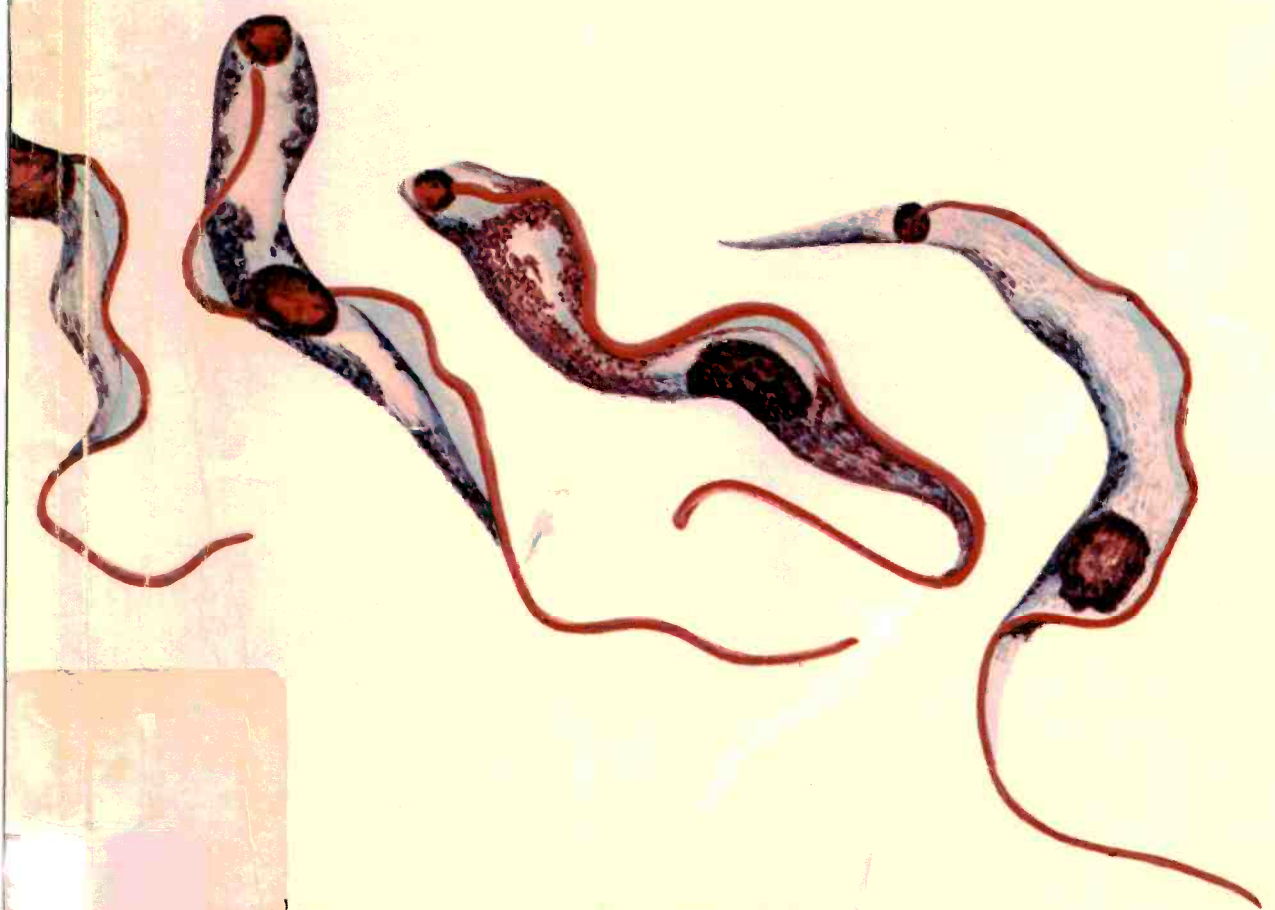
IDRC-132e

Pathogenicity of Trypanosomes

Proceedings of a workshop held at Nairobi, Kenya, 20-23 November 1978



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Editors: **George Losos[1] and Amy Chouinard[2]**

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/IDRC publication/. Compilation of workshop papers on /trypanosomiasis/ particularly in /Africa south of Sahara/ - discusses the /metabolism/ of the trypanosome /parasite/s, mechanisms of /disease transmission/, effects on /blood/ and /serum/ /protein/ levels in /cattle/, /immunology/cal aspects, /disease resistance/.

