

# The Unsuccessful Introduction of White Rhinoceros to Matusadona National Park, Kariba.

R.D. Taylor

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## INTRODUCTION

The plight of the rhinoceros, both white and black, throughout Africa, is widely known and well documented. Although the conservation status of the southern white rhino (*Ceratotherium simum simum*) is relatively secure, especially in South Africa, in Zimbabwe a small national population of approximately 200 animals requires building up. Apart from Parks and Wild Life land in the Zambezi Valley and the Sebungwe region, there is little habitat elsewhere in Zimbabwe in which to increase the distribution and population size of the species.

There is no clear evidence, as yet, that the white rhinoceros was historically widespread in the Zambezi Valley. However, it is possible that this was so during the previous century and before, particularly above the Zambezi escarpment. Selous (1881), Coryndon (1894) and others record that white rhinoceros were common in Mashonaland up until 1890, and the more recent researches of Roth (1967) and Tomlinson (1977) indicate that the species was previously widespread in the country as a whole. The Zimbabwe Department of National Parks and Wild Life Management supports the re-introduction of species previously known to have occurred in a particular locality, and wherever possible this is encouraged. Indeed, the country's present population of white rhinoceros has been built up since 1962 through introductions from Zululand, following the depletion of the indigenous population shortly after the turn of the century (Davison and Condy, 1963; Tomlinson, 1977).

A major risk to introducing white rhino to the Zambezi Valley and the northern Sebungwe was the possibility of trypanosomiasis infection. An experimental introduction was, therefore, a necessary prelude to any long-term or large-scale introductions. Because Matusadona is well protected, accessible and infested with tsetse fly, this Park was chosen for an initial introduction. White rhino for translocation became available during the widespread drought which ravaged much of the country between 1982 and 1984 (Pitman, 1983). Regrettably the experimental introduction was not conducted as planned and I report here the failure of the exercise.



**Figure 1.** Translocation of white rhinoceros in Zimbabwe from Mushandike Sanctuary and Doddieburn Ranch to Matusadona National Park during 1983.

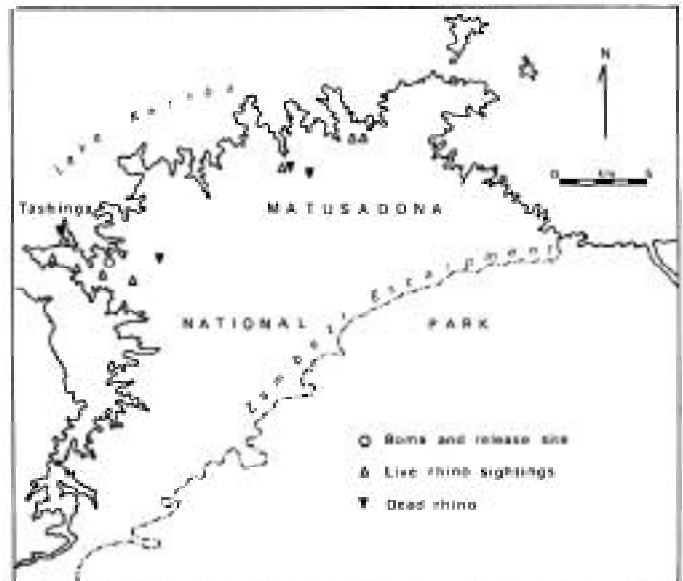
## TRANSLOCATION AND RELEASE

During the latter half of November 1983, two white rhino (one male, one female) were captured at Mushandike Sanctuary near Masvingo and translocated to Matusadona. A further three rhinos (one male and two females) were transferred from Hwange National Park where they had been held since August following their capture on Doddieburn Ranch near West Nicholson (Figure 1). All five rhino travelled in standard rhino translocation crates on trucks and trailers.

At Matusadona, the rhino were unloaded into a holding boma consisting of three pens with water and shade. The boma was constructed on open grassland on the Kariba lakeshore near Tashinga, the Park headquarters (Figure 2). Animals were fed and watered daily, fodder being freshly-cut

*Panicum repens* grass. All rhino settled into the pens fairly readily, especially the three Doddieburn rhino which had become well accustomed to pen life at Hwange.

The two Mushandike rhino were released from the holding boma six days after arrival at Matusadona. The three remaining rhino were held for only two days in the boma before their release as they had been penned for nearly three months already. All five animals left the boma site in different directions, with little fuss or difficulty.



**Figure 2.** The area of Matusadona National Park below the Zambezi Escarpment into which white rhinoceros were released.

## SUBSEQUENT LIVE SIGHTINGS AND MORTALITIES

By the end of November all five rhino had been released and regular sightings were being made in the vicinity of the release site. The first rhino to die was a Doddieburn female who died stuck in mud on the lakeshore. This accidental death gave immediate cause for concern lest a similar fate befall the others. An intensive air search located three of the four remaining rhino all within close proximity of Tashinga. Subsequent sightings indicated that the rhino were beginning to move further afield, as much as 15 km away (Figure 2). Commencement of the rainy season restricted coverage of the Park by staff so that sightings became infrequent.

On 4 February 1984, a female white rhino was seen alive near the Jenje River, but was found dead 36 hours later on 6 February, in almost the same locality. Apart from a prolonged urination, there had been no obvious symptoms of illness when the animal was initially observed on 4 February. The already advanced state of decay prevented the collection of blood or tissue material for pathological examination.

