INFECTIOUS DISEASES OF THE RHINOCEROS AND TAPIR

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The Perissodactyla is a small order of mammals comprised of only three families, the Equidae, Tapiridae, and Rhinocerotidae. More numerous in the Pleistocene, the order now contains only 15 species. The tapir and rhinoceros are physically more similar to each other than to the equids, and several rhinoceroses and tapirs share similar habitat, survival strategies, and health problems. All rhinoceroses and tapirs are considered either threatened or endangered in the wild.

The Tapiridae family contains four species: the Malayan tapir (*Tapirus indicus*) of southeast Asia, the Central American or Baird's tapir (*T. bairdii*), the South American tapir (*T. terrestris*), and the mountain tapir (*T. pinchaque*). All are browsers and, with the exception of the mountain tapir, live in lowland tropical or neotropical habitats. The mountain tapir lives at higher elevations in the Andes Mountains of South America and is considered the rarest of the tapirs.

The Rhinocerotidae family contains five species: the black rhinoceros (*Diceros bicornis*), the white rhinoceros (*Ceratotherium simum*), the greater one-horned rhinoceros (*Rhinoceros unicornis*), the Javan rhinoceros (*R. sondaicus*), and the Sumatran rhinoceros (*Didermocerus sumatrensis*). All are neotropical or tropical browsers, with the exception of the grazing white rhinoceros.

Much of the information regarding infectious bacterial, viral, and mycotic diseases of the tapir and rhinoceros is derived from descriptions of captive animals, and therefore may show a degree of species bias because of the prevalence of certain species in captivity. The information on parasitic diseases is largely derived from descriptions of organisms taken from free-living tapirs and rhinoceroses over the last 100 years, and is complicated by changing scientific names for the parasites. Generally, the tapir and rhinoceros are affected by the same pathogens and similar parasites as the other perissodactylids, the equids, and most diagnostic and therapeutic regimens are therefore based on those developed for horses.

BACTERIAL DISEASES

Salmonella is one of the most frequently reported bacterial pathogens of the rhinoceros and tapir. Most reports have described infections of captive animals, although there were two reports of deaths in recently captured black rhinoceroses. *Salmonella* appears to cause septicemia in the rhinoceros and tapir as commonly as causing enteritis. Fatal *Salmonella* septicemia has been described in the South American tapir,[39] a greater one-horned rhinoceros[40] and a juvenile southern white rhinoceros.[41] The latter showed signs of disseminated intravascular coagulation, presumably secondary to endotoxemia. The treatment of these animals with fluids, parenteral antibiotics, and steroids was unsuccessful. *Salmonella typhimurium* septicemia was described in an animal 2 days postcapture,[42] and *S. weltevredem* was isolated from the heart, blood, spleen, and intestine of an African rhinoceros that died 10 days postcapture.[12]

Salmonella enteritis occurs in both neonatal and adult animals. *Salmonella poona* was isolated from a neonatal Baird's tapir experiencing acute gastrointestinal distress. The infant was successfully treated with antibiotics and oral fluids, but continued to have positive fecal cultures after the discontinuation of antibiotic therapy.[43,44] *Salmonella* is suspected to have caused a fatal enteritis of an adult Sumatran rhinoceros. This animal exhibited profuse, bloody diarrhea, extreme pallor, and anorexia. At necropsy it had a severe, necrotizing enteritis. Fecal cultures for *Salmonella* were negative, but cultures were obtained after the animal was placed on antibiotic therapy. Cultures of elephants and horses on the zoo grounds, with similar clinical signs, grew *S. blockey*. Similarly, the soil samples from several paddocks of the Sumatran rhinoceros yielded the same organism. The occurrence of salmonellosis in the zoo coincides with the rainy season.[45]

The diagnosis of *Salmonella* infections is based on clinical signs and bacterial culture, although, as noted, the organism may not be cultured from every case. Samples should be cultured on an enteric medium, such as Hectone enteric agar, and also cultured in a selective medium, such as selenite broth.