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Contents

Trypanosomiasis of game animals

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Abstract. Wild mammals are sometimes said to be "trypanotolerant," i.e., they harbour trypanosomes but do not develop the clinical signs of trypanosomiasis. Although there are many studies that confirm the presence of trypanosomes in the blood of game animals, there are few, if any, that examine the pathological effects. At Kabete, we at the Veterinary Research Laboratory have begun studies to monitor the pathogenesis of the trypanosomiasis in animals that are trypanotolerant. Our preliminary results indicate that the animals act as reservoirs for the trypanosomes but do not suffer any adverse effects. More complete information will be forthcoming from game animals that we have raised and kept trypanosome-free.

Wild animals are usually said to be resistant to trypanosomiasis but highly susceptible to infection. In other words, the host–parasite association has evolved naturally so that susceptible wild hosts usually are free of the normal signs of disease. Healthy wild mammals may serve as reservoirs for trypanosomes and yet be resistant to trypanosomiasis.

In this paper, "resistance" means resistant to disease, not infection. Trypanotolerant is another word for the same concept; therefore, some wild animals are trypanotolerant. They are probably not tolerant to trypanosome infection in the immunologic sense because antibodies are found in their blood (Drager and Mehlitz 1978).

There are many reports showing trypanosomes in the blood and tissues of healthy game animals (Burridge et al. 1970; Ashcroft, Burtt, and Fairbairn 1959; Lumsden 1962; Hoare 1972); some of the authors have demonstrated histopathological changes in game animals infected with trypanosomes (Losos and Gwamaka 1973). Laboratory and field studies to date do not prove that game animals are resistant to trypanosomiasis; in fact, a percentage, and at times a high percentage, become diseased but are eliminated by predators.

The literature on infection and pathogenicity of trypanosomes in game animals is quite limited, although there are many reports demonstrating trypanosomes in the blood and tissues of wild animals. McCulloch (1967) found trypanosomes of the *brucei* subgroup in the blood smears of two zebra (*Equus burcheli*). The zebra had lost weight, and McCulloch considered *T. brucei* to be a contributory factor. He found trypanosomes in the blood and brain smears of the zebra but did not employ any other diagnostic procedures, e.g., to detect bacteria viruses or tissue abnormalities, to rule out the presence of other pathogens.

Wild animals as trypanosome carriers have been investigated by various workers. Ashcroft, Burtt, and Fairbairn (1959) divided game animals into two groups on the basis of susceptibility to trypanosomiasis. Group one comprised animals usually killed by *T. rhodesiense* and *T. brucei* infections, including Thomson's gazelle, dik-dik, blue forest duiker, jackal, fox, ant bear, hyrax, serval, and monkey. Group two consisted of animals usually resistant or "tolerant to infection," including the warthog, bush-pig, porcupine, and baboon. The authors were dealing with *T. rhodesiense* and *T. brucei*, and the results may have been different for strains of *T. vivax* and *T. congolense*, which are usually very pathogenic for cattle. While examining wild animals as a potential reservoir for *T. rhodesiense*, Geigy, Mwambu, and Kauffmann (1971) isolated 12 strains of the *T. brucei* subgroup: 2 from hyena, 5 from lion, 1 from warthog, 1 from waterbuck, and 3 from hartebeest. These strains were isolated from clinically healthy animals and their pathogenicity was not discussed.

There is some evidence that trypanosomes cause lesions in game animals and even kill them. For example, Losos and Gwamaka (1973) carried out a histological examination of wild animals described by Geigy, Mwambu, and Kauffmann (1971). Significant histological lesions were found in two impalas, one Thomson's gazelle, three Coke's

hartebeest, and in two lions. The lesions, which were attributed to trypanosomes, were myocarditis and meningoencephalitis and were consistent with the observations made in domestic animals (Losos and Ikede 1972).

