REVIEW ARTICLE

Zoonotic Diseases Associated with Free-Roaming Cats

R. W. Gerhold[1] and D. A. Jessup[2]

[1] Center for Wildlife Health, Department of Forestry, Wildlife, and Fisheries, The University of Tennessee, Knoxville, TN, USA
[2] California Department of Fish and Game (retired), Santa Cruz, CA, USA

Impacts

- Free-roaming cats are an important source of zoonotic diseases including rabies, *Toxoplasma gondii*, cutaneous larval migrans, tularemia and plague.
- Free-roaming cats account for the most cases of human rabies exposure among domestic animals and account for approximately 1/3 of rabies post-exposure prophylaxis treatments in humans in the United States.
- Trap–neuter–release (TNR) programmes may lead to increased naïve populations of cats that can serve as a source of zoonotic diseases.

Keywords:
Cutaneous larval migrans; free-roaming cats; rabies; toxoplasmosis; zoonoses

Correspondence:
R. Gerhold. Center for Wildlife Health, Department of Forestry, Wildlife, and Fisheries, The University of Tennessee, Knoxville, TN 37996-4563, USA. Tel.: 865 974 0465; Fax: 865-974-0465; E-mail: rgerhold@utk.edu

Received for publication March 16, 2012

doi: 10.1111/j.1863-2378.2012.01522.x

Summary

Free-roaming cat populations have been identified as a significant public health threat and are a source for several zoonotic diseases including rabies, toxoplasmosis, cutaneous larval migrans because of various nematode parasites, plague, tularemia and murine typhus. Several of these diseases are reported to cause mortality in humans and can cause other important health issues including abortion, blindness, pruritic skin rashes and other various symptoms. A recent case of rabies in a young girl from California that likely was transmitted by a free-roaming cat underscores that free-roaming cats can be a source of zoonotic diseases. Increased attention has been placed on trap–neuter–release (TNR) programmes as a viable tool to manage cat populations. However, some studies have shown that TNR leads to increased immigration of unneutered cats into neutered populations as well as increased kitten survival in neutered groups. These compensatory mechanisms in neutered groups leading to increased kitten survival and immigration would confound rabies vaccination campaigns and produce naïve populations of cats that can serve as source of zoonotic disease agents owing to lack of immunity. This manuscript is a review of the various diseases of free-roaming cats and the public health implications associated with the cat populations.

Domestic cats are a potential source of numerous infectious disease agents; however, many of these diseases are controlled in cats belonging to responsible owners through routine veterinary care, proper vaccination regimens and parasite chemotherapy. Free-roaming cats often lack the necessary preventative care to control these diseases and consequently pose a potential health threat to other domestic animals, wildlife and humans. Historically, animal control programmes have been paramount in minimizing zoonotic risk in the United States. In the 1950s, a rabies control programme began, which included mandatory rabies vaccination in dogs and animal control programmes aimed at removing free-roaming animals (Rupprecht et al., 2001). These programmes have significantly reduced the incidence of human rabies in the United States. However, in the last decade, there has been a marked reduction in social support for collection and euthanasia of free-roaming animals, particularly cats. In some areas, animal control has been turned over to private organizations that follow a 'no-kill' philosophy and routinely release free-roaming animals. Diminished resources and willingness to collect free-roaming animals have led to increasing numbers of free-roaming animals; and rabies exposure in humans remains an important public health threat.

Rabies

Since 1988, rabies has been detected more frequently in cats than dogs in the United States (Rupprecht, 2002), and in 2008 the number of rabies cases in cats ($n = 294$) was approximately four times the number of cases in dogs (Blanton et al., 2009). In 2010, rabies cases declined in all domestic animals, except for cats, which comprised 62% ($n = 303$) of all rabies cases in domestic animals (Blanton et al., 2011). In contrast, dogs accounted for 69 rabies cases, which is a 14% decrease from 2009. Although rabies is detected most frequently in various wild animals in the United States and the majority of human rabies cases in the United States are attributable to bites of rabid bats, multiple studies have disclosed that human exposure to rabies is largely associated with free-roaming cats because of people being more likely to come in contact with cats, large free-roaming cat populations and lack of stringent rabies vaccination programmes (Childs, 1990; Cole and Atkins, 2007; Roseveare et al., 2009; Eidson and Bigman, 2010). A recent case of rabies in an 8-year old girl from California in 2010 disclosed that the patient had multiple cat bites from free-roaming cat colonies near her house (Blanton et al., 2011). Although rabies RNA was unable to be collected for molecular typing, the epidemiological data highly suggest that the girl was exposed by a rabid free-roaming cat (CDC MMWR, 2012).

