Chapter 17

Perissodactyls

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INTRODUCTION

Perissodactyls are odd-toed, hoofed ungulates with an uneven number of digits. Body weight is carried on the central digit of the limb. The group includes the *Equidae*, the *Tapiridae* and *Rhinoceridae* families. The taxonomy of this group is listed in Supplemental Table e1.

UNIQUE FEATURES

Perissodactyls are monogastric (Fig. 17.1) with an extensive hindgut for fermentation by microbiota and alloenzymes. All perissodactyls lack a gallbladder. Similar to the domestic horse, the nondomestic perissodactyls have guttural pouches that are paired ventral diverticula/outpouchings from the pharynx lateral to the hyoid bones. Mammary glands are inguinal. Placentation is diffuse, microcotyledonary, and epitheliochorial (Figs. 17.2 and 17.3).

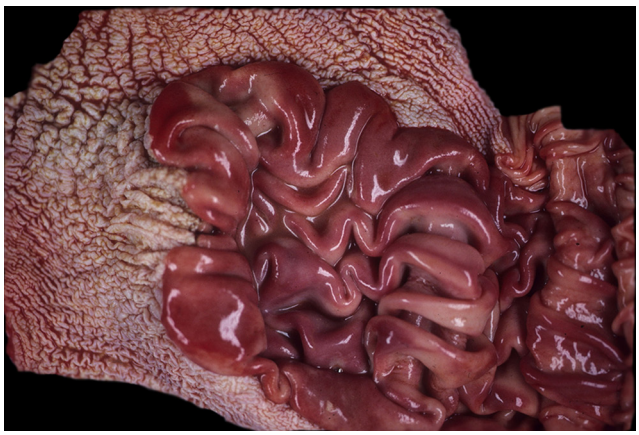

FIGURE 17.1 Normal stomach of a neonatal Indian rhinoceros. The margo plicatus is the junction between the keratinized squamous *(left)* and glandular *(right)* mucosal epithelium. *(Photo Courtesy of Smithsonian's National Zoo)*

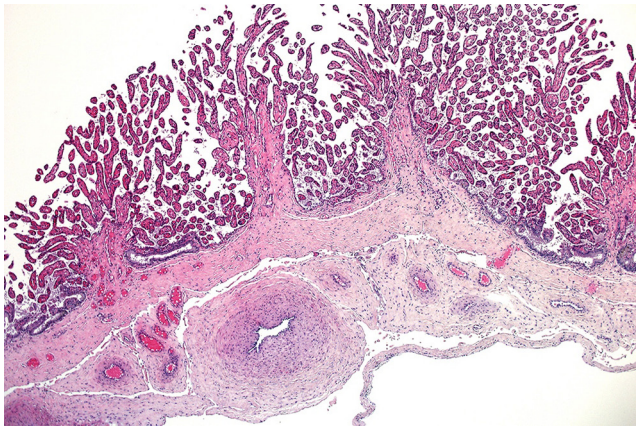

FIGURE 17.2 Normal placenta of a Grevy's zebra. The allantochorion is diffuse microcotyledonary and epitheliochorial.

Adenomatous hyperplasia of the placenta (Fig. 17.4) has been seen in greater Asian one-horned rhinoceros. The change has not been associated with placentitis or abortion.

The feet of the perissodactyla have several unique modifications. In *Equidae*, the third digit is well formed and the only weight bearing digit. In general, domestic equids have larger feet with a greater frog: 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 and has a small (reduced) squamous portion. Malayan tapirs alone have adhesions between the chest wall and lungs (Janssen et al., 1996). Female tapirs may be larger than the males. The testes are present in the inguinal canal in lateral recumbency and are otherwise cranioventral to the external anal sphincter in the slightly pendulous scrotum (Zimmerman and Hernandez, 2015). Four digits develop on

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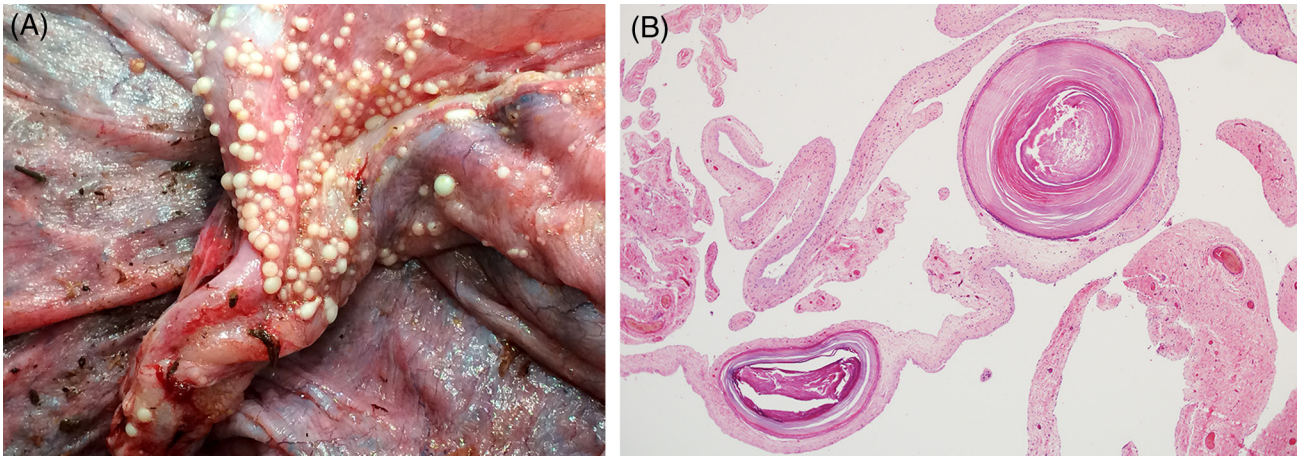

FIGURE 17.3 Normal round, yellowish, slightly raised squamous patches (pearls). (A) Concentrated toward the umbilical cord of a black rhinoceros placenta. (B) These are composed of dense, concentrically laminated, acellular keratin that is rich in glycogen.

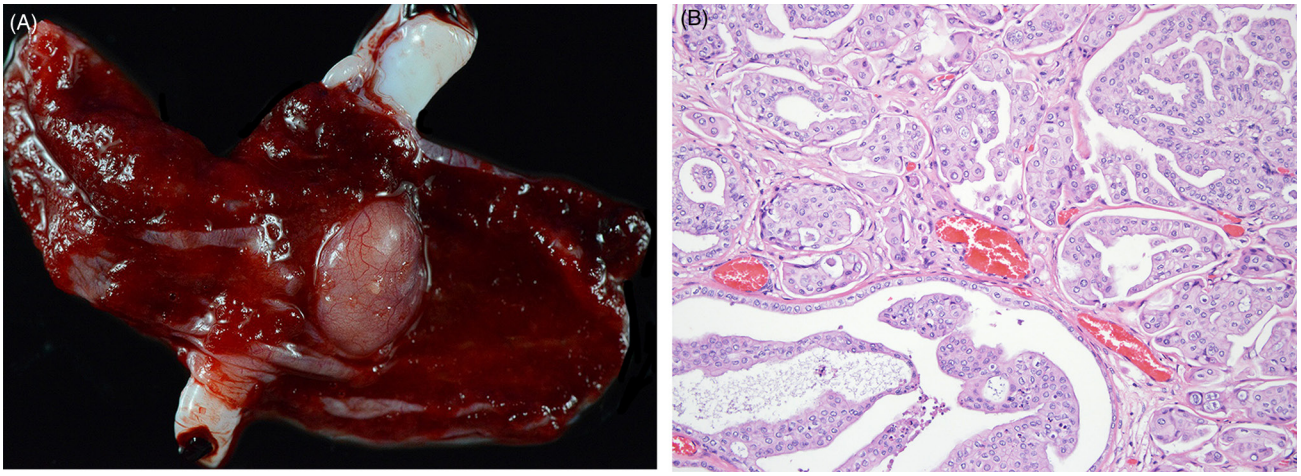

FIGURE 17.4 Adenomatous hyperplasia in the placenta of a greater Asian one-horned rhinoceros. (A) These can be grossly inapparent or appear as nodular foci. (B) Nodules are composed of variably sized glandular or cystic structures lined by cuboidal to low columnar epithelium or epithelial fronds with various amounts of fibrous stroma. *(Photo Courtesy of I. Stalis, Disease Investigations, San Diego Zoo Global)*

the forefeet, digit V being smaller and only functional on deep, soft ground; three digits develop on the hind feet. The radius and ulna, and tibia and fibula are separated.

The distinctive horn of the *Rhinoceridae* is composed of compressed keratin filaments (Fig. 17.5), anchored to the dermis over a roughened cushion of subjacent nasal bone (*Rhinoceros* spp.) (Fig. 17.6). 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 mucus glands, lymphoid nodules, vessels, and nerve endings acts as pharyngeal tonsil (Radcliffe et al., 1998). 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

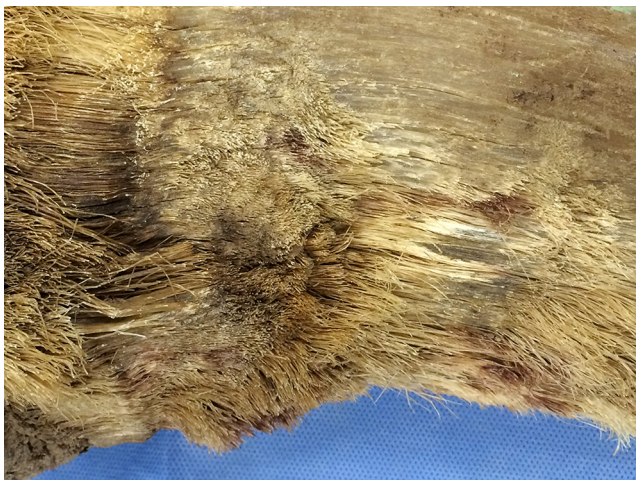

FIGURE 17.5 Normal rhinoceros horn. The horn consists of compact keratin filaments.

FIGURE 17.6 Normal Asian one-horned rhinoceros skull. Roughening is present in the nasal bone over which the horn develops.

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.

See Table e2 (Supplemental Materials) for a comparative dentition of the Perissodactyls.

Nondomestic Perissodactyls share similar clinical pathology responses with 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 1000-fold while minor acute phase proteins increase only 2–10-fold in clinically abnormal animals (Cray et al., 2013). 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 (Kock et al., 1990). 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 (Miller and Buss, 2015). Calcium carbonate crystals give perissodactyl urine a cloudy/milky appearance. This change can be intensified by diet (Janssen and Allen, 2015).

NON-INFECTIOUS DISEASES

Nutritional

Iron overload disease occurs in captive black and Sumatran rhinoceros as well as Baird's, Malayan, and Brazilian tapirs (Bonar et al., 2006; Miller, 2003; Paglia et al., 2000). The mechanisms underlying iron overload in susceptible perissodactyls is currently not well understood and is an area of active research; the pathogenesis is likely multifactorial. In general, a number of intrinsic and extrinsic factors influence iron absorption and the development of iron overload. 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. Circulating hepcidin binds ferroportin and prevents iron uptake. When hepcidin levels are high, iron remains in enterocytes and is shed in the feces. Hepcidin production is increased under certain conditions, such as inflammation from infection or autoimmune disease (Ganz and Nemeth, 2012). In conditions of iron deficiency and increased erythropoiesis, hepcidin production is down regulated to allow increased iron absorption. Deficiencies in hepcidin lead to increased iron absorption and subsequent deposition in hepatocytes, cardiomyocytes, and endocrine glands. Hemochromatosis 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 were identified across rhinoceros species. A protein polymorphism was found in the black rhinoceros alone, not the white, Indian or Sumatran. The S88T protein polymorphism is in the portion of the HFE protein that interacts with the transferrin receptor and may have allowed adaptation of the black rhinoceros to a low iron diet by aiding iron uptake (Beutler et al., 2001; Olias et al., 2012).

