RESEARCH AND REVIEW

First, do no harm: a precautionary recommendation regarding the movement of black rhinos from overseas zoos back to Africa

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Introduction

The international zoological community considers it important to maintain captive animals for a variety of reasons, including the concept that a captive population may serve as a 'safety net' in the face of *in situ* crisis (Foose 1993; Emslie and Brooks 1999), as well as a potential source of animals to re-establish or reinforce wild populations in the future.

With black rhinos managed in the wild as well as in overseas zoological parks, it would seem prudent, if not essential, to develop consensus on when it does and when it does not make conservation sense for animals to be moved from overseas captivity to protected areas in Africa.

In our opinion, moving black rhinos from overseas zoos to free-range settings in Africa can be considered in the following three situations, *but only after both thorough disease risk and cost-benefit analyses have been specifically undertaken when any such movement is proposed.*

1) When introducing new genetic material is deemed necessary for the recipient population's long-term viability, such as for populations with limited founders. This presumes a reasonable knowledge base about the history and genetic diversity of the recipient population and the genetic origins of the animal or animals being considered for reintroduction. This also presumes that the reintroduced animals will breed effectively in the wild setting.

OR

2) If or when the captive population can contribute *biologically significant numbers* to an area below its estimated ecological carrying capacity, or to an area within the species' historic range but currently devoid of black rhinos. The reasons for the absence of endemic animals must obviously have been dealt with before a reintroduction is considered. Through 'compound growth', early introduction of significant numbers of animals, particularly females, can lead to much faster population expansion, particularly in areas well below their ecological carrying capacity. Generally, one would expect that wild sources of rhinos for such supplementation would be fully utilized before overseas zoological sources would be considered.

OR

3) When captive rhino reintroduction techniques need to be studied well in advance of actually applying them on any significant scale. By testing various methods (ideally using animals considered genetically 'surplus' by the community managing captive animals), the techniques may be refined, increasing the chances of successful reintroductions if and when they are actually deemed to be a conservation intervention of choice (see 1, 2 above) in any given locale. It should be noted that intermittent releases of animals that have been hand reared in Africa for a variety of reasons offer opportunities to evaluate different reintroduction techniques.

While these three scenarios seem relatively straightforward, many caveats are buried within the prerequisite *disease risk* and *cost–benefit analyses*—which must be recognized as two discrete processes.

Our primary objectives here are to point out some unique reintroduction risks related to the state of health of black rhinos in captivity and to emphasize that the prudence of management activities with biological risks can be properly assessed only in light of the actual conservation benefits that are likely to accrue because of such activities. A more general yet useful overview is provided by the 'IUCN guidelines for reintroductions' (IUCN 1998), and veterinary information in the IUCN Veterinary Specialist Group's new 'Quarantine and health screening protocols for wildlife prior to translocation and release into the wild' (Woodford in press) may be of interest.

We further recognize that, beyond disease issues, crucial management and behavioural factors have an impact on the success of any rhino reintroduction program. Rhino managers in Africa, dealing with wild animals, not zoo-born rhinos naïve to free-ranging conditions, have learned that for optimal translocation results, large numbers of rhinos (ideally 20 or more) should be moved into an understocked or vacant protected area within a relatively short time. The numbers give any resident animals, particularly dominant bulls, less opportunity to assert themselves and injure or kill newcomers (Brett 1998). Animals translocated individually or in small groups tend to suffer higher mortalities, usually because of encounters with resident males (Brett 1998). Techniques to acclimatize and reintroduce captive-bred rhinos must of course take these issues into account.

We also feel it is important to critically evaluate financial trade-offs when it comes to the relative costs of supplementing range areas with rhinos from overseas zoos versus obtaining animals from other wild populations that are able to provide them. Finally, we note that the animals *being moved* from overseas captivity are of course exposed to an array of risks themselves during any reintroduction process. It is not within the scope of this paper to discuss, for example, the heightened disease risks that the animals being moved from captivity to African protected areas may face when exposed to what for them may be novel pathogens and parasites at their destination. This issue is also associated with various actual and potential costs (Wobeser 1994). Africa's endemic disease-causing organisms, including parasites, will obviously be novel to zoo-born animals. However, black rhinos coming from captive settings will experience disease risks beyond those of their wild counterparts even when faced with micro-organisms that would be, under normal circumstances, unlikely pathogens—if the rhinos carry with them iron loads significantly beyond normal (Weinberg 1974; Payne and Finkelstein 1978; Paglia and Dennis 1999; Weinberg 1999). This issue is discussed below. 'Normal' iron loads are defined as those seen in wild black rhinos that have never spent significant time in captivity.