From 2002 to 2006 in Georgia, 70 cats tested positive for rabies and the virus was detected more frequently in cats than in any other domestic animal (Cole and Atkins, 2007). Moreover, 17% of all confirmed human rabies exposures in Georgia were attributable to cat bites from 2004 to 2006, whereas domestic dogs comprised 5% of all confirmed human rabies in Georgia during the same time period. A separate investigation of rabies exposure in domestic animals in upstate South Carolina disclosed that free-roaming cats were disproportionately associated with potential human rabies exposure and were most frequently reported rabid among domestic exposure animals (Roseveare et al., 2009). Similarly, in New York from 1993 to 2010, cats accounted for the majority of human rabies exposure incidents (32%) and post-exposure prophylaxis (PEP) treatments (31%) (Eidson and Bigman, 2010). In Pennsylvania, rabid cat cases exceeded all cases of rabid wild animals, with the exception of raccoons, and in 2009 and 2010, rabid cat cases ($n = 56$) were tied with skunks for the second most frequently diagnosed animal (Herman, 2010). In contrast to the 56 free-roaming cat cases in 2010 in Pennsylvania, dogs, cattle and horses constituted 4, 7 and 5 cases, respectively. In 2011, numerous press releases from various county health departments have documented the presence of rabid cats including a rabid cat in Worchester County, MD: two human exposure cases in Cecil County, MD, owing to bites by a rabid cat; four human exposures in Wantage Township, NJ, owing to two rabid free-roaming cats; and two cases of human exposure owing to free-roaming cat bites in Hall County, GA. Similarly in 2012, a rabid free-roaming cat in Cherokee County, GA, led to rabies PEP treatment for at least seven people. Unfortunately, reporting to county health departments is not performed in uniform manner; thus, the actual cases of rabies exposure in humans owing to cats are likely underestimated.

Rabies virus is transmitted via saliva from one host to another primarily via a bite from a rabid animal. Following a bite of a rabid animal and virus inoculation, the virus replicates in neurons and disseminates via the nervous system. Later in the infection, the virus can be found in highly innervated organs including cornea, skin and salivary glands (Iwasaki, 1991). Rabies leads to various neurological impairment symptoms, and the disease is invariably fatal. Individuals exposed to potentially rabid animals are administered PEP, and cat exposures account for approximately 1/3 of all PEP recipients. Post-exposure prophylaxis regimen generally costs \$5000–8000 for each individual, which is mostly borne by public health agencies (Recuanco et al. 2007). Although rabies vaccination may be provided to free-roaming cats by some trap–neuter–release (TNR) programmes, it does not decrease the need for PEP because (i) cats can shed virus for a few days prior to clinical onset, (ii) the uncertainty about free-roaming cat vaccination status, (iii) the inability to determine time and route of virus exposure in the cats, and (iv) the inability to confine free-roaming cats for observation similar to dogs (Jessup and Stone, 2010; Brown et al., 2011). Additionally, Murray et al. (2009) reported rabies cases in 22 (2%) of vaccinated cats, including two cats classified as currently vaccinated, indicating that vaccine failures can occur. Moreover, TNR advocates are unlikely to administer rabies immunization of all free-roaming cats. This is significant because one rabid cat in an aggressive (i.e. furious rabies) condition can lead to multiple exposure events because furious rabid animals often seek potential hosts to bite. Rabid cats were found to exhibit aggressive behaviour (55% of cases) more frequently than dumb behaviour, which is in contrast to rabid dogs which only displayed aggressive behaviour in 33% of cases (Eng and Fishbein, 1990). Moreover, rabid cats were significantly more likely than rabid dogs to bite a person (62% vs. 36%) (Eng and Fishbein, 1990).

