robert, in a valuable report on the plague situation in Indochina presented to the second session of the Joint OIHP/WHO Study-Group on Plague, stated that plague was endemic in the following localities :

(*a*) Cochin-China (Viet-nam Sud), principally in Saigon/Cho-lon, the hotbed of the infection being located in the precincts round the central market of Saigon which had been invaded from Canton and Hong Kong in 1906 ;

(*b*) Cambodia, especially Pnom-Penh, where plague, probably imported from Saigon, had become established in 1907 ;

(*c*) Phan-thiet/Phan Rang region on the south Annam Coast, infected by ship from Saigon in 1908 ;

(*d*) Lang Bian plateau of south Indochina where the disease, imported with rice cargoes from Cho-lon, possibly also from Phan Rang, became established in 1943 or 1944.

The recent incidence of plague in the country was :

Year	*Cases*	*Deaths*
1946	52	24
1947	90	40
1948	355	105
1949	113	55

While the bubonic type was most frequently met with, occasional epidemics with pneumonic features were observed (Wilcocks,[144] Robert, communication to WHO).

R. rattus played in general the most dangerous role with *X. cheopis* as the usual vector. Most interestingly, however, Herivaux & Toumanoff,[50] investigating a limited outbreak at Saigon in 1943 (42 cases), proved the existence of an epizootic among domestic mice. These animals, while not harbouring any *Leptopsylla segnis*, were more heavily *cheopis*-infested than the rats. Only one specimen of the latter (*R. norvegicus*) was found infected.

(4) *Thailand*

Plague, probably imported by the sea-route from China (Bangxang [2]), appeared at Bangkok in 1904 and it would seem that the inland town of Korat became infected in the same year.

Though persisting in both places and eventually also involving other localities, the infection seems to have caused comparatively little havoc in Thailand. According to Wilcocks [144] a total of 1,722 cases occurred at Bangkok from 1905 to 1922. At Korat a severe outbreak (586 cases with 580 deaths) took place in 1917. Preventive measures, including house improvement, were then instituted and only 248 cases with 199 deaths were recorded from 1918 to 1934. Plague began to decline in the country in general from 1929 onwards and the disease seems to have been absent during the period from 1935 to 1937 (Bangxang [2]). However, the infection reappeared towards the close of 1938 in the north-western province of Tak and, as described by Park,[100] spread first eastwards and then to the south. Bangkok became re-infected early in 1940, but only a few cases were reported there and the port seems to have remained free from plague since that year.

Korat became once more infected in 1942, since when cases or small groups of cases have been noted every year. The recent incidence of plague in Thailand may be gathered from the following :

	Thailand		*Korat*	
Year	*Cases*	*Deaths*	*Cases*	*Deaths*
1944	57	29	13	7
1945	107	46	19	2
1946	72	33	4	2
1947	71	23	4	1
1948	122	36	8	2
1949	176	64	1	1
1950	57	10	16 (January-November)	2

Dealing with the seasonal occurrence of the disease, Bangxang [2] stated that the case incidence began to increase in September and reached its highest point in February and March. The disease was at a low level from May to September, particularly during the wet months of June and July. Human plague was almost invariably of the bubonic type, but Bangxang [2] referred to one outbreak with pneumonic features described by Braddock in 1912.

As maintained by Park,[100] *R. rattus alexandrinus* was principally involved ; it formed over 60% of the Bangkok rats as against 14% *R. norvegicus*. Infected shrews were found and might, as Bangxang [2] believed, play a role in the propagation of the infection. As everywhere in the Far East, *X. cheopis* was the principal vector.

(5) *Java*

Plague appeared in Java in 1911 but it is likely that the introduction of the infection, ascribed to the importation of rice cargoes into the port of Surabaya, went back to the previous year (November 1910). The interior of the eastern part of the island soon became invaded. While it became possible to terminate the outbreaks there through wholesale rat-proofing of the houses, the infection began to move westwards, successively affecting middle and western Java. During the period 1920-7 from 8,000 to 10,000 deaths occurred annually. A decrease of the infection-rate was noted from 1928 to 1931 but then the incidence rose again, reaching a maximum of 23,267 cases with 23,239 deaths in 1934. Then, as shown below, a gradual decline set in, which was ascribed to large-scale inoculation campaigns with Otten's live vaccine :

Year	*Cases*	*Year*	*Cases*
1935	13,022	1939	1,558
1936	6,227	1940	312
1937	3,834	1941	550
1938	2,107	1942	339

Little information is available for the period during the second World War ; van Loghem [73] maintained that generally speaking the situation 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.

As pointed out by Park,[101] 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,[147] Wilcocks [144]).

As summarized by Wilcocks,[144] 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 [123] 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,[123] the first of them (Bombay and Mysore States) had been reached by plague in 1898. Nilgiri became involved in 1903 (George & Timothy [30]). 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 [110]).

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,[34] 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,[58] Kaul [65]

TABLE II. ANNUAL MORTALITY FROM PLAGUE IN INDIA DURING THE PERIOD 1939-50

Year	Total plague mortality	Bihar	Bombay State	Central Provinces	Madras	Punjab	United Provinces	Other areas	Hyderabad State [a]	Mysore State [a]
1939	26,257	1,938	1,472	852	324	—	21,662	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	3,886
1944	21,525	834	2,514	910	1,738	61	15,454	14	5,263	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	2,894
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	98	3,904	42	3[b]	2,073	204	719	255
Totals	232,438	22,891	35,253	15,475	18,690	2,640	137,102	387	40,470	38,158

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

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 1939-50, 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 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 [65] 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.

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, where plague had been rampant up to 1923 and perennial manifestations had continued to exist until 1934, is now practically 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 [109]), and where the infection persisted until 1925, considerable outbreaks, due possibly to an importation from Bihar (Greval [42]), took place in 1948 and 1949. However, only two autochthonous cases were recorded in 1950.

Dealing with the seasonal incidence of the disease in his classical study " Twenty years of plague in India ", White [143] 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 [121] and Seal & Prasad [122] 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 in a later section of these studies, other rodents like bandicoots and mole rats (*Gunomys* sp.), 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 [132] and Payne [103] 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 (in a report presented to the second session of the Joint OIHP/WHO Study-Group on Plague) 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 was able to prove the existence of plague in three species of wild rodents (*Meriones persicus*, *Tatera indica*, and a jerboa) as well as in some weasels which were heavily infested with *X. cheopis*. This flea predominated on the rodents as well, but *Nosopsyllus fasciatus* was also found on them. A species of mice (*Mus musculus bactrianus*) was met with but commensal rats appeared to be altogether absent. It is legitimate, therefore, to include Kurdistan among the plague areas where wild rodents form the reservoir of the infection. The heavy infestation of these animals with rat fleas is a feature of particular interest.

In regard to Iraq it was stated by Kaul [65] that plague was absent during the period 1937-44 though in 1938 plague-infected rats were still found in Baghdad. The disease reappeared in February 1945, when 46 cases were reported, mainly from Amara and vicinity (Stowman [130]).

In Turkey a plague epidemic in three villages on the Syrian border took place in March-April 1947. According to Erzin & Payzin [20] 19 persons were affected of whom 14 had axillary buboes and 5 septicaemic features ; 13 of these patients succumbed. A report on this outbreak in the *Journal of the American Medical Association* [63] added that the infection, apparently present among the rats as well as among man, had been derived from Syria where plague used to occur in sporadic form among the desert nomads. Actually after the termination of the outbreak on Turkish territory 6 cases of plague were notified in the Syrian village of Varta.

(8) *Palestine*

While plague cases or even limited epidemics had been met with in Palestine after the British occupation at the end of the first World War, the disease appears to have been absent during the period 1925-40 (Stowman [130]). However, the infection, presumably imported from the Suez Canal zone, reappeared at Haifa in 1941. A considerable epizootic resulted which reduced the incidence of *R. rattus* from 63% to 2.7%, but only 10 human cases were notified.

Jaffa became involved in the winter of 1942-3 when a rat-caused outbreak led to 15 cases with 9 deaths. Only 1.5% *R. rattus* were found at the time and the incidence of *X. cheopis* (38%) seemed less than that at Haifa.

Plague persisted in both places and occasional instances of the disease were observed also at Tel Aviv. The total case incidence was 93 in 1944 and 38 in 1945 when 19 cases with 9 deaths were recorded at Jaffa from October until December. In 1946 there were only 13 cases, but the disease became epidemic at Haifa in 1947 when, including 3 cases in a secondarily involved village, 17 attacks were notified in June and July (Pollock,[106] Haddad & Valero [43]).

Europe

(1) *Ajaccio (Corsica) and Taranto (Italy)*

The essential features of the ephemeral outbreaks occurring soon after the second World War at Ajaccio and at Taranto are given in table III.

TABLE III. DETAILS OF OUTBREAKS OF PLAGUE AT AJACCIO (CORSICA) AND TARANTO (ITALY) IN 1945 [a]

Locality	Dates of occurrence	Origin	Number of cases	Number of deaths	Rats and fleas involved
Ajaccio	May-July 1945	Apparently imported from North Africa	13	10	*R. norvegicus* (90%) *R. rattus* (10%) *X. cheopis*
Taranto	September-November 1945	Possibly importation of rags by ship	29	15	*R. norvegicus* *R. rattus* *X. cheopis* *Nosopsyllus fasciatus*

[a] Bernard et al.,[11] Martorana,[83] Schulz[120]

(2) *Malta*

After an absence of over a hundred years plague appeared in Malta in 1917 when a batch of 8 cases with 4 deaths occurred among dockyard workers and their contacts. The first victim was said to have been " infected from a sick rat which he found in a box containing stores coming from Mesopotamia where the disease was epidemic " (Bernard [10]).

