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Chapter 12

Procyonidae, Viverridae, Hyenidae, Herpestidae, Eupleridae, and Prionodontidae

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INTRODUCTION

This chapter covers the diseases and pathology of multiple taxonomic groups within the order Carnivora including *Procyonidae* and several of the Feliformia carnivores. There is also considerable overlap in disease susceptibility between these groups and with the *Felidae* and *Canidae* (See Chapters 9 and 10). Feliformia carnivores include the *Hyenidae* (spotted, striped, and brown hyenas as well as aardwolf), *Viverridae* (civets, binturongs, genets, and linsang), *Prionodontidae* (a monogeneric family of linsangs), *Eupleridae* (which includes fossa, civets, mongoose, and falanouc), and *Herpestidae* (which includes mongoose and meerkats) (Zhou et al., 2017). A notable feature of the feliform carnivores is a double chambered auditory bulla composed of two bones joined by a septum (Ewer, 1973). Many of these species are listed as vulnerable or endangered (see Supplemental Materials Table e1) due to habitat loss and hunting.

The procyonids include the raccoons, kinkajous, coatis, ringtail cat, and olingos. These groups are characterized by masked faces and banded tails and are native to the western hemisphere. With the exception of the near threatened olinguito, procyonids exist in stable numbers. Raccoons have adapted to life within urban and suburban environments and population numbers in some regions are increasing (Frantz et al., 2005).

Hyaenidae are largely restricted to Africa, the Arabian Peninsula, and Asia as far east as India. Although they have a similar quadrupedal, digitigrade form to members of the suborder Caniformia in the family *Canidae*, modern taxonomic revisions place *Hyaenidae* in the suborder Feliformia. Hyenas have a powerful build with somewhat long forelimbs; this disparity in limb length is most pronounced in the brown hyena. Hyena fur is coarse and lacks an undercoat, unlike most of the canids.

The majority of knowledge about disease pathogenesis for these species has been biased toward studies of raccoons in part due to overlapping habitat and disease susceptibility with domestic dogs and cats (Klinkowski-Clark and Kutilek, 2010). For the feliform groups in this chapter, susceptibility to and potential reservoir status of several species for diseases, such as avian influenza virus and coronaviruses (e.g., SARS) have recently received renewed scientific interest (Wicker et al., 2017). Several species are commonly displayed in zoological facilities.

UNIQUE FEATURES

In general, normal gross and histologic features of the species covered in this chapter are similar to those of domestic carnivores. Dental formulas are similar and generally I 3/3, C 1/1 with more variation in the PM 3-4/3-4 and M 1-2/1-3 based on species and dietary lifestyle. The jaws and teeth of most hyenas are more robust and powerful than those of the typical carnivores. The spotted hyena, in particular, is known for the power of its jaws relative to its body size. Insectivorous species, such as the aardwolf have smaller teeth.

A notable unique feature of procyonids in general, and of raccoons specifically, is their manual dexterity in handling and manipulating items with their forepaws. In fact, the word raccoon originates from the Algonquian word ärähkun or ärähkuněm, which means "he scratches with his hands" (Collins English Dictionary, 2016). While they do

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not have true opposable first digits like primates, the pattern of innervation to their forepaws is similar to that of primates (Turnbull and Rasmusson, 1986). Procyonids do not have a cecum; ceca are rudimentary in some of the herpestids and present in viverrids (Ramsay, 2015).

Kinkajous and binturongs are the only carnivores to possess prehensile tails (Youlatos, 2003). While most carnivores utilize scent marking for territorial and reproductive communication, some viverrids have unique scent characteristics. As an example, to some, binturongs smell like buttered popcorn. This specific odor has been attributed to a volatile compound in the urine (Greene et al., 2016). The use of anal-sac gland secretion from civets for musk scent in perfume is well documented, but it is also a welfare concern due to varying husbandry standards (Anonis, 1997; Klok and Nugteren, 1973; Tolosa and Regassa, 2007).

Female spotted hyenas have unique, often confusing reproductive anatomy. They lack an external vagina because the labia are fused during development to form a pseudoscrotum. The clitoris is greatly enlarged, is erectile, and has a central urogenital canal. The internal portions of the reproductive tract are similar to other carnivores except that spotted hyenas lack a well-developed cervix. For most of the species in this chapter that have been studied, placentation is zonary and endotheliochorial. Haemophagous organs may occur early in gestation in some species but regress during pregnancy. The placenta of the hyena is unusual in that it is hemochorial (Enders et al., 2006). In males, a baculum (os penis) is vestigial or missing in all hyenas.

Numerous histopathological findings of undetermined significance are commonly seen in postmortem examination of raccoons. Because of their role as rabies reservoirs (discussed below), the nervous system of raccoons is frequently examined. Common postmortem findings include vascular mineralization; psammoma bodies in the cerebrum, meninges, and choroid plexus; and neuroaxonal degeneration in the brainstem (see section Miscellaneous). Myocardial inclusions without associated inflammation are a common histological finding in captive raccoons; there is no known effect on cardiac function (Hamir, 2011b; Hamir et al., 2007). These inclusions can be basophilic, periodic acid-Schiff (PAS) positive, and diastase resistant or eosinophilic. They are round on transverse section and cigar-shaped longitudinally, and are rarely associated with areas of fibrosis. Bridging portal hepatic fibrosis of undetermined etiology (Fig. 12.1) is a relatively common incidental histologic finding in aged raccoons.

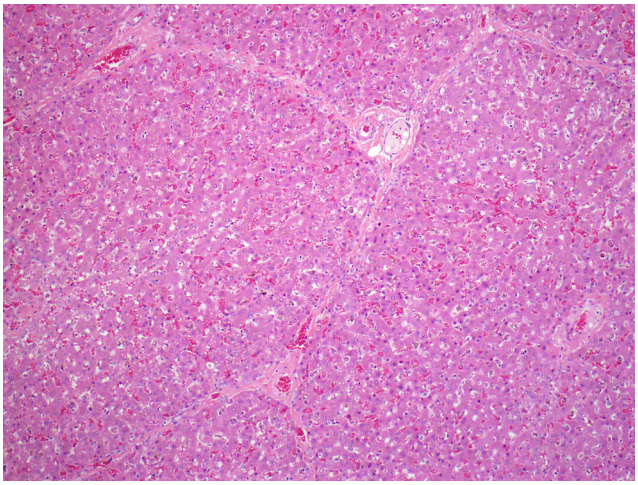

FIGURE 12.1 Hepatic portal fibrosis in a free-ranging raccoon. Thin strands of mature collagen span adjacent portal areas. This is an incidental finding that appears to increase in prevalence with age in free-ranging raccoons.

NON-INFECTIOUS DISEASES

Nutritional

The species in these groups are carnivorous but eat a wide variety of items from whole vertebrate prey, to invertebrates and eggs. Because the nutritional composition of many of the dietary items of wild populations are unknown, providing a variety of food items in managed care settings is critical for ensuring a balanced diet. **Taurine** should be a component of diets and may be a contributing factor in dilated cardiomyopathies noted in meerkats and possibly mongooses. **Obesity** is not an uncommon finding in urban and suburban raccoons that have ample access to anthropogenic food sources.

As can occur in domestic carnivores, **pancreatitis** has been reported in captive civets and meerkats (Laura et al., 2014; Naples et al., 2010). Lesions are similar to those in domestic species and include interlobular and peripancreatic neutrophilic inflammation and pancreatic and peripancreatic fat necrosis. Obesity, high fat diets, hyperlipidemia, and dietary indiscretion are considered risk factors for the development of pancreatitis in these species.

Metabolic

Metabolic disease has been reported infrequently in this group. Pancreatic islet amyloidosis is a common postmortem finding in both free-ranging and captive raccoons (Fig. 12.2). An association between this finding and diabetes, as occurs in some other species, has not been established in raccoons (Anderson and Cullor, 1982; Hamir et al., 2007). Amyloid deposition is characteristic, with abundant, amorphous, homogeneous eosinophilic material in the islets of Langerhans that exhibit birefringence in Congo-red stained sections examined under polarized light.

Toxic

Nontarget exposure to **rodenticides** are the most commonly encountered toxins in this group of carnivores, though in some cases, intentional poisoning may occur if they are perceived as pests. Both anticoagulant toxins and those that

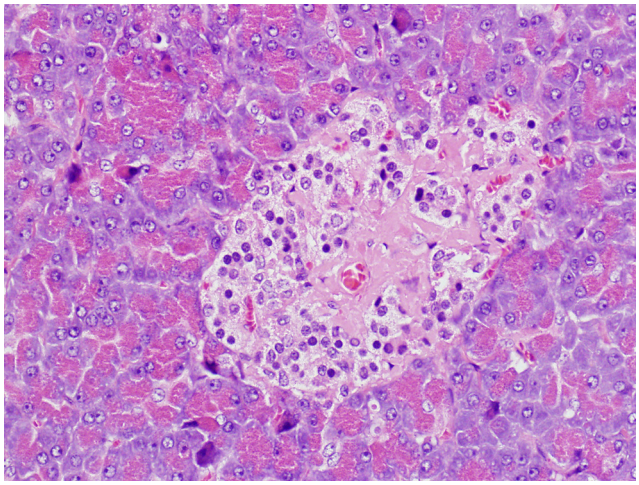

FIGURE 12.2 Pancreatic islet amyloidosis in a raccoon. In some raccoons, pancreatic islets contain small to moderate amounts of acellular, amorphous, eosinophilic fibrillar material consistent with amyloid. There is no evidence that this accumulation is associated abnormal hormone production or other physiologic or metabolic diseases.

uncouple oxidative phosphorylation are often manufactured into green-blue pellets or cakes that may be identified in the gastric or intestinal contents of poisoned individuals. Anticoagulant rodenticides include first generation (e.g., warfarin, diphacinone, chlorophacinone, and coumachlor) and second-generation (e.g., brodifacoum, bromodiolone, and difethialone) compounds. These rodenticides inhibit vitamin K reactivation, thus preventing production of vitamin K-dependent clotting factors. In one long-term, unpublished survey of free-ranging raccoons in Northern California, up to 80% of raccoons tested from a rehabilitation center over 10 years were positive for rodenticides (personal communication, M. Piazza and K. Kacmarcik). Other studies in raccoons (Daniels, 2013; Stone et al., 1999) and other free-ranging wildlife species (Gabriel et al., 2012; Rattner et al., 2014) also found frequent rodenticide exposure and indicating an important risk. Gross and histologic lesions include hemorrhage in multiple anatomic sites including the subcutis, brain, pericardial, thoracic, and abdominal cavities, and within the gastrointestinal tract. Definitive diagnosis is made detecting anticoagulant rodenticides by high-performance liquid chromatography, mass spectrometry (HPLC-MS/MS) in liver, serum samples, whole blood, or gastrointestinal contents.

