THE RELATIONSHIP OF FIELD RODENTS TO PLAGUE IN KENYA

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1. Introduction

Periodic fluctuations in the populations of field and domestic rodents are well-known phenomena in Kenya, and maximum years of abundance are followed by heavy mortality among the species concerned. These fluctuations have not, so far, shown definite periodicities, but follow years of heavy rainfall or seasons giving good growing conditions for crops.

Since 1930, attention has been paid to rodent communities and particularly to outbreaks of disease among them, with the object of finding out whether true plague was the cause of mortality among field rats following years of maximum abundance. Further, should plague be found to be the cause of mortality in field rodents, it was essential to know whether their fleas would bite man and be capable of transmitting the disease.

During this period, human plague has not ceased to occur each year in Kenya, with the years 1930–1 and 1935–6 marking epidemic periods. Also, within the last six years, two major outbreaks of epizootic disease occurred among field rodents and were investigated, together with numerous field rodents found dead at various periods.

2. Recent outbreaks of disease among field rodents

(*a*) *Outbreak of epizootic disease among* Arvicanthis *at Naivasha*

During 1930, reports were received from several districts situated in the Rift Valley that large numbers of field rodents had been found dead. Coincident with this outbreak among rats was another among sheep from some obscure cause. At first, the two outbreaks were not considered to have any connexion, the mortality among rats being accepted as plague. Several of these dead rats, which were all *Arvicanthis abyssinicus*, were examined for plague at the Laboratory, Nairobi, and were pronounced to be bacteriologically negative.

The Veterinary Research Officers, who were studying the outbreak of disease among sheep, found it was being caused by a new virus, which has been described by Daubney & Hudson (1931) under the title "Enzootic Hepatitis" or Rift Valley fever. Owing to the uncertainty in regard to the vector of this

new disease among sheep and the source of infection, coupled with the fact that the outbreak among sheep and rats appeared to be coincident, the rodent population was investigated. On the first indication of rodent mortality in 1931, a survey of rats and fleas was started and carried through until no further deaths among rats were recorded from the area. The work included taking blood smears from various organs in rats and submission of organs for histological examination and inoculation tests. The trapping of rodents was carried out both in the field and in the huts of labourers employed on farms where mortality was heaviest both in sheep and rodents.

The bacteriological examination of large numbers of rodents, histological examination of tissues and transmission experiments all proved negative. The possibility of a virus infection was also excluded, and the results of the investigation show that the cause of death among *Arvicanthis* had no connexion whatsoever with the outbreak of Rift Valley fever among sheep at the same period. Also, in view of the failure to obtain a virus from these rats, smears and various organs were submitted to Dr de Smidt, Bacteriologist, for examination. He failed to recognize any pathogenic organism likely to be the cause of death.

A. abyssinicus is the dominant field rat in the district, but whether excessive numbers or other causes operate to produce an apparent high mortality in this species remains obscure. Both sexes are equally affected and there is no abnormal infestation of fleas or other ectoparasites. During periods when there is a shortage of rain and the grass is dried up, there is a noticeable absence of field rats, but as soon as sufficient rain falls and fresh grass springs up, most of the commoner field rats are to be observed in numbers. During dry periods they exist mainly on the bark and roots of trees and shrubs. If the rains are prolonged, as they were in 1930 and 1931, the grass cover is very dense, field rodents increase rapidly, breeding rates being quite different from years of normal rainfall. Litter follows litter more frequently and survival rates are higher. These were the conditions during the period under review.

In September 1931, 1 ♂ *Otomys* was found dead in a trap, no fleas were taken. In October, 19 ♂♂, 20 ♀♀ *Arvicanthis* were taken dead in traps; 1 ♀ *Otomys*, from which 2 ♂♂ *Ctenophthalmus cabirus* were taken; and 1 ♀ *Rhabdomys pumilio* with no fleas. The individual *Arvicanthis* from which fleas were obtained are listed below:

