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Mustelids

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INTRODUCTION

The family Mustelidae, comprising the stoats, polecats, mink, fishers, wolverines, weasels, martens, badgers, and otters, is the largest family within the order Carnivora. Skunks were traditionally considered as mustelids, but recent molecular evidence places them in the family Mephitidae (Nyakatura and Bininda-Emonds, 2012; Sato et al., 2004). Globally, mustelids are free ranging in terrestrial and marine environments, are kept as pets, farmed, used as laboratory animals, and managed in zoological settings. This chapter contains information on more common diseases and syndromes affecting the members of this family.

UNIQUE FEATURES

Based on the available literature, mustelid hematology is similar to other carnivores; a few peculiarities are noted in the chapter on clinical pathology. As mammalian carnivores, the gross anatomy of Mustelidae is “familiar” to most pathologists, with the exception of reniculate kidneys, a peculiarity seen in sea and river otters. Male mustelids have a baculum (os penis) (Nowak, 1991). The tip of the baculum is curled and the urethra is relatively small in some species, making passage of a catheter difficult.

Few unique histologic features are encountered at necropsy in mustelids, or at least that have been described in the literature. Placentation in mustelids is zonary, similar to dogs and cats. Implantation sites in the ferret are presymplasmic, with extremely pleomorphic decidual cells that may be confused with an endometrial carcinoma. Ectopic adrenocortical tissue is a common finding in the abdomen of ferrets and sea otters.

NON-INFECTIOUS DISEASES

Nutritional

Associations between neurologic disease and **thiamine deficiency** in mink were first noted in the 1940s, especially

in mink fed diets containing high levels of thiaminase-containing fish (Okada et al., 1987). Additional reports include one incident in farmed ferrets, and a single captive river otter. However, it is likely that this condition could affect any member of the taxa under the right conditions. The pathogenesis of thiamine deficiency is complex, and likely related to its role as a critical cofactor for production of pyruvate decarboxylase and transketolase, with lesion distribution closely paralleling transketolase activity in the brain. The decreased activity of ATP-dependent sodium and water transport results in the intraneuronal swelling progressing to neuronal necrosis as well as vascular degeneration, edema, and hemorrhage. The gross lesions of thiamine deficiency are similar to those in other carnivores and consist of bilaterally symmetrical hemorrhage and necrosis of the periventricular gray matter, caudal colliculi, and vestibular nuclei. Histologically, vacuolar degeneration and necrosis of neurons (with accompanying gliosis) is most easily seen in nuclei within the lateral geniculate bodies, caudal colliculi, and red nuclei. In one report, lesions were present only in adult male mink but not in adult females or kits (Okada et al., 1987). Cardiac lesions, which occur in the dog and fox, have not been described in mink.

Vitamin E deficiency was first described in farmed mink in the 1940s with intermittent reports in other mustelids (Gorham and Griffiths, 1956). This disease primarily affects healthy young farmed mink in the summer and fall, and may present with no premonitory signs (Gorham and Griffiths, 1956). As an important antioxidant, vitamin E protects cellular membranes against oxidative processes initiated by free radicals, hypovitaminosis E has been associated with diets containing high levels of polyunsaturated fatty acids (improperly stored fish can contain highly unsaturated fish oils). Affected mustelids suffer from steatitis, and skeletal and cardiac muscle degeneration. Mink can also develop microcytic, normochromic anemia (Stowe and Whitehair, 1963). Gross lesions justify the early name associated with this condition, “**yellow fat disease.**” At necropsy, the abdominal skin appears

thickened and doughy, and may exude thin, watery fluid (Ford, 1961). Oxidized abdominal and subcutaneous fat is hardened and yellow-brown, and a fishy odor may permeate the cadaver in severe cases. Histologic lesions include widespread adipocyte necrosis with multifocal saponification, dystrophic mineralization, and mixed suppurative and histiocytic inflammation (Fig. 11.1). In severe cases, entire lobules may be replaced by inflammatory cells whose cytoplasm contains acid-fast positive ceroid pigment. Skeletal and cardiac muscle may exhibit varying degrees of myofiber degeneration, swelling, cytoplasmic vacuolation, fragmentation, contraction band necrosis, and fibrosis, with proliferation of satellite nuclei.

Metabolic

Shallow pinpoint **gastric erosions or bleeding mucosal ulcers** with “coffee-ground” melena are common in mustelids and exceedingly similar to published descriptions of stress-induced gastritis in humans. In affected animals, gastric lesions are grossly visible and are often concentrated in the pyloric mucosa, with an adherent surface coating of dark brown to black, partially digested blood (Fig. 11.2). Histologically, these lesions in sea otters are typically erosions not ulcers and they are characterized by wedge-shaped areas of mucosal coagulation necrosis and hemorrhage, sometimes with one or more tiny fibrin thrombi in subtending blood vessels. When present in the gastric body, these lesions often concentrate along the tips of the rugal folds. In ferrets, ulcers are often concentrated in the pylorus and may result in full thickness ulceration and significant, often fatal bleeds. In all affected mustelids, while rarely serving as a primary cause of death, gastric erosions/ulcers often

contribute to morbidity and mortality through progressive, severe blood loss, and less commonly, acute submucosal vascular rupture. A causal relationship has been proposed for the development of gastric inflammation and erosions/ulcers in older ferrets and sea otters with confirmed *Helicobacter mustelae* and *H. enhydrae* infection, respectively; however, a direct causal link has not been established (Fox et al., 1990; Shen et al., 2017).

Adrenal-associated endocrinopathy (AAE) is a very common metabolic condition in middle aged and older ferrets. In intact ferrets, seasonal secretion of gonadotropin-releasing hormone from the hypothalamus results in secretion of luteinizing hormone and follicle-stimulating hormone from the pituitary gland, which prepares the ovary and testis for reproduction. The routine practice of early castration or ovariectomy causes chronic luteinizing hormone secretion due to loss of negative gonadal feedback, resulting in hyperplastic and potentially neoplastic proliferation of primitive adrenocortical cells in the juxtamedullary region (Bielinska et al., 2006). Proliferative adrenocortical lesions may be observed in animals less than 1 year of age, and range from multiple hyperplastic nodules to infiltrative carcinoma. Corroborative clinical signs include various patterns of truncal alopecia due to the inhibitory effects of excess estrogen secretion on hair follicles, vulvar swelling in spayed females, and a return to sexual behavior in neutered animals of either sex. Aberrant adrenocortical tissue may secrete estrogen, testosterone, 17-hydroxyprogesterone, or other intermediate metabolites; serum tests for these compounds, especially in neutered animals, establish the diagnosis. Excessive cortisol levels are only rarely encountered in ferrets with this condition, so use of the term “Cushing’s disease” is inappropriate.

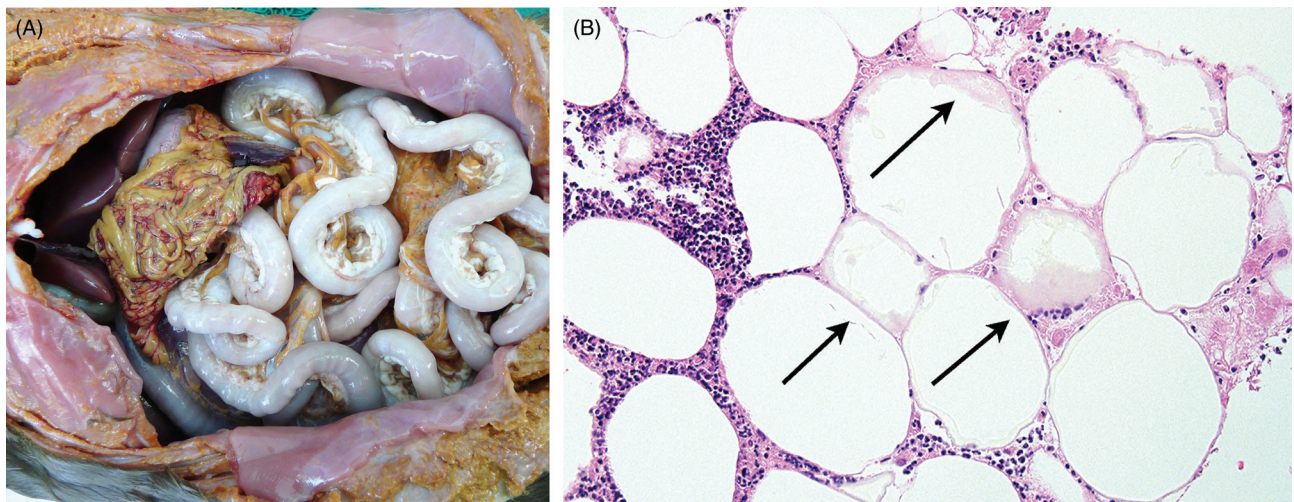


FIGURE 11.1 Steatitis due to hypovitaminosis A. (A) Gross presentation of steatitis and abdominal fat necrosis in a smooth coated otter with vitamin E deficiency. (B) Steatitis within the mesentery of a mink suffering from hypovitaminosis E. The interstices between adipocytes contain lymphocytes, plasma cells, and histiocytes. Several adipocytes exhibit saponification (arrows). (Part A: Photo Courtesy of T. Kasantikul, Zoological Park Organization and Faculty of Veterinary Science, Mahidol University)

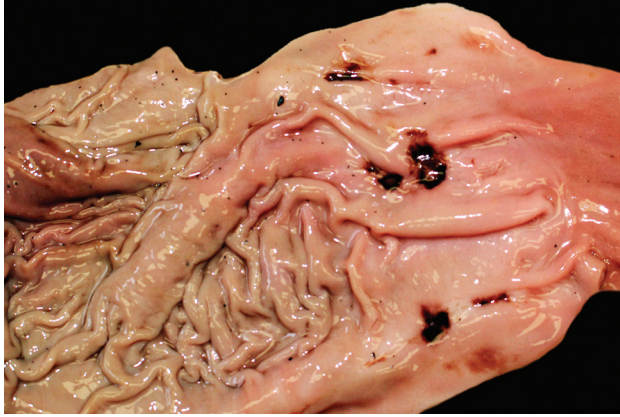


FIGURE 11.2 Multiple gastric ulcers in the pylorus of a sea otter.

appropriate. Precise classification of these tumors is not always possible or necessary; with the exception of a myxoid variant of carcinoma, tumor metastasis is uncommon, and occurs late in disease progression (Peterson et al., 2003). Adrenocortical nodular hyperplasia is also very common in sea otters and appears to be age-related; however, no associated clinical signs or clinical pathology have been reported.

