

# Epidemiological Studies of Plague in India

## 1. The Present Position

S. C. SEAL<sup>1</sup>

*Plague is apparently receding from India, but whether this recession heralds its final disappearance from the subcontinent or is merely a phase in its secular trend or is perhaps due to the effect of control measures is a matter for consideration. On the correct assessment of the present position will depend the nature of the steps to be taken now or in the future. Among the factors considered in this assessment are the possible existence of endemic plague foci in India, the clinical forms of the disease encountered, the relative frequency and epidemiology of urban and rural plague, seasonal variations in prevalence, and the likelihood of resistance of fleas to insecticides.*

Plague, which was once a dreaded disease in India and had its feet deeply rooted in her soil, now seems to be withdrawing from there in full retreat. Whether, as in Europe, this steady regression will prove to be a prologue to the final disappearance of plague from the Indian subcontinent or whether it will turn out to be merely a phase in the secular course of the disease, and to what extent it is attributable to effective measures of control and treatment, are questions for serious consideration. It is on the correct assessment of the present situation that the nature of the steps to be taken against plague now and in the future will depend.

The first authenticated plague epidemics in India in modern times occurred in 1895-96 and from 1898 onwards the disease was appreciably manifest, reaching a peak in the year 1907. Since then there has been a continuous decline in the mortality from plague, as can be seen from Fig. 1 and from the decennial mortality data presented in Table 1. From a general inspection of these data the position seems to be very reassuring, but closer analysis of the figures for 1939-57 (see Table 2) reveals that the situation deteriorated between 1942 and 1947, which was a year of peak mortality. According to Kaul (1949), this was only a temporary setback, consequent upon the food shortage during the Second World War which necessitated the movement of grain from certain central collecting-stations to various areas where food was scarce and thus may have led to the dissemination of plague infection;

however, since hardly any new areas were affected, this does not seem to be an entirely satisfactory explanation. The human exodus from West Pakistan in the last quarter of 1947 was also thought to be a possible explanation, but again on closer scrutiny it can be seen that the mortality rate for 1948 was less than half that for 1947. Only in the Punjab was there any evidence that the exodus might have influenced the mortality rate.

Similarly, Greval's editorial note in 1948 to the effect that plague was imported from Bihar could not be substantiated, as a detailed study of Calcutta plague by the present author has definitely established that it was a recrudescence of an old focus.

### POSSIBLE ENDEMIC FOCI

Sharif (1951) in his study on the endemicity of plague in India suggested that the infection was entrenched in three groups of foci in northern, central and southern India (Fig. 2).

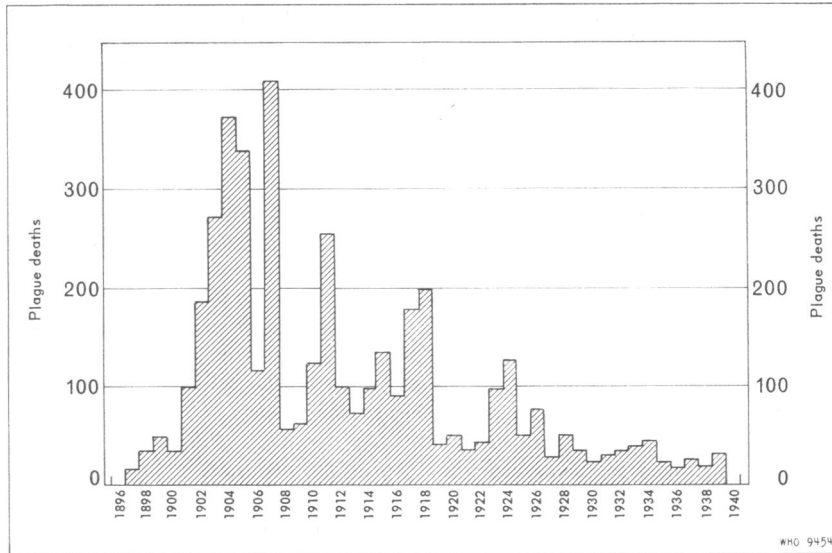
(1) The northern foci consist of three endemic centres at the foot of the Himalayas, perhaps forming part of a big sub-Himalayan focus. These centres were considered responsible for plague outbreaks in East Punjab, Uttar Pradesh (formerly United Provinces) and districts of Bihar north of the river Ganges.

