The International Development Research Centre is a public corporation created by the Parliament of Canada in 1970 to support research designed to adapt science and technology to the needs of developing countries. The Centre’s activity is concentrated in five sectors: agriculture, food and nutrition sciences; health sciences; information sciences; social sciences; and communications. IDRC is financed solely by the Parliament of Canada; its policies, however, are set by an international Board of Governors. The Centre’s headquarters are in Ottawa, Canada. Regional offices are located in Africa, Asia, Latin America, and the Middle East.

©1981 International Development Research Centre
Postal Address: Box 8500, Ottawa, Canada K1G 3H9
Head Office: 60 Queen Street, Ottawa

Karstad, L.
Nestel, B.
Graham, M.
IDRC, Ottawa CA


/IDRC publication/, /wild animals/, /research/, /animal diseases/, /disease transmission/, /livestock/, /Kenya/ — /epidemiology/, /parasitic diseases/, /infectious diseases/, /viruses/, /immunization/, /disease control/, /meat/, /food contamination/, /ruminants/, /animal production/, /environmental effects/, /list of participants/.

UDC: 591.2

Microfiche edition available
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
Contents

Foreword 5
Participants 7
Opening address
  S. Chema 11
The role of wildlife disease research in livestock development
  Lars Karstad and Barry Nestel 13
The role of wildlife in the epidemiology of foot-and-mouth disease in Kenya
  E.C. Anderson 16
Queries about rinderpest in African wild animals
  A. Provost 19
Epidemiology and control of bovine malignant catarrhal fever
  E.Z. Mushi, F.R. Rurangirwa, and L. Karstad 21
The possible role of wildlife as maintenance hosts for some African insect-borne virus diseases
  F.G. Davies 24
The possible role of wildlife in the natural history of rabies in Kenya
  F.G. Davies 28
Attempted isolation of Cytocoetes ondiri from wild ruminants in areas where bovine petechial fever is endemic
  F.G. Davies 30
The importance of wildlife in the epidemiology of theileriosis
  J.G. Grootenhuis and A.S. Young 33
Potential application of research on African trypanosomiases in wildlife and preliminary studies on animals exposed to tsetse infected with Trypanosoma congoense
The role of wild ruminants in the epidemiology of nematodiasis in Kenya
  E.W. Allonby 46
Helminths in wild ruminants in Central Africa: impact on domestic ruminants
  M. Graber 48
The role of jackals in the transmission of Echinococcus granulosus in the Turkana District of Kenya
  Calum N.L. Macpherson and Lars Karstad 53
The public health significance of cysticercosis in African game animals
  P. Stevenson, A. Jones, and L.F. Khalil 57
The value of research findings to the research director
S. Chema 62

The role of wildlife disease research in livestock production
L.J. Howard 64

Wildlife ranching in perspective
David Hopcraft 68

What ecologists think veterinarians should do
Harvey Croze 72

Discussion Conclusions 76
The Role of Wildlife in the Epidemiology of Foot-and-Mouth Disease in Kenya

E. C. Anderson

Foot-and-mouth disease (FMD) is a disease of cloven-footed animals, both domestic and wild, caused by a virus of the picornavirus group and characterized by fever and vesicle and ulcer formation in the mouth, on the feet, and on the udder and teats of females. Recovered animals may harbour the virus in the upper respiratory tract for variable periods of time depending on the species.

In considering the possible role of wildlife species in the epidemiology of FMD it is necessary to examine briefly current knowledge on the pathogenesis of the disease in the domestic animal.

Until the late 1950s, it was generally thought that the method by which cattle became infected was by ingestion — the virus entering the bloodstream through lesions in the alimentary tract. However, this concept was revised when infection by airborne virus was shown (Hyslop 1975; Eskildsen 1969). It is now agreed that natural infection takes place via the respiratory tract (Sellers 1971; McVicar 1977) through the inhalation of virus-containing aerosols. In the upper respiratory tract, the pharynx is a predilection site for virus multiplication, and it is in the pharynx that the virus may persist for long periods (Van Bekkum et al. 1959; Burrows 1966; Sutmoller and Gaggero 1965).

Following infection, virus is shed in greatest amounts during the clinical phase of the disease when lesions are present. At this time, aerosols are generated from virus released from ruptured lesions in the mouth and on the feet, teats, and udder and from virus multiplying in the pharynx. Milk and feces from newly infected animals also contain virus.