The diet of wild browsing rhinoceroses is rich in compounds that chelate iron (tannins, phytates, polyphenolic compounds), forming insoluble complexes that pass through the gastrointestinal tract unabsorbed (Paglia and Dennis, 1999). Hemosiderosis has not been described in free ranging or recently collected rhino (Kock et al., 1992a). In captivity, it is thought that lower levels of these iron binding compounds increase the amount of dietary iron available for absorption. Additionally, ascorbate (vitamin C) increases iron absorption and ascorbate rich produce can increase uptake in captive rhinoceros. The total bioavailability of inorganic iron cannot be predicted simply from the measurement of total dietary iron because absorption is affected by these dietary elements (e.g., tannins, vitamin C) (Smith et al., 1995). Black rhinos in captivity commonly experience hemosiderosis and complications of iron overload. However, captive white and Indian rhinoceros store low levels of iron but do not demonstrate ferritin and tissue iron elevations suggestive of iron storage (Miller and Buss, 2015). One difference between these species is that the black rhinoceros are browsers while white and Indian species are grazers. Based on these dietary factors and previously discussed genetic data, the black rhinoceros appears

to have adapted its iron absorption to a naturally low iron diet, and overload develops with loss of homeostasis in captivity when the diet has high iron bioavailability (Beutler et al., 2001; Ganz and Nemeth, 2012).

Lesions of iron accumulation (hemosiderosis) in rhinoceros are typically seen in the spleen, liver, small intestine, and lungs of black rhinoceros, spleen, and other organs in Indian rhinos, and intestines, liver, and lung in white rhinos (Olias et al., 2012). 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. Extensive iron deposition in both the reticulo-endothelial and parenchymal cells of multiple tissues suggests the condition is iron overload rather than the result of hemolysis. Initially, hemosiderin is deposited in Kupffer cells but over time periportal hepatocytes also have increased iron staining (Figs. 17.7 and 17.8); in severe hemochromatosis periportal fibrosis develops. Accumulation of iron in the bone marrow may cause myelodysplasia (Smith et al., 1995). In affected animals bone marrow is hypocellular and often fibrotic, with sheets of hemosiderin laden macrophages.

Iron storage disease is connected with a variety of related disease concerns. 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 (Kock et al., 1992b; Paglia and Dennis, 1999; Smith et al., 1995). 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.

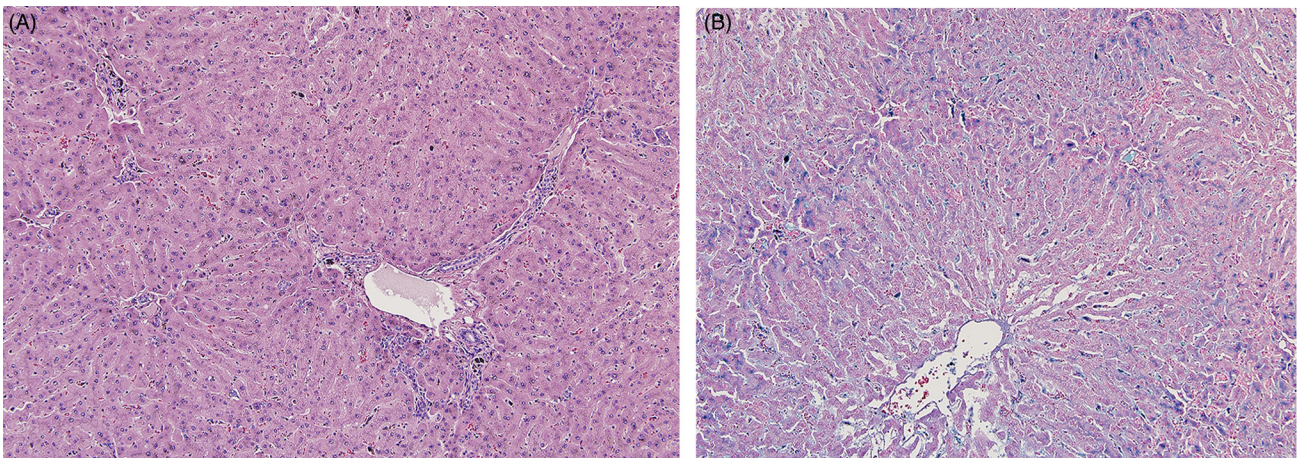

FIGURE 17.7 Mild hepatic iron overload (hemosiderosis) in a black rhinoceros. (A) Mild iron accumulation appears as granular brown intracytoplasmic pigment in intrasinusoidal Kupffer cells. (B) Iron is highlighted with special stains. Perl's iron/Prussian blue.

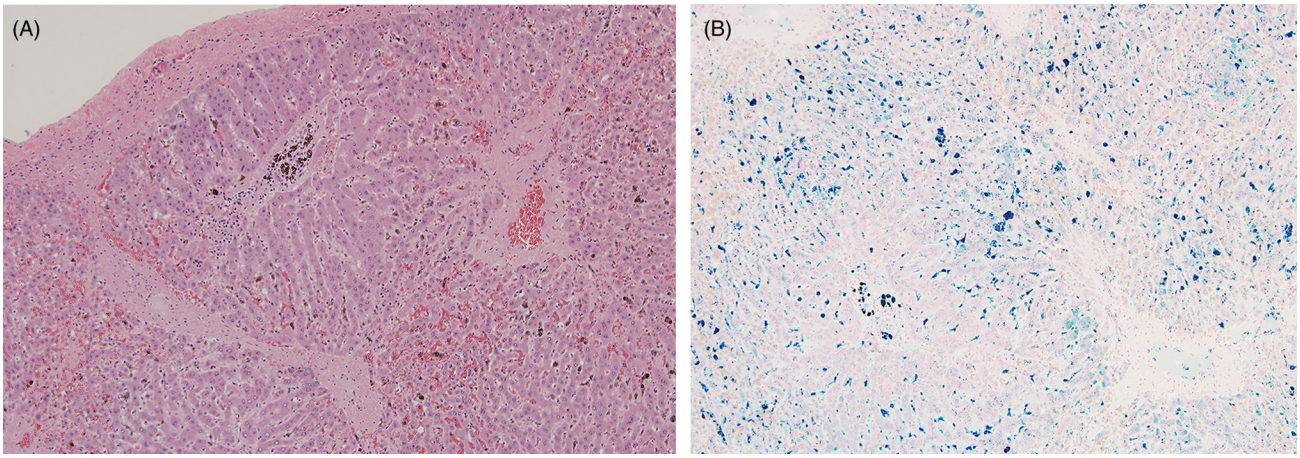

FIGURE 17.8 Moderate iron overload disease (hemosiderosis) in the liver of a black rhinoceros. (A) Moderate periportal deposits of pigment-laden Kupffer cells are associated with periportal fibrosis. (B) Compare the amount of stainable iron with mild accumulation in Fig. 17.7. Perl's iron/Prussian blue stain.

While iron storage disease and mycobacteriosis have been diagnosed concurrently, no study on the causal relationship has been performed in rhinoceros.

Serum ferritin concentration is a good estimate of total body iron stores; it is usually measured by an enzyme-linked immunoabsorbent assay (ELISA). Antibodies to equine ferritin cross react with rhinoceros ferritin making this a useful test in both species. Serum ferritin levels in black rhinoceros tend to increase over time in captivity. 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 was significantly higher in black rhinoceroses than white rhinoceroses, and the level in black rhinoceroses increased with age (Smith et al., 1995).

The pathogenesis of iron overload in tapirs has been studied less extensively and is an area of needed research. Captive tapirs develop increasing serum ferritin and have increased transferrin saturation (mountain > Baird's > Malayan) relative to free-ranging Baird's tapirs (Paglia et al., 2000).

Vitamin E (alpha-tocopherol) is important in maintaining membrane integrity relative to oxidant stress. **Hypovitaminosis E** can lead to hemolysis, which has been reported in primates, rats, and horses, and causes low bile salt secretion and reduced solubilization of dietary fat. It has been implicated in **hemolytic anemia and encephalomalacia** in black rhinoceros and **myopathy** in a Brazilian tapir (Yamini and van Veen, 1988). As with horses, low levels of vitamin E can be associated with histiocytic steatitis (Fig. 17.9). Lower circulating blood levels of vitamin E have been reported in captive versus free-ranging black rhinoceros. This may reflect a biochemical deficiency or variation in the bioavailability of different forms of the vitamin (Dierenfeld et al., 1988; Papas et al., 1991). The form of vitamin E present in the diet greatly affects its bioavailability and different species vary in the way they utilize the different forms of vitamin E. Grazers and browsers may have different dietary requirements for vitamin E so the form of supplementation should be considered for each species. For example, Dierenfeld et al. (1995) demonstrated higher vitamin E in the leaves than the twigs of the same browse plants in Zimbabwe. Vitamin E increases with chlorophyll degradation during plant maturation and would be expected to be higher in photosynthesizing leaves. Captive black rhinoceros diet is higher in produced and dried forage with little access to fresh pasture. **Degenerative myelopathy** in captive Mongolian wild (Przewalski) horses (Liu et al., 1983) is also associated with hypovitaminosis E. 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 have not been noted. The most significant lesions are in the caudal cervical and cranial thoracic spinal cord and consist of degeneration, demyelination and astrogliosis of the ventral and lateral funiculi. 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. Dietary alpha-tocopherol (vitamin E) concentrations in the feed of affected Mongolian horses, was low.

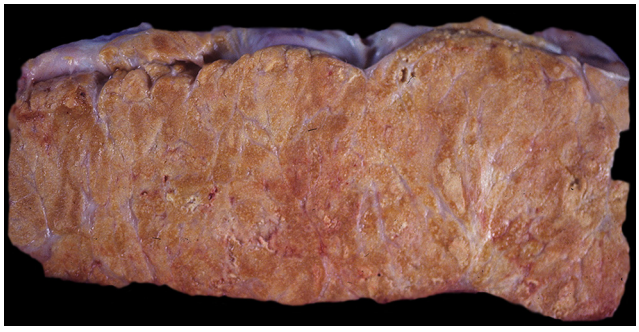

FIGURE 17.9 Chronic histiocytic steatitis due to presumed hypovitaminosis E in the fat from the abdominal body wall of an onager. The fat is discolored and may be firm. *(Photo Courtesy of Smithsonian's National Zoo)*

Microcytic, hypochromic iron deficiency anemia, has been reported in captive Malayan tapir neonates from two institutions (Helmick and Milne, 2012). Both had unremarkable neonatal examinations and blood parameters but developed anemia by one to two months of age. Notable hematologic findings were anisocytosis, polychromasia and hypochromia with acanthocytes, schistocytes, and poikilocytes. Stores of iron are 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.

Copper deficiency has been diagnosed in tapirs (Janssen et al., 1996, 1999). 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.

Obesity is a common concern in all captive equids that receive a rich diet and get relatively little exercise (Nelson, 1986).

Metabolic

Capture myopathy (exertional rhabdomyolysis) is a potential problem in all Perissodactyls but the condition is of greatest concern during and in association with capture and

translocation of rhinoceros (McCulloch and Achard, 1969). Lesions are consistent with those seen in bovids (see Chapter 5) and cervids (see Chapter 6).

Toxic

A number of chemical and biological toxins have been reported in nondomestic Perissodactyls. Most are single case reports or series.

Organophosphate toxicity has been described in Damara zebra following deworming and has been implicated in mortalities of two black rhinoceros with degenerative myopathy (McCulloch and Achard, 1969; Nelson, 1986).