(2) The focus in central India (Madhya Pradesh) comprises the watersheds of the Vindhya, Bhanrer and Maikal ranges and the Mahadeo hills.

(3) The three southern foci are situated in:  
(a) the watersheds of the Western Ghats of Bombay

<sup>1</sup> Professor of Epidemiology, All-India Institute of Hygiene and Public Health, Calcutta, India.

FIG. 1  
PLAGUE MORTALITY IN INDIA PER 100 000 POPULATION, 1896-1939



and Mysore States, (b) the watersheds located in the districts of Salem, Coimbatore, Nilgiri and Madura in Madras State, and (c) the hilly regions of Hyderabad State.

The endemic centres in southern India may have been established after the city of Bombay became infected in 1896. Bombay and Mysore States and the adjacent parts of Hyderabad State were involved in 1898 and the Nilgiris in 1903. Hyderabad city became involved in 1911, when the plague situation

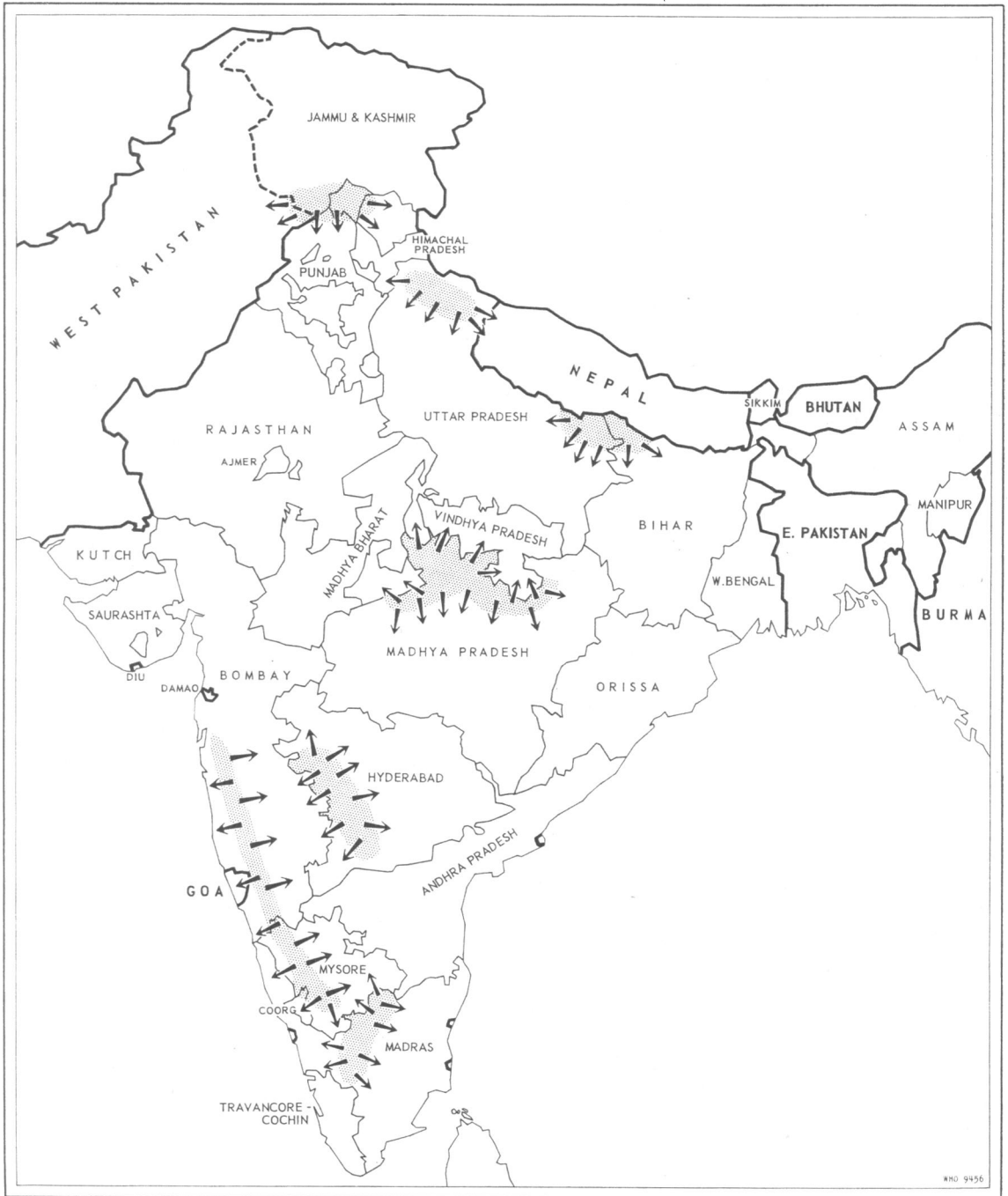
became rather serious. On the other hand, it seems possible that the endemic foci in the Himalayas were of long standing, plague infection being known in the Kumaon and Gharwal districts since 1823. The infection might have persisted there as a relic of the great pestilence of the seventeenth century and might have been responsible for occasional plague outbreaks until 1877. Thereafter, it remained latent for some time, to become active again early in the present century. In central India and Madhya