The exposure of apparently insusceptible animals or immune animals to airborne virus can also lead to infection of the upper respiratory tract. These animals do not develop clinical disease but may excrete virus for up to 7 days following exposure (Sellers et al. 1977; Donaldson 1979). The amount of exhaled virus is proportional to the amount present in the pharynx but is much less than found in clinically diseased animals.

To assess the possible role of wildlife in the persistence and dissemination of FMD the following questions must be answered: (1) What species are susceptible to infection? (2) Do they develop clinical disease and, therefore, generate large amounts of virus? (3) Which species become virus carriers following exposure? (4) Can virus carriers transmit the disease to domestic animals?

Susceptibility of Wildlife Species

An initial assessment of which species might be susceptible to FMD was made by carrying out an extensive field survey for the presence of serum antibody to the five serotypes of virus found in Kenya. Attempts to isolate virus from the throats of many of the animals were also made.

From the results of this survey (Table 1) four species, impala (Aepyceros melampus), wildebeest (Connochaetes taurinus), eland (Taurotragus oryx), and buffalo (Syncerus caffer), were selected for controlled laboratory exposure experiments. The method of exposure was either by tongue inoculation (impala, wildebeest, eland, and buffalo) or by nasal instillation (buffalo). The course of the clinical disease, the sites of multiplication, the routes of excretion of virus, and the development of the carrier state were examined (Anderson et al. 1975, 1979, 1980).

Transmission of Disease from Wildlife to Domestic Animals

Eland were found to harbour the virus in the throat for up to 32 days after exposure while buffalo remain carriers for at least 2 years and probably much
longer. Experiments were carried out where eland and buffalo were housed in close contact with susceptible cattle to see if virus transmission would occur.

Results of the field survey for the presence of virus and serum antibody are shown in Table 1. The highest incidence of antibody was found in the buffalo and antibody was found to all five serotypes. Antibody was also observed in a small proportion of eland, Grant’s gazelle, Thomson’s gazelle, wildebeest, impala, and topi. Virus (types Sat 1 and Sat 2) was isolated from 14% of the buffalo sampled but not from any other species.

Following exposure to virus in the laboratory typical clinical disease was not observed in any of the four species examined. The impala and wildebeest did not harbour the virus for longer than 7 days and the eland for 32 days, but at no time was the virus present in high titre.

In contrast, the carrier state was readily established in the buffalo using the Sat serotypes, and the virus was found in high titre in the throat for up to 3 months after exposure. The buffalo appeared to be less susceptible to type A although the carrier state with this serotype could be established. The buffalo remained carriers for at least 280 days following laboratory exposure and for at least 2 years following natural exposure.

The results indicate that the buffalo is the only wildlife species in Kenya so far recognized as likely to be involved in the persistence and transmission of FMD. Although clinical disease has been reported (Young et al. 1972) it has not been observed in Kenya, Botswana, or Zimbabwe (Hedger 1972; Condy and Hedger 1974; Falconer and Child 1975) and must be considered to be unusual. Consequently the buffalo is unlikely to be a major source of virus, but it will be a persistent source. This has been demonstrated by the isolation of Sat 3 in buffalo in Zimbabwe 15 years after the last recorded case attributable to this serotype in cattle (Hedger 1976).

One must therefore consider if the carrier buffalo can transmit the disease to cattle. In an attempt to answer this question a Sat 2 carrier buffalo was housed with two susceptible grade cattle in a loose-box measuring 6 x 4 m for a period of 25 weeks while a Sat 1 carrier was housed with two susceptible cattle for 5 months.

There was no evidence of transmission in either case. These results must be interpreted with caution as similar attempts at cattle-to-cattle transmission with cattle carriers have also failed. There is some circumstantial evidence of transmission of virus from buffalo to cattle in the field (Condy 1971) as there is of cattle-to-cattle transmission (Anderson et al. 1978).

Buffalo are numerous and widespread throughout Kenya and the incidence of carrier buffalo is undoubtedly high. The virus of FMD circulates readily within the buffalo population because of their gregarious habits. For them to transmit disease to domestic animals, close contact between the species would be necessary to allow airborne transmission to occur.


Hyslop, N. St.G. 1975. Airborne infection with the virus of FMD. J. Comp. Path. 75, 119.


