Chapter 17

Perissodactyla

Mary Duncan[a], Patricia M. Gaffney[b,*], and Pedro Enrique Navas-Suárez[c]
[a]*Saint Louis Zoo, Saint Louis, MO, United States;* [b]*San Diego Zoo Wildlife Alliance, San Diego, CA, United States;* [c]*University of São Paulo, São Paulo, Brazil*

INTRODUCTION

Perissodactyla are hoofed ungulates with an odd number of digits. Body weight is carried on the central digit of the limb. The group includes the *Equidae*, *Tapiridae*, and *Rhinoceridae* families. The taxonomy and conservation status of these groups are listed in the Digital Content (Table 17.e1). Likewise, details of unique anatomic features, including a list of comparative dentition (Table 17.e2) and related images (Figs. 17.e1–17.e5), and lists of reported neoplasms (Table 17.e3), serologically detected pathogens (Table 17.e4), endoparasites (Table 17.e5), and ectoparasites (Table 17.e6) that expand descriptions of diseases that follow below, can be found in the Digital Content.

NON-INFECTIOUS DISEASES

Nutritional

Iron overload disease occurs in captive black and Sumatran rhinoceros, but not free-ranging or recently collected rhinoceros.[74,108,136] The mechanisms underlying iron overload in susceptible perissodactyls are currently not well understood, nor is the precise significance of the condition to overall health. Iron overload continues to be an area of active research and the pathogenesis is likely multifactorial. **Hemosiderosis** is the presence of iron in a tissue, while **hemochromatosis** indicates high levels have altered histologic structure.

After absorption by duodenal enterocytes, iron is exported from the cells into the circulation (transferrin) by ferroportin. Ferroportin production is controlled by the hormone hepcidin, which is secreted by hepatocytes. Increased serum iron and the presence of interleukin-6 (proinflammatory cytokine) cause hepcidin release. Circulating hepcidin binds ferroportin and prevents iron uptake. When hepcidin levels are high, iron remains in enterocytes and is shed in feces. Hepcidin production is increased under certain conditions, such as inflammation from infection or autoimmune disease.[42,141] Hepcidin production is down regulated in iron deficiency and with increased erythropoiesis to allow for increased iron absorption. Hepcidin deficiency leads to increased iron absorption and subsequent deposition in hepatocytes, cardiomyocytes, and endocrine glands.

Hemochromatosis, or primary iron overload, in humans is generally a heritable disorder. Mutation of the hepcidin or hemojuvelin genes results in earlier iron deposition in the myocardium or endocrine organs, while mutation of the transferrin receptor-2, ferroportin, or hemochromatosis genes results in milder later onset accumulation of iron, particularly in the liver. The hemochromatosis gene (*HFE*) encodes a protein that complexes with the transferrin receptor; abnormal gene expression leads to increased iron absorption by enterocytes. The HFE protein with transferrin receptor-2 protein can also affect hepcidin expression. Hemochromatosis gene nucleotide mutations have been identified across rhinoceros species, but a protein polymorphism has been found only in the black and not the white, greater one-horned or Sumatran rhinoceros. The S88T protein polymorphism is in the portion of the HFE protein that interacts with the transferrin receptor and may have aided iron uptake in the black rhinoceros with a low iron diet.[9,133]

Black and Sumatran rhinoceroses are browsers while white and greater one-horned rhinoceroses are grazers. The diet of wild black rhinoceroses is rich in compounds that chelate iron (tannins, phytates, polyphenolic compounds), forming insoluble complexes that pass through the gastrointestinal tract unabsorbed.[136] In captivity, lower levels of dietary iron-binding compounds may increase the amount of dietary iron that is available for absorption. Ascorbate (vitamin C) increases iron absorption, so ascorbate rich produce can increase iron uptake. The bioavailability of iron cannot be predicted simply from the measurement of total dietary iron since absorption is affected by other dietary elements (e.g., tannins, vitamin C).[170] The black rhinoceros appears genetically adapted for iron absorption from a naturally low iron diet, and overload

*Deceased.

Pathology of Wildlife and Zoo Animals. https://doi.org/10.1016/B978-0-443-18437-6.00019-5

TABLE 17.E1 Taxonomy of the Nondomestic Perissodactyls[17,59,105,130,194]

Suborder: Hippomorpha		IUCN Status
Family: Equidae		
Equus caballus	Feral, free-ranging	
Equus ferus ssp. *Przewalski's*[71]	Przewalski's/Mongolian wild horse	Endangered
Equus africanus[113]	Somali and Nubian wild asses	Critically endangered
Equus hemionus[64]	Onager, Asiatic wild ass, kulan	Near threatened
Equus kiang[166]	Eastern, Western, and Southern kiangs	Least concern
Equus zebra ssp. *zebra*[56]	Cape mountain zebra	Least concern
Equus zebra ssp. *hartmannae*[46]	Hartmann zebra	Vulnerable
Equus grevyi[162]	Grevy's zebra	Endangered
Equus quagga[72]	Plains/Burchell/Grant's zebra	Near threatened
Suborder: Ceratomorpha		
Family: Tapiridae		
Tapirus bairdii[43]	Baird's/Central American tapir	Endangered
Tapirus indicus[179]	Malayan/Asian tapir	Endangered
Tapirus pinchaque[87]	Mountain/woolly tapir	Endangered
Tapirus terrestris[180]	Lowland/ Brazilian/South American tapir	Vulnerable
Tapirus kabomani	Kabomani/little black tapir	
Family: Rhinoceridae		
Ceratotherium simum[32]	White rhinoceros	Near threatened
Diceros bicornis[33]	Black rhinoceros	Critically endangered
Dicerorhinus sumatrensis[31]	Sumatran/Asian two-horned rhinoceros	Critically endangered
Rhinoceros unicornis[29]	Greater/Indian/Asian one-horned rhinoceros	Vulnerable
Rhinoceros sondaicus[30]	Javan rhinoceros	Critically endangered

TABLE 17.E2 Comparative Dentition of the Nondomestic Perissodactyls

Taxonomic Group	Dental Formula	Notes
Equidae[59]	I 3/3C 1/1 P 3-4/3M 3/3	Females have vestigial canine teeth, males have pointed canine teeth Molars are hypsodont (high-crowned) for use on abrasive grasses
Tapiridae[20,177]	I 3/3C 1/1 P 4/3-4M3/3	Juvenile tapirs have 0–1 M teeth per arcade Young adults have 1–2 M per arcade Molars are low crowned
Rhinoceridae		
Black rhinoceros, white rhinoceros	I 0/0C 0/0 P 3-4/3-4M3/3	
Sumatran rhinoceros	I 1/0 C0/1 P 3/3M3/3	Canine and incisor teeth are vestigial
Javan rhinoceros, Asian one-horned rhinoceros	I 1/1 C0/1 P 3/3M3/3	Canine and incisor teeth are vestigial

C, canine; *I*, incisor; *M*, molar; *P*, premolar.

TABLE 17.E3 An Overview of Reported Neoplasms in Nondomestic Perissodactyls

Family/Species	Tumor	Age	Sex (M = male; F = female)	Clinical Notes	Location	Notes
Equidae						
Somali wild ass[135,189] Plains zebra[88] Cape mountain zebra[93] Hartmann zebra Onager Kulan	Sarcoid			Seen in captive and wild settings	Skin, variation in distribution with species, single to multiple tumors	Possible association with bovine papilloma viruses; fibroblastic, local invasion, possible recurrence, no metastasis
Onager[143]	Pituitary gland adenoma, pars intermedia	13 years 22 years	F F	Lameness, hyperglycemia, glycosuria, curly coat over neck Intermittent lameness	Pituitary gland	Excessive endogenous cortisol circulating to the laminae of the hoof results in laminitis; increased susceptibility to secondary infections from immunosuppression
Nondomestic equids[167]	Pituitary gland, pars intermedia dysfunction					Signs similar to those described with adenoma of pars intermedia, may suggest adrenocorticotrophic hormone testing with age
Tapiridae						
Baird's tapir[11]	Embryonal rhabdomyosarcoma	22 months	F	Nasal discharge, coughing, seizure-like behavior (possibly associated with hypoxia)	Nasopharynx	Cells negative for smooth muscle actin, multifocally positive for desmin and myoglobin
Baird's tapir[70]	Sarcoid	2 years 3.6 years	M F		Pinna Pinna	Association bovine papillomavirus 1, in situ hybridization, PCR
Malayan tapir[102]	Squamous cell carcinoma	26 years	M	Immunohistochemistry negative for bovine papillomavirus 1 Resection, chemotherapy, remission	Oral/gingiva	Oral squamous cell carcinoma is rare in horses, but may be lytic and metastasize to regional lymph nodes
Lowland tapir[4]	Cutaneous hemangioma	3 years	M	Discomfort, pruritus	Prepuce	
Baird's tapir[2]	Sebaceous adenoma	15 years	F	Free ranging	Pinna	

Rhinoceridae						
Black rhinoceros[150]	Seminoma		M	Identified by ultrasonography, hemicastration	Testis	
Black rhinoceros[153]	Acute lymphoblastic leukemia	21 months	F	Sudden onset acute dyspnea and lymphadenomegaly	Blood	Heterogeneous lymphoblastic cells
Black rhinoceros[183]	Melanoma	10 years	M	Diagnosed at biopsy	Skin	Multiple, benign, well differentiated
Greater one-horned rhinoceros[183]	High grade melanocytic neoplasm	28 years	F	Fistulous wound with intermittent exudation, swelling at defect 3 years later, biopsied. Euthanized 5 years after presentation, progressive	Coronary band	Despite anaplasia, no evidence of metastasis at necropsy, mass replaced distal phalanx. Melanoma of the digit in domestic species has a poor prognosis. Humans and horses trauma nail/hoof leads to development of melanocytic neoplasms
White rhinoceros[157]	Vaginal hemangioma		F	Episodic vaginal hemorrhage, more frequent over 10 years	Vagina	Biopsy
White rhinoceros[190]	Uterine adenocarcinoma	39 years	F	Recurrent vaginal bleeding for 7 years, respiratory distress, ill-thrift, euthanized	Uterus	Over 100 L ascitic fluid present, carcinomatosis, pleural metastasis
White rhinoceros[169]	Osteochondroma	2 years	M	Progressive lameness	Distal third metacarpus	Surgically removed. Benign, originated adjacent to the physis/subarticular, at site of endochondral ossification
White rhinoceros[8]	Pheochromocytoma	46 years	M	Possible death due to hypertensive collapse	Adrenal medulla	Well-demarcated mass, round to polyhedral cells in packets, granular cytoplasm (granules positive for chromogranin-A)
White rhinoceros[47]	Squamous cell carcinoma	42 years	F	Ulcerative skin lesion. Biopsy. Temporary regression, antiinflammatory drugs, and bandaging. Recurred 2 years later	Foot pads, bilateral, hind	No metastasis at necropsy. Squamous cell carcinoma previously reported in greater one-horned rhino at horn base and white rhino at foot and flank
White rhinoceros[81]	Squamous cell carcinoma	48 years	F	Necropsy. Euthanized aged, chronic pododermatitis, and laminitis. Dental loss	Tongue	Associated with dental loss and deposition of tartar. Found at postmortem. No alteration in food intake noted

TABLE 17.E4 Serologic Evidence of Infection in Nondomestic Perissodactyls

Family/Species	Pathogen	Geographic Location	Comments
Equidae			
Grevy's zebra[59]	Equine herpesvirus 9 (EHV-9)	Captive	Asymptomatic
	Equine herpesvirus 1, 9 (EHV-1, EHV-9)[48,84]	Free-ranging population, Kenya	Enzyme-linked immunosorbent assay. EHV-1 seroprevalence similar wild and captive populations EHV-9 wild population higher seroprevalence than captive
Przewalski's horse[59]	Equine herpesvirus 2, 5 (EHV-2, EHV-5)	Captive	No clinical signs
Plains zebra[148]	Equine herpesvirus 1, 4 (EHV-1, EHV-4) African horse sickness Equine encephalosis virus	Free ranging, South Africa	No clinical disease has been associated with these viruses in free-ranging zebra
Somali wild ass[135]	Equine herpesvirus 1 (EHV-1)	Captive, Europe	
Feral horse[128] $n=242$	Japanese encephalitis virus 73% seropositive	Free-ranging, feral Misaki, Japan	Hemagglutination inhibition test, aged individuals higher titers
Tapiridae			
Baird's tapir $n=17$[54]	High titers Venezuelan equine encephalitis 12/17. *Leptospira bratislava* 5/17 seropositive	Free ranging, Costa Rica	No serological reaction for Equine herpesvirus, and *Brucella abortus*
Baird's tapir $n=23$[194]	21% seropositive Venezuelan encephalitis H1 virus 47% vesicular stomatitis, 13% West Nile virus	Captive, Panama	Antibodies not detected to equine herpesviruses-1, -2, -4, equine influenza, and equine rhinoviruses type 1 and 2
Lowland tapir $n=65$[100]	Antibodies to 10 bacterial taxa (including high prevalence for *Leptospira interrogans*) and 5 viruses (including bluetongue, infectious bovine rhinotracheitis)	Free ranging, Brazil, 2 biomes	Eastern and Western encephalitis, porcine parvovirus seropositive in one biome only
Lowland tapir $n=10$[40]	*Toxoplasma gondii* 1/10 seropositive	Free ranging, Brazil	No serological reaction noted for equine infectious anemia, *Leptospira* sp., *Brucella abortus*
Lowland tapir $n=35$[36]	91% seropositive blue tongue virus 97% seropositive porcine parvovirus 60% seropositive *Leptospira interrogans*	Free ranging, Cerrado, Brazil	No clinical signs Agar gel immunodiffusion Hemagglutination inhibition Microscopic agglutination test No evidence of seropositivity: by enzyme-linked immunosorbent test to bovine viral diarrhea, swine fever/hog cholera; by agar gel immunodiffusion to foot and mouth disease virus, equine infectious anemia virus, bovine leukemia virus, infectious bovine rhinotracheitis virus; by serum neutralization to Eastern equine encephalitis virus, Western equine encephalitis virus, infectious bovine rhinotracheitis virus, pseudorabies virus, vesicular stomatitis virus; and by serum agglutination to *Brucella abortus*

Lowland tapir $n=125$[35]	Rabies virus No seropositivity found	Free ranging, three Brazilian biomes	Rapid fluorescent focus inhibition test (neutralizing antibodies are generally not present until clinical disease is found)
Rhinoceridae			
Black and white rhinoceros $n=281$[38]	Akabane (seropositive 59.8%), blue tongue (55%), African horse sickness (27.9%), epizootic hemorrhagic disease (19.4%), parainfluenza type 3 (25.3%), bovine herpesvirus 1 (3.1%), equine herpesvirus 1 (8.8%), bovine viral diarrhea (1.2%)	Free ranging, Republic of South Africa, Namibia, Kenya	Antibodies were not detected to Rift Valley fever virus, encephalomyocarditis virus. Geographic variation in antibody prevalence and species differences were detected. Low titers may reflect continual exposure, high titers may suggest viral replication susceptibility to infection
White rhinoceros[25]	*Coxiella burnetii* Private reserve 41.1% seropositive, National park 71.0% seropositive	South Africa, free ranging	Adults (> 7 years) higher seropositivity than subadults (2–7 years). *Coxiella* spp. have been isolated from ticks, possibility of bacterial symbionts that may cross react on seroassays. Sylvatic transmission by ticks primarily suspected
Black and white rhinoceros[77] $n=65$ black $n=58$ white	*Ehrlichia ruminantium* (heartwater) rickettsia 0.03% and 56.2% seropositive black (marked variation between sites), 75.9% seropositive white	Free ranging, Zimbabwe	Competitive enzyme-linked immunosorbent assay. Suggests rhinoceros reservoir for disease, possible concern with translocations

TABLE 17.E5 Endoparasites in Nondomestic Perissodactyls

Type of Endoparasite	Parasite Species	Host Species	Lesion
Equidae			
Nematode[126,186]	*Strongylus, Strongyloides, Parascaris, Oxyuris, Dictyocaulus*	Zebra	*P. equorum* possible increased pathogenicity zebra foals
	Strongylus vulgaris[128]	Feral horse	7/7 necropsies severe arterial lesions, hemomelasma ilei, larvae in cranial mesenteric arteries, in ileum, and cecum, adult attached to ileal mucosa. Risk factor for mortality
	Parascaris equorum[128]	Feral horse	Duodenum
	Halicephalobus gingivalis	Grevy's zebra[57]	Captive, disseminated granulomatous inflammation
Cestodes[186]	*Anoplocephala* spp.		
	Anoplocephala perfoliata[128]	Feral horse	Large intestine
Trematode[112]	*Echinococcus equinus*	Przewalski's horse Plains zebra	Captive ($n=1$), necropsy hydatid cyst liver, previously *E. granulosus* strain G4 Free ranging, Namibia, no significant disease Captive ($n=1$), necropsy hydatid cysts, lung and liver
Protozoa[159,186]	*Babesia caballi, Theileria equi*	Przewalski's horse	
	Sarcocystis spp., *Trypanosoma* spp., *Theileria equi, Toxoplasma gondii, Besnoitia bennetti*		
Bot/gadfly	*Gasterophilus*		
Tapiridae			
Nematode[28,49,80,117,124]	*Strongyloides* spp., Ascarids, *Strongylus* spp. capillarids, hookworms		
	Kililuma longipene Strongyloidea	Lowland tapir	Stomach, intestines, no gross lesions
	Neomurshidia monosticha Strongyloidea	Lowland tapir	Small and large intestines
	Monodontus nefastus Ancylostomatoidea	Lowland tapir	Stomach and small intestines. No gross findings
	Physocephalus spp., *P. meridionali, P. nitidulans* Rhabditoidea	Lowland tapir	Nodular proliferative gastritis. Free ranging, Brazil
	Probstmayria tapiri Cosmocercoidea[50]	Baird's tapir	Nematode eggs in feces
	Tapironema coronatum Trichostrongyloidea	Lowland tapir	Stomach, no gross findings reported
	Tziminema unachi Strongylidae	Baird's tapir	Cecum, colon. No gross findings
Cestode[117,124,155]	*Anoplocephala mamillana, Anoplocephaloides globiceps*	Lowland tapir	Small intestine
	Paranoplocephala sp.	Mountain tapir	Captive, incidental

