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Observations on the epidemiology of Rift Valley fever in Kenya

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SUMMARY

The epizootic range of Rift Valley fever in Kenya is defined from the results of virus isolations during epizootics, and from an extensive serological survey of cattle which were exposed during an epizootic. A study of the sera from a wide range of wild bovidae sampled immediately after the epizootic, showed that they did not act as reservoir or amplifying hosts for RVF.

Virus isolation attempts from a variety of rodents proved negative. Rift Valley fever did not persist between epizootics by producing symptomless abortions in cattle in areas within its epizootic range. A sentinel herd sampled annually after an epizootic in 1968 revealed not one single seroconversion from 1969 to 1974. Certain forest and forest edge situations were postulated as enzootic for Rift Valley fever, and a small percentage of seroconversions were detected in cattle in these areas, born four years after the last epizootic. This has been the only evidence for the persistence of the virus in Kenya since 1968, and may be a part of the inter-epizootic maintenance cycle for Rift Valley fever in Kenya, which otherwise remains unknown.

INTRODUCTION

Rift Valley fever (RVF) was first described by Daubney, Hudson & Garnham in 1931. They had investigated an outbreak of disease in sheep in the Rift Valley in Kenya characterized by a heavy mortality amongst lambs and abortions in ewes. Later outbreaks showed that calves and pregnant cattle could be similarly affected, and that humans handling infected animals are easily infected and suffer influenza-like symptoms. They showed that a filterable virus was the cause and suggested that this was probably transmitted by a mosquito vector. Smithburn, Haddow & Gillet (1948) and Williams, Woodall, Corbet & Haddow (1960) isolated the virus from various mosquito species in Uganda. More recently vector studies have been made by McIntosh (1972) and McIntosh, Jupp, Anderson & Dickinson (1973) in South Africa. No vector studies have been made in Kenya.

Findlay (1931) described the susceptibility to RVF of a range of animals: primates, ruminants and rodents proved more susceptible than the others. Easterday (1965) made studies on the pathogenicity of the virus.

Epizootics involving large numbers of cattle and sheep have occurred in Kenya at irregular intervals (Scott, Weddel & Reid, 1956). The epizootics were associated with periods of greater than average rainfall (Daubney *et al.* 1931; Scott *et al.* 1956). Epidemiological data were collected by Scott *et al.* (1956) during the years

1952–4, years in which RVF occurred in many parts of the country. The last major outbreak was in 1968, and since then no virus isolations have been made from domestic animals, nor has there been any evidence of activity of RVF in any part of Kenya, other than a single case in a human (Metselaar *et al.* 1974).

In Kenya or elsewhere, there is no information on the fate of the virus in the inter-epizootic periods. The reservoir for the virus is unknown, and the physical location where the virus persists during these periods. The possibility that a forest or forest edge cycle exists involving one or more vertebrates other than domestic ruminants would seem to be high. Observations made on rodents (Mims, 1956; Weinbren & Mason, 1957; Scott & Heisch, 1959; Henderson *et al.* 1972) would seem to indicate that they are not involved. Davies examined baboon sera (*Papio anubis*) collected in Kenya in 1968–9 and was unable to find any antibody in wild caught animals. The present study was initiated to define the areas of Kenya which were affected by RVF during the last epizootic, to assess the role, if any, of wild ruminants in its natural history and to try to define the natural habitat of the virus in the inter-epizootic periods. This was done by mapping virus isolation results from blood and organs of animals, by observation of sentinel animals, and by serological tests on several groups of serum samples.

MATERIALS AND METHODS

Ecological zones

The results of the observations made from 1968 to 1974 have been related to ecological zones as classified by Pratt, Greenway & Gwynne (1966). This classification is based on characteristics of climate, vegetation and potential for agricultural use and is thus especially useful for the purpose. The principal features of the zones of the classification are as follows:

Zone II. Climate humid to dry sub-humid, moisture index not less than -10. Vegetation forests and derived grasslands and bushlands.

Zone III. Climate dry sub-humid to semi-arid, moisture indices -10 to -30. Vegetation moist woodland/bushland/wooded grassland, trees typically broad leaved *Combretum*.

Zone IV. Climate semi-arid, moisture indices -30 to -40. Vegetation dry woodland and wooded or bushed grassland, trees typically *Acacia*.

