HEALTH IMPLICATIONS OF TRANSLOCATIONS OF ENDANGERED SPECIES IN AFRICA: TRYPANOSOMIASIS IN RHINOCEROS

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Areas such as Tsavo National Park in Kenya that once harboured many black rhinoceros are now being restocked with surplus animals from a variety of sanctuaries established in the 1980's. Many of the roughly 400 black rhinoceros remaining in Kenya are presently living in sanctuaries in the highlands where tsetse flies are not present; in contrast, areas receiving rhinos are mostly in the lowlands where tsetse flies and trypanosomiasis are prevalent. The disease implications of these movements have been studied since 1989 following concerns resulting from the death of a translocated rhino at Ngulia in Tsavo West National Park. The rhino may have died as a result of complications arising from the acquisition of a *Trypanosoma brucei* infection. This case prompted detailed studies of tsetse and trypanosome epizootiology at Ngulia in conjunction with health monitoring of translocated rhino. Initial epidemiological surveys indicated this animal was moved to an area with a high tsetse density at a time of the year when both density and infection rates in flies were at a peak. Subsequent introductions were therefore planned to avoid tsetse challenge as much as possible by relocating the bomas to a more appropriate area. Within logistical constraints relating to rainfall and availability of green vegetation for feeding animals during confinement, subsequent translocations have been undertaken to minimise disease risks. This strategy has resulted in the acquisition of very few patent infections and no further deaths. To date rhinos have survived infections of *T. congolense, T. vivax* and a new genotype of *Nannomonas* parasite without the need for chemotherapy.

Although serious problems have not occurred, hematological data have revealed indications of stress in terms of moderate anemia, depressed lymphocyte and platelet counts and elevated neutrophil counts a few weeks after translocation. These effects have occurred mostly in animals moved long distances in highland to lowland translocations. Changes in blood parameters have also been pronounced in the few animals that acquired trypanosome infections, but it has been difficult to attribute causes to any single factor. Diagnostic information from antigen-ELISA tests for trypanosome antigens have also complicated a simple interpretation of conventional parasitological data. These results have suggested most animals harbour cryptic infections, particularly of *T. brucei,* and hence current routine nostic techniques only pick up parasites during an acute phase of parasitaemia.

Recently, we have also started to monitor trypanosomiasis in white rhinoceros. A small number of animals were moved from South Africa to Kenya in the early 1970's to two areas (Solio Ranch - without tsetse) and Meru National Park (with tsetse). The Meru animals did poorly. The Solio animals thrived. To test the effects of trypanosome infection on white rhino, two Kenyan-bora animals were moved from an area without tsetse to the Maasai Mara (with tsetse) in 1992. Challenge was minimised on introduction by reducing tsetse numbers by about 80% with odour-baited traps near the bomas where the animals were confined for a few months. The rhinos were then allowed to roam freely. At six months, they were both diagnosed with chronic *T. brucei* infections accompanied by mild anemia. After two years, they were still alive, apparently healthy, and had self-cured any infections. After this initial positive result, ten white rhinos were introduced directly to the Mara from South Africa in September 1994.

Unfortunately, tsetse densities increased dramatically shortly after introduction due to heavy rainfall. A few months later nearly all animals were diagnosed with active or chronic *T. brucei* infections. One animal died after a few months and another had to be treated with trypanocides. Other animals moved at the same time to Nakuru National Park (without tsetse) fared well. Although the interpretation and outcomes of these two introductions from South Africa are not final, it is clear that the white rhino is susceptible to trypanosomiasis at the time of translocation. Also, it is clear that the white rhino is a particularly good wildlife host for *T. brucei.* These results have serious implications for management plans for moving white rhinos into and out of areas with human sleeping sickness (such as the Serengeti-Mara ecosystem). Since we know very little about the efficacy of trypanocides in both black and white rhino, there are real dangers of inadvertently introducing human-infective forms of *T. brucei* to new areas when animals are moved.