TABLE 17.E5 Endoparasites in Nondomestic Perissodactyls—cont'd

Type of Endoparasite	Parasite Species	Host Species	Lesion
Trematode[117,155,193]	*Fasciola hepatica*	Lowland tapir	Death in captivity, cirrhosis
	Fasciola spp.	Malayan tapir	Eggs in feces
	Cladorchis asper (Choerocotyloididae)	Lowland tapir	Cecum
	Cladorchis pyriformis (Choerocotyloididae)	Lowland tapir	
	Schistosomiasis[193]	Lowland tapir	Granulomata liver, hemorrhagic gastroenteritis, associated with embryonated eggs
Pentastome[155]	*Armillifer moniliformis*	Malayan tapir	Nymphal cysts, wild-caught
	Linguatula recurvata[144]	Baird's tapir	Nymphs in the liver, mesentery, and small intestine
Protozoa[155,194]	*Balantidium* spp., *Giardia* spp. *Eimeria* spp.		Watery diarrhea (possibly normal flora)
	Giardia duodenalis AI genotype[92]	Lowland tapir	No clinical signs (genotype causes disease in humans)
	Babesia spp.	Malayan tapir	
	Babesia equi, suspect	Malayan tapir[194]	Icterus and anemia, recent import
	Cryptosporidium suis	Baird's tapir[194]	Watery diarrhea Captive
	Theileria equi	Lowland tapir[21]	Tick vector, intraerythrocytic
	Naegleria fowleri	Lowland tapir[91]	Meningoencephalitis, ameboid trophozoites identified by specific monoclonal antibodies
	Neospora caninum[147]	Malayan tapir	Abortus, mononuclear cell inflammation at the brain, heart, liver and lung, protozoa not seen histologically. Tissues PCR positive
	Giardia spp.[19]	Baird's tapir	
	Theileria equi	Lowland tapir	Tick vector
Rhinoceridae			
Nematode[108,155]	*Parabronema* spp. and *Dicronema versterae*	Rhinoceros	Stomach, possible tumor-like lesions
	Habronema khalili, Draschia megastoma (spirurid)	Black rhinoceros	Intestine
	Oxyuris spp. (*O. karamoja*)		Large intestine
	Probstmayria vivipara (ascarid)	Greater one-horned and white rhinoceros	
	Strongyles[131]	Greater one-horned rhinoceros	Free ranging, India; fecal egg counts
	Strongylus tremletti	Black rhinoceros	Intestine
	Strongyloides spp., *Crossocephalus* spp.	Sumatran rhinoceros	
	Grammocephalus clathrotus	African rhinoceros	Biliary system
	G. intermedius (hookworm)	Black rhinoceros	Large intestine
	Thelazid worms	Black rhinoceros	Conjunctival sacs

Continued

TABLE 17.E5 Endoparasites in Nondomestic Perissodactyls—cont'd

Type of Endoparasite	Parasite Species	Host Species	Lesion
Cestode[108,155]	*Anoplocephala* spp.		Incidental
	A. gigantea	Javan rhinoceros	
	A. vulgaris	Greater one-horned and black rhinoceros	Oribatid mite suspected cestode intermediate
	Anoplocephala spp.[131]	Greater one-horned rhinoceros	Free ranging, India; fecal egg counts
Trematode[108,155]	*Brumptia bicanda*	Black and white rhinoceros	Intestinal tract
	Paramphistomum sp.[131]	Greater one-horned rhinoceros	Free ranging, India; fecal egg counts; freshwater snail intermediate
	Fascioloides gigantica	Greater one-horned rhinoceros	Death
	Bivitellobilharzia nairi[131]	Greater one-horned rhinoceros	Nepal, schistosome more usual in elephants
Protozoa[16,97,105,108,127,155]	Trypanosomiasis *T. brucei, T. vivax,* Babesiosis	Black rhinoceros	Translocations, $n=77$; survival 52% after a year, 8.5% mortalities attributed to trypanosomiasis, and 3.4% attributed to babesiosis
	Trypanosomiasis, *T. brucei*	Black rhinoceros	Survey, translocation, $n=39$, one mortality, suggest rhino possible reservoir for disease, death on stress
	Balantidium sp., *Entamoeba* sp.	White rhinoceros	Presumed pathogenic in intestine
	Neospora caninum[163]	White rhinoceros	Abortus, multifocal hepatic necrosis with zoites. Positive polymerase chain reaction
	Gasterophilus (bot)		Bots generally esophageal region of stomach
Arthropod, larvae/bot[127,155]	*Gyrostigma pavesii, G. conjungens, Gasterophilus rhinocerontis*	African rhinoceros	Fly eggs are laid at the nares or horn base and larvae burrow into the tissue and migrate to the stomach
	Gasterophilus sumatrensis	Sumatran rhinoceros	

TABLE 17.E6 Ectoparasites of Nondomestic Perissodactyls

Family/Species	Parasite	Findings
Equidae		
Mites[186]	*Sarcoptes equi, Psoroptes equi, Chorioptes equi, Demodex equi, D. caballi*	
Ticks[186]	*Dermacentor nutalli*	Vector disease
Tapiridae		
Mites[155]	*Sarcoptes tapiri*	Pruritus, exudative dermatitis, hyperkeratosis. Progression to blindness, malnutrition and death described
	Sarcoptes tapiri, S. scabiei[92]	
Ticks	*Amblyomma testudinarium*[155]	Tapir, Indonesia
	Amblyomma brasiliense, A. cajannese, A. calcaratum, A. coelebs, A. dubitatum, A. incisum, A. latepunctatum, A. multipunctum, A. naponense, A. neumanni, A. oblongoguttatum, A. ovale, A. pacae, A. parvum, A. scalpturatum, A. tapirellum, A. pseudoconcolor, A. triste, Haemaphysalis juxtakochi, Dermacentor halli, D. latus, D. (Anocentor) nitens, Ixodes bicornis, I. boliviensis, I. scapularis, I. tapirus, Rhipicephalus (Boophilus) microplus, Ornithodorus rudis, O. tuttlei[92]	
	Ixodidae[124]	Lowland tapir, Brazil
Blood-sucking moth[155]	*Calpe eustrigata*	Only observed feeding on captive tapir and Sumatran rhino in Malaysia
Rhinoceridae		
Nematodes[75,123,127,155]	*Stephanofilaria dinniki*	Crusty skin disease, free living, Africa, nematode. Absence of disease in captivity thought to require an insect vector
	Habronema spp.	Possible larvae cause periocular lesions
Ticks[155]	*Amblyomma hebraeum, A. sparsum*	Black and white rhinoceros, tick is vector for *Ehrlichia ruminantium*, heartwater
	A. rhinoceros, A. gemme, A. marmorem	Black rhinoceros
	A. testudinarium	Rhinoceros, Indonesia
	A. crenatum	Javan rhinoceros
	A. infestum, Hyalomma walkeriaeirii, Aponomma spp., *Haemaphysalis* spp.	Sumatran rhinoceros in Malaysia
	Rhipicephalus ayrei, R. superitus, Cosmiomma hippopotamensis, Dermacentor spp., *Haemaphysalis* spp., *Hyalomma* spp.	Black rhinoceros, Kenya Ticks locate especially in folds, greatest problem as disease vectors
	D. rhinocerinus	White rhinoceros on importation
Biting flies[108,155]	*Lyperosia* spp., *Rhinomusca* spp., *Tabanus* spp., *Glossina* spp.	*Glossina* spp. are the vector for trypanosomiasis
	Chrysomyia bezziana	Old World screw worm

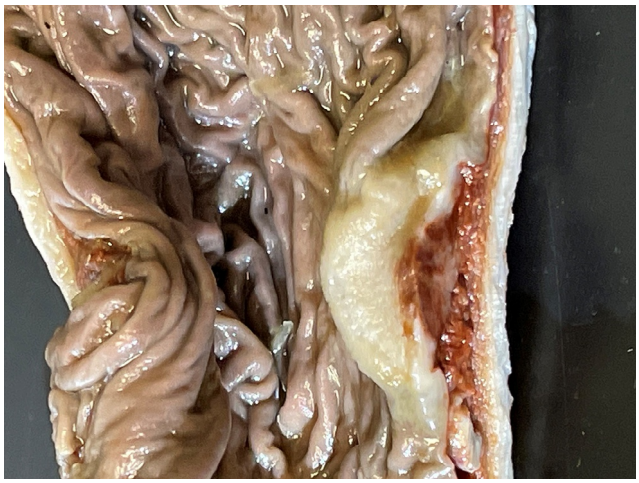

FIGURE 17.1 Hemosiderosis (iron overload) in the duodenum of a black rhinoceros. Iron accumulation appears grossly as red/brown deposits in the submucosa, particularly in the region of the common bile duct opening. This corresponds histologically to hemosiderin-laden macrophages (see Fig. 17.2; eSlides 17.e1 and 17.e2).

develops with loss of homeostasis in captivity when the diet has high iron bioavailability.[9,42]

In captivity, iron accumulation is typically seen in hepatocytes and histiocytes of the spleen, liver, small intestine (Fig. 17.1; eSlides 17.e1 and 17.e2), and lungs of black rhinoceros, the histiocytes of the spleen, and other organs in greater one-horned rhinos, and the histiocytes of the intestines, liver, and lung in white rhinos.[133] The presence of hemosiderin in histiocytes is consistent with secondary iron overload.[44] The captive white and greater one-horned rhinoceros do not demonstrate ferritin and tissue iron elevations suggestive of iron storage.[105] In hepatic hemochromatosis (iron accumulation associated with cellular degeneration, necrosis, and fibrosis) both parenchymal and reticuloendothelial cell lines are affected and cellular and tissue structure is altered. Initially, hemosiderin is deposited in periportal hepatocytes but over time increased iron staining occurs throughout the hepatic lobule and is prominent in both hepatocytes and Kupffer cells (Figs. 17.2A–D; eSlides 17.e1 and 17.e2); in hemochromatosis, periportal fibrosis develops (Figs. 17.2C, D). Iron accumulation in the bone marrow may cause myelodysplasia; marrow is hypocellular and is often fibrotic with sheets of hemosiderin-laden macrophages.[170]

The ability to assess and monitor the development of iron overload in living animals has been an ongoing quest. Serum haptoglobin levels are similar in both white and black rhinoceros suggesting iron accumulation in the black rhinoceros is unlikely secondary to hemolysis. Hepatic nonheme iron in black rhinoceroses in captivity has been shown to be significantly higher than that in white rhinoceroses, and the level in black rhinoceroses increased with age.[160,170] Measurement of serum ferritin using an enzyme-linked immunoabsorbent assay (ELISA) with antibodies to equine ferritin, which cross-react with rhinoceros ferritin, is thought to be a good estimate of total body iron stores in rhinoceros.[170] A Sumatran rhinoceros species-specific ferritin assay was developed due to inconsistent results with the equine assay. Ferritin concentration was found to vary between individuals and did not correlate well with iron overload or illness.[161] The Sumatran rhinoceros ferritin assay was subsequently validated for use in black rhinoceros. In one study, as in Sumatran rhinoceros, serum ferritin concentrations varied between individual black rhinos, and there were no correlations between serum ferritin and sex, season, age, or length of time in captivity. Ferritin is an acute-phase protein that is released in response to inflammatory and neoplastic processes; it is not specific to iron overload disease. The usefulness of serum ferritin to monitor iron overload in browsing rhinoceros appears limited.[191] Transferrin iron binding capacity has been used as a marker of body iron status, but accurate measurement has been difficult in rhinoceros.[151] Nontransferrin-bound labile plasma iron has also been assessed as a potential biomarker of iron overload, but while all Sumatran rhinos positive for labile plasma iron showed clinical signs of overload, positive black, white, and greater one-horned rhinoceros appeared healthy.

Iron overload is associated with a variety of diseases. Free iron catalyzes the production of hydroxyl free radicals, and iron overload disease may therefore predispose black rhinoceros to hemolytic anemia, mucocutaneous ulcerative diseases, and stress intolerance.[136,170] Iron accumulation disrupts physiological functions and increases susceptibility to many infectious diseases. At normal levels, iron is captured by the host and bound to protein, which deprives pathogens of this essential cation. However, organisms such as mycobacteria thrive in iron-rich environments and leukocyte phagocytic and bactericidal properties are compromised with iron excess. The influence of iron overload in infectious disease is further complicated by the form of overload (primary or secondary) that is present, with histiocytes as the main iron storage cell in secondary overload.[69,160]

Further research is needed into iron overload in tapirs as the topic has been studied less extensively. Captive tapirs develop higher serum ferritin and increased transferrin saturation (lowland > Baird's > Malayan) relative to free-ranging Baird's tapirs.[12,137,146] Hemosiderosis has not been described in free-ranging lowland tapirs.[124]

Microcytic, hypochromic iron deficiency anemia has been reported in captive Malayan tapir neonates from two institutions.[52] Both had unremarkable neonatal examinations and blood parameters but developed anemia by 1 to 2 months of age. Notable hematologic findings were anisocytosis, polychromasia, and hypochromia with acanthocytes, schistocytes, and poikilocytes. Stores of iron are

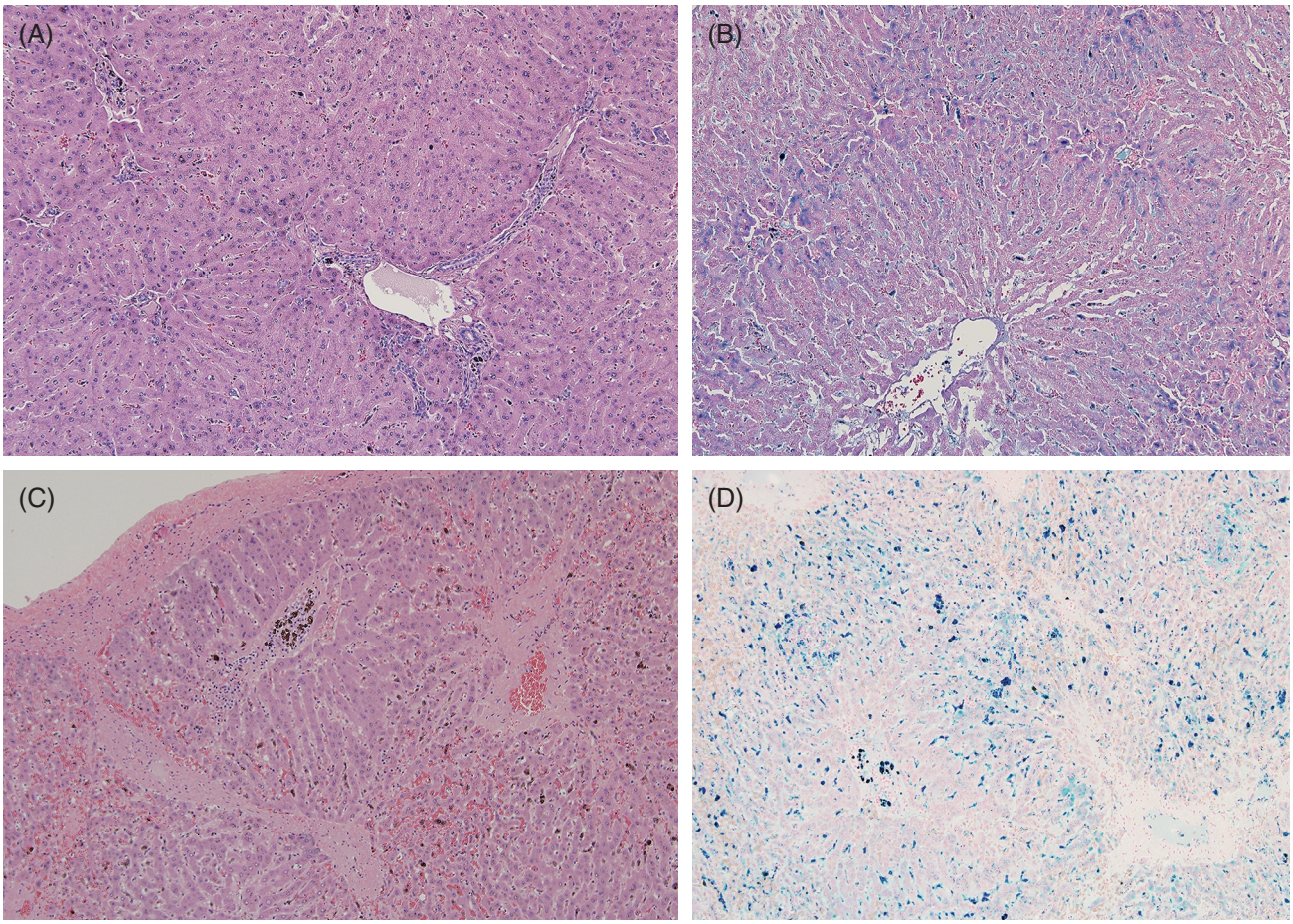

FIGURE 17.2 Mild hemosiderosis (iron overload) (A, B) and mild hemochromatosis (C, D) in the livers of black rhinoceros. (A) Mild iron accumulation appears as granular brown intracytoplasmic pigment in intrasinusoidal Kupffer cells. (B) Intracellular iron is highlighted with special stains. Perl's iron/Prussian blue. (C) Moderate iron accumulation is associated with increased numbers of periportal, pigment-laden Kupffer cells and periportal and perivascular fibrosis. (D) With special staining, the accumulation of iron can be seen to be more widespread in mild hemochromatosis than mild hemosiderosis. Perl's iron/Prussian blue (see Fig. 17.1; eSlides 17.e1 and 17.e2).

low at birth, milk has low iron content, growth is occurring rapidly, and limited access to iron sources, such as soil, may all be factors in the development of iron deficiency in these young animals.

Copper deficiency has been diagnosed in tapirs.[60,61] Although the diet may appear to have adequate levels, interaction between copper and other trace elements, such as iron, zinc, sulfur, or molybdenum, may be responsible for low bioavailability. Stillbirths and light haircoat, seen in other species with copper deficiency, have been seen in tapirs with low serum copper levels.

