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Trypanosomiasis in the black rhinoceros (*Diceros bicornis* Linnaeus, 1758)

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Summary: *A black rhinoceros* (Diceros bicornis) *moved from a tsetse-free to a tsetse-infested area in Kenya was monitored for two months following translocation. The animal acquired a* Trypanosoma vivax *infection from natural tsetse challenge, but survived without requiring treatment with trypanocides. The infection was characterised by moderately high parasitaemia, with symptoms of anaemia, leukopaenia and thrombocytopaenia. Although confirmed to be* T. vivax *through deoxyribonucleic acid hybridisation and parasite development in tsetse in the proboscis only, the parasite had unusual morphology and motility. It also failed to infect normally susceptible hosts such as cows and goats, and produced unusually low infection rates in* Glossina morsitans centralis *and* G. brevipalpis.

KEYWORDS: Disease – Glossina – Trypanosoma – Tsetse fly – Wildlife translocations – Xenodiagnosis.

INTRODUCTION

Following massive declines in numbers of rhinoceros in Africa, many populations are now being managed in order to prevent further losses. In Kenya and Zimbabwe, one strategy has been to move animals from insecure (mostly lowland) areas infested with tsetse flies (*Glossina* spp.) to safe (mostly highland) areas free of tsetse. When animals are moved in this way, they may acquire trypanosomiasis, a disease caused by the protozoan *Trypanosoma* and spread by the bite of tsetse flies.

Although not evident from surveys of wildlife (1, 4), veterinarians have often noted trypanosome infections in black rhinoceros (*Diceros bicornis* Linnaeus, 1758) and white rhinoceros (*Ceratotherium simum*). Chronic trypanosome-related health problems, including abortions, arose in white rhinoceros when animals of this species were introduced into Meru National Park in Kenya (E.C. Goss and D. Röttcher, personal communications). Similar problems appear to have arisen in other countries, but are poorly documented. In black rhinoceros, problems arising from trypanosomiasis were first noted in a capture and translocation operation in Tanzania in the early 1960s (6). Some *Trypanosoma brucei* and *T. vivax* infections became severe when rhinoceros were

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held in captivity, resulting in deaths. These observations were confirmed by Clausen during a study of 39 rhinoceros captured between 1968 and 1970 (3). *T. brucei* was detected in 18% of the rhinoceros (all sub-adults) and antibodies were found in most of the animals. In both cases, problems were encountered in treating infections with trypanocides.

These early studies, conducted when capture methods were being developed, should be interpreted with caution. Nevertheless, they suggest that rhinoceros already carrying trypanosome infections are susceptible to trypanosomiasis when stressed by capture, captivity and translocation. Hence, the acquisition of infections in immunologically naive rhinoceros which are translocated to tsetse-infested areas may also result in stress-related disease problems. The authors therefore began monitoring rhinoceros in Kenya which are moved from a tsetse-free area (Nairobi National Park) to a tsetse-infested area (Ngulia Rhino Sanctuary, Tsavo West National Park) to observe how the acquisition of infection would affect the health of the animals.

To date, details of two translocations have been reported (7). The first rhinoceros, upon being moved to an area with high numbers of tsetse, acquired a *T. brucei* infection which appeared to have contributed to its death. The second animal acquired a *T. congolense* infection when moved to an area with a low density of tsetse flies, but suffered no apparent health effects.

MATERIALS AND METHODS

Details are given here of a third translocation involving a ten-year-old male black rhinoceros captured in Nairobi Park and moved to Ngulia on 7 May 1991. The animal was held in a pen for eight weeks and monitored bi-weekly by haematology, parasitology and xenodiagnosis. Methods were similar to those reported previously (7), except that a greater variety of animals were injected with blood from the rhinoceros, and three species of tsetse fly (*G. morsitans centralis*, *G. brevipalpis* and *G. longipennis*) were used for xenodiagnosis on all occasions.

RESULTS

Data gathered at capture and at week 2 indicated that the rhinoceros was free of trypanosomes. Infection status at week 4 was equivocal, due to a technical problem. At weeks 6 and 8, wet films revealed an infection consisting of large, sluggish trypanosomes with a free flagellum and many dividing forms. When examined a few hours later, the parasite exhibited the vigorous motility of *T. vivax*, with fewer dividing forms. In thin smears, the parasite appeared fatter and more club-shaped than *T. vivax* in bovids, with a small kinetoplast which was marginal and subterminal.

Parasite identity was confirmed by deoxyribonucleic acid (DNA) characterisation, development in tsetse and failure to grow in rodents. Dot blots hybridised only with a DNA probe for East African *T. vivax*. The parasite produced

infections in tsetse in the proboscis only (17% *G. m. centralis*, 12% *G. brevipalpis*). Although *G. longipennis* prefers to feed on rhinoceros (10), in the present study it showed reluctance to feed. On two occasions, when the rhinoceros was well immobilised, only 37% of the *G. longipennis* fed (n = 76), compared to 50% of the *G. m. centralis* (n = 184), and 74% of the *G. brevipalpis* (n = 78). Surprisingly, the parasite failed to grow in rabbits, cows and goats, and did not survive in a tsetse proboscis culture system for *T. vivax*.

