DISEASES OF BLACK RHINOCEROSES IN CAPTIVITY

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A number of diseases of unusual nature and uncertain aetiology have affected black rhinoceroses *(Diceros bicornis)* in captivity. These diseases have played a significant role in limiting the growth of the captive black rhinoceros population. Haemolytic anemia is one example; in one survey of captive black rhinoceroses, it accounted for 40% of all adult deaths[21 22]. A syndrome of mucocutaneous ulcers has had a similar impact[24] and other poorly understood conditions include an apparently increased incidence of fungal pneumonia, haemosiderosis[15] and encephalomalacia[23]. Although there are few, if any, reports of these syndromes in the wild, there are reports of similar diseases occurring in black rhinoceroses shortly after capture, and thus, these syndromes may have significance for black rhinoceroses maintained in even semi-captive situations. In contrast to the black rhinoceros, the diseases reported in captive white rhinoceroses *(Ceratotherium simum)* in North America are of a more routine nature and an apparently lower incidence.

This paper will focus on the diseases of the black rhinoceros noted above. For information regarding other diseases, references regarding general medicine[8 12 16 31], capture techniques[14 29], and infectious diseases[28], and reviews of the veterinary literature[18] are available.

HAEMOLYTIC ANAEMIA

Our investigations centred on possible causes for the hemolytic anaemia. A fatal case of haemolysis at the St. Louis Zoo led to subsequent surveys that noted 47 episodes of haemolysis in 39 individual black rhinoceroses. Cases can be classified as "primary," i.e., those haemolytic events that occur without other obvious underlying disease, and "secondary," those cases that occur as agonal events in rhinoceroses dying of other causes. Although several familial groupings of affected rhinoceroses exist, no sex, age, or captive-bred vs. wild-caught patterns were evident[22]. Early reports suggested that many of the acute cases of haemolysis were associated with leptospirosis[4 30]. Indeed, with the advent of the fluorescent antibody (FA) test for *Leptospira interrogans*, many cases that were not evident by titres were positive[11]. At the present time, biannual vaccination of all black rhinoceros with leptospiral bacterins that contain the serovars *icterohemorraghiae* and *grippotyphosa* (serovars that have elicited elevated titres in two reports of rhinoceroses surviving haemolysis) has been recommended[11]. (Author's note: *Leptospira interrogans* has also been identified in the tissues of an aborted greater one-horned Asian rhinoceros *(Rhinoceros unicornis)* calf at the Bronx Zoo.)

However, not all of the cases of haemolysis could be accounted for by leptospiral infection, and a series of investigations was initiated to determine if other aetiologies or properties inherent in the black rhinoceros red blood cell (RBC) increased their susceptibility to haemolysis from a number of causes (drug exposure, bacterial infection, etc.). Various studies indicated that the anemia was unlikely to result from autoimmune disease[2], uncomplicated vitamin E deficiency[3], nor an unstable haemoglobin[6].

The most significant findings resulted from investigations into the metabolism of the black rhinoceros RBC. Initial studies focused on RBC levels of glucose-6-phosphatase dehydrogenase (G-6 PD) and other enzymes commonly recognized to cause haemolysis in man, but those levels were either normal or elevated compared to human normals[27]. However, on a more fundamental level,

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the black rhinoceros RBCs were noted to be markedly deficient in energy (ATP), when compared to other mammalian species.

The RBCs of white rhinoceroses, a species which has been apparently healthy in captivity, were also low in ATP, but they also had significantly higher levels of the enzyme catalase than black rhinoceroses[26]. The full significance of this is unknown, but further metabolic studies are underway. Interestingly enough, Dr. Paglia has hypothesized that the energy deficiency of the black rhinoceros RBC could be an adaptive characteristic for haemic parasitism in the same manner than G-6-PD deficiency is considered adaptive in man. Dr. Paglia, is presently on sabbatical in the laboratories of Dr. Eric Harley at the University of Cape Town.