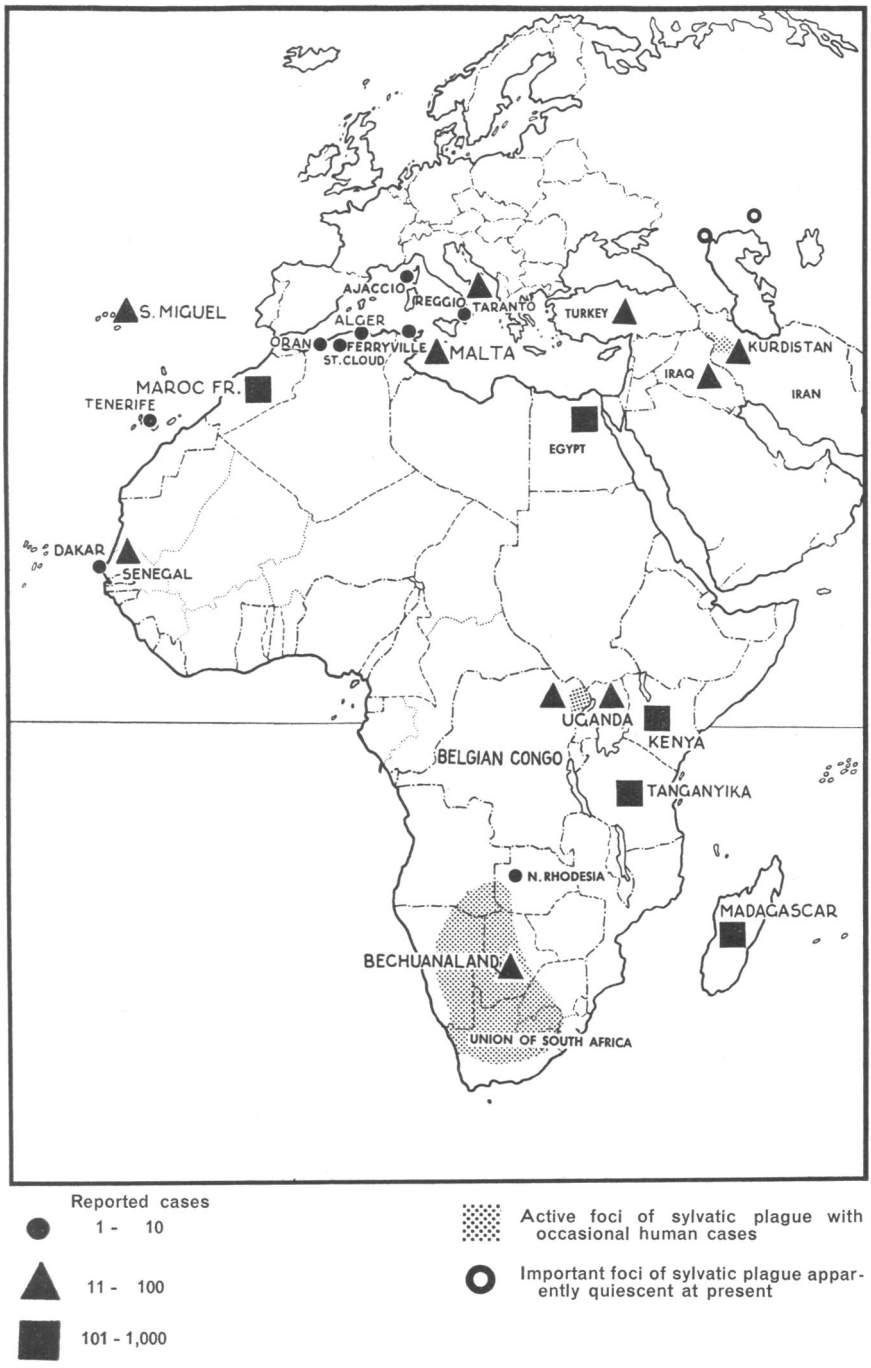
A further outbreak, ascribed to the importation of straw and hay from North Africa, took place from April to November 1936, when 25 bubonic cases with 10 deaths were recorded. An epizootic, involving mainly *R. norvegicus*, was found to be responsible for this epidemic. *Leptopsylla segnis* was the most frequent rat flea (48.75%), followed by *X. cheopis* (37.5%) ; *Nosopsyllus fasciatus* was rarer (13.75%) (Bernard [10]).

Plague again appeared in Malta in June 1945 when an epizootic which involved mainly the area of the commercial port began to manifest itself and led to an epidemic lasting until the end of the year. The 75 cases with 20 deaths recorded during this period were followed by 5 further cases with 2 deaths up to June 1946. Infected rodents (mainly *R. norvegicus*) were found up to February 1947 (Cauchi,[13] Barnett [3]).

(3) *Portugal : Azores Islands*

Plague outbreaks, often showing a high incidence of cases with pneumonic features, were recorded since 1908 in several of the Azores Islands

FIG. 2. DISTRIBUTION OF PLAGUE, 1945-8. I — EUROPE, AFRICA, AND THE MIDDLE EAST



(Wu Lien-teh [147]) but within recent years only São Miguel Island seems to have been involved. An extensive epidemic (744 cases) had occurred there in 1922-3 (Stowman [130]), but since then sporadic cases only, averaging 8 per year during the period 1942-8, have been recorded. Two cases were notified in 1949, none from January to June 1950.

Recent plague outbreaks in the Azores, suspected to be due to epizootics among field-mice (mulots), were rural in character and appeared chiefly during the seasons when crops were harvested and grain was brought to the granaries.[142]

R. norvegicus predominated amongst the commensal rodents, but black rats (mainly *R. rattus*) and *M. musculus* were also found. *Leptopsylla segnis* was the most numerous flea of these rodents, followed by *Nosopsyllus fasciatus*, *X. cheopis* being only the third most frequent (Jorge [61]).

North and North-west Africa

(1) *Egypt*

Fifty-five years after Egypt had become free from plague, the infection, possibly imported from Bombay, reappeared in 1899 at Alexandria. Port Said, the second big port of the country, became affected in 1900, Suez in 1904.

As soon as the infection was established in the two principal ports, it began to travel inland. Invading first the chief places of the provinces and districts but soon spreading to other towns and villages, plague gradually reached most localities of the Nile valley from the coast to Aswân (Wakil,[141] Makar [81]). As shown by statistics compiled by Wakil for the period 1899-1930 (see table IV), the case incidence in Upper Egypt was considerably higher than that in the ports and Lower Egypt.

TABLE IV. INCIDENCE OF PLAGUE IN EGYPT, 1899-1930

Area	Cases		Deaths		Mortality percentage
	Number	%	Number	%	
Ports and Western Frontier Province	3,797	19.6	2,146	20.9	56.5
Lower Egypt	3,201	16.5	1,245	12.1	38.9
Upper Egypt	12,388	63.9	6,881	67.0	55.5
Totals	19,386	100.0	10,272	100.0	52.9

While plague continued to be comparatively frequent in Upper Egypt where the province of Asyût had become the chief focus, the situation in

the country in general became increasingly favourable from 1935 onwards as shown by the following figures supplied by Makar : [81]

Year	*Total number of cases in Egypt*	*Cases in Asyût*	
		Number	*Percentage of total cases*
1931-5	941	389	41.3
1936-7	183	157	85.5

In 1938 only 11 cases were recorded in the country as a whole. However, while the ports remained free and Lower Egypt had an almost clear bill in 1939 and 1940, there were considerable outbreaks, involving 169 and 452 cases respectively, in Asyût.

As summarized by Stowman,[130] from 1941 onwards there was little evidence of plague even in Asyût. Unfortunately, however, as in the first, so also during the second World War a serious situation developed in the Suez Canal zone. Outbreaks commenced successively at Suez in November 1943, at Ismaîlîa and in its district in March 1944, and in May of the same year at Port Said where sporadic cases had been noted since 1940. The total incidence of plague from November 1943 to September 1944 was 712 cases (Stowman [130]). The disease reappeared in the Canal ports in February 1945, but the situation gradually improved and by August 1946 the infection had disappeared (Tomich [135]).

Alexandria, which had had a clear record since 1935, reported 15 cases with 5 deaths in January 1947. Since then, however, plague seems to have been absent from Egypt.

The plague seasons in Egypt were, according to Wakil,[141] as follows :

Zone	*Onset*	*Peak*	*End*
Upper Egypt	March	April	May
Middle Egypt	April	May	June
Nile Delta and Suez	April	June	July
Mediterranean ports	May	July	October

Thus, as in other plague-affected countries, particularly in China, the seasonal incidence of the disease in Egypt stood in correlation with the latitude in which the various foci were situated, being earliest in the south and latest in the north.

The frequency with which the different types of human plague were met with during the period 1904-28 was illustrated by Wakil [141] (see table V).

As will be noted, the pneumonic type, while rare in the coastal regions and Lower Egypt, was comparatively far more frequent in the south of the country.

Dealing with the rodents in his classical study on plague in Egypt, Wakil [141] enumerated " as the species of plague rats most commonly found associated with man " *R. norvegicus*, *R. rattus*, and *Acomys cahirinus*.

The Norway rats were very prevalent in the residential quarters of the port cities, *R. rattus* being restricted to the dock areas. *Acomys cahirinus* was rare in Alexandria and Port Said, more frequent in Suez.

TABLE V. FREQUENCY OF DIFFERENT TYPES OF HUMAN PLAGUE IN EGYPT, 1904-28

Zone	Total plague cases	Bubonic and septicaemic cases	Pneumonic cases	
			Number	%
Ports and Western Frontier Province	3,011	2,925	86	2.8
Lower Egypt	2,660	2,595	65	2.5
Upper Egypt	11,988	10,389	1,599	13.3
Totals	17,659	15,909	1,750	9.9

In Lower Egypt, *R. norvegicus* was most frequent. This species was scarce in Upper Egypt where, according to Petrie & Todd,[104] *R. rattus* formed 60.6% of the total rat population and *Acomys cahirinus* 38.3%.