Fears of disease threatening the remaining three animals prompted a search and rescue operation which was mounted over the following four days. Intensive air and ground searches failed to locate any of the rhino. Although the aerial search was abandoned, ground patrols located a further two dead rhino on 17 and 21 February. Both animals had been dead approximately 14 and 10 days respectively, indicating that all three rhino died within days of each other. Although the fifth rhino was never found, it was concluded that the animal had succumbed in similar manner.

### PROBABLE CAUSE OF DEATH

Circumstantial evidence led to the strong possibility of trypanosomiasis infection for the following reasons (C.M. Foggin, pers.comm.).

(i) Matusadona National Park is in a tsetse fly (*Glossina* spp.) infested region. All white rhino in Zimbabwe have hitherto been located in fly-free areas.

(ii) All white rhino in Zimbabwe are ex-Zululand stock which has not been exposed to tsetse fly since 1948. Therefore their tolerance to trypanosomiasis could be expected to be low or absent.

(iii) In East Africa, black rhinoceros (*Diceros bicornis*) tend to be infected with trypanosomes but which only become pathogenic when the animal is stressed (Harthoorn, 1973; Clausen, 1981). Harthoorn (1973) states that black rhino may die of acute trypanosomiasis several days after capture unless treated. Although Berenil (Bayer) (diminazene aceturate) can prevent the stress-induced formation of the disease, Clausen (1981) found that Berenil did not effect complete elimination of the trypanosomes and that, in spite of treatment, one rhinoceros in his study sample died of trypanosomiasis. This East African problem has not, apparently, been experienced in Zimbabwe and remains somewhat anomalous in the context of black rhino capture in this country. However, in a telephone conversation with Dr. C.M. Foggin, the veterinary pathologist who assisted with the Matusadona problem, Dr. A.M. Harthoorn suggested that white rhino would probably be equally susceptible to trypanosomiasis, especially with no previous exposure.

(iv) The three deaths occurred within a relatively short period, some 2-3 months after the rhinos' release and at much the same time. There was no evidence to indicate that internal parasites or malnutrition were responsible. It seems, therefore, that all three animals could have been exposed to an infectious agent at about the same time and died after incubation and morbid periods of similar duration. This fits the theory that trypanosomiasis could have been responsible.

### LESSONS FOR THE FUTURE

In any future such exercises, much more attention should be paid to monitoring the progress of translocated animals than was the case in the Matusadona exercise, especially when being moved into tsetse fly infested areas. Previous releases of white rhino have all been into tsetse tree areas. The following points especially need to be taken into consideration.

(i) A longer holding period is required prior to release. There are numerous records of the disappearance and subsequent death of a number of wild animals following immediate release into new areas. More importantly in the case of white rhino, monitoring any tsetse fly/trypanosomiasis challenge would necessitate keeping animals confined for an extended period.

(ii) Holding pens should be sited in an area where tsetse fly are present, but where the challenge is low, at least initially. In the Matusadona exercise, the pens were situated on open lakeshore grassland where tsetse fly were unlikely. The animals would have encountered tsetse fly only once released, when they moved into

the adjacent woodlands.

(iii) Blood smears should be taken as frequently as is practicable which would require a certain level of pen training. Otherwise the rhino would have to be subjected to further chemical or physical restraint before treatment can be effected if illness occurs.

(iv) Chemotherapy with Berenil should be instituted once trypanosomes are found in any quantity and the animal shows clinical symptoms of disease.

(v) An initial single prophylactic treatment with Samorin just prior to translocation may also be of value. It could then be established whether the drug both protects the rhino and allows it to develop the necessary tolerance to the disease. Confinement and observation would then have to be at least 6 months.

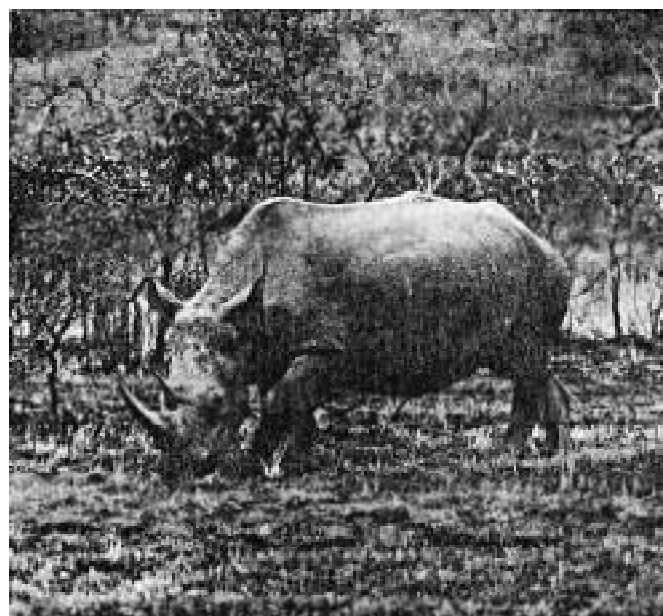
(vi) Some form of marking or tagging animals is necessary so that the rhino can be monitored subsequent to their release and more readily located if need be.

(vii) Finally, the ecological suitability of an area to new introductions should be examined critically. In the case of white rhino introductions into the Zambezi Valley, there is perhaps a need to re-examine very carefully the historical record as to the presence or absence of white rhino in the area. The species may well have been an infrequent visitor on the very edge of its range.

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Southern white rhino. F. Vollmar/WWF