Rodenticides that uncouple oxidative phosphorylation, for example, bromethalin, frequently do not cause gross lesions. Death occurs secondary to an influx of fluid into the central nervous system (CNS) with an associated increase in intracranial pressure and subsequent compression of cardiovascular and respiratory centers in the brainstem. Histologically, there is marked vacuolar degeneration of the white matter that corresponds to splitting of myelin fibers by edema (Bautista et al., 2013). There is also marked astroglial and oligodendrogial gliosis. Definitive diagnosis is made by detection of desmethylbromethalin (a metabolite of bromethalin) by HPLC-MS/MS in brain, fat, liver, or gastrointestinal contents.

Elevated tissue mercury levels have been reported in Egyptian and Javan mongooses but there was no clinical or histologic evidence of toxicity (Horai et al., 2008; Millán et al., 2008). It has been suggested that mongooses have elevated thioredoxin reductase activity, an intrinsic detoxification pathway.

Ingestion of pennies resulting in **zinc toxicosis** has been described in a captive striped hyena (Agnew et al., 1999). The animal had severe, nonregenerative anemia, jaundice, and gall bladder edema. Multifocal thrombosis was evident microscopically and associated with multifocal necrosis. The liver demonstrated centrilobular hemosiderosis, cholestasis, Kupffer cell hyperplasia, and single-cell necrosis. Multifocal tubular necrosis in the liver was associated with intraluminal casts and lymphoplasmacytic, tubulointerstitial nephritis.

Mongooses, whose diet can include snakes, have developed complex mechanisms to avoid the toxic effects of **snake venom**. Examples include toxin-neutralizing proteins in the serum that inhibit venom metalloproteinases and myotoxins, as well as genetic adaptation of the nicotinic acetylcholine receptor that prohibit binding of α-neurotoxins (Voss and Jansa, 2012).

Congenital/Genetic

A case of lysosomal storage disease, **sphingomyelin lipidosis** (**Niemann-Pick Disease**), was described in a raccoon based on histological and electron microscopic findings. These included vacuolated neurons and glial cells in the CNS as well as macrophages in numerous different organs (liver, tongue, trachea, tonsils, spleen, lymph nodes, small and large intestines, and lungs). Cytoplasmic vacuoles did not stain with Sudan black, oil red O, or PAS, and ultrastructurally, they were composed of concentrically laminated, myelin-like figures with a regular periodicity (Vapniarsky et al., 2013). An **uncharacterized glycogen storage disease** has been reported in a captive coati with progressive neurologic disease. Histologically, neurons, hepatocytes, and macrophages have distended cytoplasm containing finely granular material that ultrastructurally is composed of membranous structures within lysosomes (Chu and Loynachan, 2013).

Albinism occurs in raccoons and is linked to two independently inherited alleles (Long and Hogan, 1988). Crosses of albino raccoons and normal-colored raccoons produce offspring with a reddish-brown hair coat, referred to by some as "cinnamon" or "red" raccoons. **Congenital follicular dysplasia** has been reported in related litters of coatis (Nicolier et al., 2005). Coatis were born alopecic with only very short dark hairs, and cutaneous lichenification, crusting, and scaling. Histologically, the most significant lesion was premature cornification within the cells of the hair shaft. Other congenital lesions in raccoons are reported rarely in the literature and

include single case reports of forelimb and tail absence (Heidt, 1969), tail duplication (Vellard and Penteado, 1931), diaphragmatic hernia (Sanderson, 1960), renal hypoplasia (Mech and Anderson, 1966), and dental abnormalities (Knable and Werner, 1964). The author (M. Church) has observed a single case of an approximately 8 week old female raccoon with duplicated pelvises and two sets of hind limbs.

Age-Related/Degenerative

Aged animals have potential problems with **dental wear and attrition** as with other taxa. Dietary indiscretion can often result in damaged teeth and gastrointestinal foreign bodies in striped hyenas (R. Wack, personal communication). Similar to domestic carnivores, there are sporadic cases of both **hypertrophic and dilated cardiomyopathy** in aged species of uncertain pathogenesis (Eschar et al., 2010; Hollamby et al., 2004). Dilated cardiomyopathy appears to be somewhat common in captive mongooses (see section, Nutritional), although detailed population studies have not been conducted. In aged coatis, PAS positive, diastase-resistant, cytoplasmic globules are noted within neurons of the peripheral autonomic ganglia (Cooper et al., 2017). There was no evidence of concurrent neurodegeneration and globules appear to be an incidental aging change.

Miscellaneous

Vascular mineralization in the leptomeninges (overlying cerebrum, cerebellum, and spinal cord), choroid plexuses, and capillaries is common in the brains of adult free-ranging, especially aged, raccoons. It appears multifocally as round deposits of deeply basophilic, refractile material arranged in concentric rings in the walls of small capillaries and randomly throughout the neuropil. While these mineralized foci are sometimes associated with mild inflammation and fibrosis, the lack of associated clinical signs and lack of disruption of surrounding tissue make these foci of mineralization an incidental finding (Hamir, 2011a). Another commonly encountered lesion in the CNS of raccoons is **neuroaxonal degeneration**, which is seen consistently in the gracilis and cuneate nuclei of the medulla and is characterized by abundant vacuolated neurons and axonal spheroids. This lesion has no associated inflammation or clinical signs, and has been reported in young and adult free-ranging and captive raccoons (Hamir, 2011b; Hamir et al., 2002, 2007). Additionally, **perivascular eosinophilic cuffs**, primarily in the gray matter of the cerebrum are not uncommon in free-ranging adult raccoons. Their significance is unknown, but as with the earlier lesions, these changes in the brain are found in raccoons without neurologic signs.

Cystic endometrial hyperplasia, characterized by glandular dilation with intraluminal proteinaceous material and mild endometrial inflammation, is commonly seen in captive adult female raccoons (Hamir, 2011b; Hamir et al., 2007). **Polycystic kidneys**, in which cysts of varying sizes lined by a single layer of tubular epithelium that expanded and destroyed the renal cortex and medulla and were associated with lymphocytic inflammation, has been reported in a captive aged raccoon (Hamir and Klein, 1996). Single or multiple and extensive renal cysts are occasionally diagnosed in dwarf mongoose.

Neoplastic

As might be expected, neoplasia in procyonids and feliform carnivores has similar gross and histologic features, disease progression and prognosis to those commonly diagnosed in domestic cats and dogs. Reported neoplasms in these groups include lymphoma (Fig. 12.3), carcinomas of the urinary bladder, gastrointestinal tract (including anal sac glands), mammary gland, hepatobiliary, pulmonary, and renal systems (Aihara, 2009; Bjornson et al., 1999; Boonsri et al., 2013; Childs-Sanford et al., 2005; Chu et al., 2012; Dadone et al., 2014; Dawood, 2012; Effron et al., 1977; Goodnight et al., 2013; Hamir and Rupprecht, 1995; Hamir et al., 1996, 2008; Howard et al., 2007; Klaphake et al., 2005; Lombard and Witte, 1959; Marrow et al., 2014; Montali, 1980; Singh et al., 2005; Singleton et al., 2007; Thompson et al., 2016; Wadsworth et al., 1982; Wang et al., 2001). A single case of uterine adenocarcinoma in a captive coati associated with implantation of the contraceptive melengestrol acetate has also been reported (Chittick et al., 2001).

Thyroid neoplasms have been reported in raccoons in the United States and Germany (McCain et al., 2010). In Germany, the incidence was 64.3% (18/28) and 77.5% (31/40) in two necropsy surveillance studies of captive raccoons. Age likely plays a role in the oncogenesis, as none of the 37 free-ranging juvenile raccoons in one of the studies

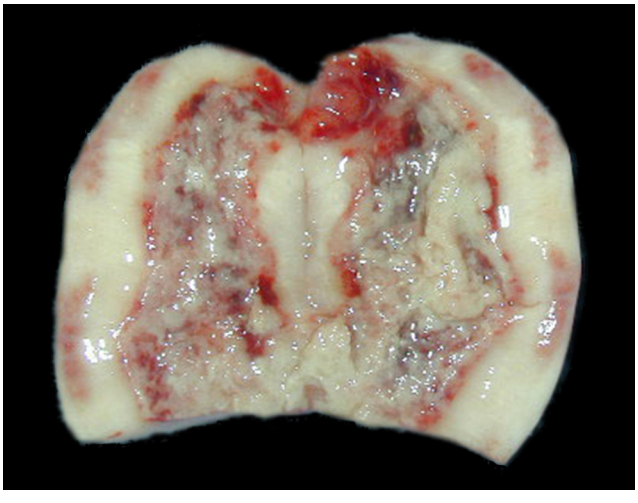

FIGURE 12.3 Uterine lymphosarcoma in a free-ranging raccoon. Longitudinal section of the uterine body with exposure of the lumen is shown. The uterine wall and mucosa contains an infiltrative soft, pale tan mass that thickens and obscures the normal layers of the uterine wall. Lymphosarcoma is a common tumor of free-ranging and captive raccoons.

had thyroid pathology. Tumors in raccoons included carcinomas, some of which metastasized (similar to dogs) and functional follicular hyperplastic lesions or adenomas (similar to cats).

