

XXIV. GENERAL CONSIDERATIONS REGARDING THE
SPREAD OF INFECTION, INFECTIVITY OF HOUSES,
ETC. IN BOMBAY CITY AND ISLAND.

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I. INTRODUCTION.

At this point the question arises: Do modes of spread, other than by means of the epizootics, exist which aid in the diffusion of the infection during epidemic prevalence of the disease—in other words, is the epidemic wholly or only partially dependent upon the epizootics?

In the following pages we shall attempt to supply an answer to this question. Before entering into details we may for the sake of clearness indicate the principal points about to be discussed.

In the first place, it is necessary to examine the statement, that the epidemic is to an appreciable extent due to infection acquired by direct contact with patients suffering from the disease. This statement involves the assumption, that the plague patient is not infrequently a source of danger to others on account of the infectivity of his excreta. Closely connected with this is the question of the infectivity of houses and the nature of the infecting agent within them. It is conceivable, and indeed it has been suggested, that the infectivity of houses is referable to contamination of the soil (*e.g.* cowdung floors) with *B. pestis* derived from the excreta of plague-infected rats or men. Again, it is conceivable that the infection resides for a time in articles, *e.g.* bedding and clothing, which have been soiled by the excreta of patients suffering from plague.

Lastly, there is the view that the infection in houses is present in an effective form in the bodies of rat fleas.

It will be noted that these problems have reference to the spread of the infection within houses. Not less important, however, is the problem of the transportation of infection to a distance in clothing or in merchandise. The question of the importation of infection into a hitherto uninfected locality altogether hinges upon the possibility of such a mode of spread.

In conclusion, we shall give the results of our experience in the matter of the occurrence of plague in domestic and other animals (excepting rat plague) and shall discuss, briefly, the significance of animal plague on the spread of the epidemic.

II. THE SPREAD OF THE INFECTION WITHIN HOUSES.

(1) *The question of the spread of infection by direct contact with a suffering case.*

In this connection it is our intention to consider the alleged spread of infection from patients suffering from the bubonic and septicaemic varieties of plague. The contagiousness of pneumonic plague¹ and its mode of transmission have never been disputed, but on account of its rarity, this type of the disease plays only a minor part in the spread of the epidemic. Our evidence with regard to the question at issue is derived from various sources and may be arranged as follows :

(a) *Experience in hospitals.*

When plague broke out in Bombay ten years ago it was not long before it was recognised that the attendants in plague hospitals remained singularly free from danger of infection, although they were brought frequently into intimate contact with patients in the acute stage of the disease. This is the more remarkable, when it is borne in mind that in the majority of fatal cases there is incontinence of urine and faeces, and that the hospital patients and the menial staff who attend them do not possess an elementary notion of the most ordinary precautions.

Our own experience amply bears out the view that the wards of a plague hospital are devoid of infectivity. Our visits to the Maratha

¹ We are aware of the possibility that cases of primary pneumonic plague may originate from septicaemic cases with secondary pneumonia, but we have no observations on the point.

plague hospital in the course of certain investigations connected with our work strongly impressed on us the truth of the dictum, that one of the safest places during the epidemic is the ward—the “acute ward” we might add—of a plague hospital. In addition certain experimental observations we have made go far, in our opinion, to prove that a plague hospital ward is devoid of infectivity and that the excreta of patients suffering from plague are not, as a matter of fact, infective from the point of view of epidemic plague.

Three modes of experimentation were adopted.

First, two guinea-pigs were introduced into the “acute” ward of the hospital in the epidemic season and were allowed to run freely about the ward. The guinea-pig, as is abundantly evident from the numerous experiments already related, is markedly susceptible to the natural infection of plague, and yet these animals, although kept in the ward for one week, remained perfectly healthy. No rat fleas were caught on them. The second experiment constituted a still more severe test. 15 guinea-pigs were confined in a flea proof godown in the laboratory compound, and bedding, recently soiled by the excreta of acute cases just before death, was added daily, each lot of bedding being kept in the godown for 24 hours. Although the experiment was continued for several weeks, and in spite of the intimate contact with this material in a confined space, none of the guinea-pigs contracted the disease. The third method was to rub the urine or faeces of acute plague cases into a scarified area on the abdomen of guinea-pigs. A considerable number of experiments were carried out but only one doubtful success (by rubbing in faeces) was obtained.

(b) The influence of imported cases on the spread of the epidemic.

