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Rift Valley Fever in Kenya: the presence of antibody to the virus in camels (*Camelus dromedarius*)

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SUMMARY

Five hundred and seventy-one camel sera collected after an epizootic of Rift Valley Fever were examined for antibody to the virus. Clinical disease had not been observed in cattle and sheep in the ecosystems shared with the camels. Positive sera with high titres of serum neutralizing antibody were found in 22 % of camels at one of the seven sampling sites.

Scott *et al.* (1963) reported the presence of neutralizing antibody to Rift Valley Fever (RVF) virus in camel sera from the Garissa and Marsabit districts of northern Kenya. They associated this finding with the previous widespread abortions in camel herds which had instigated their investigations. Antibody to RVF in camel sera has also been reported from the sub-Saharan region of Nigeria (Fagbami, Tomori & Kemp, 1973) and more recently from Egypt (Ali & Kamel, 1978; Hoogstraal *et al.* 1979) where RVF virus was also isolated from a camel (Imam, Karamany & Darwish, 1978) and the Sudan (Eisa, 1981).

Epizootics of RVF have occurred regularly in Kenya since the original report of Daubney, Hudson & Garnham (1931). These have been recognized by the abortions and the mortality which have occurred in the principal disease hosts in Kenya, which are the exotic breeds of sheep and cattle and their crosses (Scott, Weddell & Reid, 1956; Davies, 1975). Such animals are usually found in the higher rainfall areas of the highlands and at the coast, for it is in these regions that the animal production potential is greatest. The distribution of the disease was confirmed in a serological study (Davies, 1975) carried out in the immediate post epizootic period, with cattle and sheep sera from the different ecological zones of Kenya (Pratt, Greenway & Gwynne, 1966). Positive sera with evidence of quite high exposure rates were obtained from the higher rainfall zones but virtually no exposure of cattle or sheep and goats had occurred in the drier zones during the 1967–8 and 1978–9 epizootics. A few positive sera were obtained from cattle herds close to riverine forest extending from known enzootic zones into the drier areas (Davies, 1975). The report of Scott, Weddell & Reid (1963) suggests that in 1962 the epizootic caused abortions in camels. The rains of 1962 produced exceptional flooding in the semi-desert northern province of Kenya (Kenya Meteorological Department, Annual Reports). This paper records the results of serum neutralization tests with RVF virus on sera from camels collected in the period after the 1978–9 epizootic of RVF in Kenya.

Table 1. *Rift Valley Fever antibody in camel sera collected after an epizootic in Kenya*

(*Sera assayed by a microserum-neutralization test in Vero cells against* 50–75 $TCID_{50}$ *of virus.*)

Origin	Number positive	Number tested
Kulal	0	22
Ngurunit	0	109
Ol Maisor	0	91
Kisima	0	116
Garissa	0	48
Bura	0	42
Galana	32	143
Totals	32	571

Table 2. *Titres of antibody to Rift Valley Fever virus obtained with camel sera from Galana, when assayed by a microserum-neutralization test against* 50–75 $TCID_{50}$ *of the virus*

	Antibody titre*									
	20	40	80	160	320	640	1280	2560	5120	10240
Number	2	3	0	1	2	5	6	7	4	2

* Reciprocal of the dilution of serum suppressing 80% or more of the cytopathic effects of the virus.

The strain of RVF virus used in the neutralization tests was isolated from *Culicoides* during the Kenya 1978–9 epizootic (Davies & Highton, 1980). This was shown to be identical with the Kabete strain of RVF and indistinguishable by neutralization tests from strains of RVF obtained in earlier epizootics. The virus was adapted to Vero cells by three passages at limiting dilutions and then stored at −70 °C. Vero cell cultures were grown in Eagles minimum essential medium with 10% bovine serum, and sodium bicarbonate (4·4%) to adjust the pH to 7·2. Approximately 20000 cells were added in 0·1 ml aliquots to each well of a flat-bottomed polystyrene microtitre plate.

The camel sera were inactivated at 56 °C for 30 min at a 1 in 5 dilution. They were diluted from 1 in 10 to 1 in 10240 by doubling dilutions made in medium without serum in the microtitre plates. Some 50–75 $TCID_{50}$ of RVF virus was then added to each well. Control positive and negative bovine sera were included with each test series. Plates were incubated for 1 h at 37 °C before the cell suspension was added, and the plates sealed. The plates were examined daily for 5 days and the end points taken at the dilution where more than 80% of the cytopathic effects of the virus challenge dose were suppressed.