The treatment of *Salmonella* infections in captive animals is frequently unrewarding. Aggressive fluid therapy is essential, but the size and intractable nature of most rhinoceroses and many tapirs make the delivery of antibiotics and adequate volumes of fluids difficult. Parenteral antibiotic therapy ideally should be based on antibiotic susceptibility testing.

The inability to prevent animals from becoming inapparent carriers of the organism further complicates clinical and postclinical management decisions. A more satisfying approach is to attempt to prevent infection by minimizing contact of stressed or immune compromised individuals with the organism and by strict quarantine of clinical cases.

Enteritis has also been associated with infections by *Pseudomonas pyocyanea* and coliform bacteria in hand-reared rhinoceroses,[37,38,46] by *Klebsiella* and *Pseudomonas* sp. in the white rhinoceros,[38] and by a β-hemolytic *Streptococcus* in a Malayan tapir.[31] Systemic infections caused by a β-hemolytic *Streptococcus* in the tapir,[34] *Pasteurella* in the tapir,[47] and *Escherichia coli* in white and black rhinoceroses[37,48] have also been reported. In England, a Sumatran rhinoceros died of an acute bacterial toxemia caused by *K. pneumoniae*. The clinical signs included recumbency and colic.[31] *Yersinia pseudotuberculosis* has been identified as the cause of enteritis and enlarged

mesentric lymph nodes in young rhinoceroses.[32] Omphalophlebitis, in one incident associated with a valvular endocarditis caused by *E. coli*, was observed in captive white rhinoceros neonates.[27] In the Sumatran rhinoceros, local or generalized pyoderma was a consequence of hyperkeratosis, which is caused predominantly by *E. coli*.[87]

Tapirs appear especially prone to the development of mandibular abscesses, frequently referred to as "lumpy jaw," and evidence of infection has even been observed in a fossil perissodactylid.[51] The lesions appear as a unilateral, firm swelling ventral to the ramus of the mandible or in the parotid region. The abscesses might fistulate, and bony involvement is common. The pathogenesis of these lesions is unknown but has been speculated to be similar to that of lumpy jaw in cattle, in which rough feed causes oral lesions through which pathogens migrate ventrally.[44] *Corynebacterium pyogenes*, β-hemolytic *Streptococcus*,[42] *Actinomyces*,[1, 83] *Necrobacillus*, *Escherichia coli*, and tubercle bacilli[25] have been isolated from these lesions. Biopsy of the lesion is recommended, because a mandibular fibrosarcoma has also been observed in a Malayan tapir.[25]

The treatment of mandibular infections depends on the causative agent. Kuehn[38] has described IV therapy with 20% sodium iodide, three 15-ml treatments at 10-day intervals. Surgical debridement of infected soft tissue and bone is usually indicated. If there is an underlying cellulitis or osteomyelitis, prolonged treatment with parenteral antibiotics is necessary. The prognosis for complete resolution of the lesion depends on the extent of infection and on the causative agent, but is usually guarded.

Methods of prevention of these lesions include offering only leafy, nonscabrous portions of hay or pelleted feed products. The inclusion of sodium iodide in the diet appeared to prevent "lumpy jaw" lesions in several mobs of kangaroos, in the senior author's (Ramsay's) experience, and may be of benefit to tapirs, although Alexander[1] noted that a diet containing sodium iodide was poorly accepted by a South American tapir.

Clostridial infections are infrequently reported in these species. Tetanus has been described in a rhinoceros.[30] but no reports were found for tetanus in the tapir. We recommend tetanus toxoid vaccination for both these groups. A black rhinoceros, which demonstrated ataxia, weakness, and oral lesions, and subsequently died, had *Clostridium sordellii* cultured from its liver and spleen.[4] A cause of "big head" in cattle and sheep, *C. sordellii* is a common pathogen of domestic livestock. The vaccination of captive animals with a multivalent clostridial bacterin should be considered in areas endemic for *Clostridium* sp.