X. cheopis was the most prevalent species of rat fleas throughout Egypt. *Leptopsylla segnis*, which came next in order, was more often encountered in the ports than in Upper Egypt. According to data supplied by Wakil,[141] *R. rattus* was more heavily flea-infested than the Norway rats. In his opinion this factor in conjunction with the frequency of the former species of rat was partly responsible for the higher plague incidence in Upper Egypt.

(2) *Tunisia*

Plague, which had been absent from the coastal areas of North Africa west of Egypt since 1822, reappeared in the port of Tunis in 1907, imported, as Jorge [62] believed, by ship via Marseilles. Apart from a pneumonic outbreak which claimed 65 victims in 1929-30, sporadic cases only were observed in Tunis. However, the disease, appearing usually in the bubonic but occasionally in the pneumonic form, was epidemic in the south of Tunisia in 1920-1 and fairly frequent during the period 1926-31 when a total of 1,095 cases was recorded, mainly in the districts of Kairwan and Sfax (Stowman [130]). Only 37 cases were notified from 1932 until 1943, the years 1932-4 and 1942-3 being clear. However, from August 1944 until March 1945 an outbreak of bubonic plague (37 cases with 10 deaths) took place at Ferryville (Magrou [80]) and some cases were also noted at Tunis and Bizerta. Tunisia seems to have been free from plague since 1946.

It was claimed that both the 1920-1 and the 1926 epidemics had been due to the immigration of infected wild rodents from Tripolitania. However, no definite proof exists that such animals played a role in the Tunisian outbreaks. Gobert [36, 37] claimed to have found suspicious signs in field-rats and gerbils but not much reliance can be placed upon his evidence (Wu Lien-teh [148]). Ristorcelli,[112] working in a region where plague was said

to have been present in the past, could find no trace of recent epizootics. It is noteworthy, however, that some of the wild-rodent species in Tunisia, particularly the *Psammomys* which abound in the south, are highly susceptible to experimental infection.

Norway rats were by far the most frequent in Tunis, but black rats (*R. rattus alexandrinus* and *R. rattus*) and two species of mice were met with as well. The rodents found infected during the period from 1935 to 1940 and again in 1944 belonged to the following species :

Species	*Number infected*
R. norvegicus	75
R. r. alexandrinus	29
R. r. rattus	13
M. gentilis	9
M. azoricus	3
Total	129

X. cheopis was the most preponderant species of rat fleas in Tunis but *Leptopsylla segnis* and *Nosopsyllus fasciatus* were also present.

(3) *Algeria*

The importation of plague into the Algerian port of Philippeville, occurring in 1899 already (Jorge [62]), led at first to no serious consequences, the total incidence of the disease in the North African ports up to 1907 amounting to 82 cases.

Though plague manifestations became fairly frequent in Algeria from 1911 onwards, Grenouilleau [40] felt certain that they were due to repeated importation of the infection by maritime or caravan routes and not to endemicity. Up to 1935 major epidemics were observed twice only—in 1921, when 185 cases with 97 deaths were recorded at Aumale, and in 1931, when 86 cases of pneumonic plague were notified in the department of Constantine. The incidence of the disease from 1935 onwards may thus be summarized :

Year	*Total number of cases*	*Remarks*
1935	11	10 of the cases were noted at Philippeville
1936	10	3 of the cases were notified at Algiers
1937	3	All at Algiers
1939	2	,,
1940	18	,,
1944	95	94 of the cases occurred at Algiers
1945	11	Small pneumonic outbreak (8 cases) at Oran (Roux & Mercier,[116] Gordon & Knies [38])
1946	2	Both at Oran
1935-46	152	Only 2 of the cases were notified from the hinterland

Throughout these years most of the plague cases were observed during the period from August to November.

R. norvegicus was the preponderant species at Algiers, Bône, and Oran, but as usually *R. rattus rattus* and *rattus alexandrinus* were considerably more frequent in the port area of Algiers than in the city proper (Meunier [85]). To judge from not numerous observations, the two subspecies of *R. rattus* were more liable to contract plague than the Norway rats.

As reported by Grenouilleau & Carle,[41] during the period 1937-44 the rats of Algiers yielded 73.6% of *X. cheopis* as against 19.1% *Leptopsylla segnis*, and 6.4% *Nosopsyllus fasciatus*.

(4) *Morocco*

Plague in Morocco became first manifest within recent times in 1909-10 when, according to Jorge,[62] a total of 25 cases was observed in military stations of the Casablanca district. A violent epidemic, claiming 8,000-10,000 victims, took place in 1911 in the district of the Doukkalla and adjacent areas of the hinterland. During the period 1912-9 outbreaks of varying severity continued to appear in these parts and the infection spread to the ports of Casablanca and Rabat where, however, not much havoc was caused.

Further epidemics occurred from 1922-4 and again during the period 1929-35 but were not on a considerable scale. However, the plague situation became quite serious in 1940 from when onwards the case incidence was as follows :

Year	*Cases*
1940	1,099
1941	2,337
1942	583
1943	393
1944	227
1945	828

In 1940 the disease appeared first in April among tribes in the southern area of Agadir but spread in September to the Marrakech region. In 1942 Casablanca further north became affected, probably through grain transports from the southern foci. The case incidence decreased in 1942 to become fairly low in 1943 and 1944 but the infection spread during this period to Rabat, Port Lyautey, and once more to Marrakech. In the port of Casablanca, where plague had continued to exist since 1941, a minor outbreak took place in 1944, and a severe epidemic in 1945. The whole of Morocco was free in the following year (Stowman,[130] Kaul [65]).

Though plague in Morocco was prevalently bubonic in character, pneumonic cases, sometimes in groups, were seen during the 1911 outbreak and a few limited pneumonic epidemics were observed afterwards (Wu Lien-teh [147]). The disease did not show a predilection for any particular season of the year (Sanguy [119]).

Jorge [62] was inclined to think that, though plague became first rampant in the interior of Morocco, " l'origine la plus admissible serait la maritime ",

inapparent infection of the commensal rats having become established in the ports and then spreading inland. Dealing with the rat population in the ports of Morocco he came to the interesting conclusion :

" Le *norvegicus* est prépondérant au Nord et cède dans le Sud la place au *rattus*, surtout à l'alexandrin ; — c'est la transition murine du régime méditerranéen au régime de la Grande Afrique. Au Nord même, le *rattus* tend à accroître son domaine aux dépens de son rival."

R. norvegicus were found in the hinterland together with *Meriones* which were sometimes more numerous.

X. cheopis was the preponderant rat flea. *Nosopsyllus fasciatus* and very rarely *X. brasiliensis* and *X. astia* were also found.

It is of great interest to note that, in the opinion of Blanc & Baltazard,[12] *human* fleas and lice played an important, if not a preponderant role in the epidemic spread of plague in Morocco. The merits of this contention will be discussed in a later section of these studies.

(5) *French West Africa*

Though a few plague cases had been noted in a southern port in 1912 (Jorge),[62] it was in April 1914 that the infection, probably imported by ship from Casablanca, made its real entry into French West Africa in a manner characteristic of the Black Death rather than of modern African outbreaks. As often in the local manifestations of the 14th century, the initial outburst at Dakar was ushered in by a pneumonic phase and it was only 2½ months after the onset that an epizootic became manifest and the bubonic type became prevalent in man. A wide area even including the Cape Verde Islands was reached by the epidemic which, lasting until January 1915, claimed almost 9,000 victims (Wu Lien-teh [147]).

Further plague manifestations in French West Africa became practically restricted to a triangular area in Western Senegal with its tip at Dakar in the west and its northern angle near St. Louis. The incidence of the disease rose after a quiescent period in 1915 and 1916 to reach a maximum of 7,999 cases in 1920 (Damez [16]). The infection was also active from 1928 to 1930, during which period an epidemic at St. Louis described by Lefrou [70] took place, and again in 1934. Then a marked decline lasting until 1942 set in. Numbers of cases of plague from that year onwards are given below (Kaul [65]) :

Year	*Dakar (Circonscription)*	*Senegal*	*Total*
1943	32	266	298
1944	570	69	639
1945	4	54	58
1946-9	0	0	0

It will be noted that in French West Africa as well as in several of the areas dealt with above the hitherto favourable plague situation deteriorated considerably during the second World War, when the spread of the infection

was facilitated through " a certain amount of war destruction and inadequate storing facilities to meet the war emergency " (Stowman [130]).

The plague season in French West Africa fell into the period from June to August (Sorel [127]). Human plague was in general of the bubonic type but pneumonic cases were met with and were possibly frequent in some outbreaks (Wu Lien-teh [147]).

As confirmed by recent statistics from Dakar, *R. rattus alexandrinus* was by far the most frequent among the commensal rodents, *R. rattus rattus* less common, and *R. norvegicus* not numerous. However, as pointed out by Cazanove (quoted by Jorge [62]), the comparative frequency of these rodents was apt to vary in different locations : the Norway rats of Dakar frequented the port and sewers, *R. rattus rattus* was at home in European houses, *R. rattus alexandrinus* in the habitations of the indigenous population.

Plague-positive animals found at Dakar within recent years belonged to the following species :

Year	*R. rattus alexandrinus*	*R. rattus rattus*	*R. norvegicus*	*M. musculus*	*Total*
1935	10	1	1	2	14
1944	21	33	7	3	64
Totals	31	34	8	5	78

Various wild-rodent species have been found plague-infected in Senegal and it was suspected that one of them, the giant rat *Cricetomys gambianus*, played a causal role. It has to be noted in this connexion that recently all rural foci were situated on the railway line leading parallel to the coast from St. Louis to Dakar. It was assumed that the storage and transport of ground-nuts along this route proved attractive to the wild rodents.