By an imported case we mean a patient who has acquired infection in a place, *e.g.* at his work, other than the house in which he is found suffering. We would also wish it to be understood in this connection, that there is no evidence of rat mortality due to plague in the house in which the patient is found. If surveillance were kept on the relatives and attendants of such a case, it should be possible to discover whether any plague cases followed amongst the contacts. It must be admitted that the careless habits of the people, to which allusion has been made, offer abundant opportunities for direct transference of infection by contact, were such a method of infection an effective one. We have no evidence, however, that this method of infection, which we can merely conceive of as occurring, is ever effective in Bombay, and we

shall now proceed to support this conclusion by relating our experience of a number of imported cases in the outlying villages. The cases were personally investigated by members of the Commission.

In Parel village 28 cases of plague occurred in the epidemic under review. These cases are especially appropriate to our present purpose, since the epizootic in the village was a very limited one, and since the available evidence shows, that of the total cases 17 fall into the group of imported cases. In reviewing the entire series of 28 cases, it is noteworthy that only in four instances did two or more cases occur in the same house. In all the houses in this village in which plague cases occurred a number of individuals were living in the same room as the sick, but in no instance did we obtain any evidence to show that the sick communicated the disease to their attendants and friends.

In Wadhala village we inquired into two cases of plague which were imported from the City. One of the patients was brought to the village during his illness, while the other was attacked with the disease on the day after arrival. There was no evidence of rat mortality in either of the buildings in which the cases were found. Guinea-pigs placed in each of the houses remained healthy, and no cases occurred amongst the persons who were living in either of the houses.

Worli village furnished similar examples (*vide* detailed description of Worli).

(c) *Occurrence of single and multiple cases in houses and buildings.*

If plague is an infectious disease, it would certainly happen that multiple cases in a house would be common. It is obvious, then, that an investigation of the relative frequency of single and multiple cases in houses might throw light on the question of the spread of infection by contact with a suffering case.

Our own experience has been, that it is comparatively rare to find two or more cases in a house. Our attention has been specially directed to the point, because throughout the epidemics of 1906 and 1907 we made a continual endeavour to find instances of this kind with the purpose of using such houses for guinea-pig experiments.

In addition we have analysed a large mass of data relating to this question drawn from the records of the epidemics 1903—1906, inclusive. Before calling attention to the results of this analysis, we may explain that the data were abstracted from records kept by the District Registrars. Each District Registrar has a street register, which contains a list of the street numbers of every inhabited *building* in the sections

under his charge. When he is informed of a case of plague in a building (as well as of certain other diseases), a note is made of the fact in the register opposite the corresponding building number, the date of notification being also recorded. No data are, however, available in these street registers for showing the number of cases which occur in a *house* in any of the buildings. From these records we have abstracted and analysed the data relating to plague for ten of the sections in the City for four successive years.

We may now draw attention to the figures of principal interest in the table which gives the results for the year 1906 (*vide* Table I). The columns showing the average number of inhabitants per building and the average number of houses per inhabited building are important, since they give an indication of the average size of the buildings in each section. It will be observed that the yearly records have been subdivided for our purpose into two half yearly periods, January to June, and July to December. This division practically corresponds to the plague season and the off-plague season in Bombay. Attention is directed to the column which shows the average number of cases per building for each section. The columns in the table towards the right hand are important since they give the percentages of buildings in which single and multiple cases occurred in each half year.

The results which come out of a study of the tables as a whole are striking. It will be noted, in the first place, that the average number of cases per building in the epidemic months for all the sections given in the table is very low, never indeed rising to three per building.

This low average of cases per building, when considered in the light of the fact that the buildings on the whole have a large population distributed as separate families in houses within the buildings, is without doubt good evidence that the average number of cases *per house* must be a very low figure. It is only necessary to add that the columns showing the percentages of buildings which yielded single and multiple cases confirm this view.

Taken as a whole the evidence afforded by the tables seems to us to accord completely with that already adduced.

Since it cannot be questioned that multiple cases in families do occasionally occur, it becomes necessary to furnish an explanation of the source of infection of such cases.

It will readily be understood that, even if no spread of infection by contact occurs, one might still expect to meet with multiple cases if the plague rat and the rat flea be regarded as the common source of

TABLE I. 1906.