In vaccination studies, it was demonstrated that feline leukaemia virus (FeLV)-infected cats may not be able to mount adequate immune response to some rabies vaccines (Franchini, 1990). The author indicated that FeLV-infected cats should be confined strictly indoors to prevent spread of FeLV to other cats in the neighbourhood and if left outside in areas at risk of rabies, FeLV-positive cats should receive more frequent rabies vaccination (every 6 months). In a prospective study of FeLV and feline immunodeficiency virus (FIV) in Canada, the authors noted that 6% ($n = 14$) of free-roaming cats were FeLV seropositive, whereas only 2% ($n = 4$) of owned cats were FeLV seropositive (Little, 2011). The risk of being seropositive for either virus was most frequently associated with being free-roaming, followed by having access to outdoors. Owing to the threat of rabies exposure as documented above, the 2011 Compendium of Animal Rabies Prevention and Control states that stray animals including cats should be removed from the community through local health departments and animal control officials (Brown et al., 2011).

Free-roaming cat behaviour

An investigation of the demographic differences of urban groups of neutered and sexually intact free-roaming cats following a TNR procedure disclosed that the neutered groups increased significantly compared to intact groups because of higher immigration and lower emigration (Gunther et al., 2011). Additionally, the authors noted that sexually intact adult cats immigrated into the neutered groups at a significantly higher rate than the sexually intact groups. These immigrating cats were not tame and succeeded to integrate into the group, which highly suggests that these were free-roaming cats and not abandoned house cats. In addition, kitten survival in the neutered groups was significantly higher than in the unneutered groups. The authors suggested that immigrating sexually intact females had increased fertility along with increased survivorship of kittens as a population compensation response to neutered individuals. These data suggest that neutered cat groups act as attractant of sexually intact free-roaming cats, thus negating the belief that TNR programme leads to decrease in free-roaming cat populations. In a separate study, free-roaming cats changed movement patterns and habitat on a seasonal basis compared to owned cats (Horn et al., 2011). Interestingly, the free-roaming cats used more grasslands and urban areas than predicted because of available habitat. Although the owned cats were neutered, it was not considered a reason for the movement pattern differences because in a separate investigation, Guttilla and Stapp (2010) did not find a significant difference between the movement of neutered cats and intact cats. These data suggest that immigrating and habitat switching of unvaccinated cats may severely limit the protection offered by vaccination of TNR processed cats and would not abate the zoonotic threat of rabies in these groups.

Secondary mesocarnivore impacts

Free-roaming cat colony feeding stations attract wild mesocarnivores (Gehrt, 2003), potentially exacerbating human rabies exposure incidents. Raccoons, bats, skunks and various fox species are the wildlife species most frequently infected with rabies, depending on the region of the United States. By attracting mesocarnivores, feeding stations likely increase the potential interaction between humans and mesocarnivores, leading to a greater public health risk of exposure to rabies. Furthermore, raccoons harbour an intestinal nematode parasite, *Baylisascaris procyonis* (i.e. raccoon roundworm), that has caused morbidity and mortality in humans, especially children (Kazacos, 2001). Infections occur after accidental ingestion of the microscopic *B. procyonis* eggs containing embryonated larvae followed by larvae migration (i.e. larval migrans) through visceral organs, eyes and brain. The geographical distribution of *B. procyonis* is expanding from its historical range from Midwestern, Western and Northeastern United States (Kazacos, 2001). *Baylisascaris*-positive raccoons have been found in multiple states in the Southeastern United States, Canada, Europe and Japan (Kazacos, 2001; Souza et al., 2009; Blizzard et al., 2010; Yabsley et al., 2010). The finding of *B. procyonis* in raccoons only near urban areas in Georgia (Blizzard et al., 2010) is of particular interest given that managed free-roaming cat colonies are likely to be found in urban and suburban settings.