A newly recognized group of neoplastic diseases in free-ranging raccoons in the western United States are *neuroglial brain tumors*. The tumors are associated with high levels of a novel raccoon polyomavirus (RacPyV) (Church et al., 2016; Dela Cruz et al., 2013). These tumors are consistently found in the olfactory tract and bulb of raccoons, and in some cases are so extensive that they extend rostrally into the nasal cavity (Fig. 12.4). Histologically these tumors have a varied appearance, ranging from bundled spindloid cells to sheets of polygonal cells interrupted by serpentine areas of necrosis with a pseudopallisading pattern (Giannitti et al., 2014) (Fig. 12.5). Confirmation of causality in oncogenesis remains to be achieved. However, the viral oncogenic gene, Large T antigen, is highly transcribed in tumor tissue relative to host (Brostoff et al., 2014), and the RacPyV genome is maintained in cultured tumor cells and the transformed phenotype is lost if the viral genome is inhibited from binding to the host genome in vitro (Church et al., 2016a). This suggests RacPyV as having an important role in the promotion of neoplasia in neural stem cells. Serological surveys have confirmed viral exposure in free-ranging raccoons with and without tumors across the United States and Canada with, suggesting additional factors may play a role in the oncogenesis of these tumors (Church et al., 2016b).

Several case reports describe **renal carcinoma** in binturongs; all have had metastatic disease (Childs-Sanford et al., 2005; Klaphake et al., 2005; Thompson et al., 2016). In one case, both the dam and sire of the affected animal died from complications of metastatic renal carcinoma (Thompson et al., 2016). The tumors in binturongs resemble those in other species. Grossly, they can be focal or extensive and replace normal parenchyma (Fig. 12.6). Histologically, they

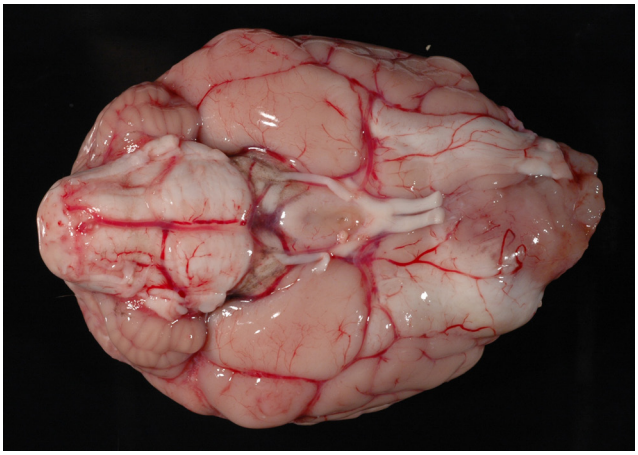

FIGURE 12.4 **Neuroglial brain tumor in raccoon.** An unencapsulated, poorly demarcated, soft, pale gray mass distorts the normal architecture of the left olfactory bulb. These tumors are consistently found in this location. In severe cases, they can extend into the nasal cavity.

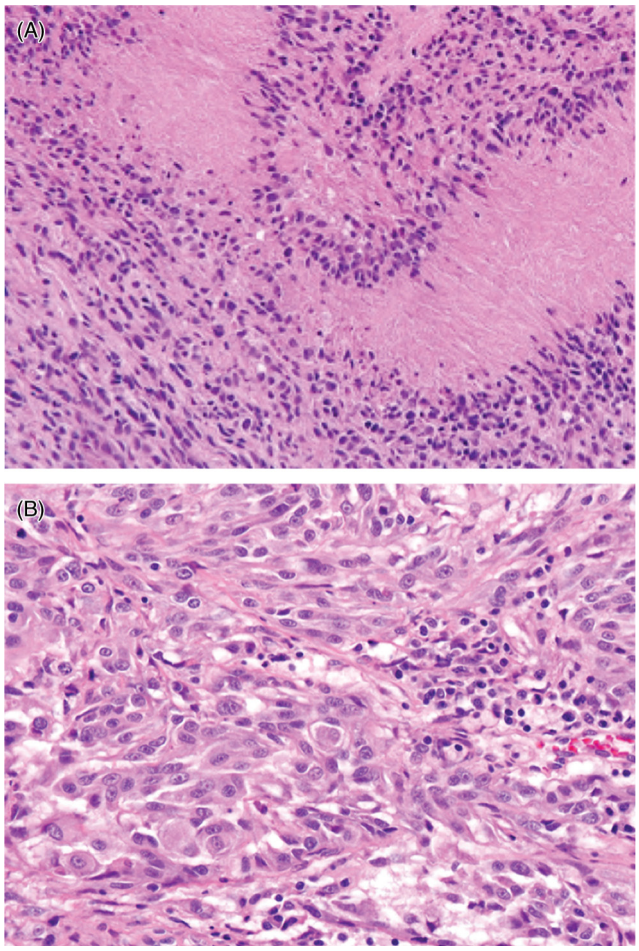

FIGURE 12.5 **Neuroglial brain tumor in a raccoon.** (A) A characteristic feature is a prominent pseudopalisading pattern of neoplastic cells. (B) Cells vary in morphology from sheets of polygonal sheets to streams of spindloid cells.

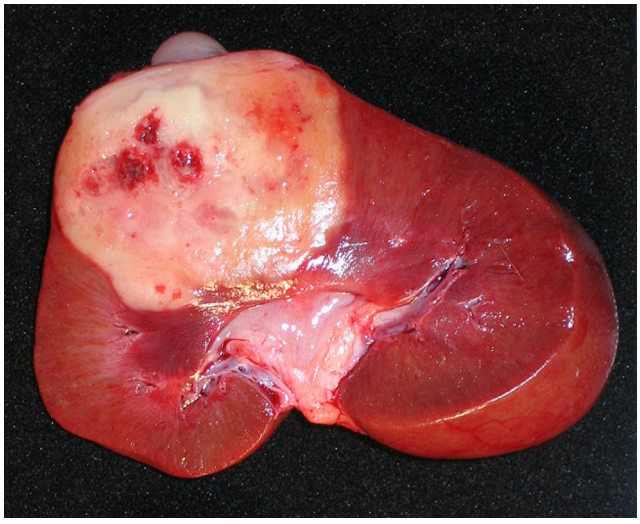

FIGURE 12.6 **Renal carcinoma in a binturong.** A focal, well-demarcated, tan mass within the cortex replaces the normal parenchyma. Metastasis is common, even with focal masses. Renal carcinomas are commonly reported in binturong. *(Photo Courtesy of C. Rodriguez, Wildlife Conservation Society)*

are composed of nests of well-differentiated to pleomorphic, cuboidal to polygonal (or spindle shaped, in the case of the sarcomatoid variant) cells sometimes arranged in irregular tubules. Metastatic disease in the lungs was present in all of the reported cases; metastases also occur in the mediastinum, pericardium, parietal pleura, muscles of the thoracic wall, adrenal glands, liver, spleen, and pancreas.

INFECTIOUS DISEASES

Procyonids and feliform carnivores are ubiquitous in their environments and share habitat and environmental resources with other nondomestic and domestic carnivores and humans. Because several of the species in this chapter act as reservoirs for a number of important multispecies or zoonotic pathogens, for example, raccoons (e.g. canine distemper virus and rabies) and civets (e.g. SARS coronavirus), surveys for pathogens that may be harbored or vectored by these species have been active areas of investigation. Unfortunately, less research has focused on the potential effects of these pathogens on their hosts. The diseases covered are primarily those for which disease is observed in wildlife species.

DNA Viruses

Infection with several viruses in the family ***Parvoviridae***, including feline parvovirus, canine parvovirus, and mink enteritis virus, occur in raccoons (Allison et al., 2013; Barker and Parrish, 2001; Kapil et al., 2010). The majority of **parvoviral disease** outbreaks are due to infection with a virus characterized to be phylogenetically positioned between canine parvovirus 2 (the initial virus that emerged in the 1970s) and 2a (one of the three subtypes, 2a, 2b, and 2c, that have emerged more recently); however, raccoons can be infected with all subtypes of canine parvovirus (Allison et al., 2013). Disease manifestation with any of these parvoviruses consists of lymphoid depletion, lymphoid necrosis, and necrohemorrhagic enteritis with sloughing of intestinal crypt epithelium and mucosal collapse ("radiomimetic" lesions) (Fig. 12.7) (Kapil et al., 2010). While most studies examining natural parvovirus infection in raccoons do not address age of affected animals, anecdotally disease tends to be more severe in young raccoon kits than in adults, similar to disease in domestic animals. Several species of civets and genets can be also be infected with canine and feline parvoviruses; similar gastrointestinal lesions and lymphoid depletion are described (Demeter et al., 2009; Wicker et al., 2017). In a survey of vehicle-killed and hunted free-ranging mongoose, almost 60% of tested individuals harbored feline parvoviral DNA (Duarte et al., 2013). However, given extremely low recovery of viral DNA and the lack of pathologic lesions, it is unclear if this species is truly susceptible to parvoviral-induced disease or is merely susceptible to infection and has the potential to act as a virus reservoir and vector.

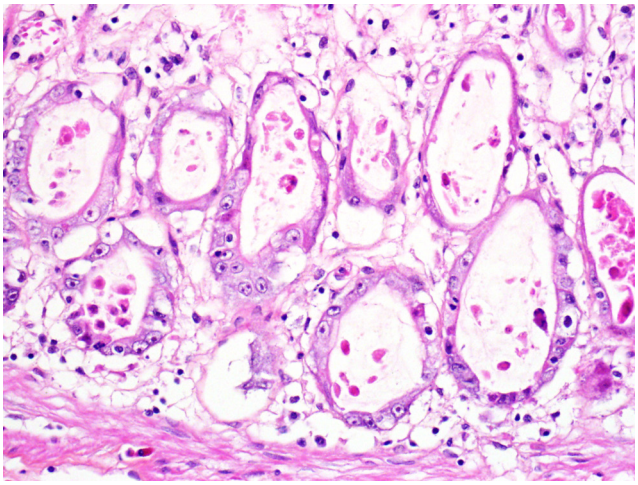

FIGURE 12.7 Parvoviral enteritis in the small intestine of a raccoon. Lesions resemble parvovirus infection in other species and include crypt dilation, crypt epithelium necrosis, and villous collapse.