Leptospira was cultured from the liver of a euthanized, adult black rhinoceros in the wild. The relation of this organism to the animal's clinical sign of being unable to rise is unknown.[65] Serological antibody titers for *L. canicola* and *L. icterohaemorrhagiae* have suggested leptospirosis as a cause of hemolytic anemia in two captive black rhinoceroses.[17] A detailed discussion of hemolytic anemia in the black rhinoceros was presented in the first article in this chapter.

Keratitis is frequently observed in the captive Malayan tapir and Sumatran rhinoceros. The cause of this syndrome is unknown, but may be infectious. Affected eyes show corneal opacity, corneal ulceration, and conjunctivitis. Affected eyes should be swabbed for culture and cytology. Ophthalmic antibiotic solutions sprayed topically are usually of benefit. This lesion is probably caused by physical trauma and aggravated by excessive exposure to sunlight, because both these species live in dense tropical jungle. Providing adequate shade for these animals is believed to be important for their eyes and overall well-being.

In 1981 and 1982, five Javan rhinoceroses were found dead in Ujong Kulon, Indonesia. All died suddenly. Severe diarrhea was reported in three rhinoceroses, and one carcass showed prolapse of the rectum and a foamy mucous discharge from the mouth and nostrils. The symptoms indirectly observed in the dying rhinoceroses were characteristic of those of anthrax. Unconfirmed reports claimed that the last Javan tigers in Ujong Kulon died of anthrax. Anthrax is endemic in Indonesia, but soil samples collected in Ujong Kulon were negative for anthrax spores.[66]

MYCOBACTERIAL INFECTIONS

There have been numerous reports of "tuberculosis" in the tapir and rhinoceros[14, 26, 32, 34, 41, 44, 52, 62, 72] in captivity and in one free-living black rhinoceros.[35] Diffuse and focal pulmonary lesions, bronchitis, pleuritis, and lymph node abscesses have been described in captive animals. A mucocatarrhal bronchiolitis, granulomas in the thoracic cavity, and an abscess in the pelvic cavity, with abscessation of draining lymph nodes, were observed in a wild black rhinoceros.[35] *Mycobacterium bovis* has been isolated from the tapir[30, 72] and two black rhinoceroses.[45] One tapir showed cavitary pulmonary abscesses.[30] One black rhinoceros, which had died, had a generalized lymphadenopathy, cavitary pulmonary lesions with purulent centers, and miliary 1- to 2-mm liver lesions. *M. bovis* was cultured from an antemortem lung biopsy from the other rhinoceros and showed the following responses to intradermal skin testing: a marked delayed hypersensitivity reaction (DHR) to mammalian old tuberculin (MOT), a moderate DHR to bovine purified protein derivative (BPPD), and a mild DHR to avian old tuberculin (AOT). It was euthanized after 6 months of treatment with isoniazid. Gross lesions in this animal resembled those described above, but no mycobacteria were cultured postmortem. Histology demonstrated suppurative pulmonary granulomas, with giant cells and alveolar wall fibrosis.[45]

Mycobacterium tuberculosis has been cultured from the lowland tapir,[30, 50] black rhinoceros,[26] and greater one-horned rhinoceros.[41] The onset of infection is insidious, with weight loss as the only initial clinical sign. A productive cough and dyspnea may appear shortly before death. Observed lesions included

chronic bronchitis, bronchopneumonia, caseous tubercles, and pulmonary hilar lymphadenopathy. *M. avium* infection has been reported in a tapir.[14]

The antemortem diagnosis of mycobacterial infections is difficult. Clinical signs are vague, and intradermal skin testing yields equivocal results.[15] Use of the enzyme-linked immunosorbent assay (ELISA) has shown some promise in zoo species but, in the evaluation of the rhinoceros and tapir, with or without evidence of mycobacterial infection, it appears that ELISA results must be evaluated in combination with those of other tests.[16] The need for standardization of testing methods in the rhinoceros has been identified.[24] The following testing methods have been suggested:[16] (1) comparative intradermal skin testing using 0.1 ml MOT, BPPD, and AOT, initially on the tail folds and repeated 10 days later in the neck if suspect reactions occur at tail site; (2) biopsy of all questionable intradermal reactions; (3) concomitant ELISA testing (serum drawn prior to skin testing) of any animal in question; (4) similar testing of available "normal" conspecifics in the collection; and (5) culture of the respiratory tract of any animal showing clinical signs.