Zone V. Climate arid, moisture indices -40 to -50. Vegetation dry woodland/bushland, trees typically *Commiphora* and *Acacia*.

Zone VI. Climate very arid, moisture indices -50 to -60. Vegetation dwarf shrub grassland or dry bushed grassland, including barren land.

Field material

Blood with anticoagulant, liver or spleen from animals that died in the field were submitted by field veterinary officers to the laboratory. Fetuses were similarly submitted for virus isolation and also the spleens from rodents purposely collected for RVF virus isolation.

The departmental records of virus isolations in other epizootics before 1968 were also used.

Sentinel herd

Cattle of a large dairy farm in an area most severely affected in the 1968 epizootic were used as a sentinel herd. A group of 65 animals were bled in 1969 and each year 30–40 animals of a new and younger age group were brought into the survey and bled at yearly intervals. From 1970 onwards paired sera were collected from all cases of abortion occurring in this herd.

Wild rodents

These were tested for the presence of virus. The animals were trapped near the site of the 1931 outbreak of RVF. Traps were inspected daily after setting and rodents were stored at $-20°$ C. until they could be dissected at the laboratory. The spleens were taken and stored at $-70°$ C. in species pools until they were processed.

Serum collections

Apart from the sentinel herd serum samples, the following groups of sera were used for antibody testing.

(*a*) Sera collected by members of the Kenya Veterinary Department and submitted to the Veterinary Laboratories for a whole variety of purposes. A deliberate bias to obtain positive sera was made by taking those specimens that were collected during and after the widespread epizootic of RVF in 1968. This approach was made to define as far as possible the boundaries of the epizootic areas.

(*b*) A random sample of sera received from aborted cattle. These sera mostly come from the higher potential grade cattle or their crosses kept on farms where the management standards are high. These farms are situated in the ecological zones II and III of highest production potential, often consisting of grassland derived from forest. The samples represent a useful countrywide monitoring system for outbreaks of RVF, of which the principal sign may be symptomless abortion (Scott *et al.* 1956). When positive sera were detected, further serum samples were obtained in an attempt to demonstrate rising titres to RVF virus.

(*c*) When monitoring of sera of the sentinel herd and sera from aborted animals was competely negative for RVF for some time, it was postulated that there could be enzootic foci within the epizootic areas. Four such areas were defined (see Fig. 1), namely the coastal forest belt, Kakamega forest, and forest near Tinderet and Elgeyo Marakwet, the latter two on the Rift Valley escarpment. Sera from these areas were obtained from cattle which had been born in 1972 and 1973 at least four years after the last outbreak of RVF. Sampling was made from different herds at up to 20 different sites in each forest zone. The areas are not involved to any great extent in improved farming with grade cattle; the cattle sampled were all of the unimproved indigenous *Bos taurus* type.

(*d*) Sera from domestic sheep were obtained from the four principal ecological zones and from animals born after the epizootic of 1968.

(*e*) Sera from sheep and goats, cattle and humans were collected in 1971 in a coastal village where a case of RVF virus infection had been observed in a human (Metselaar *et al.* 1974).

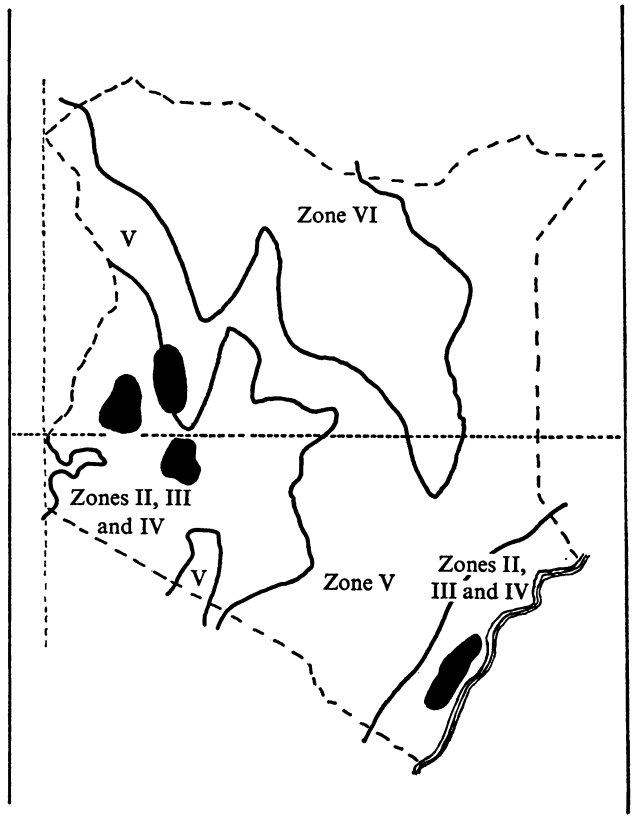

Fig 1. Forest sampling areas indicated in black.