Hypovitaminosis D is of interest across multiple species. This fat-soluble vitamin is involved in many metabolic pathways and disease processes. Circulating 25-hydroxyvitamin D is the best indicator of vitamin D in the body. Vitamin D_3 is at least 3 times more effective than vitamin D_2 in maintaining 25-hydroxyvitamin D values. Ergocalciferol (vitamin D_2) comes from the diet, plant material. Cholecalciferol (vitamin D_3) is endogenously produced in the skin on exposure to ultraviolet B radiation. Levels vary with distance from the equator and levels of sunlight and are reduced by barriers such as glass. Relative to free-ranging black rhinoceros (Zimbabwe), two individuals in managed care in North America had low circulating 25-hydroxyvitamin D. Exposure to sunlight was shown to be highly significant, despite dietary supplementation. Of note is that in other hindgut fermenters, bone growth and calcium homeostasis appear to occur independently of vitamin D.[132]

Vitamin E (alpha-tocopherol) is important in maintaining membrane integrity relative to oxidant stress. **Hypovitaminosis E** as a cause of hemolysis has been reported in primates, rats, and horses. It also causes low bile salt secretion with reduced solubilization of dietary fat. It has been implicated in **hemolytic anemia** and **encephalomalacia** in black rhinoceros and **myopathy** in a Brazilian tapir.[193] Free-ranging black rhinoceros are reported to have significantly higher plasma vitamin E than those in

managed care. The lower levels in the latter are suggested to be due to lack of access to fresh browse/grazing.[23] One study showed poor efficacy of absorption of vitamin E with most dietary supplement formulations, which may also contribute to lower vitamin E in managed care settings.[142] Grazers and browsers may have different dietary requirements for vitamin E. Higher vitamin E has been measured in the leaves relative to the twigs of the same browse plants in Zimbabwe.[22] Vitamin E increases with chlorophyll degradation during plant maturation and would be expected to be higher in photosynthesizing leaves.

Degenerative myelopathy in captive Przewalski's horses is associated with hypovitaminosis E due to low dietary alpha-tocopherol in feed.[86] Clinical presentation varies from mild ataxia to wide-based gait and stance, and uncoordinated movements of the hind limbs. Gross or histologic lesions in cardiac or skeletal muscle are not noted. The most significant lesions are degeneration, demyelination, and astrogliosis of the ventral and lateral funiculi in the caudal cervical and cranial thoracic spinal cord. Axons in the gray matter throughout the cord are swollen and degenerate. Plasma alpha-tocopherol concentration in affected horses is less than 0.03–0.08 mg/dL (normal >0.5 mg/dL; < 0.3 mg/dL is considered deficient). Similar demyelinating lesions are seen in humans with vitamin E malabsorption due to chronic liver disease and laboratory animals fed vitamin E-deficient diets.

Metabolic

Exertional rhabdomyolysis (capture myopathy) is a potential problem in all perissodactyls but the condition is of greatest concern during and in association with capture and translocation of rhinoceros.[97,149] Lesions are consistent with those seen in other species (see Bovidae, Antilocapridae, Giraffidae, Tragulidae, Hippopotamidae and Cervidae).

Toxic

A number of chemical and biological toxins have been reported in nondomestic Perissodactyls. Most are single case reports or series (Table 17.1). In one notable toxicological study of free-living lowland tapirs in Brazil, carbamates (aldicarb, carbaryl), organophosphates (diazinon, malathion, mevinphos), pyrethroids (deltamethrin, permethrin), and toxic metals (cadmium, lead) were detected in a variety of tissues, but no significant effects were linked to these findings.[99]

Congenital/Genetic

Adenomatous hyperplasia of the placenta (Fig. 17.3) has been seen in greater one-horned rhinoceros. Nodules are composed of variably sized glandular or cystic structures lined by plump cuboidal to low columnar epithelium or epithelial fronds with various amounts of fibrous stroma. The change has not been associated with placentitis or abortion.

Familial degenerative myelopathy was reported in 8 of 17 Plains zebra foals, the progeny of one stallion and two mares.[116] Foals developed progressive ataxia between 4 and 7 months of age. This was not associated with radiographic or postmortem findings to indicate narrowing of the vertebral canal which can result in spinal cord damage. Histologically, degeneration of ascending and descending tracts with bilaterally symmetrical demyelination was found in the spinal cord at the lateral and ventral funiculi. The entire cords were examined in two zebra and lesions were seen in the cervical, thoracic, lumbar, and sacral regions. Lesions also extended cranially to the medulla oblongata. Small blood vessels in the lesions showed fibrosis in the adventitia. No histologic lesions were found in the brains.

Skeletal, cardiac, and neurologic congenital defects have been reported in nondomestic perissodactyls. A **schistosomus reflexus-like malformation** was reported in a white rhino calf that was aborted at approximately 13 months of gestation.[83] The fetus exhibited spinal inversion and a ventral midline abdominal defect with externalization of the abdominal viscera. Computed tomography revealed severe scoliosis, spiral spinal rotation, multiple vertebral anomalies, hypoplasia of multiple bones of the skull, and mild prognathism. In cattle, autosomal recessive inheritance is suspected to cause the defects based on case clustering around certain bulls. The white rhino dam was captive born and had successfully delivered two normal calves sired by the same bull prior to the abortion of the affected fetus.

Congestive heart failure as a result of **persistent truncus arteriosus**, and **ventricular and atrial septal defects** has been reported in a neonatal black rhinoceros.[156] Hypoplastic pulmonary arteries and secondary right and left ventricular dilation were also noted. Persistent truncus arteriosus is characterized by a single arterial trunk arising from the ventricular outflow tract to supply the pulmonic, coronary, and systemic circulations. A septum dividing the truncus arteriosus into the aorta and pulmonary artery failed to form. The cause of persistent truncus arteriosus may be multifactorial. It is more common in certain breeds and certain human families in which it may have monogenetic inheritance. Another black rhinoceros calf that died less than 12 h after birth had congenital defects including **cardiac septal defects**, **cleft palate**, and **patent foramen ovale**.[85]

Atresia ani is a congenital embryological anomaly in which the hindgut fails to fully communicate with the perineum. The anus may be either stenotic or imperforate. Atresia ani may appear alone or in combination with rectovaginal or rectovestibular fistula. Atresia ani has caused

TABLE 17.1 Toxins Reported in Nondomestic Perissodactyls

Family/Species	Toxic Agent	Comments
Equidae		
Plains zebra[126]	Organophosphate	Postdeworming ($n=1$)
Grevy's zebra[188]	Red maple *Acer rubrum*, includes hybrid trees. Seasonal—wilted/dried leaves, gallic acid, precise toxin unknown	Hemolytic anemia, methemoglobinemia, (oxidative damage erythrocytes) $n=2$ females. Heinz body formation, leukocytosis, icterus, hemoglobinuric nephrosis, paracentral hepatic necrosis
Przewalski's horse[114]	Sycamore seed *Acer pseudoplanatus* Sycamore/sycamore maple. Toxin is hypoglycin A	Equine atypical myopathy $n=1$ foal. Sudden onset neck stretching, progressive weakness. Increased creatine kinase, increased aspartate aminotransferase, mild increase in inorganic phosphorus, neutrophilia. Brown urine—increased protein and hemo/myoglobin Multiple acyl co-A dehydrogenase deficiencies were indicated. Fatty acid oxidation in muscle mitochondria Rhabdomyolysis striated and cardiac muscles
Rhinoceridae		
Black rhinoceros[97]	Organophosphate	Degenerative myopathy, $n=2$
Black rhinoceros[39]	Vitamin D_3	Diet compounding error, diet 13–38 times expected vitamin levels; 3/10 individuals developed hypercalcemia and partial anorexia, and died over a 5-month period; of these 2/3 had mildly elevated serum 25-hydroxyvitamin D_3, 1/3 marked increase liver 25-hydroxyvitamin D_3
Rhinoceros, including white rhinoceros[105,108]	Blue-green algae *Microcystis aeruginosa*	Periodic algal blooms associated with high organic matter and low water levels. Random massive acute hepatic necrosis
Black rhinoceros	Creosote from coal tar, cresols, associated wood preservatives	20 wild-caught in creosote treated pen for >1 month. 4/20 became lethargic, anorexic, anemic, icteric. Increased aspartate transaminase, bilirubinemia, dark brown urine. Two of the four died with icterus, ulcers oral cavity and stomach, large cholestatic (green) liver, and erythroid hyperplasia of marrow at necropsy. Acute periacinar liver necrosis. Post translocation five more died with similar liver lesions[78] Two captive rhinoceros developed protracted anemia and died, ulcerative skin lesions, possible icterus, suspected creosote toxicity. Degenerative hepatopathy with green/brown pigmentation. White rhinoceros similarly housed unaffected. Fencing old telephone poles[165]
Black rhinoceros[140]	Anthracycline (doxorubicin) chemotherapy	Juvenile treated for acute lymphoblastic leukemia. Developed congestive heart failure with ventricular dilatation, congestion of lung, liver, and spleen. Doxorubicin-induced cardiomyopathy identified in humans

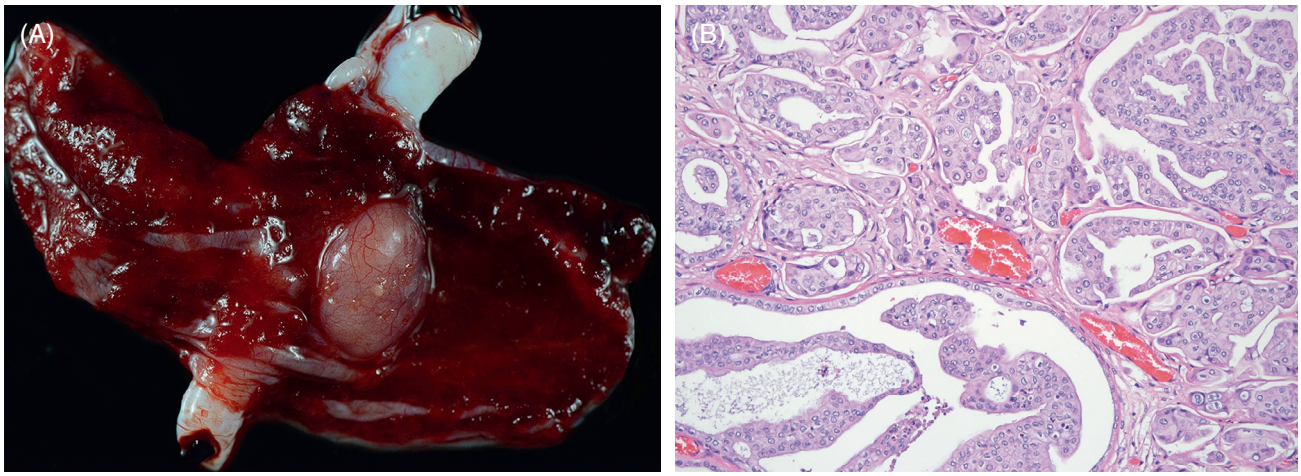

FIGURE 17.3 Adenomatous hyperplasia in the placenta of a greater one-horned rhinoceros. (A) These can be grossly inapparent or appear as nodular foci. Tissue in this case is light pink/tan and the surface is covered by multiple prominent vessels. (B) Nodules are composed of variably sized glandular or cystic structures lined by cuboidal to low columnar epithelium or epithelial fronds with various amounts of fibrous stroma.

death in neonatal tapirs; abdominal distention has been seen, but associated fistulae have not been reported.[60] Other congenital abnormalities in perissodactyls include **albinism** in a free-living lowland tapir in Brazil,[178] **leucism** in a lowland and a mountain tapir,[129,176] and coccygeal scoliosis, unilateral microphthalmia, and optic nerve hypoplasia in a Grevy's zebra foal (Figs. 17.4A–C).

Age-Related/Degenerative

Histologic features of **chronic renal disease** in perissodactyla are similar to those in other species and include mononuclear interstitial nephritis, fibrosis, tubular dilation, and tubular degeneration and loss (Fig. 17.5A) and pyelonephritis (Fig. 17.5B). Secondary changes, such as uremic mineralization in the lungs (Fig. 17.5C) or stomach (Fig. 17.5D) and gastric ulcers (Fig. 17.5E), are seen. As with sequelae to renal disease in many species, blood and urine analysis can be used for clinical monitoring. Management with a high-energy, low-protein diet may help.[105]

Degenerative joint disease is frequently seen in older animals under managed care. In a review of the osteopathology of rhinoceros, skeletons and cadavers from 27 rhinoceroses of various species were examined.[158] Twenty-two individuals had bone lesions in at least one limb. Six categories of lesions were identified with the following frequencies: enthesopathies 20/27, osteoarthritis 15/27, bone remodeling 12/27, osteitis/osteomyelitis 3/27, fracture 3/8, and subluxation 3/8. Entheses are the sites of ligament and tendon attachment to the bone surface. Response to injury at these sites includes new bone deposition. Enthesopathies were present at the dorsoproximal phalanges in particular. Osteoarthritis was most common at the distal interphalangeal joints.

Dental disease is common in aged perissodactyls. Tooth loss may be associated with tartar accumulation and gingivitis due to insufficient browse in captive diets in rhinoceros.[108] Dental disease, **mandibular and maxillary osteomyelitis**, and **facial abscesses** are common in aged tapirs. The lesions are thought to be initiated by rough feed that causes mechanical trauma and enables pathogen entry. Surveys of dentition in museum specimens have indicated markedly different patterns of wear and lesions between Malayan and lowland tapir species. Malayan tapirs are selective browsers and feed on soft vegetation. In skulls and mandibles from Malayan tapirs, dental lesions in one study were identified in 52% of the captive and 6% of the free-ranging tapirs.[20] Lesions were most common at the second, third, and fourth premolars and the first molar teeth and consisted of resorption with loss of radiodensity extending into the tooth at the cementoenamel junction. The subgingival location suggested a metabolic rather than an external etiology. Mandibular teeth were more commonly affected than maxillary teeth. Age and captivity were positively correlated with lesion development. In a study of the skulls of lowland tapirs, 24% had lesions with similar prevalence in free-ranging and captive animals.[177] The most common problems were dental fracture with associated periapical reaction in 15% of skulls, periapical reaction without dental pathology in 13% of skulls, and dental fracture with no periapical reaction in 4% of skulls. One skull from a free-ranging lowland tapir had caries-like lesions. The lesions in the lowland tapirs were most consistent with dental trauma. Increasing age was positively correlated with lesion development.

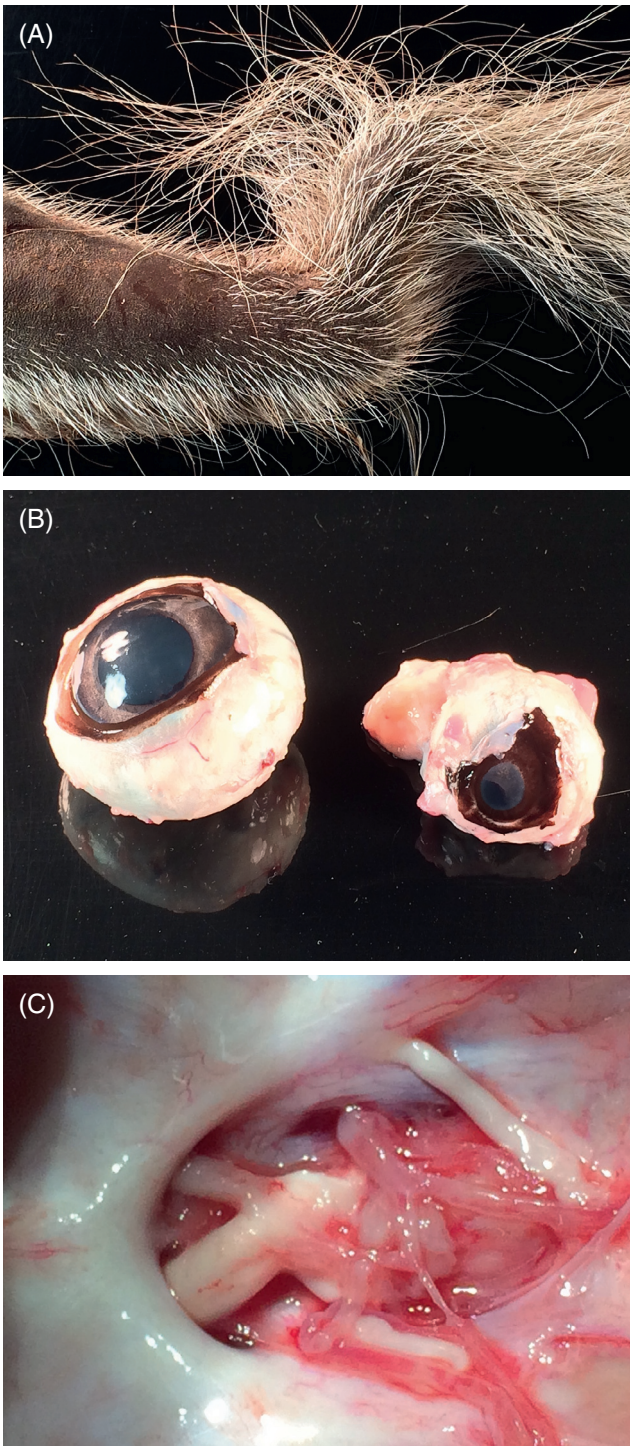

FIGURE 17.4 Multiple congenital defects in a Grevy's zebra foal. (A) Coccygeal scoliosis. There is sideways curvature of the vertebral column. (B) Unilateral microphthalmia. The eye on the right in the image is significantly smaller than the normal contralateral eye. (C) Unilateral optic nerve hypoplasia. In this image of the optic chiasm, the abnormal left optic nerve (upper left) is notably smaller than the normal right optic nerve (lower left).

Inflammatory, Non-Infectious

The density of hair follicles is low in rhino skin and there is an increased prominence of epidermal rete ridges. The dermis is composed of thick, coarse dense collagen (reticular dermis). When hemorrhage occurs in the superficial dermis, erythrocyte exocytosis through the epidermis may result in subcorneal hemorrhage or hemorrhagic crusts.[119]

A variety of skin diseases affect captive black rhinoceros. Some have frequent episodes of ulcerative skin/mucocutaneous disease alone or in association with other disease problems.