Vitamin D_3 toxicosis was implicated in the deaths of three black rhinoceros following a compounding error. Over 5 months, three of 10 rhinoceros developed hypercalcemia and partial anorexia. Terminally, increased blood urea nitrogen and creatinine were noted. At necropsy, two animals had varying degrees of mineralization in major blood vessels, myocardium, lung and kidney. Pelleted diet had levels of vitamin D_3 13–38 times greater than normal. The serum levels of 25-hydroxy vitamin D_3 were mildly elevated in two of the animals and one had markedly elevated tissue liver 25-hydroxy vitamin D_3 (Fleming and Citino, 2003).

Blue-green algae (*Microcystis aeruginosa*) intoxication in rhinoceros is associated with random massive acute hepatic necrosis (Miller, 2003). In some areas, algal blooms occur periodically in association with high organic matter and low water levels (Miller and Buss, 2015).

Red Maple (*Acer rubrum*) toxicosis was the presumed cause of *hemolytic anemia* in two female Grevy's zebra with Heinz body formation and leukocytosis (Weber and Miller, 1997). Methemoglobinemia, a factor considered to be associated with high mortality, occurred in the female with generalized icterus, hemoglobinuric nephrosis and paracentral hepatic necrosis. Hybrid red maple trees present in the holding area were the only source of identified toxin. Cases of red maple toxicity are seasonal and associated with ingestion of wilted or dried leaves that contain gallic acid. The precise toxin has not been isolated but causes severe oxidative damage to erythrocytes.

Sycamore seed (*Acer pseudoplanatus:* sycamore in the United Kingdom, sycamore maple in the United States) ingestion associated with **equine atypical myopathy** has been described in a Przewalski's horse foal (Molenaar et al., 2016). The foal presented with sudden onset of stretching of the neck and progressive weakness. Biochemistry results indicated markedly increased plasma creatine kinase, increased aspartate aminotransferase and a mild increase in inorganic phosphorus concentration. Other findings were neutrophilia and passage of dark brown urine with elevated protein and hemo/myoglobin. On histologic examination, there was rhabdomyolysis of skeletal and cardiac muscle. Organic acid analysis of the urine and the plasma acyl carnitine profile were consistent with multiple acyl Co-A dehydrogenase deficiency. Inhibition of these enzymes by hypoglycin A has been associated with equine atypical myopathy, in which fatty acid oxidation in muscle mitochondria is defective and muscle weakness develops. Seeds of the sycamore maple contain hypoglycin A; sycamore maples were present in the foal's enclosure.

Suspected **creosote toxicity** was reported in black rhinoceros approximately one month after 20 wild caught animals were moved into a creosote-treated holding pen. Four individuals became lethargic, anorexic, anemic, and icteric with elevated aspartate transaminase and bilirubinemia (unconjugated and conjugated) and dark brown urine (Kock et al., 1994). One individual died, another became moribund and was euthanized. At necropsy, both animals had icterus, ulcers in the oral cavity and stomach, hemorrhages throughout the body, hyperplastic bone marrow and enlarged, cholestatic green livers. Histologically, there was acute periacinar hepatic necrosis. Direct injury through ingestion of a corrosive substance was suggested as the cause of the ulcerations. After translocation a further five animals died with similar liver lesions. Creosote derived from coal tar, is a mixture of compounds, cresols, frequently used for wood preservation, since it is toxic to insects and fungi. Creasote toxicity was also suspected as the cause of death in two captive black rhinoceros that died after a protracted period of anemia, with possible icterus and ulcerative skin lesions. Histologically hepatocytes were degenerate, individualized and contained green/brown pigment. Ultrastructurally, the pigment appeared to be within membrane-bound phagosomes. White rhinoceros housed near the black rhinoceros were unaffected. The fencing for the black rhino exhibit only was old telephone poles that may have been treated with a wood preservative, such as creosote (Schmidt et al., 1982).

A single dose of **anthracycline (doxorubicin) chemotherapy** given to a juvenile black rhinoceros with acute lymphoblastic leukemia was thought to have been cardiotoxic (Paglia and Radcliffe, 2000). Congestive heart failure with ventricular dilatation, severe acute and chronic passive congestion of the lungs, liver and spleen were seen at necropsy. No leukemic infiltrates were found. Doxorubicin–induced cardiomyopathy in humans leads to myocardial edema and degeneration, severe acute and chronic passive congestion in the lungs, liver, and spleen, and pulmonary edema.

Congenital/Genetic

Familial degenerative myelopathy was reported in eight of 17 Burchell's zebra foals, the progeny of one stallion and two mares (Montali et al., 1974). Progressive ataxia developed between 4 and 6 months of age in the foals; there were no radiographic or postmortem findings to indicate narrowing of the vertebral canal. 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; cranially, lesions extended to the medulla oblongata. Small blood vessels in these areas 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. **Schistosomus reflexus-like malformation** was reported in a white rhino calf aborted at approximately 13 months of gestation (Lankton et al., 2014). The fetus exhibited spinal inversion and a ventral midline abdominal defect with externalization of the abdominal viscera. Computed tomography revealed severe scoliosis, a spiral rotation of the spine, multiple vertebral anomalies, hypoplasia of multiple bones of the skull, and mild prognathism. In cattle, autosomal recessive inheritance is suspected 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.

A 6-day-old black rhinoceros died with congestive heart failure as a result of **persistent truncus arteriosus and ventricular and atrial septal defects** (Reese and Edwards, 1996). Hypoplastic pulmonary arteries and secondary right and left ventricular dilation were 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. A black rhinoceros calf less than 12 h old died with **cardiac septal defects**, **cleft palate**, and **patent foramen ovale** (Lewis et al., 2016).

Atresia ani has caused death in neonatal tapirs; as with domestic species, the lesion leads to clinically apparent abdominal distention (Janssen et al., 1996). 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. In tapirs, atresia ani has been identified without associated fistula.

Age-Related/Degenerative

Chronic renal disease in aged rhino includes mononuclear interstitial nephritis and fibrosis with tubular degeneration and loss (Fig. 17.10). As with sequela to renal disease in many species, blood and urine analysis can be used for clinical monitoring. Secondary changes, such as uremic mineralization in the lungs (Fig. 17.11A) or stomach (Fig. 17.11B) and gastric ulcers. High energy, low protein diet management may help (Miller and Buss, 2015).

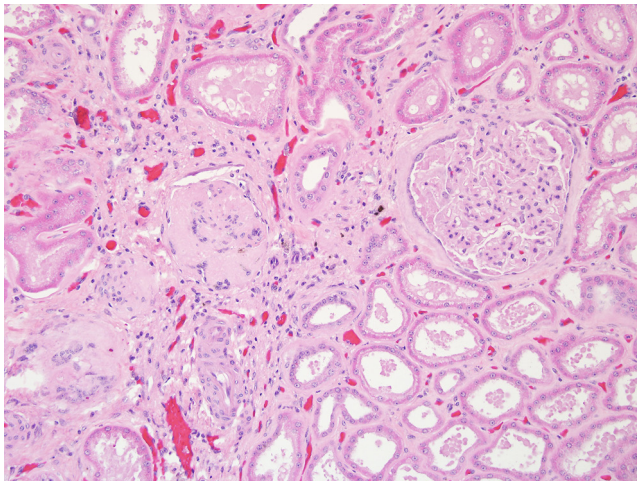

FIGURE 17.10 Chronic renal disease in black rhinoceros. Chronic nephritis is associated with interstitial and periglomerular fibrosis, glomerular fibrosis and senescence, mild multifocal tubular dilation and multifocal tubular drop-out.

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 (Regnault et al., 2013). Twenty-two individuals had bone lesions in at least one limb. The authors identified six categories of lesions 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. Enthesopathies are lesions at the bone surface at sites of ligament and tendon attachment, usually characterized by new bone deposition and thought to reflect the response of bone to forces applied to it. Enthesopathies were present at the dorsoproximal phalanges of the digits 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 (Miller, 2003). Dental disease, mandibular and maxillary osteomyelitis (lumpy jaw) and facial abscesses are common in aged tapirs. These lesions are thought to be associated with rough feed that allows pathogen entry. Surveys of dentition in museum specimens have indicated a markedly different pattern of wear and lesions between Malayan and Brazilian (lowland) tapir species. Malayan tapirs are selective browsers and feed on soft vegetation. In skulls and mandibles from Malayan tapirs, dental lesions were identified in 52% of the captive and 6% of the free-ranging tapirs (Da Silva et al., 2011). They were most common at the second, third, and fourth premolars and the first molar teeth and consisted of resorption with loss

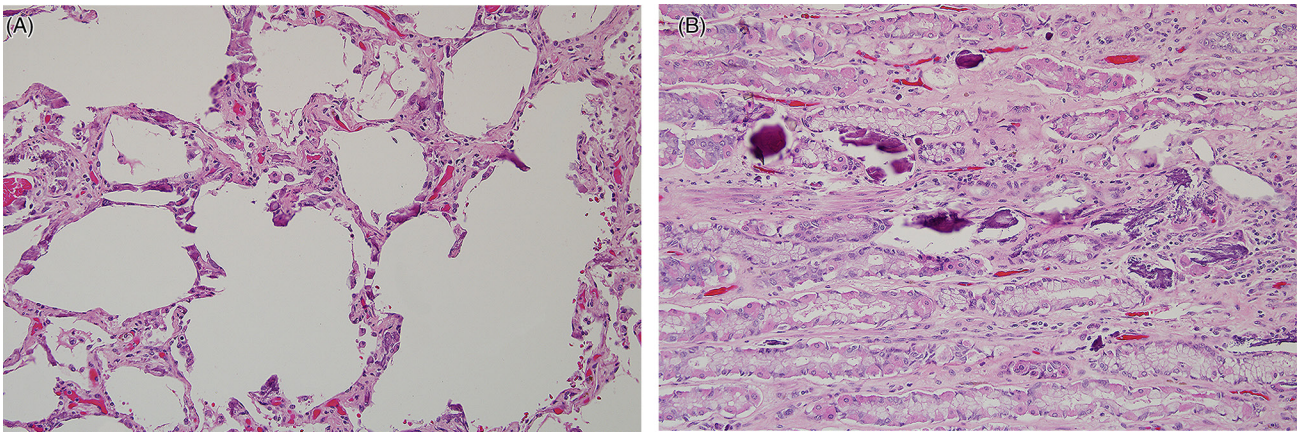

FIGURE 17.11 Chronic renal disease in black rhinoceros. Complications can include pulmonary (A) and/or gastric (B) mineralization secondary to uremia.

of substance extending into the tooth from just below the cementoenamel junction. The subgingival location suggests 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 Brazilian tapirs, 24% had lesions with similar prevalence in free-ranging and captive animals (Tjornelund et al., 2015). The most common problem was 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 Brazilian tapir had caries-like lesions. The lesions in the Brazilian tapirs were most consistent with dental trauma. Increasing age was positively correlated with lesion development.

Trauma

Fracture of cervical vertebra and femur has occurred during courtships of Somali wild ass (Pagan et al., 2009). Traumatic lesions during breeding also occur in many species of rhinoceros (Miller and Foose, 1996). **Bite wounds** are common in *Equidae*. **Cervical fractures** occur with some frequency in captive zebras after collision with enclosure fencing (Suedmeyer et al., 2006). Animals are often found dead at the fence with no history of any illness. Diagnosis is straight forward radiographically and on gross examination based on the cervical hemorrhage and disrupted vertebrae. **Rhinoceros horn avulsion** can occur if horizontal poles are used for fencing. **Poacher-related traumas** from dehorning and snares are common in wild rhinoceros. **Interspecies aggression** may occur between male tapirs housed together.

Severe decubitus ulcers with necrosis have been described in white rhinoceros. One neonate with mild angular limb deformity developed ulcers secondary to reduced mobility. Another rhino developed ulcerations due to trauma associated with cement flooring during winter housing. Particular susceptibility to pressure necrosis in the skin of neonatal rhinos has been suggested (Gandolf et al., 2006).