(*f*) Sera collected from game animals by members of the FAO Wildlife Project at Kabete during and immediately following the 1968 outbreak.

Virus isolation

Tissues were homogenized with sterile sand with chilled pestles and mortars and 10 % suspensions made in 5 % fetal calf serum or 0·5 % bovine plasma albumin in phosphate buffered saline (PBS). They were incubated with antibiotics and used as inoculum for laboratory animals and tissue culture. Plasma or serum diluted 1/10 was prepared from blood samples. Hamsters and 4–8 week old mice were inoculated intraperitoneally with 0·1 ml., and 0·1 ml. of inoculum was adsorbed onto tissue cultures for one hour. Animals were observed for signs of infection for 1 week. Virus strains isolated in animals were identified by serum neutralization tests or by direct fluorescent antibody staining after inoculation of tissue culture. Later a fluorescent antibody technique (Pini, Lund & Davies, 1971) was used directly on liver samples from affected or laboratory animals.

Tissue culture

BHK21 C13 cells were used for the tissue culture work as described by Macpherson & Stoker (1962). The cells were grown in tubes with or without flying

Table 1

Rodent species screened for virus	Number
Otomys tropicalis	61
Lemniscomys striatus	12
Rhabdomys pumilio	1
Praomys jacksonii	7
Lophuromys flavopunctatus	5
Mastomys natalensis	13
Arvicanthus niloticus	11
Total	110

coverslips in Eagle's BHK medium with 10 % tryptose phosphate broth and 10 % bovine serum. The pH of medium was adjusted to 7·2 with bicarbonate solution, and antibiotics added (100 i.u. penicillin and 100 μg. streptomycin sulphate per ml.). The maintenance medium differed from the growth medium by the addition of only 2·5 % bovine serum. After absorption of the inoculum, the cells were washed three times in PBS before adding the maintenance medium.

Flying coverslips were harvested for staining by the direct fluorescent antibody technique (FAT) to identify virus strains, as described by Pini, Lund & Davies (1973).

Serology

The mouse neutralization test (Scott *et al.* 1956) was used to screen sera together with the indirect fluorescent antibody method for RVF described by Pini *et al.* (1973.) Flying coverslips were infected with the Kabete strain of RVF virus and harvested as antigen for this test at 24–56 hr. after inoculation. The method used was as described by Pini *et al.* (1973).

This method was used to screen the wild ruminant serum samples. Positive sera were confirmed by further serum neutralization tests. Common lines of gel precipitation were shown with the game sera and bovine or ovine gamma globulins.

RESULTS

Virus isolation

The sites at which virus was isolated from affected sheep and cattle in 1968, together with earlier departmental results, have been illustrated in Fig. 2. It shows that outbreaks of RVF were almost entirely confined to the ecological zones II, III and IV, which may be considered to represent the limits of the epizootic range of RVF in Kenya.

No virus isolations were made from the spleens of 110 rodents; 7 species were represented amongst the catches (Table 1). The animals were however taken in the post-1968 epidemic period.