Aleutian disease is caused by **Aleutian disease virus** (syn. ***carnivore amdoparvovirus*** 1). It is a highly contagious parvoviral disease that naturally occurs in mustelids, especially mink and ferrets (See Chapter 11), as well as other free-ranging carnivora including skunks, foxes, and raccoons. In mink, the disease causes plasmacytic inflammation in multiple organs and fibrinoid arteritis. Antibodies to the virus have been detected in raccoons in North America; experimental seroconversion has been demonstrated but disease is not often observed, suggesting raccoons as a potential reservoir for the virus (Oie et al., 1996). Free-ranging common genets in France have also been shown to harbor antibodies to this parvovirus, but active disease has not been demonstrated (Fournier-Chambrillon et al., 2004).

Captive mongooses in Germany are reportedly extremely susceptible to **cowpox virus**, with infection rates in some outbreaks as high as 100% (Kurth et al., 2009; Schmiedeknecht et al., 2010). Cowpox-infected rodents are thought to be the virus vectors. Gross lesions include multifocal cutaneous and mucocutaneous vesicles, pustules, and ulcerations. These correspond histologically to epithelial cell necrosis with eosinophilic, intraepithelial cytoplasmatic viral inclusion bodies and associated inflammatory infiltrates. Pinpoint to coalescing foci of necrosis are also noted in the lymph nodes, liver, spleen, and the gastrointestinal tract. These gross and histologic lesions are typical of cowpox lesions reported in other species susceptible to infection (Schmiedeknecht et al., 2010).

Pseudorabies [Aujeszky's disease virus (ADV), suid herpesvirus 1] has been reported in raccoons and hyenas (Stallknecht and Howerth, 2001). It is an important

differential diagnosis in suspect rabies cases, as animals infected with either virus can present with a variety of neurological signs. Gross lesions in raccoons are usually not evident or are associated with self-trauma from the severe pruritus that is frequently seen clinically. Histologic findings include nonsuppurative encephalitis with intranuclear, eosinophilic viral inclusion bodies in neurons, and myocardial, lymphoid, and pancreatic necrosis. While 30 years ago it was thought that raccoons were a likely reservoir for ADV, it is now suspected that feral swine and European wild boar are the main reservoirs (Boadella et al., 2012; Davidson, 2006).

Oral and **genital papillomas** have been identified in a population of wild spotted hyenas at the Masai Mara Game Reserve in Kenya (Nelson et al., 2013). Spontaneous regression occurred in some animals. The microscopic and gross appearance was similar to oral and cutaneous papillomas in other species. Virus isolated from hyenas was most closely related to the lambdapapillomaviruses of cats.

RNA Viruses

Rabies virus is a single stranded RNA virus in the *Lyssavirus* genus and the *Rhabdoviridae* family. Raccoons are consistently the most commonly reported rabid wildlife species in the eastern United States, and rabies infection is considered enzootic in populations in some eastern states. In addition to causing disease in raccoons, the raccoon strain of rabies is also the most common strain diagnosed in rabid domestic companion and production animals. Raccoons infected with rabies typically display a wide range of neurologic clinical signs including, but not limited to, diurnal activity, ataxia, head-pressing, paralysis, and aggressiveness (Ruprecht et al., 2001). There are no gross lesions directly associated with rabies virus infection, though affected animals may have superficial cutaneous lesions (i.e., contusions), tooth fractures or gastric foreign bodies as a result of ataxia, incoordination, aggression, and pica. Histopathologic findings include polioencephalomyelitis with perivascular cuffing by lymphocytes and plasma cells throughout cerebrum, brainstem, and spinal cord (Hamir, 2011a; Hamir et al., 1998; Ruprecht et al., 2001). In some cases, intracytoplasmic inclusion bodies (Negri bodies) can be seen (Fig. 12.8). Inclusions are reported to be most common in the hippocampus of carnivores but they may also be abundant in other regions including but probably not limited to cortical neurons, basal nuclei, thalamus, and caudal colliculi (De Araujo et al., 2014). The gold standard for diagnosis of rabies virus infection (in any species) is direct staining with fluorescent antibody (Davidson, 2006; Hamir, 2011a; Ruprecht et al., 2001). Domestic dogs and other nondomestic canid and carnivore species can serve as reservoirs for rabies virus in different geographic locations; mongoose are recognized reservoirs in Puerto Rico (Berentsen et al., 2015; Sabeta et al., 2008; Wicker et al., 2017).

Canine distemper virus (**CDV**) is a single stranded RNA virus in the *Morbillivirus* genus, *Paramyxoviridae* family. It is an extremely important multihost pathogen that affects all carnivores (Ramsay, 2015; Williams, 2001). Case reports and periodic outbreaks of canine distemper have been reported in most of the groups included in this chapter. Some species, like raccoons are reservoirs and possible sources of newly evolved strains (Riley and Wilkes, 2015). Disease manifestation is similar to that seen in domestic dogs, and young animals are most susceptible (Williams, 2001). Evidence of infection occurs in many different organ systems notably the respiratory, gastrointestinal, lymphoid, and nervous systems and clinical signs can therefore vary and include oculonasal discharge, coughing, diarrhea, anorexia, and concurrent infections. Blepharoconjunctivitis, rhinitis, and footpad hyperkeratosis can also be seen (Beineke et al., 2015; Williams, 2001). Neurologic signs are frequently encountered and rabies is an important differential diagnosis. The most common histopathologic findings are bronchointerstitial pneumonia (Fig. 12.9A) and generalized lymphoid depletion (Fig. 12.9B). Nonsuppurative encephalitis with neuronal necrosis and demyelination can be seen anywhere in the brain but is often most prominent in the cerebellar white matter; nonsuppurative polioencephalitis has also been described in farmed civets in Thailand (Techangamsuwan et al., 2015). Eosinophilic intracytoplasmic and intranuclear inclusion bodies can be seen in many different cells including epithelial cells and viral syncytial cells in many different organ systems (e.g., enterocytes, pneumocytes, transitional epithelium of the urinary bladder, neurons, and astrocytes). Lesions in captive binturongs in the United States and Korea included interstitial pneumonia, lymphoid depletion, and footpad hyperkeratosis but interestingly, they

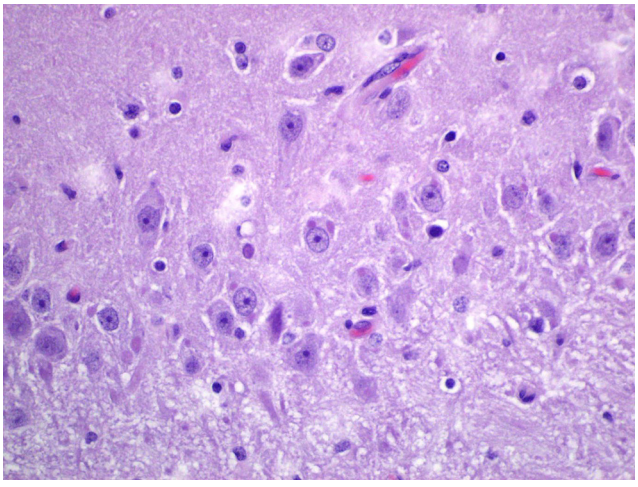

FIGURE 12.8 Rabies viral encephalitis in a raccoon. Neurons contain characteristic viral intracytoplasmic inclusion bodies (Negri bodies) with minimal associated inflammation. *(Photo Courtesy of D. McAloose, Wildlife Conservation Society)*

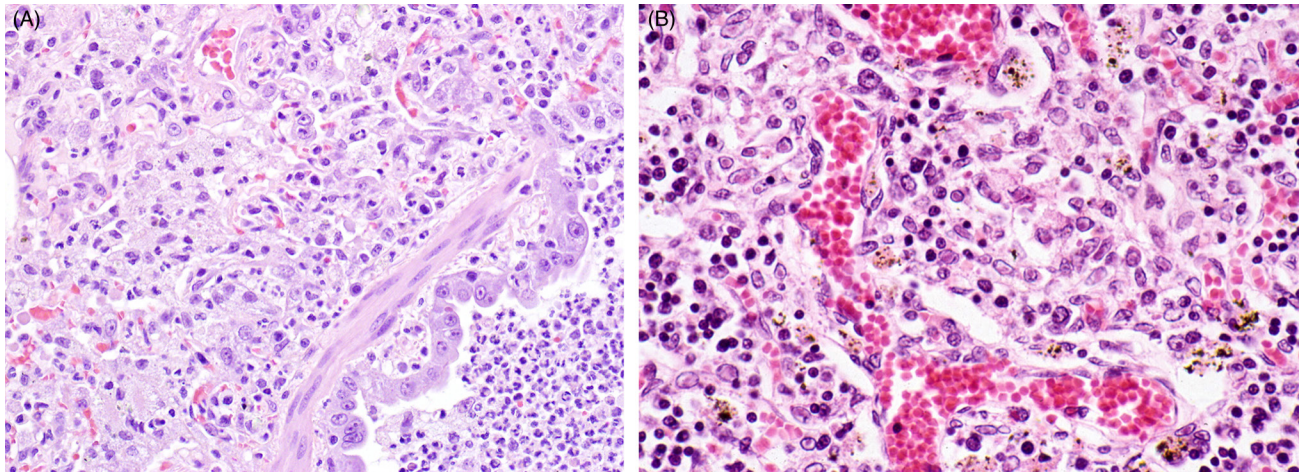

FIGURE 12.9 Canine distemper virus infection. (A) Bronchopneumonia in a raccoon. Bronchiolar epithelium contains numerous intracytoplasmic and intranuclear eosinophilic inclusions and there are rare syncytia. Alveolar type II pneumocyte hyperplasia with intranuclear and intracytoplasmic inclusions, and intraalveolar and intrabronchiolar neutrophils and foamy macrophages are also seen (B) Thymic necrosis in a hyena. Numerous histiocytes contain prominent intranuclear eosinophilic inclusions.

did not develop CNS disease (Chandra et al., 2000; Hur et al., 1999). Opportunistic infection with commensals or less common pathogens including *Toxoplasma* sp. (Dubey et al., 1992), *Sarcocystis* sp. (Kubiski et al., 2016), *Neospora* sp. (Lemberger et al., 2005), Tyzzer's disease (Wojcinski and Barker, 1986), and rabies (Hamir et al., 1998) can occur as comorbidities related to lymphoid depletion and immunosuppression (Fig. 12.10).