Middle-aged male ferrets suffering from **hyperestrogenism** may develop urinary obstruction following the development of prostatic cysts. The elevated levels of estrogen in the male ferrets results in squamous metaplasia and keratinization of prostatic glandular epithelium and accumulation of purulent material, forming large cysts. Affected individuals cannot void voluntarily, but their enlarged bladders are easily expressed manually. Left untreated, this postrenal obstruction is a life-threatening condition.

Urolithiasis is a well-documented entity in several species of mustelids, especially mink, ferrets, and river otters (Fig. 11.3). The pathophysiology is likely multifactorial, with interplay of metabolic, nutritional, dietary, and genetic factors. In farmed mink, struvite urolithiasis displays clear seasonal patterns, with pregnant females affected in the spring, and male kits affected in the fall. In the gravid female, stones may hinder parturition, resulting in dystocia and death, while in males; urethral obstruction may result from urolith-mediated blockage at the distal os penis (Gorham and Griffiths, 1956). Concurrent urinary tract infection with *Staphylococcus intermedius* is common in affected animals. In a review of 408 uroliths from pet ferrets, 202 (67%) uroliths were struvite, 61 (15%) were cystine, and 43 (11%) were calcium oxalate. Uroliths were detected more commonly in males (73%), prevalence increased with age, and most (77%) uroliths were retrieved from the bladder. Unlike mink, dogs and cats, ferret struvite uroliths were not associated with concomitant bacterial infection (Nwaokorie et al., 2011).

Nephrolithiasis is an especially common and widespread condition among free-ranging and captive otters, and less commonly, wolverines (Table 11.1). The prevalence and chemical composition of nephrolithiasis varies widely between species, with the highest prevalence observed in captive Asian small-clawed otters. As with dogs, cats and humans, older age is a common risk factor for mustelid nephrolithiasis. The high prevalence of nephrolithiasis in free-ranging and captive otter species suggests the possibility of shared heritable metabolic abnormalities.

Toxic

Oil spills are especially devastating to mustelids living in aquatic environments. The Exxon Valdez oil spill in Prince William Sound, Alaska in 1989 was of particular significance to sea otters. Up to 4000 sea otters perished following that single event, and in many areas, recovery remains incomplete. Because of the complex mix of compounds in petroleum products from differing sources, lesions, and clinical signs can vary widely. The most common impact of petroleum spills is pelage contamination, which prevents normal air trapping resulting in severe hypothermia and death.

A broad array of internal and external lesions may be observed, depending on the chemical composition of the product, the extent of postspill weathering, and the exposure dose and route. In general, more refined petroleum products that have been enriched for shorter, flammable petroleum compounds are more acutely toxic, while crude and/or weathered products are more likely to cause death via physical fouling and hypothermia. In addition to grossly apparent surface contamination, associated gross lesions include skin, ocular and oral chemical burns, physical obstruction of the nares and mouth, chemical gastroenteritis and pneumonia, interstitial emphysema, and secondary stress-related gastric ulceration and melena (Lipscomb et al., 1993). Histologic lesions may include centrilobular hepatic necrosis, and lipidosis of periportal hepatocytes and renal tubules. Nonspecific neurological, endocrine and reproductive impacts, bone marrow suppression, and tumor induction have also been recognized postexposure in mustelids (Lipscomb et al., 1993), other animals and humans.

Anticoagulant rodenticide poisoning has emerged as a significant concern for conservation and management of non-target wildlife, including mustelids. In a study in California (US), 46 of 58 (79%) tested free-ranging fishers were positive for anticoagulants, and 96% had been exposed to one or more second-generation anticoagulant compounds (Gabriel et al., 2012). Fisher deaths, including a lactating female, were directly attributed to anticoagulant toxicosis, and transplacental and/or lactational transfer was demonstrated in a fisher kit. At necropsy, affected animals were in variable nutritional condition with multifocal areas of hemorrhage and/or frank

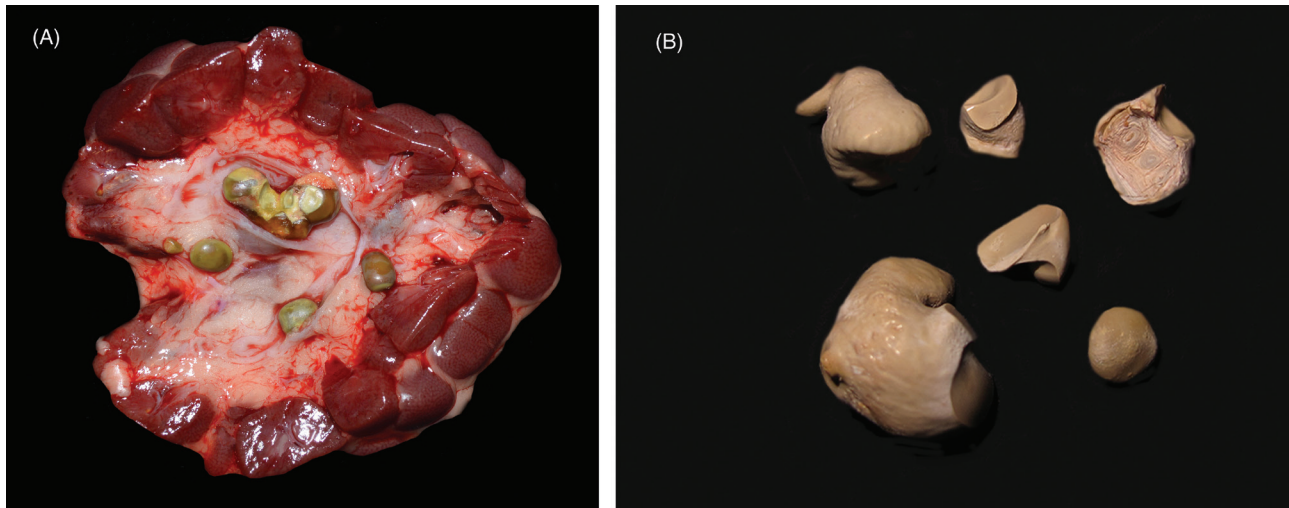


FIGURE 11.3 Urolithiasis in mustelids. (A) Uroliths in situ in an Asian small clawed otter. (B) Uroliths from the urinary bladder of a Cape clawless otter. (Part A: Photo Courtesy of K. Terio, University of Illinois Zoological Pathology Program; Part B: Photo Courtesy of B. Rideout, Disease Investigations, San Diego Zoo Global)

TABLE 11.1 Urolithiasis in Otters and Wolverines

Mustelid sp.	Captive or Free Ranging	Stone Type	Reported Prevalence	Associated Lesions	Possible Risk Factors
Asian small clawed otter ^a	Captive	Mix of urate and calcium oxalate	66%–89%	Not reported	Older age (>2 years)
Eurasian otter ^b	Free ranging	Ammonium acid urate	10.2%	Nephrolithiasis	Dietary purine intake, protein quality, and digestibility
North American river otter ^c	Free ranging	Calcium phosphate	16.2%	Not reported	Older age, capture location
North American river otter ^d	Free ranging	Uric acid	0.33%	Bilateral nephrolithiasis, expanded renal calyces, marked hydroureter, mild renal medullary loss, and cortical tubular atrophy	Not reported
Wolverine ^e	Free ranging	Ammonium acid urate with magnesium ammonium phosphate and calcium phosphate	8.9%	Nephroliths were unilateral in 87.5% of cases.	Older age (>2 years) males

^aPetrini, K.R., Lulich, J.P., Treschel, L., Nachreiner, R.F., 1999. Evaluation of urinary and serum metabolites in Asian small-clawed otters (*Aonyx cinerea*) with calcium oxalate urolithiasis. *J. Zoo Wildl. Med.* 30, 54–63.

^bSimpson, V., Tomlinson, A., Molenaar, F., Lawson, B., Rogers, K., 2011. Renal calculi in wild Eurasian otters (*Lutra lutra*) in England. *Vet. Rec.* 169, 49.

^cNiemuth, J.N., Sanders, C.W., Mooney, C.B., Offenbittel, C., DePerno, C.S., Stoskopf, M.K., 2014. Nephrolithiasis in free-ranging North American river otter (*Lontra canadensis*) in North Carolina, USA. *J. Zoo Wildl. Med.* 45, 110–117.

^dGrove, R.A., Bildfell, R., Henny, C.J., Buhler, D.R., 2003. Bilateral uric acid nephrolithiasis and ureteral hypertrophy in a free-ranging river otter (*Lontra canadensis*). *J. Wildl. Dis.* 39, 914–917.

^eOakley, M.P., Jung, T.S., Kukka, P.M., Robitaille, J.-F., 2016. Prevalence of renal calculi in wolverine (*Gulo gulo*) from northwestern Canada. *Mammal. Biol. Zeitschrift für Säugetierkunde* 81, 189–193.

blood within the thoracic and abdominal cavities. Anticoagulant exposure and/or poisoning has also been reported in free-ranging European and American mink and European otters in France, skunks from New York, stoats and weasels in New Zealand, and badgers from California (Alterio, 1996;

Fournier-Chambillon et al., 2014; Quinn et al., 2012; Stone et al., 1999).

