HEALTH DATA GAINED FROM BLACK RHINO IMMOBILIZED FOR RELOCATION

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ABSTRACT

The capture of black rhinoceros (*Diceros bicornis*) for relocation within Zimbabwe, for export, and to a lesser extent capture of black rhino in Namibia, presented opportunities to take blood samples for laboratory testing for antibodies to various diseases, to establish physiologic norms, and for electrophoretic analysis of protein markers of heterozygosity.

Both leptospirosis and vitamin E deficiency have been implicated in the "hemolytic anemia syndrome" deaths of a number of captive black rhino. Vitamin E levels in black rhinoceros we sampled confirmed the reports of Dierenfeld *et al.*, that diets of free-ranging rhino in diverse locations apparently contain considerably higher levels of that vitamin than do diets of captive animals. Its role in "hemolytic anemia syndrome" remains unproven.

A total of 60 rhino captured at 5 locations in Zimbabwe and 3 animals from Namibia were tested for agglutinating antibodies to seven or eight serovars (strains) of leptospirosis. Most rhino from Zimbabwe had titers to several serovars and 38 (63%) had titers greater than 1:100 to at least one serovar. Rhino from Namibia had little evidence of exposure to leptospiras. Microenvironmental differences at water sources in the two locations may explain this observation. Immunization and further testing may offer approaches to reducing the risk of clinical leptospirosis in relocated black rhino.

Relocation of wildlife carries the risk that diseases that threaten livestock and/or man may accidentally also be relocated. Sera were tested for antibodies that suggest previous exposure to African Horse Sickness, Rinderpest, or Foot and Mouth Disease. These samples included the 10 Zimbabwean rhino relocated to North American zoos in 1989 and 2 that went to Germany.

Electrophoretic separation of 7 blood proteins, coding for 12 loci, revealed no heterozygosity, and no differences between the 16 Zimbabwean and 3 Namibian black rhino tested.

INTRODUCTION

The 1986 African Rhinoceros Workshop in Cincinnati identified a number of promising areas for biomedical research that could enhance the health and long term survival of black rhinoceros (*Diceros bicornis*). These included research into diseases in general, hemolytic anemia and hepatic disease, optimal vitamin and mineral levels, and genetics. International Wildlife Veterinary Services recognized that large scale capture and relocation efforts aimed at reducing poaching losses in the Zambezi Valley of Zimbabwe and smaller efforts in Namibia presented the opportunity to take biological samples from free-ranging

black rhino for comparison with captive animals. Individuals and organizations that expressed an interest in obtaining samples from captive black rhino at the Cincinnati workshop were contacted and offered access to samples from free-ranging rhino. In 1989, 65 sets of serum and/or plasma samples were brought into the United States and distributed to a number of researchers associated with zoos, universities and government agencies. The research efforts to which samples were disbursed and a summary of the information gained to date are presented.

MATERIALS, METHODS AND RESULTS

Hemolytic anemia appears to be a fairly common cause of death in captive rhino and its origins are unknown. Red blood cell fragility, red cell parasites, leptospirosis, and vitamin E deficiency had been blamed.

Analysis of plasma by high performance liquid chromatography has proven an accurate method to determine circulating levels of vitamin E in black rhinoceros (Dierenfeld et al., 1988). In previous studies these authors showed that vitamin E levels in free-ranging black rhino from Zimbabwe (0.77 µg/ml) were considerably higher than those seen in captive rhino (0.18 µg/ml). Using the same techniques, Dierenfeld and coworkers analyzed an additional 34 plasma samples from rhino captured for relocation from the Zambezi valley in 1988. The mean vitamin E level was 0.54 µg/ml. In 1989 they analyzed an additional 21 plasma samples from Zimbabwe origin black rhino, most from the Zambezi Valley. The mean vitamin E level was 0.46 µg/ml. Although these means are somewhat lower than their previous published results, they are significantly higher than captive black rhino. The respective mean serum vitamin A level of the 1988 and 1989 samples were 0.04 and 0.05 µg/ml (Dierenfeld, 1989). Three Namibian desert rhino, living on very xeric plant species, had mean plasma vitamin E levels of .80 µg/ml and vitamin A levels of 0.4 µg/ml. As Dierenfeld has suggested, supplementation of captive rhino diets with vitamin E may be in order. The relationship between hemolytic anemia and vitamin E levels is still speculative.