Inflammatory, Non-infectious

A variety of skin diseases affect captive black rhinoceros. **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**) is relatively common. Affected individuals have lesions that develop over time and are of variable severity (Munson et al., 1998). Animals of all ages can be affected. Epidermal plaques progress to vesicles or pustules then erosions or ulcerations; lesions are generally bilateral, possibly symmetrical and have 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. Rhinoceroses appear depressed, anorexic and lose weight. Hypoalbuminemia, hypocholesterolemia and decreased hematocrit reflecting loss of albumin and blood through the skin may be found. In domestic species with similar gross and histologic lesions there is an association with hyperglucagonemia or hypoaminoacidemia, and low circulating amino acids that disrupt epidermal homeostasis cause ulceration or increased susceptibility to injury. Glucagonoma, diabetes mellitus, hepatic disease, hyperadrenocorticism, and hypothyroidism have been linked to the skin lesions in domestic species. In black rhinoceros, hypoaminoacidemia was not confirmed in a study in which monthly plasma levels were collected (Dorsey et al., 2010).

Histologically, skin lesions have a layered "red-white-blue" appearance: superficial parakeratosis with eosinophilic, nuclear retention (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 (Munson and Miller, 1999).

Epidermal exfoliation may be a variant of superficial necrolytic dermatopathy. The disease is associated with shedding of sheets of superficial hyperkeratotic epidermis from the thorax and flanks leaving a shiny gray surface. Intraepidermal pustules are occasionally seen with no further epidermal degeneration, that is, not as 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.

Some captive black rhinoceroses have frequent episodes of **ulcerative skin/mucocutaneous disease**, alone or in association with other disease problems. This condition is unrelated to the specific skin diseases listed earlier. The skin of rhinos has a low density of hair follicles and 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 (Munson and Miller, 1999).

Nodular collagen degeneration with dystrophic mineralization has been reported in the rhinoceros, alone or in association with chronic ulcers. 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 (Munson and Miller, 1999).

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 (Munson and Miller, 1999; Pessier et al., 2004). Lesions can resolve spontaneously; corticosteroid and cryotherapy have been effectively used to resolve lesions. 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 (Bishop et al., 2016).

Vesicular skin disease is described in tapirs. Lesions most commonly develop in the dorsal cervical and lumbosacral areas (Finnegan et al., 1993, Janssen et al., 1999) and resolve spontaneously. Resolution may be associated with fluctuating hormone levels in females (Zimmerman and Hernandez, 2015). 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. Sloughing and repair occur approximately a week after lesions first develop. Viral particles have not been identified in vesicular fluid by electron microscopy, and virus was not isolated from equine fibroblast culture. On survey, tapir (18 Malayan, 9 Brazilian, 5 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. The histologic diagnosis has features 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 not consistent with that in other species.

A survey of free-ranging Baird's tapir in Costa Rica identified **depigmentation of undetermined etiology** along the dorsum, perineum, inguinal and perioral regions (Hernandez-Divers et al., 2005). Histologically the epidermis was hyperplastic with little pigment. Mild mononuclear cell infiltrates were present in the superficial dermis and there were perivascular eosinophils. Hairs were generally in the catagen or telogen stage of development. Leukoderma/vitiligo was diagnosed due to acquired loss of pigmentation. Histologic lesions were consistent with hypersensitivity dermatitis; a specific etiologic agent was not identified but a response to biting insects was suspected. A captive black rhinoceros developed nonpigmented foci initially at the nares and lips, later multifocally at the limbs, head and trunk totaling 15% of the body surface. Multifocal hypopigmentation of the basal layer of the epidermis with pigmentary incontinence and perivascular lymphohistiocytic dermatitis were seen. Phototherapy induced repigmentation (Takle et al., 2010).

In the 1980s and 1990s, a third of captive black rhinoceros deaths in North American zoological facilities were associated with episodes of **acute intravascular hemolytic anemia.** Early cases were characterized by hemoglobinuria, lethargy, and severe anemia. Rare animals survived episodes with supportive care (Douglass and Plue, 1980; Miller and Boever, 1982). Investigated causes of hemolytic anemia

included: autoimmune hemolytic anemia, equine infectious anemia, equine viral arteritis, copper toxicosis, vitamin E deficiency, and clostridial infection (Miller, 2003). 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 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 of hemolytic anemia (see iron overload disease discussed earlier) (Paglia and Miller, 1993; Paglia and Dennis, 1999). The relationship between hemolytic anemia and iron accumulation in the tissues is still not understood (Smith et al., 1995). Histologic lesions include renal tubular casts with luminal 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 (Paglia and Miller, 1992). Several factors have been suggested in the pathogenesis of hemolytic syndromes in black rhinoceros. Erythrocyte glucose 6 phosphate dehydrogenase activity in black rhinos is normal (Paglia and Miller, 1993). **Low erythrocyte catalase activity**, which leads to decreased neutralization of reactive oxygen species and is associated with gangrenous oral ulcers in humans (Takahara's disease), is seen in black rhinoceros erythrocytes. However, enzyme levels are similar in black rhinos with or without hemolytic anemia and are similar among black, white, Asian, and Sumatran rhinos (Paglia and Tsu, 2012). Low erythrocyte adenosine kinase activity (important for maintaining ATP levels) (Paglia and Miller, 1993), hypophosphatemia (inhibits glycolysis, reducing the amount of available ATP) (Miller, 1993), and low erythrocyte ATP levels (relative to humans) have all been suggested as possibly predisposing red cells to oxidative stress (Paglia and Miller, 1993). 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 (Paglia and Miller, 1992). Rhino red blood cells have very low levels of activity for additional kinase enzymes, such as catalase and glutathione S-transferase. While there have been multiple intriguing findings, no definitive pathogenesis has been elucidated to explain cases of hemolytic anemia in rhinos. Cases in recent years have been rare; however this does confound the search for a cause.

Suspected neonatal isoerythrolysis (syn. **hemolytic icterus, hemolytic anemia**) occurred in two Baird's tapir (Wack and Jones, 1997). 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 also been reported and may be due partly to natural antibodies to erythrocytes of a different species. It is unknown whether equine colostrum is appropriate as a replacement for tapir colostrum.

Idiopathic hemorrhagic vasculopathy syndrome is a concern in black rhinoceros. Of seven reported cases, six occurred in Texas (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 and associated with a rapid fall in the hematocrit and serum phosphorus resulting in hypophosphatemia. Histologically, extravasation from small vessels occurs; neutrophilic vasculitis in the superficial limb dermis and proliferative vasculitis at the oral mucosa are seen and suggestive of immune-mediated disease. Extensive testing to identify an infectious agent has been unproductive, and the cause remains unknown (Murray et al., 1999; Paglia and Tsu, 2012). Equine purpura hemorrhagica, a sporadic noncontagious allergic reaction to streptococcal or viral antigens appears to be the closest 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 (Pagan et al., 2009). High protein diets have been proposed as a cause of laminitis in captive rhino (Miller, 2003). Additional predisposing conditions may include excessive feeding of concentrates, enteritis, chronic renal failure, and acute intravascular hemolytic anemia. The disease begins when the blood supply to the corium, the sensitive laminae of the foot, is interrupted. In severe cases, the union between the horny and sensitive laminae breaks down and progresses to separation of the nail at the coronary band. The first signs of laminitis are lameness and pain or discharge at the coronary band. It is possible for the affected nail to remain while the new nail grows and displaces it. With total separation, the nail may remain attached at the sole (Von Houwald, 2016).

Greater one-horned rhinoceroses are prone to developing **chronic foot disease**. This species lives in river plains and are "hoof-walkers," with the outer rim of the hoof wall

being more dense and carrying the animal's weight. In captivity on hard substrate, there is a tendency to develop foot problems especially if animals become overweight. In a 1996 international survey, chronic foot disease affected nearly 25% of the captive population. Male rhinos were twice as likely to suffer from chronic foot disease as females. The condition usually occurs in one or both hind feet, but all four feet are vulnerable. The hoof wall breaks down/cracks as more stress is put on the more delicate central pad than the more structurally robust outer wall. 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 and trauma promotes separation, with resulting scar tissue. The lesions are prone to secondary bacterial infection due to their location. 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 (Von Houwald, 2016).

Though not specifically reported in the literature, many non-infectious **chronic inflammatory diseases** are seen in aged individuals. With time, these lesions often have associated degeneration and mineralization. **Chronic degenerative vascular diseases,** including *arteriosclerosis and atherosclerosis* (Fig. 17.12), 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 aneurysms**, with or without rupture and hemopericardium, have been described in black rhinoceroses (Kock et al., 1991; Miller, 2003). 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 (Kock, 1996). The cause of the lesions was undetermined. Sites of chronic skin trauma can develop fibrosis and mineralization (Fig. 17.13).

Gastrointestinal tract disease is a common source of problems in the domestic horse and similar conditions are seen across nondomestic Perissodactyls (Fig. 17.14A–E). Dilation of the caudal esophagus was associated with regurgitation in a black rhinoceros whose symptoms resolved on a soft diet (Radcliffe et al., 1998). In one study, a third of adult tapir deaths involved the gastrointestinal tract (Janssen et al., 1996). Findings included intestinal volvulus, gastric and colonic impactions, colonic incarceration, oropharyngeal abscess/oral necrobacillosis, oropharyngeal trauma, acute pancreatitis, and eosinophilic enterocolitis. Enteroliths 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) (Murphy et al., 1997). Sand and shavings have caused colic and intestinal impaction in *Equidae* and *Tapiridae*, especially newborns (Zimmerman and Hernandez, 2015). Gastrointestinal problems occur with over consumption, especially if food lacks fiber as the stomach is relatively small (Zimmerman and Hernandez, 2015). 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 related dehydration. Prolapse has also been reported in rhinoceros (Abou-Madi et al., 1996; Bertelsen et al., 2004). Prolonged tenesmus, constipation, diarrhea, intestinal protozoal parasites, dystocia, urinary obstruction, proctitis, rectal tear, and space occupying

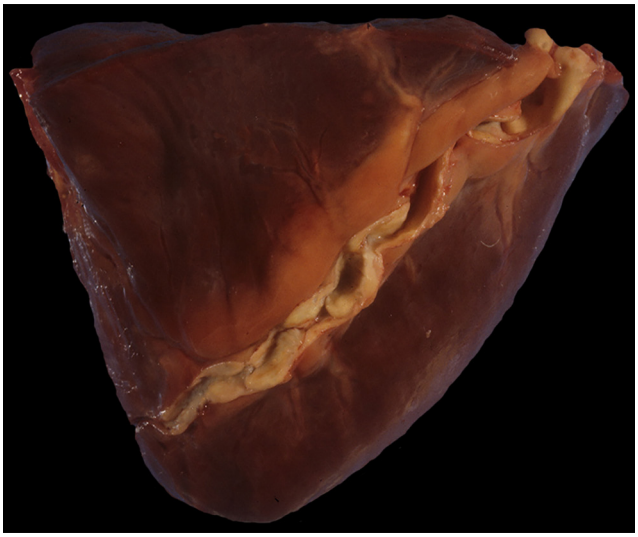

FIGURE 17.12 Arteriosclerosis in a Malayan tapir. There is moderate mural thickening of the cardiac arteries in the interventricular groove of the heart. *(Photo Courtesy of Toronto Zoo)*