	D. lypusus		*X. cheopis*		*C. cabirus*		*X. brasiliensis*	
Arvicanthis	♂	♀	♂	♀	♂	♀	♂	♀
♂ 9. x. 31	3	6	16	10	—	—	—	—
♀ 9. x. 31	—	1	—	1	—	—	—	—
♂ 9. x. 31	—	2	—	—	—	—	—	—
♀ 10. x. 31	—	4	—	1	—	—	—	—
♂ 12 x. 31	1	1	7	10	1	2	1	—
♀ 27. x. 31	—	1	—	—	—	—	—	—
♀ 30. x. 31	1	—	—	—	—	1	—	—

During November, 1 ♀ *Rhabdomys* died in a trap and had no fleas. 9 ♂♂, 12 ♀♀ *Arvicanthis* were also found dead, with the following fleas:

Arvicanthis	*D. lypusus* ♂	*D. lypusus* ♀	*X. cheopis* ♂	*X. cheopis* ♀	*C. cabirus* ♂	*C. cabirus* ♀	*X. brasiliensis* ♂	*X. brasiliensis* ♀
♂ 3. xi. 31	—	1	—	—	—	3	—	—
♂ 13. xi. 31	—	—	—	—	—	—	1	—
♂ 18. xi. 31	—	—	—	1	—	—	—	—
♂ 18. xi. 31	2	1	—	—	—	—	—	—
♀ 19. xi. 31	—	—	1	—	—	—	—	—
♂ 24. xi. 31	—	—	—	—	—	1	—	—

From a total of 277 rodents trapped, 64 had entered traps and died almost immediately, giving a percentage mortality rate among trapped rats of 23%. *Arvicanthis* were also found dead in large numbers at all parts of the farms.

The following table gives the total number of specimens of each species trapped and the flea infestations for the period of the survey:

	Total no.	No. infested	*D. lypusus*	*C. cabirus*	*X. cheopis*	*X. brasiliensis*
R. rattus	17	7	3	1	29	—
Arvicanthis	222	111	249	108	210	25
Rhabdomys	19	11	13	31	2	1
Otomys	13	7	26	12	2	—
M. coucha	1	—	—	—	—	—
Lophuromys	1	1	4	5	—	—
Soricidae	2	—	—	—	—	—
Lemniscomys	2	2	—	2	1	1
Totals	277	139	295	159	244	27

The average number of fleas per rat was 2·8 and the average for the principal species of fleas, *D. lypusus* 1·0, *C. cabirus* 0·5 and *X. cheopis* 0·9. The percentage infestation was 50·7.

The suspected vector of Rift Valley fever among sheep is the *Mansonia* (*Taeniorhynchus*) group of culicines. Precipitin tests carried out on members of this group from Naivasha are given below and are mainly of interest to show that culicines do occasionally obtain a meal from rats:

	Total no. tested	Mosquitoes found positive to blood of: Human	Rat	Sheep	Ox
M. fuscopennatus	213	7	—	—	1 (?)
M. microannulatus	240	19	1	7	1 (Human and ox)
M. versicolor	42	—	—	—	10

(*b*) *Outbreak of disease at Nairobi among* Otomys

On 15 January 1931, a report was received to say that large numbers of rats had been found dead in the Hill area, Nairobi. On investigating this occurrence, it was found that only *Otomys* were involved. Large numbers of this species were found dead and the behaviour of the remainder was similar to that observed in plague-infected house rats, an unsteady gait and a desire to climb impossible objects, or wandering about in a lethargic state. No trapping, baiting or other mechanical means were necessary to collect several hundred rats, which were moving freely over the grounds. Africans armed with

sticks followed behind the moving mass and clubbed the stupified animals. It was not possible to count the actual numbers of rats, but it can be safely estimated that between five and six hundred were killed in one day.

The phenomenon was confined to one small area. One hundred and twenty-one rats were submitted for bacteriological examination and were pronounced negative. A small recurrence was noted on 29 January. Eight rats from this outbreak were examined with similar negative bacteriological findings.

The behaviour and abnormal death rate among this species were quite exceptional, although during certain seasons they do enter traps and die suddenly from some unknown cause. Bacteriological examination has failed to establish an infecting organism and more recent work has shown that virus infection can also be excluded.

The macroscopic signs at autopsy were heavily necrosed livers, slight enlargement of the spleen, congested suprarenal glands, and the general appearance of septicaemia.