These include **superficial necrolytic dermatopathy** (also known as **superficial necrolytic dermatitis, necrolytic migratory erythema, vesicular and ulcerative dermatopathy, metabolic epidermal necrosis, mucosal and cutaneous ulcerative syndrome, hepatocutaneous syndrome**, and **ulcerative skin disease**). With this condition, individuals develop lesions of variable severity.[118] Animals of all ages can be affected. Epidermal plaques progress to vesicles or pustules that erode or ulcerate. Lesions are generally bilateral, possibly symmetrical, with a tendency to form at pressure points. Common sites are the lateral body surfaces, coronary bands, tail tip, and ear margins. Oral and nasal lesions are seen and may be more persistent. Affected rhinoceroses appear depressed, anorexic, and lose weight. Hypoalbuminemia, hypocholesterolemia, and decreased hematocrit may be found reflecting loss of albumin and blood through the skin. Similar gross and histologic lesions in domestic species are associated with hyperglucagonemia or hypoaminoacidemia. Low circulating amino acids disrupt epidermal homeostasis causing ulceration or increased susceptibility to injury. Glucagonoma, diabetes mellitus, hepatic disease, hyperadrenocorticism, and hypothyroidism have been linked to the skin lesions in domestic species. Hypoaminoacidemia was not confirmed in a study of black rhinoceros in which monthly plasma levels were assessed.[26]

Histologically, lesions have a characteristic layered "red-white-blue" appearance: superficial parakeratosis with eosinophilic keratinocytes retaining nuclei (red) overlying hydropic degeneration in the stratum spongiosum, spongiosis, and intraepidermal vesicles resulting in pallor (white), and acanthosis of the deep stratum spongiosum (blue). Intracytoplasmic inclusions seen in some cases are keratin intermediate filament aggregates as determined by electron microscopy. Dermal inflammation develops following ulceration.[119]

Epidermal exfoliation may be a variant of superficial necrolytic dermatopathy and is associated with shedding of superficial hyperkeratotic epidermis from the thorax

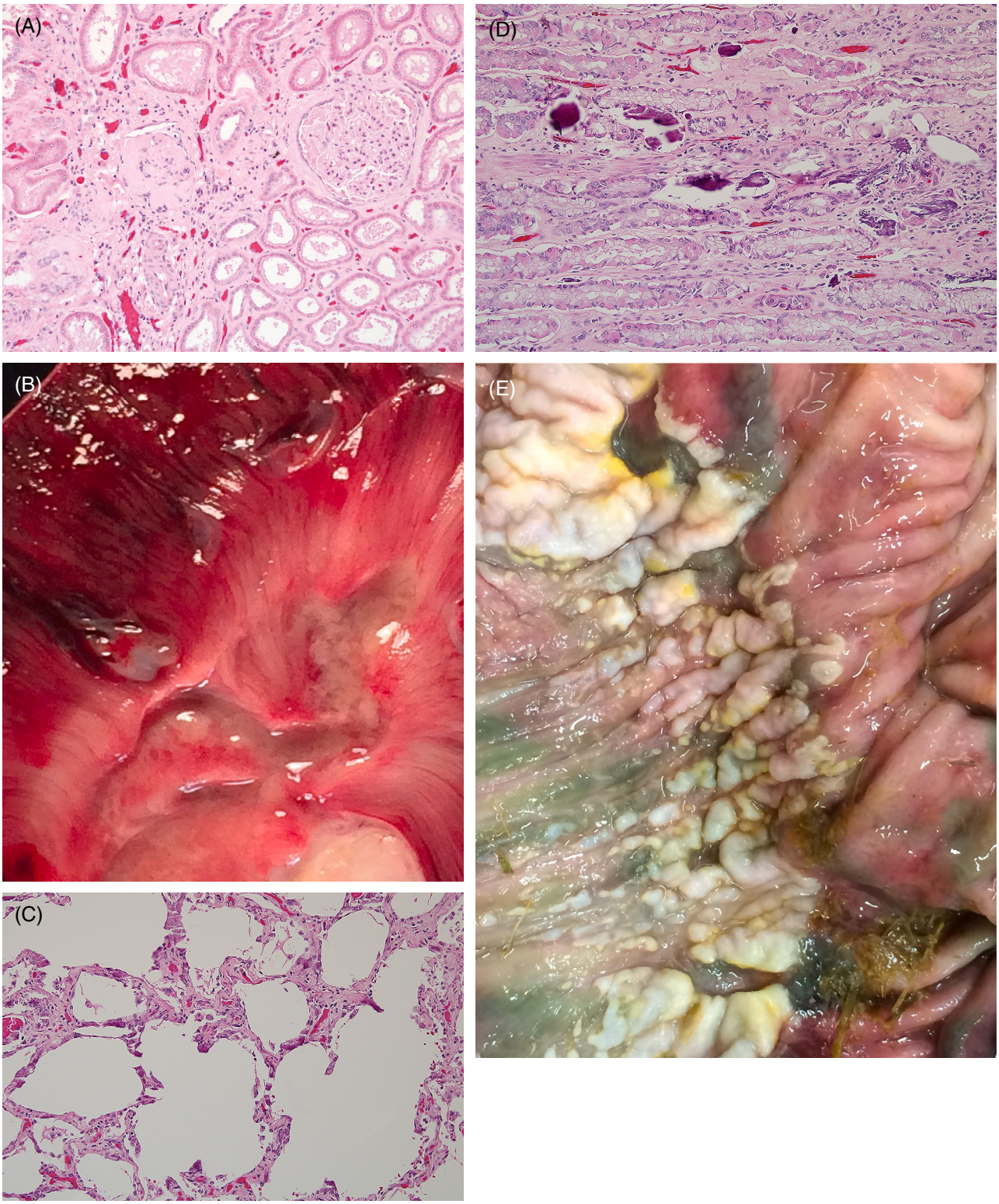

FIGURE 17.5 Chronic renal disease in a black rhinoceros (A, C, D) and Grevy's zebra (B, E). (A) Chronic nephritis is associated with interstitial and periglomerular fibrosis, glomerular fibrosis and senescence, mild multifocal tubular dilation, and multifocal tubular drop-out. (B) Pyelonephritis can appear grossly as fine tan streaks that extend from the renal pelvis to the cortex. Complications of chronic renal disease can include (C) pulmonary and/or (D) gastric mineralization (basophilic deposits) secondary to uremia and (E) gastric ulceration at the margo plicatus.

and flanks that leaves a shiny gray surface. Intraepidermal pustules are seen occasionally with no further epidermal degeneration (pustules are not a component of superficial necrolytic dermatitis). Rarely, coagulative necrosis of the epidermis may occur in association with neutrophil accumulations and colonies of cocci. Dermal inflammation is seen in these lesions.

Nodular collagen degeneration with dystrophic mineralization alone or in association with chronic ulcers has been reported in the rhinoceros. Macrophages and multinucleate giant cells may be associated with these lesions. The etiology is currently unknown. Similar changes are seen in horses with arthropod-induced injury and dogs with hyperglucocorticoidism.[119]

Eosinophilic granulomas have been described in eight captive black rhinoceros. Affected sites are the oral and nasal cavities and the skin (frequently behind the prehensile upper lip). Gross lesions appear as proliferative, exophytic masses that are prone to trauma. Histologically, the epidermis is proliferative, acanthotic, and often ulcerated. The dermis contains prominent infiltrates of eosinophils, collagen degeneration with flame figures, and marked neovascularization.[119,145] Lesions can resolve spontaneously, and corticosteroid and cryotherapy have been used to resolve lesions effectively. The condition does not appear to be seasonal or occur in related individuals. It can recur and may be seen concurrently with superficial necrolytic dermatopathy. The etiology is currently unknown, but hypersensitivity as a primary cause is suspected. Recent cases in rhinoceros have responded to antihistamines.[10]

Seasonal allergic dermatitis has been reported in related greater one-horned rhinoceros. The lesions were eosinophilic and consistent with a hypersensitivity reaction. Intradermal skin testing and allergen-specific IgE serum testing (the latter not validated for rhinoceros) identified specific allergens for hyposensitization treatments, which resulted in a positive response.[14]

Vesicular skin disease is described in tapirs. Lesions develop most commonly in the dorsal cervical and lumbosacral areas and resolve spontaneously.[37,61] Erythematous papules form, coalesce, rupture, and slough leaving ulcers. Vesicles are subepidermal and may contain neutrophils and eosinophils. The overlying epidermis is spongiotic with superficial to full-thickness necrosis. Follicles are unaffected. A narrow zone of degenerate collagen is present in the superficial dermis below the vesicles; perivascular edema and hemorrhage are present in the dermis. The histologic features are consistent with a number of conditions **including bullous pemphigoid, dermatitis herpetiformis**, junctional form of **erythema multiforme**, and the acute hemorrhagic form of **staphylococcal hypersensitivity**. However, lesion distribution and disease epidemiology in tapirs is inconsistent with those found in other species. Sloughing and repair occur approximately a week after lesions first develop. Resolution may be associated with fluctuating hormone levels in females.[194] Viral particles have not been identified by electron microscopy in vesicular fluid and virus was not isolated from equine fibroblast culture. In one survey, tapir (Malayan, lowland, Baird's) with similar clinical histories had biopsies consistent with vesicular skin disease. In Baird's tapir, the lesions were more lateral and pruritic. Many animals also exhibited neurologic signs, such as hindlimb ataxia, lameness, weakness, and syncopal episodes during periods of skin disease. Many affected tapirs also have chronic intermittent respiratory infections.

A survey of free-ranging Baird's tapir in Costa Rica identified **depigmentation of undetermined etiology** (also known as **leukoderma** or **vitiligo**) along the dorsum, perineum, inguinal, and perioral regions.[54] Histologically, the epidermis was hyperplastic with little pigmentation. Mild mononuclear cell deposits were present in the superficial dermis and there were perivascular eosinophils. Hairs were generally in catagen or telogen stage. Histologic lesions were consistent with hypersensitivity dermatitis; a specific etiologic agent was not identified but a response to biting insects was suspected. In a somewhat similar case, a captive black rhinoceros developed nonpigmented foci initially at the nares and lips and subsequently multifocally on the limbs, head, and trunk. Multifocal hypopigmentation of the basal layer of the epidermis with pigmentary incontinence and perivascular lymphohistiocytic dermatitis were seen. Phototherapy was used to induce repigmentation.[173]

In the 1980s and 1990s, 44 episodes of **acute intravascular hemolytic anemia** occurred in 36 captive black rhinoceros in North America, Europe, and Japan. Among them, 23 individuals died (~64%).[108] Early cases were characterized by hemoglobinuria, lethargy, and severe anemia. Rare animals survived episodes with supportive care.[27,109] Investigated causes of hemolytic anemia included autoimmune hemolytic anemia, equine infectious anemia, equine viral arteritis, copper toxicosis, vitamin E deficiency, and clostridial infection.[108] Mortality due to hemolytic anemia occurs in free-ranging rhinoceros in association with babesiosis and trypanosomiasis; however, none of these infections were confirmed as contributing to hemolytic anemia in affected captive black rhinoceros. Leptospirosis was diagnosed in many of the affected captive black rhinos and may have played a role in the pathogenesis of hemolytic anemia. Iron deposition in parenchymatous organs is a general finding, originally thought to be evidence for hemolytic anemia (see iron overload disease discussed earlier).[136,139] The relationship between hemolytic anemia and iron accumulation in tissues is still poorly understood.[170] Histologic findings in affected rhinos included renal tubular casts with red blood cells and protein; hepatic centrilobular fatty change and necrosis were also seen.

Hemolytic syndromes in humans are commonly caused by **enzyme defects** that impair metabolic pathways required to neutralize oxidants in red blood cells. These include hereditary deficiency of glucose 6 phosphate dehydrogenase as the most common defect and bacterial or viral infections that lead to secondary oxidant stress.[138] Several factors, including 6 phosphate dehydrogenase, erythrocyte catalase activity, erythrocyte adenosine kinase activity, and catalase and glutathione *S*-transferase, have been suggested in the pathogenesis of hemolytic syndrome cases in black rhinoceros. However, to date, no definitive pathogenesis has been elucidated to explain cases of hemolytic anemia in rhinos. Unlike humans, erythrocyte glucose 6 phosphate dehydrogenase activity in affected black rhinos is normal.[139] Erythrocyte catalase activity levels are similar in black rhinos with or without hemolytic anemia and are similar among black, white, greater one-horned, and Sumatran rhinos.[141] Low erythrocyte adenosine kinase activity (important for maintaining ATP levels),[139] hypophosphatemia (inhibits glycolysis, reducing the amount of available ATP),[107] and low erythrocyte ATP levels (relative to humans) have all been suggested as possibly predisposing red cells to oxidative stress and hemolysis.[139] Low ATP in rhino red blood cells may be rate limiting in the antioxidant activity of the hexose monophosphate shunt, which is important in the neutralization of oxidative byproducts and may contribute to mucocutaneous ulcerative disease and acute episodic hemolysis.[138] Recent cases have been rare; though further investigation of oxidative stress in black rhinoceros may inform procedures for conservation transportation events.[149]

Suspected **neonatal isoerythrolysis (syn. Hemolytic icterus)** has been reported in Baird's tapirs.[184] Neonatal isoerythrolysis is a form of immune-mediated hemolytic anemia in which maternal antibodies to red cell components inherited from the sire bind to and destroy the neonate's red blood cells. The dam develops an immune response to fetal red blood cells during her first pregnancy with an individual sire, so isoerythrolysis only occurs in subsequent offspring. Clinical signs include lethargy, inappetence, icterus, and red urine. Hemolysis in tapir foals fed equine colostrum has been reported and may be due partly to natural antibodies to erythrocytes of a different species. It is unknown whether equine colostrum is an appropriate replacement for tapir colostrum.

Idiopathic hemorrhagic vasculopathy syndrome has been reported in captive black rhinoceros in the United States. No sex bias or tendency to develop in wild-caught versus captive-born animals was noted. Signs included locally extensive swelling of the neck and limbs, lethargy, laminitis, and sloughing of the nails. Nonhemolytic anemia is seen; hematocrit and serum phosphorus fall rapidly in affected animals. Histologically, neutrophilic vasculitis in the superficial limb dermis, proliferative vasculitis in the oral mucosa, and extravasation from small vessels suggestive of immune-mediated disease are seen. Extensive testing to identify an infectious agent has been unproductive, and the cause remains unknown.[122,141] Equine purpura hemorrhagica, a sporadic noncontagious allergic reaction to streptococcal or viral antigens, appears to be the most similar condition.

Hoof lesions, such as **laminitis** and foreign bodies at the white line resulting in abscess formation, have been reported in captive equids including Somali wild ass.[135] In captive rhino high-protein diets have been suggested as a cause of laminitis.[108] Additional predisposing conditions may include excessive feeding of concentrates, enteritis, chronic renal failure, and acute intravascular hemolytic anemia. Laminitis occurs when the blood supply to the corium is interrupted. In severe cases, the union between the horny and sensitive laminae breaks down, progressing to separation of the nail at the coronary band. Signs of laminitis are lameness and pain or discharge at the coronary band. The affected nail may remain while the new nail grows in and displaces it. The nail may remain attached at the sole with total separation at the coronary band.[182]

Greater one-horned rhinoceroses are prone to developing **chronic foot disease**. The species lives in river plains and they are "hoof-walkers;" the outer rim of the hoof wall is denser and carries the animal's weight. In captivity on hard substrate, foot problems tend to develop especially if animals become overweight. Chronic foot disease was found to affect nearly 25% of the captive population in a 1996 international survey. Male rhinos were twice as likely as females to have chronic foot disease. The condition usually occurs in one or both hind feet, but all four feet are vulnerable. The hoof wall breaks down/cracks with increased stress on the more delicate central pad, and hematomas of the pad may be seen. Fissures develop in the pad behind the middle toe and excessive tissue growth occurs between the toes. Cracks in the pad initially appear as ragged edges of overgrown sole. Trauma promotes separation and scarring results. Secondary bacterial infection of the lesions may occur. A majority of cases also have overgrowth of the toenails, although it is not certain whether this is a cause or a consequence of pad separation.[182]

Though not specifically reported in the literature, many non-infectious chronic inflammatory diseases are seen in aged individuals; over time lesions degenerate and mineralize. Chronic degenerative vascular diseases, including **arteriosclerosis** (Fig. 17.6) and **atherosclerosis**, are seen in many aged captive perissodactyls. These changes are dissimilar to vitamin D_3 toxicosis (described earlier) and unrelated to vascular rupture. **Coronary artery**

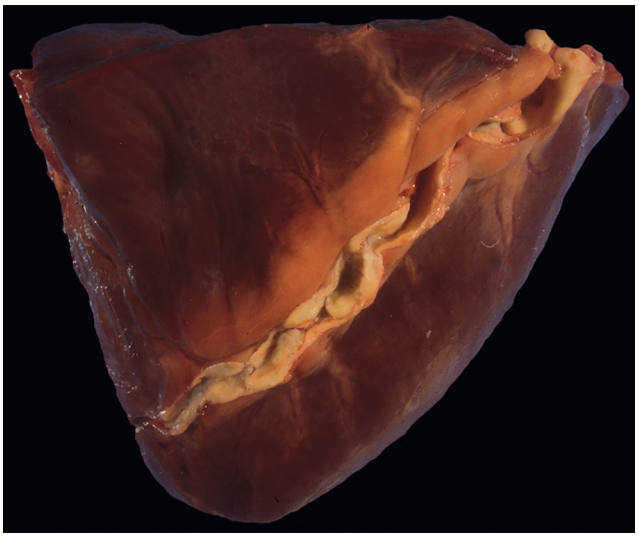

FIGURE 17.6 Arteriosclerosis in a Malayan tapir. There is moderate transmural thickening of the cardiac arteries in the interventricular groove of the heart. *(Photo Courtesy of Toronto Zoo.)*

aneurysms, with or without rupture and hemopericardium, have been described in black rhinoceroses.[73,108] Histological lesions include ulceration of the intima with superficial/luminal granulation tissue. Cardiac **Purkinje fiber degeneration, necrosis, and mineralization** have been described in free-ranging black rhinoceroses.[76] The cause of the lesions is undetermined. Sites of **chronic skin trauma** can develop fibrosis and mineralization; some have been associated with the development of calcinosis circumscripta (Fig. 17.7).

Gastrointestinal tract disease is a common source of problems in the domestic horse and similar conditions, including **colonic impaction, esophageal rupture, intestinal perforation, serositis, ulcerative colitis**, and **colonic torsion**, have been seen in a number of nondomestic perissodactyls (Figs. 17.8A–F). Dilation of the caudal esophagus in one report was associated with regurgitation in a black rhinoceros whose symptoms resolved on a soft diet.[154] In another study, a third of adult tapir deaths involved the gastrointestinal tract.[60] Findings included **intestinal volvulus, gastric and colonic impactions, colonic incarceration, oropharyngeal abscess/oral necrobacillosis, oropharyngeal trauma, acute pancreatitis**, and **eosinophilic enterocolitis**. Sand and shavings have caused colic and intestinal impaction in *Equidae* and *Tapiridae*, especially newborns.[194] Some gastrointestinal problems occurred due to overconsumption, especially if food lacks fiber as the stomach is relatively small.[194] **Rectal prolapse** has been seen in perissodactyls, most frequently in tapirs in association with poor quality dietary roughage and lack of access to water and consequent dehydration. Prolapse has also been reported in rhinoceros.[1,7] Prolonged tenesmus, constipation, diarrhea, intestinal protozoal parasites, dystocia, urinary obstruction, proctitis, rectal tear, and space-occupying lesions have been associated with rectal prolapses. Rectal prolapse was associated with marked edema and submucosal fibrosis in a greater one-horned rhino.[7]

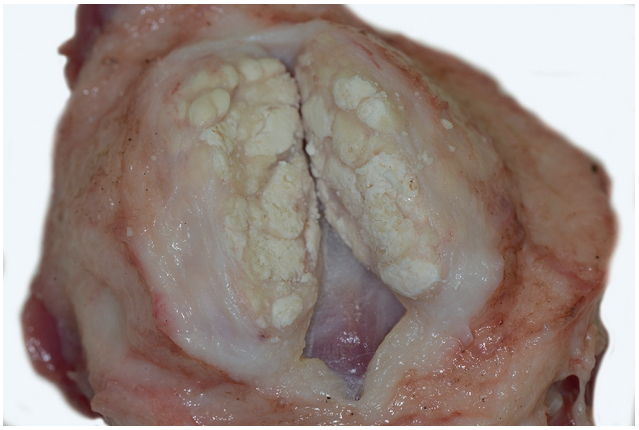

FIGURE 17.7 Calcinosis circumscripta due to chronic trauma in the skin of a white rhinoceros. Characteristic features are the nodular deposition of mineral in soft tissues such as the dermis and subcutis.