Section	No. of occupied buildings	Population	Average inhabitants per building	Average no. of tenements per inhabited building	Total no. of buildings of which yielded plague cases				Average no. of plague cases per building		No. and percentage of buildings which yielded														
					January to June	July to December	January to June	July to December	January to June	July to December	1 case	2 cases	3 cases	4 cases	5 to 10 cases	11 cases and over									
Dongri	535	32,663	61	9.1	435	32	197	24	2.2	1.3	49.7	83.3	15.7	8.3	2	31	2	35	1	14	1	16	1	3	1.5
Umarkhadi	1,041	53,610	51	11.2	516	36	276	29	1.8	1.2	58.7	86.2	23.2	6.9	2	64	2	19	1	14	1	17	—	—	—
Kumbharwada	642	32,784	51	—	354	3	235	3	1.5	1.0	71.0	100.0	18.3	—	43	3	14	—	5.9	—	3	—	8	—	—
Khara Talao	575	26,935	47	9.6	363	8	171	7	2.0	1.1	59.6	100.0	21.6	—	37	7	—	8	—	9	—	14	—	1	—
Mandvi	915	38,158	42	9.3	521	44	264	33	2.0	1.3	57.6	69.7	20.8	—	55	8	—	23	1	15	1	18	—	1	—
Market	902	35,305	39	8.0	407	20	244	18	1.7	1.1	63.9	94.4	22.9	—	56	17	—	15	1	9	—	8	—	—	—
Fansaewadi	802	29,240	36	3.9	460	17	185	14	2.5	1.2	49.7	78.6	24.3	21.4	41	3	—	21	—	7	—	21	—	3	—
Chakla	812	29,362	36	7.5	312	7	206	7	1.5	1.0	66.0	100.0	20.9	—	43	—	—	19	—	7	—	1	—	—	—
Bhuleshwar	1,123	28,129	34	6.8	635	25	347	20	1.8	1.2	57.6	75.0	22.8	20.0	79	4	—	34	1	16	—	18	—	—	—
Dhobi Talao	1,126	36,594	33	5.2	567	10	277	8	2.0	1.2	56.0	87.5	20.9	—	58	—	—	28	1	14	—	17	—	5	—
Khetwadi	1,255	33,579	27	4.5	247	10	145	10	1.7	1.0	70.3	100.0	11.7	—	102	10	—	12	—	6	—	8	—	—	—

TABLE II.

Serial No.	Address	Dates of attack of cases	History of dead rats	Remarks
1	60, Bellasis Road	4 plague cases between 8/3/07 and 12/3/07	2 dead rats about 5/3/07	On 16/3/07, 7 rat fleas got on 1 guinea-pig.
2	159, Queen's Road	6 plague cases between 12/2/07 and 19/2/07	Many dead rats before cases	On 20/2/07, 11 rat fleas got: 3 of these contained plague bacilli in stomach contents.
3	11-13, Kombhal Lane	3 plague cases all on 25/3/06	Dead rat in room. Date?	White rat in cage unprotected from fleas died of plague.
4	85, Bunganga	(a) 3 plague cases all on 5/4/06 (b) 2 " " both on 5/4/06	(a) None (b) Dead rats on 4/4 and 10/4/06	(a) Nil. (b) 25 rat fleas got on guinea-pig; this guinea-pig died of plague.
5	4, Kalachawki Rd	4 plague cases—2 on 13/4/06 2 on 14/4/06	Dead rats found; date?	72 rat fleas got on 2 guinea-pigs both of which died of plague.
6	22, Navroji Hill North	(a) 3 plague cases—2 on 14/4/06 1 on 15/4/06 (b) 3 " " 14/4, 15/4, 16/4/06	(a) Dead rats in adjoining room; date? (b) Dead rats in adjoining room	(a) 8 rat fleas got on 2 guinea-pigs. (b) 47 rat fleas got on 2 guinea-pigs.
7	1, Ripon Road	4 plague cases, 15/4, 2 on 16/4, 1 on 17/4/06	Dead rat; date?	170 rat fleas got on 2 guinea-pigs, one of which died of plague.
8	Jubilee Mills Sewri (cotton godown)	5 plague cases all about 17/3/07	Several dead rats before cases	On 25/3/07, 6 rat fleas got on 2 guinea-pigs, one of which died of plague.
9	96, Cavel (infection in kitchen)	Cooks { 1st case, 17/3/06, left day of attack 2nd case, 19/3/06, removed to hospital Cook's assistant, 3rd case, 12/3/06, removed same day Child in house, 4th case, 20/3/06 Assistant in kitchen, 5th case, 21/3/06	None	106 fleas got on 2 guinea-pigs, one of which died of plague.

infection. We think that such cases are to be explained in this way. In confirmation of this view it may be noted, that in our experience the evidence regarding rat mortality was much stronger in multiple cases than in single cases in a room. Another important point is, that as a general rule the persons in the multiple cases were attacked almost simultaneously, as if by a common infecting agent. In order to illustrate these points we have brought together in Table II the essential details of a series of multiple cases which occurred in a number of badly infected houses. From a study of these cases, in the light of our knowledge of the incubation period of the disease, it is evident, that all are aptly explained on the view of a common source of infection: indeed in some this is the only possible explanation. Moreover, in nearly every instance a history of dead rats was obtained and in several instances the houses were proved to be infective, the infectivity being associated with the presence of rat fleas in unusual numbers within them.