Mortality among rodents from unknown causes is a well-known phenomenon throughout the world and gives rise to epizootics when the population is at the height of reproductivity and numerical strength. A parallel exists in this instance with *Otomys*. This species is never present in very great numbers, its density fluctuating with rainfall and grass growth. During very dry years it is very difficult to obtain specimens. January 1931 was noteworthy for its heavy cover of grass over the country after the heavy rainfall of sixty inches in 1930, and *Otomys* increased rapidly everywhere.

(*c*) *Other reported outbreaks of disease in field rodents*

It has been extremely difficult to obtain accurate information concerning outbreaks of rodent mortality, but two epizootics have been reported from competent and reliable observers. In both instances, field rodents alone were involved.

Both epizootics were reported to occur in the Rift Valley. Both observers identified *Arvicanthis* as the rodent concerned. The first outbreak occurred outside Menengai station in 1933, in the old reputed plague area of 1927, when *Arvicanthis* was accused of being responsible for the outbreak of true plague. Twelve rats from this outbreak were submitted to bacteriological examination and returned negative. No prophylactic inoculations were carried out in the human population in the area and no cases of the disease occurred. An estimate of 4000 dead rats was made for the collection.

The other outbreak occurred in 1935 between Eburru and Gilgil, in close proximity to a labour concentration camp. An estimate of 1500 dead rats was made for this collection. None were submitted to bacteriological examination. No prophylactic measures were undertaken in the labour camp and no human cases of plague occurred.

3. The health of rodent communities

There are apparent differences in types of lesion, disease and mortality rates, in different rodent communities, such as those which have their normal habitat in buildings and those whose habitat is confined to the field, respectively.

The normal health of house rats is well maintained, judging purely from the absence of positive findings in routine blood smears and cultures, excepting during periods when *Bacillus pestis* becomes epizootic. Naked-eye observations for lesions in the organs of *Rattus* have been carried out daily for long periods but suggestive signs are absent. *Rattus* captured in the Nairobi area are, however, heavily infested with the larval stages of *Taenia taeniaformis* (Bat.) and *Taenia tenuicollis* (Rud.), the infection being as high as 90% in captured rats. The larvae of these species are large and lie in a sac of epithelium attached to the liver, being conspicuous objects at post-mortem examinations. The role of these parasites in lowering the host's resistance to infection cannot be ignored, owing to the abnormal rate of infection. The worms are probably derived from domestic cats, large numbers of which are kept in African and Asian houses to control the rat population.

The normal health of rodents in field communities can also be regarded as well maintained, judging by the absence of known endoparasites, although outbreaks of disease peculiar to themselves have been found to occur as reported, for *Otomys* in Nairobi and *Arvicanthis* at Naivasha.

There is, however, a more or less regular and typical diseased condition affecting field rats which generally assumes a form of severe mottling of the liver and kidney, accompanied by distinct colour changes, such affected rodents being recognizable at a glance on dissection. Macroscopically, the condition is obvious, but microscopical blood examination and cultures from organs have all proved to be negative. Histological study of mottled organs does show that a severe necrosis takes place. The rats in which the condition is found are generally those that enter traps overnight and are collected dead in the morning. The condition has also been noted in those rats whose vigour or liveliness is below normal.

The diseases of wild rodents, forming a check upon numbers, have been viewed generally as being of a periodic nature associated with overcrowding in the population. This view is partially substantiated by the mortality of field rats at Nairobi and Naivasha, but, so far, no definite periodicity can be given to field rodent mortality rates for Kenya. On the other hand, the diseased condition peculiar to field rodents remains more or less a constant factor, cases occurring throughout the year. There seems to be no form resembling an epizootic except in isolated instances. Plague among house rats in endemic areas and the obscure disease among field rodents appear to be analogous and of an enzootic nature.

As most of the known bacterial organisms causing rodent diseases can be ruled out and other conditions likely to set up this diseased condition appear to be absent, it remains that a virus may prove to be responsible. To test this possibility, some experiments have been carried out with extracts from mottled organs. The result of examinations and transmission tests carried out by the Veterinary Laboratory Staff with material obtained from the Naivasha outbreak among *Arvicanthis* had already proved negative.

