CHAPTER 9

Musteloid diseases: implications for conservation and species management

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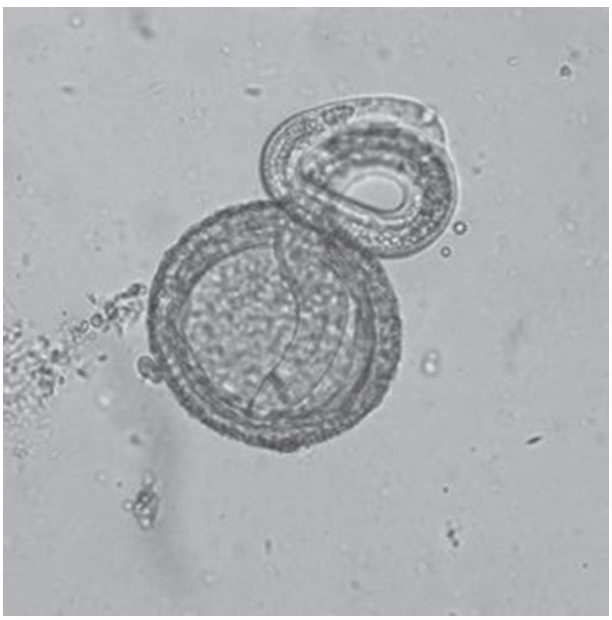

Larva of *Baylisascaris procyonis* hatching from an egg. © US Centers for Disease Control and Prevention

Introduction

As with all living organisms, the Musteloidea are susceptible to a wide range of infectious diseases, arising from prion, viral, bacterial, protozoan, or multicellular pathogens. Furthermore, they also suffer from various non-infectious organic diseases, causing patho-physiological change to bodily tissues or organs, such as cancers, heart disease, and genetic disorders.

Symptoms can be acute or chronic; they can be localized, affecting a specific system or bodily function (e.g. conjunctivitis); disseminated through the body (e.g. metastasizing cancers); or systemic, affecting the entire body (e.g. viraemias). The immunological resilience of individuals also differs depending on intrinsic factors, such as age, nutrition, and co-infections; in turn linked to habitat, diet, and ecomorphology. Immature individuals are especially susceptible to pathogens, and without sufficient offspring recruitment population decline will ensue (Hudson et al. 2002).

From a conservation ecology perspective, when we see a population in demise, or conservation measures

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fail, too often we ignore the potential involvement of disease, especially if the symptoms are cryptic or the aetiology obscure. This is likely because of the inevitability of 'death by natural causes'. Yet wildlife diseases often arise from distinctly unnatural sources, such spill-over from our pets, our farming practices, our pollution, and our intrusion into wild habitat exacerbating exposure (Sih 2013). Habitat fragmentation that impinges on host dispersal and recolonization further amplifies effects at the population level. Diseases also travel with their hosts, and/or with their vectors; thus introducing an alien species into a new region or ecosystem can take with it infections that then afflict endemic community members. Although disease is often foremost among drivers of population regulation, it is often the forgotten agent when modelling a species' potential range, with a view to habitat management or reintroductions. If the species in question is susceptible to an unrealized disease (or vector) in that region, it will never thrive there.

At the population level, a background level of disease is inevitable, especially among wild animals. While these enzootic diseases may have an incipient effect on population structuring and immunological evolution, it is often sudden outbreaks of epizootic disease that cause the catastrophic population crashes that flash bright on the conservationist's radar. Moreover, diseases confined to their wild hosts usually garner less attention than those zoonotic conditions able to spread to humans, or even spill-over into our livestock. Eco-toxicology is pertinent here too; be that organochlorine and polychlorinated biphenyl (PCB) contamination of watercourses poisoning otters, or prey-derived secondary poisoning of mustelids arising from exposure to rodenticides. This is a subject in its own right, however, where poison damages tissues and organs just as surely as being impacted by a car—thus subtly different to those diseases we discuss here, which are caused by biological agents, or result from the dysfunction of the host's own biology. Consequently, we minimize our account of ecotoxins to only the most significant cases.

Inevitably the literature on musteloid diseases is greatest for those most prominent species, typically those living in proximity to people. Nevertheless, this does not imply that disease is any less influential in the ecology of less well-known species; it just reflects how much work remains to be done in this field. To simply list every disease able to afflict the superfamily Musteloidea would be as dull as it would compendious. Instead, we focus here on infectious pathogens that cause significant illness, or 'dis-ease'; that is, conditions that overcome the host's immune defences sufficiently to cause patho-physiological and behavioural symptoms, which may either go into remission, leaving antibodies, or cause the host's demise. Importantly, when a disease is kept sub-patent by the host's immune system it does not always imply the body is not working hard to achieve this competency, incurring energetic costs. Aside from systemic diseases, all wild animals are beleaguered by a diversity of ectoparasites. These are too numerous to include comprehensively here, and often cause only mild discomfort; thus we restrict mention only to ectoparasites vectoring pathologic disease.

We describe the prevalence and pathogenicity of major diseases relevant to musteloids, and discuss implications for each subfamily, in turn. We are, however, inevitably constrained by limited literature in several instances, where we stress that 'the absence of evidence does not confer evidence of absence' (*modus tollens*; see Noonan et al. 2015c) and that less-researched species likely suffer from considerably more diseases than we realize. In this sense, this chapter is neither comprehensive, nor should it be used diagnostically—rather it emphasizes the inter-relationship between disease and species ecology. In particular, we draw attention to instances where the fingerprints of humanity are implicated in disease aetiology.

Morbid mink

Although starting with 'mink' may appear taxonomically or ecologically random, from a disease perspective, mink are the foremost researched musteloid. This is due to the long history of farming ('ranching') American mink (*Neovison vison*) in captivity for their valuable fur (see Fraser et al., Chapter 16, this volume; Maran et al., Chapter 17, this volume), and thus the need to maintain the health of these animals under conditions conducive to high infectious transmission rates.

Aleutian Mink Disease Virus (**AMDV** causing **AMD**) is a highly contagious parvovirus (also termed 'mink contagious plasmocytosis'—Bloom et al. 1994), able to infect all musteloids, under experimental conditions. First identified in farmed mink in 1956, contrary to perceptions, AMD has no connection to the Aleutian Islands. Rather the pathogen was named after the Aleutian coat colour gene that gives mink a gun-metal grey coat, prized by fur farmers—although subsequently the virus has spread into all colour types. In the 1950s–60s, mink farmers made

their own distemper vaccines by homogenizing tissue from distemper-infected mink, which led to the rampant spread of AMDV in farmed mink, and quickly to farmed ferrets.

The pathogenicity of AMDV varies with the musteloid species infected, the individual's immune status and general health, its genotype (Guzmán et al. 2008), and the strain of the virus involved. Among mink, strains range from non-virulent, through low-virulence isolates to which only Aleutian coat colour varieties succumb, through to highly virulent strains, such as Utah-1, Danish-K, and Ontario isolates (Nituch et al. 2012), which lead to over 90% mortality within two weeks of exposure (Alexandersen et al. 1994).

Morbidity arises from antibodies (immunoglobulins) being unable to neutralize the virus (Steinel et al. 2001), thus immune complexes form and deposit in various tissues, causing glomerulonephritis, arteritis, splenomegaly, and ultimately bone marrow suppression, renal failure, and death, usually in no more than a few months. Infected females also suffer reduced fertility and abortion. The virus can be spread in urine, faeces, saliva, and blood, and as a sexually transmitted disease, and vertical transmission to offspring occurs during pregnancy (Nituch et al. 2012). Kits suffer acute pneumonia that causes 98–99% mortality in fur-farm outbreaks (Marcaccini et al. 2008). Not all animals that are infectious exhibit clinical signs, however, making disease management difficult.

Although mink farmers vaccinate kits against botulism, distemper, and enteritis, and respond to outbreaks of pneumonia with vaccination, due to inability of antibodies to neutralize AMDV, there is currently no effective vaccine or treatment for AMDV, despite ongoing research (Castelruiz et al. 2005). Consequently, the only way to manage AMDV on mink ranches is through antibody testing and selective culling (Nituch et al. 2012).

Fur farming was banned in the UK in 2000, but this should not imply to readers from the UK that fur farming is in demise—far from. As of 2015 there were 5000 fur farms in the EU (Fur Europe 2015), accounting for 63% of global mink pelt production (Fur Commission USA; see also Fraser et al., Chapter 16, this volume). The US is the fifth largest mink producer (Denmark is number one). Remarkably, however, the Canadian Province of Nova Scotia (NS; where author CN happens to live) stands out as having a particularly large investment in mink farming. With a human population of <1 million, NS produces around 1.4 million mink pelts per year (Medel 2013), valued at around USD150 million—one quarter of the agricultural revenue raised in the Province (Bundale 2013). In contrast, prior to the 2000 ban, the UK's 11 mink farms produced 100,000 pelts annually.

Not only do mink escape fur farms and spread AMDV directly, but in Canada contaminated ranched mink manure is used extensively as a field fertilizer, without restriction. Unsurprisingly, Farid (2013; see also Farid et al. 2010) found that Nova Scotia (NS) has unprecedented levels of AMD in its wildlife. Examining spleen homogenates (via PCR and antibodies), Farid found not only that 56 of 60 wild American mink sampled across NS were AMDV positive, so were 43 of 61 short-tailed weasels (*Mustela erminea*; aka stoats, or ermine), two of eight striped skunks (*Mephitis mephitis*), two of 11 North American river otters (*Lontra canadensis*), nine of 85 raccoons (*Procyon lotor*), and two of 10 bobcats (*Lynx rufus*). Crucially, Cho and Greenfield (1978) found that of 74 of 120 AMDV seropositive 'feral' mink trapped in Ontario, 80% had a non-progressive infection, showing that asymptomatic mink can live long enough to reproduce and transmit infection.

AMDV also infects American mink outside of their native range. Wells et al. (1989) reported that 85% of former British mink farms tested had Aleutian disease present, with up to 40–90% prevalence. Although the farms are now closed, escapees have carried AMDV into the wild. Yamaguchi and Macdonald (2001) found 14 of 20 seropositive feral mink in the Upper River Thames region (UK). Ten years later, Harrington et al. (2012a) reported that eight of 12 mink shot (for invasive species control) were AMDV positive.

In Spain, Mañas et al. (2001) describe escaped American mink displacing smaller, native European mink (*Mustela lutreola*) not only through direct competition (see Maran et al., Chapter 17, this volume), but also due to introducing AMDV. In southwestern France, Fournier-Chambrillon et al. (2004a) detected AMDV in 17 of 75 American mink and 12 of 99 European mink, as well as in 16 of 145 polecats (*Mustela putorius*), four of 17 stone martens (also called beech martens, *Martes foina*), one of 16 pine martens (*Martes martes*), and three of 68 common genets (*Genetta genetta*). In Iceland, Skírnisson et al. (1990) reported 13 of 394 wild American mink with AMDV antibodies. Knuuttila et al. (2009) describe three mink farming-related waves of AMDV among wildlife in Finland. Ultimately, it seems that AMDV has spread to all regions where American mink are now found.

Mink Enteric Virus (MEV) is another prevalent mink parvovirus, where infection in farmed mink is

controlled using a three-way vaccine against MEV, botulism, and *Pseudomonas*. MEV is most closely aligned with feline panleukopaenia (a problem for raccoons—below), and is genetically and antigenically distinct from AMDV. It is transmitted via the faecal–oral route with an incubation period of four–eight days, although the virus can survive in the environment for several months. Symptoms include watery mucoid bloody diarrhoea, resulting from a dilated, flaccid hyperaemic small intestine. Anorexia, dehydration, and death soon follow.

Mink are also very susceptible to **Canine Distemper Virus (CDV)**, a morbillivirus able to infect a wide variety of carnivores. This can be a serious disease; 50% of adult domestic dogs and 90% of pups contracting CDV die. It is a form of influenza that can spread through aerosol, as well as direct routes, but the virus dies quickly in the environment. CDV outbreaks in (ranched) mink must be differentiated from pseudo-distemper, or tyrosinaemia II—a rapidly degenerative and fatal genetic disease (Sanford 1988). Clinical signs include an ocular discharge, hyperaemia, and swollen, crusty skin on the feet, abdomen, and throat. Ultimately CDV affects the central nervous system, causing severe convulsions and vocal 'wailing' as neurological symptoms progress. Many, but not all, of the infected individuals that develop clinical signs die, but progression of the disease is slow. Infected animals without clinical signs can develop immunity and survive, but can infect other susceptible mustelids, enabling widespread transmission among free-living animals (McDonald and Larivière 2001). CDV can be managed among ranched mink by the use of live vaccine, although vaccine-induced canine distemper has been reported (Sutherland-Smith et al. 1997). Cunningham et al. (2009) describe four dead wild mink with microscopic lesions and viral inclusions consistent with CDV in the Florida Everglades—subsequently confirmed by reverse transcription PCR. Working in southwestern France, Philippa et al. (2008) found antibodies to canine distemper virus (CDV) in 9% of 127 European mink and 5% of 12 American mink, as well as in several other sympatric mustelid species.