Disease-risk assessment

Much has been written on disease risk assessment and its implications in conservation decision-making, and we will not review this material here (see, for example, Wolff and Seal 1993; Armstrong and Seal 2000). Three primary sets of skills are needed to ad-

dress disease-risk concerns associated with moving animals for conservation purposes: 1) disease biology as applied to animals being considered for movement and to any recipient populations, 2) data analysis and decision facilitation, and 3) communications for accurately conveying risk-analysis results to decision-makers (Armstrong and Seal 2000). Armstrong and Seal (2000) also recognize that 'a zero risk tolerance philosophy does not meet the needs for decision making in conservation programs' and acknowledge that 'there is not a comprehensive agreed, unified, broadly applicable set of tools such as protocols, models, policies, guidelines to assist assessment of disease risk associated with needed animal movement decisions'.

Anyone who has participated in thorough risk-assessment exercises knows that they are generally time-consuming and somewhat frustrating processes—usually due more to what the participants do *not* know than related to what they do know. In short, disease-risk assessment is often, uncomfortable as it may seem to decision-makers trying to apply the results, a subjective interpretation of available information by the collective expertise consulted. While computerized modelling exercises can be helpful and are always being refined, the bottom line is that in all too many cases there is much we do not know about the range of diseases affecting or potentially affecting the species of interest, particularly in the wild. So we are forced to make decisions in the face of uncertainty in the real world.

Disease and the captive black rhino

This leads us to the 'first, do no harm' precautionary recommendation in the case of the black rhino. Black rhinos in captivity have suffered from an extraordinary range of syndromes that appear related to the fact that they are captive; these conditions are seldom, if ever, observed in the wild. Conditions include acute episodic haemolytic anaemia, chronic non-haemolytic anaemia, superficial necrolytic dermatopathy, haemosiderosis, haemochromatosis, central nervous system degeneration (leukoencephalomalacia), idiopathic and toxic hepatopathies, stress-induced sudden death, a haemorrhagic disease of the microvasculature (idiopathic haemorrhagic vasculopathy syndrome), a noticeable susceptibility to fungal pneumonias and other infectious agents (*Mycobacterium*, *Salmonella*, *Leptospira*), and other as yet incompletely understood disorders (Miller 1993, 1994; Paglia 1994; Lung et al. 1998; Munson et al. 1998; Paglia and Dennis 1999; Murray et al. 2000; Paglia et al. 2001). In the absence of known causes, most of these disorders remain enigmatic, but accumulating evidence suggests that at least some may be related to dietary management (Dierenfeld et al. 1995; Smith et al. 1995). More specifically, these conditions may be sequelae of diet-related iron overloads that develop progressively in captivity and appear to affect only captive black and Sumatran rhinos, which browse, and not captive white or Indian rhinos, which graze (Smith et al. 1995; Paglia and Dennis 1999; Paglia and Radcliffe 2000; Paglia et al. 2001). Further research, which is ongoing, is essential if we are ever going to be able to better address the plethora of biomedical concerns outlined in this paper.

While we believe the basic problem of iron overload in captivity is related to dietary factors, one of the primary consequences of iron overload in humans and quite likely in black rhinos is increased susceptibility to infectious diseases, or increased virulence of micro-organisms of all types in iron-overloaded hosts (Weinberg 1974; Payne and Finkelstein 1978; Paglia and Dennis 1999; Weinberg 1999). In many instances, it appears as if the animals become immunocompromised.

Two disease conditions in black rhinos merit particular attention here: idiopathic haemorrhagic vasculopathy syndrome (IHVS), which may or may not have an infectious aetiology, and tuberculosis—both *Mycobacterium tuberculosis*, the usual agent of human TB, and *Mycobacterium bovis*, so-called bovine tuberculosis or BTB.