TABLE 1  
MORTALITY FROM PLAGUE IN INDIA DURING THE PERIOD 1898-1957, ARRANGED IN PERIODS OF 9-11 YEARS

Period	Total deaths from plague	Total population in each period <sup>a</sup>	Specific mortality rate per 100 000	Plague deaths as percentage of total deaths between 1898 and 1957	Average annual percentage of total deaths
1898-1908 (11 years)	6 032 693	3 291 915 990	183.3	47.47	4.32
1909-1918 (10 years)	4 221 529	3 155 926 382	133.8	33.22	3.32
1919-1928 (10 years)	1 702 718	3 283 195 808	51.9	13.40	1.34
1929-1938 (10 years)	422 880	3 619 458 716	11.7	3.33	0.33
1939-1948 (10 years)	268 596	3 965 924 896	6.8	2.11	0.21
1949-1957 (9 years)	59 059	3 287 649 065	1.8	0.46	0.05
<b>Total</b>	<b>12 707 475</b>				

<sup>a</sup> Populations were first calculated for each year by inter-census correction and then added together for different periods. From 1948 onwards, the population of the areas forming Pakistan is excluded.

FIG. 2  
 ENDEMIC PLAGUE FOCI IN INDIA ACCORDING TO SHARIF (1951)



Arrows indicate only the direction of radiation of plague, not the actual course of its progress.

TABLE 2  
ANNUAL DEATHS FROM PLAGUE IN DIFFERENT STATES IN INDIA, 1939-57<sup>a</sup>

Year	Andhra	Assam	West Bengal	Bihar	Bombay	Hydera- bad	Madhya Pradesh	Madras	Mysore	Uttar Pradesh	Vindhya Pradesh <sup>b</sup>	Punjab	Other areas <sup>c</sup>	Total	Rate per 100 000
1939	—	0	0	1 938	1 472	6 758	852	324	2 352	21 662	—	0	9	26 257	6.88
1940	—	0	0	1 040	5 573	7 500	283	1 169	2 593	11 725	—	0	9	19 799	5.13
1941	—	0	0	129	5 311	2 713	761	1 725	5 417	4 035	—	0	22	11 984	3.08
1942	—	0	0	108	680	657	129	701	3 776	8 953	—	0	6	10 577	2.67
1943	—	0	0	266	715	1 498	144	4 885	3 886	7 556	—	1	11	13 578	3.38
1944	—	0	0	834	2 514	5 263	910	1 738	5 357	15 454	—	61	14	21 525	5.29
1945	—	0	0	1 523	11 779	6 631	575	1 644	8 016	14 024	—	203	3	29 751	7.21
1946	—	0	3	8 689	3 405	4 026	189	2 254	2 894	18 206	—	245	6	32 997	7.84
1947	—	0	11	13 204	3 081	1 791	2 902	3 078	1 502	51 455	—	1 905	8	78 937	18.61
1948	—	0	18	2 142	1 305	811	2 860	978	1 128	13 722	—	211	16	23 191	7.02
1949	—	0	57	2 155	1 139	2 103	3 475	151	982	9 875	—	241	19	20 197	5.78
1950	—	0	3	1 449	146	719	5 568	42	255	10 231	196	3	201	18 813	5.33
1951	—	0	8	3	7	98	475	60	542	12 959	14	0	12	14 178	3.97
1952	—	0	16	0	2	1	457	12	272	3 107	18	0	20	3 905	1.08
1953	—	0	0	0	0	0	541	6	56	762	20	0	0	1 385	0.378
1954	18	0	0	0	0	0	0	6	115	157	—	0	0	296	0.08
1955	28	0	0	0	0	0	0	0	137	29	—	0	0	194	0.052
1956	14	2	0	0	0	0	0	1	52	5	—	0	0	74	0.019
1957	0	0 <sup>d</sup>	0	0	0	0	0	1	15	1	—	0	0	17	0.0044

<sup>a</sup> Dashes (—) indicate no record; 0 indicates no death.