Domestic cats can be a source of infection for native wildlife. Contact or consuming domestic cats can be a threat to native predators. Consumption of free-roaming cats by cougar or panther (*Felis concolor*) poses a risk of FeLV transmission, and suspected cases of domestic cat-transmitted FeLV in wild felids have been reported in California and Florida (Jessup et al., 1993; Cunningham et al., 2008). Genetic analysis of the FeLV virus associated with mortality in 5 Florida panthers indicated that the virus envelope sequence was nearly identical indicating the source or the infection was likely from a single domestic cat (Brown et al., 2008).

Endoparasities

Domestic and wild felids are the definitive host for several zoonotic parasites, including the protozoan *Toxoplasma gondii* and the ascarid *Toxocara cati*. Similar to *B. procyonis* of raccoons, the host defecated eggs (*Toxocara*) or oocysts (*Toxoplasma*) of these parasites are extremely environmentally resistant (Long, 1990; Kazacos, 2001), and human infections can occur months or possibly even years after the cat has excreted the parasite egg. For this reason, cat faeces-contaminated playgrounds, garden soil, sandboxes and other outdoor recreational areas may serve as a source of infection for humans (Holland and Smith, 2006; Lee et al., 2010). The prevalence of *T. cati* was higher in urban areas than rural areas, and soil samples from urban parks contained a higher proportion of *T. cati* compared to the canine *Toxocara*, *Toxocara canis*. These data suggest that the higher levels of *T. cati* are associated with free-roaming cats in urban areas. *Toxocara cati* infections have been associated with visceral and ocular larval migrans and can result in permanent ocular damage in infected humans (Lee et al., 2010).

In toxoplasmosis, humans are infected primarily by ingestion of sporulated oocyst in cat faeces-contaminated soil or water or tissue cysts in undercooked or raw meat (Elmore et al., 2010). Nutter et al. (2004) reported a higher seroprevalence of *T. gondii* in free-roaming cats than pet cats, with the lowest prevalence in cats kept indoors. Similar results were found among free-roaming cats in Sri Lanka and Seoul, Korea (Kulasena et al., 2011; Lee et al., 2011). Contact with infective *T. gondii* oocysts in cat faeces has been shown to be a primary risk factor for human toxoplasmosis (Elmore et al., 2010).

For many years, the risk of infection from oocysts has been dismissed as considerably less common than infection from ingestion of undercooked or raw meat. Recently, a *T. gondii* embryogenesis-related protein antibody (TgERP), which is sporozoite specific, has been developed, which allows for serological distinction between oocyst and tissue cyst infection given that sporozoites are only present in oocysts (Hill et al., 2011). The TgERP can be detected within 6–8 months post-infection allowing for detection of oocyst infection in acute stage infections. Of 163 individuals in acute stage infection, 103 (63%) were positive for TgERP indicating that the majority of human infection was attributable to oocyst infection (Hill et al., 2011). *Toxoplasma* infections can manifest as ocular diseases, neurological impairment and lead to blindness, abortions and birth defects, particularly hydrocephalus, in humans (Dubey and Odening, 2001). Toxoplasmosis is also a significant risk for individuals receiving immuosuppressive therapy, transplant recipients and is a major cause of systemic infection and death for immunosuppressed (e.g. HIV/AIDS) patients (Elmore et al., 2010). An increased risk of schizophrenia, autism, Alzheimer's and other neuro-inflammatory diseases has been proposed with *T. gondii* infection (Fekadu et al., 2010; Prandota, 2010), but further research is needed to fully understand the neurological effects of *T. gondii*. Toxoplasmosis is also a major disease issue for wildlife and has been documented in multiple wild avian and mammalian species, especially marine mammals and Australian marsupials (Dubey and Odening, 2001; Dubey, 2002; De Thoisy et al., 2003; Lindsay and Dubey, 2007). In addition, toxoplasmosis is an important cause of abortion in domestic animals including sheep and goats.