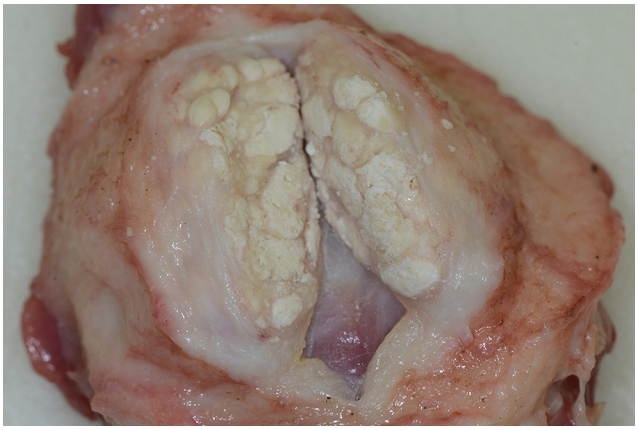

FIGURE 17.13 Calcinosis circumscripta due to chronic trauma in the subcutis of the thorax in a white rhinoceros. Sequela to chronic trauma includes fibrosis and mineralization characterized by the nodular deposition of mineral in the soft tissues. *(Photo Courtesy of Disease Investigations, San Diego Zoo Global)*

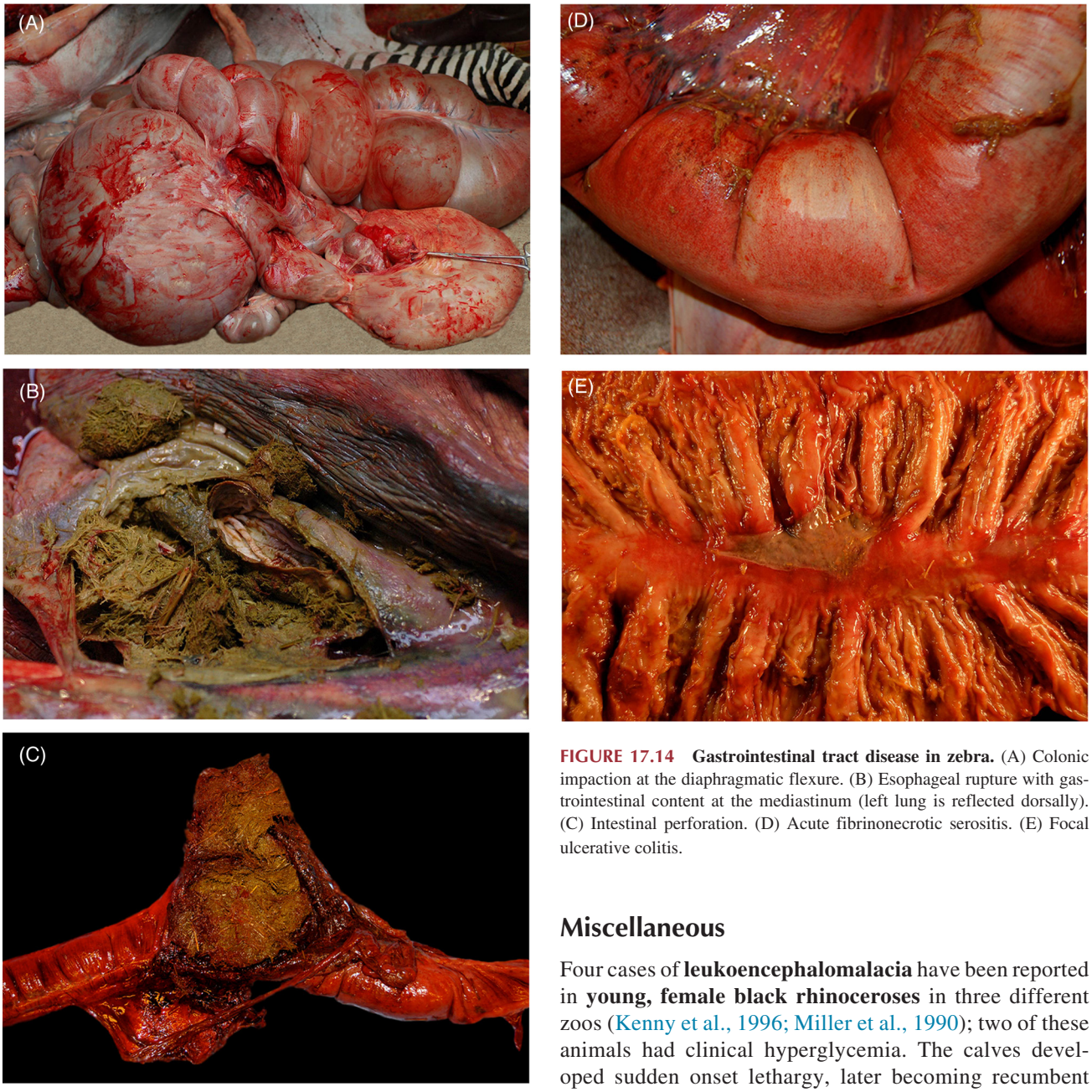

FIGURE 17.14 Gastrointestinal tract disease in zebra. (A) Colonic impaction at the diaphragmatic flexure. (B) Esophageal rupture with gastrointestinal content at the mediastinum (left lung is reflected dorsally). (C) Intestinal perforation. (D) Acute fibrinonecrotic serositis. (E) Focal ulcerative colitis.

lesions have been associated with rectal prolapse. Rectal prolapse was associated with marked edema and submucosal fibrosis in an Indian rhino (Bertelsen et al., 2004).

Corneal cloudiness due to **keratitis** in captive tapirs may be associated with excessive light exposure, trauma, or herpesvirus (Janssen et al., 1996; Janssen, 2003; Ramsay and Zainuddin, 1993; Zimmerman and Hernandez, 2015). 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

Four cases of **leukoencephalomalacia** have been reported in **young, female black rhinoceroses** in three different zoos (Kenny et al., 1996; Miller et al., 1990); two of these animals had clinical hyperglycemia. 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. The fourth case developed acute neurologic signs:

circling, head tilt, head pressing, continuous vocalization, hypermetria, ptyalism, and apparent blindness (Kenny et al., 1996). Computed tomography scans showed areas of reduced density in the cerebrum and thalamus, dilation of the ventricles, 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. The dam in 1 case 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 (Miller and Buss, 2015).

Neoplastic

A variety of neoplasms occur in Perissodactyls, the number and frequency of which may not be completely represented by the published literature. Leiomyoma of the female reproductive tract and cutaneous sarcoids are described below; other reported neoplasms in nondomestic Perissodactyls are listed in Supplemental Materials Table e3.

Leiomyoma, a benign smooth muscle mass, is found with relative frequency in the female reproductive tract of aged zoo species, especially greater Asian one-horned rhino. In a clinical study 100% of greater Asian one-horned rhinoceros over 12 years of age have reproductive tract masses, most likely leiomyomas (Hermes et al., 2014). The number and size of masses increase with age. The masses are particularly at the cervix and vagina, and can be single or multiple. At necropsy leiomyomas are generally well-demarcated, white, and firm. The average gestational length of the white rhinoceros is 16 months and in the wild, a single offspring is produced every 2–3 years, making a wild female pregnant for much of its life with the uterus under the influence of progesterone. Increased progesterone levels have a protective effect against the development of leiomyomas (Wilson et al., 2010; Hermes et al., 2014). An association with acyclicity has been suggested as a predisposing factor in captive rhinos.

Sarcoids are unencapsulated, poorly demarcated fibroblastic skin tumors that invade locally, may recur after surgery, but do not metastasize. Spindle cells within the dermis are arranged in streams and whorls and form well to poorly demarcated masses. The overlying epidermis is frequently hyperplastic with long rete pegs. Equine sarcoid are associated with bovine papilloma virus (BPV) type 1 infection (also see section, Viruses), which has also been reported from sarcoids at the eyelid, nose and inguinal region in captive Burchell's zebras (Löhr et al., 2005). Sarcoids at the ear, prepuce and mammary gland in Somali wild asses are thought to be virus-associated (Pagan et al., 2009)

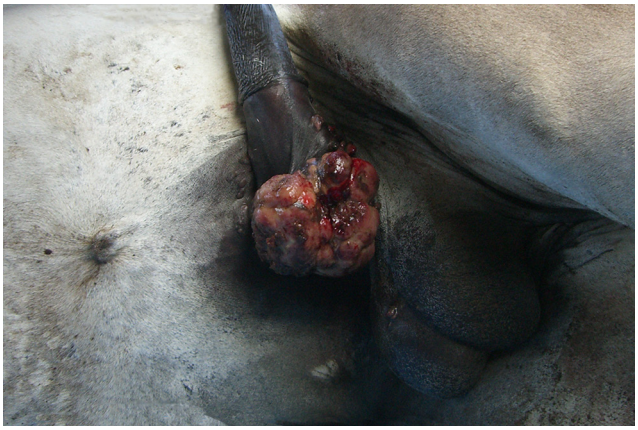

FIGURE 17.15 Preputial sarcoid in a Somali wild ass. These are exophytic, flat to fleshy tumors that may, as in this case, have an ulcerated surface. *(Photo Courtesy of S. Citino, White Oak Conservation)*

(Fig. 17.15). They have also been reported in wild Cape mountain zebra, particularly along the ventral abdomen and legs, in two reserves in South Africa with up to 53% prevalence (Marais et al., 2011).

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 clusters, and is typically drawn from collections and serosurvey data. In addition to descriptions of notable diseases, also see Tables 17.1 and 17.2 and Supplemental Tables e4–e6. OIE 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, glanders (*Burkholderia mallei*), contagious equine metritis (*Taylorella equigenitalis*), equine piroplasmosis, and dourine (*Trypanosoma equiperdum*). Equids are also assumed to be susceptible to Borna virus and vesicular stomatitis virus infections (Walzer, 2003).

Viruses

Perissodactyls are susceptible to **rabies** (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 that was suspected to have been bitten by a feral dog. Approximately one month later, the foal died after experiencing neurological disease, for 2 days. Rabies was confirmed by direct fluorescent-antibody testing (Lankau et al., 2012).

TABLE 17.1 Viral Infections of the Perissodactyls

Family/Species	Viral Pathogen	Comments
Equidae		
Grevy's zebra[a]	Equine herpesvirus 1	Captive, abortion, perinatal foal mortality, virus neutralization, DNA restriction endonuclease analysis
Onager, zebra[b]	Equine herpesvirus 1	Captive, abortion onager (multifocal necrosis spleen and liver), suspected myelitis zebra
Nondomestic equids[c]	African horse sickness	Captive. Epizootic Europe with mortalities following introduction wild-caught zebra. *Equus zebra burchelli,* considered the natural host for the virus, rarely develops signs. *Culicoides* spp. are insect vectors
Tapiridae		
South American and Malayan[d]	Foot and mouth disease virus	Captive, Europe Interdigital lesions
Malayan and mountain[d]	Herpesvirus	Captive, fever, keratitis, erosion to ulceration at mucocutaneous junction 3/6 mortalities
Indian[e]	Equine herpesvirus 1, presumptive	Captive, pregnant female died hemorrhage, and necrosis multiple organs
Rhinoceridae		
Black rhinoceros[e]	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[d]	Poxvirus	Captive, vesicles, and pustules

[a]*Wolff, P.L., Meehan, T., Basgall, E.J., Allen, G.P., Sundberg, J.P., 1986. Abortion and perinatal foal mortality associated with equine herpesvirus type 1 in a herd of Grevy's zebra. J. Am. Vet. Med. Assoc. 189(9), 1185–1186.*
[b]*Montali, R.J., Allen, G.P., Bryans, J.T., Phillips, L. G., Bush, M., 1985. Equine herpes virus type 1 abortion in an onager and suspected herpesvirus myelitis in a zebra. J. Am. Vet. Med. Assoc. 187, 1248–1249.*
[c]*Phillips, L.G., 1999. Infectious diseases of equids. In: Fowler, M.E., Miller, R.E. (Eds.), Zoo and Wild Animal Medicine, Current Therapy, fourth ed., W.B. Saunders Company, Philadelphia, Pennsylvania, pp. 572–574.*
[d]*Ramsay, E. C., Zainuddin, Z. Z., 1993. Infectious diseases of the rhinoceros and tapir. In: Fowler, M.E. (Ed.), Zoo and Wild Animal Medicine, Current Therapy, third ed., W.B. Saunders Company, Philadelphia, Pennsylvania, pp. 459–466.*
[e]*Göltenboth, G., Busch, W., Jenschke, J., Oces, Ä., Wittstatt, U., 1996. Herpesvirus infection in an Indian tapir (Tapirus indicus) and in a black rhinoceros (Diceros bicornis): Case reports. In: Proceedings of the American Association of Zoo Veterinarians, Puerto Vallarta, Mexico, pp. 18–21.*

Fatal **encephalomyocarditis virus (EMCV)** infections have been identified in tapirs. White foci and streaks within the myocardium correspond histologically to areas of cardiomyocyte degeneration and mineralization and interstitial lymphoplasmacytic infiltrates with fewer neutrophils (Fig. 17.16A,B). Mild to moderate congestion of the lungs and hepatic centrilobular vacuolar degeneration and degeneration may also be seen. Infection is confirmed by PCR amplification (Vercammen et al., 2017). Several fatal cases of EMCV have also been described in rhinoceroses (Miller, 2003). While lesions have not been specifically described, viral presence has been confirmed in heart, spleen and other viscera. Virus is excreted in feces and urine of rodents, the natural hosts for the virus. Management of this virus in captive settings should focus on pest control.