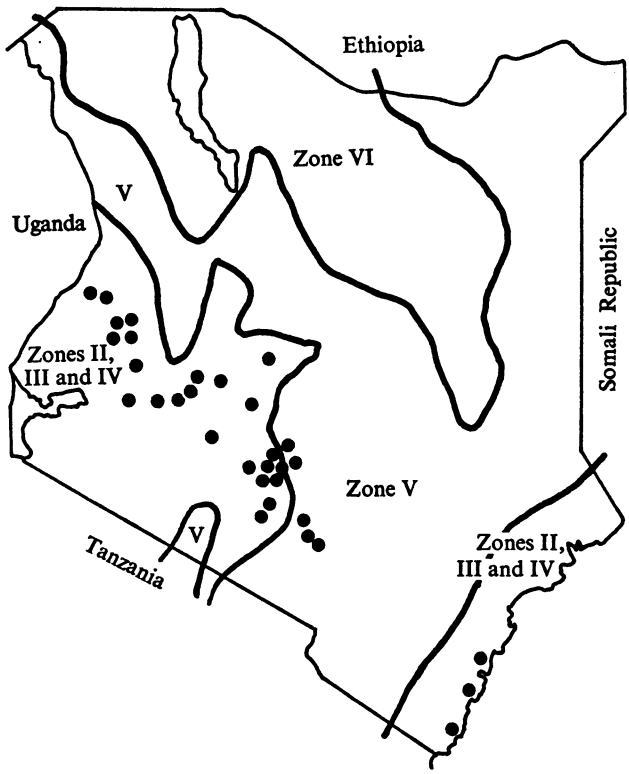

Fig. 2. Disease sites (●), up to 10 isolations per site.

Serological results

Sentinel herd

The results of the examination of sera from the sentinel herd show that of those alive in 1968, 81 % have antibody to RVF. The antibody titres show the same unnatural distribution with a double peak, as those described by Capstick & Gosden (1962) in a flock of sheep. During the years 1970–4 there has not been a single seroconversion to RVF amongst the animals born after 1968, nor any anamnestic response amongst those alive in 1968. This herd would appear to be outside the enzootic area for RVF in Kenya. It was however in the midst of an area worst affected in the 1968 epizootic.

All cases of abortion have been examined for antibody to RVF and none have been found with a rising titre to RVF virus.

Cattle survey

The results of the examination of sera from domestic cattle are recorded in Table 2 and illustrated in Fig. 3. Positive sites correlate fairly accurately with those sites where virus has been isolated from clinical cases. They are within ecological zones II, III, IV and V. The only anomalous results are from the Tana river district which is explained by the presence of heavy riverine forest and the permanent swamplands of the Amboseli basin. While these are in ecological zone V

Table 2. *The distribution of sera with antibody to RVF virus*

	Zone (no. of locations defined per zone in parentheses)				
	II	III	IV	V	VI
Domestic cattle, *Bos indicus* and *taurus*	193/1532 (122)	215/1425 (236)	106/712 (89)	16/444 (16)	0/67 (1)
Wildebeest, *Connochaetes taurinus*	—	—	2/62	—	—
Coke's hartebeest, *Alcelaphus buselaphus*	—	—	1/47	—	—
Thomson's gazelle, *Gazella thomsonii*	—	1/70	0/62	—	—
Grant's gazelle, *Gazella grantii*	—	—	2/33	—	—
Reedbuck, *Redunca fulvorfula*	—	0/24	—	—	—
Impala, *Aepyceros melampus*	—	—	0/33	—	—
Waterbuck, *Kobus ellipsiprymnus*	—	1/19	0/25	—	—
Buffalo, *Syncercus caffer*	0/19	0/16	0/27	—	—
Oribi, *Ourebia ourebia*	—	0/14	—	—	—
Eland, *Taurotragus oryx*	—	—	0/11	—	—
Topi, *Damaliscus korrigum*	0/36	—	—	—	—

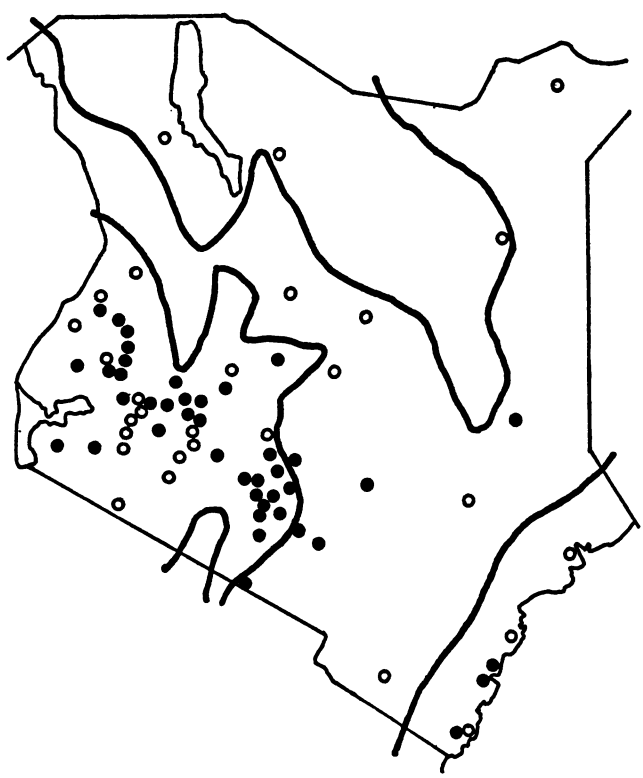

Fig. 3. ●, serological positive sites; ○, serological negative sites.