As mentioned earlier, viverrid species have been reported to be susceptible to a large number of viruses, raising the concern that they could serve as vectors for zoonotic disease. Specifically, civets were implicated as the source of **severe acute respiratory syndrome coronavirus** (**SARS-CoV**) in humans (Chan and Chan, 2013; Ge et al., 2013; Li et al., 2006). Civets are sensitive to illness from this virus, and given the short duration of associated pathological lesions, they likely represent an amplification host rather than a reservoir host. In an experimental study, viral RNA was detected by in situ hybridization (ISH) in the lung, small intestine, and cerebrum, and was associated with interstitial pneumonia, lymphoid depletion, and neuronal degeneration (Xiao et al., 2008).

Several viverrid species are also susceptible to **influenza virus** infection, which is transmissible not only between animal species but is also zoonotic. Severe interstitial pneumonia, in the absence of other infectious agents, was reported in a captive binturong infected with H1N1 influenza A during a global pandemic in 2009 (Schrenzel et al., 2011); captive Owston's civet and Palm civets have been reported to suffer illness from infection (Wicker et al., 2017); and an H5N1 outbreak in captive Owston's civets caused respiratory and neurologic disease and death in the three affected individuals (Roberton et al., 2006).

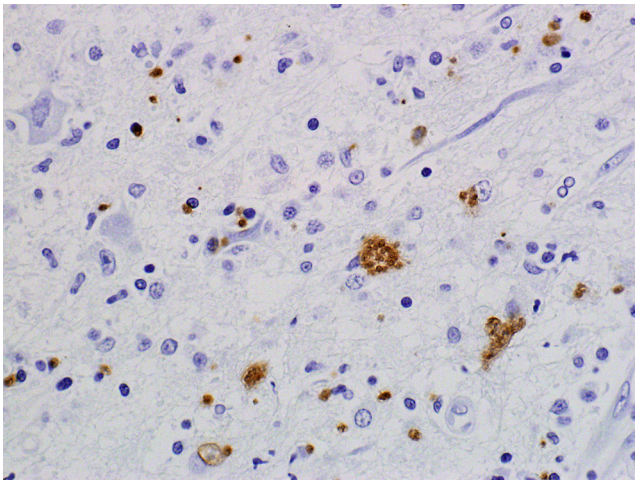

FIGURE 12.10 ***Sarcocystis neurona*** **encephalitis in a raccoon concurrently infected with canine distemper virus (CDV).** Protozoal infections are common secondary infections in raccoons debilitated by CDV infections. In the center of the image is a mature schizont with merozoites peripherally arranged in a characteristic pattern. Immunohistochemistry using antibodies against *Sarcocystis neurona*.

Bacteria

Many of the species covered in this chapter are susceptible to and are important species in the epidemiology of mycobacterial infections, particularly free-ranging mongoose and meerkats. The original studies of **mycobacteriosis** in meerkats identified *Mycobacterium tuberculosis* or *M. bovis*, members of the *M. tuberculosis* complex, as the causative organisms. However, more recent molecular analyses have identified a novel tuberculous mycobacterial species, ***M. suricatta***, in meerkats and the closely related ***M. mungi*** in mongoose (Alexander et al., 2002, 2010; Parsons et al., 2013). ***M. microti*** has also been reported in meerkat (Palgrave

et al., 2012). Epidemiological studies in wild meerkats suggest that mycobacterial infections spread locally within clusters of closely interacting individuals within a group, particularly among grooming individuals (Drewe et al., 2011). The pattern of lesions suggests infection is via inhalation. Social behaviors are also thought to be important in the transmission of *M. mungi* in banded mongoose. High levels of *M. mungi* organisms are found in anal gland and nasal planum of mongoose, suggesting olfactory related behaviors as being important in transmission (Alexander et al., 2016). Infections have also been reported in civet and genet, and spoligotyping identified close similarity to ***M. bovis*** strains from buffalo suggestive of spillover infections (Katale et al., 2017).

Lesions in meerkats are typical of tuberculous mycobacterial infections and include characteristic multifocal to coalescing granulomas in the lung, liver, lymph nodes, spleen, kidneys, and skin (Drewe et al., 2009). Histologically, granulomas contain numerous epithelioid macrophages with few lymphocytes. Multinucleated giant cells and neutrophils, as well as fibrosis, are rare findings. Acid-fast bacilli are common within lesions but many macrophages contain only a single bacillus. In mongoose, *M. mungi* lesions are common in the skin of the nasal planum and nasal cavity (Fig. 12.11). In mongoose, lesions in the lung were adjacent to vessels and thus fit a pattern of hematogenous spread rather than inhalation (Alexander et al., 2016). Similar to meerkats, fibrosis is minimal and central caseous necrosis within granulomas is only variable. In contrast, reported *M. bovis* lesions in mongoose consist of granulomas with central necrosis and mineralization in the lungs and lymph nodes; intralesional bacilli are rare (Brüns et al., 2017).

Both *M. bovis* and the nontuberculous ***M. avium*** have been detected in the tissues of free ranging raccoons (Miller and Sweeney, 2013; Witmer et al., 2010). Experimentally, raccoons develop granulomatous lymphadenitis, with characteristic granulomas (Palmer et al., 2002). Though not a significant disease in free-ranging raccoons, given their wide distribution and their intimate contact with domestic animals, raccoons could be a reservoir species for *M. bovis* (Witmer et al., 2010). Additional case reports of mycobacteriosis include ***M. intracellulare*** infection in a captive binturong (Adamvoicz et al., 2017) and ***M. goodii*** in a spotted hyena (van Helden et al., 2008). In the latter, infection caused bronchial lymphadeomegaly and severe, multifocal and regionally extensive, pneumonia with consolidation. Histologically, multifocal to coalescing, pyogranulomatous inflammation with multinucleated giant cells and intralesional acid-fast positive bacilli were present.

Raccoons are both susceptible to and may serve as a reservoir for a number of ***Leptospira*** species serotypes. Several serologic surveys in the United States demonstrate that raccoons commonly have circulating antibodies to

FIGURE 12.11 ***Mycobacterium mungi*** **rhinitis in a mongoose.** The nose is a common site for infection with this bacterium in mongoose. Lesions may be within the nasal cavity or within the skin of the nares. *(Photo Courtesy of C. Sanderson and K. Alexander, Department of Fish and Wildlife Conservation, Virginia Tech)*

a number of *L. interrogans* serovars including *grippotyphosa, autumnalis*, *hardjo,* and *icterohemorrhagiae* (Junge et al., 2007; Raizman et al., 2009; Richardson and Gauthier, 2003). Some studies have confirmed nephritis in association with the bacteria by using PCR and immunohistochemistry, indicating that raccoons are susceptible to disease and do not simply serve as asymptomatic reservoirs (Duncan et al., 2012; Hamir et al., 2001; Koizumi et al., 2009). Leptospiral infections have also been reported in mongoose and genets, with *L. interrogans* serovar *icterohemorrhagiae* being the most commonly identified strain (Millán et al., 2009; Moinet et al., 2010). Histologic lesions are similar to those in other species and consist of lymphoplasmacytic interstitial nephritis with fibrosis and tubular ectasia (Fig. 12.12A). Intralesional organisms can be identified in silver stained sections as well as by immunohistochemistry (Fig. 12.12B).

Severe swelling of the face, head, and neck; marked mandibular lymphadenomegaly and abscessation with rupture and drainage; respiratory distress; ocular discharge; ataxia; lethargy; and occasionally death were the hallmark findings of an outbreak of ***Streptococcus equi*** **subsp.** ***ruminatorum*** (Lancefield group C) in spotted hyenas in the Ngorongoro Crater in Tanzania (Höner et al., 2006; Speck et al., 2008). Histologically, suppurative inflammation and edema with intralesional colonies of Gram positive cocci were seen in the soft tissues and nodes in the head/neck; internal draining or aspiration with necrotizing and suppurative pneumonia and similar intralesional bacteria has also been described. The bacterium was cultured from affected and unaffected hyenas, suggesting a possible carrier state. It has also been cultured from Burchell's zebra, which are a prey species and the suspected source. Clinical signs in the hyenas were

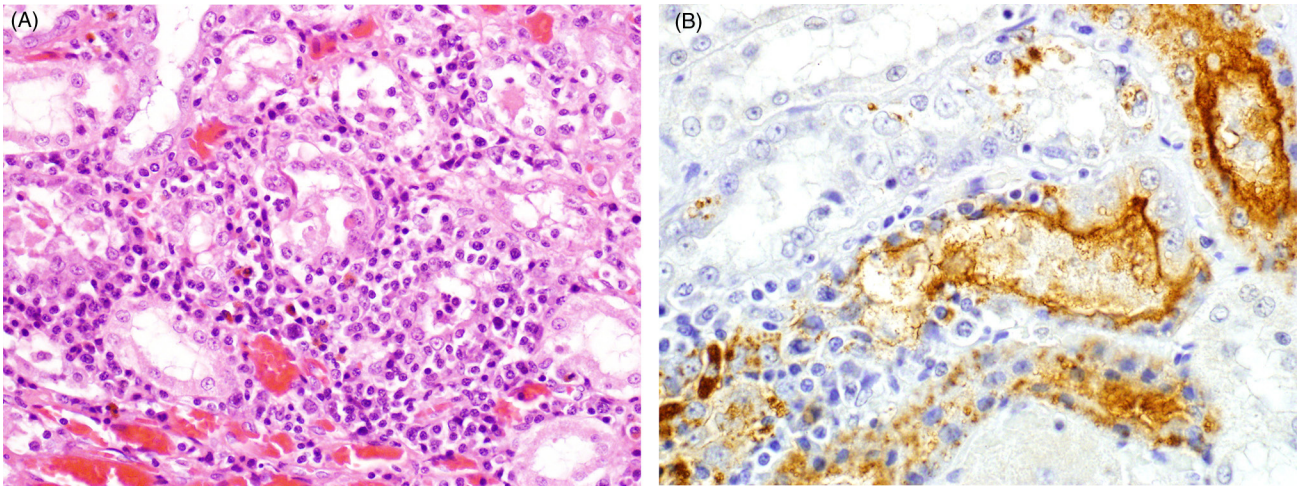

FIGURE 12.12 Leptospiral nephritis in a raccoon. (A) Interstitial lymphoplasmacytic nephritis centered on renal tubules is associated with tubular epithelial necrosis. (B) Immunohistochemistry using antibodies against leptospiral antigens demonstrating strong positive staining in tubular epithelial cells and tubular lumens. Immunohistochemistry using antibodies against *Leptospira* spp.

similar to a disease colloquially called "strangles" in domestic horses caused by the related bacterium *Streptococcus equi*.