Pseudodiverticulosis with jejunal rupture and peritonitis has been reported in a captive-born Przewalski's horse.[112] **Enteroliths** have also been described in nondomestic perissodactyls. They develop when mineral deposits in layers around a nidus. Diet acidification may decrease formation. Tapir fecal enteroliths are composed of hydrated phosphates, vivianite (Fe), and newberyite (Mg) rather than struvite (struvite is more common in horses).[121]

Corneal cloudiness due to **keratitis** in captive tapirs may be associated with excessive light exposure, trauma, or herpesvirus.[58,60,155,194] The nocturnal nature of Malayan tapirs and the dense vegetation of the habitat of these and Sumatran rhino may predispose these species to keratitis in the captive setting when managed with less shade and encouraged to shift their wake/sleep cycle.

Miscellaneous/Multifactorial

Fracture of cervical vertebra and femur has occurred during courtships of Somali wild ass.[135] Traumatic lesions during breeding also occur in many species of rhinoceros.[111] **Bite wounds** are common in *Equidae*. **Cervical fractures** occur with some frequency in captive zebras after collision with enclosure fencing.[172] Animals are often found dead at the fence with no history of any illness. Diagnosis is straightforward radiographically and on gross examination based on the cervical hemorrhage and disrupted vertebrae (Fig. 17.9). **Rhinoceros horn avulsion**

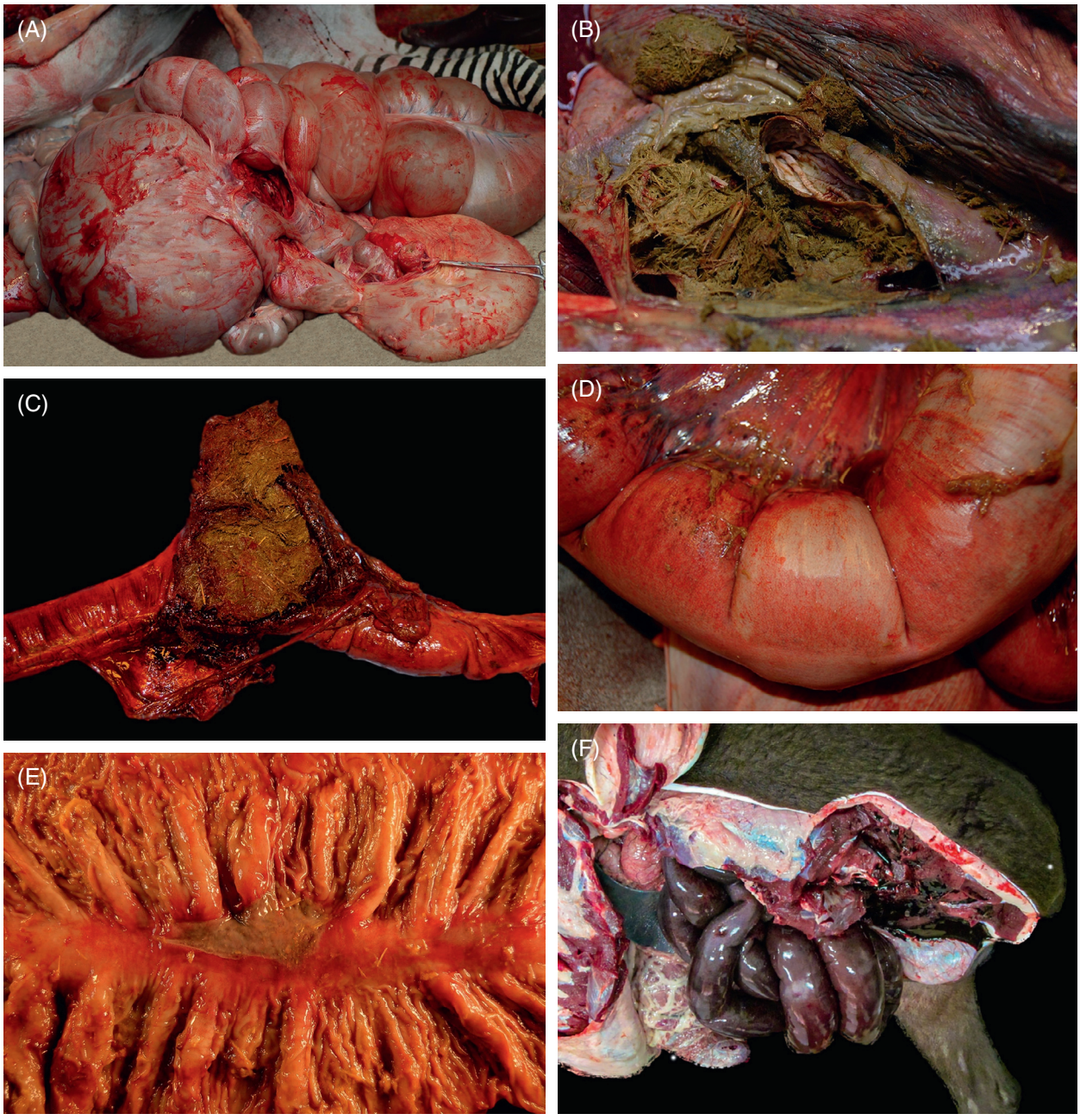

FIGURE 17.8 Gastrointestinal tract disease in perissodactyls. (A) Colonic impaction at the diaphragmatic flexure in a Grevy's zebra. (B) Esophageal rupture with intrathoracic gastrointestinal content at the mediastinum (left lung is reflected dorsally) in a Grevy's zebra. (C) Intestinal perforation in a Grevy's zebra. (D) Acute fibrinonecrotic serositis in a Grevy's zebra. (E) Focal ulcerative colitis in a Grevy's zebra. (F) Colonic torsion in a lowland tapir. *(Part F: Photo Courtesy of Zoológico de Sorocaba, Brazil.)*

can occur if horizontal poles are used for fencing. **Poacher-related trauma** from dehorning and snares are common in wild rhinoceros. **Interspecies aggression** may occur between male tapirs housed together. Death of free-ranging tapir occurs frequently through collision with **motor vehicles**.[65,98]

Obesity is a common concern in all captive equids that receive a rich diet and get relatively little exercise.[126]

Severe decubitus ulcers with necrosis have been described in white rhinoceros. One neonate with mild angular limb deformity developed ulcers secondary to reduced mobility. Particular susceptibility to pressure necrosis in the skin of neonatal rhinos has been suggested.[41] In another report, a rhino developed ulcerations due to trauma associated with cement flooring during winter housing.

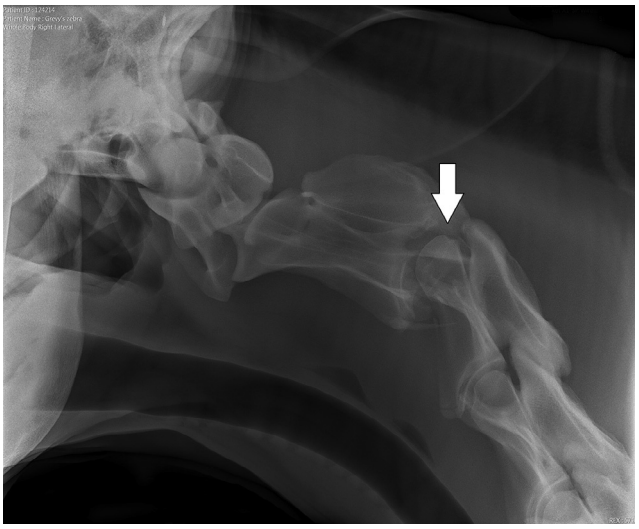

FIGURE 17.9 Cervical vertebral column trauma in a Grevy's zebra. Radiograph showing C2 vertebral displacement with facet fracture.

Ischemic distal tail necrosis after presumed trauma and entrapment of the extremity necessitated amputation in a black rhinoceros (Figs. 17.10A, B).

Leukoencephalomalacia has been reported in young, female black rhinoceroses in three different zoos; two of these animals had clinical hyperglycemia.[68,110] The calves developed sudden onset lethargy, later becoming recumbent and comatose. One calf developed epistaxis and hyperresponsiveness. Cloudy meninges, yellow-green foci in the frontal lobes, and subdural hemorrhages were seen grossly. White matter cavitation was seen in coronal sections. Histologically, edema, hemorrhage, neutrophil infiltration, and focal necrotizing vasculitis were present. The cerebrum was most severely affected. The pattern of encephalomalacia was similar to that seen in horses that ingest moldy feed contaminated with the fungus *Fusarium moniliforme*, which produces several toxins including fumonisins. No mold toxin was identified in feed in the affected rhinos. The fourth case developed acute neurologic signs: circling, head tilt, head pressing, continuous vocalization, hypermetria, ptyalism, and apparent blindness.[68] Computed tomography scans showed areas of reduced density in the cerebrum and thalamus, ventricular dilation, and dense areas in the caudate nuclei compatible with hemorrhage. Grossly, cavitating necrosis of the white matter was seen in the cerebral hemispheres. Histologically, demyelination was the main lesion with minimal reaction. In one case, the dam had been severely traumatized in the last trimester of pregnancy with possible compromise of blood circulation to the fetal brain. Increased age of the dam, trauma, and/or excess maternal iron stores were suggested as possible contributing factors.[105]

Neoplastic

The number and frequency of neoplasms occurring in perissodactyls may not be fully represented in the published literature. **Leiomyoma** of the female reproductive tract and cutaneous **sarcoids** are described below. Other reported neoplasms in nondomestic perissodactyls are listed in Table 17.e3.

Leiomyoma, a benign smooth muscle tumor, is found with relative frequency in the reproductive tract of female aged zoo species, especially greater one-horned rhinoceros. In one clinical study, 100% of greater one-horned rhinoceros over 12 years of age had reproductive tract masses on ultrasound examination.[53] The number and size of masses increase with age. The masses are particularly common at the cervix and in the vagina. They can be single or multiple and are generally well demarcated, white, and

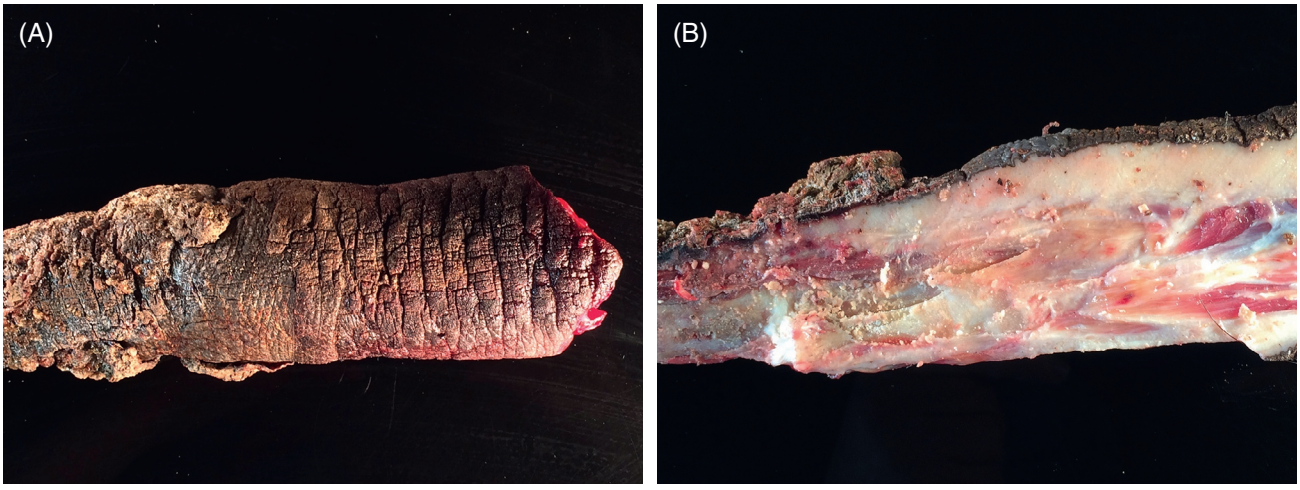

FIGURE 17.10 Ischemic necrosis (undetermined cause) of the distal tail of a black rhinoceros. (A) Depressed distal surface with crusted dried serocellular deposits to the left of the image. (B) Central segmental pallor of the striated muscle is consistent with necrosis.

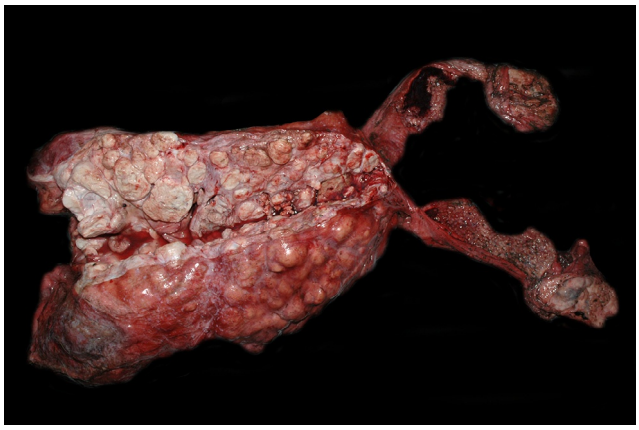

FIGURE 17.11 Uterine leiomyomas in a greater one-horned rhinoceros. The uterine body, which is opened longitudinally along its long axis, is markedly expanded by numerous, variably sized, firm, demarcated, coalescing, light tan masses that obscure the uterine lumen (see eSlide 17.e3). *(Photo Courtesy of the Wildlife Conservation Society.)*

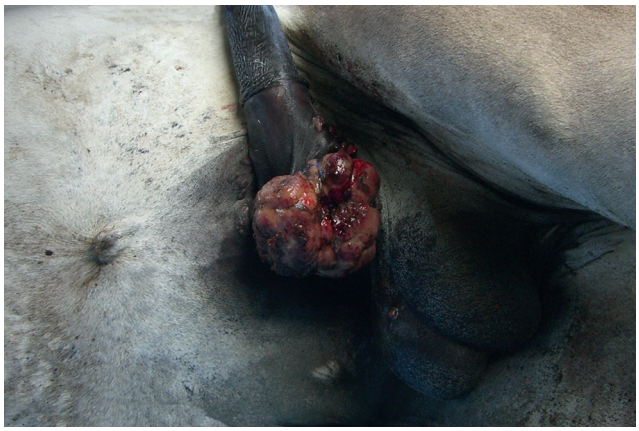

FIGURE 17.12 Preputial sarcoid in a Somali wild ass. These are exophytic, flat to fleshy tumors that may, as in this case, have an ulcerated surface (see eSlides 17.e4–e6). *(Photo Courtesy of S. Citino, White Oak Conservation.)*

firm. The wall of the uterus can be markedly distorted (Fig. 17.11; eSlide 17.e3) and cause compression or dislocation of adjacent abdominal viscera. The average gestational length of the white rhinoceros is 16 months. In the wild, a single offspring is produced every 2–3 years, so the uterus of a wild female is generally under the influence of progesterone. Increased progesterone levels are protective against the development of leiomyomas, while acyclicity has been suggested to predispose captive rhinos to their development.[53,190]

Sarcoids are unencapsulated, poorly demarcated fibroblastic skin tumors that invade locally and may recur after surgery, but that do not metastasize. Spontaneous regression has been reported in young individuals with mild lesions. Sarcoids are composed of spindle cells within the dermis that are arranged in streams and whorls and form well to poorly demarcated masses. The overlying epidermis is frequently hyperplastic with long rete pegs. Equine sarcoids associated with **bovine papillomavirus (BPV) types 1, 2, and 13** infection have been identified (see "DNA viruses" section). Sarcoids have been reported at the eyelid, nose, and inguinal region of a captive Plains zebra with BPV type 1. Virus was demonstrated in the tumor with in situ hybridization of the inguinal mass.[88] Sarcoids at the ear, prepuce, and mammary gland in Somali wild asses have also been described and are also thought to be virus-associated (Fig. 17.12; eSlides 17.e4–e6).[135] A survey of sarcoid cases in African equids in European zoo collections suggests prevalence varies with equid species. A genetic predisposition has been suggested in domestic horses and captive Somali wild ass; in the latter this is because the population is descended from 17 wild-caught individuals.[189] Sarcoids have also been reported in wild Cape mountain zebra in two reserves in South Africa. The prevalence was high (up to 53%) and lesions were most frequent on the ventrum and legs.[93] Free-ranging zebra populations with greater genetic diversity seem less likely to be affected by sarcoids.[164] Two cases of sarcoid at the pinna of captive Baird's tapir with possible association with BPV type 1 have been reported.[70] Papillomas have been identified on rhinos. An association with viral infection has not, to date, been identified.