<sup>b</sup> No data for 1954 onwards as the State was amalgamated with Madhya Pradesh.

<sup>c</sup> There has been no plague since 1950 in Rajasthan or in Kashmir and Jammu. No plague has been recorded in Orissa

<sup>d</sup> 5 cases were recorded, but no deaths.

Pradesh, plague started after the infection of Bombay in 1896. The endemic foci of Sharif are supposed to be the result of this importation of the infection, but it is difficult to say whether the existence of all these foci can be substantiated by factual data.

The annual plague deaths in different States in India since 1939 are given in Table 2, from which it will be seen that the principal plague-affected states in India since 1939 have been Bihar, Bombay, Hyderabad (now in Andhra State), Madhya Pradesh, Madras, Mysore, Uttar Pradesh and the Punjab. Within this period, West Bengal and Assam have been newly affected, owing in the former to a recrudescence of plague and in the latter to a new invasion.

The main feature to be noted is the gradual reduction in the number of plague deaths in the earlier part of the period, followed by an increase which reached its peak in 1943 in Madras, in 1945 in Bombay and Mysore, in 1947 in Bihar, Madras (second peak), Uttar Pradesh and the Punjab, in 1949 in West Bengal (Calcutta only) and in 1950 in Madhya Pradesh. No plague death has been recorded in the Punjab since 1951; in Bihar since 1952; in West Bengal, Bombay, Vindhya Pradesh and Hyderabad since 1953; in Madhya Pradesh since 1954; and in Andhra Pradesh and Assam since 1957. The last three States in which human plague deaths were recorded up to 1957 are Uttar Pradesh, Mysore and Madras. No plague death has been reported so far in 1958.

#### PLAGUE IN GAUHATI, ASSAM

Except for some cases imported during the exodus from Calcutta in the earlier part of the first plague epidemic in 1902-04, no human or rat plague was ever reported in Assam until 1956. In November of that year epizootic plague was detected for the first time in the town of Gauhati, the gateway of the State. Following this enzootic plague, four human cases with two deaths were recorded (Seal & Bose, 1957). Here then was virgin terrain for the development of the infection, but prompt and extensive application of DDT and prophylactic immunization prevented any epidemic. In the following year, also, several cases (at least five) were reported from another area and from Shillong, the capital of Assam. The only possible clue as to how the infection might have been introduced was that a large consignment of mustard seed in gunny bags had arrived in Gauhati by goods

train from a town in Uttar Pradesh. The first human case, believed to have been unreported, occurred in one of the labourers sleeping inside the godown where the seeds were stacked, and the first known case occurred in the house of the merchant who owned the godown. The transfer of infected fleas or rats through this merchandise was therefore suspected, but no positive evidence could be obtained.

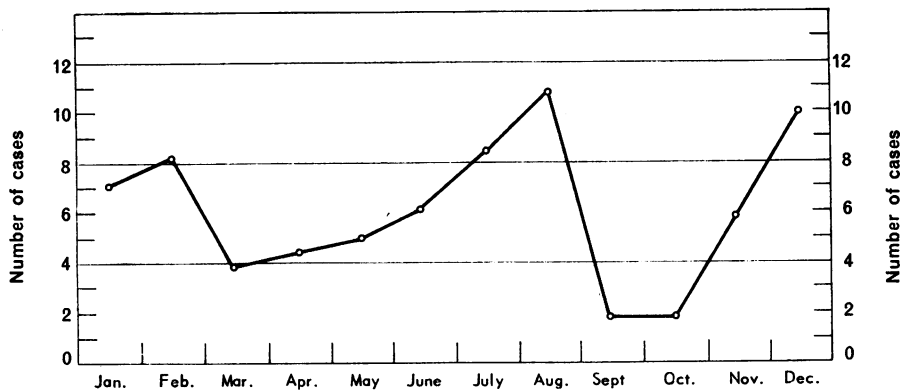
#### CLINICAL FORMS OF PLAGUE IN INDIA

Plague is essentially bubonic in India. True septicaemic plague is rare except for accidental laboratory infections (two such instances are known to the author); some bubonic types have septicaemic manifestations. Primary pneumonic plague is also rare; it generally occurs after lung involvement in a bubonic-septicaemic case leading to plague pneumonia, and subsequent contacts of such cases may develop primary pneumonic plague. Such outbreaks have been known to occur in India (Seal, 1949a; Seal & Prasad, 1949), but they have generally remained confined to a single family or a few families only. The incidence of pneumonic plague in India is generally below 1% and has never exceeded 3% in any year since 1895.