In 1984, 3000 mink died from pneumonia over 33 farms in one month along the south coast of Sweden (Klingeborn et al. 1985). The cause was identified as **influenza A virus** (possessing avian H10N4 surface antigens) possibly arising from seabirds being attracted to outdoor mink cages by mink food—one of the first examples of avian flu infecting a mammal population. Although ranched ferrets are particularly susceptible to less virulent strains of influenza, including human strains, influenza was not identified in mink in the USA until 2010. An outbreak on a mink farm can quickly produce over 90% seropositives. Infection causes coughing and sneezing, which can cause mortality among juvenile mink, combined with progression to more pathological secondary bacterial infections, such as *Pseudomonas aeruginosa*, causing acute haemorrhagic pneumonia—although this can be treated by vaccination (Hunter and Prescott 1982).

There are several other mink viral diseases that cause economic losses on ranches, but these are rare in wild populations. For example, Aujeszky disease (pseudorabies), arising from porcine herpes-virus—this causes severe and rapidly fatal seizures in mink fed tainted pork products (although eradicated from British pigs); Shaking Mink Syndrome (SMS: an astrovirus)—a meningoencephalitis affecting juveniles with low mortality; and Epizootic Catarrhal Gastroenteritis (a coronavirus) prevalent in 100% of Danish mink farms—most prevalent among dark colour morphs, occurring during times of stress.

Other disease types noted for this extensively researched musteloid include: **Mink Chronic Wasting Disease** (**CWD**), or **Transmissible Mink Encephalopathy (TME)**; a prion disease arising among farmed mink fed infected beef (related to Bovine Spongiform Encephalopathies [BSE])—although mink appear to have a barrier to primary oral challenge with scrapie (Ovine Spongiform Encephalitis) (R. Harrington et al. 2008). Also arising from toxic feed, **botulism** outbreaks occur frequently in farmed mink (Lindström et al. 2004) necessitating vaccination as standard.

Protozoan diseases include occasional outbreaks of **toxoplasmosis** in ranched mink (Frank 2001; see Sea otters, below) and in the wild. Harrington et al. (2012a) report that six of 12 American mink tested positive for ***Toxoplasma gondii*** antibodies in their Upper Thames study. In County Wicklow, Ireland, O'Crowley and Wilson (1991) found seven of 15 American mink trapped tested positive for *T. gondii* (but no cysts were found in brain necropsies). Minor parasitic diseases include sarcocystis, with Dubey and Hedstrom (1993) reporting acute infections leading to meningoencephalitis and substantial mortality. Outbreaks of coccidiosis are also commonplace among ranched mink (Myers et al. 1980), but respond well to sulphonamide antibiotics. As we shall elaborate more for European badgers (*Meles meles*), these protozoans can impact juvenile survival severely in wild populations. In southwestern France, Moinet et al. (2010) report **leptospirosis** antibodies, arising from the spirochete bacterium

Leptospira interrogans, in 73 of 99 European mink and 64 of 74 American mink, plus 87 of 144 European polecats, 17 of 19 stone martens, and 14 of 19 pine martens. In Ireland, ***Cryptosporidium*** was successfully amplified by PCR in four of 81 'wild' mink faecal samples (Stuart et al. 2013).

Among various minor nematodes, notable is ***Skrjabingylus nasicola***, which infects the nasal sinuses of mink and many other mustelids, as well as skunks (*Mephitis* spp.), with a raccoon variant. Hansson (1968) found 45% of 126 mink with *S. nasicola* in Sweden, while Harrington et al. (2012a) found one incidence in 12 mink post-mortemed in Oxfordshire, UK. A total of 261 mink skulls from Ontario, examined by Bowman and Tamlin (2007) exhibited reduced braincase volume, due to the swelling of frontal sinus from *S. nasicola* infection—but only in males, suggesting infection may be sex-biased. Larvae are ingested by mustelids when foraging on terrestrial gastropods, or mice, shrews, frogs, or snakes (intermediate hosts), which then penetrate the abdominal wall and moult into worms, which migrate through the abdomen to the spinal column and into the sinuses.

A notable helminth (among many minor ones) of species-specific significance is the lung fluke ***Paragonimus kellicotti***, infecting 16 of 105 wild mink in Ontario (Ramsden and Presidente 1975), with 44% prevalence among wild mink screened in Ohio by Gesinski et al. (1964). This parasite is restricted by the distribution of the first intermediate host *Pomatiopsis lapidaria*, an amphibious snail, found in southeastern and Midwestern regions of US and southern Ontario, Canada, although crayfish are also an effective intermediate host.

Organic diseases among ranched mink include osteosarcoma (Mikaelian et al. 1998; a condition noted also among wild sea otters [*Enhydra lutris*]—Fernandez et al. 2012). Nursing sickness, a simple metabolic stress disorder, is also a major cause of mortality among ranched breeding female mink. Clausen et al. (1992) reported 14% mortality of 1774 lactating females in Denmark. Mink farms in Ontario (n = 48) report that 56% of their 0.2–10.1% mortality rate among lactating females was attributable to nursing sickness (Schneider and Hunter 1993).

Queasy weasels: small carnivorous mustelids

Many other carnivorous mustelids succumb to the same infections as mink, but with different levels of importance to their overall biology. In addition, a variety of other parasites and pathogens afflict this group, where again the greatest detail is known about the *Mustela* species most often kept in captivity, the ferret, *Mustela putorius furo*.

Ferrets have a long history of being used by people to hunt rabbits, stretching back over 2000 years (from mtDNA; Davison et al. 1999). Increasingly today, however, they are kept as exotic pets. Ferrets are also bred commercially for use as experimental models for lab research on human influenza (Matsuoka et al. 2009, Maher and DeStefano 2004) and cystic fibrosis (Li and Engelhardt 2003), and were important for research into the 2009 H1N1 'swine flu' epidemic, as well as investigations of SARS (Chu et al. 2008). As a consequence of careful health controls in a laboratory setting, ferrets provide further insight into the range of diseases to which *Mustela* spp. are all likely susceptible.

Ferrets can contract the majority of viral diseases described for mink (Kiupel and Perpiñán 2014), although exposure to these diseases in laboratory settings, or when homed as a pet, limits their incidence. Ferrets are very susceptible to **Canine Distemper Virus (CDV)** and are commonly used for CDV immunological research and vaccine development (e.g. Von Messling et al. 2003). This high susceptibility means that when CDV vaccines are administered to ferrets/polecats they must be strongly attenuated; this proved an issue with early work vaccinating black-footed ferrets (*M. nigripes*) as part of a reintroduction programme in US prairie States (Carpenter et al. 1976; Biggins and Ead, Chapter 15, this volume).

The **ferret ADV** strain differs from the American mink AMDV strain, and can lie dormant in ferrets until they become stressed or injured—even then progressing more slowly, and producing less severe histopathologic lesions. Severe cases do occur, however, involving clinical hypergammaglobulinaemia, CNS lesions, and lymph node degradation, causing impaired proprioception, and ultimately paresis ataxia. Welchman et al. (1993) conducted a serological survey of 446 ferrets owned by ferret club members in the USA and discovered 8.5% seropositives.

Ferrets provide a lab animal model for research and vaccine development relating to **Feline Panleukopaenia Virus (FPV)** and the related **Mink Enteric Virus (MEV**) (Kilham et al. 1967). Natural infections of farmed ferrets with these non-ADV parvoviruses have, however, not been detected serologically (Veijalainen 1986), and parvovirus infection has not been described in non-domestic ferrets (black-footed ferrets, or polecats *Mustela eversmanii* or *Mustela putorius*).

Ferrets are especially susceptible to gastrointestinal symptoms. In 1993, severe, highly contagious, life-threatening diarrhoea was reported in ferrets attending pet shows in the eastern US. Because of the character of the diarrhoea, this disease became known as 'green slime disease'. It was suspected that the cause was a coronavirus, and because of the intestinal signs, this disease was officially named **Epizootic Catarrhal Enteritis (ECE)**; clinical symptoms include necrosis of villus enterocytes, villus atrophy, fusion, and blunting; and lymphocytic enteritis (Williams et al. 2000). Another coronovirus, **Feline Infectious Peritonitis (FIP),** was reported by Garner et al. (2008) involving 23 cases of show ferrets from the US and Europe suffering from systemic pyogranulomatous inflammation, palpable intra-abdominal masses, anorexia, diarrhoea, and, occasionally, hind limb paresis.

Other infectious and prevalent gastrointestinal infections of ferrets include ***Campylobacter*** (Fox et al. 1988), ***Cryptosporidium*** (Rehg et al. 1988), ***Helicobacter mustelae*** (Fox et al. 1990), and **coccidiosis**, caused by ***Eimeria furonis*** (B. Williams et al. 1996; Sledge et al. 2011) (see European badgers, below).

Aside from pathogen-driven diseases, ferrets can suffer from **adrenal disease** (adrenal gland tumour—or hyperadrenalcorticism; Johnson-Delaney 2006), **insulinoma** (cancer of the pancreas; Weiss et al. 1998) and **lymphoma** (cancer of the white blood cells; Li et al. 1995).

An important pathological condition that is well-known among ferrets, and is likely pervasive across the mustelids, is connected to induced ovulation (Macdonald et al., Chapter 6, this volume). Among low-density, diffuse mustelid populations there is a risk that a female might ovulate wastefully, without finding a male to fertilize her. To resolve this risk, it appears that all mustelids, in which the trait has been investigated, have evolved induced ovulation (IO) (Amstislavasky and Ternovskaya 2000). In domestic ferrets, if females (jills) fail to mate, prolonged high levels of oestrogens, during unterminated oestrus, can cause systemic bone marrow suppression, leukopaenia, and thrombocytopaenia, with 50% of jills developing aplastic anaemia, which can lead to haemorrhaging and death. These high oestrogen levels also lead to secondary uterine infections in about half of unmated jills, where the cervix remains partially dilated, allowing bacterial infections (*Escherichia coli, Staphylococcus* sp., *Streptococcus* sp., and *Corynebacterium* sp.) to develop into pyometra and fatal toxaemia (Batchelder et al. 1999). In captive ferrets, this entire cascade can be prevented by the 'jill jab'—a progesterone (proligesterone) injection that terminates oestrus (or by mating with a vasectomized male, assuming breeding is not an option). The literature is sparse on how failure to mate can impact wild mustelids—where objectively, unrestrained by captivity, females will seek out mates whenever possible. Nevertheless, wild polecats, black-footed ferrets, etc., are close genetic relatives to domestic ferrets; Johnson et al. (1999) report pyometra in a Siberian polecat—indeed all unmated female mustelids could be at risk.

Given the susceptibility of pet ferrets, and those used in biomedical research, to these conditions, despite veterinary care and controlled environments, it is clear that the incidence in closely related wild *Mustela* species is likely to be substantial, although formal investigations are few. Crucial too is to emphasize that these small mustelids carry little energy reserve and any inappetence, or inability to hunt, is likely to cause their rapid demise in the wild.

Black-footed ferrets

The potential for disease to impact wild mustelid populations is brought into sharp focus by the endangered, and thus thoroughly researched, black-footed ferret, *Mustela nigripes*. Black-footed ferret populations are highly dependent on prairie dog (*Cynomys* spp.) colonies as prey, thus numbers declined precipitously through the 1900s as prairie dogs were eradicated as a nuisance to agriculture. Black-footed ferrets were believed to be extinct, until in 1981 a relict population was discovered (Biggins and Ead, Chapter 15, this volume). Williams et al. (1988) describe how two of six black-footed ferrets caught in 1985, to commence captive breeding, exhibited severe pruritus, hyperkeratosis, and progressive loss of body condition consistent with **CDV**. Subsequently the remaining four also came down with CDV symptoms, developing intermittent diarrhoea and respiratory disease. Upon post-mortem, meningoencephalitis was apparent and CDV was isolated from these ferrets with paramyxoviral nucleocapsids observed in their faeces. None of these black-footed ferrets had antibodies to CDV in their sera, implying CDV exposure was always fatal; however, antibodies were identified in the sera of sympatric American badgers (*Taxidea taxus*) and coyotes (*Canis latrans*). Ultimately the entire colony of black-footed ferrets from which these individuals were captured died (the cause of which is unknown but may have been plague – see below); the few remaining

individuals were taken into captivity (see Biggins and Ead, Chapter 15, this volume).