IHVS, first reported in 1995, presents clinically as an acute non-haemolytic anaemia owing to extensive haemorrhage into skeletal musculature and subcutaneous tissues, often associated with extensive swelling of soft tissues (for example, neck, shoulders, extremities), respiratory stridor and dyspnea, laminitis, oronasal ulcers and stressful events (Lung et al. 1998; Murray et al. 2000). The clinical characteristics and underlying pathology, a haemorrhagic microvasculitis, are most consistent with an immune-complex disorder of the type often initiated in humans and other mammals by infectious agents such as the streptococci that have been isolated from about half of the affected rhinos. Attempts to detect IgG and IgA immune complexes with rhino-specific reagents have thus far been unsuccessful, but the possibility of an

undetected infectious component remains. All but one of the seven cases described (Murray et al. 2000) occurred in Texas, again perhaps hinting at an infectious agent. Since we do not know what is causing or triggering IHVS, we obviously cannot test for it as part of pre-shipment or quarantine screening protocols, nor can we guess whether 'it' can be transmitted by arthropods or other potential vectors.

Tuberculosis, for which there is as yet no completely reliable antemortem diagnostic test in rhinos, can be caused by several types of mycobacteria, the strains of which may or may not resemble those found in places like South Africa's Kruger National Park in terms of pathogenicity, host predilections, drug-resistance profiles, and so on. While BTB is now present in Kruger Park and several other protected areas in Africa and affects a wide range of wildlife species, to our knowledge no confirmed cases have been reported in wild rhinos.

Meanwhile, bovine as well as human tuberculosis have been reported in African rhinos in overseas zoological parks, as have *M. avium* and other paratuberculous strains (Stetter et al. 1995). These cases have often eluded a variety of diagnostic techniques employed while the animals were still alive. A black rhino with minimal clinical signs other than weight loss, and harboring *M. tuberculosis* as eventually proven by nasal cultures and PCR amplification, had 17 negative cultures over a 12-month period, including one specimen obtained endoscopically, yet recently died with extensive necrotizing granulomatous pneumonia. It is unclear whether this represents an example of enhanced TB virulence in an iron-loaded host or simply reflects a general tendency for rhinos to suppress outward signs of weakness or vulnerability, almost regardless of the extent of underlying disease. The zoological community is now at a heightened state of alert regarding tuberculosis, not only because of cases in rhinos, but perhaps more so because of the number of cases being recognized in captive elephants over the past few years (Mikota et al. 2000). It is also important to note that tuberculosis is a zoonotic disease, meaning it can be transmitted not only among animals but back and forth between animals and people, an obvious public health concern for zoos and the agencies that regulate them (Dalovisio et al. 1992).

Excess iron in captive black rhinos may well be a major factor contributing to enhanced susceptibility to a wide range of infectious agents, all of which are dependent on host iron for replication and metabolism. Recent reviews of iron overload and its relationship to a wide variety of human diseases, including infections, note that nearly 50 microbial genera, including *Mycobacterium,* contain strains that are more pathogenic in iron-loaded hosts (Moyo et al. 1997; Weinberg 1999).

One might ask whether excess iron will dissipate once a captive black rhino is reintroduced to Africa. Unfortunately, mammals lack any effective physiological mechanisms for excreting such excess iron. Judging both by necropsy pathology and by quantitative serum and tissue assays, black rhinos born in or recently brought into captivity have normal iron stores, but these stores begin rising progressively with time in captivity and can increase 10-fold in three years or even less. Longer periods in captivity can result in adult rhinos with iron loads 100-fold or greater than normal.

In the absence of excretory mechanisms, simple interdiction of additional uptake, such as by release into a natural habitat with a presumably normal diet, unfortunately would not affect excessive iron burdens that the animals would carry from captivity into the wild. These burdens could be modified only by active intervention involving physical removal: 1) by pharmacological chelators, which are used in humans with haemochromatosis but which would be prohibitively expensive in rhinos, or 2) by repetitive phlebotomies, or controlled bleedings, which would be practical only in very tractable, tolerant and chute-trained animals. It seems reasonable to conclude that, for a variety of reasons, if there are scenarios in which it makes conservation sense to reintroduce black rhinos from captivity to the wild, younger animals would generally be more sensible candidates than animals that have been captive for many years. The question 'How many years is too many?' merits further analysis.