Though *X. cheopis* was prevalent, in Senegal as in Morocco a possible role of other fleas in the spread of human plague was suspected. Attention was paid in this respect not only to *Pulex irritans* but also to *Synosternus pallidus* which, though rare on the rats, abounded in human habitations (Kartman [64]). The relative importance of these three flea species in the spread of the infection will be discussed in a later section of these studies.

Central Africa

(1) *Uganda*

When considering the plague situation in Central Africa, it is advisable to deal first with Uganda, where most probably the ancient African centre of the infection mentioned before was situated. It serves as a corollary for this assumption that, as soon as observations became possible in modern times, plague foci of long standing were detected in Uganda (Roberts [113]).

Though there was little evidence for the continued existence of the disease in the Protectorate at the close of the last and quite early in the present century, there can be little doubt that the infection was inapparent

rather than altogether absent. Moreover, it was claimed that during this period the construction of the Uganda Railway, lasting from 1896 to 1901, led to an importation of plague from India. Be this as it may, it is certain that from 1903 to 1908 most of the ports on Lake Victoria became involved and that by 1906 the infection had become so firmly entrenched that from then until 1949 it was never absent from Uganda in any year (Hopkins [53]).

According to figures culled from the report of Hopkins,[53] the mortality reported as due to plague in Uganda during the three decades from 1910 to 1939 was as follows :

1910-9	31,305
1920-9	17,410
1930-9	11,387
1910-39	60,102

The situation which, as shown above, had gradually improved from 1920 onwards became quite favourable during the period from 1940 to 1949 :

Year	*Cases*	*Deaths*
1940	278	268
1941	218	213
1942	354	338
1943	19	0
1944	7	7
1945	4	4
1946	3	3
1947	1	1
1948-9	0	0
1940-9	884	834

As was emphasized by Hopkins,[53] the endemic foci in Uganda from which the plague outbreaks originated were invariably situated in rural localities and not in townships.

To judge from statistics showing the monthly incidence of the disease from 1935 to 1947, cases occurred throughout the year without showing a regular predilection for any particular season. Though human plague was mainly bubonic in character, cases with pneumonic features were met with, recently by Hennessey.[49]

It is interesting to note that, in significant contrast to the original foci in Central Asia, plague seems not to have become entrenched among the wild-rodent species in Uganda or elsewhere in Central Africa.

In recent times *R. rattus* formed the principal reservoir of the infection in Uganda ; it was in many of the plague areas practically the only rat found in human habitations and endemicity never became established in regions where this species was absent. However, *R. rattus* did not seem to have invaded Uganda before the beginning of the present century so that it could not have been responsible for the plague outbreaks occurring before that time. Its place must then have been taken by *R. (Mastomys)*

coucha ugandae which continues to be the most abundant hut-rat in the parts of the Protectorate not invaded by the black rat (*R. rattus*), and which has a flea fauna similar to that of the latter. It is likely that *R. coucha* continued to take a limited part in the causation of plague (Hopkins [53]).

Xenopsylla brasiliensis was, in the opinion of Hopkins, " the normal initiator " of the plague outbreaks in Uganda which, as noted above, invariably started in rural localities. *X. cheopis*, nearly always confined in the plague areas to large townships, helped to carry on the epidemics when such places became invaded, but seemed in general not a vector of great importance in Uganda.

(2) *Kenya*

The earliest plague outbreak on record in Kenya, occurring in 1902 at Nairobi, though not necessarily autochthonous, was probably of local origin because, as pointed out by Roberts,[113] Mombasa, the only port through which the infection could have entered, had been free from the disease for years and remained so until 1912.

From 1906 onwards plague began to spread in Kenya but up to the present its incidence has remained usually below the level reached in Uganda (Roberts [114]). As summarized by Kaul,[65] 600-1,000 cases per year occurred in Kenya from 1926 to 1931, below 300 cases annually from 1932 to 1937, sporadic instances of infection only from 1938 to 1940. However, as shown by the following statistics, the situation became comparatively serious in 1941 and 1942 :

Year	*Cases*	*Deaths*	*Year*	*Cases*	*Deaths*
1941	781	196	1946	35	11
1942	754	333	1947	55	16
1943	17	13	1948	30	16
1944	18	7	1949	5	2
1945	56	21			

It should be noted that in Kenya as in Uganda the endemic foci of plague were situated in rural areas.

A comparatively high incidence of cases with pneumonic features was recorded in some outbreaks, recently by Plum,[105] but attention must be paid in this connexion to the following statement (Hunter [57]) :

" The disease is mostly bubonic in type : septicaemic and pneumonic cases are common, true inspiration pneumonia is rare ".

In a recent study on the transmission of plague in Kenya, Roberts [114] reached the conclusion that *R. rattus* was the only rodent involved and that *X. cheopis* and *X. brasiliensis* served as vectors. Human infection was intense in the *cheopis*-infested areas, probably because this flea which was associated with rats living underground was in far closer and more constant contact with man than *X. brasiliensis* which infested the roof-rats.

(3) *Tanganyika*

Since the old trade route from Uganda to the coast led through Tanganyika (formerly German East Africa), it is not surprising to find evidence for the early existence of plague in the latter territory. Outbreaks in the Uhehe county of the southern province of Iringa seem to have taken place as early as 1886 and 1889, and the existence of endemic plague in that area was confirmed by the German observers of the 1903-4 outbreak at Iringa (Wu Lien-teh [147]). Another endemic focus was detected in 1897 by Koch and Zupitza in Kisiba, a locality between the Kagera River and Lake Victoria (Wu Lien-teh [146]).

Further and sometimes fairly extensive outbreaks continuing in the then German territory from 1906 to 1919 included epidemics in the Gasseni district on the eastern slopes of the Kilimanjaro in 1912 (Lurz [74]), in the Mwanza district and eastern Lake Victoria area in 1913, and at Mwanza township and Dar el Salaam in 1914. After the British occupation, manifestations of the disease continued to be frequent in the central and Lake provinces up to 1928. Except in 1931, when 238 cases were recorded, plague was inconspicuous or even absent during the period from 1928 to 1936. The incidence of the disease from then onwards was as follows :

Year	*Cases*	*Deaths*	*Locality involved*
1937	72	47	Lake province
	61	17	Central province
	2	2	Iringa
	135	66	
1938	3	0	Northern province
1941	2	2	" "
1948	311	174	Central province
1949	18	14	" "

To judge from figures showing the monthly plague incidence from 1936 to 1949, epidemics in Tanganyika usually ran their course during the period from February to July, reaching their peak in March and April. The bubonic type was prevalent but pneumonic manifestations were recorded by the German observers, particularly by Lurz.[74]

Black rats, recently identified as *R. rattus alexandrinus*, appear to be the only rodents playing a causative role in the Tanganyika outbreaks with *X. brasiliensis* as the probable vector.

(4) *Belgian Congo*

In compiling this summary on the plague situation in the Belgian Congo, advantage was taken not only of the voluminous published literature, but also of an excellently documented report received in July 1950, in answer to the inquiry instituted by the WHO Expert Committee on Plague, from the Inspecteur-Général de l'Hygiène, Ministère des Colonies, Brussels, Dr. A. Duren.

Plague exists in the Belgian Congo in two foci situated in the extreme north-east of the colony near the Uganda border—the Lake Albert focus and the Lake Edward focus.

Nosogeographically the first of these foci forms part of the plague areas in Uganda, because Lake Albert, which marks the border between that territory and the affected region in the Belgian Congo, served as a means to convey the infection rather than as a bar to its progress. For this reason it seems probable that, though the presence of plague in the Lake Albert focus was detected in 1928 only, the disease had been of long standing.

Cases in the Lake Edward focus were first notified in 1938. The invasion of this area was evidently due to an importation of the infection from the Lake Albert region, possibly through plague fleas which had been carried by passengers or in goods arriving from there by motor-truck (van Riel & Mol [111]).

The incidence of the disease in the two foci is shown in table VI.

TABLE VI. INCIDENCE OF PLAGUE IN LAKE ALBERT AND LAKE EDWARD AREAS, BELGIAN CONGO, 1928-50

Period	Lake Albert focus		Lake Edward focus	
	Cases	Deaths	Cases	Deaths
1928-37	133	—	—	—
1938-47	249	226	107	93
1948-50 [a]	28	27	22	21

[a] Until the end of June 1950. Cases continued to occur during the second half of the year.

It will be noted that thus far the plague morbidity in the Belgian Congo was low. The maximal case incidence observed in any year was 65 in the Lake Albert focus (in 1939) and 29 in the Lake Edward focus (in 1941). The case fatality-rate on the other hand was high, amounting since 1938 to 91.3% in the former focus and to 88.4% in the latter. Commenting upon the severity of the infection, the report to WHO stated that instances of ambulatory plague seemed to be altogether absent in the Belgian Congo. Cases with lung features were observed and were apparently frequent in the Lake Albert focus in 1939 (van Hoof [52]) when, as noted above, the incidence of the disease was higher than usual. Plague cases seem to be most frequent during the period from April or May to September or October.

Several wild-rodent species, particularly *Arvicanthis abyssinicus*, have been found naturally infected in the Belgian Congo, but, since these animals had been obtained in the vicinity of villages where rat plague existed, they had apparently become secondarily involved.

In the Lake Albert focus *Rattus (Mastomys) coucha ugandae* was by far the most frequent rodent found in human habitations (98%). Among the few other rodents found *Arvicanthis abyssinicus* was comparatively the most conspicuous. It is interesting to note that quite recently an invasion of *R. rattus alexandrinus* has been noted in the Kasenyi region, but so far no instance of infection has been found in this species. *Rattus coucha ugandae* seems therefore the sole reservoir of plague in the focus.