Early CDV vaccines were insufficiently attenuated, and vaccine killed black-footed ferrets (Carpenter et al. 1976), but thanks to successful testing on very similar Siberian polecats (*Mustela eversmanii*, the black-footed ferrets' nearest relative), subsequently, black-footed ferrets have been vaccinated against CDV (Wimsatt et al. 2006). A decision was taken not to manage *Eimeria* infections in this captive colony of black-footed ferrets (although coccidiosis does occasionally kill some individuals), because once released they would be exposed to the endogenous parasite. ***Cryptosporidium*** has also proven a problem for the back-footed ferret reintroduction programme, but is believed to be exotic, rather than originating in the native population (Gompper and Williams 1998). Interestingly, Emerson (1964, cited in Gompper and Williams 1998) records a species of biting louse infesting black-footed ferrets (and sympatric weasels), *Neotrichodectes minutus*, which has become extinct since black-footed ferret numbers went through this stringent bottleneck. Canine distemper may pose a threat to free-ranging black-footed ferrets but compelling evidence is lacking.

Plague is a serious issue issue for black-footed ferret recovery (Barnes 1993; Antolin et al. 2002), reducing their prairie dog prey availability by up to 90% during epizootics, but also infecting black-footed ferrets directly. This sylvatic plague is caused by the bacterium ***Yersinia pestis***—introduced inadvertently to the US. This is the same pathogen that was primarily responsible for three known world pandemics of plague in humans. Plague is managed by dusting prairie dog colonies with deltamethrine (Seery et al. 2003) to reduce the flea vectors, and more recently by deploying vaccine baits to prairie dogs (Mencher et al. 2004) as well as by vaccinating released ferrets against infection, using F1-V fusion protein vaccine (Matchett et al. 2010; see Biggins and Ead, Chapter 15, this volume).

Other black-footed ferrets diseases include **human influenza, rabies, tuberculosis, leptospirosis, neoplasia**, and **botulism** (Carpenter et al. 1981), even occasional **amyloidosis**, probably arising from a genetic predisposition among the founder population supporting the captive breeding programme (Garner et al. 2007). Nevertheless, with so few black-footed ferrets remaining and facing more major disease risks, these pathogens are less significant, overall. Although less thoroughly researched, similar diseases prove problematic for other, more abundant, polecat species.

Polecats

The disease status of the European polecat (*Mustela putorius*) is relatively well-established, again due to a history of being bred in captivity for its valuable fur (fitch) used to trim coats, make hats etc. In addition to **CDV** and **influenza** they are prone to **malignant tumours, abscesses** (especially around the head), and **hydrocephaly**. Philippa et al. (2008) report that 20% of 210 polecats carried CDV antibodies in their study in southwest France. European polecats also carry ***trichinosis, leptospirosis, toxoplasmosis***, and ***adiaspiromycosis***, as well as being susceptible to rabies during epizootics (see Raccoons/Skunks).

Steppe (or Siberian) polecats (*Mustela eversmanii*) have similar primary disease susceptibility, including also ***tularaemia*** (see Raccoons, below) and ***pasteurellosis*** in weakened individuals, and can be infected experimentally with ***Y. pestis*** (Williams et al. 1991). Křivanec and Otčenášek (1977) reported that 19 of 26 steppe polecats and 22 of 72 *M. putorius* they examined in Siberia were suffering from **pulmonary adiaspiromycosis**. Less is known about the marbled polecat (*Vormela peregusna*), although phylogenetically and ecologically it would be expected to succumb to this same suite of diseases, and to particular regional endemism; for example they have been found to carry ***trichinellosis***, contracted from infected pigs, in China (Wang et al. 2007).

Several pathologic **bacteria** (notably *Erysipelothrix rhusiopathiae*) and a variety of **helminths** are also recorded for polecats—for example Shimalov and Shimalov (2002), found helminth infection in 34 of 40 European polecats examined in Belarus. **Coccidia** species also infect polecats, such as *Eimeria ictidea* (Dubbelde 2011), and the recently discovered *Isospora eversmanni* and *I. pavlovskyi*, in China (Yi-Fan et al. 2012), where coccidians are typically associated with juvenile morbidity and mortality (see Badgers).

Stoats and weasels

The literature on infectious disease for weasels and stoats/ermine is sparser. Wild stoats (or short-tailed weasels *Mustela erminea*) and (least or common) weasels (*M. nivalis*) are not prone to CDV (but see Almberg et al. 2010) but can contract it in captivity, or experimentally (Ek-Kommonen et al. 2003). Small mustelids are also not very susceptible to rabies (McDonald and Larivière 2001); just 11 of 2851 incidences of rabies reported by Krebs et al. (2003) from 1960–2000, in the US, involved these two *Mustela* spp. (23 of 2851

involved domestic ferrets). They have also proven relatively resistant to tularaemia, although epizootics can impact the availability of vole prey substantially (Hörnfeldt 1978).

Among **bacterial** conditions, between one in three (Twigg et al. 1968) and one in eight weasels (Michna and Campbell 1970) sampled in Britain were positive for ***Leptospira* spp**. which causes **Weil's disease** in humans. *Pasteurella multocida*, the causal agent of haemorrhagic septicaemia in domestic stock, has been isolated from *Mustela* spp. (Rosen 1981), as has ***Bartonella*** spp. in 33 of 45 stoats examined by McDonald et al. (2000), which damages red blood cells (Breitschwerdt and Kordick 2000). Stoats, and more commonly ferrets, can also contract *Mycobacterium bovis*, but at very low rates (Delahay et al. 2002). None of 33 stoats examined in Britain between 1971 and 1986 tested positive for *M. bovis* (MAFF 1987). These small burrow hunters are also prone to fungal infections with ***Chrysosporium parvum***, leading to **adiaspiromycosis**. Křivanec and Otčenášek (1977) report infection in 30 of 76 stoats and 17 of 53 weasels.

As with mink, infection with the nematode ***Skrjabingylus nasicola*** can be especially serious for stoats and weasels, but causes less skull perforation than in mink (Hansson 1968). Hansson (1968) reports 91 of 172 stoat skulls and 31 of 35 polecat skulls exhibiting diagnostic cranial deformity in Sweden. Males tended to exhibit higher prevalence than female (significantly so only for *M. erminea*), typically over 10 nematodes per individual, with up to 52 recorded in a single stoat. *S. nasicola* erodes the nasal sinuses, causing skull perforation, although there is little evidence that infection causes serious morbidity, even to heavily infested individuals (King 1977), although it could cause convulsions in extreme cases.

Diseases and parasites of the long-tailed weasel (*Mustela frenata*), Egyptian weasel (*M. subpalmata*), and Japanese weasel (*M. itatsi*) are likely to be similar, but are not well reported. Similarly, reports of pathogens among larger mustelids (wolverine [*Gulo gulo*], tayra [*Eira barbara*] etc.) are very anecdotal. Certainly traumatic injuries from road traffic or railway accidents, as well as hunting, seem to be a far more major cause of death in wolverines than does disease (Mörner et al. 2005).

Marten maladies

Martens (*Martes* spp.) and fishers (now *Pekania pennanti*, although formerly *Martes*; and included here due to ecological similarity) are semi-scansorial inhabitants of forest ecotopes, that tend to be less strictly carnivorous than other mustelids (Zhou et al. 2011b).

The literature on marten diseases is especially patchy, even compared to a lack of systematic study in other musteloids. **CDV** is the viral disease most often mentioned, although it seems an occasional epizootic that most individuals survive to produce antibodies, rather than a substantial influence on species population dynamics. Tavernier et al. (2012) describe how 30 sick beech martens (also known as stone martens; *Martes foina*) were admitted to a wildlife rescue centre in eastern Flanders. Symptoms included conjunctivitis (80%), rhinitis (100%), dyspnoea with forced abdominal respiration (75%), and generalized muscular tremors/convulsions (75%); all were immobile, often screaming, and either died or were euthanized within three days. PCR sequencing identified CDV as the cause, involving a strain originating from martens in Germany. Pavlacik et al. (2007), screening mustelids for CDV in the Czech Republic, report that, among 21 sick animals, one of 18 stone martens was CDV positive, whereas two pine martens (*Martes martes*) and a European badger screened negative. CDV antibodies were detected in seven of 21 stone martens and one of 20 pine martens tested by Philippa et al. (2008) in southwest France; notably pine marten were the only mustelids (of five species, 481 individuals) not positive for **Canine Infectious Hepatitis** (**CIH**; caused by Canine Adenovirus, CAV)—two stone martens were positive. CIH can be a serious disease (Addison et al. 1987; Stephenson et al. 1982) killing 10–30% of infected dogs, especially young pups (Kahn and Line 2010).

Martens can also contract **AMDV** (parvovirus), with Fournier-Chambrillon et al. (2004a) reporting antibodies in four of 17 stone martens, and one of 16 pine martens. Notably, however, there is a surprising paucity of literature on American marten (*Martes americana*), although Kerr et al. (2005) report that they, and fishers, are susceptible to AMDV. Spriggs et al. (2016) mention that American marten are susceptible to **Canine Parvovirus (CPV)** (along with CDV); and in Germany, Frölich et al. (2005) found CPV antibodies in four of 13 stone marten sera samples and one of two pine martens. Nine of 67 American martens tested with PCR by van de Rakt (2013) were positive for **herpes virus**, of various strains. With supportive care, 68–92% of dogs with CPV enteritis survive and develop lifelong immunity. Infectious CPV can persist outdoors for many months and possibly years, if protected from sunlight and desiccation (Kahn and Line 2010).

Outbreaks of **rabies** can affect martens. In Germany, Geisel et al. (1980) examined 124 beech martens displaying unusual levels of fearlessness and aggression; 23 were positive for rabies. Since this time, Germany has been declared officially rabies free, although Müller et al. (2004) did record spill-over of European ***Bat Lyssavirus Type 1*** into a stone marten in Germany. In North America, although martens are susceptible to rabies, they are not a significant reservoir (US Centres for Disease Control and Prevention [CDC], www.cdc.gov).

As with American mink, trans-species infection with highly pathogenic **avian influenza virus (**subtype H5N1), has been noted in the stone marten, causing diffuse non-suppurative panencephalitis with perivascular cuffing and neuronal necrosis (Klopfleisch et al. 2007).

Among protozoan pathogens, screening 282 wild carnivores in Spain, Sobrino et al. (2007) found ***T. gondii*** antibodies in 17 of 20 stone martens and four of four pine martens (and 26 of 37 badgers). Similarly, in the Czech Republic, Hrková and Modrý (2006) used PCR to diagnose *T. gondii* in three of 61 *Martes* spp. No *Neospora caninum* was found in these martens, although fungal ***Encephalitozoon cuniculi*** infection occurred in two of 61. Antibodies to ***Leptospira interrogans*** were detected in 19 of 19 stone martens and 14 of 19 pine martens examined by Moinet et al. (2010). Spriggs et al. (2016) mention both toxoplasmosis and leptosirosis occurring in American martens.

Nematode infections in martens do not feature strongly in the literature, although they are prone to '**lungworms**': Olsen (1952) reports 18 of 63 American martens that contracted ***Crenosoma* sp.**, while ***Capillaria* sp.** occur in both stone martens (Millán and Ferroglio 2001) and American martens (Foreyt and Lagerquist 1993). Bacteriologically, the stone marten is an unusual but competent host of ***Francisella tularensis*** (Origgi et al. 2013), causing **tularaemia**, while the American marten is similarly an atypical but competent host of plague-causing ***Yersinia pestis*** (see Black-footed ferrets).

The literature on lesser-known marten species is too sparse to enable meaningful commentary; however, in areas with similar endemic disease, other *Martes* spp. will likely be susceptible to a similar range of pathogens.

Fisher

Knowledge on fisher (*Pekania pennanti*) pathogens benefits substantially from screening linked to reintroduction programmes (Powell et al., Chapter 11, this volume). Brown et al. (2008) conducted an exemplary study in the Hoopa Valley Indian Reservation in northwestern California, sampling 76 fishers caught 115 times between December 2004 and March 2007. As with martens, **CDV** is prevalent among fishers, with antibodies present in five of 98 exposed (see also Gabriel et al. 2006; Keller et al. 2012). In addition 28 of 90 had been exposed to **CPV**, four of 95 to CAV-2, five of 96 to Canine Herpes Virus (CHV), 24 of 102 to ***Borrelia burgdorferi* (Lyme disease)**, 45 of 77 to ***T. gondii*** (and *Sarcocystis* spp.; see Larkin et al. 2010), and 60 of 79 to ***Anaplasma phagocytophilum*** (tick-vectored bacteria, causing fever). None of 34 samples had detectable antibodies against *Yersinia pestis* or *Bartonella* spp. (bacteria); interestingly, four of 99 serum samples had been exposed to mosquito-vectored **West Nile Virus**. PCR confirmed current CDV infections in 18 of 98 individuals, with three of 78 currently suffering from *A. phagocytophilum.* Exposure to *T. gondii* was significantly more common in females (69%) than males (49%). Other pathogen exposures did not vary with sex or age, except CPV—increasing with age. Of 15 radio-tracked fishers found dead during the study there was no clear link between pathogen exposure and mortality. The authors stress that because these viruses can be transmitted through saliva, nasal discharge, and faeces, traps and handling equipment must be disinfected as good practice during trapping or reintroduction programmes. Emphasizing the importance of understanding an animal's pathogen load in reintroduction projects, Peper et al. (2016) screened 58 fishers for CDV antibody prior to their release into Pennsylvania, although only five of 58 showed a weak-positive antibody reaction, and none exhibited clinical signs of CDV.