At the present time, suggested treatment of acute cases of haemolysis is supportive and includes penicillin and possibly dihydrostreptomycin (in the event that it is leptospiral-induced), parenteral vitamin E (to assist in the maintenance of membrane stability), enteral and/or parenteral phosphorus supplementation (as many of the chronically haemolytic individuals have become hypophosphataemic)[7], and possibly blood transfusion. The latter has been attempted in two black rhinoceros as preliminary findings suggest that rhinoceroses do not have inherent antibodies to other blood groups of their species.

MUCOCUTANEOUS ULCERATIVE SYNDROME

Oral, nasal and cutaneous ulcers have been frequently reported in black rhinoceros, and in captivity, often lead to debilitation and can progress to death. Infection with *Stephanofilaria dinniki*, the most common cause of skin ulcers in wild black rhinoceroses, has not been identified in captive animals. At the present time, the ulcerative syndrome has been identified in 45 black rhinoceroses in North America[24]. A typical case starts with raised plaques that progress to vesicles and subsequent ulcers. Often they start over points of wear (where early lesions are difficult to differentiate clinically from scrapes) and peripheral areas (eg, ear tips, coronary bands) and in severe cases may progress to cover larger areas (up to 70% of the body surface) over the lateral and ventral thorax and abdomen. Typically, the lesions are bilaterally symmetrical.

Histologically, the initial lesions are characterized as superficial necrolytic dermatitis. When present, inflammatory changes are found in association with ulcers. At the present time, the aetiology remains unknown. Bacterial isolates from the lesions have been variable and most likely reflect secondary infection; neither viral inclusions nor viral particles have been noted in the cases examined histologically (and viral culture has been negative in two additional cases). Additionally, there has been no evidence of autoimmune disease in two cases that were examined using anti-porcine, anti-equine or anti-rhinoceros immunoglobulin. Due to the similarity with superficial necrolytic dermatitis, the possibility of concurrent liver disease, nutritional deficiencies, and/or endocrine abnormalities are being evaluated.

In many of the less extensive cases, the ulcers resolve spontaneously. Others have noted an apparent marked response to corticosteroid therapy[26]. That therapy is associated with an increased incidence of fungal pneumonias (primarily *Aspergillosis* species, see below), however, and should be used only in cases that are clearly life-threatening.

FUNGAL PNEUMONIA

The author knows of at least nine cases of fungal pneumonia in black rhinoceroses - seven due to infection with *Aspergillus* species, three *Phycomyces* species (there was one dual infection). Four of the fungal infections occurred after corticosteroid therapy, three were associated with other chronic illnesses (two with end-stage lesions of the mucocutaneous syndrome), and two cases were "spontaneous," i.e., no other illness or immunosuppressive factors were evident. Cases in animals on concurrent corticosteroid therapy occurred after even apparently low dose therapy (eg, 1 $mg.kg^{-1}$ for several days) and so extreme caution should be used whenever immunosuppressive drugs are used in this species. Fungal pneumonias are unusual in most mammalian species and when they

occur, are most commonly associated with immunosuppression. In the cases that occurred while undergoing corticosteroid therapy, it is not clear if those drugs aggravated pre-existing immunosuppression, or if black rhinoceroses are particularly sensitive to the effects of this class of drugs. Studies to better characterize the immune status of black rhinoceroses are proposed in order to address this issue and to possibly identify a serological test for antemortem diagnosis.

At the present time, treatment is speculative. Prophylaxis has been attempted with thiabendazole[7] and itraconizole[8]. The latter was administered at the rate of 13 $mg.kg^{-1}$ with no signs of ill effects; however, at this time, the cost of this medication is prohibitive.

ENCEPHALOMALACIA

Encephalomalacia has been reported in three black rhinoceros calves and one 2-year-old animal. All were female. Clinical signs varied from somnolence and hyperthermia to hyperexcitability[13 23]. Three died during their episodes and one was euthanized subsequent to becoming a "dummy" calf. Histologically the lesions were notable for massive white matter necrosis (leucoencephalomalacia), and in some areas, adjacent gray matter was also affected. Evidence of inflammation was evident only in the older lesions where presumably it was a reaction to necrosis. It is believed that the variable neurological signs may simply reflect which areas of the brain in each individual were most severely affected.