The treatment of mycobacterial infections is controversial because of the difficulty in making a definitive diagnosis and the unknown efficacy of antimycobacterial drugs in these species. For the rhinoceros, the use of isoniazid has been suggested.[21] 15 to 25 mg/kg/day PO for 1 year, then 10 to 15 mg/kg/day PO for the second year. Isoniazid, 7 g/day PO for 6 months, did not eliminate mycobacterial lesions in a female black rhinoceros, but was possibly the cause of negative cultures posteuthanasia.[15] Frolka[20] has described a hygienic and therapeutic regimen that prevented the spread of *M. bovis* in a collection of tapirs.

It has been speculated that many of the mycobacterial infections of captive animals are acquired postcapture, in the countries of origin, where human tuberculosis is common. If so, control of mycobacterial infections might be accomplished by scrupulous quarantine and testing of imported specimens, using various evaluation techniques (see earlier).

VIRAL INFECTIONS

Poxvirus infections have been described in both captive black and white rhinoceroses. One report suggested that the virus is related to a fowlpox virus.[46] The evaluation of more recent infections has indicated that the virus appears to be more closely related to cowpox virus.[37] Lesions include vesicles and pustules of various sizes on the flank, abdomen, and medial thigh. Ulcerations may also be present at the mucocutaneous junctions and the mucosa of the esophagus and forestomach.[28] Therapy includes supportive care and the treatment of secondary bacterial or fungal infections.

A herpesvirus infection was described in a group of captive Malayan and mountain tapirs. Clinical signs included fever, keratitis, erosions, and ulcers at the oral and nasal mucocutaneous junction and coronary band, and dermatitis. Some coronary band lesions undermined the soles of the feet, causing lameness, and the coalescence of skin lesions produced seborrheic eczema in a few animals. Three of six clinically ill animals died, despite symptomatic treatment.[31]

Foot-and-mouth disease was documented in South American and Malayan tapirs in an outbreak at a European zoo. The principal signs were interdigital lesions.[30] Foot-and-mouth disease is known to affect ruminants but does not affect equids, and has not been reported in the rhinoceros.

No reports of equine encephalitides could be found, but the vaccination of tapirs against Venezuelan, eastern, and western equine encephalitis in endemic areas has been recommended.[38]

Several diseases observed in the rhinoceros are speculated to be caused by viruses, but no causative agents have been identified. An acute vesicular dermatitis occurred in a captive female black rhinoceros, which led to the animal's death. It did not appear contagious, because another rhinoceros housed with the affected animal showed no clinical signs.[27] Papillomas have been observed in black and greater one-horned rhinoceroses.[32] In domestic animal species, papillomas are known to be caused by papovaviruses, but there have been no attempts to identify viral antigens in rhinoceros papillomas.

MYCOTIC INFECTIONS

Dermatophytosis has been described in both the rhinoceros and tapir. *Microsporum gypseum*, *M. canis*, and *Trichophyton tonsurans* have been cultured from the tapir.[5,40] *M. canis* causes severe infections in the mountain tapir, producing alopecia of the body and proximal extremities. The lesions are covered with yellowish scruff and respond to griseofulvin, 10 mg/kg for 50 days.[40] *Trichophyton mentagrophytes* was identified as a cause of dry eczematous lesions in a herd of white rhinoceroses, held indoors after importation. Topical therapy with etisazol alleviated the problem.[42] A case of dermatomycosis in a white rhinoceros, caused by *Trichophyton* sp., resolved without treatment when the animal was released from its winter quarters.[71]

Pulmonary aspergillosis has been identified in two black rhinoceroses.[23] *Aspergillus fumigatus* and *Absidia corymbifera* were cultured from lesions in these animals. *Fusarium*, *Alternaria*, and *Absidia* sp. have been identified as secondary invaders of superficial skin lesions.[33] *Pityrosporon pachydermis* was described as the cause of exfoliative dermatitis in greater one-horned rhinoceroses.[43]