In addition, fisher recovery programmes present another potential vector of rabies. While only one of 41 fishers tested by Larkin et al. (2010) was positive for **rabies** (a male with the Eastern Raccoon strain), the risks involved in reintroducing fishers into new regions are evident. **Mustelid Herpes Virus (MHV)** seems rare in fishers (see European badgers, below), although it contributed to the death of a fisher suffering cutaneous ulcers in a zoo in Quebec (Gagnon et al. 2011). **Lungworm** nematode infections also blight fishers; cause of death in 10 of 167 fishers (representing 10 of 21 natural mortalities) in California was attributed to interstitial pneumonia or bronchopneumonia, from combined lungworm and bacterial infections (Gabriel et al. 2015).

Blighted badgers

European badgers (*Meles meles*) are among the most thoroughly researched musteloids, where the majority of studies have focused on their role in **bovine tuberculosis (bTB)** epizootiology (e.g. Macdonald et al. 2015c; Abdou et al. 2016). Furthermore, there is a substantial body of work examining how other pathogens interact with badger population dynamics.

Woodroffe and Donnelly (Chapter 20, this volume) discuss the socio-politics and management of bTB in European badgers at length, and so we mention this here only for completeness, from a pathological perspective. bTB is a bacterial infection caused by ***Mycobacterium bovis***, that primarily causes disease of the respiratory system, resulting in tubercles forming within the lungs, or other organs (including the skin, in the case of bite wounds). These granulomas can form lesions, from which *M. bovis* bacilli can be transmitted, typically expelled as a coughed aerosol, or contracted through bite wounding (Corner et al. 2011), and from faeces sniffing (Tinnesand et al. 2015).

The European badger is an excellent host for *M. bovis*, able to support persistent chronic infection for long periods, only advancing to severe morbidity once immunity is compromised (Corner et al. 2011), and infected animals can reproduce successfully. In the Republic of Ireland, as part of a large-scale bTB management programme (2007–2013), 1447 of 10,231 of the badgers culled were bTB positive, diagnosed from basic post-mortem with infection confirmation by tissue culture; although a significant decrease in bTB prevalence from 26% to 11% occurred across this time series (Byrne et al. 2015b). In a smaller scale Irish study by Murphy et al. (2010), 78 of 215 badgers were culture positive after advanced post-mortem techniques were used, 26 of 215 exhibiting gross lesions. That is, 66.7% of confirmed infected animals exhibited no gross pathology. In the UK, in the Randomised Badger Culling Trial (RBCT), conducted across the southwest of England, 1020 of 6432 badgers (16%) were confirmed positive for *M. bovis* by culture or histopathology; 393 had gross lesions, thus 61.5% of infected animals did not have gross pathology (Jenkins et al. 2008a). Despite the low sensitivity of ante-mortem tests, and benefiting from using three tests in parallel (see Woodroffe and Donnelly, Chapter 20, this volume), 156 of 294 badgers tested by Carter et al. (2012) in a bTB hotspot (Gloucestershire, UK) were positive. If an animal hosts an infection below the threshold of assiduous detectability, it will not be experiencing any associated morbidity. While we stress that the true infection prevalence in the RBCT data (and the badger management data from Ireland; Byrne et al. 2015b) is probably on average double what was detected, due to low diagnostic sensitivity (Crawshaw et al. 2008; Murphy et al. 2010), even with an infectious burden of around 50%, badger populations can demonstrably maintain or even increase population density. Nevertheless, non-symptomatic animals could represent a potential 'ticking bomb' reservoir if populations become stressed or perturbed.

One theory relating to susceptibility is that because European badgers spend a lot of time digging and residing below ground they are exposed to many non-pathogenic environmental bacteria, lessening their immune systems' sensitivity to *Mycobacteria.* Nitric oxide and Toll-like Receptor-9 (TLR9) are critical components of macrophage anti-TB responses, yet Bilham et al. (2017) report an atypical immune deficiency among badgers, where their macrophages do not produce nitric oxide in response to TLR agonists.

Although advanced lesions are characteristic of bTB, smaller lesions can be confused with **adiospiromycosis** caused by the saprotrophic fungus ***Emmonsia parva*** (formerly *Chrysosporium parvum*), to which badgers (and other musteloids) are susceptible; although this causes little pathology (Křivanec et al. 1976). The fungus ***Histoplasma capsulatum*** has also been detected in badgers (Jensen et al. 1992; Bauder et al. 2000)—a risk for cavers or researchers entering caves (notably a disease of bats). Other causes of lung disease include **lungworms** (*Aelurostrongylus falciformis*, Davidson et al. 2006; *Crenosoma melesi*, Magi et al. 1996; *Capillaria aerophila*, Hancox, 1980), and occasional pulmonary **osseous metaplasia** (Gallagher and Clifton-Hadley 2000).

Before we leave bacteria, badgers can develop severe gingivitis and periodontal disease, especially as they age. Bacteria entering the bloodstream through the gums can adhere to the inner lining of the heart and mitral valve producing, plaques causing potentially life-threatening endocarditis (Lockhart et al. 2009). This is a condition observed during post-mortem of badgers in Wytham Woods, Oxford (C. Newman, pers. obs.).

Viral diseases are less well-researched in European badgers. Badgers seem quite resistant to **CDV**: Frölich et al. (2000) detected no CDV antibodies in badger sera, in Germany, but did establish CDV presence in lung tissue, by PCR; in England 0 of 468 badgers were seropositive for CDV (Delahay and Frölich

2000). Among adenoviruses, we found no records of badgers suffering from CAV (Duarte et al. 2013), or hepatitis virus. **Mustelid Herpes Virus (MusHV-1)** is, however, common (King et al. 2004) with Sin et al. (2014) detecting MHV in 354 of 361 blood samples by qPCR in Wytham Woods, Oxford. Adults had lower MHV intensity than cubs, and MHV infection intensity impaired weight/length ratio, although no symptomology was established. MHV infection intensity was not associated with MHC heterozygosity; herpes viruses can evade immune detection and establish lifelong infections through MHC down-regulation. A new polyomavirus, tentatively named *Meles meles polyomavirus 1* (MmelPyV1) has also been discovered in this population (Hill et al. 2015).

Rabies can afflict European badgers, although prior to the elimination of rabies in Europe it was a minor host, after foxes (*Vulpes vulpes*), raccoon dogs (*Nyctereutes procyonoides*), stone martens, etc. (Wandeler et al. 1974). Nevertheless, badger populations were decimated by den gassing to control the disease (Wandeler et al. 1974). In Zhejiang Province, China, seven people died from being bitten by sluggish rabid badgers they attempted to capture (two badgers were eaten) in an epizootic, 2002–2004 (Zhenyu et al. 2007).

Badgers seem quite robust to parvoviruses; Leimann et al. (2015) detected no **AMDV** among badgers in Estonia, although Knuuttila et al. (2015) found seven of 26 badgers with antibodies present in Finland. AMDV was not present in badger samples screened in Wytham Woods, Oxford (C. Newman, pers. obs.). Duarte et al. (2013) detected three of three badgers with **CPV** by PCR in Portugal, while Barlow et al. (2012) report parvovirus enteritis in five cubs in the UK, the cubs suffering severe diarrhoea and eventually dying. Indeed, European badgers seem especially prone to enteric infections, although most arise from protozoan infections.

Coccidial gut parasites occur commonly (Anwar et al. 2000), both ***Isospora melis*** and highly pathogenic ***Eimeria melis*** causes diarrhoeal enteritis, epithelial sloughing, steatorrhoea, and malaise, and consequently (primarily among cubs that lose fluid) malabsorption and chachexia (chronic wasting). Newman et al. (2001) found 106 of 159 cubs infected with *E. melis*, whereas prevalence dropped to 21 of 242 among adults. Badger faecal samples (n = 50) collected in Ireland by Stuart et al. (2013) were positive for *Isospora*-like oocysts (8/50), as well as *Uncinaria criniformis* (20/50), and *Eucoleus aerophilus* (= *Capillaria aerophila*; 3/50).

When juvenile coccidial infections coincide with shortages of food and water, typical during dry spring weather (Newman et al., Chapter 21, this volume), cub cohort mortality rates peak at over 90%, with mortality compounded by malnutrition (Newman et al. 2001; Nouvellet et al. 2013). In years of plenty, over 50% of the cohort will typically survive, despite similar infectious intensities (Macdonald and Newman 2002). Cubs surviving infection, however, suffer stunted growth of 5–7% (Newman et al. 2001). In response to coccidial infection badger cubs exhibit unexpectedly precocious development of antioxidant capacity. Bilham et al. (2013) found that in a year with a dry spring, the mean antioxidant capacity of 16-week-old cubs was equivalent to that of prime-age adults (1–5 years old). ***Coccidiosis*** is likely an important disease for juveniles of many musteloid species; every vertebrate species that has ever been examined intensively, over a broad geographic range, has been found to have at least one coccidian species unique to it, and can have more (Duszynski and Upton 2001). While studies mention coccidia in other musteloids (e.g. raccoons—Yakimoff and Matikaschwili 1933; ferrets—B. Williams et al. 1996), the full impact on juvenile survival and recruitment remains to be properly established.

Anwar et al. (2006) report that 63 of 90 badgers tested positive for ***T. gondii*** antibodies, but without specific pathology. Two blood protozoans, *Babesia missiroli* (203/263) and *Trypanosoma pestanai* (20/263—flea-transmitted, see Lizundia et al. 2011) have also been detected (Macdonald et al. 1999)—again without evident pathology. In addition, European badgers can host a variety of helminths, described by Torres et al. (2001) and Millán et al. (2004); with Hancox (1980) providing a comprehensive list of badger parasites. Notably, European badgers appear unusually resistent to mange mites, compared with sympatric foxes (Balestrieri et al. 2006).

American badger

The details and implications of disease in American badgers (*Taxidea taxus*) are less well resolved, although Quinn et al. (2012) provide thorough disease screening for twelve American badgers from California, with serological evidence of **CDV** (9/12; CDV has been noted as the cause of death for American badgers in captivity—see Armstrong 1942), ***T. gondii*** (6/12), ***Francisella tularensis*** (3/12; bacterial agent of tularaemia), ***Anaplasma phagocytophilum*** (1/12; bacterium causing tick-borne 'pasture' fever, with zoonotic infection causing human granulocytic anaplasmosis) and

Bartonella spp. (3/12). They also report anticoagulant rodenticides brodifacoum and bromadiolone in badger tissues, commonly used to control rodent pests. Goodrich et al. (1994) report successful CDV vaccination in five of six American badgers, which developed antibodies, whereas the five control badgers they were housed with did not, implying that the vaccine virus was not transmitted.

The American badger is a competent **rabies** host, but prevalence seems very rare at just 40 of 2851 cases investigated by the CDC, 1960–2000 (Krebs et al. 2003), among secondary host species excluding raccoons, skunks, and foxes (see below).

O'Toole et al. (1993; 1994) found **subepidermal vesiculobullous filarial dermatitis**, caused by the nematode *Filaria taxideae*, in 51 of 64 American badgers killed as part of the black-footed ferret programme. One of five badgers tested in western Dakota was positive for *Yersinia pestis*, causing sylvatic **plague** prevalent among black-footed ferrets (Dyer and Huffman 1999).

Taxidea can contract ***Sarcocystis*** sp. (Cawthorn et al. 1983) although we found no studies mentioning coccidiosis, explicitly (cf. *Meles*). There are anecdotal mentions of American badgers being investigated as a possible host for *M. bovis*, but with negative results, for example, Schmitt et al.'s (2002) study in Michigan; although 340 of 65,000 white-tailed deer harboured bTB. Bruning-Fann et al. (2001) also found no bTB in American badgers. Philippa et al. (2004) found no AMDV in *Taxidea* in Canada. References to other viruses, such as CPV, CAV, and MHV are also vague in the limited literature, precluding any conclusive discussion.

Other badgers

Studies of diseases in tropical badgers are even more sporadic. Although honey badgers (*Mellivora capensis*) have not been found infected with host CPV, **FPV** has been detected (Steinel et al. 2001; see Raccoons, below); and we note that honey badgers are a minor host of **rabies** among a broad guild of Carnivora in Africa (e.g. Röttcher and Sawchuk 1978). Similarly, Zhou et al. (Chapter 13, this volume) report **rabies** in Chinese ferret badgers (*Melogale moschata*) (S. Zhang et al. 2009; Liu et al. 2010), as well as being implicated as a zoonotic vector of **severe acute respiratory syndrome coronavirus** (**SARSCoV**—Guan et al. 2003), which has led to badger species being culled in China.