(d) *The question of the transmission of infection from a septicaemic human case to the attendants of such a case by the agency of the human flea.*

Experimenting with human fleas (*P. irritans*) we obtained three successful transmissions out of 38 experiments, *the fleas having previously been fed on selected septicaemic rats* (vol. VII. p. 413). We have also shown that multiplication of plague bacilli takes place in the stomach of the human flea. Taking into consideration the evidence relating to the spread of infection by direct contact adduced above, and further taking into account the slight septicaemia as observed microscopically and by cultural methods in human cases compared with that in rats (vol. VI. pp. 521, 527), we think that transmission of infection from man to man by means of the human flea is probably a very infrequent occurrence.

We may add that there is in our view still less reason to believe that infection is transmitted by infected human fleas to rats in houses, because we know that this species of flea is very particular in its choice of host, that it does not live well upon the rat, and that it will only attack this animal in the absence of its proper host.

- (e) *The question as to whether an epizootic amongst rats in houses is alone sufficient to account for a widespread dissemination of infection throughout a locality.*

It is our experience that a widespread dissemination of infection in houses may result from an epizootic in the rats, even when conveyance of infection by direct contact with sick occupants of the houses is rigidly excluded. This conclusion is based upon a study of plague infection in an evacuated village, namely, Sion Koliwada (*vide* report on this village). In this village an experiment was carried out, which showed that a large proportion (at least 45%) of the total buildings became infective in consequence of an epizootic amongst the house rats. The possibility that infection by direct contact with sick animals played a part in the spread of the infection was definitely excluded by their isolation in the houses.

- (f) *Conclusion derived from a consideration of the evidence which has been brought forward on the question under discussion.*

A review of the whole of the evidence bearing upon the question at issue leads us to conclude, that contact with plague cases, although a conceivable mode of spread of infection, yet, as a matter of fact, plays no part in the spread of the epidemic.

- (2) *The question of the infectivity of houses.*

A. *General considerations.*

Most investigators are agreed that the infection of plague is characteristically present in buildings, in other words, that plague is a place infection. Thus, it has been an oft-repeated observation, especially in India, that healthy persons, who have not otherwise been exposed to infection, have contracted plague after visiting houses vacated because of the disease. Again, it is well known that the evacuation of an infected village by its inhabitants and their removal to a temporary camp, if only a short distance away, is one of the best measures for checking an outbreak of plague. A case of this kind happened in Sion Koliwada village. When plague broke out in this village, almost all the inhabitants voluntarily vacated their houses and went to live in a camp of rude huts only about 200 yards distant. One or two cases of plague occurred after the people were in camp, but these we had reason to attribute to infection received during a visit to the vacated houses for domestic purposes.

B. *The infectivity of houses.*

(a) *The nature of the infecting agent in houses.*

The conclusion that infection by direct contact with a suffering case plays no part in the epidemic spread of the disease, that is, that the excreta of a plague patient have little or no infective properties, is a very important one, because it simplifies the problem of the infectivity of houses and the nature of the infecting agent within them. It is evident that, having come to this conclusion, no importance can be attached to contamination of the soil by such excreta. For, if excreta in a fresh state on clothing and bedding possess no infective properties, it would appear very improbable, that the transference of bacilli from this source after being deposited on, *e.g.* a cowdung floor, would prove effective. From similar considerations, and taking into account the relatively small bulk of rat excreta compared with human excreta, we conclude, further, that contamination of any part of a house or its furnishings by the excreta of rats plays no part in the spread of the epidemic. Apart from reasoning of this kind certain experiments carried out by us in infected houses strongly support the view that the infectivity of houses cannot be referred to soil, contaminated by infection in the form of excreta, either of men or of rats, deposited casually on any part of floors. We refer to experiments, in which susceptible animals—guinea-pigs, white rats, monkeys—were confined in cages of special construction in which the animals were protected from possible contamination of the soil. Moreover certain experiments in which guinea-pigs were confined in wire cages appeared to us to be more frequently successful, than they could possibly have been if the infection, assuming it to be effective, had been thus casually deposited.

The arguments adduced above force us, thus, to seek for the source of the infectivity of houses in some intermediate agent, which is capable of conveying the infection from rats to man. Our view is that this intermediary is the rat flea and that the infectivity of houses is due to the presence within them of infected rat fleas. It is unnecessary in this place to enter fully into the evidence for this conclusion, but we may refer the reader to certain papers which have already been published dealing with experiments in plague houses.

*Infectivity of Houses**(b) Circumstances which may be accepted as evidence of infectivity in houses.*

Definite proof of infectivity is forthcoming, when one or more of the following results are obtained :

(1) The discovery of one or more dead plague-infected rats in any part of a house.

(2) The death from plague of a guinea-pig allowed to run free in a house.

