Observations on the epidemiology of ephemeral fever in Kenya

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SUMMARY

Ephemeral fever antibody was found in domestic cattle in Kenya across a wide range of ecological zones, from highland forests and grasslands to desert and semi-desert thorn scrub. Antibody was found in several species of game animals, notably waterbuck and buffalo, where over 50% of the samples showed antibody to EF. Evidence was obtained to show that the virus had been cycling in these wild ruminant populations between epizootics in domestic cattle.

INTRODUCTION

Ephemeral fever (EF) has been recorded in Kenya since 1913–14 when the clinical syndrome was described and reproduced by the subinoculation of blood from infected animals. In Kenya there has been an association of epizootics of the disease with years of greater than average rainfall. Recent observations have shown that more local outbreaks occur within a period of a few weeks following local rains, and a further outbreak occurred in the Rift Valley adjacent to saline lakes with no record of recent rains (Davies & Walker, 1974a).

The disease is seen in both *Bos indicus* and *B. taurus*; it is not usually associated with any mortality other than that produced by secondary factors, notably the lack of water for recumbent animals in range conditions. Dairy herds suffer a significant loss in milk production during epizootics (Macfarlane & Haig, 1955; Theodoridis, Giesecke & du Toit, 1973) and this is the principal economic importance of the disease in Kenya.

When the study was initiated an hypothesis was made that, should the major vector of EF be a *Culicoides* midge, the distribution of specific antibody would show the same general pattern as that to bluetongue virus in Kenya, which is *Culicoides* transmitted (Walker & Davies, 1971; Davies & Walker, 1974b). Mosquito transmitted virus infections in Kenya have a more restricted range. It was to test this hypothesis that the study was initiated. Subsequently EF virus was isolated from a pool of wild caught *Culicoides* (Davies & Walker, 1974a).

No natural or amplifying hosts for EF have been demonstrated other than domestic cattle (Snowdon, 1971; St George et al. 1973). The clinical syndrome has been described in domestic water buffalo in the Philippines (Topacco et al. 1937). This survey includes the results of screening a variety of game animals to examine the possibility that they play a part in the natural history of this virus infection

MATERIALS AND METHODS

Tissue culture

The tissue cultures used and their method of preparation and maintenance were as described in the previous paper (Davies, 1975).

Virus strains

An Australian strain of EF virus kindly supplied by Dr W. A. Snowdon was used at first for the neutralization tests. This was later replaced by the Kenya strain K 86/73 (Davies & Walker, 1974a), which was shown to be serologically indistinguishable from the Australian virus. This strain produced cytopathic effects in BHK cells 36-48 h. earlier than the Australian BHK adapted virus.

Neutralization tests

These were carried out in infant mice and BHK cells according to the method of Snowdon (1970). A slight modification was that sera were diluted 1/2 in phosphate buffered saline before mixing with the appropriate virus dilution, the final serum dilution was thus 1/4. Between 100 and 400 mouse LD 50 or TCID 50 were used as the screening dose for antibody.

Sera

A total of 758 bovine sera which had been collected at 36 different sites by the Kenya Veterinary Department for a variety of purposes during the years 1968–72 were used for the survey. These were used to outline the distribution of antibody which followed the epizootic of EF which occurred in 1968. Sera from game animals were obtained from the collection made by the FAO Wildlife Project at Kabete, and by Dr E. C. Anderson., WIRFMD*, Embakasi, Kenya. All sera were inactivated at 56° C. for 30 min. before use.

Ecological zones

The sampling sites are described in the ecological zones following the classification by Pratt, Greenway & Gwynne (1966) which is particularly useful for the purpose, as it is based on climate, vegetation and land use, and is thus related to the potential of the area for stock farming. The principal characteristics of the zones have been summarized in the previous paper (Davies, 1975).

RESULTS

Distribution of antibody

Fig. 1 shows the distribution of antibody to EF virus in cattle in various parts of Kenya. Positive sera were found across the whole range of ecological zones examined, and it may be emphasized that this involves a range from semi desert thorn scrub to temperate type highland grass habitat. Positive sera were obtained at each of the thirty-six sites sampled. A further analysis of the results is shown in Table 1.

* Wellcome Institute, Research on Foot and Mouth Disease.

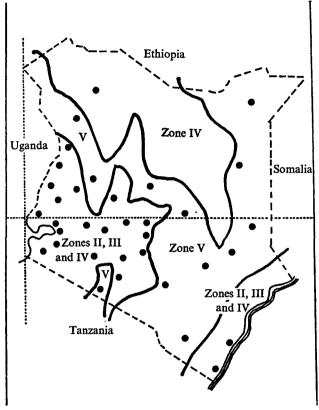


Fig. 1

Table 1. The number of cattle serologically positive to EF virus described by ecological zone

	II (9)*	III (10)	IV (9)	V (6)	VI (2)
Domestic cattle	119/225	151/241	57/153	71/109	21/30

^{*} Figures in parentheses indicate sampling sites.