Leptospirosis (Lepto) is known to cause intravascular hemolysis and hepatitis. Lepto has been implicated in hemolytic anemia problems of nine captive black rhino (Jessup *et al.*, 1991). Microscopic agglutination titers for serovars of *L. interogans* were determined at the National Animal Disease Center (NADC) on sera from 60 wild-caught black rhinoceros from five locations in Zimbabwe and three samples from Namibia. This study will be more fully reported elsewhere (Jessup *et al.*, 1991). Briefly, free-ranging black rhino from the Zambezi Valley frequently had relatively high titers to a variety of leptospirosis serovars. Thirty eight of 60 rhino (63%) sampled in Zimbabwe had titers to one or several serovars of leptospirosis of at least 1:100. Titers as high as 1:400 were seen in 9 of 37 (25%) in one group of animals tested. Namibian animals from a desert environment did not have significant titers.

Leptospiras are usually passed between animals via urine contaminated water and they survive best in warm alkaline water and mud. It would seem logical that leptospiras are more common in some moist jungle environments than in desert environments, where the rodent fauna and other potential carriers may account for the differences in prevalence between locations. Leptospirosis may be a naturally occurring disease of black rhino in the Zambezi Valley but clinical cases have not yet been reported. The effects of leptospiras on free-ranging rhino and those destined for relocation need further investigation.

Dr. Mike Worley of the San Diego Zoo CRES has tested serum and plasma from free-ranging rhino from Zimbabwe and Namibia for antibodies to and antigens cross reactive with hepatitis viruses. His data will be presented separately.

Sera from 26 Zimbabwean black rhino and 3 animals from Namibia were tested for antibodies to African Horse Sickness (AHS), Foot and Mouth Disease (FMD), Rinderpest (RP) by enzyme linked immunosorbent assay, virus infection associated antigen and fluorescent

antibody neutralization, respectively, at the USDA Foreign Animal Disease Diagnostic Laboratory on Plum Island, New York. No evidence of previous exposure to any of these viruses was found (Yedloutschnig, 1990). These samples included the 10 rhino shipped to the United States and the 2 shipped to Germany in 1989. As several outbreaks of Foot and Mouth Disease occurred in Zimbabwe during 1989 around the time the rhino were shipped, this is rather reassuring information.

Kock and his coworkers recently documented many of the normal metabolic and physiologic parameters of free-ranging black rhino in Zimbabwe (Kock *et al* ., 1990b; 1990c). Although some of these values are influenced by the physiologic stresses of capture, they should be of comparative value to zoo veterinarians. Serum and plasma for additional trace element and metabolite levels were sent to Dr. U.S.Seal. No samples were analyzed.

When wild populations decline sharply, and particularly when captive breeding strategies may become part of species survival, genetic questions come to the forefront. The basic questions usually are how much heterozygosity is present in natural populations; is there evidence distinct races or sub-species exist; and how can existing heterozygosity (genetic diversity) best be preserved. Although pedigrees may be used to answer these questions for captive animals, they can seldom be applied to truly free-ranging animals. When 7 blood proteins coding for 12 genetic loci from Zimbabwe and Namibian black rhino (19 individuals) were separated by electrophoretic means, no heterozygosity was found (May, unpublished report 1990). These findings agree with those of Melnick. This preliminary data should not be over interpreted and should be supplemented by additional samples, by checking additional loci, and by utilizing more advanced methods such as analysis of mitochondral and nuclear DNA sequences.

DISCUSSION

Two of the ten black rhinoceros that were relocated to the United States (Agrippa and Marongora) in 1989 died on ranches in Texas. In neither case has a firm cause of death been established. Hepatocellular degeneration and cholestasis was present in the former animal and an acute mild hepatopathy along with enteritis, pneumonia, nephritis and stomatitis in the latter. Although the availability of blood chemistry and hematology data and extra sera or plasma taken at capture did not help establish a diagnosis or successful treatment program in these two cases, the value of this kind of reference data should be apparent. Samples taken at capture should serve as the beginning of each animals health record and data base whenever black rhino are shipped internationally. The veterinarian accompanying international shipments of wild-caught black rhino should take the responsibility to see that each animal's health records are complete, starting from the day of capture and that those records are delivered to the recipient institution.

Gaps in our knowledge of black rhino health problems continue to be filled. The opportunities to sample significant numbers of endangered free-living black rhino are rare. IWVS is willing to cooperate with any reasonable request for samples from free-ranging black rhino that may help answer significant health questions.

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