In short, even if we employ excellent screening and quarantine procedures for both sending and receiving black rhinos for reintroduction, we run the risk of a captive rhino appearing completely healthy and still serving as a Trojan horse for a strain of TB or BTB or another infectious agent not previously seen in rhinos in Africa. Can we quantify this risk? No. Is there less risk if we move captive rhinos into locales within the historic range of the species but currently devoid of endemic rhinos? Yes, particularly over the short term. All of these issues point to the

obvious fact that we must put more thought into analysing *the conservation benefits* of any such proposed reintroduction before it is undertaken. This type of management intervention is not a trivial one. Should we bring zoo black rhinos into *continentally key or important* wild black rhino populations? Given our incomplete understanding of the true nature of many of the disorders affecting captive black rhinos and of the mechanisms involved**,** we would strongly argue that, at the present time, we should not.

Discussion and recommendations

Disease risks should be assessed case by case in light of specific anticipated conservation benefits before any black rhino reintroductions from zoos are undertaken. All other things being equal, younger (within reason) captive animals are in general likely to pose lower disease risks to any recipient population of conspecifics than older animals from captive settings. Hypothetically, it might make sense to move a particular animal from an overseas zoo to a small, isolated reserve for black rhino restoration or as part of a program to develop better captive black rhino reintroduction methods. It would be ill advised to move that same animal into a continentally key or important black rhino population. *Of course, either way, subsequent movements of animals within Africa could result in any given animal from a captive facility, or the pathogens it brought with it and then passed on to vectors or to other conspecifics, or both, eventually ending up somewhere else.* This is an issue that IUCN AfRSG and the SADC Rhino Program should consider as they assemble guidelines regarding the movement of rhinos within Africa.

We acknowledge that the available information regarding disease incidence and prevalence in wild African rhino populations still has many gaps. We recognize, for example, that the bovine tuberculosis already present in ecosystems important to African rhinos remains a concern meriting further research. AfRSG and SADC will of course consider diseases and vectors already known to be present in different parts of Africa as they develop their translocation guidelines. The need for thorough disease risk assessments *before wild rhinos are moved within Africa* is an extremely important topic, but is beyond the scope of this paper.

Cost effectiveness must be assessed for any black rhino supplementation plan. A comparative overview of the cost effectiveness of *in situ* versus *ex situ* black rhino conservation programs, using costs per rhino as a measure, suggests that conservation in the wild has been more cost effective than overseas captive breeding (Currie unpubl.). Add to any overseas zoo-to-Africa black rhino reintroduction project the various costs associated with quarantine on shipping and receiving ends, transportation, and infrastructure and staff time associated with an acclimatization process likely to require months to years for a zoo rhino to successfully adapt to a plethora of 'lifestyle differences' in its new home, and one is faced with the reality that such operations are quite expensive compared with the sourcing of rhinos within Africa whenever biologically and politically possible. (Moves within Africa, however, should of course be accompanied by some of the same precautionary procedures.) Again, this reality emphasizes the need to carefully evaluate the real conservation benefits anticipated from such a zoo-to-wild exercise before proceeding. For the foreseeable future, it does seem likely that supplementing from stock within Africa will generally be lower in cost than importing rhinos from overseas zoos.

The demand for captive animals for restocking wild areas will likely be largely a function of the number of surplus wild black rhinos that are available, which has been limited in recent years. However, there appears to be a growing number of wild populations across several rhino range states wherein population performance seems to have declined as rhino densities have grown closer to estimated longer-term ecological carrying capacities. A number of these populations are showing such classic signs of density-dependent reduction in reproductive performance as long intercalving intervals, low ratios of calves (animals < 3.5 years old) to adult females and lower population growth rates. Given the growing number of potential donor populations in Africa and the fact that improved biological management to maintain high metapopulation growth rates is a key component of most national black rhino management plans, the densities of many of these currently suboptimally performing populations may be actively reduced by capture and translocation as wildlife officials attempt to increase overall reproductive performance. Hopefully, more wild black rhinos will thus become available for restocking.

Finally, while it is not a primary subject of this paper, we want to acknowledge that rhinos in overseas captive facilities have usually been secured

through formal international agreements with the supplying range countries. The legal ownership and disposition issues surrounding wild-caught zoo rhinos as well as their progeny need to be understood and respected as the types of movements discussed in this paper are contemplated.

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