Morbidity in cattle

The morbidity in a sentinel herd of cattle in 1968 was 74 %. This was determined by serological confirmation of the clinical records which were maintained. Both Bos indicus and taurus were susceptible to clinical disease in the field, and there was no apparent difference in the morbidity rates determined by seroconversion.

Antibody in wild ruminants

The results of screening the sera from wild ruminants are shown in Table 2. Wherever possible the sera examined were those from animals shot immediately after the 1968 epizootic and negative results in most species can be considered significant even though the sample numbers are not very large. Both waterbuck and buffalo seem to be involved in the natural history of this virus as amplifying or reservoir hosts. A further analysis of the samples from these two species (Table 3) shows that both species have been involved in the interepizootic maintenance

Table 2. Results of serum neutralization tests in wild ruminants against EF virus

Species	Ecological zone	$egin{array}{c} \mathbf{Number} \\ \mathbf{tested} \end{array}$	Number positive	%
Buffalo, Syncercus caffer	II & III	72	39	54
Waterbuck, Kobus ellipsiprymnus	III & IV	31	19	61
Wildebeest, Connochaetes taurinus	II	32	3	9
Hartebeest, Alcelaphus buselaphus	III	36	1	2.8
Impala, Aepyceros melampus	III	22	0	0
Grant's gazelle, Gazella grantii	III & IV	24	0	0
Thomson's gazelle, Gazella thomsonii	III & IV	38	0	0
Eland, $Taurotragus$ $oryx$	IV	4	0	0
Oryx, Oryx beisa	IV	3	0	0

Table 3. Analysis of serological results in buffalo and waterbuck populations, demonstrating the seroconversion of animals in the interepizootic period

Species	Alive during cattle outbreaks	Alive only between outbreaks
Waterbuck	7/12	12/19
Buffalo	9/15	30/57

of this virus in Kenya. In 1968 a widespread epizootic of this disease occurred over the whole country. No further clinical cases of the disease were observed until late 1972, although evidence of the persistence of the virus was obtained by seroconversions in a sentinel herd in 1971. Table 3 seems to indicate that the virus of EF was persisting in the waterbuck and buffalo populations. Both buffalo and waterbuck which were born after the 1968 epizootic were found to be positive on neutralization tests.

DISCUSSION

Antibody to EF in Kenya is found across the wide range of varying ecological habitat. A similar picture emerged from a study of the distribution of antibody to blue tongue (BT) virus in a wide range of animal species and of the principal vectors of BT virus in Kenya (Walker & Davies, 1971; Davies & Walker, 1974b). This might reasonably be considered to support the hypothesis that *Culicoides* are the vectors of EF in Kenya. There is evidence of the widespread distribution of the blood feeding species of *Culicoides* (Walker, personal communication) which are known to feed on domestic cattle.

The finding that sera from wild game species, notably buffaloes and waterbuck, show neutralizing activity against EF is interesting. The screening virus titre of at least 100 mouse LD 50 or TCID 50 which in one series of buffalo tests was 400 mouse LD 50, seems to support the view that the test was detecting virus specific

neutralizing antibody. The possibility that the test might be detecting non-specific neutralizing substances in sera was discussed at length by Snowdon (1971), who concluded that this was most unlikely. These results are discussed on the assumption that specific antibody has been found in these wild ruminant species. The finding is highly significant. In Kenya many extensive ranching systems and even dairy farms carry considerable wild game populations. In 1968 when the last countrywide epizootic of EF occurred in Kenya, challenge of buffalo and waterbuck presumably occurred but there were no reports of any clinical disease in these species. In the absence of evidence to the contrary EF in game must be considered to be a subclinical infection. The antibody titres seem to indicate that a viraemia occurs and until pathogenesis experiments have been carried out it is reasonable to suppose that this viraemia is similar to that in cattle and capable of infecting an arthropod vector.

After the clinical cases in cattle in 1968, no further disease was seen until late 1972. The serological results from different age groups of buffalo and waterbuck show that virus was cycling in these animals in this interepizootic period. It is reasonable to conclude that they are involved as reservoir and amplifying hosts for this virus. Further evidence from a sentinel herd study has shown that cattle are also reservoirs in this interepizootic period, for seroconversions have been shown to have occurred when no clinical disease was recognized (Davies, unpublished data). The infection may have been sub-clinical. A similar observation has been made by Snowdon (1971) in Australia.

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