Wildlife Disease Research and Economic Development

Proceedings of a workshop held in Kabete, Kenya, 8 and 9 September 1980

Editors: Lars Karstad, Barry Nestel, and Michael Graham

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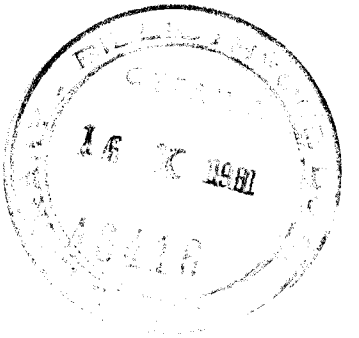

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The Possible Role of Wildlife as Maintenance Hosts for Some African Insect-Borne Virus Diseases

F. G. Davies[1]

The coastal strip of Kenya has been inhabited for many years, while it is only over the last 400 years that the highland areas have been populated by the movement of peoples from the south and the north. The natural fauna, the wildlife inhabiting the forest and bushed grasslands, now share their habitat with the domestic ruminants brought by the Nilotic and Bantu peoples. Many insect-borne viruses had established maintenance cycles in the forest and savanna habitat. These involved the natural fauna of the region and showed little or no evidence of disease. The coming of the domestic stock several centuries ago and the importation of more exotic species this century introduced many new potential disease hosts, as did the coming of the people themselves. The impact of these intrusions has become more and more interesting and relevant to the development of the region, both for animal production and for the health of the human population.

Diseases such as bluetongue in the wool sheep, Nairobi sheep disease, Rift Valley fever, ephemeral fever, and lumpy skin disease were recognized in Kenya with the development of the agricultural industry. African horse sickness appeared in horses brought into the territory. The causative viruses were suspected to be transmitted by insects and the elucidation of the natural maintenance cycles for these viruses is a subject of considerable interest and relevance to their ultimate control.

At Kabete, investigations were carried out on the distribution of diseases, and of antibodies and vectors where these were known, in relation to the ecological zones in Kenya. The antibody surveys were carried out both in the disease host populations and also among the natural fauna of the region. Some of the diseases initially behaved in an epizootic manner and later contracted to persist in enzootic maintenance cyles in limited habitats. Fundamental studies were carried out, in particular with *Culicoides* (Walker 1976), to determine their distribution, population biology, feeding and breeding habits, and the foraging ranges. Attempts were also made to isolate viruses.

Investigations were made of the role of wildlife in these diseases. Initially, efforts were made to isolate viruses from wild game animals killed for control purposes; more than 100 samples were inoculated into sheep, BHK cell cultures, infant mice, and embryonated eggs. Two passages were made in the latter three systems. This work did not result in any virus isolations (Davies 1980). The serological investigations generally were carried out with one test but the results were always validated by a further serological test for the same disease.

Bluetongue

Sera from a wide range of wild ruminant species collected in many different ecological zones contained both fluorescent and type-specific neutralizing antibody to bluetongue virus. Animals such as the buffalo (*Syncerus caffer*), wildebeest (*Connochaetes taurinus*), kongoni (*Alcelaphus buselaphus cokei*), and various gazelles (*Gazella* spp.) are probably important maintenance hosts for bluetongue virus in Kenya and many other parts of Africa. Large proportions of their populations have shown evidence of challenge by bluetongue virus (Davies and Walker 1974; Davies, unpublished). In the absence of cattle, which have replaced the wild ruminants in many developed parts of the country, it is considered that they play the major role in the virus maintenance cycle. Blood meal analysis of the species of *Culicoides*, which are considered important vectors of bluetongue, show that they feed principally upon wild and domestic ruminants (Walker and Boreham 1976). Where agricultural development has largely excluded wild species, domestic cattle assume the principal role in the maintenance

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of the virus (Davies 1978). There are large parts of Africa where agricultural developments in the next 50 years may introduce disease hosts. The bluetongue virus maintenance cycle probably already exists in those countries.

Ephemeral Fever

Ephemeral fever occurs in Kenya in the form of epidemics which sweep across large parts of the country (Davies et al. 1975). Between the epidemics, limited foci of disease may be encountered and there is evidence of seroconversions in sentinel herds. An examination of a range of wild ruminant sera showed evidence of neutralizing antibody in 54% of buffalo, 62% of waterbuck, 9% of wildebeest, and 2.8% of hartebeest. These samples had been collected from the higher potential agricultural areas of the country through to the more arid acacia savanna grasslands. Antibody is also found in the domestic cattle population in these zones. A further interesting observation was that there were seroconversions in waterbuck and buffalo that were not alive during the previous epizootic of the disease. This is evidence that the virus was cycling in the wild ruminant population during a period when no clinical disease was diagnosed in cattle (Davies et al. 1975). These animals may be important maintenance hosts for this virus in Kenya.

African Horse Sickness (AHS)

The only Equidae in Kenya before the importation of horses were the zebras (*Equus* spp.). As soon as horses were imported into the country in this century, large numbers died of the disease. This is a classic situation of a virus finding a disease host. No disease has ever been recognized in the wild zebra population, and attempts to isolate the virus from zebra blood have proved unsuccessful. Antibody has been found in zebra sera by complement fixation, fluorescent antibody, and neutralization tests (Davies and Lund 1974; Davies and Otieno 1977; Davies, unpublished).