The rodents found in the huts of the Lake Edward focus were *R. rattus alexandrinus* (52%), *R. coucha ugandae* (32%), and *Arvicanthis abyssinicus* (14%). So far the infection has not been detected in the last species, so that the two first-mentioned rodents appear to be responsible for the plague manifestations in the area.

Both *X. cheopis* and *X. brasiliensis* which are considered as the vectors of plague in the Belgian Congo occurred in the Lake Albert focus, the former forming a large majority of the rat fleas in the north and west of the area, the latter being almost exclusively present in the east and south.

In the Lake Edward focus *X. brasiliensis* was largely preponderant.

The wild-rodent flea *Dinopsyllus lypusus* has been found capable of transmitting plague under experimental conditions ; *Ctenocephalides felis strongylus*, which in the Belgian Congo infests man in place of *P. irritans*, was unable to do so.

Madagascar

For a study of the plague situation in Madagascar it was possible to consult, in addition to the voluminous literature, a most valuable summary prepared by the Direction des Services sanitaires in Tananarive for the WHO Expert Committee on Plague and containing information up to July 1950.

Plague was introduced into Madagascar in November 1898, when a ship bringing a rice cargo from India served as the vehicle of the infection. In the following years outbreaks continued to appear in the major ports but never caused much havoc.

The infection was again introduced into the port of Tamatave in March 1921, leading to an epizootic and 107 bubonic cases with 71 deaths. In June of the same year pneumonic plague appeared at Tananarive on the high plateau of the island and claimed 46 victims in the course of three weeks. Four months afterwards foci of bubonic plague became manifest in adjacent localities and signs of rat infection began to be found. Since that time the disease has persisted on the high plateau, practically all parts of which became successively involved.

Occasional outbreaks were noted in the ports as well, but these never caused much harm and soon terminated. Further, while the outbreaks on the coast continued to be bubonic in nature, the infection showed on the high plateau a marked tendency to lead to secondary lung involvement

and thus to produce foci of pneumonic plague. Though no major outbreaks of pneumonic plague occurred, the incidence of this form of the disease was invariably considerable, averaging according to Le Gall [69] 29% during the period 1935-9. Occasionally, the incidence of pneumonic plague was higher, for instance amounting to 56.2% in 1946 (Favarel [25]).

The plague incidence in Madagascar from 1933 to 1950 was as follows :

Year	*Cases*	*Year*	*Cases*
1933	3,933	1942	181
1934	3,605	1943	234
1935	3,493	1944	184
1936	2,006	1945	185
1937	916	1946	278
1938	630	1947	274
1939	681	1948	240
1940	754	1949	143
1941	272	1950	153

It will be noted that from 1936 onwards the incidence of the disease decreased to reach a level of less than 300 cases per year in 1941. Large-scale inoculation campaigns with the live vaccine of Girard and Robic had been started in November 1935 (Robic [115]) and were no doubt responsible for this decrease of the morbidity. However, as recently pointed out by Lepage,[71] the incidence of the disease from 1941 to 1948, though moderate, had remained fairly uniform so that apparently a level had been reached which could not be lowered any more through inoculation campaigns alone. However, application of DDT was started and led to a further decrease in incidence as shown by the following figures :

Period	*Cases*
1st quarter of 1948	135
„ „ „ 1949	61
„ „ „ 1950	34

Lepage also pointed out that during the early months of 1950 no plague case had been recorded in the Emyrne district where DDT had been used most systematically.

The incidence of bubonic plague on the Madagascar high plateau was maximal during the period from 1 December until 31 March, at low ebb during June and July. Pneumonic cases on the contrary were comparatively more frequent from July to December and less numerous during the hot season from December to February (Le Gall [69]).

R. rattus alexandrinus and *R. rattus frugivorus* are the species of commensal rats commonly met with on the coast as well as on the high plateau, but the former predominates. *R. rattus rattus* is preponderant in the forests ; it occasionally enters store houses but is hardly ever found inside human habitations.

No evidence has been forthcoming thus far that wild rodents play a role in the causation of plague, but one species, *Brachytarsomys albicauda*,

living mainly in the forests, has been found rather susceptible to experimental infection.

There can be no doubt that the black rats, in the first line *R. rattus alexandrinus*, form the plague reservoir in Madagascar. *X. cheopis* is the only flea recognized there as vector of the infection.

South Africa

(1) *Union of South Africa*

In compiling this summary, advantage has been taken not only of an ample literature but also of a valuable report on the plague situation in the Union of South Africa from 1935 to April 1950, prepared by D. H. S. Davis for the WHO Expert Committee on Plague.

Plague gained a foothold in the Union of South Africa in 1900 when, on account of the Boer War, large amounts of forage had to be imported from infected South-American ports. As summarized by Mitchell,[90] during the period from 1900 to 1902 serious epidemics took place at Cape Town, Port Elizabeth, East London, and other centres in Cape Province, as well as at Durban and Pietermaritzburg. Further outbreaks occurred in 1903 at King Williams Town, Queenstown, and elsewhere, followed in 1904 by a considerable epidemic with an initial pneumonic phase in Johannesburg and its vicinity. The infection persisted there as well as in Port Elizabeth and East London until 1905, but seemed to be altogether absent from the Union from 1906 to 1911. In 1912 an outbreak, due evidently to a re-importation of the infection from eastern ports, occurred at Durban. As was afterwards realized, that epidemic marked the end of the initial plague period in the Union of South Africa which, because it was characterized by the occurrence of rat-caused outbreaks in urban areas, has been termed the " murine " phase (Davis [18]).

From 1914 onwards outbreaks were noted in remote rural localities, first in Cape Province, from 1916 onwards also in the Orange Free State, whence the infection spread in 1917 to a neighbouring district of Transvaal. The total incidence from 1914 to 1918 (when two fatal cases were recorded in the Orange Free State) was 189 cases with 132 deaths. Though it was suspected that wild rodents might be responsible for these manifestations of the disease, proof for this assumption could be obtained only in 1921 (Mitchell [90]).

It was formerly believed that a gradual propagation of plague to the rural areas had taken place, the striped mouse (*Rhabdomys pumilio*) contracting the infection in the outskirts of the infected urban centres and passing it on to other wild-rodent species (Thornton [133]). Though such a spread took place occasionally (Mitchell [90]), it was of main importance that a direct transport of infected rats and fleas by rail or other means of traffic led to the establishment of three primary distributing centres of wild-rodent plague which were situated in south-western Transvaal and

north-western Orange Free State, in the Cape midlands, and in the Uitenhage district near Port Elizabeth respectively (Fourie,[27] Davis [18]).

As the result of an expansion of the infection from these primary foci, a vast area, comprising more than half of South Africa, from the Cape to Barotseland in Northern Rhodesia became affected. It would seem that a spread beyond the limits thus reached is not to be expected (Davis [18]).

The incidence of the disease from 1 July 1919 to the end of June 1949 was as follows :

Period	*Cases*	*Remarks*
1919-25	556	Expansion of plague from the original foci due to a continued spread of plague among wild rodents.
1925-31	466	Outbreaks mainly in new areas, as a result of major extensions of the enzootic area in the karroo (Cape midlands) and northern Orange Free State. Spread also into South-West Africa and Bechuanaland.
1931-7	687	Epidemic in the Orange Free State. Extension and consolidation of the infection in other parts of the affected areas.
1937-49	646	Continuation of perennial or almost perennial outbreaks in hyperenzootic areas situated in the northern Orange Free State, a district on the Transkei border in the eastern Cape area, and in the Uitenhage/Port Elizabeth districts. Sporadic manifestations in the other parts of the enzootic area (Davis [18]).

The incidence of human plague in the Union of South Africa was found to be highest during a season lasting from December to April. However, the wild-rodent epizootics being unaffected by seasonal changes, epidemics have occurred in all months of the year (Davis, communication to WHO). Bubonic manifestations preponderated, but pneumonic outbreaks have been recorded both during the " murine " phase and subsequently in the wild-rodent foci, recently by Gale [29] and Clark & Goldberg.[14]

Though, as stated by Davis in his communication to WHO, over 100 species and subspecies of rodents and other small animals are at risk of infection and some 60 species of fleas have to be regarded as actual or potential vectors, the main reservoir of wild-rodent plague in the Union of South Africa is formed by two gerbils, *Tatera brantsi* and *Desmodillus auricularis*, associated with *Xenopsylla philoxera* (*X. eridos* auctt., not of Jordan and Rothschild) and *X. piriei* respectively. Human infection was in most instances not directly derived from the wild rodents or through their fleas, the semi-domestic *R. (Mastomys) coucha natalensis* and *R. rattus* acting as intermediaries between the primary gerbil reservoirs and man. *R. (Mastomys) coucha natalensis* carried a mixed fauna of wild and commensal rodent fleas, while *R. rattus* met with in rural areas was infested with *X. brasiliensis*, the flea considered as the main vector of human plague in the Union.

Though, as noted above, rodents with semi-domestic or domestic habits were apt to become involved, instances of a transition of the infection from the wild rodents to urban rat populations have been almost totally absent thus far. Only one outbreak at Port Elizabeth in 1938 is on record where a relatively short-lived focus was established amongst the commensal rats and gave rise to 28 cases. Positive rats were found also at Johannesburg in 1943, but no general epizootic resulted (Davis, communication to WHO). Plague in the Union of South Africa appeared thus within recent times almost invariably in rural localities, where as a rule a few cases only occurred at one and the same time. Under these circumstances it is not surprising to find that, according to Ferguson,[26] 1,005 individual outbreaks took place from 1920 to 1949. Since he recorded the occurrence of 2,361 cases during this period, the case incidence per outbreak averaged 2.3.