Sarcoids (see Neoplastic above) in horses have been associated with **bovine papilloma virus type 1 and 2 infections**. Viral DNA has been detected in the nuclei of the neoplastic mesenchymal cells of zebra sarcoids (Löhr et al., 2005). Late viral antigens are not found in sarcoids and neutralizing antibodies to the virus are not found in horses suggesting the infection is nonproductive in this (probable) nonpermissive host. Mature viral particles are found in the upper keratinizing layers of the epithelial tumor in bovids, and the immune system response results in limited progression of the lesion. The mechanism of transmission of bovine papilloma virus to the 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.

Bacteria

Sporadic ***Mycobacterium bovis*** and ***M. tuberculosis*** (OIE listed reportable) infections have been reported in captive rhinoceros (Miller, 2003; Miller et al., 2016a; Ramsay and Zainuddin, 1993). Most cases have been subclinical and only diagnosed during post mortem examination; advanced cases may present with chronic progressive respiratory disease (Fig. 17.17). Isolated cases of *M. bovis* or *M. tuberculosis* have also been reported in free-ranging rhinos (Espie et al., 2009; Miller et al., 2017). The presence of granulomas

TABLE 17.2 Mycotic Infections of the Perissodactyls

Species/Family	Fungal Pathogen	Comments
Equidae[a]	*Trichophyton*	
Grevy's zebra[a]	Phaeohyphomycosis	Captive, mortality
Tapiridae[b]	*Microsporum gypseum, M. canis, Trichophyton tonsurans*	Dermatophytosis Alopecia in a mountain tapir
South American tapir[b,c]	*Coccidioides immitis*	Wild caught from endemic area. Case report, captive. Pulmonary granulomata in association with amebic meningoencephalitis.
White rhinoceros[b]	*Trichophyton mentagrophytes*	Group housed after recent capture
White rhinoceros[d]	*Malassezia pachydermatis Candida parapsilosis*	Captive. Pruritus
Black rhinoceros[b]	*Aspergillus fumigatus, Absidia corymbifera*	Pulmonary aspergillosis
Black rhinoceros[e]	Aspergillosis most frequent	Survey captive US, *n* = 8, pneumonia at necropsy, often concurrent disease, 2/8 mycobacteriosis.

[a]*Walzer, C., 2003. Equidae. In: Fowler, M.E., Miller, R.E. (Eds.). Zoo and Wild Animal Medicine, fifth ed., Saunders-Elsevier Science, Saint Louis, Missouri, pp. 578–586.*
[b]*Ramsay, E. C., Zainuddin, Z-Z., 1993. Infectious diseases of the rhinoceros and tapir. In: Fowler, M.E. (Ed.) Zoo and Wild Animal Medicine, Current Therapy, third ed., W.B. Saunders Company, Philadelphia, Pennsylvania, pp. 459–466.*
[c]*Lozano-Alarcón, F., Bradley, G.A., Houser, B.S., Visvesvara, G.S., 1997. Primary amebic meningoencephalitis due to Naegleria fowleri in a South American tapir. Vet. Pathol. 34, 239–243.*
[d]*Bauwens, L., De Vroey, C., De Meurichy, W., 1996. A case of exfoliative dermatitis in a white Southern rhinoceros (Ceratotherium simum). J. Zoo Wildl. Med. 27, 271–274.*
[e]*Weber, M., Miller, R.E., 1996. Fungal pneumonia in black rhinoceros (Diceros bicornis). In: Proceedings of the American Association of Zoo Veterinarians, Puerto Vallarta, Mexico, pp. 34–36.*

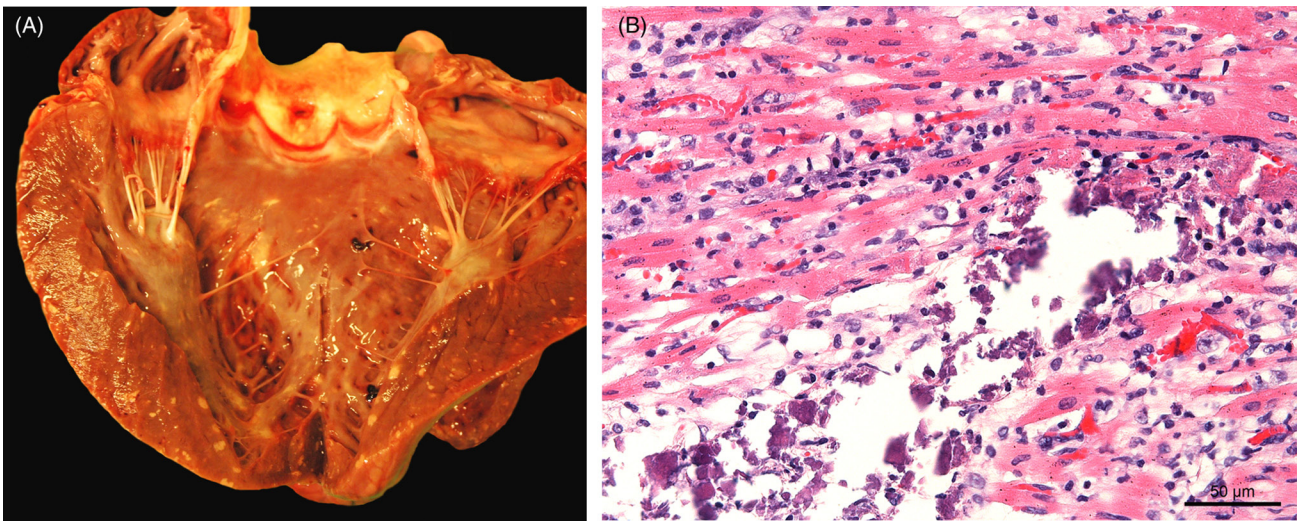

FIGURE 17.16 **Encephalomyocarditis virus infection in the heart of a tapir.** (A) Multifocal white discoloration is present throughout the myocardium. (B) Discoloration corresponds to foci of myocardial degeneration, mineralization, and mild inflammation. *(Reprinted with permission from Vercammen et al., Open Vet J. 7:100-1-3, 2017).*

in the lungs and lymph nodes should raise suspicion for mycobacteriosis. Diagnosis is confirmed by bacterial culture and PCR analysis. ***Mycobacterium orygis*** has also been reported in a free-ranging Indian rhinoceros in Nepal. Granulomas were present in the lungs and molecular methods confirmed the diagnosis (Thapa et al., 2016).

Mycobacterium pinnepedii has been isolated from captive pinnipeds worldwide and in the Southern hemisphere from wild individuals. A captive Malayan tapir housed adjacent to sea lions and transferred between European collections developed respiratory signs and weight loss. Sputum was positive for acid fast bacilli, and necrogranulomas

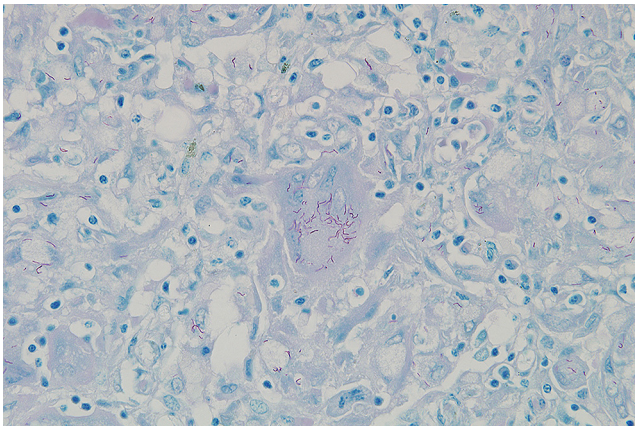

FIGURE 17.17 Pulmonary mycobacteriosis in a black rhinoceros. Histiocytic pneumonia is associated with numerous intracytoplasmic, acid fast positive bacilli in multinucleate giant cells and histiocytes., Ziehl-Neelsen.

were present in the lungs and mesenteric lymph nodes at necropsy. Spoligotyping was positive for *M. pinnepedii*. Three additional tapir that were seropositive and subsequently euthanized had granulomatous lymphadenitis. Bacterial culture and spoligotyping confirmed *M.pinnepedii* in these three animals (Jurczynski et al., 2011). Three lowland tapirs at a Brazilian zoo died with pulmonary granulomas (Murakami et al., 2012). 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.

Paratuberculosis or Johne's disease caused by ***M. avium* subspecies *paratuberculosis*** is an OIE listed reportable, contagious disease typically seen ruminants. Infection causes histiocytic to granulomatous ileitis and enteritis. Clinical signs include progressive weight loss, debilitation, and occasionally death. Johne's disease is not generally seen in Perissodactyls; however, the bacterium was isolated from the feces of a wild-caught black rhino with diarrhea and weight loss (Bryant et al., 2012). Due to the conservation value of the individual, antimycobacterials were administered until fecal shedding was no longer seen.

Anthrax (an OIE listed reportable disease), 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 may increase risk of spore acquisition. Vaccination has been conducted in some parks in Kenya and Namibia. Efficacy has not yet been determined (Janssen and Allen, 2015; Miller and Buss, 2015; Walzer, 2003).

Leptospirosis occurs worldwide, especially in moist environments. Infection is associated with rodent transmission and water contamination. Leptospirosis has been 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 (Jessup et al., 1992). 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 (Douglass and Plue, 1980). In another study, antibodies to 8 serovars of *Leptospira interrogans* were reported in 63 free ranging and 29 captive black rhinoceros. The free-ranging black rhinoceros had low antibody levels and serovars varied with region and ecosystem. Fluorescent antibody testing was positive for *L. interrogans* in the liver of three of four black rhinoceros that died of hemolytic anemia and another rhinoceros that died as a complication of ulcerative skin disease (Jessup et al., 1992). In the absence of hemolytic anemia, *L. interrogans* infection has also been associated with peracute depression, anorexia, hind leg trembling, dysuria, glucosuria, gastrointestinal discomfort and decreased fecal output, and acute tubular necrosis and subacute periportal hepatitis in captive black rhinos (Neiffer et al., 2001), and with abortion in an Indian rhinoceros (Miller and Foose, 1996).

Salmonellosis is a common cause of septicemia and enteritis in rhinoceros and tapir (Ramsay and Zainuddin, 1993), and of abortion and genital tract infection of equids (Walzer, 2003). 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 at an American zoo that died with gastroenteritis and sepsis (Kenny et al., 1997). Other reported signs of salmonellosis include lethargy, partial anorexia, epistaxis, ulcerative skin lesions and/or diarrhea in a captive female and two male rhinos; Tokay geckos released into the building were the suspected source of infection. Gross lesions in these rhinos included fibrinous pleuritis, a massive 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 (Kenny, 1999), 11% of fecal samples were culture positive for *Salmonella* spp. Some animals in this survey 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 a period of nearly 3 years by fecal culture and PCR identified periodic shedding in four of six rhinoceros during that time period (Miller et al., 2008). Recently four clinical cases of salmonellosis in black rhino calves were reported. Two recovered from self-limiting diarrhea with treatment, but two calves died from associated

sepsis thought to have been related to low-level transfer of maternal immunity and primiparity (Love et al., 2017).