#### URBAN AND RURAL PLAGUE IN INDIA

Plague is both urban and rural in India, the latter predominating. It appears that plague has failed to gain a foothold in many of the towns of India, perhaps owing to untoward climatic conditions and the lack of any efficient flea vector (as in Madras and Assam). Regular heavy annual floods may also be responsible for keeping certain States (e.g., Bengal) free from plague. Another factor which may play an important part is the distribution and proportion of various types of rodents. Given a suitable flea vector, a large proportion of *R. rattus* will make for easier and quicker spread of plague among human beings than a similar proportion of other rodents. On the other hand, replacement of one rodent by others, as in Bombay, may disturb the balance and plague may recrudescence or be imported with consequent severe outbreaks. The development of natural immunity among commensal rats is another factor which may prevent an outbreak or make for simmering enzootic plague. However, given a suitable climate, a sufficiently large rodent population, effective vectors and the plague bacillus, the infection may often become firmly entrenched

FIG. 3  
PNEUMONIC PLAGUE IN CALCUTTA, 1904-07



among the rats of towns or cities and persist there for many years. If such towns or cities are of commercial importance, having traffic connexions with other towns and rural areas, they might spread plague infection and create secondary plague-distributing centres for further propagation of the disease.

The epidemiology of rural plague differs from that of urban plague in one essential respect—namely, that the rural plague may be dependent upon infection of commensal, peridomestic or even wild rodents and is usually initiated by the importation of that infection. The problem of the persistence of plague in rural areas remains a problem for study in India. Kunhardt (1912) suggested the notion of “incomplete” and “complete” plague. But recent observations made by the present author in connexion with city plague suggest that there is every possibility of infection being carried over by the rat population during inter-epidemic periods, possibly independently of the size of the locality or of the rat community. And, indeed, there are villages in the Cumbum valley where plague used to appear year after year. However, not only may rural plague be the result of importation from a plague-affected urban centre, but the contrary may also be equally possible—i.e., plague may spread from an affected rural centre to the towns through grain traffic or by means of other communications, as observed by Pollitzer in China (Pollitzer, 1954). Again, plague may shift from one village to a neighbouring one and can come back again to the first village after two or three years' interval. Plague workers have put forward the notion of area-wide endemicity to explain this phenomenon. According

to Sharif (1951) the slow type of epidemic killing fewer rats persists longer than the severe epidemic causing heavy rat mortality. Baltazard, from his recent observations in Uttar Pradesh,<sup>1</sup> suggests that plague is not endemic there but that it shifts from one place to another through infection of contiguous colonies of field rodents, eventually infecting the commensal rats of a village on its path, thus causing a rat epizootic and human cases. In fact, multiple factors are involved and the same factor may not be equally operative in all places. At least three essential factors must occur together for epizootic plague, which may or may not be followed by human cases, to arise. These are: (1) presence of the plague bacillus in the active or passive reservoirs, (2) susceptible rodents and (3) an efficient vector species. Climate and season also play a secondary role.

#### SEASONAL VARIATION

In bubonic plague the optimum conditions of temperature and humidity are approximately represented by a mean temperature of 68°-77° F (20°-25° C) in association with a relative humidity of the order of 60% and above. (Local studies have, however, shown a slightly higher range). There is a marked decrease in the incidence of plague if the mean temperature rises above 90° F (or about 32° C). The largest numbers of cases are found to occur in years of highest relative humidity. Pneumonic cases are generally interspersed with bubonic, and never exceed 3% of the total. The seasonal incidence of pneumonic cases as seen in Calcutta in the period 1904-07 is shown in Fig. 3. It may

<sup>1</sup> See the article by Baltazard & Bahmanyar on page 169 of this issue.

thus be maintained that the climatic factors, by reason of their influence upon the transmission of infection, are capable of determining the season of the year in which plague epidemics will be most likely to occur. The season influences the numerical importance and longevity of rat fleas and the multiplication of the plague organism in both rats and fleas. The temperature in rat burrows is generally 5°-10° F lower than that of the outside atmosphere, and rats rarely develop septicaemia below 50° F (10° C).