Coccidioidomycosis caused the death of a wild-caught, 25-year-old captive South American tapir. Death followed a 3-week history of weight loss and 3 days of lethargy, dyspnea, and anorexia. Necropsy lesions included discrete to coalescing granulomas of the liver and a hilar lymphadenopathy. The animal was captured in Ecuador, an endemic area for *Coccidioides immitis*, and shipped to a zoo in the south-

east United States, an area not endemic for coccidioidomycosis. It was speculated that the organism lay dormant in the animal for 20 years prior to the development of clinical disease.[10]

PARASITIC DISEASES

Perissodactylids can suffer from endoparasitic and ectoparasitic infestation, and from protozoal enteric parasites and hemoparasites.

Ectoparasites

Arthropods

Sarcoptes tapiri is a potentially pathogenic parasite of tapirs. In young animals, infestations are severe and may become secondarily infected. Adults usually demonstrate only small areas of alopecia, unless stressed. Lesions generally begin on the belly and inner limbs and progress to the sides of the torso and back, becoming large, erythematous hairless patches, with skin thickening and crust formation. Pruritus may be severe and can lead to secondary trauma. Exceptional cases may show proliferative dermatitis around the eyes, ears, and mouth, which interferes with hearing and the prehension of food. Severe infestations produce general debilitation and may cause death.[36] Diagnosis is by demonstration of the mite in the epithelium or crust by skin scraping. Treatment with 1 gallon of toxaphene (Cooper-Tox) in 150 gallons of water has been recommended, two sprayings 2 weeks apart.[36] Alternatively, ivermectin, 0.2 mg/kg SC or IM, should be effective.[15] Infected animals should be quarantined from all stock until the infection is cleared.

A large number of *Amblyomma* ticks infest rhinoceroses. Adult *A. hebraeum* and *A. sparsum*, vectors for *Cowdria ruminantium*, the agent for heartwater disease, have been recovered from white and black rhinoceroses.[15,37] *A. rhinoceros*, *A. gemme*, and *A. marmorem* have also been described as parasites of black rhinoceroses.[38] *Amblyomma testudinarium* was identified as an ectoparasite of the rhinoceros and tapir in Indonesia,[37] and *A. crenatum* was described as infesting the Javan rhinoceros.[39] *A. infestum*, *Hylaomma walkeriaeirii*, *Aponomma* sp., and *Haemaphysalis* sp. have been found on Sumatran rhinoceroses in peninsular Malaysia.[36]

Rhipicephalus ayrei, *R. superitus*, *Cosmiomma hippopotamensis*, and *Dermacentor*, *Haemaphysalis*, and *Hyalomma* sp. have been found on black rhinoceroses from Kenya.[15,43,71] *D. rhinocerinus* has been identified on white rhinoceroses imported into the United States.[71] Ticks generally gather in the tail folds, around the ears and eyes, and around the genitals. Ticks are seldom of pathogenic significance by themselves. The major veterinary concern is their role as vectors for hemoparasites and other pathogens of domestic animals. A major reason for antitick treatment is restriction of the importation of nonendemic diseases carried by ticks on imported rhinoceroses. The use of 1.0 and 0.5% flumethrin pour-on has proved successful for the treatment of translocated black rhinoceroses, with the lower concentration being more suitable because of the larger treatment volume.[15] It is assumed that similar therapies are as effective and safe in the tapir.

Biting flies of the genera *Lyperosia*, *Rhinomusca*, *Tabanus*, and *Glossina* have been reported as causes of irritation for the black rhinoceros.[15,71] *Glossina* sp., tsetse flies, are of particular importance as vectors of trypanosomes. The production of proliferative, periocular lesions seen in captive and wild black rhinoceroses were speculated to have been caused by *Habronema* larvae; these responded to treatment with topical antibiotics and steroids.[36] *Tabanus* sp. and *Chrysomyia bezziana* affect captive Sumatran rhinoceroses in peninsular Malaysia.