Histology or specific diagnostic tests were not supportive of vitamin E-induced malacia, polioencephalomalacia, or viral infections (eg., encephalomyocarditis virus or equine encephalitis). The histologic pattern most closely resembled that of leucoencephalomalacia due to ingestion of food contaminated with the mould *Fusarium moniliforme*, however, feedstuffs ingested prior to the onset of the neurological symptoms were not available for analysis.

The variable clinical presentation of this disease emphasizes the importance of collecting brain and spinal cord tissues from all rhinoceros deaths, particularly those in which a diagnosis is not readily evident. It is possible that this syndrome is under-diagnosed due to the difficulties in removing a rhinoceros brain.

HAEMOSIDEROSIS

Accumulation of iron has been noted in tissues of captive black rhinoceroses[15 33], and has been shown to be positively correlated with length of time in captivity[16]. At the present time it is uncertain if this may represent a chronic stage of haemolysis or nutritional deficiencies/excesses in the captive diet.

OTHER DISEASES

Black rhinoceroses are susceptible to tuberculosis. Recommendations for the most effective testing regimens for this disease have been limited by a lack of positive individuals. In the past, two black rhinoceroses infected with *Mycobacterium bovis* and one exposed to those individuals were positive on intradermal testing with MOT (mammalian old tuberculin) and ELISA[18]. More recently, a black rhinoceros at the Detroit Zoological Park who was culture-positive for *Mycobacterium tuberculosis*, was positive when 0,1 ml of USDA bovine tuberculin was administered intradermally in the eyelid, and this animal was also positive on ELISA testing. It has been our recommendation that any suspicious or positive tuberculin test be followed by acid-fast culture of gastric lavage or tracheal wash samples.

In maintaining black rhinoceroses in captivity, it is advisable to avoid exposure to creosote and other phenolic compounds. Exposure to these and possibly other chemicals may induce and/or contribute to a syndrome of liver necrosis and failure. Epidemiology suggests that several recent black rhinoceros deaths in North America have been associated with exposure to creosote. Initial clinical

signs are related to liver failure, including marked hyperbilirubinemia (both direct and indirect bilirubin are elevated). Terminally, mucocutaneous ulcers and haemolytic anaemia may develop (it is unclear if these signs are from the toxic exposure or simply agonal events as has been noted in other black rhinoceroses with chronic diseases). The signs of liver necrosis and skin ulcers are similar to those previously reported in black rhinoceroses exposed to creosote in North America and southern Africa[1 6 16 32].

SUMMARY

Integral to many of the projects noted above has been comparative data from white and greater one-horned Asian rhinoceroses (and hopefully to make future comparisons with the Sumatran rhinoceros *(Didermocerus sumatrensis)*). Data from these rhinoceroses is useful for comparison with the diseases of the black rhinoceros and additionally, for establishing baseline data for these species. Frozen (-75°C) sera and tissue banks have aided such comparisons, and allowed retrospective analysis as knowledge and assay availability improve. Presently, over 400 sera and tissue samples from 25 black, white and greater one-horned Asian rhinoceroses are stored at the St. Louis Zoological Park. The establishment of regional sera/tissue banks elsewhere is strongly encouraged.

In summary, although much progress has been made in understanding the diseases of the black rhinoceros, much remains to be learned. Under the auspices of the Rhinoceros Taxon Advisory Group (TAG) and the black rhinoceros Species Survival Plan (SSP), research into the diseases of black rhinoceroses remains an active and ongoing effort. In August 1993, a meeting of a diverse group of specialists interested in the medicine of rhinoceroses was organized by Dr. Evan Blumer, Research Coordinator for the Rhinoceros TAG. The meeting identified priority areas for future research. Areas deemed of vital significance included further studies of nutrition, stress, mucocutaneous ulcerative disease, comparative cellular metabolism, management issues, and enhancement of intra- and interregional cooperation in sample and data acquisition and storage. Presently, these recommendations are being organized, interested researchers and institutions identified (eg, Dr. Ellen Dierenfeld, Nutritional Advisor to the Black Rhinoceros SSP Committee, has proposed a coordinated series of projects to address nutrition), and steps taken so that the TAG may seek funding for priority projects. Hopefully, this enhanced effort will result in a better understanding of the medicine and management of this critically endangered species.

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