At capture and at week 2, erythrocyte indices and platelet counts were within normal ranges (5). In Ngulia, leukocyte counts were half of those measured at capture, mostly due to lymphopaenia. Lymphocytes reached a low of 1.1×10^9/l at week 6 compared to 4.4×10^9/l at capture. At week 4, the rhinoceros was anaemic (packed cell volume of 31% vs 47% at capture) and thrombocytopaenic (platelet count of 108×10^9/l vs 500×10^9/l). Anaemia was not associated with haemolysis (2), nor with the presence of other haemoparasites. A large increase in erythrocyte size occurred at week 4, presumably reflecting increased erythropoiesis (median 93.0 fl vs 81.5 fl at capture, measured values).

At week 6, the infection reached high parasitaemia (6×10^8/l). This was accompanied by recovery in erythrocyte indices (packed cell volume of 43%), but continuing low lymphocyte (1.1×10^9/l) and platelet (80×10^9/l) counts. At week 8, the clinical picture remained depressed with persistent high parasitaemia (10^8/l). The animal was released without treatment and was still alive in December 1992.

DISCUSSION

These recent results confirm previous reports of trypanosomiasis in stressed rhinoceros (3, 6). Rhinoceros can apparently develop the same disease symptoms found in other animals (anaemia, leukopaenia, thrombocytopaenia), despite their ability to thrive in areas with tsetse. The rhinoceros studied in the present experiment was able to cope with infection under minimal veterinary care. In other circumstances, infection could be life-threatening, particularly for animals exposed to multiple stresses when released shortly after capture. The stress of immobilisation and transport, the change in diet and surroundings, the acquisition of tick-borne parasites (e.g. *Babesia*, *Theileria*), the change in social status, etc. could all modify the ability of rhinoceros to cope with infections. Given these potential hazards, it is recommended that animals moved to tsetse-infested areas be monitored for one to two months prior to release. This would allow for intervention with trypanocides if necessary.

Another important finding is the atypical nature of the parasite causing anaemia in this rhinoceros. Although tentatively identified as *T. vivax*, it displayed unusual morphology and failed to infect bovid hosts. The rate of transmission to tsetse was also unusually low. These features suggest that isolation and characterisation of rhinoceros parasites may require special efforts. Similar problems were experienced by Clausen, who isolated unusual *T. brucei* parasites (3). It is therefore recommended that appropriate perissodactyl hosts (e.g. donkeys, horses) be used for research on rhinoceros parasites. This would allow the development of techniques for testing trypanocides to supplement the meagre literature (8, 9).

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TRYPANOSOMOSE DU RHINOCÉROS NOIR (*DICEROS BICORNIS* LINNAEUS, 1758). – S. Mihok, R.O. Olubayo et S.K. Moloo.

Résumé *: Un rhinocéros noir* (Diceros bicornis) *a été observé pendant deux mois après avoir été transféré d'une zone indemne de trypanosomose vers une zone infestée, à l'intérieur du Kenya. L'exposition naturelle aux glossines a provoqué une infection par* Trypanosoma vivax, *mais l'animal a survécu sans que son état n'ait nécessité l'administration de trypanocides. L'infection s'est caractérisée par une parasitémie modérée accompagnée de symptômes d'anémie, de leucopénie et de thrombocytopénie. Bien que l'appartenance du parasite à l'espèce* T. vivax *ait été confirmée par hybridation de l'acide désoxyribonucléique et par un développement limité à la trompe de la glossine, sa morphologie et sa motilité étaient inhabituelles. De même, les hôtes qui y sont normalement sensibles (bovins et caprins) n'ont pas été infestés et les taux d'infestation ont été anormalement faibles chez* Glossina morsitans centralis *et* G. brevipalpis.

MOTS-CLÉS : Déplacements de la faune sauvage – Glossine – Maladie – Mouche tsé-tsé – Trypanosome – Xénodiagnostic.

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TRIPANOSOMIASIS DEL RINOCERONTE NEGRO (*DICEROS BICORNIS* LINNAEUS, 1758). – S. Mihok, R.O. Olubayo y S.K. Moloo.

Resumen*: Un rinoceronte negro* (Diceros bicornis) *fue observado durante dos meses tras haber sido transferido de una zona sin tripanosomiasis a una zona infestada, en el interior de Kenia. La exposición natural a las glosinas provocó su infección por* Trypanosoma vivax, *pero sobrevivió sin haber necesitado que se le administraran tripanocidas. La infección se caracterizó por una parasitemia moderada acompañada de síntomas de anemia, de leucopenia y de trombocitopenia. Si bien se confirmó la pertenencia del parásito a la especie* T. vivax *por hibridación del ácido desoxirribonucleico y por un desarrollo limitado a la trompa de la glosina, su morfología y su motilidad no eran las habituales. Por otra parte, los huéspedes normalmente sensibles (bovinos y caprinos) no fueron infectados y las tasas de infección fueron anormalmente bajas en* Glossina morsitans centralis *y* G. brevipalpis.

PALABRAS CLAVE: Desplazamientos de fauna salvaje – Enfermedad – Glosina – Mosca tse-tsé – Tripanosoma – Xenodiagnóstico.

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