Microcystin, a potent cyanotoxin, was identified as a cause of icterus and massive hepatic necrosis for sea otters in central California (US). Livers from affected animals

were swollen, friable and hemorrhagic, and histologic lesions included hepatocellular necrosis, apoptosis, hepatocellular vacuolation, and hemorrhage (Miller et al., 2010).

Domoic acid (DA) intoxication is postulated as a cause of toxic cardiomyopathy and heart failure in southern sea otters (Kreuder et al., 2005). Based on multivariate statistical analyses, domoic acid exposure appears to be a significant risk factor for myocarditis and dilated cardiomyopathy in sea otters (Kreuder et al., 2005). DA and shigatoxin exposure without associated pathology has been observed in Alaskan sea otters (Lefebvre et al., 2016).

Botulism is a serious food-borne illness of ranched mink. Feeding processed feeds or offal containing high levels of **type C botulinum toxin** has been implicated in numerous outbreaks in multiple countries, each of which affected hundreds or thousands of animals (Lindström et al., 2004). Extensive mortality of unvaccinated mink can occur within 18–96 h of consuming toxin-laden food characterized by acute muscular incoordination and stiffness, progressing to limb paralysis, and culminating in respiratory paralysis and death (Gorham and Griffiths, 1956). PCR assays are currently available to identify *Clostridium botulinum* type C bacteria and the type C botulinum toxin. Ferret deaths from botulism have also been reported.

Congenital/Genetic

A variety of putative congenital diseases occur sporadically in diverse mustelid species (Table 11.2).

Cardiomyopathy is a common finding in ferrets and has recently been identified in striped skunks in captive collections (Benato et al., 2014). This finding is commonly seen in certain bloodlines of pet ferrets. In the domestic ferret, it is the most common cause of cardiac disease and may be seen in ferrets ranging from 6 months to geriatric individuals. Both dilatative and hypertrophic forms have been identified in this species; dilatative forms being more common. Thoracic and abdominal effusions may be seen in these cases.

The classic histologic lesion associated with cardiomyopathy is myofiber loss and fibrosis. Mild lesions are often first identified in perivascular areas throughout the ventricles, and to a lesser extent, the interventricular septum. Foci of myocardial fibrosis consistent with cardiomyopathy are seen in increasing prevalence in older animals that often show little or no outward evidence of cardiac disease; the degree of fibrosis often does not closely correlate with clinical impairment. Concurrent evidence of cardiac insufficiency, such as chronic hepatic congestion, pulmonary siderosis, or effusion is helpful in corroborating the histologic diagnosis of cardiomyopathy.

Age-related/Degenerative

Age-related pathology is common in mustelids. These include **periodontal disease** (Fig. 11.4), **degenerative**

joint disease, old, healed **bone fractures**, and rarely, **intervertebral disk disease**. For male southern sea otters, healed baculum fractures and fractures or luxations of the digits of the hind feet are relatively common sequela to a lifetime of territorial fighting.

Age-related ocular disease has been described in ferrets and mink. **Cataracts** are common in older ferrets, and several studies have described incidence in genetically distinct laboratory populations. Persistent fetal vasculature has been implicated as a cause in young ferrets, but in most animals, cataracts develop in middle age and progress over time (Lipsitz et al., 2001). **Central retinal degeneration** is also relatively common in older ferrets and mink. It is characterized by degeneration of rods, cones, and outer retinal layers (Hadlow, 1984). In ferrets, retinal degeneration may accompany cataract formation. Older ferrets may also spontaneously develop glaucoma with buphthalmia. Chronic corneal edema is seen in pastel mink, resulting from **spontaneous degeneration of the corneal endothelium**. The condition is persistent, bilateral, and is rarely associated with ulceration (Hadlow, 1987).

A variety of presumed congenital **cystic urogenital lesions** have been described from older ferrets, as they require time to grow to a size where they are detectable (Li et al., 1996). Urethral gland distention and cyst formation occurs in older female mink, leading to pressure atrophy and urethral wall degeneration (Hadlow and Race, 1981). Predisposing causes are unknown.

Trauma

In free-ranging mustelids, trauma is the most common cause of mortality, with **traffic deaths** and **ballistic trauma** being most prevalent for terrestrial species, and **interspecific predation** as a common cause of traumatic death for sea otters. A recent study of small mustelids in the United Kingdom identified vehicular trauma was the most common cause of death for stoats and polecats, while domestic cat predation exceeded deaths from vehicular trauma by almost 3:1 for weasels (Simpson et al., 2016). Common predators for black-footed ferrets include coyotes, owls, and badgers. Entrapment in fishing line, hooks, nets, and traps used to catch crabs or fish is a cause of death for some riverine and oceanic mustelids. Predation of sea otters by great white sharks and killer whales has been incriminated as a cause for the declines in Southern and Northern sea otters, respectively (Doroff et al., 2003; Tinker et al., 2015).

Inflammatory Non-infectious

Cardiomyopathy is a common finding in ferrets and southern sea otters, and was recently reported in captive striped skunks (Benato et al., 2014). This syndrome first appears in ferrets that are ≥ 6 months, with increasing prevalence in geriatric animals. Both dilatative and hypertrophic forms have been

TABLE 11.2 Congenital Diseases in Mustelids

Condition	Species Affected	Lethal	Lesions	Predisposing Factors
Neural tube defect ^a	Ferret	Yes	Spina bifida, cranioschisis, anencephaly, inencephaly	Color dilution
Deafness ^b	Ferret	No	Deafness which parallels symmetry of albino coloration	Color dilution
Polycystic kidney ^c	Mink	Yes	Cysts in collecting ducts, all kits died within 4 months.	None reported
Mullerian duct cysts ^d	Otters	No	Mullerian duct cysts identified on the serosa of the spermatic duct (72%)	None reported
Ehlers-Danlos syndrome ^e	Mink	No	Skin fragility, hyperextensibility, laxity	Autosomal recessive
Hydrocephalus ^{f,g}	Mink	Yes	Cranial distention, incoordination, severe lack of growth. Kits died before 6 weeks	None reported
Tyrosinemia ^h	Mink	No	Hyperkeratosis, hemorrhagic pododermatitis, corneal edema, granulomatous nephritis	Autosomal recessive—lack of hepatic tyrosine aminotransferase
Chediak-Higashi syndrome ⁱ	Mink	No	Abnormal storage granules in leukocytes, platelets, hepatocytes, renal tubular epithelium, neurons, and endothelial cells. Partial oculocutaneous hypopigmentation due to clumping of melanocyte granules. Impaired NK function, peripheral neuropathy	Color dilution, frameshift mutation in <i>LYST</i> gene
Retinal dysplasia ^j	Otter	No	Retinal folding, rosette formation, and detachment	Decreased Vitamin A, increased dieldrin

^aWilliams, B.H., Pope, E.J., Hart, R.A., Harris, R.K., 1994. Inencephaly and other neural tube defects in a litter of ferrets (*Mustela putorius furo*). *Vet. Pathol.* 31, 260–262.

^bPiazza S., Abitbol M., Gnirs K., Huynh M., Cauzinille L., 2014. Prevalence of deafness in association with coat variations in client-owned ferrets. *J. Am. Vet. Med. Assoc.* 244, 1047–1052.

^cHenriksen P., 1988. Polycystic disease of the kidney in related mink. *J. Comp. Pathol.* 99, 101–104.

^dRoos, A.M., Agren, E.O., 2013. High prevalence of proposed Müllerian duct remnant cysts on the spermatic duct in wild Eurasian otters from Sweden. *PLOS One* 8, 384660.

^eHegreberg, G.A., Padgett, G.A., Ott, R.L., Henson J.B., 1970. A heritable connective tissue disease of dogs and mink resembling Ehlers-Danlos syndrome of man. *Skin tensile strength properties.* *J. Invest. Dermatol.* 54, 377–380.

^fCorham, J.R., Griffiths, H.J., 1956. *Diseases and parasites of minks.* US Department of Agriculture.

^gStowe, H.D., Whitehair, C., 1963. Gross and microscopic pathology of tocopherol-deficient mink. *J. Nutr.* 81, 287–300.

^hSanford, S.E., 1988. Ontario. Tyrosinemia II (Pseudodistemper) in mink. *Can. Vet. J.* 29, 298.

ⁱAnistoroaei, R., Krogh, A., Christensen, K., 2013. A frameshift mutation in the *LYST* gene is responsible for the Aleutian color and the associated Chediak-Higashi syndrome in American mink. *Anim. Genet.* 44, 178–183.

^jWilliams, D., Simpson, V., Flindall, A., 2004. Retinal dysplasia in wild otters (*Lutra lutra*). *Vet. Rec.* 155, 52–56.



FIGURE 11.4 Extensive tooth wear with pulp exposure and tooth loss in an aged Northern sea otter.

identified in ferrets, but dilative disease is more common. Thoracic and abdominal effusion may be present, and histological lesions include myofiber loss and fibrosis. Mild lesions are often first identified in ventricular perivascular regions and the interventricular septum. The extent of fibrosis on histopathology may not correspond with the severity of clinical signs. In southern sea otters, cardiomyopathy was identified as the third most common primary cause of death in one review of mortality patterns in free-ranging animals (Kreuder et al., 2005). In contrast, this condition is uncommon in Alaskan sea otters. Grossly, affected animals exhibit variably dilated, orange/white-mottled myocardium, often with evidence of left and/or right heart failure (Fig. 11.5). Dilated cardiomyopathy (DCM) is presumed to be the end stage of this chronic condition in animals that have survived for longer periods. However, as with ferrets, the extent of myocardial dilation, inflammation, and fibrosis may

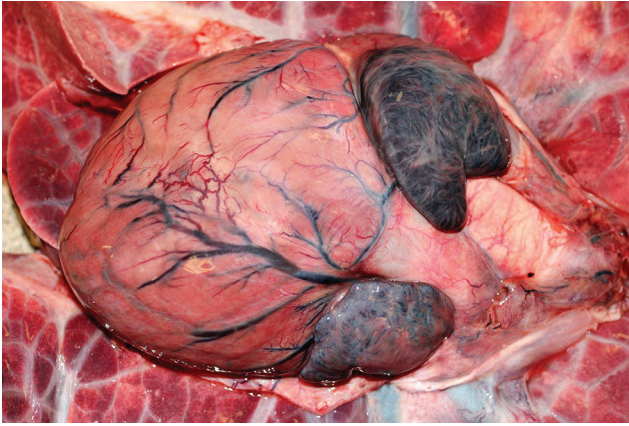


FIGURE 11.5 Cardiomyopathy in a Northern sea otter. Biventricular pallor as well as dilation of the right ventricle are present.

not always correspond with the severity of clinical diseases. On histopathology, sea otter cardiomyopathy is characterized by mild to severe lymphocytic to pleocellular myocarditis with progressive myofiber loss and replacement fibrosis. In a prior epidemiological study, exposure to *Sarcocystis neurona* and DA were significant risk factors for myocarditis in sea otters, but only DA exposure was a risk factor for DCM (Kreuder et al., 2005). The lesions associated with sea otter cardiomyopathy are similar to DA-associated cardiac disease of California sea lions (See Chapter 23).