(3) The capture of plague-infected fleas in a house, that is to say, the demonstration of abundant bacilli microscopically indistinguishable from *B. pestis* in the stomach contents of fleas caught on guinea-pigs which are allowed to run free in a house.

(4) The death from plague of a previously healthy guinea-pig in a flea proof cage, to which animal fleas caught on a dead rat or on guinea-pigs allowed to run free in a house had been transferred.

Presumptive evidence of infectivity rests upon (1) the occurrence of one or several plague cases in a house, and (2) a definite history of mortality amongst rats in the house, especially during the plague season. The evidence is strengthened when multiple cases occur in a house associated with a history of rat mortality. We may add that the capture of a large number of rat fleas, in our experience roughly from 20 to 200, on guinea-pigs placed in suspected houses affords a presumption of infectivity. It must be kept in mind, however, that the number of fleas caught in houses proved to contain infection depends largely upon whether the guinea-pig is put into the house shortly after the death of the rats or at a later period. The largest numbers are obtained in the former case.

Again, even if there is no history of dead rats in a house, the discovery of plague rats or even a history of dead rats in the building, in which the house is situated, must be held to be matter of evidence in an inquiry into the infectivity of houses in buildings. Although necessarily of less value as evidence the occurrence of plague rats in the adjoining gully or in the vicinity of a building must also be considered as carrying weight in a similar inquiry. It must be noted that in this statement we are referring to the conditions which obtain in Bombay.

(c) Certain features of the infectivity of houses.

It is noteworthy that the infection in houses is frequently localised to a part, sometimes even a very small part, of a house and that the

infection, as one might expect, varies in "concentration" in different houses.

As to the first point, it has been our experience that the infection may be confined to a single room in a house consisting of several rooms. We may cite the case of a severe outbreak (five cases) in a family living in 96 Cavel (see Table II). In this house the infection was confined to the kitchen. No fewer than 106 fleas were got on two guinea-pigs placed in this room, one of the animals subsequently dying of plague.

In a paper on the life history and habits of fleas it will be pointed out that plague sick rats frequently harbour fleas in unusual numbers, and that such rats in their wanderings are apt to leave a trail of infected fleas behind them. Fleas, if dropped under these circumstances in the living room of a house, might easily prove a source of danger to man. The danger is, however, much greater in the immediate neighbourhood of a rat dead of plague, because here the infection is in a concentrated form on account of the larger number of fleas which remain at or near the spot.

The statement that the degree of infectivity of houses is proportional to the number of rats which die of plague in them would seem to require no proof.

(d) Duration of infectivity of houses.

The duration of infectivity of houses is probably very variable, depending as it does on the persistence of the infection amongst the rats. In one instance which came under our notice the interval between the discovery of the first and the last plague rat was as long as 13 days.

It has also to be kept in mind that houses are liable to reinfection by rats when the epizootic is very widespread, as in Bombay.

It has been alleged that the infection may persist in a house or in a locality apart from rats. The underlying idea in this belief appears to be that the *B. pestis* is able to live for long periods, in soil for example, and that the infection may continue in a latent form in this medium, until the next plague season when it breaks out afresh in a virulent form. Associated with this highly speculative assumption is the idea that the conditions in certain houses are especially favourable for the persistence of infection, so that these houses are attacked with plague year after year. It seems to us that statements of this kind are entirely without value, unless supported by systematic and long continued observations of the course of the infection amongst the rats. Further, the whole of our experience in Bombay is opposed to the view that the

infection persists for any length of time in a house or locality apart from infection amongst the rats. A number of houses which were plague infected during the epidemic of 1906, were experimentally investigated at regular intervals during a period of one year but no evidence of persistence of infection was forthcoming in any of them, nor did plague occur in them during the subsequent epidemic season.

C. *The infectivity of buildings.*

In Table III figures are set forth which show the incidence of plague on persons living on the various floors of buildings for the whole of Bombay. It will be noted that the population living on the various floors is expressed as percentages of the total population and that similarly the plague cases investigated by us have been grouped according to their occurrence on different floors, the resulting numbers being also expressed as percentages of the total number of plague cases. Comparison of the percentage figures for each floor makes it evident that they correspond in a remarkable manner. Similar figures have been calculated for 21 of the sections and generally speaking they confirm the accuracy of the results for the whole of Bombay. We think it, therefore, justifiable to make the general statement that the incidence of plague on persons living on different floors of buildings is the same.

Further, it has been our experience that when infection is present in several houses in a building the incidence of plague on these houses appears to have followed no definite course. This irregularity is readily explicable from the point of view of the epizootic. Apart from infection amongst the rats it would seem impossible to explain why one house in a building should be infected rather than another since the conditions within the houses are, as a rule, identical.