In addition to the above parasite species, human infections with domestic cat hookworms, including *Uncinaria stenocephala*, *Ancyclostoma tubaeforme, A. brazilense* and *A. ceylanicum*, have been reported (Bowman et al., 2010). After defecation, hookworm eggs hatch and the infectious filariform larvae can penetrate the skin of animals or human hosts. Infective larvae can cause skin lesions known as cutaneous larva migrans (CLM) and less frequently pneumonitis, muscle infection and ocular manifestations. Occasionally, *A. ceylanicum* can develop into an adult hookworm in humans and cause abdominal discomfort (Prociv, 1998). Several reports of human infections of feline hookworm infections have been reported from soil under houses or on beaches that cats defecate upon. Approximately 75% of free-roaming cats in Florida were positive for *A. tubaeforme*, and 33% were positive for *A. braziliense* (Anderson et al., 2003). In 2006, 22 people were diagnosed with CLM at a Miami-Dade County children's camp. Although free-roaming cats were found in the vicinity of the camp, the source of the infection was not determined (CDC MMWR, 2007). In 2010, contaminated cat faeces was responsible for at least seven confirmed and eight unconfirmed human hookworm infections in Miami-Dade County from contaminated beaches (Personal communication Miami Dade health Department). In both of these incidents, the County public health department bore the expense and responsibility of trapping the free-roaming cats and removing faeces from the contaminated areas to minimize further human infections.

Ectoparasites and vector-borne diseases

Ectoparasites of domestic cats, especially the cat flea (*Ctenocephalides felis*), are important in transmission of zoonotic diseases. Three major flea-associated diseases of cats in the United States include cat-scratch disease (CSD), flea-borne typhus and plague (McElroy et al., 2010). Cat-scratch disease or bartonellosis is caused by the gram-negative bacterium *Bartonella henselae*. Cats are the primary source of the bacteria; however, they are inapparent carriers and thus appear healthy. Animal to animal and animal to human infection occurs by exposure of an open wound, from a scratch or bite, or *B. henselae*-contaminated flea faeces. Fleas acquire *B. henselae* from a previous bloodmeal from an infected cat. Symptoms in human with CSD include fever, headaches and regional lymph node enlargement, and the disease is one of the most frequent diagnoses of benign lymphadenopathy in children and young adults (McElroy et al., 2010). Atypical complications including encephalitis, retinitis and endocarditis occur in 5–15% of CSD-infected humans (Chomel et al., 2004), and recently *Bartonella* spp. infection has been associated with chronic rheumatic symptoms, clinically similar to chronic Lyme disease, in humans (Maggi et al., 2012). Seroprevalence of *B. henselae* in cats ranges from 14 to 93% (Nutter et al., 2004; Case et al., 2006; Lappin et al., 2006), and free-roaming cats had a significantly higher seroprevalence than pet cats (Nutter et al., 2004).

In addition to CSD, cat fleas are potentially able to vector rickettsial diseases including murine typhus (*Rickettesia typhi*) and a closely related zoonotic disease agent, *Rickettesia felis* which are potential human health threats wherever cat, rat or flea populations are dense (Case et al., 2006). Similar to CSD, cats are inapparent carriers of *R. typhi*, and outbreaks have been associated with free-roaming cat colonies in Hawaii (Jessup, 2004). Other reported cases of murine typhus in the United States are focused in central and south-central Texas and Los Angeles area (Adams et al., 1970; Sorvillo et al., 1993). In the Los Angeles *R. typhi* focus, 90% ($n = 9$) of collected cats were seropositive for *R. typhi* antibodies, whereas no seropositive cats ($n = 21$) were found in the control areas where no human infections were reported (Sorvillo et al., 1993). Flea suppression is the first public health action often initiated; however, failure to control free-roaming cat populations can lead to future disease outbreaks.

Additionally, human bacterial diseases including tularemia, caused by *Francisella tularensis*, and plague, caused by *Yersinia pestis*, have been associated with direct contact with cats or cat fleas (Liles and Burger, 1993; Gage et al., 2000; McElroy et al., 2010). Approximately, 8% of plague cases in the United States are associated with transmission from cats, and cases of cat exposure associated plague are reported year round where flea-associated cases are generally restricted to warmer months (Gage et al., 2000). Cats frequently develop the pneumonic form of plague, which is considerably more infectious to humans in close contact, and results in rapidly progressive and frequently fatal disease. Both tularemia and plague can cause various symptoms and potentially lead to fatal respiratory disease or multiorgan failure in both humans and other animals (Spagnoli et al., 2011). It is suggested that in addition to harbouring infected fleas, cats preying on infected rodents can contain the bacterial agents of tularemia and plague in their mouths and potentially transmit the bacteria to humans via bites or scratches.