A number of inflammatory diseases that are suspected to have an underlying **immune-associated** etiopathogenesis have been reported over the past 25 years in ferrets. Of these, **inflammatory bowel disease** is common in middle-aged and older ferrets. It is characterized by lymphocyte infiltration, epithelial damage, and marked villous blunting and fusion. **Eosinophilic gastroenteritis** is associated with systemic hypereosinophilia, and inflammatory damage to the gastrointestinal tract, abdomen, and thoracic cavity. Eosinophil aggregates within lymph nodes and various organs may produce Splendore-Hoeppli material, and abundant eosinophils are apparent in the vasculature of affected animals. Within the last decade, a syndrome of **idiopathic systemic myofasciitis and/or myositis** has been identified. Characteristic findings include severe pyrexia and neutrophilia, and infiltration of skeletal muscles (almost always including the esophagus) by abundant neutrophils in the absence of demonstrable infectious agents (Garner et al., 2007a). Rarely, **erythema multiforme** and other autoimmune skin diseases have been identified in domestic ferrets.

Masticatory myositis of presumed immune-mediated etiology has been documented in mink. The condition is characterized by myodegeneration and myonecrosis of the temporal and masseter muscles with a prominent eosinophilic infiltrate and diffuse subcutaneous edema over the dorsal head (Needle et al., 2014). Mink masticatory muscles also possess unique type 2M fibers, suggesting a similar pathogenesis to canine masticatory myositis.

Systemic or regional **amyloidosis** has been identified in numerous mustelids, but documentation is most complete in mink in which serum amyloid A (SAA) protein has been sequenced. In one review, 24% of 68 mink >3 years old were positive for amyloid, compared with 3% prevalence for 68 mink < 3 years. SAA is most common in the cervical salivary gland interstitium, but is also detected in liver, spleen kidney, heart, adrenal, tongue, stomach, intestine, pituitary, and pancreas (Schwartz et al., 1971). A study of black-footed ferrets from eight US zoological institutions revealed SAA in the renal glomeruli and other tissues in association with proteinuria and emaciation (Garner et al., 2007b). Amyloidosis has also reported in a skunk, beech marten, badger, and Eurasian otter (Elhensheri et al., 2012; Jakob, 1971; Scaglione et al., 2013).

Hemorrhagic enteropathy is frequently identified in river and sea otters. Histologically, this condition is characterized by prominent blood pooling in the intestinal mucosa and submucosa, with minimal damage to the mucosal epithelium; associated bacterial infection has not been observed (Lauckner, 1985). A similar syndrome has been identified in oiled river otters. Considering the absence of opportunistic pathogens in such cases, underlying stress, shock, and/or cardiac insufficiency should be considered, depending upon case findings.

Neoplastic

Neoplasms are common in mustelids, and parallel the breadth and variety seen in mammalian domestic species. Available literature suggests that tumor prevalence varies, even between closely related species, but this may be in part due to incomplete data. There have been extensive reviews of neoplasia in some species, especially the ferret, but for many free-ranging mustelids, reports of neoplasia are scant and consist of only single cases. Fig. 11.6 is an illustration of the classical nature of neoplasia presentation in mustelids. Table 11.3 includes a list of a number of commonly reported tumors in several species of mustelids.

INFECTIOUS DISEASES

DNA Viruses

Multiple **herpes** viruses infect mustelids. **Pseudorabies** has been identified as a causative agent of neurologic disease in mink. The incubation period is generally is 3–4 days, and clinical signs include hypersalivation, vomiting, depression, and coma. Microscopically, pseudorabies infection in mink is characterized by fibrinoid degeneration of vessels in the central nervous system (CNS), myocardium, and oropharynx. This differs from the nonsuppurative encephalitis typical of other species (Quiroga et al., 1997). **Herpes simplex** has been identified as the cause of nonsuppurative and necrotizing

encephalitis with neuronal and glial inclusions in a striped skunk; experimental intravenous inoculation of additional skunks resulted in systemic necrosis that was most severe in the liver and adrenal glands (Charlton et al., 1977).

Mustelid herpesvirus-1 in a male fisher caused dermal ulcers on the muzzle and plantar pads (Gagnon et al., 2011). Histologically, the border of the ulcers contained clusters of cells with basophilic to amphophilic nuclear inclusions; a pan-herpesvirus nested PCR polymerase assay and DNA sequencing confirmed the infection. Mustelid herpesvirus-1 was also identified from fibroblast cultures, and viral-specific IgG was isolated from wild badgers in the British Isles (King et al., 2004). In 2012, a novel **mustelid herpesvirus-2** was identified in oral ulcers from northern sea otters stranding

during the 1989 Exxon Valdez spill (Tseng et al., 2012). This virus was subsequently isolated in oronasal secretions from apparently healthy northern sea otters.

Multifocal raised, variably pigmented **papillomas** have been reported in the gingiva and lips of southern sea otters infected with **Enhydra lutris papillomavirus-1 (EIPV-1)** (Ng et al., 2015) (Fig. 11.7). Microscopically, koilocytes and intranuclear inclusions were apparent in the mucosal stratum granulosum and spinosum (Figs. 11.8 and 11.9). Immunohistochemical staining using antibodies raised to bovine papilloma virus revealed EIPV-1 labeling in both intranuclear inclusions and intracytoplasmic keratin granules, and virus-infected cells were scattered throughout the stratum granulosum and stratum spinosum.

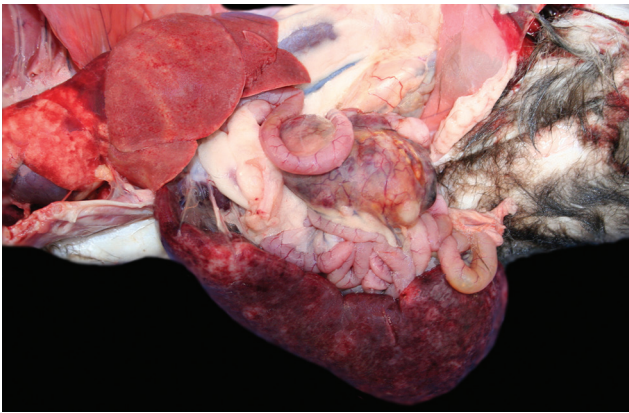


FIGURE 11.6 Lymphoma in a ferret. The enlarged spleen and mesenteric lymph nodes demonstrate the “classical” presentation of this neoplasm. (Photo Courtesy of F. Del Piero, Louisiana State University)

RNA Viruses

At least two **morbillivirus** (family *Paramyxoviridae*) strains have been implicated in mustelid infections worldwide. These are **canine distemper virus (CDV)** and a morbillivirus similar to phocine distemper virus (PDV) in northern sea otters; CDV is more prominent and clinically significant (Goldstein et al., 2009; Mos et al., 2003; Pavlacik et al., 2007; Tavernier et al., 2012). Susceptibility and case fatality rates vary among species, with black-footed ferrets being very sensitive, and striped skunks comparatively resistant (Williams et al., 1988). Veterinarians must remain vigilant for distemper outbreaks following modified live virus (MLV) vaccination of wildlife (e.g., black-footed ferrets), as live vaccines developed for use in dogs and cats can cause fatal disease in other species (Carpenter et al., 1976).

TABLE 11.3 Common Neoplasms in Mustelids

Species	Neoplasm
Ferret ^a	Islet cell tumor; adrenocortical hyperplasia, adenoma, and adenocarcinoma; cutaneous basal cell tumors, mast cell tumor, apocrine adenomas and adenocarcinoma, chordoma
Black-footed ferret ^b	Apocrine gland tumors, renal tubular neoplasms, biliary cystadenoma, and carcinoma
Mink ^{c,d}	Anal sac carcinoma, carotid body tumor
Skunk ^e	Hodgkin's like lymphoma
Siberian weasel ^f	Fibrosarcoma, interstitial cell tumor
Sea otter ^{g,h}	Soft tissue sarcomas, uterine and cervical leiomyomas, lymphoma

^aWilliams, B.H., Wyre, N.R., 2017. Neoplasia. In: *Ferrets, Rabbits, and Rodents: Clinical Medicine and Surgery*. Elsevier, Philadelphia.

^bLair, S., Barker, I.K., Mehren, K.G., Williams, E.S., 2002. Epidemiology of neoplasia in captive black-footed ferrets (*Mustela nigripes*), 1986–1996. *J. Zoo Wildl. Med.* 33, 204–213.

^cHadlow, W.J., 1985. Carcinoma of the anal sac glands in ranch mink. *Vet. Pathol.* 22, 206–218.

^dHadlow, W.J., 1986. Carotid body tumor: an incidental finding in older ranch mink. *Vet. Pathol.* 23, 162–169.