An unusual ectoparasite of the captive black rhinoceros and Malayan tapir in southeast Asia is a skin-piercing, bloodsucking moth, *Calpe eustrigata*.[2] It has been observed feeding on captive animals, but has not been seen in the wild. Because the Malayan tapir is endemic to the area, it has been presumed that the *Calpe* moth may feed on free-living tapirs, and possibly also on endemic rhinoceros species.

Nematodes

Stephanofilaria dinniki has been frequently reported as a cause of dermatitis in wild and newly imported black rhinoceroses.[45,62,69,76] Lesions are an ulcerative, proliferative dermatitis, caudal to the point of the elbow and on the flanks. The lesions are centrally depressed, raised peripherally, and usually covered with a crust. They may be pruritic, and secondary trauma, caused by scratching or oxpeckers (birds that perch on the rhinoceros and eat parasites and crusts) may lead to secondary bacterial infection. The occurrence is seasonal, with manifestations most severe in the summer and regression in the winter.

Histology demonstrates microfilaria in the superficial lymphatics and interstitial spaces and inflammatory infiltrates of plasma cells and lymphocytes. Adult nematodes were not found in the ulcerative portion of the lesion, but were occasionally noted in the periphery of the lesions. The infection is believed to be transmitted by certain species of biting flies and therefore is not seen in long-term captive animals. Diagnosis is made on the basis of clinical signs and histology. Lesions usually resolve spontaneously, but may be treated with topical preparations.[15] Infections have not been described in the white rhinoceros, which is sympatric with the black rhinoceros in areas endemic for this nematode.

Endoparasites

Arthropods

Larvae of *Gyrostigma pavesii* are commonly found in the stomachs of both African rhinoceros species. *Gyrostigma pavesii* was observed in a captive white rhinoceros in Malaysia, but natural infections with African *Gyrostigma* sp. have not been reported outside the African continent.[36] *Gyrostigma conjungens*

and *Gasterophilus rhinocerontis* occur in the black rhinoceros.[11] *Gasterophilus* larvae have also been found in the stomach of imported southern white rhinoceroses, but the infection apparently cleared, without treatment, in the Northern hemisphere.[27] *Gasterophilus sumatrensis* has been described from larvae found at necropsy of a captive Sumatran rhinoceros.[11] Larvae are laid by adult flies at the nares or horn base and migrate through tissues to the stomach. Clinical signs are minimal, and treatment with an organophosphate compound, such as 75% dimethylhydroxytrichlorethyl phosphate, has been described.[11] No reports of bot infections in tapir have been published.

Platyhelminths and Pentastomes

Fasciola hepatica has been associated with hepatic cirrhosis and the death of a captive South American tapir.[6] Trematodes in the bile duct were an incidental finding in a captive Malayan tapir.[27] The death of a young captive greater one-horned rhinoceros has been attributed to *F. gigantica*.[71] *Brumptia bicanda* is a trematode of the intestine of black and white rhinoceroses. Its life cycle is unknown, and no attempts at treatments have been described.[41]

Tapeworms infect the tapir and rhinoceros, but are seldom of clinical importance. Captive mountain tapirs can be infected with *Paranoplocephala* sp., and respond to chlorsalicylamide, 100 mg/kg PO.[30] Tapeworms of the genus *Anoplocephala* are found in captive and wild rhinoceroses. *A. diminuta* and the enormous *A. gigantea*, which can grow to 20 feet long, have been described in the Javan rhinoceros.[56] *A. vulgaris* was found and thought to have contributed to overall debilitation in a captive greater one-horned rhinoceros.[41] This parasite has also been described in free-living black rhinoceroses.[73] The treatment of tapeworms in rhinoceroses includes praziquantel, niclosamide,[32] or chlorsalicylamide, 160 to 220 g/rhinoceros, once.[41] Orabatid mites are believed to be the intermediate host for these tapeworms, and may be controlled by not leaving bedding in the exhibit for extended periods of time or by periodic flaming of the yards.[41]

Several cysts containing the nymphs of the pentastome *Armillifer moniliformis* have been recovered from a wild-caught Malayan tapir.[42]