Table 3. *Serum samples from aborting cattle* 1968–74, *screened for antibody against RVF*

Year	Sera examined	Positive	Percentage positive of those examined	Ratio
1968	537	180	33·5	1:2·9
1969	582	22	3·8	1:26
1970	192	7	3·6	1:27
1971	252	9	3·6	1:28
1972	192	0	0	0
1973	189	0	0	0
1974	130	0	0	0

they are not characteristic for that zone, and could properly be described as local islands of type II ecological zone.

In the survey there was no apparent difference between the positive percentages of *Bos indicus* or *taurus* populations, they are therefore presented together.

Wild ruminants

Table 2 also shows the results of screening of a variety of game animals whose habitat was generally in the ecological zones III and IV. Most of the samples were obtained in 1968 and 1969 immediately after the major epizootic in Kenya. The few positive animals detected by FAT and confirmed positive by neutralization tests were shot a few miles from known disease sites for RVF in the epizootic.

Sera from aborting cattle

The results are shown in Table 3. The high proportion with antibody in the outbreak year of 1968 indicates the significance of the disease economically. It must be recorded that only a very insignificant number of sera from the total of those aborting ever reach the laboratory. In the years 1969, 1970 and 1971 a ratio of positive to total sera examined of 1:27 appears. The positive sera were in nearly all cases from animals alive in 1968 or were of vaccinal origin. In 1972, 1973 and 1974 no positive sera were detected from this sample series. It would not appear that RVF was causing symptomless abortion in any of the years after the 1968 epizootic.

Results of screening sera from the postulated enzootic foci

These results are recorded in Table 4. Positive sera were found at each of the postulated enzootic foci. The percentages positive were low, between 0·9 and 3·2 %. As the sera were all from animals born in 1972 and 1973 this is evidence of the involvement of domestic cattle in the interepizootic cycle for RVF virus. The cattle were all *Bos taurus*, of an unimproved type with low production potential.

Table 4. *Results of screening cattle against RVF from postulated enzootic foci*

District	Ecological zones	Sites	Positive	%
Coastal forest	II and III	20	4/447	0·9
Kakamega forest	II	20	4/274	1·5
Tinderet forest	II	4	2/63	3·2
Elgeo Markwet forest	II	20	3/284	1·1

Table 5. *Sheep sampled in the post-epizootic period, and screened for antibody to RVF*

	Ecological zone			
	II (9)*	III (8)	IV (8)	V (11)
Domestic sheep	0/137	0/121	0/136	0/262

* Figures in parentheses show the number of sampling sites.

Interepizootic maintenance of virus in sheep

The results of screening samples obtained in the interepizootic period are shown in Table 5. The screening of sera from these areas has not revealed any with antibody to RVF, no positive samples have been seen from sheep since 1968.

The sera which were collected at the time of the single human case at the coast (Metselaar *et al.* 1974) from 65 sheep and goats, 52 cattle and 22 humans, in the village of the affected person, were all negative for antibody to RVF.

DISCUSSION

The most interesting problem relating to RVF in Kenya and elsewhere has been to define the interepizootic maintenance cycle for the virus. The results reported in this paper indicate that domestic cattle have been involved in this cycle at certain forest and forest edge situations within the far more extensive epizootic range of the disease. However the percentage of seroconversions in the age groups sampled was low and varied between 0·9 % and 3·2 % (average 1·7 %). Whether this proportion is sufficient for the maintenance of the virus without some further host is arguable. At least as likely is that a basic cycle between one or more unknown vertebrate hosts and arthropods is going on unobserved during interepizootic periods, and only occasionally vertebrates other than the natural host are infected. Usually these are dead end infections. In Kenya the seroconversions in domestic animals and the human case reported by Metselaar *et al.* 1974 could be examples of such dead end infections. Under certain circumstances however further spread occurs. The periods of heavier-than-average rainfall with which the epizootics of RVF in Kenya are associated (Daubney *et al.* 1931; Scott *et al.* 1956) presumably provide conditions for an extension from this forest cycle. The disease appears in the ecological zones II, III and IV in cleared and developed open grassland savannah type country. The water pans and swamps created by the rainfall

allow of an enormous increase in the population of the opportunist mosquito species which are capable of transmitting RVF (McIntosh, 1972): their feeding cycles overlap with those of forest edge species and once infected a very rapid amplification of virus in the vector and susceptible cattle and sheep populations can occur. A large proportion of animals have a viraemia with no clinical signs (Haig, 1951).