^eSmith, D., Barker, I., 1983. Four cases of Hodgkin's disease in striped skunks (*Mephitis mephitis*). *Vet. Pathol.* 20, 223–229.

^fZöller, M., Aeffner, F., Müller, J., Mätz-Rensing, K., Kaup, F.-J., 2008. Neoplasia in the Siberian weasel (*Mustela sibirica*): two case reports of fibrosarcoma and interstitial cell tumour. *Eur. J. Wildl. Res.* 54, 15–20.

^gNewman, S., Smith, S., 2006. Marine mammal neoplasia: a review. *Vet. Pathol.* 43, 865–880.

^hBurek-Huntington, K.A., Mulcahy, D.M., Doroff, A.M., Johnson, T.O., 2012. Sarcomas in three free-ranging Northern sea otters (*Enhydra lutris kenyoni*) in Alaska. *J. Wildl. Dis.* 48, 483–487.



FIGURE 11.7 Gingival papillomas in a sea otter. Clusters of white raised plaques on the gingiva and lips are characteristic of *Enhydra lutris papillomavirus-1* infection.

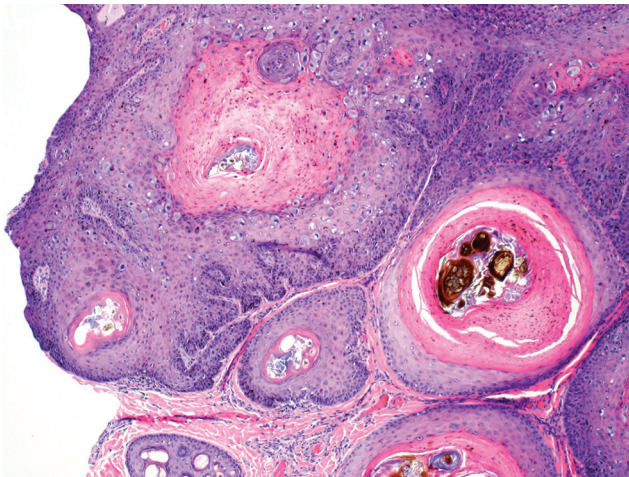


FIGURE 11.8 Oral mucosa of a sea otter infected with *Enhydra lutris papillomavirus-1*. Clusters of koilocytes and intracellular edema are present in oral papillomas in sea otters.

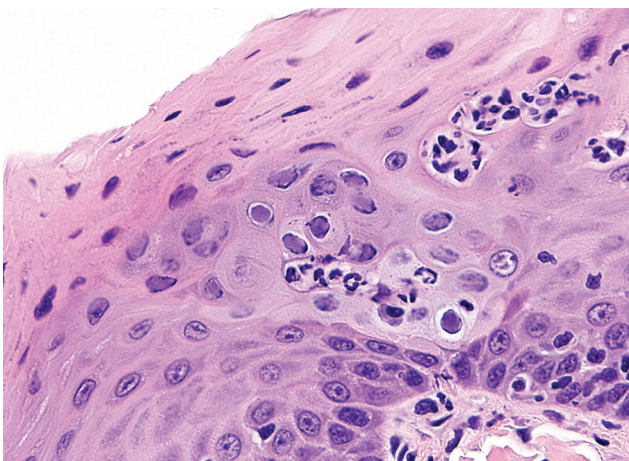


FIGURE 11.9 Oral mucosa of a sea otter infected with *Enhydra lutris papillomavirus-1*. There are characteristic intranuclear viral inclusions.



FIGURE 11.10 Marked hyperkeratotic of the footpads and periocular skin of a skunk infected with canine morbillivirus. (Photo Courtesy of R. Ossiboff, Cornell University)

Clinical signs of CDV in mustelids are similar to those in canids and include anorexia, oculonasal mucopurulent discharge, multifocal dermal hyperkeratosis (Fig. 11.10), emaciation, severe pruritis, intermittent diarrhea, pneumonia, and rapid death (Mos et al., 2003; Williams et al., 1988). Clinical signs in mustelids often progress faster than in dogs, with ferret-adapted strains causing death in as little as 12 days. Microscopic lesions include bronchointerstitial pneumonia, alveolar syncytia, mild to moderate meningoencephalitis, and variable numbers of intracytoplasmic and intranuclear, eosinophilic viral inclusions in epithelial and syncytial cells as well as neurons and glial cells. Due to the rapid disease course, sick animals may be seronegative for CDV (Mos et al., 2003; Williams et al., 1988). CDV immunohistochemistry and PCR are available in most of the diagnostic laboratories. Concurrent infections are common secondary to CDV-associated immunosuppression (Kubiski et al., 2016). Although a prior serological survey found no evidence of CDV infection in Alaskan and California sea otters, CDV-associated mortality has been reported for a northern sea otter from Washington State (Hanni et al., 2003; Lance et al., 2004). Fatal CDV cases have been reported in fisher (Keller et al., 2012).

A second morbillivirus, similar to PDV was detected by PCR in northern sea otters but association with clinical disease was not established. PDV seroprevalance was as high as 41% in sea otters from some areas of coastal Alaska.

Although reports are sparse for some species, all mustelids are presumed to be susceptible to rabies virus infection. In many parts of the world, mustelids serve as important reservoirs for host-adapted rabies viral strains, especially striped skunks in the United States, and ferret badgers in Southeast Asia (Chang et al., 2015). Based on anecdotal reports, river otters are commonly rabies-positive in some eastern US state rabies monitoring programs. However,

many of these cases are not reported in the peer-reviewed literature, which leads to the misperception of rabies being uncommon in the species. In most cases, affected animals were described as unusually aggressive or tame, and often bit or approached humans prior to euthanasia and rabies testing. In one report from Massachusetts (US), 3 of 8 river otters submitted for testing were rabies-positive, while a published review of historical cases revealed 24 confirmed cases in US river otters from 1971 to 1994. Rabies was also diagnosed in a river otter intended for a reintroduction program, illustrating the importance of pathogen screening in wildlife transportation and release programs (Serfass et al., 1995). Vaccination has dramatically reduced case prevalence in zoos; any unexpected behavioral signs or sudden death, especially in unvaccinated animals, necessitates consideration of rabies, in part due to significant human health risk.

Influenza viruses are zoonotic pathogens with a broad host range that includes canids, horses, marine mammals, and mustelids. Ferrets are susceptible to both Type A and B influenza stains, and are commonly used as models for influenza research (Zitzow et al., 2002). Free-ranging striped skunks and ranched mink have been implicated as potential conduits for influenza viral amplification and spread from infected humans and other animals. Based on the necropsy findings and experimental infection, both groups may shed influenza virus for extended periods, facilitating transmission (Britton et al., 2010; Englund et al., 1986; Root et al., 2014). Clinical signs of influenza in mustelids are similar to humans: malaise, serous nasal discharge, and lower respiratory tract disease due to viral infection and/or secondary, opportunistic bacterial infections. In free-ranging skunks infected with highly pathogenic H1N1 influenza virus, lesions and clinical signs included purulent nasal exudate, splenomegaly and severe pneumonia characterized by heavy, dark red to purple lungs. Microscopic examination revealed moderate rhinitis and severe bronchointerstitial pneumonia (Britton et al., 2010). Influenza A encephalitis has also been reported in a stone martin that had diffuse nonsuppurative panencephalitis with perivascular cuffing, multifocal gliosis, neuronal necrosis, and focal necrosis of pancreatic islet cells (Klopffleisch et al., 2007). Both northern and southern sea otters have been reported as seropositive for influenza A infection, but associated clinical disease or pathology was not seen (Li et al., 2014).

Numerous **enteric viruses** have been associated with diarrhea in mustelids. **Rotaviruses** have been identified in young ferrets and mink, with group A and C strains infecting ferret kits (Wise et al., 2009). Prewaning diarrhea syndrome of mink kits in Europe has been purported to be caused by mink **calicivirus** or **astrovirus** (Hammer et al., 2012). **Coronaviruses** are best described in the mink and ferret, where they can be clinically silent, or can cause

epizootics characterized by rapid spread of diarrhea through naive colonies. The associated clinical disease is called **epizootic catarrhal gastritis** in mink, and **epizootic catarrhal enteritis (ECE)** in ferrets. Morbidity can be up to 100% in naive individuals, but overall mortality tends to be low. Viral cytopathic effects are most severe for enterocytes at the tips of intestinal villi, resulting in marked villous blunting and fusion, followed by lymphocytic infiltration into the subtending lamina propria. In ferrets, these lesions may be apparent up to 8 months postinfection. Feline coronavirus anti-gp70 antibody successfully cross-reacts with ferret coronavirus; immunohistochemical staining is readily demonstrated within villous epithelium, although it wanes with time. In ferrets, a fatal **feline infectious peritonitis (FIP)-like syndrome** was recently correlated with ferret enteric coronavirus infection. While unproven, the pathophysiology of feline enteric coronaviral infection characterized by viral mutation within infected individuals that facilitates persistence within macrophages is likely for ferrets as well. Affected ferrets develop multifocal granulomas, especially in the spleen, liver, and lymph nodes that contain ferret coronavirus (FeCov) antigen. The majority of cases resemble the “dry form” of FIP, with fewer cases resembling the “wet” form (Garner et al., 2008).