Viruses

Cats have been implicated as potential vectors of other diseases not historically associated with felines, including SARS and H1N1 and H5N1 avian influenza as evidenced by natural and experimental infection of domestic cats (Kuiken et al., 2004; Songserm et al., 2006; Thiry et al., 2007; Anonymous, 2011). In the experimentally infected cats, excreted virus was transmitted to sentinel cats demonstrating horizontal transmission and suggesting cats can be involved in epidemiology and transmission of the virus (Kuiken et al., 2004). Cats have been infected with H5N1 through ingestion or close contact of infected birds as well as intratracheal and intra-oral infection of a human isolated virus strain (Thiry et al., 2007). Additionally, cats have been found to be subclinically infected with H5N1 (Leschnik et al., 2007), and more research is needed to determine the role cats may play in the epidemiology and spread of avian influenza.

Conclusion

The information in this review highlights the serious public health diseases associated with free-roaming cats and underscores the need for increased public health attention directed towards free-roaming cats. Diseases including rabies, toxoplasmosis, cutaneous larval migrans and various vector-borne diseases have been shown to be associated with free-roaming cats. Rabies exposure in human is disproportionally associated with free-roaming cats compared to other domestic animals. This fact should be of paramount concern to public health officials because of the high mortality rate of clinical rabies and the significant cost of PEP in exposed people. Furthermore, TNR programmes can increase immigration and kitten recruitment, which would lead to naïve populations of cats that would be a source for zoonotic diseases including rabies and toxoplasmosis. While citizens who are concerned about the perceived improved welfare of cats in TNR programmes may be very vocal in their support of free-roaming cat populations, local, county and state legislative and medical officials need to understand the economic and public health threats associated with various policies and laws associated with free-roaming cat populations. Further resources are needed to educate the public, the medical community and public health officials about the zoonotic disease potential associated with free-roaming cats.

References

Adams, W., R. Emerson, and J. Brooks, 1970: The changing ecology of murine (endemic) typhus in Southern California. *Am. J. Trop. Med. Hyg.* 19, 311–318.

Anderson, T. C., G. W. Foster, and D. J. Forrester, 2003: Hookworms of feral cats in Florida. *Vet. Parasitol.* 115, 19–24.

Anonymous, 2011: H1N1 influenza turns up again in cats. *J. Am. Vet. Med. Assoc.* 238, 964.

Blanton, J. D., K. Robertson, D. Palmer, and C. Rupprecht, 2009: Rabies surveillance in the United States during 2008. *J. Am. Vet. Med. Assoc.* 235, 676–689.

Blanton, J., D. Palmer, J. Dyer, and C. Rupprecht, 2011: Rabies surveillance in the United States during 2010. *J. Am. Vet. Med. Assoc.* 239, 773–783.

Blizzard, E., C. Davis, S. Henke, D. Long, C. Hall, and M. Yabsley, 2010: Distribution, prevalence, and genetic characterization of *Baylisascaris procyonis* in selected areas of Georgia. *J. Parasitol.* 96, 1128–1133.

Bowman, D. D., S. P. Montgomery, A. M. Zajac, M. L. Eberhard, and K. R. Kazacos, 2010: Hookworms of dogs and cats as agents of cutaneous larva migrans. *Trends Parasitol.* 26, 162–167.

Brown, M., M. Cunningham, A. Roca, J. Troyer, W. Johnson, and S. O'Brien, 2008: Genetic characterization of Feline Leukemia virus from Florida Panthers. *Emerg. Infect. Dis.* 14, 252–259.

Brown, C., L. Conti, P. Ettestad, M. Leslie, F. Sorhage, and B. Sun, 2011: Compendium of animal rabies and control, 2011. *J. Am. Vet. Med. Assoc.* 239, 609–617.

Case, J. B., B. Chomel, W. Nicholson, and J. E. Foley, 2006: Serological survey of vector-borne zoonotic pathogens in pet cats and cats from animal shelters and feral colonies. *J. Feline Med. Surg.* 8, 111–117.