Nematodes

Strongyloides sp. and strongylids are commonly found in the tapir.[60] Ascarid ova have also been found in the feces of the mountain tapir.[32] The presence of *Capillaria hepatica* was an incidental finding in the liver of a South American tapir, and *Brachyclonus indicus* is thought to have caused the death of a Malayan tapir.[60] Nematode parasite infections are diagnosed by fecal flotation and by the identification of ova or larvae. They may be treated PO with mebendazole, 8 mg/kg, or thiabendazole, 50 to 60 mg/kg. These treatments should be given once and repeated in 2 to 3 weeks. Alternately, levamisole, 10 mg/kg PO, or ivermectin, 0.2 mg/kg PO, can be given once.

Rhinoceroses are infected with a wide variety of nematode parasites. The ascarid *Probastamayria vivipara* has been found in captive greater one-horned and white rhinoceroses.[32] Strongylids, *Strongyloides*, and *Crossocephalus* sp. infect the Sumatran rhinoceros. *Parquilonia brumpti* and *Murshidia*, *Buisoon*, and *Khalilia* sp. are found in the small intestines of black rhinoceroses.[41] *Memphisia*, *Henryella*, *Kiluluma*, and *Quilonia* sp., and *Oxyuris karamoja*, *Parabronema rhinocerotis*, and *Pteridopharynx omoensis* have been identified in the large bowel and cecum of the black rhinoceros.[41, 73, 74] *Strongylus tremletti* was reported to cause massive intestinal infections in the black rhinoceros.[47]

The hookworm *Grammocephalus clathrotus* has been associated with bile stones in the bile duct of both African rhinoceros species,[74] and *G. intermedius* was reported in the large bowel of the black rhinoceros.[41] The spirurids, *Drascheia megastoma* and *Habronema khalili*, have also been identified in the intestine of the black rhinoceros.[41] *Capillaria* have been found in the white rhinoceros[73] and thelazid worms were reported in the conjunctival sac of free-living black rhinoceroses.[65]

Fecal egg counts may increase markedly whenever rhinoceroses are kept crated or the enclosures are not cleaned thoroughly and regularly. The diagnosis and treatment of enteric nematode parasites in the rhinoceros are similar to those described for the tapir. We have safely used mebendazole in the rhinoceros.

A captive South American tapir showed clinical signs and necropsy lesions suggestive of schistosomiasis.[45] Occasional bouts of bloody diarrhea culminated in a fatal episode of diarrhea and dark red urine. Granulomatous hepatitis and a hemorrhagic enteritis were found, with nonoperculated eggs associated with the lesions. The animal had been captive-born in the southeastern United States and held in collections in Florida, Ohio, and Michigan prior to its death. It was speculated to have been infected with *Heterobilharzia americana*, based on the histology of the lesions, the morphology of the ova, and the animal's geographical origin. A nutritional myopathy was also observed.[45]

Protozoans

Enteric Parasites

The enteric protozoal flora of tapirs may include ciliophora, mastigophora, and *Balantidium* and *Giardia* sp. The latter two organisms are also known to cause watery diarrhea in the tapir, which can be treated with chlortetracycline, paromomycin, or diiodohydroxyquin.[22, 60] *Balantidium* sp. and trophozoites of Entamoeba sp. have been found and presumed to be pathogenic in the white rhinoceros.[32, 59] Ciliated protozoa and *Phalodinium*, *Arachnodinium*, *Monoposthium*, and *Rhinozeta* sp. are found as commensal flora of both the black and white rhinoceros.[77, 78]

Hemoparasites

Intraerythrocytic *Babesia* sp. have been found in the Malayan tapir and both African rhinoceros spe-

cies. The infected tapir was a recently imported animal and exhibited antemortem signs of icterus and anemia. The babesial organisms were not identified as to species, but resembled those of *Babesia equi*.[80] Large *Babesia* and *Theileria*-like piroplasms have been found in healthy black and white rhinoceroses.[5,7,8] These two organisms were speculated to be pathogenic, in combination with a trypanosome infection, in recently captured black rhinoceroses.[47]