Other notable bacterial diseases in Perissodactyls include **Clostridial infections**, **tetanus** due to ***C. tetani*** infection of a hoof abscess in an onager, and ***C. welchii*** with wound infection and septicemia in Somali wild ass (Pagan et al., 2009). Additionally, three white rhinoceros died unexpectedly and were presumed to have enterotoxemia from *C. perfringens*; the main findings were mild abdominal exudate, splenic and multifocal gastrointestinal congestion, and adrenal hemorrhage (Citino et al., 2017). **Strangles**, suppurative lymphadenitis with ***Streptococcus equi***, particularly in association with the pharynx and respiratory tract, was found in a cluster of deaths in Przewalski horses reintroduced into Southwestern Mongolia. Association with a harsh winter and wolf predation were suspected as predisposing factors (Robert et al., 2005). **Bacterial vegetative valvular endocarditis** was reported in a captive, aged, wild-caught male white rhinoceros, that developed immobility and related decubital ulcers. There was roughening at the left atrioventricular valve and ventricular endocardium. Histologically, myocardial degeneration was evident with the vegetative endocarditis. Fibrosis and degeneration in the liver and chronic nephritis were seen. ***Streptococcus dysgalactiae* subsp. *equisimilis*** and ***S. ovis*** were isolated from multiple sites. The decubitus ulcers were the suspected portal of bacterial infection (Houszka et al., 2014).

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 (Dillehay and Silberman, 1991). 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, US (Terio et al., 2003). Granulomatous lesions were often disseminated, but the respiratory system was most often affected (Fig. 17.18A,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 (Terio et al., 2003). 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 (Wallace et al., 2009).

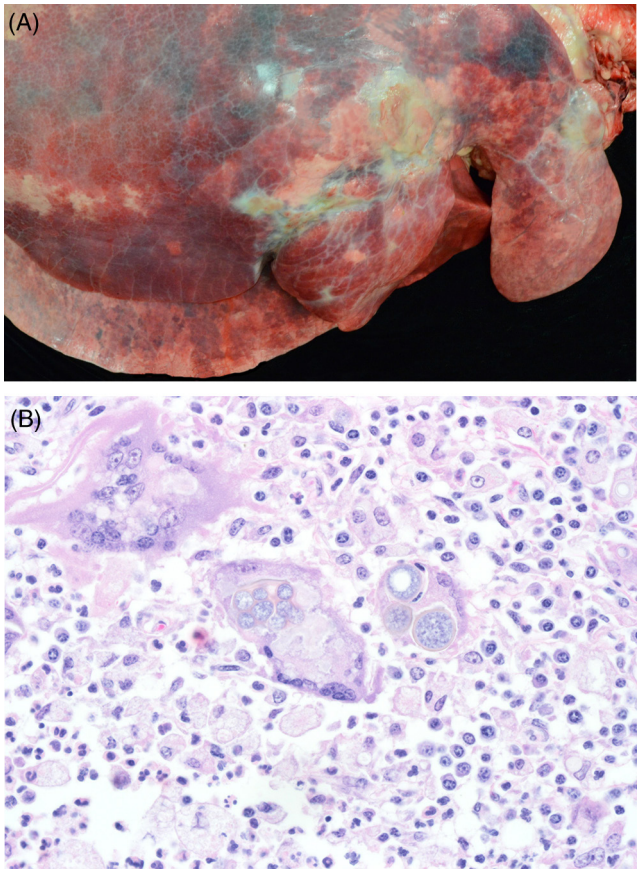

FIGURE 17.18 Coccidioides immitis infection in the lung of a Przewalski's horse. (A) Multiple yellow white pyogranulomata are present in the parenchyma. (B) *Coccidioides immitis* associated with multinucleate giant cells, plasma cells, and neutrophils in a section of Przewalski's horse lung. *(Photos Courtesy of Disease Investigations, San Diego Zoo Global)*

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. may be normal flora of rhinoceros skin; they have been cultured from the skin of unaffected individuals (Bauwens et al., 1996).

Metazoa

Stephanofilaria dinniki has been reported in free ranging black and white rhinos with crusty, dry raised areas of dermatitis. Histologically, serocellular crusts with neutrophils, bacteria and debris, ulcerative dermatitis with epidermal adult filarial nematodes, intraepidermal necrosis, and dermal granulomas and lymphohistiocytic nodules were present. Acanthosis and rete ridges were present at the margins (Kock et al., 1990). 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 (Munson and Miller, 1999). Crusts fall off to reveal red areas and leave depigmented scars. White rhinos have larger lesions averaging 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 lifecycle of *S. dinniki* is unknown but a bloodsucking arthropod is required to complete the cycle. Poor body condition and heavy rainfall are thought to lead to recurrence of the lesions. Red-billed oxpeckers (*Buphagus erythrorynchus*) may aid in parasite spread and cause ulceration (Mutinda et al., 2012).

Halicephalobus gingivalis has been identified in a captive female Grevy's zebra that appeared blind and demonstrated abnormal behavior (Isaza et al., 2000). Bilateral uveitis and associated normocytic hyperchromic anemia, 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 along with marked granulomatous inflammation of the ciliary body and choroid with synechiation were present. Larvae with adults were associated with the inflammatory infiltrates. Only female adults have been identified in infections suggesting parthenogenic reproduction.

Schistosomiasis was found at necropsy of a juvenile captive Brazilian tapir with a history of occasional bloody diarrhea. Multiple granulomas were present in the liver especially periportally with eosinophils and multinucleate giant cells 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 (Yamini and van Veen, 1988).

Protozoa

Piroplasmosis is an infectious, tick-borne disease caused by one of two parasites, *Babesia caballus* or *Theileria equi*, which attack and destroy red blood cells. Infection may occur seasonally with the vector tick lifecycle. Translocation may induce disease by spreading pathogens and modulates the effects of stress hormones on the immune system allowing proliferation of piroplasms in the host.

In equids, severe regenerative hemolytic anemia and jaundice are seen in acute cases, while chronic cases may be subclinical carriers. Gross findings include pulmonary edema and splenic congestion, and 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 horses reintroduced to Southwestern Mongolia, piroplasmosis was implicated as the cause of death of stallions and a stillborn foal (Robert et al., 2005). A wild-caught Somali wild ass stallion was diagnosed with babesiosis (Pagan et al., 2009). In cograzing donkeys and wild Grevy's zebra in Kenya, all zebra tested positive for *T. equi* (Hawkins et al., 2015).

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 Kenya, *Babesia bicornis* has not been detected while *Theileria bicornis* was found to have 49% prevalence in white rhinoceros and 43% in black rhinoceros. As previously stated, these infections have an increased importance because of conservation efforts related to animal relocations (Otiende et al., 2015).

Sarcocystis neurona has been isolated from domestic horses (*Equus caballus*) with equine protozoal myeloencephalitis. The opossum *Didelphis virginiana* is the definitive host of *Sarcocystis falcatula* and may be the definitive host of *S. neurona.* A captive Grant's zebra 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. *Sarcocystis neurona* was identified by immunohistochemical staining of merozoites (Marsh et al., 2000).

Klossiella equi is a coccidian 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 (male) and macrogametocytes (female). When pronuclei of micro and macrogametocytes fuse a zygotye/sporont develops, sporogony. Sporozoites develop in the sporocysts, sporocysts are released into the lumen and pass out in the urine. The cycle is completed when sporocysts are ingested, sporozoites are released into the digestive tract, penetrate the wall, enter the blood and travel to the glomeruli. The coccidian has been an incidental finding in sections of kidney from Hartmann's mountain zebra (Suedmeyer et al., 2006) and Przewalski's horses (Robert et al., 2005).

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* (Sommanustweechai et al., 2010). The parasite may be transmitted horizontally or vertically. Source of infection was not identified, but was likely ingestion of oocysts. Stillbirth due to neosporosis in a white rhinoceros has been documented in South Africa. Abortion is the most common sign of infection in the intermediate host. Definitive hosts (dogs) most commonly have paresis and paralysis of the hindlimbs.

Necrosuppurative meningoencephalitis due to infection with ***Naegleria fowleri*** has been reported in 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 (Lozano-Alarcón et al., 1997).

E-SLIDES

17.e1 **Iron overload, black rhinoceros, liver and lung.** Kupffer cells in sinusoids, concentrated periportally, contain brown pigment consistent with hemosiderin (confirmed Perl's iron staining). Lesser pigment granules in hepatocytes (hemosiderin by Perl's iron stain). 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. (see Figs. 17.7, 17.8 and 17.e2). eSlide: VM05018

17.e2 **Iron overload, black rhinoceros, liver and lung, Perl's iron stain.** 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. Further hemosiderin-laden macrophages are seen, especially in capillaries in the lung. Perl's. (see Figs. 17.7, 17.8 and 17.e1). eSlide: VM05019

17.e3 **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. (see Fig. 17.17 and 17.e4). eSlide: VM05021

17.e4 **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. Ziehl-Neelsen. (see Fig. 17.17 and 17.e3). eSlide: VM05020

17.e5 **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. (see Fig. 17.15). eSlide: VM05022

17.e6 **Sarcoid, Grevy's zebra, haired skin.** Multifocal 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. (see Fig. 17.15). eSlide: VM05023

17.e7 **Sarcoid, Grevy's zebra, haired skin.** 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. (see Fig. 17.15). eSlide: VM05024

17.e8 **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

E–ONLY CONTENTS

TABLE e1 Taxonomy of the Perissodactyls[a,b,c,d,e]

Suborder: Hippomorpha	
Family: Equidae	
Equus ferus przewalskii	Przewalski/Mongolian wild horse
Equus africanus	Somali and Nubian wild asses
Equus hemionus	Asiatic wild ass, kulan, onager
Equus kiang	Eastern, Western, and Southern kiangs
Equus zebra zebra	Cape mountain zebra
Equus zebra hartmannae	Hartmann zebra
Equus grevyi	Grevy's zebra
Equus quagga burchelli	Plains/Burchell/Grant's zebra
Suborder: Ceratomorpha	
Family: Tapiridae	
Tapirus bairdii	Baird's/Central American tapir
Tapirus indicus	Malayan/Asian tapir
Tapirus pinchaque	Mountain/woolly tapir
Tapirus terrestris	Brazilian/Lowland/ South American tapir
Tapirus kabomani	Kabomani/little black tapir
Family: Rhinoceridae	
Ceratotherium simum	White rhinoceros
Diceros bicornis	Black rhinoceros
Dicerorhinus sumatrensis	Sumatran/Asian two-horned rhinoceros
Rhinoceros unicornis	Indian/Asian one-horned rhinoceros
Rhinoceros sondaicus	Javan rhinoceros

[a]Nowak, R.M., 1999. Walker's Mammals of the World, sixth ed., The Johns Hopkins University Press, Baltimore and London.
[b]Janssen, D.L., Allen, J.L., 2015. Equidae. In: Miller, R.E., Fowler, M.E. (Eds.). Fowler's Zoo and Wild Animal Medicine, Vol. 8, Elsevier, Saint Louis, Missouri, pp. 559–567.
[c]Zimmerman, D.M., Hernandez, S., 2015. Tapirdae. In: Miller, R.E., Fowler, M.E. (Eds.). Fowler's Zoo and Wild Animal Medicine, Vol. 8, Elsevier, Saint Louis, Missouri, pp. 547–559.
[d]Cozzuol, M.A., Clozato, C.L., Holanda, E.C., Rodrigues, F.H.G., Nienow, S., de Thoisy, B., Redondo, R.A.F., Santos, F.R., 2013. A new species of tapir from the Amazon. J. Mammol. 94, 1331–1345.
[e]Miller, M.A., Buss, P.E., 2015. Rhinoceridae (Rhinoceroses). In: Miller, R.E., Fowler, M.E. (Eds.). Fowler's Zoo and Wild Animal Medicine, Vol. 8, Elsevier, Saint Louis, Missouri, pp. 538–547.