As a follow-up of some apparently low-titre neutralizing antibody titres in elephant sera, a number were screened by a complement-fixation test (Davies and Otieno 1977). Some 84% were found to contain complement-fixing antibody to AHS antigen. The significance of this is not understood and it may be due to another virus closely related to AHS. There is little doubt, however, that wild zebra play an important role in the maintenance and amplification of AHS virus in East Africa.

Rift Valley Fever

This is one of the most pathogenic zoonoses known and was first identified in the Rift Valley in Kenya in 1931 (Daubney et al. 1931). The disease principally affects cattle, sheep, and goats in an epizootic manner at intervals of up to 10 years. Man is accidentally infected, generally by contact with the diseased animals. A survey of wild ruminant sera collected after an epizootic in Kenya in 1968 showed that there was very little involvement of the wild ruminant populations. A very small and insignificant proportion of wildebeest and hartebeest were found to contain antibody. This was not surprising, for the game animals generally inhabit areas which are outside the epizootic range of the disease (Davies 1975a). Buffalo infected experimentally with the virus developed a viraemia and one of four animals aborted in a manner similar to that expected in cattle (Davies and Karstad 1980).

Between epizootics, the virus maintenance cycle contracts into certain forest regions of Kenya. It is not clearly known what vertebrate is involved in the maintenance cycle. Cattle are involved to a certain extent where they graze at the forest edge (Davies 1975a) and certain wild ruminant species in the forest may also be involved. Buffalo are likely hosts in such areas, but it has not been possible to confirm this (Davies and Karstad 1980). Further investigations have included baboons (*Papio anubis*) (Davies et al. 1972), green monkeys (*Cercopithecus aethiops*) (Davies and Onyango 1978), birds (Davies and Addy 1979), and rodents. No evidence for the involvement of these species has been obtained. The problem of defining the interepizootic vertebrate hosts remains.

Lumpy Skin Disease

There is no direct evidence to show that the virus is transmitted by a biting insect, however, the circumstantial evidence strongly supports this view (Diesel 1949; MacOwan 1959). The capripox viruses have cross relationships with one another and epidemiological investigations using serological tests suffer the disadvantage that any antibody detected may be due to a sheep and goat pox virus, or at low titres to cowpox. The latter however does not occur naturally in Kenya. There is no evidence of any infection of the wildlife populations in areas where epidemics of sheep and goat pox have occurred (Davies 1975b).

Lumpy skin disease was first recognized in Kenya in 1957 when it appeared in epizootic form, although with a much lower morbidity than was experienced

in South Africa. Since that time, the disease has occurred sporadically in most years with only one or two clinical cases recognized. These have always been in certain high altitude districts adjacent to a large forest area and frequently in farms excised from the forest. Because the capripox viruses have a very narrow host range, it was thought likely that the wildlife maintenance hosts must exist in the larger wild ruminants (e.g. buffalo) found in such habitats. In sera from such ecological zones and those contiguous with them, neutralizing antibody was found in a high proportion of the sera examined (Davies, unpublished). Buffalo sera from areas where the disease has not occurred did not contain antibody. In samples from Uganda taken in a year after an epizootic of the disease, a high proportion contained neutralizing antibody to lumpy skin virus. None of the 166 buffalo sera contained antibody to a strain of cowpox virus. If the antibody detected in the buffalo sera is in fact specific, these animals could be important reservoirs for the disease.

Nairobi Sheep Disease

This is a tick-borne disease of sheep and goats transmitted principally in Kenya by *Rhipicephalus appendiculatus* (Montgomery 1917; Davies 1979a). This tick feeds largely upon cattle and the larger wild ruminants. Smaller proportions feed upon sheep and goats but generally they are less preferred feeding hosts than bovids. A survey of wildlife sera, collected in areas where this tick was common, showed few with any antibody to the virus, and then only at a very low titre. These low titres were confirmed by complement fixation, fluorescent antibody, and indirect hemagglutination tests (Davies 1979b) and it was considered likely that they were cross reactions with other viruses. This suspicion was given support by the finding that Nairobi sheep disease was probably identical with a virus, Ganjam, from India and reacted on fluorescent antibody tests with Congo, Dugbe, and some other tick-borne viruses (Davies et al. 1978). There are reports of antibody to Congo and Dugbe viruses in wild ruminant sera detected by agar gel precipitation.

The conclusion from this work is that wild game animals do not play any role in the maintenance of this virus, supporting a hypothesis that it is an introduced virus. Sheep and goats are not indigenous to East Africa but may have been brought from India to the coast years ago. The noninvolvement of game animals in sheep and goat pox indicates that this also may have been introduced. The remainder, bluetongue, ephemeral fever, African horse sickness, and lumpy skin disease, are likely to be indigenous to the East African ecosystems and be maintained in the wild game populations of the region. Rift Valley fever also is an indigenous virus, utilizing the forest habitat for maintenance and probably some wild vertebrate species.

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