Trypanosoma organisms were found at necropsy of a Malayan tapir.[46] *Trypanosoma* sp. are endemic in the black rhinoceros in East Africa.[8,11] *T. vivax* and *T. brucei* have been reported in wild rhinoceroses in Tanzania and Kenya.[11,47] Postcapture deaths occurred in infected animals, despite treatment with diminazene aceturate (1.5 to 3.0 g) after capture. It is unclear, however, whether these deaths resulted from parasite infection or capture myopathy. A suggested treatment for clinical infections is pyrithidium bromide, 3.0 g, and diminazene aceturate, 6.0 g.[81]

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CAPTURE AND TRANSLOCATION OF THE FREE-RANGING BLACK RHINOCEROS: MEDICAL AND MANAGEMENT PROBLEMS

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Approximately 65,000 black rhinoceroses (*Diceros bicornis*) inhabited Africa south of the Sahara in the early 1970s. Because of poaching and habitat loss, this number has dwindled to about 2000, a decline of over 95%. Despite a 17-year ban on the sale of rhinoceros horn, as mandated by the Convention of International Trade in Endangered Species (CITES; see Appendix), poaching has persisted and, in some areas, intensified. Of the free-ranging black rhinoceroses in Africa, 95% now reside south of the Zambezi River, with about 430 in Zimbabwe, 400 in Namibia, and 750 in South Africa.

Poaching in Zimbabwe and Namibia has led to aggressive antipoaching practices, including translocation and horn removal. In Zimbabwe and Namibia, more than 400 animals have been translocated to private ranching and wildlife estate lands since 1986.[1,2,12] Some have been recaptured for relocation after escape or for investigation and treatment of medical problems, and over 150 have been dehorned as an antipoaching measure.

The capture and translocation of black rhinoceroses in Zimbabwe has resulted in an unacceptable mortality rate of 13 to 20% since 1986. A similar mortality rate of 22% has been recorded with capture and translocation operations in Namibia. In Zimbabwe, most deaths occurred within 1 week to 12 months after capture and, although many were caused by traumatic injury (e.g., horn loss, fighting), a significant number remain unexplained.[9,11] Investigations into the effects of capture, boma confinement, and translocation[5] have begun to identify contributing factors in these unexplained mortalities. The black rhinoceros appears to be less sensitive to common capture complications that occur during or shortly after chemical immobilization, but is more vulnerable to the effects of confinement and translocation afterward.[4] Whereas overt clinical manifestations of peracute and acute capture stress and myopathy are not apparent, physiological responses occurring soon after capture indicate otherwise. The changes in these parameters suggest an increased predisposition to the effects of further stress (e.g., trauma, nutritional changes and imbalances, oxidant stressors,* infections).[4] Based on these findings, an adaptive management approach has been adopted in Zimbabwe and Namibia that attempts to reduce the high mortality rate associated with capture and translocation. Significant improvements in management may be accomplished in these three areas: capture, boma confinement, and translocation.

CAPTURE AND TRANSPORT

Regardless of the methods used, capture and transport of the black rhinoceros should be undertaken only by experienced personnel.[3] Depending on terrain, logistics, and economic factors, rhinoceroses can be darted either from the ground or from a helicopter. Ground darting requires considerable coordination, the use of a fixed-wing aircraft, and bush skills. Helicopter darting is preferable if large numbers of animals are to be captured in a short period. Human and animal safety is of paramount importance.

Minimizing Stress

The first step in lessening the stress experienced by black rhinoceroses during capture is short, smooth induction of anesthesia. Work in Namibia has established the value of using high doses of etorphine HCl (M99, C-Vet), 4 to 4.5 mg, combined with azaperone (Stresnil, Janssen Animal Health), 250 to 300 mg, and hyaluronidase (Hyalase, Fisons Pharmaceuticals), 4500 U in producing rapid induction times of less than 4 minutes.[12] Research in Zimbabwe has

*Paglia, D and Miller, E. Personal communication, 1992.