Aleutian mink disease (AMD) is an extremely important condition caused by **parvovirus** infection of mink; infection is also reported in ferrets, free-ranging otters, and skunks. AMD antibodies have been detected in mink, ferrets, otters, weasels, fisher, martens, raccoons, polecats, genets, and foxes (Allender et al., 2008). All color phases of mink may be infected, but color-dilute varieties genetically related to Aleutian strains are especially susceptible. Clinical infection is often associated with high antibody titers. Over months to years, chronic infection can produce high levels of circulating antigen-antibody complexes that are deposited in host basement membranes and cause systemic arteritis and glomerulonephritis. Infected mink often have lymphoplasmacytic hepatitis and nephritis, and are especially susceptible to death from glomerulonephritis. Multifocal hemorrhage may occur due to vascular damage, thrombocytopenia, and coagulation disorders related to severe monoclonal gammopathy; acute, severe hematuria may distend the bladder, resulting in a “cherry-like” appearance. Striped skunks often present differently, with lymphoplasmacytic nephritis, hepatitis, perivascularitis, and neuronal degeneration, but without glomerulonephritis. Counter-immunoelectrophoresis (CIEP) testing remains the gold standard for AMD diagnosis, especially in large, captive facilities, although ELISA tests are available. The clinical course of AMD in Aleutian strains of mink may be as rapid as 4 months, with environmental stress often accelerating disease progression. For other color strains and ferrets, a 2-year subclinical (and potentially infectious) phase is common.

Mink viral enteritis is caused by a parvovirus closely related to feline panleukopenia virus. A recent report identified this disease as the third most common cause of death in ranched mink (Wilson et al., 2015). Disease presentation is similar in cats and mink, with mink enteritis virus causing necrosis of crypt epithelium, lymphocytes, and leucocyte progenitors in bone marrow. As with feline panleukopenia, younger animals are more severely affected. Asymptomatic carrier mink can excrete virus over a year postinfection (Bouillant and Hanson, 1965). Transspecies infection may also occur; with mink susceptible to feline panleukopenia virus, and raccoons susceptible to mink virus enteritis (Barker et al., 1983). Vaccination is protective and is typically administered to kits at approximately 6 weeks of age.

Canine parvovirus-2c infection was identified in a zoo collection of Asian small-clawed otters. Infection resulted in inappetence, lethargy, vomiting, and diarrhea, and one fatality (Gjeltsema et al., 2015). An endemic parvovirus has been identified in southern sea otters but no disease association has been established (Siqueira et al., 2017).

Astrovirus infection has been identified as the cause of a neurological condition in Scandinavian mink known as **shaking mink** (Blomström et al., 2010). Infected animals exhibit shaking, ataxia, and a staggering gait in association with nonsuppurative encephalomyelitis. Brain homogenates from infected mink produced similar signs when inoculated into healthy animals; metagenomic screening identified the causative agent as an astrovirus. Astroviruses are also associated with neurological disease in cattle and humans.

Infectious canine hepatitis, an **adenovirus**, has been identified in striped skunks and a captive Eurasian river otter, resulting in fatal hepatitis (Karstad et al., 1975; Park et al., 2007).

Bacteria

Mycobacteria are common pathogens in zoo settings; infection is also documented in free-living mustelids. Feral ferrets in New Zealand are recognized reservoirs of bovine tuberculosis, and while they can amplify and transmit the disease to conspecifics and cattle, the rate of ferret/bovine transmission is unknown (Byrom et al., 2015). A similar situation exists for wild badgers in the United Kingdom, where vaccination and culling are employed to minimize livestock transmission. *Mycobacterium bovis* infections in free-ranging mustelids (and an OIE reportable disease in cattle) may not be as profound as for other species; caseating granulomas develop but infections may be mild or limited to microgranulomas that can be easily missed during gross necropsy. In contrast to badgers, in which pulmonary granulomas dominate, hepatic granulomas (and microgranulomas) are the most common lesions in ferrets; lymph node granulomas are common in both species (Pollack, 2012). *M. bovis* has also been reported in multiple species of zoo and

farmed mustelids, where transmission is enhanced due to chronic fecal shedding by other exhibited animals, or when fur farms feed potentially contaminated feedstuffs.

Numerous subspecies of **atypical mycobacteria** (*M. avium*) have been detected in mustelid species, including domestic ferrets (Pollack, 2012). *M. celatum* infection is associated with focal or disseminated granulomas in pet ferrets (Pollack, 2012). *M. avium* infection is also reported in Eurasian badgers in Spain and the UK. *M. kumamotoense* and *M. avium* subsp. *avium* infections have been reported in stoats and weasels in the United Kingdom (Simpson et al., 2016). In Portugal, several badgers, Eurasian otters, and beech martens killed by vehicles were positive for *M. avium* subspecies *paratuberculosis*. This bacterium has also been detected in North American skunks (Corn et al., 2005).

An outbreak of *Clostridium perfringens* type A resulted in mortality in young black-footed ferrets, and was attributed to dietary change and overeating (Schulman et al., 1993). Affected animals displayed marked gastric distention and dyspnea, and robust Gram-positive bacilli lined the necrotic gastric mucosa on histology. A second instance of gastric dilatation killed two adult black-footed ferrets at the same institution, but no bacteria were recovered (Bronson et al., 2007).

Streptococcal infections have been reported in several mustelid species, often as part of mixed bacterial flora infecting wounds and bite injuries. Lancefield group C *S. zooepidemicus* infection has been associated with valvular endocarditis in ferrets, and *S. bovis* has been detected in mink (Pedersen et al., 2003). Because distinct bacterial species can have overlapping biochemical properties (e.g., *S. bovis* and *S. lutetiensis* sp. nov. described below), molecular characterization is advised. Numerous cases of bacterial discospondylitis have been reported from mink farms; *Streptococcus* sp. is most commonly isolated from these lesions. Affected animals exhibit posterior paresis associated with suppurative intervertebral discospondylitis and myelitis, and vertebral lysis or proliferation.

Streptococcus lutetiensis sp. nov., a member of the *S. bovis/equinus* group, was formerly identified as *S. infantarius* subsp. *coli* in infected Northern sea otters (Hinse et al., 2011; Poyart et al., 2002). This bacterium was the major cause of death in 30% of Northern sea otters in one study of over 600 necropsied animals between 2004 and 2010. A similar bacterial strain infects southern sea otters at much lower levels (Counihan et al., 2015). *S. lutetiensis* sp. nov. is a primary pathogen for northern sea otters, and infection is associated with fatal **meningoencephalitis** and infective endocarditis that occurs in prime-age animals with no preexisting conditions (Counihan-Edgar et al., 2012; Counihan et al., 2015). Some infected animals die acutely in good nutritional condition with meningoencephalitis. Others die from chronic, severe vegetative valvular endocarditis of the left atrioventricular and/or aortic valves, often with a dilated, thin-walled left ventricle (Fig. 11.11), massive pulmonary edema,

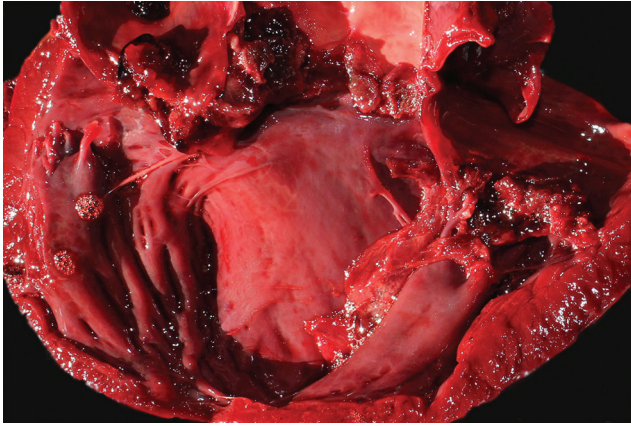


FIGURE 11.11 Valvular endocarditis due to *Streptococcus lutetiensis* sp. nov in a Northern Sea Otter. Diffuse fibrinosuppurative valvulitis and vegetative endocarditis.

effusion, and multifocal thromboemboli with coagulation necrosis in the myocardium, kidneys, spleen, or descending aorta (secondary to saddle thrombus). Coinfection with multiple species of *Bartonella* sp. was identified in both northern and southern sea otters, but infection prevalence was not correlated with the observed lesions (Carrasco et al., 2014).

In contrast, *S. phocae* and related beta-hemolytic streptococci has been identified as major opportunistic pathogen of Southern sea otters (Bartlett et al., 2016). Lesions include sepsis and abscess formation, and may also include meningoencephalitis (Fig. 11.12).

Mustelids have recently been identified as natural hosts of *Staphylococcus delphini*, one of three members of the *S. intermedius* group. Mink, badgers, and ferrets are consistently culture-positive for *S. delphini* group A (Guardabassi et al., 2012). *S. delphini* infections cause hypersecretory diarrhea in ferret kits, while *S. intermedius* infection is associated with cutaneous adenitis and mastitis in mink (Hunter and Prescott, 1991; Sledge et al., 2010).

Bacterial pododermatitis is a common and important problem in farmed mink, especially under conditions of poor husbandry. Acute disease appears as multifocal footpad and nailbed ulcers; chronic infections typically present as foot pad hyperkeratosis. Opportunistic *Staphylococcus* spp. (including *S. canis*, *S. intermedius*, and *S. delphini*), and *Arcanobacterium phocae* (associated with facilities feeding seal meat) are the most common isolates (Chalmers et al., 2015; Jespersen et al., 2015).