(2) *Basutoland*

The recent plague incidence in Basutoland, which nosogeographically forms a part of the enzootic areas in the Union of South Africa, was as follows :

Year	*Month*	*Cases*	*Deaths*
1935	December	9	9
1936	January-February	7	7
1937-41	—	0	0
1942	February	10	4
1943	January, March, June	27	10
1944	—	0	0
1945	November	8	4
1946-8	—	0	0
1949	November-December	92	?

(3) *Bechuanaland*

Though Ngamiland in the Bechuanaland Protectorate was reached in 1928 by a wave of wild-rodent plague spreading from the north-west Cape area and the enzootic then established culminated in epizootics in 1934-5 and possibly also in 1939-40, throughout this period only two instances of human plague were recorded—in April 1935. However, the reappearance of an epizootic in 1944 led to a major epidemic which, lasting from October to December, resulted in 304 cases with 156 deaths. Sporadic cases continued up to March 1945 (Davis [17]). The further incidence of plague was as follows :

Year	*Cases*	*Deaths*
1945	18	16
1946	68	57
1947	2	2
1948	0	0
1949	24	20

As stated in the Bechuanaland Protectorate Medical and Sanitary Report for 1949,[8] signs of an active epizootic were noted during that year in several

areas. The Gobabis district in South-West Africa, close to the Bechuanaland frontier, was also found to be affected and several fatal cases of human plague were observed there at the end of 1949.

Gerbil species (*Tatera* and *Desmodillus*) formed the permanent reservoir of the infection which, as will be perceived, was apt to flare up every fourth or fifth year. *R. rattus* and *M. musculus* appeared to be absent from Bechuanaland, but the multimammate mouse (*R. coucha*) acted as the intermediary between the wild rodents and man.

The gerbils were found to be infested with *X. eridos* (*X. philoxera*) and its close relative *X. hipponax*. Both these species occurred in addition to *X. brasiliensis* on the multimammate mice, but the last-mentioned flea seemed to be responsible for the majority of human infections (Davis [17]).

(4) *Northern Rhodesia*

Suspicious outbreaks were reported in the Luangwa Valley of Northern Rhodesia in 1917 and 1918, but their plague nature seems not to have been confirmed (report prepared for the WHO Expert Committee on Plague). More recently, however, the infection appeared in the Barotse Province which was evidently invaded by wild-rodent plague from the south through the Kalahari (Davis [19]).

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 (incomplete)

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 [19]). 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. eridos*, *X. hipponax*, and occasionally *Dinopsyllus lypusus*, which was the only flea infesting the swamp-rats. It is interesting that *X. eridos*, 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 *eridos*, might be a vector, no undue stress ought to be laid upon the absence of the latter flea.

R. coucha 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 [19]).

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 [93])

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 [28]).
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 [147]).
Totals		448	286	

(*b*) *Infections contracted from wild rodents or through their fleas* (after Link [72])

State	*Year of detection of:* *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	5	2
Arizona	1938	1950	1	1
Totals	1908-50		90	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,[60] Meyer [88]). Meyer [88] 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 section 2 of these studies, 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 [67] 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 [72]) :

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

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

FIG. 3. DISTRIBUTION OF PLAGUE, 1945-8. I — THE AMERICAS

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 amongst these animals has been found. Discussing this problem, Meyer [87] 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,[23] 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 [93] endorsed this opinion but also incriminated sage-brush voles and certain meadow mice.

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 [86]). 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 [147]), and that the presence of plague amongst 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 [56]). 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,[89] Mohr [93]).

Working recently on a Californian ranch round which wild-rodent plague was present, Meyer & Holdenried [89] 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
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,[22] 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,[44] 360 of the 397 patients seen from 1899-1933 and all 17 observed from then up to 1944 died.

(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 [32]).

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 in 1936, 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 [55]). 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. tuberculatus*, and *Oropsylla rupestris*, three species of fleas found able by Eskey & Haas [23] to convey plague under experimental conditions.

It is reassuring that according to Humphreys & Campbell [55] 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 [33]).

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 remains involved to date. As stated by Isaac Riaz,[59] the two affected districts cover an area of 1,000 km^2 (386 square miles) —1% of Venezuelan territory.

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

Miranda State

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

Total cases 1910-50 = 211

Aragua State

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

At Caracas *R. norvegicus* formed 98% of the rat population and *X. cheopis* 95% of the rat fleas as against 3.4% *X. brasiliensis* (Hecht [46]).

The commensal species (*R. norvegicus*, *R. rattus rattus* and *rattus alexandrinus*) were also present in and around the settlements of the plague-affected rural areas and were no doubt responsible for the appearance of plague in man, *X. cheopis* and *X. brasiliensis* serving as vectors. However, Isaac Riaz [59] postulated with much reason the existence of wild-rodent foci where the infection was carried over during the often prolonged periods of its absence from human settlements. Actually, instances of natural plague were found in two wild-rodent species, *Heteromys anomalus* and *Sigmodon hirsutus*. Isaac Riaz pointed out in this connexion that the fleas of the genus *Polygenis*, which formed the bulk of the fauna on the wild rodents, were particularly apt to produce smouldering enzootics because they do not become blocked when ingesting plague bacilli. It is, however, of interest that sometimes a high percentage of *Xenopsyllae*, particularly *X. brasiliensis*, was found on the wild rodents (Hecht,[47] Isaac Riaz [59]). An increased incidence of the latter flea was noted in June 1943, one month before the appearance of the outbreak in Aragua State. The presence of 52.2% *X. brasiliensis* and 20.3% *X. cheopis* in May 1942 did not lead to an epidemic, but one might venture to suggest that a high prevalence of *Xenopsyllae* on the wild rodents was of importance only when at the same time plague was active amongst these animals. The necessity for a coincidence of these two factors might explain why in the rural areas of Venezuela years with manifestations of human plague are separated by long periods of quiescence.

(2) *Brazil*

It is generally accepted that plague, imported by the sea-route, first appeared in Brazil in 1899 when Santos and a few months afterwards São Paulo, lying inland from that port, became infected. From then onwards up to 1906 Rio de Janeiro, Fortaleza in the State of Ceará, Pernambuco, Rio Grande do Sul, and other ports became successively involved. From 1907 onwards the infection began to spread to inland cities and towns but disappeared since 1934 rapidly from these centres while persisting in rural areas which remain in part involved to date (Barreto & Castro [7]). The total incidence of the disease from 1899 to 30 June 1949 is shown by the following approximate figures (Moll &

O'Leary ; [94] A. Castro, communication to WHO Expert Committee on Plague) :

Period	*Cases*
1899-1929	5,638
1930-4	535
1899-1934	6,173

Period	*Cases*	*Deaths*
1935-9	1,223	490
1940-4	812	205
1945-9	1,040	182
1935-49	3,075	877

Total cases 1899-1949 = 9,248

Details of the plague incidence in Brazil from 1935 onwards are given in table VII (A. Castro, communication to WHO Expert Committee on Plague).

TABLE VII. NUMBER OF CASES OF AND DEATHS FROM PLAGUE IN BRAZIL, 1935-49

Year	States of								Total	
	São Paulo		Rio de Janeiro		Minas Geraes		North-East Brazil			
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
1935	2	1	—	—	—	—	569	232	571	233
1936	31	21	—	—	—	—	328	115	359	136
1937	1	0	—	—	—	—	35	15	36	15
1938	—	—	12	8	—	—	134	53	146	61
1939	4	2	—	—	—	—	107	43	111	45
1940	—	—	—	—	—	—	255	53	255	53
1941	—	—	7	4	—	—	295	83	302	87
1942	—	—	—	—	3	0	32	7	35	7
1943	—	—	—	—	—	—	66	22	66	22
1944	—	—	—	—	—	—	154	36	154	36
1945	—	—	—	—	—	—	192	42	192	42
1946	—	—	—	—	34	23	298	48	332	71
1947	—	—	—	—	7	3	81	8	88	11
1948	—	—	—	—	—	—	386	54	386	54
1949[a]	—	—	—	—	—	—	41	4	41	4
1935-49	38	24	19	12	44	26	2,973	815	3,074[b]	877

[a] Until 30 June

[b] One recovering case in Sergipe State in 1946 not included

It will be noted that except for one outbreak in Minas Geraes State in 1946-7 which has been dealt with by Macchiavello & Martins de Almeida,[79] from 1943 onwards north-eastern Brazil alone was involved. The situation there is given in table VIII.

As shown by table VIII, the situation was serious in four of the six States concerned, particularly in Pernambuco.

As was to be expected, the seasonal incidence of plague in São Paulo in the south of Brazil was different from that farther north. Most of the cases of bubonic plague in São Paulo occurred during the sultry days of summer with the peak in January (Oliveira,[98] Moll & O'Leary [94]). In marked contrast to this a pneumonic plague outbreak at São Paulo in 1936 took place during the winter in July (de Moura Albuquerque [95]), and the same held true of an earlier pneumonic plague epidemic observed at Santa Maria in the State of Rio Grande do Sul in 1912 (Wu Lien-teh [147]).

The period of maximal plague incidence in Rio de Janeiro was from September to January, commencing thus earlier than in São Paulo (Rangel, quoted by Moll & O'Leary [94]).

As shown by monthly figures supplied to the WHO Expert Committee on Plague by Castro for the period from 1935 to 1949, the seasonal plague incidence in the north-east of Brazil was not as clear-cut as in the south of the country :

Month	*Number of cases*
January	233
February	194
March	235
April	190
May	103
June	75
July	403
August	312
September	372
October	364
November	250
December	242

Still, the incidence of plague in north-eastern Brazil during these years was comparatively highest from July to October, thus roughly corresponding to the seasonal period mentioned by Barreto [6] for Pernambuco. He claimed an earlier onset for Ceará which is situated farther north (May-June) and stated that in both States the period of maximal plague incidence coincided with the harvest season.