III. THE TRANSPORTATION OF INFECTION TO A DISTANCE.

(1) *Transportation of infection in clothing or merchandise.*

If we can exclude modes of spread of the infection of plague other than the rat flea, we must conclude that the transportation of infection to a distance is attributable solely to the conveyance of the infection in the rat flea. A little reflection suffices to show that transportation of infection in this medium is not only conceivable, but that under certain circumstances it may be a very likely contingency.

TABLE III.

Incidence of plague on persons living on ground floor, 1st floor, etc.

	Ground floor		1st floor		2nd floor		3rd floor		4th floor		5th floor		6th floor		7th floor		Total
	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	
Bombay																	
Population	351,429	48.9	189,066	26.3	107,827	15.0	50,786	7.0	16,382	2.3	2958	0.4	184	0.02	18	0.003	718,650
Plague cases	4,879	49.2	2,637	26.6	1,369	13.8	714	7.2	188	1.8	28	0.3	5	0.05	83	0.8	9,898

TABLE IV.

Incidence of plague on buildings classified according to number of storeys.

	Buildings with ground floor only		Buildings with ground floor and 1 storey		Buildings with ground floor and 2 storeys		Buildings with ground floor and 3 storeys		Buildings with ground floor and 4 storeys		Buildings with ground floor and 5 storeys		Buildings with ground floor and 6 storeys		Buildings with ground floor and 7 storeys		Numbers on which calculated	
	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total	Number	P.c. on total		
Bombay																		
Total Buildings	20,608	53.6	7,147	18.4	5,149	13.3	3,341	8.6	1,904	4.9	618	1.6	74	0.2	2	0.05	38,843	
Buildings plague infected	1,212	19.0	1,174	18.4	1,416	22.2	1,452	22.7	796	12.5	276	4.3	54	0.8	—	—	6,380	

(a) Transportation in merchandise, grain, etc.

In an account of the bionomics of the rat flea to be published later various modes of dispersal of fleas are indicated. Two of these have a bearing upon the subject under discussion. They are: (1) dispersal of fleas with the host, when the latter is carried in merchandise, and (2) dispersal by means of merchandise, grain, clothing, etc., the host, however, not being transferred with the fleas.

With regard to the first point, we may note that we have seen rats dive, as it were, into bags containing grain, so that the bags could be moved without any evidence of the presence of rats within them.

It is further obvious that merchandise and grain, which have been visited by rats, may have rat fleas (possibly infected fleas) deposited in them, so that these fleas might be transferred to distant places. Examination on one occasion of bran, which was kept in a bin with a loosely fitting lid in a rat infested room, revealed the presence of numerous rat fleas in the bran. In this connection it must be noted that adult fleas in the absence of any host to feed upon rapidly die, generally in five days.

(b) Transportation of rat fleas in clothing.

Infected fleas may be transported in this way: (a) in the clothes of a person who has been for a time in a plague-infected house, and (b) in bundles of clothing or bedding removed from an infected house.

(a) A reference to a previous paper ("A note on man as a host of *P. cheopis*," vol. VII. p. 472) shows how readily and in what large numbers rat fleas may under certain circumstances come on to man. The experiments cited in this paper indicate that rat fleas may often be transported in this way from place to place, especially from plague-infected houses, where they are more likely to take to man because of the absence of their true host. During our visits to infected houses in Bombay City we had many opportunities of noting that we carried away fleas on our persons and on our clothing. These fleas were generally human fleas, but occasionally they proved to be *P. cheopis*.

(b) The following experiments which were carried out at the end of the epidemic of 1906 are of interest:

Bundles of clothing, bedding, etc. were sent to the laboratory from houses in the City in which plague cases had occurred. The bundles after being opened out were kept in a flea proof godown for several days, being replaced by fresh ones as they arrived. Along with the

clothing guinea-pigs were placed in the godown. In some instances the animals were allowed to run free, in other cases they were placed in pairs in cages, the control animal being protected from fleas either by means of a layer of tanglefoot or by a curtain of wire gauze.

In all 26 free guinea-pigs were exposed in the godown for an average period of about four days each. The result of the experiment was that three fleas (two rat fleas and one human flea) were caught on the animals and that one of the free guinea-pigs died of plague, the bubo being in the neck. On the tanglefoot of one of the cages three fleas were caught. The stomach contents of one of these fleas, a human one, contained abundant bacilli indistinguishable from *B. pestis*.

It would appear that the guinea-pig which died of plague was infected by fleas, because we proved in a similar experiment that clothing and bedding recently soiled with the excreta of plague cases possessed no infective properties. The experiment therefore shows:

- (1) that rat fleas may be transported to a distance in bedding and clothing removed from plague houses; and
- (2) that such rat fleas may prove infective if transferred to a susceptible animal in the place to which they are carried.