Listless lutrines: otter diseases

United in their dependence upon the aquatic ecotope, the otters are actually a diverse tribe, spread from the northern tundra to the tropics, and ranging from solitary to group-living societies (Macdonald and Newman, Chapter 6, this volume; Macdonald et al., Chapter 1, this volume). The most familiar species are the Eurasian (*Lutra lutra*) and North American river (*Lontra canadensis*) otters—for which the range and dynamics of prevalent diseases are best known.

The US Forest Service lists several viruses infecting North American river otters, including **rabies, CDV, PPV**, and **CIH**—all transmittable to (and from) pet dogs. From 1960 to 2000, however, only 45 of 2851 rabies cases investigated by the CDC involved river otters (Krebs et al. 2003); nevertheless this does not imply it is not a significant issue within localized otter populations. Among 64 river otters scheduled for relocation in New York State, Kimber et al. (2000) detected the presence of antibodies against CDV, **canine herpesvirus-1**, and **CPV-2**.

North American river otters are sympatric with American mink, with guild overlap, and so exposure to **AMDV** would seem inevitable. Nevertheless, Bowman et al. (2014) found no evidence of AMD in otters in Ontario, leading them to speculate that AMDV spillsover primarily from mink farm manure composting, rather than direct mink presence in rivers. Kimber et al. (2000) report that American river otters are quite resistant to experimental AMDV infection, although in Nova Scotia, where mink manure composting is widespread (see above), two of 11 river otters tested positive (Farid 2013).

As part of a reintroduction programme, the Oklahoma Department of Wildlife Conservation found that two of 10 otters screened, pre-vaccination, for release had **feline rhinotracheitis** antibodies present (caused by feline herpesvirus-1); one also had antibodies to **feline calcivirus** (Hoover et al. 1985). Symptoms of rhinotracheitis include increased mucus production, nasal discharge, coughing, sneezing etc., which usually resolves within 3–4 days (in cats), although secondary bacterial sinusitis can follow. Otters are also susceptible to the related parvovirus**, feline panleukopaenia** (Kimber and Kollias 2000; see Raccoons).

Enteric protozoans reported by Kimber and Kollias (2000) include the frequent incidence of ***Isospora*** (coccidian), with ***Sarcocystis*** in Eurasian river otters (Gjerde and Josefsen 2015). A total of 46 of 103 otters screened in North Carolina had antibodies to

Toxoplasma gondii, which causes a substantial issue in the UK, infecting 107 of 271 Eurasian otters examined by Chadwick et al. (2013), probably via domestic cat faeces. Despite swimming in contaminated waters, otters are not particularly susceptible to ***Giardia***, although occasional cases have been noted, along with *Cryptosporidium* (Gaydos et al. 2007). Otters are not a significant host of *Trypanosoma cruzi*.

In terms of nematodes, Lankester and Crichton (1972) found nine of 118 otters suffering from ***Skrjabingylus 'lutrae'*** in Ontario, with 50 of 373 skulls examined by Addison et al. (1988) bearing cranial lesions. Otters also have a specific nematode, ***Dracunculus lutrae***, infecting the subcutaneous fascia on the legs and causing erythema and pruritis—potentially leading to skin ulcers (Crichton and Beverly-Burton 1973). These authors found 26 of 72 and 177 of 203 otters examined in two studies in Ontario to be heavily infected; 17 of 25 individuals exhibited *Dracunculus* infection in Arkansas (Tumlison et al. 1984). Simpson et al. (2009) report the potentially damaging bile fluke ***Pseudamphistomum truncatum*** in road traffic accident carcasses examined in the UK.

Fungal diseases can also be an issue for river otters, notably **pulmonary coccidiodomycosis** (aka 'valley fever') and **adiaspiromycosis** (Simpson and Gavier-Widen 2000), to which the Mustelidae appear to be particularly susceptible (Simpson 2016). Although most infections are subclinical, this can cause granulomatous lung lesions leading to fatalities (Simpson and Gavier-Widen 2000). In addition, otters can contract purulent pleuritis, resulting in an accumulation of pus in the pleural cavity, and purulent peritonitis, causing abdominal inflammation (Kimber and Kollias 2000).

A note on ecotoxicology: the effects of PCBs, organochlorides, endocrine disrupting chemicals, mercury etc., in natural water courses (e.g. Jönsson et al. 1993; Pountney et al. 2015) is a further serious issue for otters (and mink), where otters have even been proposed as sentinel biomonitors (Carpenter et al. 2014). This is, however, a subject in its own right—beyond the scope of our chapter here.

Tropical otter species are less well researched (Gallant 2007), especially their diseases. This by no means implies that they suffer from fewer, or less serious, pathogens, simply that disease remains a substantial unknown in tropical otter conservation management. Certainly, similar diseases to those affecting northern river otters afflict tropical otters in captivity, for example, Gjeltema et al. (2015) report an outbreak of **canine parvovirus type 2c** in a group of nine Asian small-clawed otters (*Aonyx cinereus*), where one otter died, despite treatment. De Bosschere et al. (2005) report CDV in captive Asian small-clawed otter. Schenck and Staib (1995; see also Schenck 1999) mention giant otters' (*Pteronura brasiliensis*, Groenendijk et al., Chapter 22, this volume)—especially cubs—susceptibility to **CPV**, spread by feral dogs.

The IUCN have identified that infectious diseases threaten the viability of both the South American marine otter (*Lontra felina*) (Pedersen et al. 2007) and the southern river otter (*Lontra provocax*), due to **CDV** circulating in the species ecosystem (Sepúlveda et al. 2014; Valenzuela-Sanchez and Medina-Vogel 2014). The presence of invasive North American mink, which escaped from fur farms, appears to act as a 'bridging host' from dogs to otters (Sepúlveda et al. 2014).

Sickly sea otters: diseases in the marine environment

Sea otters (*Enhydra lutris*) are an 'Endangered' species (Macdonald et al., Chapter 1, this volume; Estes et al., Chapter 23, this volume) and disease plays a major part in their population dynamics. In southern Monterey Bay, Kreuder et al. (2003) found that diseases caused by parasites, bacteria, or fungi (plus diseases without a specified aetiology) were the cause of death in 67 of 105 freshly deceased (or euthanized, due to serious morbidity, n = 27), beach-cast southern sea otters. Protozoal encephalitis, caused by ***Toxoplasma gondii***, as well as ***acanthocephalan***-related disease (thorny-headed parasitic worm), were especially important, accounting for 40 of 67 mortalities. Examining 162 sea otter carcasses, Mayer et al. (2003) identified that although infection with the acanthocephalan ***Corynosoma enhydri*** (n = 153) never caused significant damage to the host's intestine, even at high infectious intensity, ***Profilicollis* spp**. (n = 75) were the cause of death in 21 carcasses; either directly, due to perforation of the intestinal wall and peritonitis (n = 16), or indirectly, due to inhibition of host nutrient uptake, or depletion of host energy reserves required to fight chronic infections (n = 5). There was also a significant age-class effect; *Profilicollis* spp. was the mortality agent primarily for juvenile and old-adult females. Notably, 47% of these carcasses were infected by three species of **Digenean flatworms**, sometimes reaching massive infectious intensity (>3000 per cm^2). Ecologically, mortality patterns also relate to foraging conditions on the sea-bed, where sandy areas support larger populations of definitive acanthocephalan hosts, such as surf scoter

ducks (*Melanitta perspicillata*). As with badgers (above), climate also proves influential, with these ducks—and thus otter infections—being more prevalent during El Niño years.

T. gondii infection in sea otters exposes the subtleties of how disease can operate: infection is significantly associated with cardiac disease, plus fatal shark bites are over three times more likely to be the cause of death for individuals with pre-existing *T. gondii* encephalitis—where toxoplasmosis is well-recognized to enhance vulnerability to predation in a variety of species (Berdoy et al. 2000). While all warm-blooded mammals can contract toxoplasmosis (with 30–50% seroprevalence in human populations), the life cycle of this apicomplexan requires a felid as its definitive host, to complete its life cycle; seemingly unlikely in a marine environment. So where do these toxoplasma oocysts come from? Sea otters do not prey on known intermediate hosts for *T. gondii*, and vertical transmission appears to play a minor role in maintaining infection (Conrad et al. 2005). The most likely source of infection is, therefore, via environmentally resistant oocysts, shed in the faeces of domestic and wild felids and transported through freshwater runoff into the marine ecosystem, thence concentrated in filter-feeding marine invertebrates consumed by sea otters. Along the California coast, Miller et al. (2002) found a rate of 49 of 116 *T. gondii* seroprevalence for live otters, and 66 of 107 for dead otters, but with spatial clustering at distinct sites. Contrary to presumptions, this study found no association between seropositivity and human population density, or exposure to sewage entering the sea. Rather sea otters sampled near areas of maximal natural freshwater runoff (though potentially contaminated) were approximately three times more likely to be seropositive than otters sampled in areas of low flow.

Another protozoan, ***Sarcocystis neurona***, causes illness and death in southern sea otters (Miller et al. 2010). Unlike enzootic *T. gondii*, however, *S. neurona* is epizootic. Miller et al. (2010) describe an outbreak in Morro Bay, California that resulted in 40 sick or dead otters being recovered along an 18km stretch of coastline; the largest spike in monthly mortality over a 30-year monitoring period. Of these animals, 94% subsequently tested positive for *S. neurona* during this outbreak. As with many epizootiological outbreaks, other contributory factors seem to have been involved. The event was preceded by a major rainstorm, with terrestrial runoff potentially the source of infectious oocysts; an abundance of razor clams likely drew foraging otters inshore to the affected area; and concurrent exposure to the marine biotoxin domoic acid (produced by marine algae, concentrated in shellfish) could have exacerbated susceptibility to *S. neurona* and enhanced the neurological symptoms.

MHV can affect sea otters; with *MusHV-1* (a novel gamma virus related to *MHV-1* infecting *Meles* spp.) first noted among those northern sea otters necropsied, or admitted for treatment, after the 1989 Exxon Valdez oil spill in Alaska; many exhibiting oral ulcerations and plaques with epithelial eosinophilic intranuclear inclusions (Tseng et al. 2012). Subsequently, testing of live otters in the Kodiak Archipelago confirmed the general prevalence of *MusHV-2*. Other disease screening connected to the Exxon spill did not reveal any evidence of adenovirus, coronavirus, or feline rhinotracheitis (Spraker 1990); although this does not imply any fundamental resistance to these viruses. Additionally, AMDV does not seem to be a mortality risk for sea otters, although Goldstein et al. (2009) uncovered strong serologic evidence that, subsequent to the 2002 phocine distemper epizootic that killed >30,000 harbour seals in the North Pacific, 30 of 77 live-captured sea otters sampled in the Kodiak Archipelago had been exposed to a **PDV-like morbillivirus**.

With respect to treating sea otters, and exposure to zoonotics, the California Department of Fish and Game stress that first responders should take caution against splash infections with marine *Brucella* bacteria and the fungus *Coccidioides immitis*, which causes potentially serious 'Valley Fever' in humans (Huckabone et al. 2015).

Before we leave sea otters, we must mention how they exemplify that the loss of genetic diversity, or 'bottlenecking', can reduce ability to resist disease. Extensive over-hunting of sea otters in the eighteenth and nineteenth centuries (see Harrington et al., Chapter 7, this volume) has resulted in reduced genetic variation and inbreeding depression (Larson et al. 2002). This hampers the recovery of present-day populations because they exhibit an unusually high susceptibility to cancers and neoplasms, such as mammary adenocarcinoma (Newman and Smith 2006).

Procyonid pathogens: raccoons and their kin

Raccoon (*Procyon lotor*) diseases are a significant source of concern because, of all the musteloids, raccoons associate most closely with people, and carry diseases presenting substantial risks to us. Information on

raccoon disease epidemiology is thus important for developing control programmes to manage raccoon–human and raccoon–domestic animal interactions. Undoubtedly, other procyonids are susceptible to similar diseases, however studies are much fewer.

Perhaps the raccoon-specific pathogen of greatest human concern is the **raccoon nematode, *Baylisascaris procyonis.*** Prevalence is high among especially juvenile raccoons in North America (noting again age-susceptibility effects), ranging from 70% to 90% (Sorvillo et al. 2002), but *B. procyonis* exhibits little pathology within the definitive raccoon host—simply repeating a cycle of enteric infection, development, and egg production. Reproduction of the nematode cannot occur in paratenic (intermediate) hosts; instead larvae penetrate the gut wall and are chased through organs and tissues by the intermediate host's immune system, as the body attempts to encyst and destroy the parasite. Although human cases are rare, they can be severe if larvae invade the eye (*ocular larva migrans*)—leading to blindness—or even fatal if larvae invade vital organs (*visceral larva migrans*), or the brain (*neural larva migrans*). Spread via the faecal–oral route, raccoon latrines are the principal source of contamination infecting people. The propensity for young children to explore the environment orally, and to ingest eggs, is reflected in a median age of infection of 13 months (Murray and Kazacos 2004). A study in Chicago reported subclinical seropositivity in 30 of 389 children 1–4 years of age (Roussere et al. 2003). Furthermore, domestic pets such as cats and dogs can become infected. Page et al. (1998) report that 14% of the raccoon latrines they investigated in Indiana included infectious faeces. Despite these risks, which are increasing as raccoons invade urban environments (drawn, in particular, to swimming pools; Bradley and Altizer 2007), and the severity of neurologic symptoms, only 25 cases of human *B. procyonis* were documented by the CDC in the US to 2012, 16 leading to severe eosinophilic meningitis, with six fatalities. Nevertheless, due to the resistance of these nematode eggs to heat and disinfectants, *B. procyonis* is even a concern as a bioterrorism agent (Gavin et al. 2005). Other musteloids can carry species-specific *Baylisascaris* spp., including the kinkajou (*Potos flavus*), infected with *B. potosis*; however, no other *Baylisascaris* spp. pose such a risk to humans.