INFECTIOUS DISEASES

The literature on infectious diseases of nondomestic perissodactyls consists primarily of single case reports or case series of disease and serology of captive animals. In addition to descriptions of notable diseases below, see Table 17.2 (Viral infections), Table 17.3 (Mycotic infections), and Tables 17.e4–17.e6 (Serologic evidence of infection, Endoparasites, and Ectoparasites, respectively) for lists of additional reported infections. The World Organization for Animal Health (WOAH) listed, reportable diseases of specific global concern for equids include African horse sickness, Western and Venezuelan equine encephalitis viruses, equine influenza, equine herpesvirus-1, equine infectious anemia, equine viral arteritis, vesicular stomatitis virus (VSV), glanders (*Burkholderia mallei*), contagious equine metritis (*Taylorella equigenitalis*), equine piroplasmosis, and dourine (*Trypanosoma equiperdum*). A recent outbreak of VSV in a group of captive white rhinos demonstrated their susceptibility to this virus. Nondomestic equids are also assumed to be susceptible to Borna virus.[186]

TABLE 17.2 Viral Infections Reported in Nondomestic Perissodactyls

Family/Species	Viral Pathogen	Comments
Equidae		
Grevy's zebra	Equine herpesvirus 1 (EHV-1)[48,192] Unusual alphaherpesvirus—lacks strong host specificity	Captive, abortion, perinatal foal mortality, virus neutralization, DNA restriction endonuclease analysis Clinical disease rare, zebra considered natural host, recommend separation from other susceptible species[84]
	Equine herpesvirus 9 (EHV-9)[48,84] (closely related to EHV-1) Unusual alphaherpesvirus—lacks strong host specificity	Captive, 3/7 severe acute disease, 2 deaths, fibrinonecrotic pneumonia, hyaline membranes, nested PCR herpesvirus in lung Clinical disease rare, zebra natural host, Grevy's possible reservoir, recommend separation from other susceptible species
Onager, zebra[115]	Equine herpesvirus-1	Captive, abortion onager (multifocal necrosis spleen and liver), suspected myelitis zebra
Nondomestic equids[148]	African horse sickness	Captive, epizootic in Europe with mortalities following introduction wild-caught zebra. Plains zebra considered the natural host for the virus, rarely develops signs. *Culicoides* spp. are insect vectors
Tapiridae		
Lowland and Malayan tapirs[155]	Foot and mouth disease virus	Captive, Europe interdigital lesions
Malayan and mountain tapirs[155]	Herpesvirus	Captive, fever, keratitis, erosion to ulceration at mucocutaneous junction 3/6 mortalities
Malayan tapir[45]	Equine herpesvirus 1 (EHV-1), presumptive	Captive, pregnant female died, hemorrhage and necrosis multiple organs
Malayan tapir[181]	Encephalomyocarditis virus	Captive, unexpected death 5 month female
Rhinoceridae		
Black rhinoceros[45]	Herpesvirus	Captive (same collection as tapir with EHV-1), one death with gastrointestinal tract ulcers, 3 cases cutaneous ulcers. Herpesvirus on electron microscopy
Black and white rhinoceros[155]	Poxvirus	Captive, vesicles and pustules
White and greater one-horned rhinoceros	Vesicular stomatitis virus	Captive, United States Swellings to ulcers at nostrils, lips, and oral cavity. Cracks, ulcers, sloughing tissue at feet

DNA Viruses

Sarcoids (see "Neoplastic" section) in horses have been associated with **bovine papillomavirus types 1, 2, and 13** infection. Viral DNA has been detected in the nuclei of the neoplastic mesenchymal cells of zebra sarcoids.[88] Late viral antigens are not found in sarcoids. Neutralizing antibodies to virus are not found in horses, suggesting infection is nonproductive in this (probable) nonpermissive host. Mature viral particles are found in the upper keratinizing layers of these epithelial tumors in bovids; the immune system response results in limited progression of the lesion. The mechanism of transmission of bovine papillomavirus to zebras has not been identified. Sarcoids do not produce virions, but viral DNA and RNA may be detected, and BPV type 1 and 2 major transforming protein E5 is expressed. Of 12 skin lesions from Somali wild ass at 10 European collections, BPV type 1 was detected in eight and BPV type 2 was detected in one by PCR.[189]

RNA Viruses

Perissodactyls are susceptible to **rabies virus** infection (may be locally or regionally reportable) and, similar to other susceptible mammal species, they can die after developing clinical neurologic disease. Infection has been reported in a zebra foal housed at a safari lodge in Kenya

TABLE 17.3 Mycotic Infections Reported in Nondomestic Perissodactyls

Species/Family	Fungal Pathogen	Comments
Equidae[186]	*Trichophyton*	
Grevy's zebra[24,186]	Phaeohyphomycosis	Captive, mortality
Przewalski's horse[174]	*Coccidioides immitis*	Captive, granulomatous lesions, respiratory system predisposed, males overrepresented, intraspecies aggression suggested
Tapiridae[155]	*Microsporum gypseum, M. canis, Trichophyton tonsurans*	Dermatophytosis. Alopecia in a mountain tapir
Lowland tapir[91,155]	*Coccidioides immitis*	Wild caught from endemic area. Case report, captive. Pulmonary granulomata in association with amebic meningoencephalitis
Rhinoceridae		
White rhinoceros[155]	*Trichophyton mentagrophytes*	Group housed after recent capture
White rhinoceros[5]	*Malassezia pachydermatis, Candida parapsilosis*	Captive, pruritus
Black rhinoceros[155]	*Aspergillus fumigatus, Absidia corymbifera*	Pulmonary aspergillosis
Black rhinoceros[187]	Aspergillosis most frequent	Survey captive United States, $n=8$, pneumonia at necropsy, often concurrent disease, 2/8 mycobacteriosis
Black rhinoceros[185]	*Coccidioides immitis*	Captive, United States, chronic progressive lameness

that was suspected to have been bitten by a feral dog. Approximately 1 month later, the foal died after experiencing neurological disease for 2 days. Rabies was confirmed by direct fluorescent-antibody testing.[82]

Fatal encephalomyocarditis virus (EMCV) infection has been identified in a tapir. White foci and streaks in the myocardium correspond histologically to areas of cardiomyocyte degeneration and mineralization, and interstitial lymphoplasmacytic and occasionally neutrophilic inflammation (Figs. 17.13A, B). Mild to moderate congestion of the lungs and hepatic centrilobular vacuolar degeneration may also be seen. Infection is confirmed by PCR.[181] Several fatal cases of EMCV have also been described in rhinoceroses.[108] While lesions have not been specifically described, viral presence has been confirmed in the heart, spleen, and other viscera. Virus is excreted in the feces and urine of rodents, the natural hosts for the virus. Management of the virus in captive settings should focus on pest control.

Vesicular stomatitis virus (VSV) has been diagnosed in southern white and greater one-horned rhinoceroses as the cause of significant clinical disease (personal communication, P.M. Gaffney). PCR testing confirmed VSV-New Jersey strain infection. Gross findings were localized to mucous membranes and mucocutaneous junctions of the head, or the nails and coronary bands of limbs. On the nostrils, lips, and in the oral cavity, lesions varied from swellings to erosions to ulcers. At the distal limbs, lesions progressed from cracks to ulcers to separation and sloughing of foot pads (Fig. 17.14A). Histologic appearance of sloughed or debrided tissue revealed inflamed to necrotic epidermis, intercellular edema, and cellular dissociation (spongiosis) of the stratum spinosum, progressing to individualized, necrotic keratinocytes within enlarging vesicles. Formation of pustules following necrosis, and secondary bacterial infection, may occur (Fig. 17.14B). Some individuals seroconverted, and VSV nucleic acid was detectable in lesions for at least 6 weeks after the development of gross lesions.

Bacteria

Sporadic ***Mycobacterium bovis*** and ***M. tuberculosis*** (WOAH-listed reportable diseases) infections have been reported in captive rhinoceros.[103,108,155] Most cases have been subclinical and only diagnosed during postmortem examination. Advanced cases may present with chronic progressive respiratory disease. Isolated cases of *M. bovis*

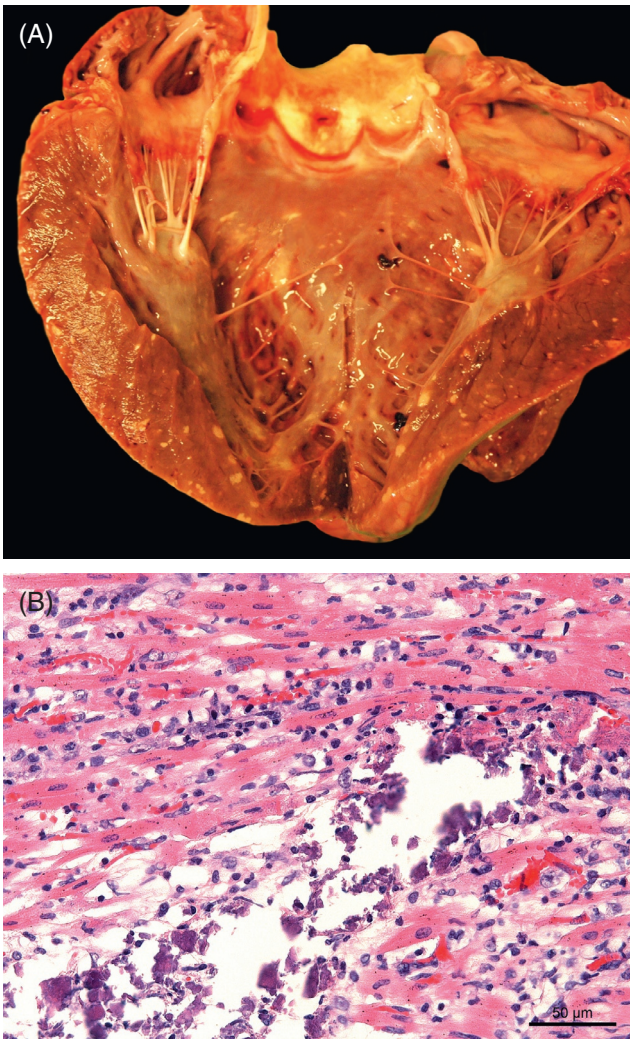

FIGURE 17.13 Encephalomyocarditis virus infection in the heart of a tapir. (A) Multifocal white discoloration is present throughout the myocardium. (B) Discoloration corresponds to foci of myocardial degeneration, mineralization, and mild inflammation. *(Reprinted with permission from* Vercammen F, Bosseler L, Tignon M, Cay AB. Encephalomyocarditis virus in a captive Malayan tapir *(Tapirus indicus). Open Vet J*. 2017;7(2):100–103.*)*

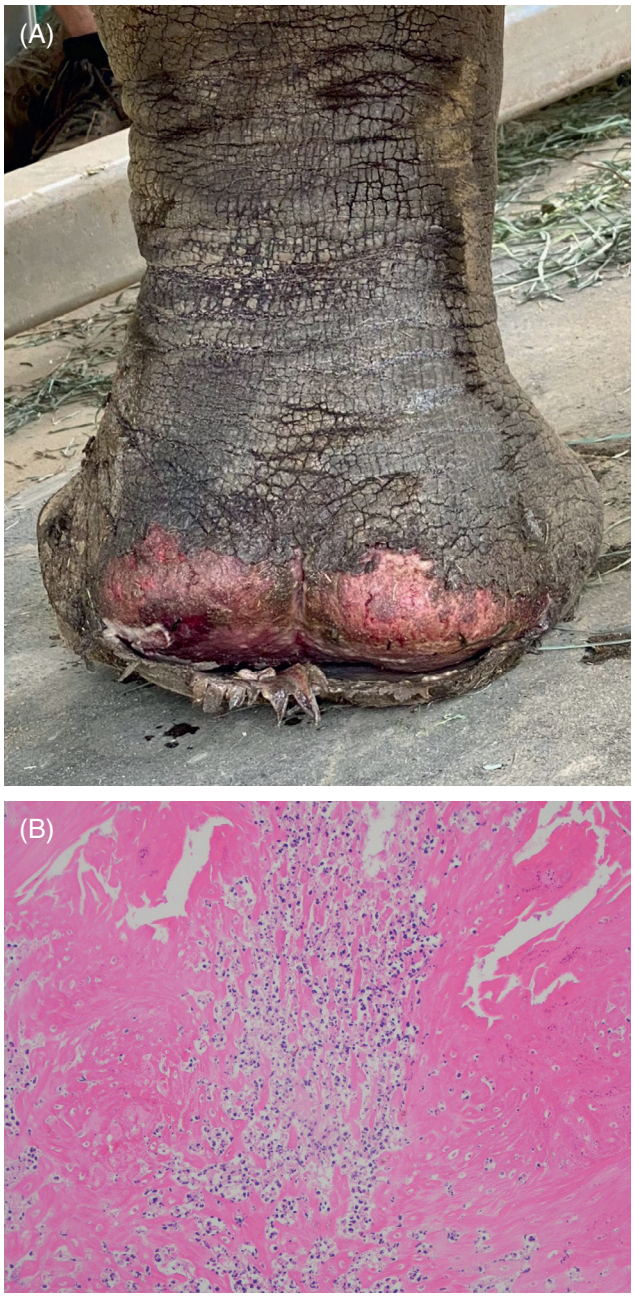

FIGURE 17.14 Vesicular stomatitis virus in the skin of a southern white rhinoceros. (A) Locally extensive ulceration is present along the junction between the skin of the foot and the foot pad. (B) This corresponds to severe epidermal necrosis and neutrophilic inflammation with abundant pyknotic and karyorrhectic debris.

or *M. tuberculosis* have also been reported in free-ranging rhinos.[34,106] The presence of granulomas in the lungs and lymph nodes should raise suspicion for mycobacteriosis (Fig. 17.15; eSlides 17.e7 and 17.e8). Diagnosis is confirmed by bacterial culture and PCR analysis. ***M. orygis*** was recently described in the ***Mycobacterium tuberculosis* complex**. Infections have occurred in East Africa, the Arabian Peninsula, and South Asia. It was reported in a free-ranging Indian rhinoceros in Nepal with pulmonary granulomata and confirmed by molecular methods.[175] A greater one-horned male rhino that was captive-born in India and imported to the United States as a juvenile had a period of lethargy and inappetence as a young adult. A year later the problem recurred and progressed to pelvic limb paresis, ataxia, and gluteal and epaxial muscle atrophy. Fibrohistiocytic to pyogranulomatous inflammation centered at the sublumbar lymph nodes and extending into a sciatic nerve and the cauda equine was identified on gross examination. Lymph node culture and polymerase chain reaction isolated *M. orygis*.[90]

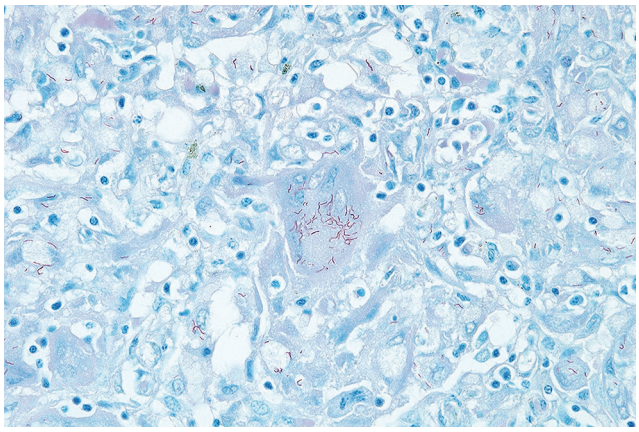

FIGURE 17.15 Pulmonary mycobacteriosis in a black rhinoceros. Histiocytic pneumonia is associated with numerous intracytoplasmic, acid fast positive bacilli in multinucleate giant cells and histiocytes. Ziehl-Neelsen (see eSlides 17.e7 and 17.e8).

Mycobacterium pinnipedii has been isolated from captive pinnipeds worldwide and in the Southern Hemisphere from wild individuals. A captive Malayan tapir, previously housed adjacent to sea lions, developed respiratory signs and weight loss. Sputum was positive for acid-fast bacilli. Necrogranulomas were present in the lungs and mesenteric lymph nodes at necropsy. Spoligotyping was positive for *M. pinnipedii*. Three additional tapirs were seropositive and subsequently euthanized. Granulomatous lymphadenitis was found, and bacterial culture and spoligotyping confirmed *M. pinnipedii* in these three animals.[63] Three lowland tapirs at a Brazilian zoo died with pulmonary granulomas.[120] Polymerase chain reaction of bronchoalveolar lavage samples from two animals was positive for a *M. tuberculosis* complex strain commonly associated with regional human cases of tuberculosis.

Disease caused by the ***Mycobacterium avium–intracellulare*** **complex** bacteria is becoming more prevalent, including lesions of lymphadenitis, pulmonary granulomata, and disseminated infections. Infections in Perissodactyla include those in a breeding pair of captive-born lowland tapirs from different origins. Both were initially negative within intradermal skin testing. However, both developed mild respiratory signs within a few months of their pairing, and nontuberculous mycobacteria were present among bacteria recovered from sputum. Clinical signs resolved completely in the male with antibiotic treatment, while the female continued to have mild signs. A female offspring born a year later developed respiratory signs at 3 weeks; similar respiratory signs developed in a male offspring born a year later. The female calf recovered with antibiotic treatment but signs progressed in the male calf, which was euthanized. Necropsy revealed chronic bronchopneumonia. Bacterial culture was positive for ***M. avium*** **subspecies** ***hominissuis***.[95] The remaining tapirs showed weight loss and respiratory disease, and *M. avium* ssp. *hominissuis* was cultured from bronchoalveolar lavage and identified by PCR testing. Following a long treatment course the group tested negative. The source of mycobacteriosis was thought to be environmental.[94]

Paratuberculosis or **Johne's disease** caused by ***M. avium*** **subspecies** ***paratuberculosis*** is a WOAH-listed reportable disease that is typically seen in ruminants. Clinical signs include progressive weight loss, debilitation, and occasionally death. Infection causes histiocytic to granulomatous ileitis-enteritis. Johne's disease is uncommon in perissodactyls. However, the bacterium has been isolated from the feces of a wild-caught black rhino with diarrhea and weight loss.[13] Due to the conservation value of the individual, antimycobacterials were administered until fecal shedding was no longer detected.

Anthrax, a WOAH-listed reportable, zoonotic disease due to infection with ***Bacillus anthracis***, is a significant problem in the free-living *Equidae* and *Rhinoceridae* in Africa and is a significant threat to wild populations of Grevy's zebra. In periods of drought, increased trauma to the oral cavity from dry or woody browse may increase the risk of spore acquisition. Infected animals may be asymptomatic and die unexpectedly. Bloody discharges from the mouth, nostrils, ears, or anus; rapid bloating of the carcass; incomplete rigor mortis; and dark unclotted blood should raise one's index of suspicion for infection. Differential diagnoses include several other important infectious diseases including African horse sickness, botulism, Rift Valley fever, and snake bite. Vaccination has been conducted in some parks in Kenya and Namibia. Efficacy has not yet been determined.[59,105,186] Transmission of anthrax in herbivores is assumed to occur by ingestion of spores. A recent study found the potential for infection by inhalation during dust bathing was unlikely.[3] Caution should be used in the handling and necropsy of suspect cases due to the risk of zoonotic transmission. Cytology is a recommended early diagnostic test that should be performed prior to other sample collection or necropsy as it may confirm infection with the encapsulated, square-end bacilli in suspect cases and inform risk assessment (see Wildlife Necropsy).