Yersinia pseudotuberculosis infection has been described in captive meerkats with necrotizing enteritis, hepatitis, and splenitis (Nakamura et al., 2015). Inflammation was predominately neutrophilic and surrounded two morphologic forms of the bacteria: bacilli and larger globular bodies that were positive for *Y. pseudotuberculosis* O4 antigen by immunohistochemistry.

An unclassified ***Mycoplasma*** species causes epiphysitis and periostitis in captive and rehabilitating raccoons (Whithear, 2001). Clinical findings and gross lesions include joint swelling and abscessation of the extremities. *Mycoplasmosis* can be successfully treated with long term antibiotic administration, even though with resolution, infection has the potential to cause permanent skeletal deformities if it occurs in a young, growing animal. One study detected Mycoplasma sp. in conjunctival swabs of healthy, free-ranging raccoons, so it is unknown if disease is the result of opportunistic infection of a commensal organism (Pinard et al., 2002).

Anthrax, due to ***Bacillus anthracis***, is a significant infectious, zoonotic disease. It is typically associated with ungulates, though most mammals, including carnivores that prey upon infected animals or carcasses, are thought to be susceptible. It is a large, spore-forming, Gram-positive, rod-shaped, bacterium with characteristic blunt ends and a clear capsule. Infection has been reported in captive genet (Ikede et al., 1976). Lesions included systemic hemorrhages as well as hemorrhage around the larynx and trachea, similar to what has been described in felids (See Chapter 10). Histologically, hemorrhagic necrosis of the intestinal villi was associated with bacterial invasion. Lymph node and splenic sinusoids contained numerous characteristic bacilli. Ingestion of contaminated meat was the presumed route of infection.

Fungi

Fungal disease is rarely reported in these species. Case reports include **histoplasmosis (*Histoplasma capsulatum*)** in two raccoon kits with disseminated, systemic infection and granulomatous pneumonia, lymphadenitis, splenitis, enteritis, nephritis, encephalitis, and thymitis with intrahistiocytic yeast (Clothier et al., 2014); ***Trichophyton mentagrophytes*** alopecic dermatitis in a captive adult female spotted hyena and her two cubs (Hahn et al., 2003); and fatal meningoencephalitis due to ***Encephalitozoon cuniculi*** in captive meerkats (Ramsay, 2015).

Metazoa

There are a number of metazoan parasites described in the procyonids and feliform carnivores without associated disease. Wicker et al. (2017) provide an excellent review of viverridae pathogens and the reader is referred to that article for more information regarding parasites infecting that group.

Baylisascaris procyonis is a nematode parasite that is commonly found in the small intestines of free-ranging raccoons. Raccoons are the natural host, and infection is only rarely associated with disease. In aberrant hosts, including other mammalian and avian species as well as humans, larval migrans can cause severe disease. A related roundworm, ***B. potosis***, was recently described in captive kinkajous. Its zoonotic potential, if any, is currently unknown (Tokiwa et al., 2014). ***Physaloptera rara*** and ***Gnathostoma procyonis*** are also common in raccoons. Infection with these gastric nematodes is also typically asymptomatic, but ulcers may

be seen in heavily parasitized individuals (Davidson, 2006). The subcutaneous nematode of raccoons, ***Dracunculus insignis***, can cause edema and inflammation in the subcutis and skeletal muscle fascia, and extrusion of the female worm through the skin may result in an ulcerative wound (Davidson, 2006). These worms must be distinguished from larvae of the subcutaneous tapeworm ***Spirometra mansonoides***, which are smaller and flatter than *D. insignis.*

Angiostrongylus species are metastrongyle nematode parasites that are found in the cardiovascular and respiratory system of definitive hosts and utilize (typically) a gastropod as an intermediate. ***Angiostrongylus dujardini*** infection has been described in a group of captive meerkats (Eleni et al., 2016). Affected animals had congested lungs and yellow foci in the lungs and liver. Histologically, inflammation in the lungs surrounded parasite eggs and larvae and adults were present in pulmonary arteries. Inflammation in the liver was variable. As cases occurred concurrently with other animals housed at the facility, consumption of infected gastropod intermediate hosts was presumed.

Ancyclostoma sp. and ***Arthrocephalus sp***. have been reported in mongoose, civets, hyena, and racooons (Seguel and Gottdenker, 2017). Raccoons have also been reported to be infected with ***Necator, Arthrostoma*** and ***Uncinaria spp***. While local tissue damage, reduced growth rates, and anemia have been reported in other species, reports of associated disease in the groups covered in this chapter are limited.

Eleven species of **acanthocephalans** have been reported in free-ranging North American raccoons (*Arythmorhynchus frassoni*, *Centrorhynchus conspectus*, *Centrorhynchus spinonsus*, *Centrorhynchus* sp., *Echinorhynchus gadi*, *Moniliformis* sp., *Neoechinorhynchus cylindratus*, *Oligacanthorhynchus tortuosa*, *Plagiorhynchus cylindraceus*, *Profilicollis botulus*, *Southwellina hispida*, and *Macracanthorhynchus ingens). M. ingens* is the only species for which the raccoon is the definitive host (Richardson, 2014). This particular acanthocephalan can also infect kinkajous. Despite this thorny headed worm's large size, it does not commonly cause disease in raccoons. *O. tortuosa* have been reported to cause severe ulceration at the site of attachment in isolated cases (Richardson et al., 1992).

Protozoa

Meerkats appear to be highly susceptible to ***Toxoplasma gondii*** infection and multiple outbreaks with high mortality have been described in captive groups (Basso et al., 2009; Burger et al., 2017; Juan-Sallés et al., 1997). Clinical signs include respiratory distress and neurologic signs. The lungs are congested and fail to collapse, corresponding histologically to interstitial pneumonia. Pale foci that correspond to areas of necrosis occur in multiple sites, commonly the lung, spleen, and lymph nodes. Histologically, necrosis with neutrophilic to pyogranulomatous inflammation surrounds free and intracytoplasmic, individual and clusters of zoites in tissues with gross evidence of infection as well as those that appear grossly normal. Hepatitis and nonsuppurative encephalitis are not uncommon lesions associated with toxoplasmosis. Infection can be confirmed by immunohistochemistry or molecular methods. Infection is often presumed to occur through ingestion of contaminated feed. Typing of isolates from one outbreak identified clonal type III lineage, although the types present within potential source populations were not described (Basso et al., 2009).

In raccoons infected with CDV, *Toxoplasma gondii*, *Neospora caninum*, and *Sacrocystis neurona* are not uncommon secondary opportunistic infections (see CDV in RNA Viruses section; Fig. 12.10). Seroprevalence of exposure to ***Toxoplasma gondii*** is relatively high in free-ranging raccoons and lower ringtails in North America, with seropositivity rates of ~80% and ~20%, respectively, depending on the geographical location (Hamir, 2011a; Suzán and Ceballos, 2005). The pyogranulomatous pneumonia seen in raccoons with CDV and *Toxoplasma* coinfections are similar to those described in other species. *Toxoplasma gondii* has also been transmitted readily in an experimental setting to raccoons (Hamir, 2011a).

Leishmania infantum infection has been reported in free-ranging genets but disease and associated clinical signs have not been described (Del Rio et al., 2014). Serum biochemistry abnormalities, such as elevated globulins, have been reported in some individual animals but are inconsistent findings (Millán et al., 2015).

Enteric coccidia including two species of *Eimeria* (*E. nuttali* and *E. procyonis*) and one species of *Isospora* have been described in the raccoon (Dubey et al., 2000). Though the significance of these pathogens on a population level is unclear, marked enteritis associated with parasitism contributed to the mortality of the raccoons in the published cases.

Free-ranging raccoons have a high seroprevalence in antibodies to ***Trypanosoma cruzi***, though associated disease is extremely rare (Yabsley and Noblet, 2002).

Babesia sp. have been reported in coatis, raccoons, genets, spotted hyena, civets, and meerkats (Alvarado-Rybak et al., 2016; Wicker et al., 2017). Some individuals have evidence of splenomegaly and anemia associated with infection. Coinfections with other hemoparasites such as ***Hepatozoon sp.*** in hyena and ***Cytauxzoon sp.*** in meerkat have also been identified by PCR, but the clinical impact of these infections were not described.

Additional protozoa of hyenidae are presented in Supplemental Table e2.