Trypanosome infections in wild animals may be influenced by the distribution of the tsetse fly vectors. Weitz and Glasgow (1956) showed that tsetse flies preferentially feed on certain game species, avoiding hartebeest, zebra, or topi and, in the case of *G. morsitans, G. swynnertoni*, and *G. austeni*, seeking blood from *suidae*. Thus, pigs may have played an important role in maintaining a variety of tsetse flies in the area studied.

Game animals play a significant role in the epidemiology of human trypanosomiasis because they act as reservoirs for trypanosomes. Heisch, McMahon, and Manson-Bahr (1958) isolated *T. rhodesiense* from a naturally infected bushbuck, which on inoculation into humans produced clinical disease.

As in domestic animals, trypanosomes in wild animals may localize in tissues, and hematologic examination for parasites is not sufficient to rule out their presence (Lumsden 1962). On the other hand, finding trypanosomes in the blood is an indication of infection but not necessarily of disease.

Experimental Studies

In the last few years, personnel at the Wildlife Disease Section, Kabete, isolated trypanosomes from eland and began a study of pathogenicity (Karstad, Grootenhuis, and Drevemo 1974). My work is a continuation of their work. We have encountered one major problem: finding susceptible, trypanosome-free wild animals. To solve this problem, we have bred a few species in captivity and now have offspring of eland, buffalo, waterbuck, wildebeest, and bushbuck. Using a syringe (mechanical), we have transmitted trypanosomes from game animals to domestic animals and from free-living game animals to captive wild animals both in the field and in the laboratory. In the field, game animals were drug immobilized; blood was taken from their jugular or peripheral veins and inoculated into mice and goats to make isolates. Trypanosomes were detected using the standard trypanosome detection methods and were stored in liquid nitrogen for further investigation in the laboratory.

Two eland heifers born in captivity were inoculated with the sixth mouse-passage of a strain of *T. congolense* isolated from a captive eland in the Kiboko area. They developed parasitemia of plus two (+ 2) (up to 10 trypanosomes observed per field) 7 days post inoculation. The animals were under observation for a period of 90 days. During this period there were no significant changes in blood parameters, and their temperatures remained within normal limits. They eventually eliminated the trypanosomes from their bodies and are now clinically healthy. We did not determine the impact of the trypanosomes on their tissues, organs, etc. because, at the time, we had very few experimental animals and could not afford to sacrifice any of them. In future, we plan to examine the immunologic responses also.

Discussion and Conclusions

The experimental eland heifers were highly susceptible to trypanosome infection but did not seem to be affected by the disease. It is possible that the strain of *T. congolense* had become adapted to eland and was not pathogenic for them. It is also possible that the *T. congolense* lost its pathogenicity in the passages through mice (six passages); however, our observations are similar to those reported by Karstad, Grootenhuis, and Drevemo (1974) who inoculated *T. congolense* into captive eland. In that study, the eland did not show any adverse effects despite continuous parasitemia for 2 1/2 months.

Our field and experimental data on game animals have revealed that buffalo and giraffe are the main reservoirs of *T. congolense* at Kiboko. Captive eland in a tsetse-infested paddock were found to be infected with trypanosomes, but several free-living eland were not. Also, repeatedly, clinically healthy buffalo have exhibited *T. vivax* infections, isolates of which have been very pathogenic for cattle.

There are a number of problems that emerge in the study of game animal trypanosomiasis. The ideal situation would be to conduct this kind of study in game animals not subjected to confinement, as capture and confinement subject game animals to abnormal stresses. The drug immobilization and the stresses of captivity probably influence the development of disease in the animals, and it is hoped that breeding and raising them in a tsetse-free environment will provide more valid observations.

Although game animals have been shown to be reservoirs of trypanosomes pathogenic to our livestock, the pathogenicity of the trypanosomes to game animals themselves has not been sufficiently investigated. The challenge that faces immunologists at the moment is to analyze the immune responses of game animals and to extract information that can be used to save the lives of millions of domestic animals.