It ought to be added that these experiments were carried out at an unfavourable time, namely, towards the end of the epidemic, so that they give no indication of the frequency with which rat fleas may be transported in clothing, during the period of the epidemic when these insects are especially numerous.

(2) *Importation of infection into a hitherto uninfected locality.*

From the discussion of the transportation of infection to a distance we are led naturally to consider the question of the importation of infection into a hitherto uninfected locality.

In the first place, we would point out that in whatever way rat fleas are transported, whether in clothing or merchandise, they will select, when carried to their new surroundings, either their true host, *i.e.* the rat, or the next best available animal. If then infected fleas are imported into a house they will by preference attack the rat rather than the human occupants of the house. It is apparent from the account we have given of the rat infestation of houses in Bombay, that under such circumstances opportunities for transference of infected rat fleas to rats in houses are abundant. It would appear, then, that the introduction of infected rat fleas into a hitherto uninfected locality may lead to

serious consequences by giving rise to an epizootic amongst the rats. We may note, (1) that many chances render uncertain the effective transference of infection to rats by importation, (2) that the most favourable time for such infection to act effectively is when the conditions for epizootic prevalence are most favourable, namely, during the period of the rise of the epizootic, and (3) that in Bombay infection carried in this manner probably affects *M. rattus* more often than *M. decumanus*, since *M. rattus* is the species most intimately associated with man. As an example of the uncertainty of importation by infected fleas we may cite several cases which were investigated in Parel village. Seven rooms, in which nine imported cases were found, were tested by means of guinea-pigs. In one of these rooms a guinea-pig died of plague. From the available evidence we concluded that this guinea-pig was infected by rat fleas imported to the village from Bombay by the patient.

In Bombay excellent opportunities are afforded of observing the importation of infection by human agency in the case of the outlying villages, Sion, Wadhala, and Worli, which were specially investigated by us. These villages were indeed selected for study for the reasons that they occupied isolated positions and that their inhabitants followed an employment (as fishermen or agriculturists) which kept them for the most part confined to their villages. For these reasons it was considered that it might prove a comparatively easy matter to narrow down the inquiry into the origin of the epizootic and epidemic and to trace the infection, if imported, to a portion of Bombay City which was at the time infected. In our view the outbreaks of plague in these villages are due to a chance importation of infection from the City. Our reasons for so thinking are as follows:

First a systematic and extensive examination of the rats in the villages failed to reveal plague either in an acute¹ or in a chronic form, amongst these animals during the off-plague season.

Secondly, our own observations during two years and a study of the history of the outbreaks in previous years clearly show that the incidence of plague in these villages is extremely erratic, both in regard to time and place, as if due to a chance importation. Thus, one part or indeed the whole of a village may be badly infected one year but may escape altogether in the following year.

A complete account of the observations made in the villages is

¹ See p. 842.

given elsewhere. We may refer to four cases of imported infection, which illustrate the point under discussion, namely :

(a) *The origin of the epizootic in Sion Koliwada village in the plague season of 1906.*

(b) *The first plague-infected rat in Wadhala village.*

(c) *The case of Jankibai in Worli village.*

(d) *Case I in Parel village.*

In conclusion, we would point out that the carrier of the infection may not contract the disease, as the Sion and Wadhala cases show. It is interesting to compare this fact with instances, in which a guinea-pig allowed to run free in a house escaped infection, although the fleas taken on it, when transferred to a guinea-pig in the laboratory, killed the latter with plague.

IV. THE QUESTION OF THE OCCURRENCE OF PLAGUE IN DOMESTIC AND OTHER ANIMALS (EXCEPT RATS).

We have already noted that when rat fleas are starved they will readily attack any animal which is available to feed upon. From this consideration and from our observations in Bombay it would appear that the occurrence of plague in animals other than rats is to be explained solely by transference of infection from the rat to these animals by means of the rat flea.

We are of opinion that animal plague is of little or of no importance, if only for the reason that instances of this kind occur very seldom, at least in Bombay.

We have observed natural plague in guinea-pigs, rabbits and monkeys.

Liston described an epizootic of plague amongst guinea-pigs in Bombay which occurred in 1903, and again in 1905. These epizootics were associated with a history of dead rats, and rat fleas were found on the guinea-pigs. In 1906, an epizootic of plague broke out amongst a stock of guinea-pigs and rabbits in the laboratory. Plague-infected rats were found by us in the runs, and rat fleas were taken on the animals.

Only one suspicious case of plague in a cat has come to our notice. This animal had a purulent bubo in the neck, but no growth of *B. pestis* was obtained from the pus.

Epidemic amongst the monkeys in Victoria Gardens.

This epidemic occurred in the zoological and botanical gardens in the City.