Helicobacter mustelae is a ubiquitous bacterial pathogen of ferrets. It is associated with atrophic and lymphocytic gastritis; affected ferrets are used as models for *Helicobacter pylori* infection in humans (Fox et al., 1990). Infection in animals results in hypochlorhydria, vomiting, and diarrhea; most commonly in animals that are >3.5 years old. While not generally fatal, chronic helicobacteriosis contributes to debilitation in older ferrets. Histologically, lesions are typically seen in the pylorus, where the pH is optimal for

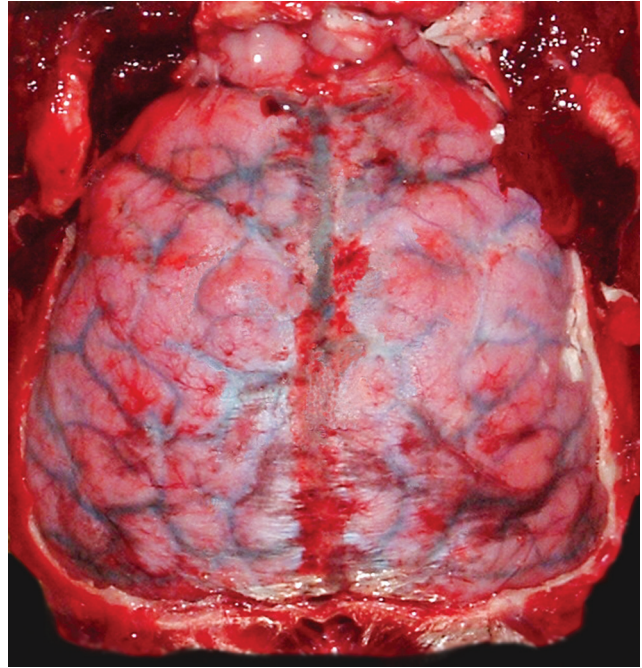


FIGURE 11.12 Fibrinosuppurative meningitis due to *Streptococcus phocae* infection in a Northern sea otter. (Photo Courtesy of C. Goertz, Alaska Sea Life Center)

bacterial colonization, and consist of lymphoplasmacytic gastritis with marked attenuation of gastric glands. *H. mustelae* infection has not yet been causally linked to gastric ulcers, a common condition in affected animals. Silver stains will demonstrate the presence of extracellular spiral bacteria within the mucus layer.

A second mustelid *Helicobacter* species has been isolated from southern sea otters; *H. enhydrae* sp. nov (Shen et al., 2017). In one study, this bacterium was isolated from postmortem gastric tissues of 29% of tested sea otters, and 58% were PCR-positive for a novel *Helicobacter* sp. that was closely related to *H. mustelae* and genetically distinct from other marine mammal *Helicobacter* spp. Histological changes in sea otter gastric tissues range from mild cystic degeneration of gastric glands, to severe mucosal erosions and ulcers with associated silver stain-positive bacilli. Although spatial associations with gastric lesions were reported, *H. enhydrae* infection has not yet been confirmed as a cause of gastric ulcers and inflammation in sea otters.

Lawsonia intracellulare infection can cause proliferative colitis in ferrets, especially young males. In contrast with hamsters, pigs, and horses, where the ileum is preferentially affected, *L. intracellulare* infection in ferrets is primarily associated with colonic pathology. Clinical infection is characterized by bloody, liquid stool. Grossly, the mucosa has a thickened, corrugated appearance, due to marked hyperplasia of colonic enterocytes. As the rapidly proliferating colonic enterocytes are immature, affected areas are largely devoid

of goblet cells, and a marked lymphocytic infiltrate is present in the lamina propria. Silver stains reveal numerous bacteria within the apical cytoplasm of proliferating enterocytes.

Pseudomonas aeruginosa can cause epizootics of necrotizing hemorrhagic pleuropneumonia and septicemia in farmed mink. First described in 1953, this highly contagious bacterium spreads rapidly on affected farms (Gorham and Griffiths, 1956). Peracute death is common and losses may approach 75%, so 6–8 week old mink are routinely vaccinated. Gross necropsy reveals cranioventral bronchopneumonia with copious bloody fluid on cut surface. Microscopic lesions include prominent vasculitis, alveolar hemorrhage, and abundant bacteria within alveoli and capillaries, with similar lesions in tracheobronchial and hilar lymph nodes. A similar lesion pattern has been identified in mink infected with hemolytic *E. coli*, although *E. coli*-associated pneumonia often presents with a more diffuse pattern (affecting the entire lung), fewer bacterial colonies, and more alveolar edema than hemorrhage (Salomonsen et al., 2013).

Striped skunks are commonly *Leptospira*-positive in the United States and Canada, and infections are often clinically silent. For example, in Ontario, Canada, 42% of skunk kidneys were positive for *Leptospira* on immunohistochemistry, suggesting striped skunks as possible reservoirs (Shearer et al., 2014). Serologic evidence of *Leptospira* infection has also been documented at relatively high frequency (over 65%) in European mustelids in European and American mink, European polecats, pine martens, and stone martens. High seropositivity in mustelid predators and skunks likely reflects high prevalence of infection in rodent prey (Moinet et al., 2010). Rare sea otter deaths due to leptospirosis have been reported, with occasional clustering of cases. Gross lesions may be absent or include renal petechiae. Microscopically, moderate to severe multifocal interstitial nephritis is associated with intratubular clumps of spiral bacteria on silver stains. *L. interrogans* seroprevalence was 2% for wild-captured southern sea otters, 10% for sick, stranded southern sea otters, and 0% for northern sea otters (Hanni et al., 2003). Although river otters are commonly seropositive, *Leptospira*-associated disease is rare. Chronic leptospirosis can be associated with abortion and chronic renal disease.

Sylvatic plague, caused by *Yersinia pestis*, is an important cause of mortality for endangered black-footed ferrets. The primary prey for black-footed ferrets is the prairie dog; prairie dog colonies and their fleas serve as reservoirs for *Y. pestis*. As part of in situ conservation programs, colonies located in areas where black-footed ferrets are concentrated or released are dusted with pesticides to control fleas to reduce disease risk. At necropsy, plague-infected ferrets display lesions similar to infected wild felids, with prominent vasculitis and necrohemorrhagic inflammation and perivascular accumulations of bacilli in submandibular and mesenteric lymph nodes (Williams et al., 1994). Vasculitis also occurs in the skin and lungs, resulting in subcutaneous

hemorrhage and pulmonary edema, respectively. Myriad peri- and intravascular bacilli may be seen in other organs. The Siberian polecat appears to serve as a suitable model for plague in black-footed ferrets (Williams et al., 1991). A *Y. pestis*-like bacterium has also been detected in feral stoats in New Zealand via PCR; this population exhibited a high prevalence of idiopathic pneumonia (McDonald et al., 2004). The related bacterium *Y. pseudotuberculosis* has been incidentally isolated from otters, martens, polecats, and mink with no associated lesions (Nikolova et al., 2001).

Fungi

Published reports of mustelid infection by **dimorphic fungi** consist mainly of single case reports, with the exception of *Coccidioides* spp. (Fig. 11.13) and *Emmonsia* spp., suggesting comparatively low pathogenicity for mustelids, or low exposure. Table 11.4 lists reported fungi in mustelids.

Parasites

A variety of parasites have been identified in the Mustelidae. Some are unique to the family; many are seen in other wild-life species. Supplemental Table e1 lists common and important metazoan and protozoan parasites in this family. This list is not all-inclusive; it simply reflects those that are reported in the literature. Expanded information on many of these parasites is available in the online Supplemental Materials. A metazoan parasite of note in the mustelids is *Skrjabinogylus nasicola*. When infections are moderate to severe, there can be considerable remodeling of the frontal bone. In spite of this, the parasite is not a significant mortality factor in mustelids. Its lifecycle includes a mollusk intermediary host and a shrew or small rodent as a paratenic host. Adult worms are found in the nasal and frontal sinuses of mustelids. *Skrjabinogylus lutrae* is a related nematode found in the sinuses of otters (Fig. 11.14).

Systemic infection with the apicomplexan protozoal parasite *Toxoplasma gondii* can cause significant morbidity and mortality in mustelids, especially in free-ranging southern sea otters and black-footed ferrets (Burns et al., 2003; Miller et al., 2008a). Wild and domestic felids are the only known definitive hosts, and mustelids serve as accidental, intermediate hosts. Terrestrial run-off from urban and agricultural areas is thought to transport the parasite to coastal marine waters, where invertebrates serving as prey for sea otters ingest and accumulate the parasite (Arkush et al., 2003; Miller et al., 2002, 2008b). In one study, 52% of necropsied southern sea otters, and 38% of live-sampled animals were seropositive to *T. gondii* (Conrad et al., 2005). Up to 16% mortality has been attributed to toxoplasmosis in fresh southern sea otters carcasses; subclinical infection is also common (Miller et al., 2004). No gross lesions are reported. Microscopic lesions include nonsuppurative

TABLE 11.4 Fungal Disease in Mustelids

Agent	Species Affected	Lesions
<i>Coccidioides</i> sp.	Sea otter ^a , river otter ^b	Pyogranulomatous pleuropneumonia with pleural effusion, granulomatous lymphadenitis, encephalitis, dermatitis, peri- and myocarditis, ophthalmitis
<i>Histoplasma capsulatum</i>	Sea otter ^c , skunk ^d	Emaciation, pyogranulomatous lymphadenitis, splenitis
Adiaspiromycosis	Mink, pine marten, stoat, sable, weasel Eurasian otter ^e , badger, polecat ^f , skunk ^g	Granulomatous pneumonia, granulomatous lymphadenitis
<i>Cryptococcus</i> spp.	Ferret ^h	Granulomatous pneumonia, rhinitis, ophthalmitis
Dermatophytes	Mink ⁱ	Chronic hyperplastic dermatitis with hyperkeratosis and furunculosis

^aHuckabone, S.E., Gulland, F.M., Johnson, S.M., Colegrove, K.M., Dodd, E.M., Pappagianis, D., Dunkin, R.C., Casper, D., Carlson, E.L., Sykes, J.E., 2015. *Coccidioidomycosis and other systemic mycoses of marine mammals stranding along the central California, USA coast: 1998–2012*. *J. Wildl. Dis.* 51, 295–308.

^bKimber, K.R., Kollias, G.V., Dubovi E.J., 2000. Serologic survey of selected viral agents in recently captured wild North American river otters (*Lontra canadensis*). *J. Zoo Wildl. Med.* 31, 168–175.

^cBurek-Huntington, K.A., Gill, V., Bradway, D.S., 2014. Locally acquired disseminated histoplasmosis in a Northern sea otter (*Enhydra lutris kenyoni*) in Alaska, USA. *J. Wildl. Dis.* 50,389–392.