The lesions observed in rodents at autopsy are very constant in respect to the mottling of organs, although colour changes show considerable variation with the advancement of the condition.

The *liver* is the organ most frequently noted to be mottled and as showing the greatest changes, especially in coloration. There is usually no enlargement of the organ. The condition of the liver approximates closely to that described for animals which have died of a virus infection, particularly Rift Valley fever: (*a*) dark red spots on darker red background; (*b*) dark red spots on salmon pink background; (*c*) light red spots on buff colour; (*d*) liver becomes entirely buff coloured and very friable, with irregular very light areas; and (*e*) a liver apparently normal in colour will show a marked honeycomb appearance on close examination.

The honeycomb appearance is apparently the primary condition and the buff coloured with irregular red areas the advanced stage. In rodents which have died in traps there is often found a severe necrosis of the whole liver and so far this has not been observed to be a focal process. When the liver is cut, the apparent surface lesions are found to extend throughout the whole substance of the organ.

Spleen. Only slight enlargement of the spleen has been noted to accompany the changes in the liver. Splenic enlargement has been noted in several instances but it does not appear to bear any relation to the diseased condition under discussion. The presence of haemorrhages beneath the capsule has been noted.

Kidney. This organ presents features similar to the condition observed in the liver. A honeycomb mottling is of frequent occurrence and very distinct. Colour changes show considerable variation, even to buff colour. The suprarenals are frequently pinkish and congestion takes place.

Lymphatic system. The mesenteric lymphatic glands are generally enlarged and haemorrhages occur. The supra-inguinal glands are invariably enlarged with haemorrhages present and acute congestion of vessels.

Generative organs. The testicles present a very congested appearance but no changes have been observed in the female reproductive organs.

Circulatory system. Haemorrhages have been found in the lung, the pericardium and over the left ventricle of the heart. Subpericardial haemorrhages are frequent. Extreme congestion is usual in most vessels.

Central nervous system. Congestion of the meningeal vessels and sometimes of the cerebral vessels.

Alimentary system. No naked-eye lesions visible in the alimentary tract and no congestion in the intestines.

Rapid decomposition of carcases is a notable feature of the condition. The principal changes noted in post-mortem examinations of affected rodents are mainly concerned with colour variations in the liver and kidney.

Experiment 1.

7. i. 32. *Arvicanthis* with congested suprarenals and showing pitting and mottling of liver in its early stages. Post-mortem appearances very suggestive of a diseased condition. The liver, spleen, kidneys and suprarenals kept in 50% glycerine and *N* saline in refrigerator for 24 hr.

8. i. 32. Two guinea-pigs each inoculated intraperitoneally with 1 c.c. of 50% glycerine extract and two pigs each inoculated intraperitoneally with 1 c.c. saline extract.

25. ii. 32. Animals killed. No lesions or changes observed at autopsy.

Experiment 2.

7. i. 32. *Arvicanthis* with liver in advanced stage of necrosis, buff yellow colour, slight irregular patches. Spleen normal, suprarenals normal. Same extractions and inoculations as in Exp. 1 carried out.

25. ii. 32. Animals killed. No lesions or changes observed at autopsy.

Experiment 3.

8. i. 32. *Mastomys coucha* with congestion in subcutaneous tissues, supra-inguinal lymphatic glands enlarged and heavily congested. Liver, spleen and kidney apparently normal. Suprarenals deep pink and large area haemorrhaged. Tissues pulped in *N* saline and 33% glycerine and two guinea-pigs inoculated intraperitoneally, also part kept in ice-chest for 24 hr. and inoculated into two white rats on following day.

25. ii. 32. Animals killed. No lesions or changes observed.

Experiment 4.

9. i. 32. *M. coucha* with lesions similar to those described in previous experiment (3) and similar inoculations carried out in one pig and one white rat.

25. ii. 32. Animals killed. No changes or lesions noted.

Experiment 5.