While, as noted above, occasional pneumonic epidemics occurred, bubonic plague was largely preponderant, 95.7% of the cases observed in Brazil from 1936 to 1945 being of this type as against 2.16% pneumonic and 1.7% septicaemic cases (Barreto & Castro [7]). The plague mortality

TABLE VIII. CASES OF AND DEATHS FROM PLAGUE IN THE STATES OF NORTH-EASTERN BRAZIL, 1935-49

Period	Alagoas		Bahia		Ceará		Parahyba		Pernambuco		Piauhy		Total	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
1935-9	74	23	79	31	317	95	14	6	673	297	16	6	1,173	458
1940-4	256	36	126	39	108	21	—	—	312	105	—	—	802	201
1945-9[a]	53	8	319	48	249	44	23	3	354	53	—	—	998	156
1935-49	383	67	524	118	674	160	37	9	1,339	455	16	6	2,973	815

[a] Until 30 June

was generally low and, as will be discussed in a subsequent section of these studies, atypically mild cases were frequent.

Though, as will be recorded in a later section of these studies, natural plague has been found in a whole series of Brazilian wild rodents, it was held by Macchiavello [75] and other observers that these animals did not form a primary reservoir of the infection but were secondarily or even accidentally involved.

While *R. rattus* was preponderant in the rural areas of the north-east, Norway rats were most common in some of the ports and in São Paulo State. In Rio de Janeiro this species and *M. musculus* were more frequent than *R. rattus* and *rattus alexandrinus*. Though *X. brasiliensis* occurred everywhere side by side with *X. cheopis*, the comparative frequency with which the former flea was present on the rats increased pari passu with the latitude of the regions in question (Barreto & Castro [7]). *X. cheopis* was considered as the plague vector in Brazil (Macchiavello [75]).

(3) *Argentina*

Apparently by-passing Buenos Aires and the Argentinian ports on the Parana River, plague became manifest first in South America at Asuncion, situated far inland in Paraguay, which had been reached in April 1899 by an infected steamer. From this original focus the disease was soon carried back to Rosario and other river ports in Argentina, at the end of the year also to Buenos Aires.

The initial period of the infection thus established was followed first by a stage during which plague was carried inland by rail, then by further progress of the pest to remote regions in the interior where wild-rodent foci were created. A wide area, extending from the provinces of Jujuy

and Salta on the Bolivian border in the north to Patagonia in the south, became thus gradually involved (Sussini [131]).

As pointed out by Barrera,[5] generally speaking the inland foci of plague in Argentina fell into two groups :

(*a*) Those in the central part of the country, particularly in the provinces of Rio Negro, La Pampa, Mendoza, and San Juan, with a sparse population and no agriculture. Since grain stores which might have attracted the rodents to the settlements were absent, contact with infected animals was restricted to chance meetings in the fields, and the incidence of human plague was consequently low.

(*b*) Those in the north, especially in Santiago del Estero, Tucuman, and Salta, where accumulation of agricultural products attracted the rodents to the settlements and houses, and the incidence of human cases was accordingly higher.

The number of plague cases in Argentina from 1899 to 1930 amounted to about 6,200 (Moll & O'Leary [94]). The recent incidence of the disease was as follows :

Year	*Cases*	*Deaths*	*Year*	*Cases*	*Deaths*
1931	37	24	1941	56	28
1932	61	19	1942	38	19
1933	63	25	1943	2	1
1934	45	40	1944	107	63
1935	15	10	1945	?	3
1936	31	21	1946	10	3
1937	20	18	1947	4	0
1938	15	8	1948	12	4
1939	5	4	1949	0	0
1940	228	192			

Note : In 1940 the provinces of Santiago del Estero, Cordoba, and Tucuman were mainly involved, in 1944 Salta and Jujuy Provinces.

Though these figures show the trend of the infection rather than the exact incidence of the disease, which was probably higher, they indicate that on the whole the plague situation in Argentina was favourable within recent years. The ports were free after 1931 with the exception of a small outbreak (8 cases with 3 deaths) in November 1946 at Buenos Aires (Moll & O'Leary [94]). Considering the large extent of the areas involved, the morbidity in the rural districts was as rule a rather low and, moreover, as is characteristic of the manifestations caused by wild rodents, human cases were usually not grouped together but appeared in numerous foci which were independent of one another (Outes,[99] Villafañe Lastra et al.[140]). It must be noted, however, that pneumonic cases were comparatively frequent and that repeated though usually limited pneumonic plague epidemics were observed. Miyara et al.[92] recorded 27 such outbreaks with a total of 222 deaths and one instance of recovery for the period 1913-38.

Villafañe Lastra et al.[140] noted the interesting fact that within recent years there was a notable increase of outbreaks taking place in autumn and winter whereas formerly the plague incidence had been highest in summer.

Among the commensal rodents of Argentina, Norway rats were predominant in the towns as well as in the rural areas. *R. rattus rattus* was less common, *R. rattus alexandrinus* least frequent, except in the coastal provinces. *X. cheopis* was the prevalent flea species on these animals (Moll & O'Leary [94]).

As summarized by Macchiavello,[78] natural plague was found in the following wild-rodent species of Argentina :

Cavia pamparum
Galea musteloides (2 subspecies)
Caviella (Microcavia) australis (2 subspecies)
Graomys griseoflavus (2 subspecies)
Hesperomys murillus cordovensis
Lepus europaeus
Sylvilagus brasiliensis gibsoni

Two of the fleas found on these rodents, *Delostichus* (formerly *Parapsyllus*) *talis* (on *Caviella australis*) and *Polygenis* (formerly *Rhopalopsyllus*) *platensis cisandinus* (on *Gr. griseoflavus*) have been found capable of transmitting plague by their bites and attacking man (Barrera [4]).

The relative importance of the commensal rats and of the wild rodents, as well as the question of a transition of plague from the former to the latter or vice versa have been the subjects of much debate. No doubt can exist that originally the infection spread from the commensal rats (probably from *R. norvegicus*) to the wild-rodent species (Villafañe Lastra et al.[140]), and it is also certain that foci have become established where wild rodents alone are responsible for the causation of human plague as well as for the perpetuation of the infection. However, in other localities a spread of the disease from the wild to the commensal rats was observed and the latter then played a subsidiary or even a preponderant role in the causation of human attacks. Barrera [5] maintained that opportunities for contact between the wild and the commensal rodents were present, particularly in the agricultural areas of North Argentina where both were attracted by the grain stores, and a study of the available literature confirmed that involvement of the commensal rats in the chain of infection was far more frequently noted there than in the central provinces.

(4) *Bolivia*

Though the presence of plague in southern Bolivia was bacteriologically confirmed in 1928 only, there can be little doubt that outbreaks, due primarily to an importation of the infection from North Argentina, have occurred since 1921. The somewhat incomplete information on these manifestations of the disease, culled from Moll & O'Leary's compilation

as well as from other sources (Veintemillas,[139] Mealla,[84] Prado Barrientos,[107] Siles,[125] Cors,[15] Benavides [9]), may thus be summarized :

Area	*Year*	*Season*	*Cases*	*Deaths*	*Remarks*
Tarija Department	1921	January-July	1,525	642	Several localities involved
	1921-2	December-May	375	300	Several localities involved ; 87 of the cases were pneumonic
	1937-8	December-March	90	18	Preceded by rat epizootic
	1943	June	10		Outbreak at Moreta
	1944		12		Infected rats still found in 1946
Vallegrande Province	1928	June-July	300	88	Bacteriologically confirmed
	1929-34				Suspicious outbreaks
	1935	July	12	9	Two foci
Tomina Province	1933	January		800	In the Department of Chuquisaca
		June		Over 100	
	1934-7	December-February			Yearly outbreaks
	1938-40		140	81	Rat epizootics present
	1944		5		In the Department of Chuquisaca
	1946		1		In the Department of Chuquisaca
Santa Cruz	1938	August-October	150	62	At Choreti and Camiri
	1943		10		At Gutierrez
	1944		5		
	1945		79	40	
	1946		12		
El Palmar (Chaco Boliviano)	1938	April-July	90	50	

While, as noted above, a rat epizootic was responsible for the 1937-8 outbreak in the Tarija Department and the same held true for at last some of the subsequent epidemics, the cause of the earlier plague manifestations in Bolivia has not been elucidated. Signs of rat epizootics seemed then to be absent and, though in later years a few wild rodents were found to have succumbed to plague (Alvarado,[1] Macchiavello [78]), there was no evidence to prove that the infection had become entrenched amongst them. It was postulated therefore that human parasites might have been responsible for the early spread of the disease, the more so because the people used to crowd together to hold prolonged wakes over the dead. Since similar claims have been made elsewhere, it is proposed to deal in a later section of these studies in a general manner with the role of human parasites in the spread of plague.

(5) *Peru*

As Moll & O'Leary [94] state :

"Peru has the unhappy distinction of having had more plague than any other American country, and no plague-free years since its introduction."

Following its importation by the sea-route into the port of Callao in 1903, the disease spread along the coast of Peru, most of the principal ports becoming infected within two years, and eventually invaded 10 of Peru's 20 departments, as well as the three special provinces of Tumbes, Callao, and Moquegua. The situation became worst in the coastal departments of Lambayeque, Libertad, and Lima, as well as inland in Cajamarca (Eskey,[21] Moll & O'Leary [94]). The former three areas remained infected to date whilst Cajamarca reported cases in 1948.