Sera were obtained from a semi-arid zone of *Acacia* savannah (moisture index −30 to −42), an arid zone of dry thorn bush (moisture index −42 to −51), and very dry semi-desert of dwarf shrubs or very dry grassland (moisture index −51 to −57). The places of origin of the sera are shown in Table 1. The collections include all age groups, and were made in 1980 and 1981 following a RVF epizootic.

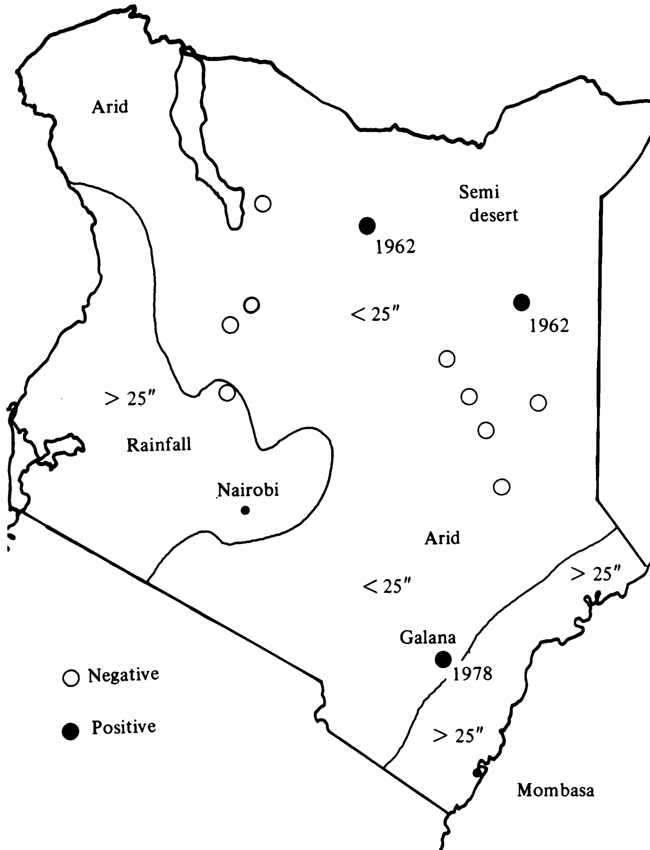

Fig. 1. Sampling sites for RVF antibody in camels in Kenya.

The numbers of sera found to have antibody to RVF virus at the different sample sites are shown in Table 1, and the titres obtained from the positive animals are shown in Table 2. The geographical distribution of the sampling sites in the present study are shown in Fig. 1. The sites found positive in 1962 (Scott, Weddell & Reid, 1963) are included for comparison. Of the 571 camel sera tested, 32 were found to contain serum neutralizing antibody to RVF virus and these were confined to sera from one sampling site. Sera positive for RVF antibody were found in cattle at this site after the 1968–9 and 1978–9 epizootics of RVF, but in less than 10% of those sampled. The area is dry thorn bush close to the Galana river, which extends with a riverine forest belt from the coast, where RVF is enzootic. The titres were comparable with those obtained in positive cattle and sheep sera assayed by the same technique. The five low titres (from 20 to 40) were found in camel foals and may be due to the persistence of maternally derived antibody.

Clinical disease which has been caused by RVF virus in camels has not been described, although virus has been isolated from an apparently naturally infected animal (Imam, Karamany & Darwish, 1978). Experimental infection did not produce any disease (O. L. Wood, personal communication). Abortions have been attributed to RVF infection in camels on the basis of retrospective serology (Scott

et al. 1963; Ali & Kamel, 1978). There were no abortions in the herd showing high titres of antibody to RVF in this study.

Clinical RVF has not been diagnosed in sheep, goats, nor cattle in the drier semi-desert parts of Kenya. Serological investigations have confirmed that in two recent epizootics, a few animals had been infected with RVF in these zones. The positive cattle sera were found adjacent to riverine habitat extending from a RVF enzootic area, the positive camel sera came from the same area. This paper and that of Scott *et al.* (1963) show the potential for RVF to occur in some semi-desert zones. This is an important observation in the study of the ecology of RVF, and suggests that the virus may persist in islands of enzootic habitat in such areas, or be introduced during epizootics. Some depressions are known to be flooded in exceptionally heavy rains, which occur on a 15–25 year cycle. They could be important in the ecology of RVF in semi-desert zones.

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