9. i. 32. *M. coucha* with slightly enlarged spleen. Liver slightly mottled and very friable. Superficial inguinal glands enlarged and congested and subcutaneous vessels also heavily congested. Suprarenals pink and congested. Extracts made in 33% glycerine and *N* saline and inoculated intraperitoneally into white rat and guinea-pig.

25. ii. 32. Animals killed. Negative.

Experiment 6.

13. i. 32. Male *M. coucha* heavily infested with fleas, following being taken: 8 ♂♂, 5 ♀♀ *X. cheopis*, 2 ♂♂, 1 ♀ *C. cabirus* and 25 ♂♂, 21 ♀♀ *D. lypusus*. There was also a maggot of *Cordylobia anthropophaga* taken from a hindleg. The supra-inguinal glands were enlarged and congested, subcutaneous veins heavily congested. Spleen slightly enlarged. Liver deeply mottled and salmon pink colour. Testicles with blood vessels heavily congested. The spleen was ground up in 1 c.c. *N* saline and inoculated intraperitoneally into a guinea-pig. Part of the liver was ground up in 1·5 c.c. *N* saline and the other part in 0·5% sodium citrate solution and inoculated into pigs.

26. ii. 32. Animals killed. Negative.

Experiment 7.

16. i. 32. *M. coucha* with 7 ♂♂, 2 ♀♀ *X. brasiliensis* and 2 ♂♂, 2 ♀♀ *L. segnis*. The liver was very friable, colour ranging from salmon to yellow buff, post-mortem appearance suggestive of very advanced stage of the disease. The subcutaneous vessels were congested and supra-inguinal glands enlarged and haemorrhaged. Spleen normal. Kidneys honeycombed and deep salmon colour, almost buff; supra-renals pinkish and vessels congested. Lungs very pale and a large haemorrhage present. No abnormal quantity of pericardial fluid. Part of the liver was ground up in 1·5 c.c. *N* saline and 0·5% sodium citrate solution respectively and inoculated intraperitoneally into two white rats.

9. iii. 32. The rat injected with 1·5 c.c. saline extract found dead. Subcutaneous vessels congested. Suprarenals red, kidneys enlarged and dark. Spleen subnormal. Liver with slight necrotic foci over surface. Cultures from organs on plain and blood agar were sterile. Histological examination of organs negative except for necrosis of liver. Cause of death, septicaemia.

The cause of this diseased condition in rodents is obscure. Neither bacteriological, protozoal or histological examinations have proved effective in giving any clue to the problem. Assuming that a virus infection is present, it is certainly indicative that two different sets of workers and materials have failed to transmit the disease, and such a source seems to be ruled out.

The pathological condition observed in field rodents, giving rise to a fatal septicaemia, occurs mainly after heavy rainfall periods and during vegetative abundance following such periods.

4. DISCUSSION

It has been extremely difficult to reconcile statements made in regard to plague in South African field rodents with what is to be observed in Kenya, particularly since the fauna of both areas is very similar. Identical rodent and flea species, guilty in the South, are guiltless in the rest of Africa. The field rodent communities of East Africa are equally in close contact with highly endemic areas and from historical evidence (Roberts, 1935) have been so for much longer periods than South Africa. There is also an interchange of fleas between them and domestic rats.

The species of rodents most commonly mentioned as having an association with plague in South Africa are *Rhabdomys pumilio*, *Arvicanthis abyssinicus* and *Mastomys coucha*. In hyperendemic areas in Kenya, these species are usually extremely rare and sometimes even absent.

The replies to the "Questionnaire on Plague in Africa and the role of wild and domestic rodents in its propagation", issued by the Office Internationale d'Hygiène Publique for 1934, throws considerable light on the distribution of plague among rodents. The extract in the *Tropical Diseases Bulletin* states that: "It is interesting to note that the only country reporting permanent signs of plague infection among wild rodents is the Union of South Africa, but that it is without sign of any important extension."

Authoritative bacteriological examination has not yet confirmed the statement that field rodents in any outbreak in East Africa have been killed by plague. Dr de Smidt, Bacteriologist, Kenya, who has carried out an immense amount of work on plague for many years, has not yet seen a field rat naturally infected with the disease. During 1930–1, when large numbers of field rats were dying in and around Nairobi, he examined a total of 2750 field rats of the commonest species, *M. coucha*, *Arvicanthis* and *Otomys*, with negative results (*Kenya Ann. Med. Rep.* 1931).