Ectoparasites

All species in this chapter are susceptible to **sarcoptic mange** caused by the mite ***Sarcoptes scabei*.** Lesions have been described in native raccoons in the United States (Fitzgerald et al., 2004) and also in introduced invasive populations in Germany (Rentería-Solís et al., 2014). Affected animals are severely pruritic and have regional alopecia and crusting dermatitis. Diagnosis is usually made by identifying mites on skin scrapes, though they can also be identified histologically in sections of skin. In addition to *Sarcoptes* sp., ***Notoedres* sp.** infections have been identified in free-ranging and captive civets (Ninomiya et al., 2003; Olivieri et al., 2015). Affected civets were pruritic and had severe alopecia and crusting lesions. Histologically affected skin was acanthotic and hyperkeratotic with intralesional mites. Hair follicles were parakeratotic and many were devoid of hair. Dermal inflammation was mild. Diagnosis can be confirmed by histopathology and microscopic evaluation of skin scrapings.

Visceral pentastomiaisis due to ***Armillifer armillatus*** nymphal migrans has been diagnosed in a zoo-housed, brown (Greve, 2017) and a striped hyena (Dechkajorn et al., 2016); ***A. armillatus*** and ***A. moniliformis*** have also been reported in aardwolf (Christoffrsen and De Assis, 2013) and *A. moniliformis* has been reported in civets and binturong (Wicker et al., 2017). Snakes are the definitive and canids are the intermediate hosts. Encysted nymphs in the striped hyena were present throughout the omentum, mesentery, diaphragm, intestines, liver, spleen, kidneys, and urinary bladder, where they were surrounded by a thin fibrous capsule and mild, chronic or neutrophilic (intestinal) inflammation. Diagnosis of pentastomiasis is typically based on identification of the characteristic parasites on gross necropsy examination. Histology and PCR with DNA sequencing support the gross diagnosis and in the case of the latter, it can be used to speciate the parasite.

Fleas are a common, generally species-specific parasite. Free-ranging raccoons in the United States are frequently infested with *Orchopeas howardi*, though they can also be infested by fleas from the *Ctenocephalides* genus (Hunter et al., 1979; Monello and Gompper, 2009). Raccoons are also frequently infested with the **louse** *Trichodectes octomaculatus*. Infestation by these ectoparasites rarely causes disease. Species of ***Ixodes*** tick are important vectors for *Babesia sp.* infections in genets and raccoons (Alvarado-Rybak et al., 2016).

Prions

Raccoons have been experimentally infected with three types of transmissible spongiform encephalopathies including transmissible mink encephalopathy, sheep scrapie, and chronic wasting disease (Hamir et al., 2007). Results suggest susceptibilty to transmissible mink encephalopathy and sheep scrapie but resistance to chronic wasting disease. No natural cases have been reported.

E-SLIDES

12.e1 **Neuroglial tumor, raccoon, olfactory peduncle.** The neuroparenchyma of the olfactory peduncle is markedly expanded and replaced by this infiltrative neoplasm. There are large areas of necrosis, though true palisading of neoplastic cells at the edges of these areas is not noted. Neoplastic cells are arranged in dense sheets, and demonstrate moderate anisocytosis and anisokaryosis with frequent mitoses. Aggregates of lymphocytes and foci of mineralization are scattered throughout the neoplasm. (see Fig. 12.5). eSlide: VM04951

12.e2 **Leptospirosis, raccoon, kidney.** Multifocally, the interstitium contains aggregates of lymphocytes and plasma cells centered on renal tubules. Some tubules are necrotic and or have sloughed cellular debris within the lumen. Intralesional organisms can be identified by silver stains or immunohistochemistry. (See Fig. 12.12). eSlide: VM05043

12.e3 **Canine Distemper Virus pnuemonia, Raccoon, Lung.** There is a multifocal bronchopneumonia that is associated with intranuclear and intracytoplasmic viral inclusions within epithelial cells. In multiple affected areas, there are syncytia. Inflammation consists predominately of alveolar histiocytes and neutrophils. Affected alveoli are lined by plump type II pneumocytes. (See Fig. 12.9). eSlide: VM05050

12.e4 **Parvoviral enteritis, Raccoon, small intestine.** Throughout the sections, small intestinal crypts are necrotic, villi are blunted and collapsed. Crypts are dilated and lined by attenuated to discontinuous epithelium with luminal cellular debris and neutrophils. Few crypts have piled, disorganized epithelium. Large numbers of eosinophils (a common cell in raccoons) are also present. Loss of superficial epithelium is an artifact. (See Fig. 12.7). eSlide: VM05230

E-ONLY CONTENTS

Table e1 Taxonomy and Conservation Status of Procyonidae and Feliform Carnivores

Family	Genus	Species	Common Names (Eng)	Red List Status[a]	Year Assessed[b]	Population trend
Eupleridae	*Cryptoprocta*	*ferox*	Fossa	VU	2016	Decreasing
Eupleridae	*Cryptoprocta*	*spelea*	Giant Fosa, Giant Fossa	EX	2015	
Eupleridae	*Eupleres*	*goudotii*	Eastern Falanouc, Fanalouc	VU	2016	Decreasing
Eupleridae	*Eupleres*	*major*	Western Falanouc	EN	2016	Decreasing
Eupleridae	*Fossa*	*fossana*	Spotted Fanaloka, Fanaloka, Malagasy Civet	VU	2015	Decreasing
Eupleridae	*Galidia*	*elegans*	Ring-tailed Vontsira, Malagasy Ring-tailed Mongoose, Ring-tailed Mongoose	LC	2015	Decreasing
Eupleridae	*Galidictis*	*fasciata*	Broad-striped Vontsira, Broad-striped Mongoose, Malagasy Broad-striped Mongoose	VU	2016	Decreasing
Eupleridae	*Galidictis*	*grandidieri*	Grandidier's Vontsira, Giant-striped Mongoose, Grandidier's Mongoose	EN	2015	Decreasing
Eupleridae	*Mungotictis*	*decemlineata*	Bokiboky, Malagasy Narrow-striped Mongoose, Narrow-striped Mongoose	EN	2015	Decreasing
Eupleridae	*Salanoia*	*concolor*	Brown-tailed Vontsira, Brown-tailed Mongoose, Malagasy Brown-tailed Mongoose	VU	2016	Decreasing
Herpestidae	*Atilax*	*paludinosus*	Marsh Mongoose, Water Mongoose	LC	2015	Decreasing
Herpestidae	*Bdeogale*	*crassicauda*	Bushy-tailed Mongoose	LC	2016	Unknown
Herpestidae	*Bdeogale*	*jacksoni*	Jackson's Mongoose	NT	2015	Decreasing
Herpestidae	*Bdeogale*	*nigripes*	Black-legged Mongoose, Black-footed Mogoose	LC	2015	Decreasing
Herpestidae	*Bdeogale*	*omnivora*	Sokoke Dog Mongoose, Sokoke Bushy-tailed Mongoose	VU	2016	Decreasing
Herpestidae	*Crossarchus*	*alexandri*	Alexander's Cusimanse	LC	2015	Decreasing
Herpestidae	*Crossarchus*	*ansorgei*	Ansorge's Cusimanse, Angolan Cusimanse	LC	2015	Decreasing
Herpestidae	*Crossarchus*	*obscurus*	Common Cusimanse, Cusimanse, Long-nosed Cusimanse	LC	2015	Unknown
Herpestidae	*Crossarchus*	*platycephalus*	Flat-headed Cusimanse, Cameroon Cusimanse	LC	2016	Unknown
Herpestidae	*Cynictis*	*penicillata*	Yellow Mongoose	LC	2015	Stable
Herpestidae	*Dologale*	*dybowskii*	Pousargues's Mongoose, Savanna Mongoose	DD	2015	Unknown
Herpestidae	*Helogale*	*hirtula*	Somali Dwarf Mongoose, Desert Dwarf Mongoose, Ethiopian Dwarf Mongoose	LC	2015	Unknown
Herpestidae	*Helogale*	*parvula*	Common Dwarf Mongoose, Dwarf Mongoose	LC	2015	Stable
Herpestidae	*Herpestes*	*auropunctatus*	Small Indian Mongoose	LC	2016	Unknown
Herpestidae	*Herpestes*	*brachyurus*	Short-tailed Mongoose	NT	2016	Decreasing

Table e1 Taxonomy and Conservation Status of Procyonidae and Feliform Carnivores (*Cont.*)

Family	Genus	Species	Common Names (Eng)	Red List Status[a]	Year Assessed[b]	Population trend
Herpestidae	*Herpestes*	*edwardsii*	Indian Gray Mongoose, Common Mongoose, Gray Mongoose	LC	2016	Stable
Herpestidae	*Herpestes*	*flavescens*	Kaokoveld Slender Mongoose, Angolan Slender Mongoose, Black Mongoose, Black Slender Mongoose	LC	2015	Unknown
Herpestidae	*Herpestes*	*fuscus*	Brown Mongoose, Indian Brown Mongoose	LC	2015	Stable
Herpestidae	*Herpestes*	*ichneumon*	Egyptian Mongoose, Large Gray Mongoose	LC	2016	Stable
Herpestidae	*Herpestes*	*javanicus*	Javan Mongoose	LC	2016	Unknown
Herpestidae	*Herpestes*	*naso*	Long-nosed Mongoose	LC	2015	Decreasing
Herpestidae	*Herpestes*	*ochraceus*	Somali Slender Mongoose, Somalian Slender Mongoose	LC	2015	Unknown
Herpestidae	*Herpestes*	*pulverulentus*	Cape Gray Mongoose, Small Gray Mongoose	LC	2015	Stable
Herpestidae	*Herpestes*	*sanguineus*	Common Slender Mongoose, Slender Mongoose	LC	2016	Stable
Herpestidae	*Herpestes*	*semitorquatus*	Collared Mongoose	NT	2015	Decreasing
Herpestidae	*Herpestes*	*smithii*	Ruddy Mongoose	LC	2016	Unknown
Herpestidae	*Herpestes*	*urva*	Crab-eating Mongoose	LC	2015	Decreasing
Herpestidae	*Herpestes*	*vitticollis*	Stripe-necked Mongoose, Striped-necked Mongoose	LC	2016	Stable
Herpestidae	*Ichneumia*	*albicauda*	White-tailed Mongoose	LC	2015	Stable
Herpestidae	*Liberiictis*	*kuhni*	Liberian Mongoose	VU	2016	Decreasing
Herpestidae	*Mungos*	*gambianus*	Gambian Mongoose	LC	2016	Stable
Herpestidae	*Mungos*	*mungo*	Banded Mongoose	LC	2016	Stable
Herpestidae	*Paracynictis*	*selousi*	Selous's Mongoose	LC	2016	Unknown
Herpestidae	*Rhynchogale*	*melleri*	Meller's Mongoose	LC	2015	Unknown
Herpestidae	*Suricata*	*suricatta*	Meerkat, Slender-tailed Meerkat, Suricate	LC	2015	Stable
Hyaenidae	*Crocuta*	*crocuta*	Spotted Hyaena	LC	2015	Decreasing
Hyaenidae	*Hyaena*	*hyaena*	Striped Hyaena	NT	2015	Decreasing
Hyaenidae	*Parahyaena*	*brunnea*	Brown Hyaena	NT	2015	Stable
Hyaenidae	*Proteles*	*cristata*	Aardwolf	LC	2015	Stable
Nandiniidae	*Nandinia*	*binotata*	African Palm Civet, Tree Civet, Two-spotted Palm Civet	LC	2015	Unknown
Procyonidae	*Bassaricyon*	*alleni*	Eastern Lowland Olingo	LC	2016	Decreasing
Procyonidae	*Bassaricyon*	*gabbii*	Northern Olingo	LC	2016	Decreasing
Procyonidae	*Bassaricyon*	*medius*	Western Lowland Olingo	LC	2016	Decreasing
Procyonidae	*Bassaricyon*	*neblina*	Olinguito	NT	2016	Decreasing
Procyonidae	*Bassariscus*	*astutus*	Ringtail	LC	2016	Unknown
Procyonidae	*Bassariscus*	*sumichrasti*	Cacomistle, Central American Cacomistle	LC	2016	Unknown
Procyonidae	*Nasua*	*narica*	White-nosed Coati, Coatimundi	LC	2016	Decreasing