Leptospirosis occurs worldwide, especially in moist environments. Infection is associated with rodent transmission and water contamination. Leptospirosis was associated with early cases of hemolytic anemia in captive black rhinoceros. In one survey, leptospirosis was considered the cause of hemolytic anemia in 9 of 31 cases.[62] At necropsy, red fluid was found in the pericardial sac, peritoneum, and urinary bladder, and marked edema was noted throughout the omentum, lungs, and perirenal tissues. Leptospirosis was implicated by microscopic agglutination test. Blood collected from rats trapped on zoo grounds where two rhinos died from leptospirosis had significant titers

for ***Leptospira icterohaemorrhagiae***. Rats were considered to be the vectors in these cases.[27] In another study, antibodies to 8 serovars of ***L. interrogans*** were reported in free-ranging and captive black rhinoceros. The free-ranging black rhinoceros had low antibody levels and serovars varied with region and ecosystem. In other infections, *L. interrogans* was the cause of death in black rhinoceros that died of hemolytic anemia and in another rhinoceros that died as a complication of ulcerative skin disease.[62] Peracute depression, anorexia, hind leg trembling, dysuria, glucosuria, gastrointestinal discomfort, and decreased fecal output in the absence of hemolytic anemia have also been associated with *L. interrogans* infection in captive black rhinos[125] and with abortion in an Indian rhinoceros.[111]

Salmonellosis is a common cause of septicemia and enteritis in rhinoceros and tapir[155] and of abortion and genital tract infection of equids.[186] Asymptomatic shedding may occur and antibiotic resistance may quickly develop with transfer of plasmids between *Salmonella* species. Salmonellosis has been reported in wild black rhinoceros with diarrhea during translocations. ***Salmonella enterica*** **subspecies** ***arizonae*** was cultured from three black rhinoceros from one American zoo that died with gastroenteritis and sepsis.[67] Other reported signs of salmonellosis include lethargy, partial anorexia, epistaxis, ulcerative skin lesions, and/or diarrhea in a captive female and two male rhinos. In one report, Tokay geckos released into a building housing rhinos were the suspected source of salmonellosis (but instead *S. eastbourne* was isolated from the intestine of one of two geckos euthanized). Gross lesions of salmonellosis in these rhinos included fibrinous pleuritis, a large lung abscess in one, and resorptive bone lesions in the distal digits. In one survey of salmonellosis conducted across rhinoceros species from 1990 to 1997,[66] 11% of fecal samples were culture positive for *Salmonella* spp. Some animals suspected of having clinical salmonellosis had leukopenia, hemoconcentration, and hyperproteinemia. In four, sepsis and death were attributed to salmonellosis. Monthly surveillance for salmonellosis in a captive, asymptomatic herd of black rhinoceros for nearly 3 years by fecal culture and PCR identified periodic shedding in four of six rhinoceros.[104] This is important from a management perspective as periodic, intermittent testing is often needed to confirm the presence or clearing of infection and shedding. Clinical cases of salmonellosis in black rhino calves have also been reported. Clinical signs can include self-limiting diarrhea or sepsis-related death (death may be related to low transfer of maternal immunity and primiparity).[89]

Other notable bacterial diseases in perissodactyls include **tetanus** due to ***Clostridium tetani*** of a hoof abscess in an onager, and bacterial sepsis secondary to ***C. welchii*** wound infection in a Somali wild ass.[135] Unexpected death, presumably from ***C. perfringens*** enterotoxemia has been reported in white rhinoceros; the main findings were mild abdominal exudate, splenic and multifocal gastrointestinal congestion, and adrenal hemorrhage.[15] ***Streptococcus equi*** causing soft tissue swelling due to suppurative lymphadenitis that is most often associated with infection of the pharynx and respiratory tract, strangles, was the cause of a cluster of deaths in Przewalski's horses reintroduced into Southwestern Mongolia. A harsh winter and wolf predation were suspected as predisposing factors.[159] **Bacterial vegetative valvular endocarditis** was reported in a captive, aged, wild-caught male white rhinoceros that developed immobility and decubital ulcers. There was roughening at the left atrioventricular valve and ventricular endocardium. This corresponded histologically to proliferative inflammation on the valve leaflet (vegetative endocarditis) and associated myocardial degeneration. Fibrosis and degeneration in the liver and chronic nephritis developed as sequelae. ***Streptococcus dysgalactiae*** subsp. ***equisimilis*** and ***S. ovis*** were isolated from multiple sites. The decubitus ulcers were the suspected portal of bacterial infection.[55]

Anaplasma phagocytophilum is a gram-negative rickettsial organism that is transmitted by *Ixodes* spp. ticks. The rickettsial has a tropism for granulocytes and forms morulae in their cytoplasm that appear as granular inclusion bodies. Anaplasmosis has been reported in Przewalski's horses in managed care based on the presence of intracytoplasmic morulae in cytologic or hematologic preparations and PCR assay of whole blood. Signs included dehydration, pyrexia, and lethargy with possible ataxia and contusions.[168]

Coxiella burnetti, the cause of **coxiellosis** (also known as **Q fever**), is most commonly reported in ruminants and most often causes abortion. Infection has been described in captive primiparous white rhinoceroses.[6] The most characteristic histologic lesion is the presence of gram-negative intracellular bacteria in necrotic placental trophoblasts and macrophages. PCR testing of frozen placenta can confirm the presence of *Coxiella burnetti* DNA. Vaginal shedding from infected female rhinos has been shown to last several weeks. Transmission is usually by aerosols. The disease may be subclinical and reproductive failure may suggest an underlying or developing problem. Human cases of coxiellosis are reportable and the zoonotic implications of the disease should be considered. In human infections, acute infection is characterized by phase II antibodies and chronic infection by phase I antibodies on serology. Serological study showed similar phase antibody patterns in rhinoceros dams.

Fungi

Phaeohyphomycosis is an opportunistic, dematiaceous (pigmented) fungal infection caused by a variety of fungal species. These fungal organisms are ubiquitous in soil and woody plants. Systemic phaeohyphomycosis is rare.

Vascular invasion is infrequent and often associated with immunosuppression. Infection usually originates in the respiratory system following inhalation, while cutaneous disease follows wound infection. Infection in a young adult, captive male Grevy's zebra caused sudden onset weight loss, lethargy, and hypothermia.[24] Focal pyogranulomatous pneumonia, myocarditis, and pericardial effusion were associated with intralesional, 3–6 µm diameter moniliform fungal hyphae. Yeast forms were found in the heart.

Coccidioides immitis is a dimorphic fungus that is present in the soil and may become airborne. Transmission is usually by inhalation of arthroconidia. Most infections are asymptomatic and resolve spontaneously. **Coccidioidomycosis** was the leading cause of death in a captive population of Przewalski's horses between 1980 and 2000 that were housed in Southern California, United States.[174] Abscesses, granulomas, pyogranulomas, and/or granulomatous inflammation with intralesional, thick-walled, 20–40 µm diameter, fungal spherules, and internal endospores were often disseminated, but the respiratory system was most often affected (Figs. 17.16A, B). Cases occurred more commonly in males and younger individuals, possibly due to intraspecific aggression and stress in the bachelor herds. Skeletal muscle lesions in the hind quarters were consistent with bite wound contamination or systemic spread to an inflamed vascular area. Multiple genetic lineages of Przewalski's horse were affected, while other equid species at the facility were unaffected. Przewalski's and domestic horses had similar numbers of CD3, CD4, CD5, CD8, and CD21 lymphocyte subsets by flow cytometry. Lymphocyte blastogenesis assay responses to the generic T-cell mitogen concanavalin A were alike but lymphocytes of two Przewalski's horses failed to proliferate in response to coccidioidomycosis antigen, suggesting a poor immune response to this specific pathogen.[174] Coccidioidomycosis was also identified incidentally in pulmonary and lymph node granulomas and in the synovium of a black rhinoceros with chronic progressive lameness. Initial serological testing was negative, though a positive titer was noted at the time of euthanasia.[185]

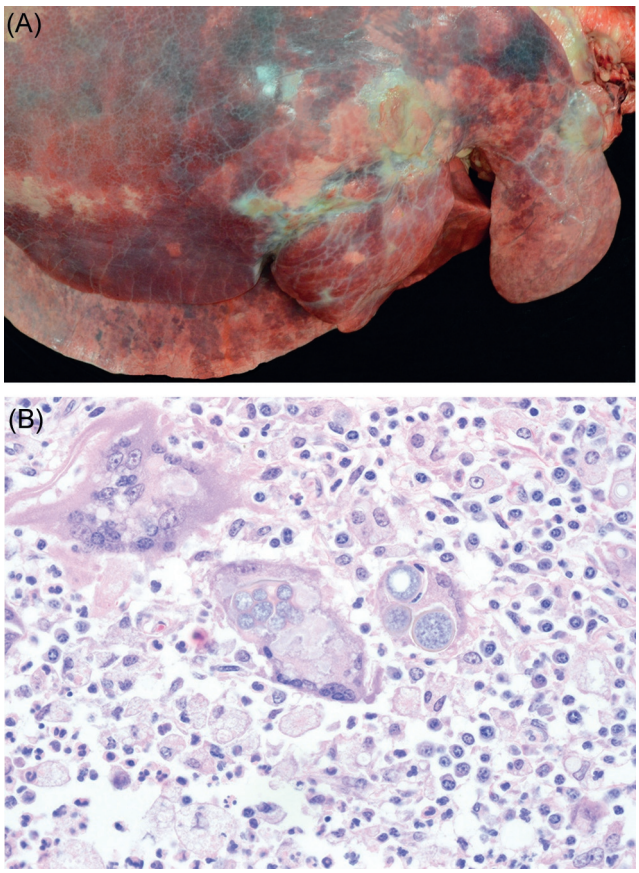

FIGURE 17.16 *Coccidioides immitis* infection in the lung of a Przewalski's horse. (A) Multiple yellow-white pyogranulomas are present throughout the parenchyma. (B) Histiocytes and neutrophils are admixed with lymphocytes and plasma cells. Fungal spherules containing endospores are present within multinucleate giant cells.

Concurrent infection with ***Malassezia pachydermatis*** and ***Candida parapsilosis*** has been described in a white rhinoceros that developed pruritic, erosive lesions up to 20 cm in diameter along the dorsum. Lesions were hyperemic with exudate. Cytologic examination of the exudate was positive for *Malassezia* spp. type yeasts. Biopsy showed exudative epidermitis. Fungal culture confirmed concurrent infection. *Malassezia* spp. have been cultured from the skin of unaffected individuals suggesting it may be part of the normal flora of rhinoceros skin.[5]

Metazoa

Stephanofilaria dinniki has been reported in free-ranging black and white rhinos with crusty, dry raised areas of dermatitis (Fig. 17.17). Histologically, ulcerative dermatitis and lymphohistiocytic nodules with serocellular crusts and intralesional, epidermal adult filarial nematodes are associated with necrosis, dermal granulomas, and mixed inflammation. Acanthosis and rete ridge formation are present at lesion margins. Chronic lesions have thick granulation tissue deposits in the dermis and there is marked inflammation with eosinophils, histiocytes, and multinucleate giant cells. Both adults and microfilariae can be seen in lesions. There is a pattern of seasonal variation in infection, with pruritus and erythema in the summer and resolution in the winter.[75] Crusts fall off to reveal red areas and leave depigmented scars. White rhinos have larger lesions that average 23 cm in diameter relative to 15 cm in black rhinos. Black rhino lesions are typically in the shoulder and ventral thorax, while white rhino lesions are seen at the rump. The life cycle of *S. dinniki* is unknown but a bloodsucking arthropod is required to complete the cycle. Poor body condition and heavy rainfall are thought

FIGURE 17.17 *Stephanofilaria dinniki* infection in the skin of a black rhinoceros. Irregular raised crusty lesions with multifocal ulceration are present along the lateral body wall. *(Photo Courtesy of S. Chege, Vetinwild.)*

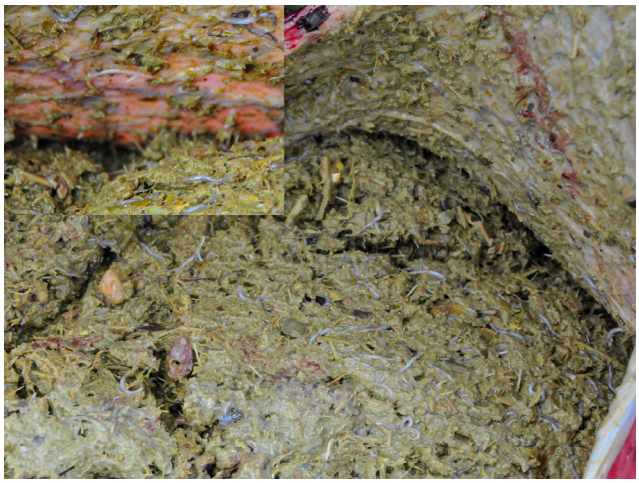

FIGURE 17.18 *Kiluluma longipene* infection in the stomach of a lowland tapir. Numerous slender, white nematodes are admixed within the gastric content. *(Photo Courtesy of the Lowland Tapir Conservation Initiative.)*

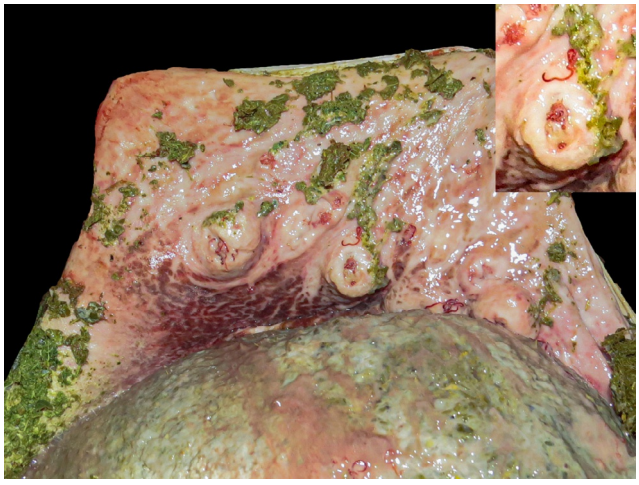

FIGURE 17.19 *Physocephalus* spp. infection in the stomach of a lowland tapir. Multiple raised nodules in the gastric wall contain a central cavitation with slender, intralesional red nematodes. *(Photo Courtesy of the Lowland Tapir Conservation Initiative.)*

to lead to recurrence of the lesions. Red-billed oxpeckers may aid in parasite spread and cause ulceration.[123]

Halicephalobus gingivalis has been reported in a captive female Grevy's zebra that appeared blind and demonstrated abnormal behavior.[57] Bilateral uveitis and associated normocytic, hyperchromic anemia, and elevated total protein and globulin with elevation of beta globulin fraction were noted. Disseminated granulomatous inflammation was found in the kidneys, heart, eyes, uterus, and lymph nodes in association with rhabditiform nematode infection. Cataracts and marked granulomatous inflammation of the ciliary body and choroid with synechiae were also seen. Larvae with adults were associated with the inflammatory infiltrates. Only female adults have been identified in infections suggesting parthenogenic reproduction.

Gastric nematodiasis due to ***Kiluluma longipene*** (Fig. 17.18) and ***Physocephalus*** spp. have been reported in free-ranging tapir. The latter is associated with proliferative mucosal nodules with central cavitations and intralesional slender red worms (Fig. 17.19).

Cestode infections have been identified nondomestic perissodactyla including tapir, Przewalski's horses, and rhinoceros (Figs. 17.20A, B). Reports include descriptions of several species of ***Anoplocephala***. Most reports list these parasites, even when loads are high, as incidental findings.

Schistosomiasis was found in a juvenile captive lowland tapir with a history of occasional bloody diarrhea. Multiple, often periportal, granulomas were present in the liver and eosinophils and multinucleate giant cells were associated with pigmented embryonated eggs. Hemorrhagic gastritis and enteritis were seen grossly and many trematode eggs were seen histologically within villi. Trematodes of schistosomiasis are intravascular; eggs are shed in the mesenteric venous plexuses and may backwash into the portal system or penetrate the intestinal villi.[193]

Protozoa

Piroplasmosis is an infectious, tick-borne disease that is caused by ***Babesia caballus*** or ***Theileria equi***. The parasites attack and destroy red blood cells. Infection may occur seasonally and parallels the vector tick lifecycle. Host

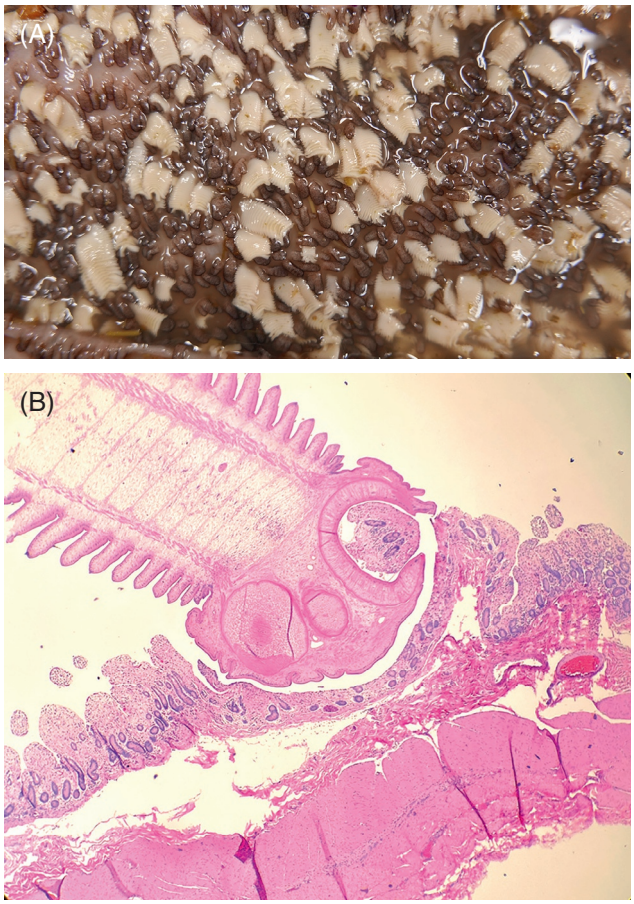

FIGURE 17.20 Enteric cestodiasis in a greater one-horned rhinoceros. (A) Myriad short, white cestodes fill the lumen and are admixed with luminal content. (B) Characteristic features of cestodes include a thick tegument, segmented body, lack of a body cavity and intestine, and multiple suckers on the scolex (head).

translocation may induce disease by spreading pathogens and modulating the effects of stress hormones on the immune system, allowing piroplasm proliferation.