The total incidence of plague in Peru up to 1949 may be summarized thus :

Period	*Cases*	*Annual average*
1903-12	8,865	886
1913-22	6,922	692
1923-32	4,642	464
1933-42	1,087	109
1943-9	655	93
1903-49	22,171	472

Note : The maximal yearly incidence was in 1908 (1,691 cases). A second peak (1,200 cases) was reached in 1926.

As shown by the classical study of Eskey,[21] the central part of Peru, situated between the 7th and 13th degrees of latitude and specially the areas between the 7° and 9° with an average annual mean temperature between 69°F (20.5°C) and 71°F (21.7°C), suffered most from plague. There the infection spread rapidly to rural as well as urban communities, produced more cases than elsewhere in the country, and showed little tendency to disappear spontaneously. However, the degree of rat infestation of the houses was also of importance. Thus the ports of Paita and Mollendo, though situated well away from the zone where the climate favoured the spread of plague, suffered heavily because their wooden buildings were attractive to the rats. Lima, on the contrary, though open to inroads of the infection as far as the climatic conditions were concerned, had a lesser morbidity than these two ports because of having better-class houses.

Generally speaking, the annual plague epidemics in Peru tended to reach their peak during the summer months. However, there as elsewhere the plague seasons fell into an earlier period in areas where the winter months were warm than in localities with a colder climate (Eskey [21]).

Human plague in Peru was mostly bubonic. Pneumonic plague was rare in general but one outbreak, claiming 21 victims in the department of Junin, was mentioned by Moll & O'Leary.[94]

As stated by Eskey,[21] *R. norvegicus*, *R. rattus*, and *R. rattus alexandrinus* were common in the towns of northern Peru, while in the central and southern coastal areas as a rule Norway rats greatly exceeded the other two species. In the rural districts near Lima *R. norvegicus* and *R. rattus alexandrinus* were found. *X. cheopis* was the most common rat flea and, according to Eskey, the only important plague vector.

Ramos Díaz,[108] investigating a plague outbreak in the mountainous region of Lambayeque, found that the epidemic was preceded by an epizootic among the guinea-pigs kept in the house of the first patient. The rats (*R. rattus*) in this locality lived in the fields but visited the houses at night, thus coming in contact with the guinea-pigs. The latter were infested with *P. irritans* as well as *X. cheopis*. Ramos Díaz was of the opinion that the former flea conveyed the infection to man and also believed that transport of *P. irritans* in garments might lead to sporadic human infections without the intervention of rats.

Though formerly wild rodents had been found naturally plague-affected in Peru, the infection was apparently not entrenched among such species. Recently, however, Macchiavello [77, 78] was able to prove the existence of a wild-rodent focus on the Peruvian-Ecuador border where squirrels (*Sciurus stramineus nebouxi*) and some other species were found to be involved. The flea of the former rodent, *Polygenis litargus*, was found to transmit the infection.

(6) *Ecuador*

After having reached Guayaquil by the sea-route in 1908, plague not only persisted in that port until 1930 but soon began to invade both coastal and inland areas of Central Ecuador. The province of Loja in the south also became involved eventually, but this was due to repeated importation of the disease from adjacent endemic areas in Peru.

The progress of plague in Ecuador up to 1940 has been well illustrated by a table compiled by Moll & O'Leary : [94]

Locality	*Period*	*Cases*	*Remarks*
Coastal Zone :			
Guayaquil	1908-39	7,921	After an absence from 1931 to 1934 plague was reintroduced in 1935 to last until 1939.
Provinces of Los Rios El Oro and Guayas (except Guayaquil)	1909-39	416	According to Sáenz Vera,[118] two small outbreaks, due to importation from Peru, took place in El Oro in 1940 and 1944 ; cases were once more reported there in 1950
Manabi Province	1913-37	337	
Central Zone :			
Chimborazo Province	1909-40	1,335	Infection continues to exist up to the present
Tungarahua Province	1916-29	187	
Leon Province	1926, 1929	68	
Canar	1933	45	Plague had been imported from Peru (Sáenz Vera [118])

Southern Zone :			
Loja Province	1921-40	1,411	Plague, which has probably been present since about 1919, continues to occur to date

Having become exclusively rural in character, the infection persists in Ecuador, mainly in the mountainous areas of Chimborazo and Loja Provinces, as shown by the following data :

Year	*Cases*	*Deaths*	*Year*	*Cases*	*Deaths*
1941	39	14	1946	45	19
1942	5	2	1947	21	—
1943	15	9	1948	40	38
1944	36	11	1949	19	5
1945	38	16	1950	27 *	—

* Until 30 September

In general, plague in Ecuador was most rampant in December, of low incidence in June. However, in Loja Province outbreaks occurred chiefly in the dry season from May to December (Moll & O'Leary [94]).

Whilst generally bubonic cases were most common, the incidence of pneumonic plague was comparatively high in the mountainous areas. Sáenz Vera [117] noted in this connexion in 1941 that, while only 43 out of the 8,000 odd cases observed in the coastal areas since 1908 were pneumonic, there were 194 instances of pneumonic plague (22.2%) among the 874 cases recorded in Chimborazo Province from 1913 onwards, invariably in rural localities. According to Murdock [96] three pneumonic epidemics took place in Ecuador during 1939 :

Period	*Province*	*Locality*	*Deaths*
January-February	Chimborazo	Riobamba	17
April	„	Columbe	14
September	Loja	Cofradia-Loja	At least 7

Except in the case of pneumonic plague, the plague mortality in Ecuador was as a rule low. Two peculiar forms of the disease, angina pestosa (tonsillar plague) and viruela pestosa (plague-pox) were met with there.

While *R. norvegicus* was preponderant in Guayaquil, *R. rattus alexandrinus* and *rattus* were more frequent than the first-mentioned rat in the sierra towns which had become infested only after the construction of the railway (Martinez [82]). As maintained by Macchiavello,[76] commensal rats (*R. rattus rattus* and *rattus alexandrinus*) were also prevalent in the rural areas of the interior. He considered that these rodents were of prime importance in the causation of the plague outbreaks there, the guinea-pigs, amply bred in the houses for culinary purposes, playing merely an auxiliary role. While in the mountains as well as elsewhere in Ecuador *X. cheopis* was the common vector of the infection, Macchiavello came to the interesting conclusion that at altitudes over 9,100 feet (2,770 m), where conditions

were unfavourable for that flea, *Nosopsyllus londiniensis* took an important part. Since its vector capacity was not high, human cases tended to be sporadic.

Apart from the above-mentioned focus comprising adjacent parts of Ecuador and Peru, wild rodents played no role in the causation of plague in the former or the latter country.

* * *

As will be gathered from the account rendered above, in many of the countries dealt with the incidence of plague has markedly decreased within recent years and in some the disease has ceased to be manifest for the present. However, even apart from the fact that some areas remain seriously involved, it is not yet possible to be complacent about the plague situation in the world.

As its history teaches, plague has often shown a decline even if left alone but was apt to flare up again in due course. Hence, while appreciating the strenuous efforts now often made to combat the scourge, one should be careful not to ascribe to the measures implemented what might really be the outcome of a periodicity of the infection.

While human plague has become a quite preventable as well as an almost always curable disease, it is melancholy to see that on account of administrative or fiscal difficulties it is still often impossible to take full advantage of the modern methods of treatment and prevention.

Moreover, even universal success in these directions will not eradicate the infection which will continue to lurk among the rodents unless effective action against these pests can be combined with the therapeutic and prophylactic work.

There is hope that, in addition to the campaigns undertaken for the specific purpose of plague control, general progress in health and wealth will gradually prove inimical to the commensal rats. It is to be feared, however, that the vast primary reservoirs of the infection among the wild rodents will remain unassailable for a long time to come.

SUMMARY

A brief review of the early history of plague is given in the first part of the article. The origin of the disease is debatable—it may have been in Central Asia or Central Africa—but the first satisfactory evidence regarding the prevalence of plague concerns the outbreak of 542 A.D. This pandemic lasted fifty to sixty years and

RÉSUMÉ

Dans la première partie de cet article, l'auteur donne un aperçu de l'histoire ancienne de la peste. L'origine de cette maladie prête à discussion : elle peut être recherchée soit en Asie centrale, soit en Afrique centrale ; les premiers témoignages sérieux sur l'apparition de la peste se rapportent à l'épidémie de 542 après J.-C.

spread throughout the world. The next reliable evidence concerns the " Black Death " of the 14th century, after which the pandemic became firmly entrenched among rats, so that frequent or perennial epidemics occurred in many countries in the following centuries. During the 17th century the disease disappeared from Europe, apart from occasional outbreaks, but in the 18th and 19th centuries plague became established in China and Burma.

The second part consists of a general appreciation of the situation in those areas of the world where plague continues to exist or was recently present. Numbers of cases are given, where possible, the route of invasion discussed, and the types of rodents and vectors named.

Cette pandémie dura cinquante à soixante ans et s'étendit sur tout le monde connu. Les données ultérieures auxquelles on peut ajouter foi concernent la « Peste noire » du XIV^{e} siècle. Après cette époque, la pandémie prend le caractère d'une enzootie sévissant parmi les rats et devient, au cours des siècles suivants, la source d'épidémies fréquentes ou d'un état d'endémicité pesteuse dans de nombreux pays. Durant le XVII^{e} siècle, la maladie disparaît de l'Europe, exception faite de certaines épidémies de caractère occasionnel ; en revanche, au XVIII^{e} et au XIX^{e} siècles, elle s'implante en Chine et en Birmanie.

Dans la deuxième partie de l'article, l'auteur expose, d'une manière générale, la situation dans les régions du monde où la peste persiste et dans celles où elle a été récemment constatée. L'auteur donne, lorsqu'il est possible, des indications sur le nombre des pestiférés, il examine par quelles voies l'épidémie se propage et indique les types de rongeurs et de vecteurs incriminés.

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