A major zoonotic protozoan (haemoflagellate) infecting raccoons is ***Trypanosoma cruzi*** (Walton et al. 1958), the causal agent of American **trypanosomiasis** or **Chagas disease**. *T. cruzi* is an important public health concern in Latin America, where 10–12 million people are estimated to be infected (Morel and Lazdins 2003). Infection can progress to chronic phase myocarditis in humans and dogs, and thus is of both medical and veterinary importance. Yabsley and Noblet (2002) found 104 of 221 raccoons from South Carolina and Georgia seropositive for *T. cruzi* antibodies, especially along coastal regions, with females appearing to be more susceptible than males. This study also screened for *Leishmania* antibodies, but all raccoons tested negative. Hancock et al. (2005) found a comparable *T. cruzi* antibody seroprevalence in 153 of 464 raccoons over three years in a suburban area of Virginia near Washington, DC.

While humans fear *Baylisascaris procyonis* and Chagas disease, the leading pathogenic cause of death among raccoons is **distemper**, where they are susceptible to both **CDV** and **feline distemper**, or **feline panleukopaenia (FPV)**; quite different viruses, but equally contagious and potentially fatal, especially among juveniles. FPV from domestic cats poses a threat to raccoons in urban settings, with fomites (substrates able to maintain pathogens in the environment) including urine, faeces, and even fleas. Symptoms include fever, diarrhoea, malabsorption, dehydration, and anaemia. CDV, spread largely to raccoons by unvaccinated domestic dogs in urban settings, tends to outbreak in cyclical epidemics among raccoon populations. Roscoe (1993) identified seventeen epizootics of CDV involving at least 615 raccoons between 1977 and 1991 in New Jersey. He noted no specific sex or age bias among the infected. Lednicky et al. (2004) further draw attention to how outbreaks of different strains of CDV caused different extents of mortality among raccoons in urban Chicago, with an emergent strain of CDV variant A75/17 causing large syncytia (a multinucleate protoplasmic mass) in brain and/or lung tissue.

In west-central Illinois, a study by Mitchell et al. (1999) undertook extensive raccoon disease screening and found that 85 of 368 were seropositive for CDV; furthermore, 222 of 459 were seropositive for ***Leptospira interrogans***, 82 of 479 were seropositive for **pseudorabies virus** (**PV**), and 108 of 379 seropositive for ***T. gondii***. For each of these infections, seroprevalence increased significantly with age. In addition, seroprevalence for *T. gondii* was higher during the spring (73%) than in autumn (33%).

Raccoons are also susceptible to bacterial diseases, and are notable sentinels for **tularaemia** and **leptospirosis** zoonoses (Duncan et al. 2012). Tularaemia is caused by the bacterium *Francisella tularensis*, which occurs throughout North America, Europe, and Asia,

but not in the Southern Hemisphere (Mörner 1992). Vectored by arthropods, most notably several tick species, symptoms include lymph node necrosis and necrotizing granulomas. While tularaemia type-B is aquatic, type-A infects terrestrial mammals (wildlife, pets, and people), often leporids; however, small rodents and rabbits die quickly from infection, while larger hosts, such as raccoons (and skunks) survive with the disease, and can bring infection to public sites. Leptospirosis is spread through contaminated urine. In 90% of human infections with *Leptospira* bacteria, only mild flu-like symptoms present; however, more potent forms include Weil's disease, which can cause organ failure and death. From necropsy of 65 raccoons in Colorado, Duncan et al. (2012) found 20 positive for *Leptospira* from immunohistochemistry, of which one was PCR positive; no gross lesions were evident, but lymphoplasmacytic interstitial nephritis and pulmonary silicosis were seen. Animals with kidney inflammation were seven times more likely to be positive for *Leptospira.* Only one raccoon was positive for *Francisella*, from both IHC and PCR, and exhibited localized histiocytic cells within a pulmonary granuloma.

Raccoons are also a common host for various ticks, transmitting Lyme disease (Ouellette et al. 1997), of substantial zoonotic concern in many parts of North America (www.cdc.gov/lyme), where again, this issue is heightened by raccoons' close affinity for people.

Raccoon rabies

While many mammals, and especially carnivores, are susceptible to **rabies** (Figure 9.1; and, notably, see discussion on skunks), again the raccoon is of especial concern because of its close association with people. It is important to emphasize too that while rabies is almost always fatal in humans, mortality rates among naturally infected dogs and cats are 53% and 29% respectively—although if the disease reaches the furious stage it is always fatal (Tepsumethanon et al. 2004).

Raccoons are the most frequently reported wildlife host of rabies in the USA, accounting for 36.5% of the total 6154 rabid animal cases investigated by the CDC in 2010 (Figure 9.2) and 32.8% of the 6031 cases in 2011; followed by skunks (2010: 23.5%; 2011: 27.0%), bats (23.2%; 22.9%), foxes (7.0%; 7.1%), along with other wild animals, including rodents and lagomorphs (1.8% in 2010) and cats (5% in 2011). Only two cases of rabies involving humans occurred in 2010—both contracted via bats—with six cases in 2011, three derived from bats; three cases of canine rabies were acquired outside of the USA (Blanton et al. 2011; 2012). The first human death connected to raccoon rabies occurred in Virginia in 2003 (CDC 2003).

Raccoon rabies was much less prevalent in the US prior to 1950, but by the 1970s the incidence of raccoon rabies began to rise, especially in Florida and Georgia. In 1977, a variant of raccoon rabies, distinct from the southern variant, was detected in Virginia and West Virginia; this has since spread north along the eastern seaboard to Ontario, Canada, and converges with the southern variant in North Carolina.

In response to the arrival of rabies in Ontario, Canada, in 1999 (Rosatte et al. 2001), three control strategies were implemented: population reduction (PR), trap-vaccinate-release (TVR), and oral rabies vaccination with baits (ORV). Overall, 1202 raccoons (and 337 striped skunks) were captured and euthanized, achieving an 83–91% population density reduction in the target areas (from

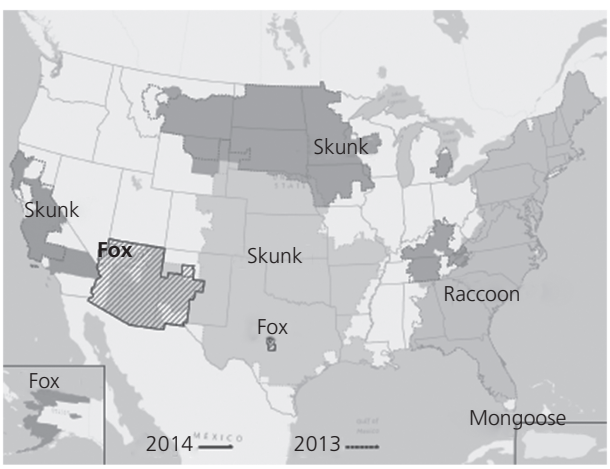

Figure 9.1 Distribution of major rabies virus variants among mesocarnivores in the United States and Puerto Rico, 2008 to 2014. Source: Monroe et al. (2016) © US Centers for Disease Control and Prevention, available from www.cdc.gov.

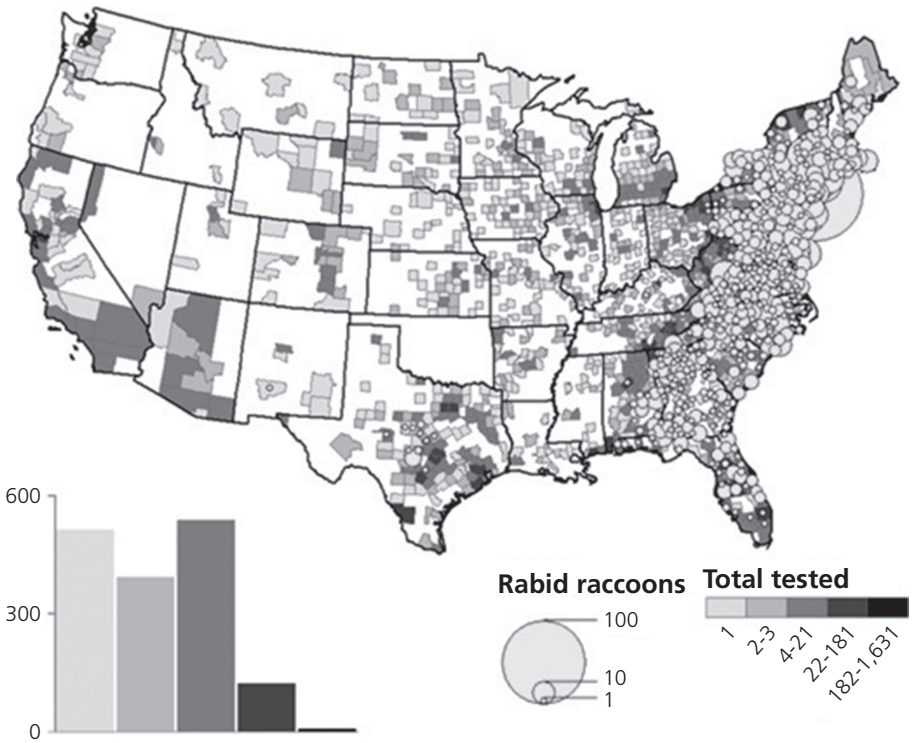

Figure 9.2 Rabid raccoons reported in the United States during 2014. Source: Monroe et al. (2016) © US Centers for Disease Control and Prevention, available from www.cdc.gov.

5.1–7.1 per km^2 to 0.6–1.1 per km^2), although all of these tested negative for rabies at post-mortem. Additionally, 1759 received intramuscular vaccination (along with 377 skunks and 856 domestic cats). To further contain the outbreak, about 81,300 baits, containing Raboral® V-RG oral rabies vaccine, were dropped by air in September 1999, to create an 8–15 km wide buffer zone (1200 km^2 area) of vaccinated raccoons immediately beyond the PR and TVR zones (see Esposito et al. 1988). At the time, this operation cost CAD 363,000.

During 1999–2003, raccoons accounted for 125 of 127 cases of rabid animals investigated in Ontario (Rosatte et al. 2006); however, these control measures have kept rabies prevalence far below levels seen in USA jurisdictions, where raccoon rabies is epizootic. In 2006 raccoon rabies variant accounted for just five of 229 (2.2%) of the recorded instances of rabies in Canadian wildlife (Veterinary Virology Dept., University of Saskatchewan). Because raccoons infected with rabies typically die within a few days, it is likely that the majority of incidences go unnoticed—reported cases are usually linked to aberrant behaviour, including aggression, fighting with dogs, vocalizations, malaise, and ataxia, and in some regions instances where raccoons are pierced by porcupine (*Erethizon dorsatum*) quills, implying a poorly judged attack. Seventy-eight percent of rabid raccoons in Ontario were adults, mostly three years of age or less, and 22% of rabid adult female raccoons had given birth in that breeding season. Winter denning and breeding seasons were associated with the annual peaks in outbreaks, where several morbid animals were often found in the same barn, implying cross-infection. Interestingly, of 291 serum samples from raccoons collected in an epizootic area of the mid-Atlantic States between 1982 and 1983, eight had titres of antibody to rabies virus (complete neutralization) of ≥1:25 (Jenkins et al. 1988), suggesting that some animals can survive infection.

Outside of North America, expatric raccoon populations are of concern in Germany because even though rabies has been eliminated from terrestrial wildlife, should rabies ever re-emerge, the extraordinarily high localized densities of raccoons in urban areas, notably around Kassel (Hesse), would be a risk factor (Vos et al. 2012). The only other country to have an expatric

introduced raccoon population is Japan (see Macdonald et al., Chapter 1, this volume), where rabies was eliminated in 1957. High urban densities of raccoons in Tokyo suburbs would, however, also pose a significant risk factor if rabies returned (as it has into South Korea; Kim et al. 2006), because these raccoons carry no rabies antibodies (Inoue et al. 2004). Of note, 2000 people die every year from rabies in China.