In equids, severe regenerative hemolytic anemia and jaundice are seen in acute cases, while chronic cases may be seen in subclinical carriers. Gross findings include pulmonary edema and splenic congestion. Histologically, renal tubular necrosis and disseminated hemosiderin-laden macrophages are noted. Transplacental infection may cause abortion. Aborted fetuses have high levels of parasitemia. In a mortality survey of Przewalski's horses reintroduced to Southwestern Mongolia, piroplasmosis was implicated as the cause of death of stallions and a stillborn foal.[159] Babesiosis has been reported in a Somali wild ass stallion.[135] In cograzing donkeys and wild Grevy's zebra in Kenya, all zebra tested positive for *T. equi*.[51]

Piroplasmosis in black rhinoceros, caused by ***Babesia bicornis*** and ***Theileria bicornis***, has been shown to be fatal, especially in small and isolated populations in Tanzania and South Africa. In one study, *B. bicornis* was not detected in Kenya, but *T. bicornis* had a 49% prevalence in white rhinoceros and 43% in black rhinoceros. Infections have an increased importance because of conservation efforts related to animal relocations.[134]

Sarcocystis neurona has been isolated from domestic horses with **equine protozoal myeloencephalitis**. The opossum, *Didelphis virginiana*, is the definitive host of *Sarcocystis falcatula* and may be the definitive host of *S. neurona*. Similar lesions have been described in a captive Grant's zebra that developed acute ataxia, weakness, and depression. Cerebrospinal fluid was positive for antibodies to *S. neurona* on Western blot. Perivascular cuffing with mononuclear cells was noted focally at the brainstem. *S. neurona* was identified by immunohistochemical labeling of intralesional merozoites.[96]

Klossiella equi is a coccidian parasite of the renal parenchyma of equids. Its complete lifecycle is unknown. Schizogony (asexual reproduction) is thought to occur in the endothelial cells of Bowman's capsule. Merozoites are released and enter the proximal convoluted tubules to develop into second-generation schizonts. Merozoites are released into the lumen and enter cells of the loop of Henlé, where they develop into microgametocytes and macrogametocytes. When pronuclei of micro and macrogametocytes fuse, a zygote/sporont develops, sporogony occurs, and sporoblasts form that develop into sporocysts. Sporocysts are released into the lumen and pass out in the urine. The cycle is completed when sporocysts are ingested and sporozoites are released into the digestive tract, penetrate the wall, enter the blood stream, and travel to the glomeruli. The coccidian has been described as an incidental finding in sections of kidney from Hartmann's mountain zebra[172] and Przewalski's horses.[159]

Disseminated **neosporosis** was found on histopathologic examination of tissues from an adult, wild-caught white rhinoceros that died unexpectedly. At necropsy, hepatomegaly with multifocal necrosis, erosions to ulcerations of the stomach, and enlargement of the mesenteric lymph nodes were seen. Histologically scattered foci of necrosis with tachyzoites were present in liver, adrenal cortex, kidney, and intestine. Immunohistochemistry and polymerase chain reaction confirmed infection with ***Neospora caninum***.[171] Parasite transmission may be horizontal (ingestion) or vertical (transplacental). A source of infection was not identified but was likely through ingested oocysts. Abortion is the most common sign of infection in the intermediate host and cases of abortion have been reported in a Malayan tapir and a white rhinoceros.[147,163] Definitive hosts (dogs) most commonly have paresis and paralysis of the hindlimbs.

Necrosuppurative meningoencephalitis due to infection with ***Naegleria fowleri*** has been reported in a captive South American tapir. Inflammation was associated with perivascular ameboid trophozoites. Confirmation was by

monoclonal antibody testing. In humans, infection is associated with water contact. Olfactory neuroepithelium is the portal of entry for the organisms.[91]

ADDITIONAL RESOURCES

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Full references for this chapter can be found online at https://doi.org/10.1016/B978-0-443-18437-6.00019-5.

UNIQUE FEATURES

All perissodactyls have guttural pouches. These are paired ventral diverticula/outpouchings from the pharynx lateral to the hyoid bones. See Table 17.e2 for comparative dentition of the nondomestic Perissodactyls. Perissodactyls are monogastric with both a keratinized squamous and glandular portion. As in domestic equids, the margo plicatus is at the junction between the two sections (Fig. 17.e1). They have an extensive hindgut for fermentation by microbiota and alloenzymes. Perissodactyls lack a gallbladder.

Mammary glands are inguinal. Placentation is diffuse, microcotyledonary, and epitheliochorial (Fig. 17.e2). Squamous plaques, small nodular aggregates of concentrically laminated, acellular keratin, are a normal finding (Figs. 17.e3A, B). They are often most common near the attachment of the umbilical cord to the placenta. Expected in necropsies of neonates are the remnants of the retracting umbilical arteries with clot formation at the lateral ligament of the urinary bladder, the vessels that supplied the placenta (Fig. 17.e4).

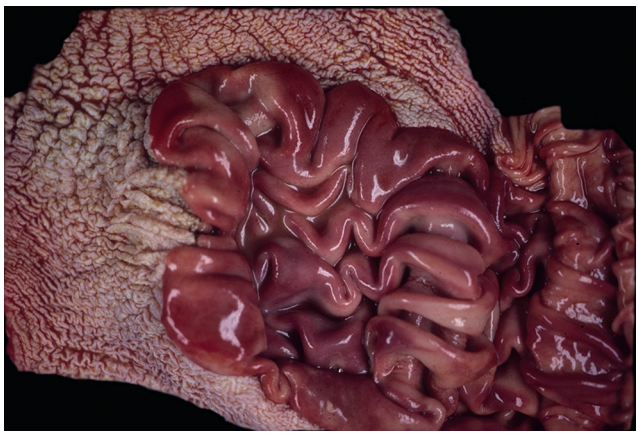

FIGURE 17.E1 Normal stomach of a greater one-horned rhinoceros. The margo plicatus is the junction between the keratinized squamous (left) and glandular (right) mucosal epithelium. *(Photo Courtesy of Smithsonian's National Zoo.)*

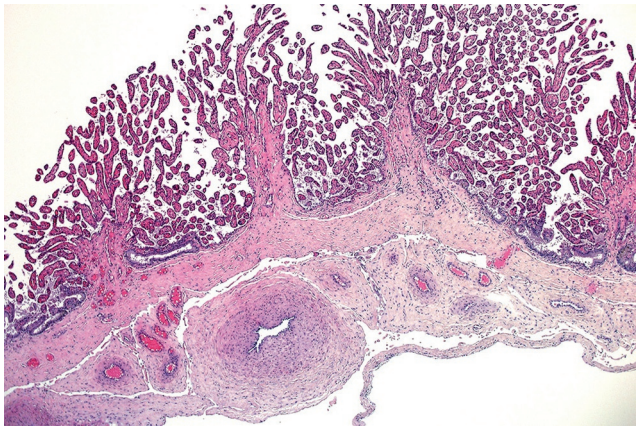

FIGURE 17.E2 Normal placenta from a Grevy's zebra. The allantochorion is diffuse, microcotyledonary, and epitheliochorial.

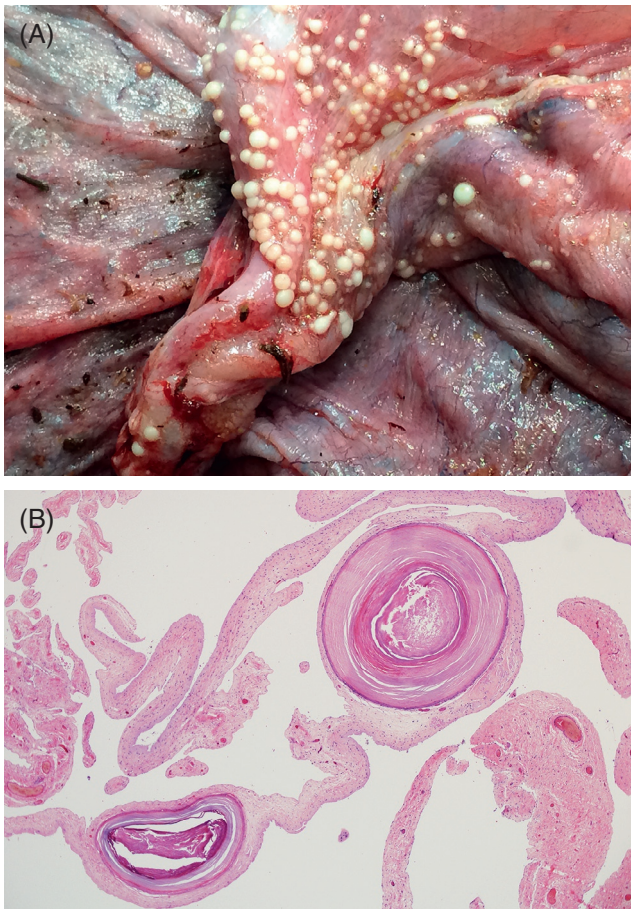

FIGURE 17.E3 Normal squamous plaques in the placenta of a black rhinoceros. (A) Round, yellowish, slightly raised squamous patches (pearls) are a normal finding and are often concentrated toward the umbilical cord. (B) These are composed of dense, concentrically laminated, acellular keratin that is rich in glycogen.

The feet of Perissodactyla have several unique modifications. In *Equidae*, the third digit is well formed and is the only weight-bearing digit. In general, domestic equids have larger feet with a greater frog-to-sole ratio than nondomestic equids. The radius and ulna and the fibula and tibia are fused.

In tapirs, the fleshy proboscis is a distinguishing feature that is more elongate in New World species. The stomach is small with a reduced squamous portion. Only Malayan tapirs have adhesions between the chest wall and lungs, though both parietal and visceral pleura are normally thick.[60] The kidneys are not lobulated.[152] Female tapirs may be larger than males. The testes are located in the inguinal canal in lateral recumbency and are otherwise cranioventral to the external anal sphincter in the slightly pendulous scrotum.[194] There are four digits in the forefeet.

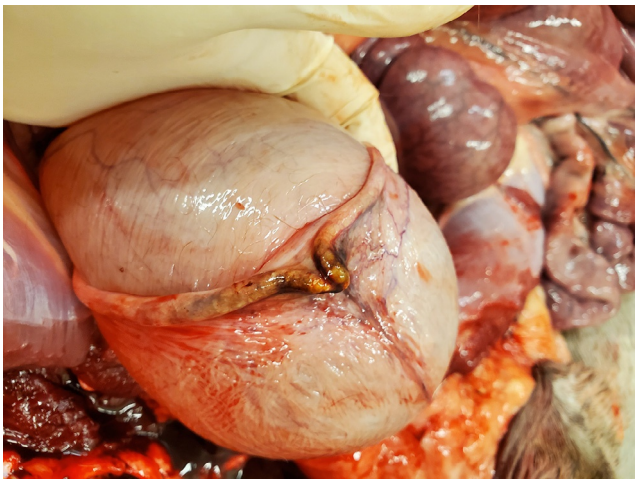

FIGURE 17.E4 Normal postpartum umbilical vessels in a Grevy's zebra. Expected pattern of umbilical artery retraction and clotting and lateral ligaments on the surface of the urinary bladder in a 3-month-old Grevy's zebra foal.

Digit V is small and only functional on deep, soft ground. Three digits are present in the hind feet. The radius and ulna and tibia and fibula are not fused.

The distinctive horn of the *Rhinoceridae* is anchored to the dermis over a roughened cushion of subjacent nasal bone (Fig. 17.e5A) and is composed of compressed keratin filaments (Fig. 17.e5B). When present (in the taxa *Ceratotherium*, *Diceros*, and *Dicerorhinus*), the second horn is more caudal on the frontal bones of the skull. An epipharyngeal bursa, an aggregate of mucous glands, lymphoid nodules, vessels, and nerve endings, acts as a pharyngeal tonsil.[154] In the male, the vesicular gland is multisacculate. The penis is caudally directed while retracted. The testes are not descended into a scrotum. The kidneys are lobulated. Skinfolds are more prominent in Asian species and the Sumatran rhino has a shaggy coat. In all *Rhinoceridae*, three weight-bearing digits (II, III, IV) are present on the fore and hindfeet and there is a fatty fibrous cushion just above the sole of the foot. The radius and ulna and tibia and fibula are distinct (not fused).

Nondomestic perissodactyls have similar clinical pathology responses to the domestic horse. Acute phase proteins are sensitive biomarkers that are highly conserved across species and increase with infection, neoplasia, and trauma. Major acute phase proteins can increase 1,000-fold while minor acute phase proteins increase only 2- to 10-fold in clinically abnormal animals.[18] In one study, an immunoassay showed serum amyloid A levels to be significantly higher in clinically abnormal white rhinoceros in managed care than clinically normal individuals; levels appeared to be of prognostic value.[101] Differences in clinical pathology values are seen between free-ranging and captive animals due to variation in the stressors of sample collection. Significant biochemical differences are also noted between adult and subadult black rhinoceros.[79] These include higher creatine phosphokinase in adults and higher creatine phosphokinase in males than females (presumably due to greater muscle bulk in males). Hypophosphatemia has been identified in captive black rhinoceros and linked to hemolytic anemia.[105] Calcium carbonate crystals give perissodactyl urine a cloudy/milky appearance; the change can be intensified by diet.[59]

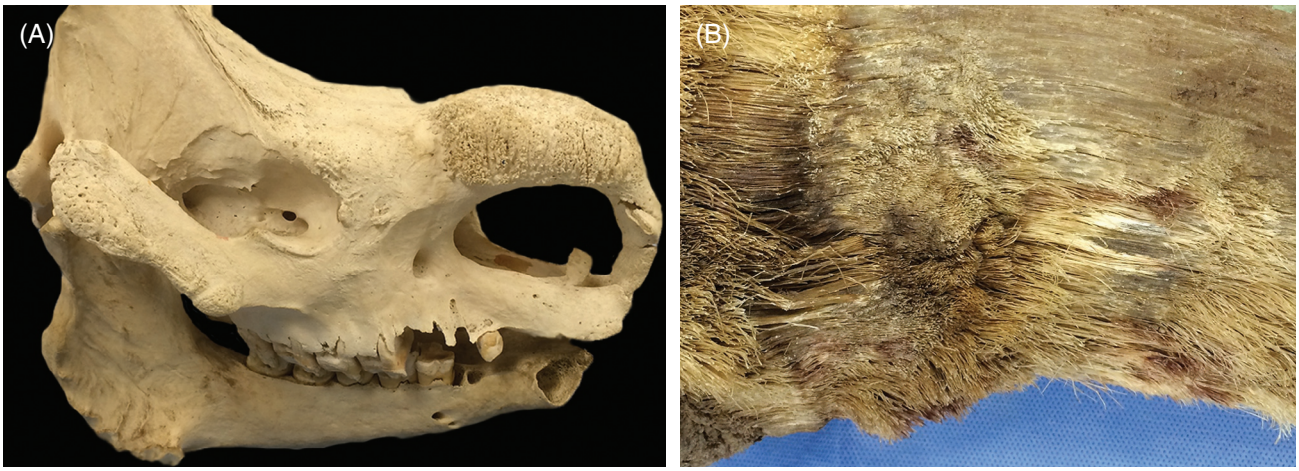

FIGURE 17.E5 Normal greater one-horned rhinoceros skull (A) and horn (B). (A) Roughening is present in the nasal bone over which the horn develops. (B) The horn consists of compact keratin filaments.

E-SLIDES

17.e1 **Iron overload, black rhinoceros, liver.** Brown pigment consistent with hemosiderin is present in the sinusoids of periportal Kupffer cells. Lesser amounts of similar pigment is present in hepatocytes. Also seen is periportal to bridging fibrosis and multifocal hemorrhage and necrosis, particularly at limiting plates. Pigment is also seen in monocytes in pulmonary capillaries. eSlide: VM05018

17.e2 **Iron overload, black rhinoceros, liver, lung. Perl's Iron.** Perl's iron stain highlights iron accumulation/hemosiderin in the liver, particularly in Kupffer cells periportally, in sinusoids, and to a lesser extent in hepatocyte cytoplasm. Hemosiderin-laden macrophages are also seen in the capillaries in the lung. eSlide: VM05019

17.e3 **Leiomyoma, Sumatran rhinoceros, uterus.** Expanding the myometrium is a large, unencapsulated, demarcated proliferation of densely organized, well-differentiated smooth muscle cells with abundant eosinophilic to fibrillar cytoplasm and elongate to polypoid, normochromatic nuclei; mitotic figures were not seen in twenty 40X fields. The tumor is moderately well vascularized. The adjacent myometrium and endometrium are moderately compressed. eSlide: VM05084

17.e4 **Sarcoid, Grevy's zebra, haired skin.** The tissue contains a locally extensive ulceration in association with a mildly exophytic poorly demarcated dermal mass. The mass is comprised of amphophilic spindle cells haphazardly arranged between dermal collagen bundles and extending to the cut margin. Cell boundaries are poorly delimited and have abundant amphophilic cytoplasm. Nuclei are oval to elongate. Rare multinucleated cells and no mitotic figures are seen. eSlide: VM05022

17.e5 **Sarcoid, Grevy's zebra, haired skin (pinna).** Within the skin there is multifocal expansion of the dermis by a poorly demarcated mesenchymal mass that separates collagen bundles. Spindle cells are poorly delimited, and have abundant amphophilic cytoplasm and elongate nuclei. eSlide: VM05023

17.e6 **Sarcoid, Grevy's zebra, haired skin (pinna).** There is multifocal ulceration and expansion of the dermis by a poorly demarcated mesenchymal mass that separates collagen bundles. Spindle cells are poorly delimited, have abundant amphophilic cytoplasm, and elongate nuclei. eSlide: VM05024

17.e7 **Mycobacteriosis, black rhinoceros, lung, spleen, liver.** In the lungs, there is an overall reduction in the open alveolar pattern with intralveolar eosinophilic substance (edema) and macrophages. Scattered histiocytic nodules with contain multinucleated cells and mineral; subpleural fibrosis is also seen. Throughout the spleen there is pigment deposition in the capsule and occasionally in the trabeculae and throughout the red pulp (hemosiderosis); similar pigment is present in hepatic Kuppfer cells and hepatocytes. eSlide: VM05021

17.e8 **Mycobacteriosis, black rhinoceros, lung, spleen, liver. Ziehl-Neelsen.** Acid fast positive bacilli are seen in low numbers most frequently in multinucleated giant cells in the lung. eSlide: VM05020

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