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Chemotherapy of Trypanosomiasis Research Project,
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Failure of *Trypanosoma (Nannomonas) simiae* to infect camels (*Camelus dromedarius*)

Short communication

E. ZWEYGARTH, D. RÖTTCHER, D. SCHILLINGER

Trypanosoma (N.) simiae has been found in the blood of dromedaries (Di Domizio, 1929) where it causes fatal infections (Pellegrini, 1948). The second member of the *Nannomonas* subgenus, *T. (N.) congolense*, causes an acute disease in camels with a very high mortality (Leese, 1927; Bennett, 1933; Härter et al., 1985). Morphologically both parasites are similar and may cause confusion. The natural reservoir of *T. (N.) simiae* are wild suids (warthog and bush-pig). They share the environment with camels in Africa and are a common species especially in Islamic countries where they are not hunted. They live not far from watering points which camels visit and are an important food source for biting flies such as tabanids and tsetse flies. However, in tsetse-infested areas wherever we investigated tsetse-borne camel trypanosomiasis, it was invariably caused by *T. (N.) congolense* or *T. (Trypanozoon) brucei brucei*. We therefore decided to re-investigate the role of *T. (N.) simiae* in camels.

For the experiments 3 male camels (*Camelus dromedarius*) were available. They had been purchased from Rumuruti/Laikipia District (Kenya) and were stabled at the Veterinary Research Laboratory Kabete. The camels were fed with commercially available cubes and with hay; water and salt lick were provided ad libitum. The animals were checked repeatedly for the absence of trypanosomes by parasitological and serological tests for at least four weeks prior to the experiments.

Two different stocks of *T. (N.) simiae* were used for experimental infections. The KETRI 2431 stock derives from the EATRO 1875, which was isolated from a *Glossina austeni* in Ukunda (Kenya) in 1970. A pig infected with this stock dies within 3–6 days, after trypanosomes have been found in the wet blood film. The second stock, CP 1916, was isolated from a bush pig in April 1985 in Ukunda (Kenya), this produces a more chronic infection in pigs. Both stocks

were non-infective for mice. Goats could be infected but self-cured after about 2 months. Stock KETRI 2431 is distinct from *T. (N.) congolense* with respect to its isoenzyme pattern (Dr. R. Brun, Swiss Tropical Institute, Basel, personal communication).

The camels were each inoculated subcutaneously with 5×10^7 organisms which were obtained minutes earlier from infected pigs by venopuncture. The wet film technique, and the haematocrit centrifuge technique (Woo, 1969) were applied for the examination for parasites. The body temperature was monitored daily.

In none of our camels we could detect trypanosomes nor any sign of a trypanosome infection, such as a rise in body temperature or a drop in the packed cell volume. Both stocks did not even cause a transient parasitaemia. After infection with the first stock, the animals were checked for 3 weeks. They were then inoculated with the second stock at a similar dose. As a final examination after both experiments, a pig was inoculated with a pooled blood sample from all the camels, but it did not become positive.

The behaviour of *T. (N.) simiae* in different animal species has been used to differentiate between the two members of the subgenus *Nannomonas*. However, this is not necessarily a valid criterion to distinguish *T. (N.) simiae* from *T. (N.) congolense* since a variety of different susceptibilities in the same species have been reported (Stephen, 1966). Recently, Gashumba et al. (1986) compared *T. (N.) simiae*, *T. (N.) congolense* and *T. (Trypanozoon) brucei brucei* strains by isoenzyme patterns and found that *T. (N.) simiae* is distinct from the others.

From our experiments it seems very unlikely that *T. (N.) simiae* is pathogenic for camels, at least as far as our stocks are concerned.

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