(*Continued*)

Table e1 Taxonomy and Conservation Status of Procyonidae and Feliform Carnivores (*Cont.*)

Family	Genus	Species	Common Names (Eng)	Red List Status[a]	Year Assessed[b]	Population trend
Procyonidae	*Nasua*	*nasua*	South American Coati	LC	2016	Decreasing
Procyonidae	*Nasuella*	*meridensis*	Eastern Mountain Coati	EN	2016	Decreasing
Procyonidae	*Nasuella*	*olivacea*	Western Mountain Coati	NT	2016	Decreasing
Procyonidae	*Potos*	*flavus*	Kinkajou	LC	2016	Decreasing
Procyonidae	*Procyon*	*cancrivorus*	Crab-eating Raccoon	LC	2016	Decreasing
Procyonidae	*Procyon*	*lotor*	Northern Raccoon	LC	2016	Increasing
Procyonidae	*Procyon*	*pygmaeus*	Pygmy Raccoon, Cozumel Island Raccoon, Cozumel Raccoon, Cozumel Raccoon Bear	CR	2016	Decreasing
Viverridae	*Arctictis*	*binturong*	Binturong, Bearcat	VU	2016	Decreasing
Viverridae	*Arctogalidia*	*trivirgata*	Small-toothed Palm Civet, Three-striped Palm Civet	LC	2016	Decreasing
Viverridae	*Chrotogale*	*owstoni*	Owston's Civet, Owston's Banded Civet, Owston's Banded Palm Civet, Owston's Palm Civet	EN	2016	Decreasing
Viverridae	*Civettictis*	*civetta*	African Civet	LC	2015	Unknown
Viverridae	*Cynogale*	*bennettii*	Otter Civet, Otter-civet, Sunda Otter Civet	EN	2015	Decreasing
Viverridae	*Diplogale*	*hosei*	Hose's Civet, Hose's Palm Civet	VU	2015	Decreasing
Viverridae	*Genetta*	*abyssinica*	Ethiopian Genet, Abyssinian Genet	DD	2016	Unknown
Viverridae	*Genetta*	*angolensis*	Miombo Genet, Angolan Genet	LC	2016	Unknown
Viverridae	*Genetta*	*bourloni*	Bourlon's Genet	VU	2015	Decreasing
Viverridae	*Genetta*	*cristata*	Crested Genet, Crested Servaline Genet	VU	2015	Decreasing
Viverridae	*Genetta*	*genetta*	Common Genet	LC	2015	Stable
Viverridae	*Genetta*	*johnstoni*	Johnston's Genet	NT	2016	Decreasing
Viverridae	*Genetta*	*maculata*	Large-spotted Genet, Blotched Genet, Central African Large-spotted Genet, Panther Genet, Rusty-spotted Genet	LC	2016	Unknown
Viverridae	*Genetta*	*pardina*	Pardine Genet, West African Large-spotted Genet	LC	2016	Unknown
Viverridae	*Genetta*	*piscivora*	Aquatic Genet	NT	2015	Decreasing
Viverridae	*Genetta*	*poensis*	King Genet	DD	2015	Unknown
Viverridae	*Genetta*	*servalina*	Servaline Genet	LC	2016	Unknown
Viverridae	*Genetta*	*thierryi*	Hausa Genet	LC	2015	Unknown
Viverridae	*Genetta*	*tigrina*	Cape Genet, Cape Large-spotted Genet, South African Large-spotted Genet	LC	2015	Stable
Viverridae	*Genetta*	*victoriae*	Giant Genet	LC	2016	Unknown
Viverridae	*Hemigalus*	*derbyanus*	Banded Civet, Banded Palm Civet	NT	2015	Decreasing
Viverridae	*Macrogalidia*	*musschenbroekii*	Sulawesi Civet, Brown Palm Civet, Sulawesi Palm Civet	VU	2015	Decreasing
Viverridae	*Paguma*	*larvata*	Masked Palm Civet, Gem-faced Civet, Himalayan Palm Civet	LC	2016	Decreasing
Viverridae	*Paradoxurus*	*hermaphroditus*	Common Palm Civet, Mentawai Palm Civet	LC	2016	Decreasing

Table e1 Taxonomy and Conservation Status of Procyonidae and Feliform Carnivores (*Cont.*)

Family	Genus	Species	Common Names (Eng)	Red List Status[a]	Year Assessed[b]	Population trend
Viverridae	*Paradoxurus*	*jerdoni*	Brown Palm Civet, Jerdon's Palm Civet	LC	2016	Stable
Viverridae	*Paradoxurus*	*zeylonensis*	Golden Palm Civet, Golden Dry-Zone Palm Civet, Golden Wet-Zone Palm Civet, Sri Lanka Brown Palm Civet, Sri Lanka Mountain Palm Civet, Sri Lankan Golden Striped-backed Palm Civet	LC	2016	Unknown
Viverridae	*Poiana*	*leightoni*	West African Oyan, Leighton's Linsang, West African Linsang	VU	2015	Decreasing
Viverridae	*Poiana*	*richardsonii*	Central African Oyan, African Linsang, Central African Linsang, Richardson's Linsang	LC	2015	Unknown
Viverridae	*Viverra*	*civettina*	Malabar Civet, Malabar Large-spotted Civet	CR	2016	Decreasing
Viverridae	*Viverra*	*megaspila*	Large-spotted Civet	EN	2016	Decreasing
Viverridae	*Viverra*	*tangalunga*	Malay Civet, Malayan Civet, Oriental Civet	LC	2016	Stable
Viverridae	*Viverra*	*zibetha*	Large Indian Civet	LC	2016	Decreasing
Viverridae	*Viverricula*	*indica*	Small Indian Civet, Oriental Civet	LC	2015	Stable

[a] CR, Critically endangered; DD, data deficient; EN, endangered; EX, extinct; VU, vulnerable; NT, near threatened; LC, least concern. [b] Status is periodically reviewed, see www.IUCNredlist.org for more detail and current status.

Table e2 Protozoal Infections Reported in Hyenidae and Protelinae

Species	Name	Country/Region	Sample/Test	Gross Findings (Nx)	Histologic Findings (Nx)	References
Aardwolf	*Babesia* sp.	South Africa/Northern Cape Province	Blood/Smear	NA	NA	Peirce et al. (2001)
Spotted hyena	*Babesia* sp.	Zambia/South Luangwa and Liuwa Plain National Parks	Blood/Smear, PCR	NA	NA	Williams et al. (2014)
Brown hyena	*Babesia* sp. related to *B. lengau*	Namibia, South Africa	Blood, skin/PCR	NA	NA	Burroughs et al. (2017)
Spotted hyena	*Babesia* sp. related to *B. lengau*	Namibia, South Africa	Blood, skin/PCR	NA	NA	Burroughs et al. (2017)
Spotted hyena	*Hepatozoon* sp.	Tanzania/Serengeti National Park	Tissue/Histology, PCR	NGL	Cyst-like structures containing protozoa in lung, heart, liver, spleen, kidney, lymph node, stomach, skeletal muscle; interstitial pneumonia; necrotizing myocarditis	East et al. (2008)
Spotted hyena	*Hepatozoon* sp.	Zambia/South Luangwa and Liuwa Plain National Parks	Blood/Smear, PCR	NA	NA	Williams et al. (2014)
Brown hyena	*T. gondii*	Czech and Slovak Republics/Multiple zoos	Serum/Serology	NA	NA	Sedlák and Bártová (2006)
Aardwolf	*Trypanosoma cruzi*	US/San Antonio Zoological Gardens and Aquarium	Tissue/Histology, EM	Left ventricular dilated myocardiomyopathy	Necrotizing lymphoplasmacytic, histiocytic myocarditis with intracellular parasites	Fletcher and Hubbard (1985)

CDV, Canine distemper virus; EM, electron microscopy; IFAT, indirect fluorescent antibody test; IHC, immunohistochemistry; MAT, modified agglutination test; NA, not applicable; NGL, no gross lesions; NHL, no histologic lesions; NX, necropsy; PCR, polymerase chain reaction; RFLP, restriction fragment length polymorphism.

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