At high latitudes the atmospheric temperature attains the critical level only during the late summer and early autumn, so that it is at this season of the year that a plague epidemic is liable to occur at that latitude. A decrease in latitude is therefore associated with earlier occurrence. In the subtropical region, on the other hand, where either the temperature factor or the humidity factor is unfavourable during the summer, the plague epidemics have a vernal periodicity (as in northern India).

An analysis of the first twenty years' records of plague epidemics in different parts of India since 1896 suggests two principal seasonal manifestations of epidemic intensity in a year. First, there is a broad-based single wave, starting in autumn or late autumn and rising to a peak in March or April. In this category are generally included the provinces of northern India with the Punjab in the north-west and Bengal in the north-east. Bombay city also falls in this category. Secondly, there are double waves which may be further subdivided as follows: (a) a main autumnal wave with a secondary rise in the early months of the year, e.g., Hyderabad and Mysore States; (b) a main spring or late spring wave with secondary rise in the autumn, e.g., Kashmir and North-West Frontier Province; (c) almost equal waves in the autumn and spring, e.g., Bombay Province and State, Central Provinces and central India; and (d) a main peak occurring in the early months of the year with an occasional secondary rise in the autumn, e.g., Madras Province<sup>1</sup> (see Table 3). In brief, the peak of the spring wave is delayed more and more as one moves from the Bombay area towards the north, north-east or any higher altitude, e.g., Kashmir; and also there is a tendency for the double wave to give way to the single one. On the other hand, the peak comes earlier as one moves towards the south or south-east. For instance, in some outbreaks in North-West Frontier

Province the peaks occurred in May or even in June, whereas they occurred in January or February in Madras Province. The two months which show the least incidence in India are June and July, and also January and February where the winter is severe.

In Calcutta, out of 20 epidemic waves since 1896, four reached their peak in March, twelve in April and four in May. In the whole of northern India the common experience is that the maximum plague mortality occurs at the end of the cold season and the disease tends to die out with the onset of the hot weather, because at that time the humidity is inimical to the survival of the rat flea and to its power to transmit infection. The most favourable period of the year is therefore autumn and spring, particularly the latter, when the fleas are seen to come out of the rodents' burrows and great epidemics of bubonic plague are liable to occur.

#### RESISTANCE IN FLEA POPULATION

Comparative pulicidal values of Cyanogas, DDT and BHC have shown that DDT is the most efficient and economical insecticide of the three (Wagle & Seal, 1953) so far as the common Indian species of fleas are concerned. These are *Xenopsylla cheopis*, *X. astia*, *Nosopsylla fasciatus* and *Leptopsylla segnis* (Sharif, 1948). A number of papers reporting the resistance of certain insects to DDT during recent years have been reviewed by Busvine (1957), and Kilpatrick & Fay (1952) have reported resistance in the cat flea, *Ctenocephalides felis*, which dropped markedly or was completely eliminated with the use of 5% chlordane dust. But as reported by Wilson et al. (1957) neither DDT nor chlordane was any longer effective against the fleas in 1956.

#### *Xenopsylla cheopis*

Perhaps the first failure of DDT against *X. cheopis* was reported from Ecuador in 1950 by Vera (1953). Control failures observed in two localities in the fifth year of spray also involved *Pulex irritans*, *Nosopsyllus londinensis*, *N. fasciatus*, *Rhapalopsylla clavicularis* and *Polygenis* sp.

The World Health Organization has received reports since that date of possible resistance developing against DDT from as many as 32 States. These reports led the organization to issue a warning to all countries to watch for the development of resistance in insects including fleas.

Among the various explanations suggested for the development of resistance the following may be

<sup>1</sup> Only a few cases were recorded in the city of Madras during the last pandemic.