Tropical procyonids

Vector-borne diseases tend to be more common in tropical latitudes (Jones et al. 2008), and as warmer conditions spread polewards, with climate change (Lafferty 2009; Newman et al., Chapter 21, this volume), so too does the range of various tropical diseases. Nevertheless, knowledge of disease in tropical wildlife—outside of charismatic megafauna—is limited, and this is very apparent among the non-raccoon procyonids. Indeed, it is hard to give a realistic impression of the range of diseases in this group, where trawling the literature risks over-emphasizing minor conditions, while a lack of systematic studies likely under-emphasizes the prevalence and pathology of, especially, viral diseases known to impact raccoons.

Kinkajous (*Potos flavus*) are possibly the best-researched low-latitude procyonid (see Brooks and Kays, Chapter 26, this volume), and are known to suffer from several diseases similar to those established in raccoons. Given that carnivores are broadly susceptible to the same suite of viruses (Appel 1987), it seems highly plausible that kinkajou populations could suffer from similar viruses to raccoons; definitive records are, however, lacking. While screening vampire bats (*Desmodus rotundus*) for rabies virus in Peru (2002–2007), Condori-Condori et al. (2013) identified infection in a single kinkajou, but were unable to determine whether this infection arose from a single spill-over from a bat reservoir or an emerging host shift with ongoing transmission within kinkajous; they did, however, flag concern about the ability of kinkajous to host rabies.

All procyonids seem susceptible to **CDV** infection (Deem et al. 2000). Nevertheless, there are no reports of distemper in wild kinkajou populations, although Kazacos et al. (1981) caution that two young kinkajous, vaccinated with modified live-virus canine distemper, developed severe neurological symptoms, from which one recovered, while the other had to be euthanized, due to severe convulsions. The susceptibility of kinkajou to **FPV** purported by Miller (1961) still remains unsubstantiated.

Overstreet (1970) reports the occurrence of ***Baylisascaris procyonis*** in kinkajou in Colombia; the first record of *B. procyonis* in South America and from a wild non-raccoon host, although confirmed in captive kinkajou in Guyana (Tokiwa et al. 2014). It is, however, impossible to get a sense of the epidemiological impact of this disease on kinkajou populations in general from such limited data.

While Yabsley and Noblet's (2002) negative result for ***Leishmania*** (a trypanosome) antibodies in raccoons is not conclusive for raccoons a whole (especially Central American populations), natural *Leishmania* infection has been reported for kinkajous in Panama (Thatcher et al. 1965). Pajot et al. (1982) reported that one of seven kinkajous screened in French Guiana was infected with *Leishmania braziliensis guyanensis*; Linnaeus's two-toed sloth (*Choleopus didactylus*) appears to be the main mammalian host with seven of 15 infected. Vectored by phlebotamous sandflies, leishmaniasis causes cutaneous ulceration, mucucutaneous ulcers in the mouth and nose, and can progress to a visceral ulcerative stage. Notably, screening tissue from four kinkajous and an olingo (*Bassaricyon* sp.) in Panama, Herrer et al. (1966) found no evidence of *Trypanosoma cruzi* infection. Captive kinkajous, in zoos, are occasionally susceptible to bacterial leptospirosis infections (Lilenbaum et al. 2002), although not recorded in the wild (Bunnell et al. 2000). Buckley (1930) describes an account of the lungworm *Crenosoma potos* in the kinkajou.

Knowledge on coati (*Nasua* spp.) diseases, and especially population impacts, is similarly limited. Working in Brazil, Nunes and Oshiro (1990) give the first report of ***Trypanosoma evansi*** in the ring-tailed coati (*Nasua nasua*), prevalent in four of 16 individuals tested—the same strain isolated from local capybaras (*Hydrochoerus hydrochaeris*) and dogs. Ring-tailed coatis are also susceptible to ***Trypanosoma cruzi***. Using a miniexon molecular assay, Herrera et al. (2008) identified high serum prevalences and high parasitaemias of three distinct *T. cruzi* genotypes among 158 individuals, with single infections by TCII (32.1%), TCI (28.0%), and Z3 (7.1%), as well as mixed infections by TCI/TCII (10.7%) and TCI/Z3 (3.6%). Seven of 20 white-nosed coatis, tested by PCR in Costa Rica, were also positive for *T. cruzi* (Mehrkens et al. 2013), all of which were also positive for *Mycoplasma* and *Babesia*. And because coatis attain high local densities and inhabit a variety of habitats, coatis may play a major role in the maintenance and dispersion of *T. cruzi*.

In terms of viral infections, coatis can contract **FPV** (Johnson and Halliwell 1968), but there appears to be

no literature describing the implications of parvoviruses in wild populations. Miller (1961) documents **CDV** in coatis, and three of 10 white-nosed coatis (*Nasua narica*) that died of natural causes while being tracked by Valenzuela (1998) were thought to have died from CDV (see also Hass and Valenzuela 2002), although this was not confirmed; significantly the other seven died from severe **scabies**, caused by the cat mite, *Notoedres cati*. Rabies also occurs in white-nosed coatis; three individuals found dead in Cancún, Mexico tested positive for rabies (Aréchiga-Ceballos et al. 2010), with another individual formerly cohabiting with these exhibiting antibodies to rabies—although the outbreak was thought to be of bat origin.

Reports of diseases among the less well-known olingos and the cacomistle (*Bassariscus sumichrasti*) are even sparser—although they are likely prone to similarly viral and trypanosome diseases. Pinto et al. (2009) give a first report of *Taenia mustelae* (cestode) parasitizing the bushy-tailed (or northern) olingo (*Bassaricyon gabbii*); it also infects raccoons in North America.

Sickly skunks

We have already alluded to skunks being the second most important terrestrial wildlife host of rabies in the USA, after raccoons, accounting for about 25% of annual incidences (CDC, www.cdc.gov). Skunks are actually the predominant host in the north- and south-central States, where the skunk species involved relates to regional rabies distribution patterns, and skunk rabies co-occurs with raccoon rabies in the east (Charlton et al. 1988) (Figure 9.1). In Canada, striped skunks (*Mephitis mephitis*) are the main terrestrial rabies host, accounting for 22 of 44 positive wildlife cases investigated by the Canadian Food Inspection Agency (CFIA) in 2014; 10 of these cases arose in Saskatchewan, 11 in Manitoba. Skunk rabies is typically epizootic and often cyclical.

Looking at the population level in detail, Greenwood et al. (1997) found that one of 23 striped skunks radio-tracked in North Dakota in 1991 became rabid, whereas in 1992, 30 of 50 became rabid. The survival rate of skunks from April to August 1991 was 0.85, but only 0.17 during April to July 1992. During the 14 days of clinical disease, preceding death, skunks reduced their hourly distance travelled by 50% and covered just 1497 ± 281 m per night, versus 2318 ± 281 m in the pre-clinical phase. Eleven of 36 skunks found dead or dying from rabies were located below ground. Time-series regression analysis, performed by Guerra et al. (2003), showed that skunk rabies variant was not, however, cycling independently among skunks in the Eastern USA, 1990–2000, rather rabid raccoon numbers predicted skunk epizootics with a one-month lag.

To establish better the extent to which striped skunks are a reservoir for emerging zoonotic infections, Britton et al. (2017) conducted molecular testing on 50 individuals necropsied in British Columbia, Canada. They found 43 of 50 with **AMDV** present, two of which had AMD symptoms. No rabies or CDV was detected; however, significantly, two skunks exhibited bronchopneumonia caused by **influenza A (H1N1) virus**, suggesting that skunks may represent a target population for reverse zoonosis of this strain of influenza A virus, linked to the 2009 H1N1 epidemic (see Britton et al. 2010). Working in Ontario, Nituch et al. (2015) also detected high rates of AMDV, with antibodies in 143 of 347 of striped skunk serum samples tested, along with AMDV nucleic acids in 14 of 40 skunk spleen samples tested by PCR—probably arising through spill-over from mink. By contrast, they found no AMDV antibodies in 144 raccoon blood samples.

CPV is another viral disease afflicting skunks. Even isolated on California's Santa Cruz Island, three of 31 spotted skunks (*Spilogale gracilis amphiala*) tested seropositive for CPV (Bakker et al. 2006), with one skunk positive for canine heartworm. None of these skunks showed evidence of exposure to canine adenovirus, canine herpesvirus, *Leptospira* bacteria, pseudorabies virus, or CDV—all infections to which skunks can be prone (Diters and Nielsen 1978). Distemper is an issue for skunks; 17 of 36 skunks evaluated for **CDV** antibodies by Woolf et al. (1986), in Illinois, tested positive, supported by diagnostic brain lesion evidence on post-mortem in 17 of 91 individuals. Symptomatically, it can be difficult to tell apart death from rabies or distemper, and so this type of confirmatory testing can be important. Karstad et al. (1975) report two cases of acute, fatal, **CIH** (CAV) in young striped skunks trapped in southern Ontario, after suffering convulsions and lethargy, rapidly progressing to coma.

While only one of 53 striped skunks studied by Ferguson and Heidt (1981) in Arkansas tested positive for rabies, 21 of 45 were positive for bacterial **leptospirosis** and 10 of 45 were positive for **toxoplasmosis**; none were positive for tularaemia. Again, these authors stressed the human health hazard this posed because these skunks used public areas. Prescott et al. (2002) suggested that an increase in the incidence of leptospirosis, and a change in serovar antigenicity among domestic dogs receiving veterinary care in Ontario, was

likely due to increased contact with urbanized populations of skunks and raccoons. Britton et al. (2015) reported nine of 49 striped skunks with *Leptospira interrogans* and 43 of 50 with *Salmonella* spp. Similarly, Ferris and Andrews (1967) isolated *Leptospira pomona* from eight of 75 striped skunks, with nine of 32 carrying antibodies (see also Roth et al. 1963). Interestingly, they report a significant relationship between infectivity (both isolations and serologic titres) with colder and wetter parts of the year—implying that leptospirosis patency is connected to general stress in skunks. Note—leptospirosis protection can be achieved in pet dogs through vaccination.

Tularaemia servoprevalence of 25.7% was detected in striped skunks (and raccoons) by a study by the Harvard School of Public Health, in Martha's Vineyard, Massachusetts—although with no apparent pathology (Berrada et al. 2006). They postulated that skunks contracted infections from hosting dog ticks (*Dermacentor variabilis*), carrying a mean 43.4 ticks each (n = 31). Again, this shows how skunks (and raccoons) pose a zoonotic threat when utilizing public spaces.

Among other bacterial infections, Hwang et al. (2002) report that among three radio-tracked striped skunks found dead in Saskatchewan, *Streptococcus equisimilis* was responsible in one instance, causing necrotizing purulent pneumonia. For the second, suppurative meningoencephalitis was the cause of death, while the third succumbed to *Streptococcus equisimilis* and *Streptococcus canis*, causing purulent myocarditis and pyothorax. Although opportunistic, this illustrates the range of pathogens causing mortality in wild animals. Striped skunks can also contract ***Sarcocystis neurona*** (Dubey et al. 2002; Burcham et al. 2010). Additionally, there are ad hoc mentions of striped skunks (e.g. Goble 1942; Webster 1964) suffering from lungworm (*Crenosoma* spp.).

Skunks occasionally exhibit infection with protozoan **trypanosomiasis**. Brown et al. (2010) detected antibodies to *T. cruzi* in four of 41 striped skunks from Arizona and Georgia. In a rare study from Argentina, two of 49 Molina's hog-nosed skunk (*Conepatus chinga*) were infected with *T. cruzi*, with electrophoretic isoenzyme patterns identical to those found in local people (Pietrokovsky et al. 1991). This is of concern, because with ongoing deforestation these skunks increasingly tend to move into towns and villages, and their role in the epidemiology of Chagas disease could increase.

The literature on Old World skunks, termed stink badgers *Mydaus* spp. (although a taxonomic anachronism—see Zhou et al., Chapter 13, this volume) is too limited to be conclusive, although they will likely be susceptible to a similar range of diseases, notably rabies (Joseph et al. 1978), endemic to the Indonesian archipelago.

Disease management

Given the range of diseases for which musteloids can be important hosts, disease control programmes are important from an epidemiological and conservation perspective, and to curb zoonotic transmission. While the issues faced by black-footed ferrets, due to plague and CDV, illustrate the most severe depression of a musteloid species by disease, there have, to date, been no recorded musteloid extinctions driven by disease. The threat posed by CDV, originating among escaped mink, to South American marine and river otters (above) is, however, of serious current concern, as is disease among southern sea otters, stalled at approximately 3000 animals, hampered by high prevalence and intensity of *T. gondii* infection (Lafferty and Gerber 2002; Miller et al. 2002; Lafferty 2015). At the other end of the spectrum, it is the abundance of certain musteloid species, hosting disease, which is the problem. For example, managing raccoons and skunks infected with rabies costs the US government USD245–510 million per year to manage (CDC, www.cdc.gov); similarly European badgers contribute to the epidemiology of bTB in cattle, a problem costing around GBP99 million per annum to manage in Great Britain (2013–14; Defra 2014b).