^dWoolf A., Gremillion-Smith, C., Sundberg, J.P., Chandler, F.W., 1985. Histoplasmosis in a striped skunk (*Mephitis mephitis* Schreber) from southern Illinois. *J. Wildl. Dis.* 21, 441–443.

^eMalatesta, D., Simpson, V.R., Fontanesi, L., Fusillo, R., Marcelli, M., Bongiovanni, L., Romanucci, M., Palmieri, C., Della Salda, L., 2014. First description of adiaspiromycosis in an Eurasian otter (*Lutra lutra*) in Italy. *Vet. Italia* 50, 199–202.

^fTinker, M.T., Hatfield, B.B., Harris, M.D., Ames, J.A., 2015. Dramatic increase in sea otter mortality from white sharks in California. *Mar. Mammal Sci.* 32, 309–326.

^gBurek, K.A., 2001. Mycotic diseases. In: Williams, E.S., Barker, I.K. (Eds.), *Infectious Diseases of Wild Mammals*, third ed. Iowa State University Press, Ames, Iowa, pp. 518–531.

^hMorera, N., Hagen, F., Juan-Sallés, C., Artigas, C., Patricio, R., Serra, J.I., Colom, M.F., 2014. Ferrets as sentinels of the presence of pathogenic *Cryptococcus* species in the Mediterranean environment. *Mycopathologia* 178, 145–151.

ⁱOvery, D.P., Maron-Lopez, F., Muckle, A., Bourque, A., Lund, L., MacHattie, D., Lopez, A., 2015. Dermatophytosis in farmed mink (*Mustela vison*) caused by *Trichophyton equinum*. *J. Vet. Diagn. Invest.* 27(5), 621–626.

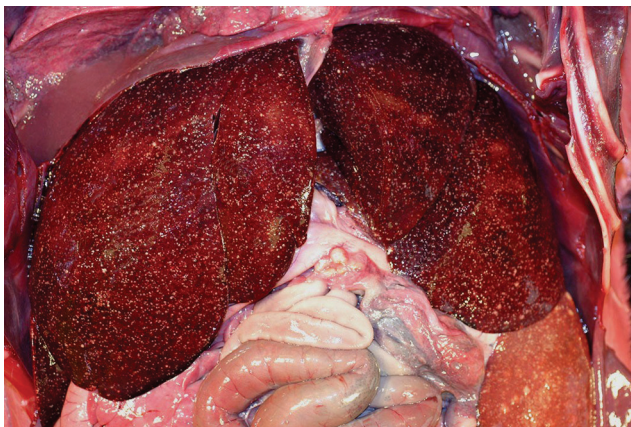


FIGURE 11.13 *Coccidioidomycosis* in a Southern sea otter. Hepatic and splenic granulomas are seen in the liver and spleen of a Southern sea otter infected with *Coccidioides immitis*.

meningoencephalitis, myocarditis, and lymphadenitis. Less commonly reported conditions are placentitis, abortion, and a single case of toxoplasmosis that was associated with congenital brain malformation (Miller et al., 2008b).

In an epizootic of toxoplasmosis in captive black-footed ferrets at a zoological park in 1992, clinical signs in adults and kits included anorexia, lethargy, corneal edema, and ataxia; two adults and six kits died acutely (Burns et al., 2003). Toxoplasmosis was confirmed by immunohistochemistry and ultrastructural examination.

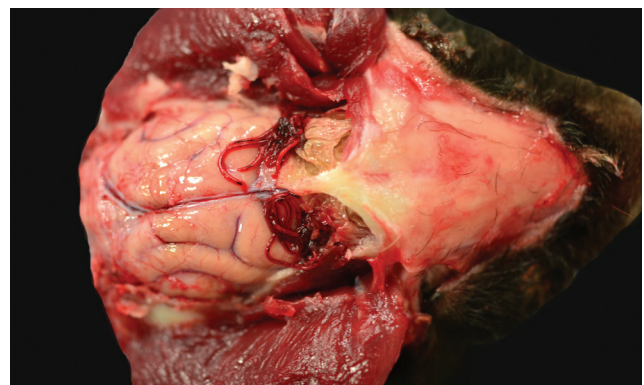


FIGURE 11.14 *Skrjabingylus nasicola* adults in the sinuses. (Photo Courtesy of E.Edmondson, Colorado State University)

Additional adult ferrets succumbed to chronic toxoplasmosis, characterized by development of chronic progressive posterior paresis and posterior ataxia 6–69 months after the epizootic began. Meningoencephalitis or meningoencephalomyelitis was identified in all cases. Although the source was not identified, frozen uncooked rabbit was considered as the most likely source. *Toxoplasma* spp. infection has also been reported in ferrets, steppe polecats, and mink (Burns et al., 2003). Skunks are commonly infected, but clinical disease has not been reported.

Prions

Transmissible mink encephalopathy (TME) is a spongiform disease diagnosed in farmed mink in numerous countries. First identified in 1947, the most recent US outbreak occurred in 1985. While the origin of TME is unconfirmed, the cause is presumed to be consumption of scrapie prions from sheep, likely via contaminated feed. In mink, disease

spreads through biting or cannibalism, with an incubation period of 6–12 months, and a clinical course of 2–8 weeks. Affected animals exhibit ataxia, aggression, soiling of the nest, and self-mutilation. Histologically, there is marked spongiform degeneration and cerebral astrocytosis, especially the frontal cortex and less pronounced in the caudal brain ([Imran and Mahmood, 2011](#)).

E-ONLY CONTENTS

TABLE e1 Commonly Reported Parasites of Mustelids

Metazoa	Species Affected	Lesions
<i>Proficollis</i> spp.	Sea otter ^j	Intestinal perforation, peritonitis
<i>Corynosoma enhydri</i>	Sea otter ^k	No clinical disease reported
<i>Pseudoterranova decipiens</i>	Northern sea otter ^{l,m,n}	Gastritis with perforation, peritonitis.
<i>Physaloptera</i> spp.	Skunk, ^o badger ^p grison ^q	Catarrhal gastritis, disease usually subclinical
<i>Dracunculus insignis</i>	Marten, ^r otter ^s	Granulomatous cellulitis and fasciitis
<i>Dioctophyme renale</i>	Mink ^t	Hematuria, granulomatous nephritis with hydronephrosis. Usually found in the right kidney
<i>Skryabynglus nasicola</i>	Skunk, mink, ermine, stoat, weasel, polecat ^u	Catarrhal to eosinophilic sinusitis, atrophy of sinusoidal bone with deformity; clinical disease is apparently rare.
<i>Baylisascaris</i> spp.	Badger, ^v skunk weasel, sea otter ^w	Eosinophilic and necrotizing meningoencephalitis, visceral larval migrans
<i>Capillaria</i> spp.	Mink, marten skunk ^x	Varies from no disease to catarrhal gastritis, eosinophilic tracheitis, and lymphocytic esophagitis
<i>Dirofilaria immitis</i>	Ferret, otter ^y	Right heart failure, pulmonary congestion and siderosis, chronic hepatic congestion.
<i>Filariataxidea</i>	Badger, ^z skunk	Subepidermal vesiculobullous dermatitis. Lesions are more hyperkeratotic in skunks.
<i>Versteria</i> sp. (cestode)	Ermine, mink ^{aa}	No clinical disease
<i>Trogloremaacutum</i>	Ferret, polecat, marten, badger, skunk ^{bb}	Lytic lesions of the nasal sinuses
Protozoa		
<i>Toxoplasma gondii</i>	Sea otters, ^a black-footed ferret (BFF), ferret, steppe polecats, mink. ^b	Nonsuppurative meningoencephalitis, myocarditis, and lymphadenitis; placentitis, abortion
<i>Sarcocystis neurona</i>	Sea otter, ^c fisher, ^d skunk ^e	Subcutaneous hemorrhage, lymphadenopathy, chemosis, histiocytic or lymphoplasmacytic myocarditis, meningoencephalitis, and myelitis, interstitial pneumonitis, hepatitis, splenitis, and retinochoroiditis
<i>Neospora caninum</i>	Polecats, badgers, ferrets, mink ^f	Subclinical meningoencephalitis
<i>Eimeria</i> spp.	Ferret ^{g,h}	Necrotizing enteritis with bloody diarrhea, infection of gallbladder, and biliary epithelium
<i>Cryptosporidium</i> spp.	River otter ⁱ	No clinical disease reported
<i>Giardia</i> spp.	River otter ⁱ	No clinical disease reported
Ectoparasites		
<i>Sarcoptes</i> spp.	All mustelid species	Proliferative, hyperkeratotic, and eosinophilic dermatitis
<i>Otodectes cynotis</i>	Ferret	Ceruminous otitis with minimal pruritis

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^bBurns, R., Williams, E.S., O'Toole, D., Dubey, J., 2003. *Toxoplasma gondii* infections in captive black-footed ferrets (*Mustela nigripes*), 1992–1998: clinical signs, serology, pathology, and prevention. *J. Wildl. Dis.* 39, 787–797.

^cDubey, J.P., Thomas, N.J., 2011. *Sarcocystis neurona* retinochoroiditis in a sea otter (*Enhydra lutris kenyoni*). *Vet. Parasitol.* 183, 156–159.

^dGerhold, R.W., Howerth, E.W., Lindsay, D.S., 2005. *Sarcocystis neurona*-associated meningoencephalitis and description of intramuscular sarcocysts in a fisher (*Martes pennanti*). *J. Wildl. Dis.* 41, 224–230.

^eDubey, J.P., Hamir, A.N., Niezgoda, M., Rupprecht, C.E., 1996. A *Sarcocystis neurona*-like organism associated with encephalitis in a striped skunk (*Mephitis mephitis*). *J. Parasitol.* 82, 172–174.

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TABLE e1 Commonly Reported Parasites of Mustelids (Cont.)

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- ^rSeville, R.S., Addison, E.M., 1995. Nongastrointestinal helminths in marten (*Martes americana*) from Ontario, Canada. *J. Wildl. Dis.* 31, 529–533.
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