TABLE 3  
NUMBER OF PLAGUE OUTBREAKS RECORDED IN ANY MONTH IN THE PERIOD 1898-1918 IN DIFFERENT PARTS OF INDIA

Province or city	Years of study	Months												Months of lowest incidence	Start of rise	Remarks		
		J	F	M	A	M	J	J	A	S	O	N	D					
1. Bombay City	20	—	—	7	13	—	—	—	—	—	—	—	—	—	—	June-July or Nov.-Dec.	Nov.-Dec. or Jan.	During first six years rise was in February; in remaining years generally in January.
2. Bombay Province	21	3	4	11	2	—	—	—	—	2	16	1	—	—	June-July	July-Aug. and Jan.	Double rise: autumn and spring.	
3. Bombay States	20	1	3	11	4	—	—	—	—	3	13	2	—	—	June-July	July-Aug. and Jan.	Double rise: autumn and spring.	
4. Hyderabad States	21	4	6	2	—	—	—	—	—	—	9	3	4	—	June-July	July-Aug. or Jan.	Irregular double rise; fall of peak often continued until June-July or next year.	
5. Mysore States	20	7	2	—	—	—	—	—	3	4	7	4	—	—	May-June	July and Jan.	Generally continuous fall from autumn peak.	
6. Madras Province	20	12	4	—	—	—	—	—	1	4	1	2	1	—	May-July	Aug.-Sept. or Dec.-Jan.	Double rise in 8 instances with main rise in winter.	
7. Central Provinces	20	1	8	5	—	—	—	—	—	1	7	3	2	—	June-July	Aug. or Jan.	Double rise in 10 instances; equal number of peaks in autumn and winter.	
8. Central India	16	1	5	9	—	—	—	—	—	2	11	2	—	—	May-July	Aug. or Jan.	Double rise in all years.	
9. Rajputana States	21	—	1	10	7	—	—	—	—	1	2	1	—	—	June-July	Aug.	Double rise in 4 instances only. Main rise in spring.	
10. Punjab	21	—	—	3	15	3	—	—	—	—	—	—	—	—	July-Aug.	Sept.-Oct.	Peaks only in spring.	
11. Kashmir	18	—	—	2	7	7	—	—	—	—	1	1	1	—	July-Aug.	Oct. or Nov.	Double rise in 3 instances only, but peaks very small. Main rise in the late spring.	
12. North-West Frontier Province	10	—	—	—	1	5	2	—	—	—	—	1	2	—	Jan.-March	March or Oct.-Nov.	Double rise once only with very small peak in December.	
13. United Provinces	19	—	—	18	1	—	—	—	—	—	—	—	—	—	June-July	Sept.	Peak only in early spring.	
14. Bengal (including Bihar)	20	—	—	13	6	1	—	—	—	—	—	—	—	—	June-July or Aug.-Dec.	Aug.-Sept. or Jan.-Feb. (later years)	Peak only in spring.	
15. Calcutta	20	—	—	4	12	4	—	—	—	—	—	—	—	—	July-Oct. or Aug.-Dec.	Nov.-Dec. or Jan.-Feb. (later years)	Peak only in spring.	



TABLE 4  
SUSCEPTIBILITY OF *XENOPSYLLA CHEOPIS* TO DDT,  
GAMMA-BHC AND MALATHION

Date of experiment	Insecticide concentration	Percentage mortality	Median lethal concentration
June-July 1957	DDT	1.0 %	83.3
August 1957		0.5 %	
September 1957	Gamma-BHC	0.1 %	83.3
		0.05 %	
October 1957	Malathion	0.25 %	80.0
November 1957		0.1 %	

mentioned. (1) There may be weak links in the mode of action of the chlorinated synthetic insecticides. (2) The persistent residue of these insecticides may impose a type of selection on the insect population which favours the elimination of the susceptible and the survival of the resistant. (3) The use of insecticides has been increased on an unprecedented scale so that the insect population exposed to selection has been bigger than ever before.

## RÉSUMÉ

La peste semble en voie de régression dans l'Inde. On peut se demander, sans pouvoir répondre, s'il s'agit du prélude à une disparition définitive, comme ce fut le cas pour l'Europe, d'une phase d'un cycle séculaire ou le résultat des mesures de lutte. Le fait est que, durant les années 1898 à 1908, le pourcentage des décès annuels par peste était d'environ 4 par rapport au nombre total des décès, et que ce chiffre s'était abaissé à 0,05 durant la période 1949-57. Il faut relever cependant que la baisse n'a pas été continue, et qu'une brève recrudescence s'est produite en 1945-47. En 1956, un nouveau foyer est apparu en Assam, mais l'infection ne s'y est pas maintenue.