Outside of these notable examples of economic or zoonotic significance, work on musteloid pathogens is typically limited to the fields of veterinary parasitology and clinical immunology, with a lack of coherent studies considering ecological implications (as the somewhat eclectic data supporting this chapter neatly illustrate). Contributing to this is the fact that decisions about disease management are often made by politicians, rather than scientists (see Tompkins and Wilson 1998)—never more clearly illustrated than with the UK badger–bTB debate (Wilkinson 2007). This can often leave ecologists inadequately informed, or disenfranchised, in the management of virulent pathogens (see Woodroffe 1999; Woodroffe and Donnelly, Chapter 20, this volume). There is, however, an epidemiological basis and generalized framework for disease control broadly applicable across the breadth of musteloid diseases.

While heterogeneities in the age, sex, and genotypic susceptibility of individuals within a population must be taken into account (e.g. Woolhouse et al. 1997),

in essence, the success of an intervention relates to whether the disease reproductive number, R_0 (the basic reproductive ratio of a disease, i.e. the number of secondary infections arising from an infectious case; Diekmann et al. 1990; Cox et al. 2005) can be reduced below one, which will lead to the decline and eventual eradication of infection within the population (Haydon et al. 2006). In terms of practical management, several strategies can be used to reduce R_0, depicted in Figure 9.3.

i) Host density regulation

Reducing host population density through active **culling**, hunting, or **poisoning** has been implemented widely in attempts to manage disease transmission from musteloid species. **Proactive culling** (Figure 9.3) generally refers to large, non-selective culling of whole populations to reduce (but not necessarily eliminate) host species density across the management region, irrespective of the animal's disease status (see Woodroffe and Donnelly, Chapter 20, this volume). **Selective culling** is based on testing individuals and culling only test-positive animals ('test and cull'), or by selecting out sub-populations within a target population to cull (for example, in reaction to spill-over infection in another host of interest). Examples include the culling, snaring, and poisoning of ferret badgers (and other meso-carnivores) in China in response to rabies outbreaks (Gong et al. 2012; see Zhou et al., Chapter 13, this volume); and the culling of invasive American mink in Spain to alleviate the spread of AMDV into native European polecats, European mink, and otters (Melero et al. 2010). Culling, supported by **vaccination**, is the major approach taken to attempt to reduce the transmission of *M. bovis* between European badgers and cattle in Ireland (Griffin et al. 2005; Byrne et al. 2014b) and the UK (Woodroffe and Donnelly, Chapter 20, this volume). Culling combined with vaccination is used in North America to control rabies spread by raccoons and skunks (Rosatte et al. 2001; Rosatte et al. 2006). In New Zealand introduced ferrets, stoats, and weasels are culled and poisoned to reduce predation of rare endemic ground nesting birds (Barlow and Norbury 2001; Clapperton 2001; King et al., Chapter 10, this volume), although this is expensive and labour intensive. Related to these mustelid issues in New Zealand, an interesting and potentially important application concerns the idea of using pathogens to reduce, or eradicate, introduced mustelids. McDonald and Larivière (2001) discuss how ADV, MEV, and CDV have potential to reduce populations of stoats, weasels, and ferrets

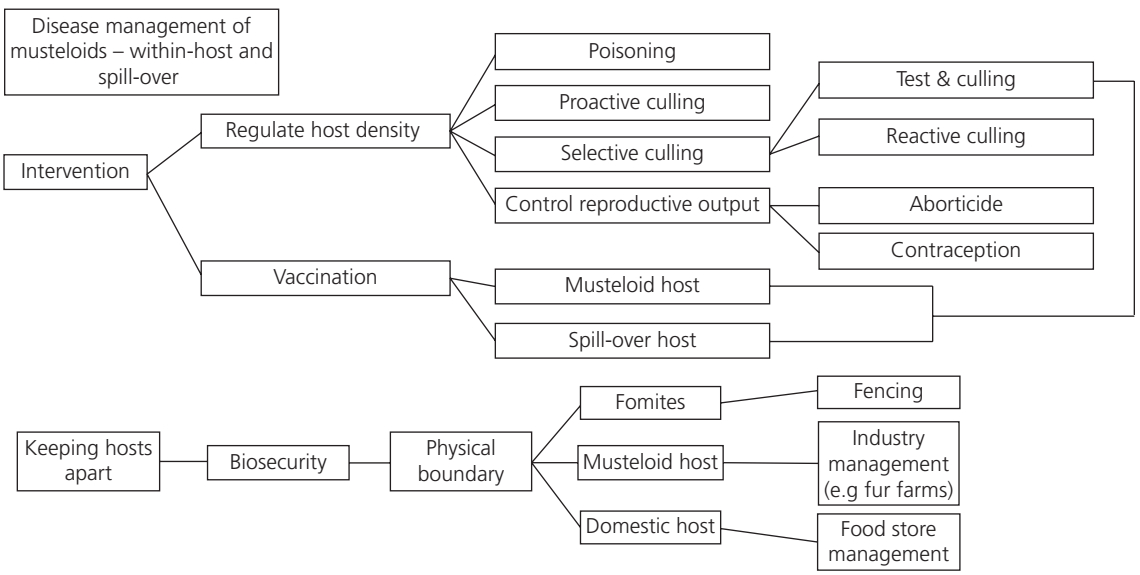

Figure 9.3 Invasive (intervention) and non-invasive (keeping hosts apart) approaches for the management of disease in musteloid hosts. Invasive interventions regulate the density of susceptible hosts (through culling, suppressing reproductive output, or vaccination), reducing spill-over to other (domestic animal/human/other species of interest) hosts. Biosecurity aims to reduce risk of spill-over, especially from wildlife to domestic hosts, through physical barriers between hosts or fomites. Inspired by Gortazar et al. (2014).

substantially, although risks to non-target species must be considered very thoroughly. Host-specific ectoparasites, such as the mite *Trichodectes ermineae*, nematodes such as *Skrjabingylus nasicola*, and bacteria such as *Helicobacter mustelae* and *Bartemella* spp. could have potential to be used for the delivery of targeted fertility control agents (chemosterilants; **controlling reproductive output**), but again, these authors sound caution. Norbury (2000) echoes this view, but stresses that the use of disease and chemosterilant agents seems more sustainable than trapping and poisoning. Furthermore, while **aborticide/sterilization** or **contraception** (Tuyttens and Macdonald 1998; Abdou et al. 2016) may be more politically and socially acceptable than culling, they tend to have a slower impact on density (Abdou et al. 2016).

ii) Vaccination

When introduced invasive species are involved (e.g. *Mustela* spp. in New Zealand), or where abundant species are considered 'pests' (e.g. raccoons in North America), population reduction may be justified. In other situations, disease management through population reduction may not be acceptable, because of detrimental impacts to species of conservation concern (Littin and Mellor 2005); for example, black-footed ferrets (Pedersen et al. 2007), or southern river otters (Breed et al. 2009). Indeed, for black-footed ferrets, **vaccination** against CDV and sylvatic plague (*Yersinia pestis*) has proven an essential conservation tool (see Biggins and Ead, Chapter 15, this volume).

Even with widespread abundant species, there may be a strong public opposition to population reduction (e.g. badgers in Great Britain; Woodroffe and Donnelly, Chapter 20, this volume) due to welfare concerns and a culture of fondness towards certain species (species 'mythologies'; Grant 2009). In such situations, non-lethal, and often non-invasive, techniques are required (Tuyttens and Macdonald 1998). Furthermore, in some situations, population control is simply ineffective and impractical due to the biology of the species under control—for example, lethal control of raccoons and stoats can result in relatively minor impacts on population abundance, with populations rebounding through increases in fecundity in surviving populations, supplemented by inward migrations (Rosatte 2000; McDonald and Harris 2002; Rosatte et al. 2007).

For a vaccination programme to achieve R_0 below one requires **'herd immunity'** whereby non-immunized individuals benefit from the vaccination of conspecifics by dilution, reducing their likelihood of interacting with another infectious individual (Anderson and May 1985; Byrne et al. 2012b; Carter et al. 2012). In European badgers, simulation modelling inferred that more than 40–50% immunization coverage needs to be achieved per annum to eradicate bTB (Wilkinson et al. 2004). This can be challenging at large spatial scales due to difficulties trapping the population (Byrne et al. 2012b; Tuyttens et al. 1999). In 2003, 14 eastern American states implemented large-scale oral rabies vaccine (ORV) programmes, primarily aimed at raccoons, by distributing 7,776,181 baits from the air, over 109,052 km^2, resulting in 71.3 baits per km^2 (Slate et al. 2005); a strategy that has been broadly successful in halting the spread of rabies westwards (Slate et al. 2009). Due to high contact rates between raccoons modelling suggests that 85% coverage is necessary to redress rabies prevalence (Reynolds et al. 2015), where a minimum of 71% has been achieved thus far (Rosatte et al. 2009). Currently rabies management among skunks is limited by a lack of effective oral rabies vaccine bait, resulting in interventions limited to trap-vaccinate-release (Slate et al. 2005; Slate et al. 2009; Rosatte et al. 2009; Fehlner-Gardiner et al. 2012). Vaccination can also be used in combination with test and cull operations, known as test-vaccinate/cull-release (TVR) operations (Abdou et al. 2016).

iii) Keeping hosts apart

Disease can also be managed by isolating hosts. When a pathogen is associated with livestock this contributes to ***biosecurity***; minimizing the risk of infection spill-over, or spill-back, between wildlife and the domestic host. Excluding badgers from farmyards has been attempted to reduce the potential spill-back transmission of bTB to cattle at this key point of contact (Wilson et al. 2011), although, in reality, such schemes have often proven surprisingly difficult to implement effectively. Fencing off **fomites** (e.g. badger setts or latrines) may be another practical intervention to reduce transmission at pasture (Ward et al. 2010). Similarly, hygiene practices, to prevent the inadvertent transmission of pathogens on work boots or vehicles, are good practice. Complete physical isolation between wild populations from other hosts (spill-over) is, however, practically impossible for small carnivores and certainly unlikely to be cost effective (Woodroffe et al. 2006; Gortazar et al. 2015). Nevertheless, known sources of infectious contact, such as the spread of

AMDV in mink manure in Nova Scotia (Farid 2013), can be alleviated through more informed and responsible farming practices—with calls to halt this practice and find an alternative way to dispose of the 37,000 tonnes of manure two million mink produce per year (Perennia 2013).

Conclusions

Wobeser (2007) compares disease in wildlife populations to an iceberg, where we tend only to see the tip of the full implications involved. Eighty percent of animal pathogens present in the USA have a wildlife component (Miller et al. 2013). This a particular issue for the musteloids because, unlike many larger carnivores, in many instances they persist in their established ecosystem roles, while also insinuating themselves into human activities—parks, agriculture, even our back yards (Daszak et al. 2000).

We have elaborated here essentially the 'most studied' examples of musteloid diseases, involving cases of zoonotic bio-safety, agricultural spill-over, and conservation importance in the Musteloidea. Nevertheless this review is the first to attempt to consolidate such disease data for the superfamily. Why should this be so? Specifically, this superfamily is diverse and challenging to synthesize, with over 90 members, but more generally, disease biology is a subject divided. Epidemiologists look at the transmission of communicable pathogens; veterinarians, medics, public health, and environment services establish the symptoms and treatment of diseases; immunologists look at physical responses and develop screening protocols; but it is only when these disciplines are unified that the importance of diseases for conservation ecology can be fully realized. Related to this, systematic surveillance and disease monitoring are essential (Gortazar et al. 2015), reliant upon: (i) the ecology and behaviour of wildlife hosts and vectors, (ii) how isolated or contagious host populations are, (iii) the single or multi-host characteristics of the pathogen—spill-over and spill-back, (iv) the availability of suitable diagnostic tools, and (v) the attitude of the stakeholders involved—which also varies regionally (after Gortazar et al. 2015). All factors set against ongoing pressures and interventions facing musteloids (and other wildlife), such as harvesting/culling, habitat loss, urban encroachment, introductions (of musteloids, competitors, and pathogens), farming practices, species control measures, and conservation strategies. Crucially, only with more systematic monitoring can the absence of infection be demonstrated reliably (OIE WofAH 2011), enabling pathogen emergence in formerly healthy populations to be identified with confidence (Gortazar et al. 2014).

To these ends, Gortazar et al. (2015) advocate the 'One Health' approach; an international collaborative initiative aimed at reducing risks of infectious diseases at the Animal-Human-Ecosystem interface (FAO/OIE/WHO/UNSIC/UNICEF/WorldBank 2008). It is thus beholden upon ecologists, in the interests of informed species management, as well as the evolutionary processes involved in host–pathogen dynamics, to seek to adopt such principles and utilize all sources of information; not only with respect to the musteloids, but to wildlife generally.

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