The monkey house is built of stone with a high masonry plinth. The floor throughout is of patent stone and is quite impermeable to rats. There is a central passage with four cages on each side, in which the monkeys are confined. The cages are separated from one another by solid walls and are closed in in front by iron bars. For purposes of description they are numbered in the diagram, I—VIII.

The history of the epidemic is as follows:

On 11/4/06 two dead rats, which had been found that morning in the monkey house, were sent to the laboratory for examination and proved to be plague infected.

On visiting the gardens the same evening we ascertained that one of the rats had been found in cage I and the other in cage VI. The occupants of the different cages as we found them at this time are given in the table accompanying the diagram. It is to be noted that the monkey which inhabited cage I had changed places with the monkeys from cage II, and that the lemurs from cage VI had been removed to cage VII, the langurs from the latter cage taking their place.

The first plague death amongst the monkeys occurred on 17/4/06, when a black ape was found dead in cage IV. This was soon followed by the death of a Bonnet monkey in cage I on 19/4/06, a langur in cage VI on 20/4/06, another black ape in cage IV on 21/4/06, and a pig-tailed monkey in cage V on 27/4/06 (*vide* diagram).

It will be seen that the rats were found six days before the death of the first monkey and 16 days before the death of the last. Two more plague rats were found at a later date, namely, on 8/5/06, one in the passage outside cage I and the other in cage II on 10/5/06. Cage II was at this time inhabited by a single monkey, a crab-eating monkey, which did not contract the disease. It is noteworthy that in this small epidemic four species of monkeys became infected, viz.

- (1) Bonnet monkey (*Macacus sinicus*),
- (2) Black ape (*Cynopithecus niger*),
- (3) Langur (*Semnopithecus entellus*),
- (4) Pig-tailed monkey (*Macacus nemestrinus*).

It only remains to add that fowls, ducks, pigeons, goats, sheep, oxen, buffaloes and horses are common animals in Bombay, but that in no

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single instance have we observed natural plague in any of them, nor do our observations lead us even to suspect that these animals play any part in the spread of the epidemic.

Diagram of Monkey House and Table showing inhabitants.

V 0 27/4/06	+ 11/4/06 VI 0 20/4/06*	VII	VIII
Pig-tailed Monkey		*Langur	
2 Black Apes			+ 8/5/06
0 17/4/06 IV 0 21/4/06	III	II + 10/5/06	I 0 19/4/06* + 11/4/06

Bonnet Monkey

+ Plague-infected Rat.
0 Plague-infected Monkey.

Inhabitants of cages on 11/4/06.

- Cage I. 6 Bonnet Monkeys, *Macacus sinicus*, transferred from Cage II on 11/4/06.
- Cage II. 1 Ourang-utang, *Simia satyrus*, transferred from Cage I on 11/4/06. Segregated on 19/4/06.
- Cage III. 3 Baboons, *Cynocephalus hamadryus*.
- Cage IV. 6 Black Apes, *Cynopithecus niger*.
- Cage V. 1 Bonnet Monkey, *Macacus sinicus*. 2 Pig-tailed Monkeys, *Macacus nemestrinus*.
- Cage VI. 3 Langurs. *Semnopithecus entellus*, transferred from Cage VII on 11/4/06.
- Cage VII. 4 Lemurs, *Lemur macaco*, transferred from Cage VI on 11/4/06.
- Cage VIII. 4 Malbronck Monkeys, *Cercopithecus cynosurus*.

V. SUMMARY AND CONCLUSIONS.

(1) The question of the alleged spread of infection by direct contact with a suffering case has been discussed. Our observations in a plague hospital and with material obtained from this hospital lead us to conclude that such a mode of spread does not exist. Support is given to this view by a consideration of the influence of imported cases on the spread of the epidemic and by an investigation of the relative frequency of single and multiple cases in houses and buildings. We have, further, referred to our experience that a rat epizootic is alone sufficient to account for a widespread dissemination of infection throughout a

locality. A review of the whole of the evidence on this point brings us to the conclusion that contact with plague cases plays no part in the spread of the epidemic.

(2) In discussing the question of the infectivity of houses, evidence has been brought forward which points to the rat flea being the transmitting agent of infection from rat to man. Further, reasons have been given for the view that plague does not persist in a locality apart from infection amongst the rats.

(3) From arguments brought forward in the discussion of the two previous questions we conclude that the epidemic is wholly dependent upon the epizootics.

(4) It has been shown that infection may be transported to a distance by means of rat fleas in clothing or merchandise and that such infection, when imported into a hitherto uninfected locality, may give rise to an epizootic in the rats.

(5) Our observations lead us to conclude that plague in domestic animals in Bombay either does not occur or occurs so seldom that it cannot be said to possess any significance from an epidemiological standpoint.