

HEARTWATER CARRIER STATUS IN WILD AFRICAN RUMINANTS

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*Il y a longtemps que l'on suspecte le rôle des ruminants sauvages dans l'épidémiologie de la cowdriose, maladie aiguë transmise par les tiques et due à *Cowdria ruminantium*, une rickettsie. Cependant ceci n'a été démontré que pour peu d'espèces. Cet article présente des résultats expérimentaux pour 4 espèces : l'eland du Cap (*Taurotragus oryx*), la girafe (*Giraffa camelopardalis*), le grand kudu (*T. strepsiceros*) et le gnou bleu (*Connochaetes taurinus*). Pour chaque espèce, on a reproduit une infection expérimentale et l'état de porteur a été démontré par transmission à des animaux sensibles avec la tique *Amblyomma hebraicum*. Ces espèces peuvent donc jouer le rôle de réservoir. Leur introduction dans d'autres pays doit se faire avec prudence.*

INTRODUCTION

The role of wildlife in heartwater epidemiology, while generally believed to be important, has never been clearly defined. Though as many as 28 wild species have been implicated as hosts of *C. ruminantium*, the rickettsial agent of heartwater in domestic ruminants that is transmitted by *Amblyomma* ticks, very few have been conclusively proven to be susceptible to infection, mainly due to inadequate diagnosis of infection. Experimental studies have demonstrated susceptibility in blesbuck (*Damaliscus albifrons*) (Neitz, 1935; 1937), African buffalo (*Syncerus caffer*) (Andrew and Norval, 1989), black wildebeest (*Connochaetes gnu*) (Neitz, 1935; du Plessis, cited in Oberem and Bezuidenhout, 1987), and white-tailed deer (*Odocoileus virginianus*) (Dardiri et al., 1987). Natural cases of heartwater, in which *C. ruminantium*-like inclusions have been demonstrated in brain smears prepared at post mortem, have been reported in eland (Young and Basson, 1973), sitatunga (*Tragelaphus spekei*) (Okoh et al., 1986), Java deer (*Cervus timorensis russa*) (Poudelet et al., 1982), springbuck (*Antidorcas marsupialis*) (Neitz, 1944), steenbok (*Raphicerus campestris*) (Jackson and Andrew, 1994), elephant (*Loxodonta africana*) (Okewole et al., 1993), water buffalo (*Bubalus bubalis*) (Mohan, 1968), Indian spotted deer (*Axis axis*) (Oyejide and Olaleye, 1984), and the Kafue lechwe (*Kobus lechwe kafuensis*) (Pandey et al., 1992). A PCR assay based on the *C. ruminantium* MAP1 gene (van Vliet et al., 1994) has also detected *C. ruminantium* DNA in bone marrow from asymptomatic, wild tsessebe (*Damaliscus lunatus*), waterbuck (*Kobus ellipsiprymnus*) and impala (Kock et al., 1995). However, diagnosis of field cases may be compromised by the existence of unknown conditions or agents producing similar pathology, thus experimental trials using defined infective material is the most conclusive method of determining susceptibility.

The increase in wildlife conservation efforts worldwide, which is often associated with the translocation of wild species from heartwater-endemic to heartwater-free areas, provides ample opportunity for the spread of the disease and its vectors into the new regions, such as the American mainland where competent vectors already exist (Barre et al., 1987). Identification of potential wildlife reservoir species is therefore an essential pre-requisite to the development of sound regulatory procedures for the translocation and importation of animals. In this paper we describe studies into the susceptibility to *C. ruminantium* infection of four species of African wild ruminants, namely eland (*Taurotragus oryx*), giraffe (*Giraffa camelopardalis*), kudu (*Tragelaphus strepsiceros strepsiceros*) and blue wildebeest (*Connochaetes taurinus*).

MATERIALS AND METHODS

Four eland, four wildebeest, five kudu, obtained from a heartwater-free area of Zimbabwe, and two giraffe from a heartwater-endemic area were exposed to experimental *C. ruminantium* infection. Three of the eland, kudu and wildebeest, and one of the giraffe were inoculated with approximately 10^8 live *C. ruminantium* organisms derived from bovine endothelial cell culture. This dose was lethal to simultaneously infected small ruminants. The remaining animals of each species were each exposed to infection by the feeding 20 infected *A. hebraicum* adult male ticks in cloth bags attached to their backs. The male ticks had been previously infected at the nymphal stage by feeding on a sheep experimentally infected with *C. ruminantium*. The infection rates of these tick batches were determined by PCR analysis (Peter et al., 1995) to be approximately 95%, and ticks from the same batches successfully transmitted infection to small ruminants.

To detect infection in the wildlife, unfed, uninfected *A. hebraicum* nymphs or adult males were allowed to feed in cloth bags on the backs of the animals at intervals after infection. After one week of feeding the adults were detached and transferred to susceptible small ruminants to test for the transmission of heartwater. Similarly, the engorged nymphs were collected, allowed to moult to the adult stage and then fed on small ruminants. The recipient animals were monitored daily for clinical signs and heartwater was confirmed by examination of brain smears prepared by biopsy during the febrile reaction or at post mortem. Serum was also collected from the wildlife prior to and at 2-3 weeks after infection. The sera was analysed by immunoblot as described previously (Mahan et al., 1993).

RESULTS

No visible signs of clinical illness were observed in any of the wildlife after exposure to infection. Ticks fed on the eland and the wildebeest at 15 and 128 days after infection transmitted heartwater to goats. *C. ruminantium* was

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also transmitted from the giraffe at 85 days post infection, and from kudu at 24 days post infection. Transmission of *C. ruminantium* from animals infected by ticks was demonstrated only in one of the kudu and one giraffe; however serological analysis indicated that *C. ruminantium* infection was established in all of the eland and wildebeest, and at least two of the five kudu. Both giraffe were seropositive prior to experimental infection, possibly due to earlier *C. ruminantium* exposure.

DISCUSSION

This study brings to eight the number of wild species that have been proven experimentally to be susceptible to *C. ruminantium* infection. Seven of these species can become carriers of infection after recovery, highlighting the significant role that these species, and potentially other wildlife, play in heartwater epidemiology. The ability of species tested here to be infected by ticks, and transmit infection to ticks is epidemiologically relevant as they are important natural hosts for *Amblyomma* ticks within heartwater-endemic regions of Africa. Previously, blesbuck (Neitz, 1937) and buffalo (Andrew and Norval, 1989) were the only wild species known to capable of infecting the vector. The absence of clear clinical signs of heartwater after infection suggests that certain wildlife may be innately resistant to clinical disease, possibly as a result of long term genetic selection.

The existence of a reservoir of infection in wildlife will make heartwater difficult to control or eradicate even under situations of intensive acaricide treatment of domestic livestock. In addition, the movement of infected wild animals raises the risk of infection of new areas and the introduction of vectors. Translocations of potentially infected animals over long distances into heartwater-free areas, for example the United States, should be carefully screened and measures taken to prevent infection of the tick population, if vectors are known to occur locally.

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