La peste — essentiellement bubonique dans l'Inde — est surtout rurale. Elle semble ne pas s'être établie dans certaines villes de l'Inde, en raison peut-être de conditions climatiques ou de l'absence de la puce vectrice (comme à Madras et en Assam). La peste rurale diffère de la peste urbaine en cela qu'elle peut être entretenue par les rongeurs commensaux, péri-domestiques et même sauvages, tandis que, dans les villes, ce sont les rats commensaux et les cas importés qui jouent le principal rôle.

In India no actual resistance of the flea population has been reported. Recently, however, after a recrudescence of plague in certain villages in Mysore the flea population seems to have increased considerably in spite of DDT (Y. K. Subrahmanyam—personal communication, 1958). The Arthropod-borne Diseases Sub-Committee of the Indian Council of Medical Research proposed in 1957 that the question of resistance of rat fleas in India should be investigated. Accordingly Sen in collaboration with the present author made some preliminary studies using the Busvine and Nash technique.

Table 4 summarizes the results of six replicates (eight specimens each) at different doses of DDT, gamma-BHC, and Malathion. The test insect was *X. cheopis*, the local vector species. These experiments show that the rat flea, *X. cheopis*, as bred in the laboratory from local strains is fully susceptible to DDT, BHC and malathion used at the proper doses. It now remains to be seen whether fleas collected from the field where anti-flea work has been going on for some time would behave in the same way as those bred in the laboratory. It may prove necessary to alter the present strategy of attack against these insects in the near future by using newer insecticides.

La période de fréquence maximum paraît en relation avec la latitude et la saison. La fréquence décroît lorsque la température moyenne s'élève à environ 32° C, et les cas les plus nombreux ont été observés dans les années où l'humidité relative est la plus élevée. L'influence de la saison est plutôt en rapport avec la densité et la longévité des puces du rat, et la multiplication du bacille pesteux soit chez le rat, soit chez la puce. La vague automnale est de plus en plus retardée à mesure que l'on avance de Bombay vers le nord et le nord-est, et, à l'altitude du Cachemire, la double vague — vernale et automnale — tend à se fondre en une vague unique. D'autre part, le clocher de la courbe de fréquence se trouve plus tôt dans l'année à mesure que l'on se dirige vers le sud ou le sud-est. La maladie cède en général au début de la saison chaude.

Aucun cas spécifique de résistance des puces au DDT n'a été démontré dans l'Inde, bien que cet insecticide y soit utilisé depuis plusieurs années. Toutefois, la récente recrudescence de la peste dans l'Etat de Mysore a été mise en relation avec une éventuelle résistance du vecteur, aussi y a-t-il lieu d'envisager pour l'avenir l'emploi d'autres méthodes de lutte contre les puces, ou celui d'autres insecticides.

## REFERENCES

- Busvine, J. R. (1957) *Trans. roy. Soc. trop. Med. Hyg.*, **51**, 11
- Busvine, J. R. & Nash, R. (1953) *Bull. ent. Res.*, **44**, 371
- Greval, S. D. S. (1948) *Indian med. J.*, **83**, 137
- Kaul, P. M. (1949) *Epidem. vital Statist. Rep.*, **2**, 142
- Kilpatrick, J. W. & Fay, R. W. (1952) *J. econ. Ent.*, **45**, 284
- Kunhardt, J. C. G. (1912) In: *Proceedings of the Second All-India Sanitary Conference . . . 1912*, Simla, vol. 3, p. 48
- Pollitzer, R. (1954) *Plague*, Geneva (*World Health Organization : Monograph Series*, No. 22)
- Sharif, M. (1948) In: *Report of the Haffkine Institute for the years 1944-46*, Bombay, p. 64
- Sharif, M. (1951) *Bull. Wld Hlth Org.*, **4**, 75
- Seal, S. C. (1949a) *Calcutta med. J.*, **46**, 167
- Seal, S. C. (1949b) *Indian med. Gaz.*, **84**, 162
- Seal, S. C. & Bose, P. N. (1957) *Indian J. publ. Hlth*, **1**, 119
- Seal, S. C. & Prasad, G. (1949) *Indian med. Gaz.*, **84**, 408
- Sen, P. (1958) *Bull. trop. Med. Calcutta*, **6**, 14
- Vera, C. S. (1953) *Bull. Wld Hlth Org.*, **9**, 615
- Wagle, P. M. & Seal, S. C. (1953) *Bull. Wld Hlth Org.*, **9**, 597
- Wilson, H. G., Keller, J. C. & Smith, C. N. (1957) *J. econ. Ent.*, **50**, 365