TABLE e2 Comparative Dentition of the Perissodactyls

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

C, Canine; I, incisor; M, molar; P, premolar.
[a]Janssen, D.L., Allen, J.L., 2015. Equidae. In: Miller, R.E., Fowler, M.E. (Eds.). Fowler's Zoo and Wild Animal Medicine, Vol. 8, Elsevier, Saint Louis, Missouri, pp. 559–567.
[b]Da Silva, M-A.O., Kortegaard, H.E., Choong, S.S., Arnbjerg, J., Bertelsen, M.F., 2011. Resorptive tooth root lesions in the Malayan tapir (*Tapirus indicus*). J. Zoo Wildl. Med. 42, 40–43.
[c]Tjornelund, K.B., Jonsson, L.M., Kortegaard, H., Arnbjerg, J., Nielsen, S.S., Bertelsen, M.F., 2015. Dental lesions in the lowland tapir (*Tapirus terrestris*). J. Zoo Wildl. Med. 46, 363–366.

TABLE e3 An Overview of Reported Neoplasms in Perissodactyls

Family/Species	Tumor	Age	Sex	Clinical Notes	Location	Notes
Equidae						
Somali wild ass[a] Burchell's zebra[b] Cape mountain zebra[c]	Sarcoid			Seen in captive and wild setting	Skin, variation in distribution with species	Possible association with bovine papilloma virus 1; fibroblastic, local invasion, possible recurrence, no metastasis
Onager[d]	Pituitary gland adenoma, pars intermedia	13 years (1) 22 years (2)	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
Tapiridae						
Baird's tapir[e]	Embryonal rhabdomyosarcoma	22 months	F	Nasal discharge, coughing, seizure-like behavior (possibly associated with hypoxia)	Nasopharynx	Cells are negative for smooth muscle actin, and multifocally positive for desmin and myoglobin
Malayan tapir[f]	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
Rhinoceridae						
Black rhinoceros[g]	Seminoma		M	Identified by ultrasonography, hemicastration	Testicle	
Black rhinoceros[h]	Acute lymphoblastic leukemia	21 months	F	Sudden onset acute dyspnea and lymphadenomegaly	Blood	Heterogeneous lymphoblastic cells
Black rhinoceros[i]	Melanoma	10 years	M	Diagnosed at biopsy	Skin	Multiple, benign, well-differentiated
Asian one-horned rhinoceros[i]	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. Digital melanoma domestic animals poor prognosis. Humans and horses trauma nail/hoof leads to development of melanocytic neoplasms.
White rhinoceros[j]	Vaginal hemangioma		F	Episodic vaginal hemorrhage, more frequent over 10 years	Vagina	Biopsy
White rhinoceros[k]	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[l]	Osteochondroma	2 years	M	Progressive lameness	Distal third metacarpus	Surgically removed. Benign, originated adjacent to the physis/subarticular, at site of endochondral ossification.

(*Continued*)

TABLE e3 An Overview of Reported Neoplasms in Perissodactyls (*Cont.*)

Family/Species	Tumor	Age	Sex	Clinical Notes	Location	Notes
White rhinoceros[m]	Pheochromo-cytoma	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[n]	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 Asian rhino at horn base and white rhino at foot and flank.
White rhinoceros[o]	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.

[a]Pagan, O., Von Houwald, F., Wenker, C., Steck, B.L., 2009. Husbandry and breeding of Somali wild ass *Equus africanus somalicus* at Basel Zoo, Switzerland. In: Fisken, F.A. (Ed.) International Zoo Year Book. The Zoological Society of London, London, vol. 43, pp. 198–211.
[b]Lőhr, C.V., Juan-Sallés, C., Rosas-Rosas, A., Parás Garciá, A., Garner, M.M., Teifke, J.P., 2005. Sarcoids in captive zebras (*Equus burchelli*) colon association with bovine papilloma virus type 1 infection. J. Zoo Wildl. Med. 36, 74–81.
[c]Marais, H.J., Page, P.C. 2011. Treatment of equine sarcoid in seven Cape mountain zebra (*Equus zebra zebra*). J. Wildl. Dis. 47, 917–924.
[d]Peel, A.J., Bouts, T., Flach, E., Rivers, S., Routh, A. 2009. Pituitary pars intermedia dysfunction (equine Cushing's disease) in an onager (*Equus hemionus onager*). J. Zoo Wildl. Med. 40, 773–780.
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TABLE e4 Diseases with Serologic Evidence in Perissodactyls

Family/species	Pathogen	Geographic Location	Comments
Equidae			
Grevy's zebra[a]	Equine herpesvirus-9	Captive	Asymptomatic. Progressive encephalitis in a polar bear.
Przewalski'shorse[a]	Equine herpesvirus-2, 5	Captive	No clinical signs.
Zebra (*Equus burchelli*)[b]	Equine herpesvirus-1, 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[c]	Equine herpesvirus-1	Captive, Europe	
Tapiridae			
Baird's *n* = 17[d]	*Leptospira bratislava* 5/17 seropositive, high titers Venezuelan equine encephalitis 12/17	Free-ranging, Costa Rica	No serological reaction for *Brucella abortus*, Equine herpesvirus
Baird's *n* = 23[e]	21% seropositive Venezuelan encephalitis H1 virus 47% vesicular stomatitis, 13% West Nile virus, antibodies to equine herpesviruses-1, 2, 4, equine influenza and equine rhinoviruses type 1 and 2.	Captive, Panama	
Brazilian *n* = 10[f]	*Toxoplasma gondii* 1/10 seropositive	Free-ranging, Brazil	No serological reaction noted for *Leptospira*sp., *Brucella abortus*, equine infectious anemia
South American *n* = 65[g]	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.
Rhinoceridae			
Black and white rhinoceros *n* = 281[h]	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
Black and white rhinoceros *n* = 65 black, *n* = 58 white[i]	*Cowdria 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.

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TABLE e5 Endoparasitic Diseases of the Perissodactyls

Type of Endoparasite	Parasite Species	Species Identified in	Lesion
Equidae			
Nematode[a,b]	*Strongylus, Strongyloides, Parascaris, Oxyuris, Dictyocaulus* (zebra)		*P. equorum* possible increased pathogenicity zebra foals
	Halicephalobus gingivalis	Grevy's zebra[c]	Captive, disseminated granulomatous inflammation
Cestodes[b]	*Anoplocephala* spp.		
Bot/gadfly	*Gasterophilus*		
Protozoa[b]	*Babesia caballi, Theileria equi*	Przewalski's horse	
	Sarcocystis spp., *Trypanosoma* spp., *Theileria equi, Toxoplasma gondii, Besnoitia bennetti*		
Tapiridae			
Nematode[d]	*Strongyloides* sp., Ascarids, capillarids, hookworms		
Cestode[e]	*Paranoplocephala* sp.	Captive mountain tapir	Incidental
Trematode[e,f]	*Fasciola hepatica*	South American tapir	Death in captivity, cirrhosis
	Schistosomiasis		Granulomata liver, hemorrhagic gastroenteritis, associated with embryonated eggs
Pentastome[e]	*Armillifer moniliformis*	Wild-caught Malayan tapir	Nymphal cysts
Protozoa[e,g]	*Balantidium* sp., *Giardia* sp.		Watery diarrhea (possibly normal flora)
	Babesia sp.	Malayan tapir	
Malayan[g]	*Babesia equi*, suspect		Icterus and anemia, recent import
Baird's[g]	*Cryptosporidium suis*	Shanghai zoo	watery diarrhea
South American[h]	*Theileria equi*		Tick vector, intraerythrocytic
South American[i]	*Naegleria fowleri*		Meningoencephalitis, ameboid trophozoites identified by specific mononclonal antibodies
Rhinoceridae			
Nematode[e,j]	*Parabronema* sp. and *Dicronema versterae*	Rhinoceros	Stomach, possible tumor-like lesions
	Habronema khalili, Draschia megastoma (spirurid)	Black rhinoceros	Intestine
	Oxyuris sp. *(O. karamoja)*		Large intestine
	Probstmayria vivipara (ascarid)	Greater one-horned and white rhinoceros	
	Strongylus tremletti	Black rhinoceros	Intestine
	Strongyloides sp., *Crossocephalus* sp.	Sumatran rhinoceros	
	Grammocephalus clathrotus	African rhinoceros	Biliary system
	G. intermedius (hookworm)	Black rhinoceros	Large intestine
	Thelazid worms	Black rhinoceros	Conjunctival sacs

TABLE e5 Endoparasitic Diseases of the Perissodactyls (*Cont.*)

Type of Endoparasite	Parasite Species	Species Identified in	Lesion
	Anoplocephala sp.		Incidental
	A. gigantea	Javan rhinoceros	
	A. vulgaris	One-horned and black rhinoceros	Oribatid mite suspected cestode intermediate.
Cestode[e,j]	*Fascioloides gigantica*	Asian one-horned rhinoceros	Death
Trematode[e,j]	*Brumptia bicanda*	African rhinoceros	Intestinal tract
	Babesiosis, trypanosomiasis, theileriosis	Rhinocerus	Mortalities associated with translocations
Protozoa[e,j,k,l,m,n]	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	White rhinoceros calf	
	Gasterophilus (bot)		Bots generally esophageal region of stomach
Arthropod, larvae/bot[e,k]	*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.
	Gastrophilus sumatrensis	Sumatran rhinoceros	

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TABLE e6 Ectoparasites of Perissodactyls

Family/Species	Parasite	Findings
Equidae		
Mites[a]	*Sarcoptes equi, Psoroptes equi, Chorioptes equi, Demodex equi, D. caballi*	
Ticks[a]	*Dermacentor nutalli*	Vector disease
Tapiridae		
Mites[b]	*Sarcoptes tapiri*	Pruritus, exudative dermatitis, hyperkeratosis. Progression to blindness, malnutrition and death described.
Ticks[b]	*Amblyomma testudinarium*	Tapir Indonesia
Blood-sucking moth[b]	*Calpe eustrigata*	Only observed feeding on captive tapir and Sumatran rhino in Malaysia.
Rhinoceridae		
Nematodes[b,c,d]	*Stephanofilaria dinniki*	Crusty skin disease, free-living, Africa, nematode. Absence of disease in captivity thought to require an insect vector.
	Habronema sp.	Possible larvae cause periocular lesions.
Ticks[b]	*Amblyomma hebraeum, A. sparsum*	White and black rhinoceros, tick is vector for *Cowdria ruminantium*, heartwater
	A. rhinoceros, A. gemme, A. marmorem	Black rhinoceros
	A. testudinarium	Rhinoceros Indonesia
	A. crenatum	Javan rhinoceros
	A. infestum, Hyalomma walkeriaeirii, Aponomma sp., *Haemaphysalis* sp.	Sumatran rhinoceros in Malaysia
	Rhipicephalus ayrei, R. superitus, Cosmiomma hippopotamensis, Dermacentor sp., *Haemaphysalis* sp., *Hyalomma* sp.	Black rhinoceros Kenya Ticks locate especially in folds, greatest problem as disease vectors.
	D. rhinocerinus	White rhinoceros on importation
Biting flies[b,d]	*Lyperosia* sp., *Rhinomusca sp., Tabanus sp., Glossina* sp.	*Glossina sp.* are the vector for trypanosomiasis
	Chrysomyia bezziana	Old World screw worm

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