The species of fleas which feed on man and thus could possibly transmit the disease is of prime importance in the study of the epidemiology of the disease. It is to be feared there has been a strong tendency in the past to rely on theories based on insufficient evidence. The flea, *Dinopsyllus lypusus*, may be taken as an example. Symes & Hopkins (1932) and Symes (1932) declared this flea to be a known plague carrier and possibly the initiator of outbreaks in Kenya. They based their statements on the work of Mitchell, Pirie & Ingram (1927) in South Africa, who, in one experiment, induced *D. lypusus* to feed on human blood, and in another experiment, successfully transmitted plague from rodent to rodent. From this, *D. lypusus* was regarded as the vector of "field" plague to labourers working on farms at Nakuru in 1927. That the species of rodents from which the flea was taken were never confirmed as having suffered from plague does not appear to have been taken into account, nor was any further confirmation given to show under what circumstances this flea fed voluntarily on man.

Two important facts upon which reliance can be placed in the interpretation of the role of field rodents in the incidence of plague are (*a*) the number of field rodents which are found suffering from the disease, and (*b*) the power of transmitting the disease to healthy rodents through fleas taken from field rats during epizootics.

In regard to (*a*) above, Dr de Smidt's figures furnish a reply, and for (*b*) the following facts are suggestive. During an outbreak of plague in Nairobi in 1930–1, live fleas were collected from rats caught in huts and the surrounding area and were transferred to healthy rats in attempts at transmission. Plague was transmitted by *X. cheopis* and *X. brasiliensis* obtained from *Rattus* during the epizootic. From field rats in the same area, also suffering from an epizootic disease, 469 *C. cabirus* and 816 *D. lypusus* were collected and tested for transmission of plague to healthy rats. Every experiment failed.

The isolated and sporadic nature of plague in Kenya offers unique opportunities for the study of the disease, as the fundamental causes of each outbreak are not obscured by epidemics nor is there spread from an infected focus.

Types of housing and the general hygiene of endemic areas play no part in the origin or propagation of the disease. They are entirely dependent on individual domestic habits, in so far as any person neglectful in regard to food, particularly its storage and wastage, offers an unlimited supply of food to a

dense house-rat population. It is this high house-rat density that is the constant factor in endemic areas.

Rat harbourage facilities, which are often quoted in connexion with high rat densities, are of little importance in the causation of the disease. In the presence of an easily accessible food supply rats will find somewhere to harbour and in its absence the best harbourage offers no attraction.

5. SUMMARY

1. A study of the literature of East Africa on the incidence of plague in field rodents shows that in no single instance has evidence been offered to prove they have any relation to the disease.

2. Recent epizootics in field rats in Kenya are described.

3. The health of house and field rats is discussed, with an account of the work carried out on a septicaemic condition present in field rats.

4. The results of bacteriological examination of 2750 field rats and attempted transmission experiments with 1285 field fleas, both investigations conducted during a period when there was heavy mortality among them, failed to establish the presence of *B. pestis*.

At the same time, mortality among the *Rattus* population, proved bacteriologically to have been caused by *B. pestis*, enabled successful transmissions to be accomplished with fleas from that species.

5. In the absence of any confirmatory bacteriological evidence to prove that epizootics among field rats are caused by *B. pestis*, and the failure to find natural infection among field fleas, together with the knowledge of their aversion to feed on man and lack of opportunity to develop such tastes, the theories that field rodents and their fleas "are definitely assisting in the perpetuation and spread of plague" or that they "are possible factors in initial outbreaks of plague" (Thornton, 1930) appear to be fallacious.

6. Plague in Kenya is distinguished by its isolated and sporadic nature, even in endemic areas, and it is not dependent on types of housing or the general hygiene of inhabited areas, but on individual domestic habits. Any individual, neglectful in regard to food, particularly its storage and wastage, is the person responsible for the disease, by offering an unlimited supply of food to a dense rat population and encouraging its survival.

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