Previous work in Kenya (Scott *et al.* 1956) has been carried out during and immediately after epizootics. The results reflect an epizootic and immediately post-epizootic situation which reveal evidence of widespread inapparent infection, and some abortions as the outbreaks persist for 1–3 years. Their studies were not carried out in a true interepizootic period.

In South Africa Kaschula (1953) was able to demonstrate seroconversions in a cattle herd in the Knysna area with no clinical disease other than occasional abortion. It is thought that these results were obtained in the years after 1950 when clinical RVF was prevalent in many parts of South Africa again as an epizootic. Studies in Uganda have been carried out primarily with the vector in true enzootic areas (Smithburn *et al.* 1948). This work was done in a forest similar to that at Kakamega postulated as an enzootic focus in Kenya. Virus was isolated from several species of ground level feeding mosquitoes, no antibody was found in 72 wild monkeys killed in the same area, and they were unable to incriminate any vertebrate in this forest edge. Other work in Uganda has been carried out at Lunyu which is near Entebbe on the shores of Lake Victoria. Mims (1956) and Weinbren & Mason (1957) both suggested that rodents were important reservoirs for RVF in this area. Henderson *et al.* (1972) working during the 1968 epizootic were unable to confirm this suspicion, and their work may be considered important supporting evidence, with that of Scott & Heisch (1959), Woodall & Williams (1960) and Gear *et al.* (1951), that rodents play no part in the maintenance cycle for RVF. No virus was isolated from the rodent material in this work.

The work of Pellisier & Rousselet (1954) with monkeys in Brazzaville showed 12 with complement fixing antibody of 113 sera examined. These animals were housed however. Davies, Clausen & Lund (1972) tested 333 baboon sera from Kenya in the year after an epizootic; all were negative. A further 400 African green monkey sera collected in 1974 in Kenya have been tested for antibody to RVF and all were negative (Davies, to be published). Together with the negative results of Smithburn *et al.* (1948), it seems unlikely that primates are involved in the maintenance cycle for RVF.

Wild game have been incriminated in the cycle by Smithburn *et al.* (1948) and Maurice & Provost (1969). The latter demonstrated haemagglutinating antibody in the sera of 16 out of 33 wild bovidae in Chad. The results from Kenya would not support the view that they play a significant part as reservoir hosts for RVF. Bluetongue antibody is widespread in wild bovidae in Kenya (Davies & Walker, 1974*a*) as is antibody to ephemeral fever virus in buffalo and waterbuck (Davies, Shaw & Ochieng, 1975). It is thought they are involved in the maintenance cycles for these viruses, both of which are *Culicoides* transmitted (Walker & Davies, 1971; Davies & Walker, 1974*a*, *b*). Antibody to RVF was found in only very few game

animals close to outbreaks of disease in domestic animals in 1968. None has been found subsequently in 36 topi killed in ecological zones II and III close to coastal forest which is considered to be enzootic for RVF, and in 119 reed buck, waterbuck and buffalo, some of which were killed in habitats similar to those postulated and proved to be enzootic for RVF.

There is evidence from South Africa (Gear *et al.* 1951; Alexander & Dickson, 1951) and from Kenya (Daubney & Hudson, 1932) that wild game are susceptible to RVF. It is probable that they are disease hosts in the same way as domestic ruminants.

Alexander (1957) reported on the susceptibility of the Sudan dioch to RVF virus, and incriminated this bird's migration as responsible for virus introductions along the Rift Valley. Findlay (1931) was unable to infect domestic fowls or other birds, and Davies (unpublished data) was unable to produce viraemia in chickens inoculated with 10^7 mouse LD 50 of the Kabete strain of RVF virus.

Unless a small number of ongoing infections in cattle such as demonstrated by the seroconversions reported in this paper could be sufficient to maintain the virus cycle, the problem of the primary vertebrate host of RVF virus has still to be solved.

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