CDC Morbidity, Mortality Weekly Report, 2007: Outbreak of cutaneous larva migrans at a children's camp – Miami, Florida, 2006. 54, 1285–1287.

CDC Morbidity, Mortality Weekly Report, 2012: Recovery of a patient from clinical rabies- California, 2011. 61, 61–65.

Childs, J. E., 1990: Urban cats: their demography, population-density, and owner characteristics in Baltimore, Maryland. *Anthrozoos* 3, 234–244.

Chomel, B., H. J. Boulouis, and E. B. Breitschwerdt, 2004: Cat scratch disease and other zoonotic *Bartonella* infections. *J. Am. Vet. Med. Assoc.* 224, 1270–1279.

Cole, D., and L. Atkins, 2007: Rabies: the epidemic continues in Georgia. *Ga. Epidemiol. Rep.* 23, 1–4.

Cunningham, M., M. Brown, D. Shindle, S. Terrell, K. Hayes, B. Ferree, R. T. McBride, E. Blankenship, D. Jansen, S. Citino, M. Roelke, R. Kiltie, J. Troyer, and S. O'Brien, 2008: Epizootiology and management of feline leukemia virus in the Florida puma. *J. Wildl. Dis.* 3, 537–552.

De Thoisy, B., M. Demar, C. Aznar, and B. Carme, 2003: Ecologic correlates of *Toxoplasma gondii* exposure in free-ranging neotropical mammals. *J. Wildl. Dis.* 39, 456–459.

Dubey, J. P., 2002: A review of toxoplasmosis in wild birds. *Vet. Parasitol.* 106, 121–153.

Dubey, J. P., and K. Odening, 2001. Toxoplasmosis and related infections. In: Samuels, W. M., M. J. Pybus, and A. A. Kocan (eds), Parasitic Diseases of Wild Mammals, pp. 478–519. Iowa State University Press, Ames, Iowa.

Eidson, M., and A. Bigman, 2010: Terrestrial rabies and human postexposure prophylaxis, New York, USA. *Emerg. Infect. Dis.* 16, 527–529.

Elmore, S. A., J. L. Jones, P. A. Conrad, S. Patton, D. S. Lindsay, and J. P. Dubey, 2010: *Toxoplasma gondii*: epidemiology, feline clinical aspects, and prevention. *Trends Parasitol.* 26, 190–196.

Eng, T. R., and D. B. Fishbein, 1990: Epidemiologic factors, clinical findings, and vaccination status of rabies in cats and dogs in the United States in 1988. *J. Am. Vet. Med. Assoc.* 197, 201–209.

Fekadu, A., T. Shibre, and A. J. Cleare, 2010: Toxoplasmosis as a cause for behavior disorders- overview of evidence and mechanisms. *Folia Parasitol.* 57, 105–113.

Franchini, M. 1990. Die Tollwutimpfung von mit felinem Leukämivirus infizierten Katzen. Veterinary Dissertation. Zürich University.

Gage, K. L., D. T. Dennis, K. A. Orloski, P. Ettestad, T. L. Brown, P. J. Reynolds, W. J. Pape, C. L. Fritz, and L. G. Carter, 2000: Cases of cat-associated plague in the Western US, 1977–1998. *Clin. Infect. Dis.* 30, 893–900.

Gehrt, S. D. 2003. Raccoon (*Procyon lotor*) and allies. In: Feldhamer, G. A., B. C. Thompson, and J. A. Chapman (eds), Wild Mammals of North America, 2nd edn, pp. 611–634. Johns Hopkins University Press, Baltimore, Maryland, USA.

Gunther, I., H. Finkler, and J. Terkel, 2011: Demographic differences between urban feeding groups of neutered and sexually intact free-roaming casts following a trap-neuter-return procedures. *J. Am. Vet. Med. Assoc.* 238, 1134–1140.

Guttilla, D., and P. Stapp, 2010: Effects of sterilization on movements of feral cats at a wildland-urban interface. *J. Mammal.* 91, 482–489.

Herman, K. 2010. An Update on the Incidence of Rabies in Pennsylvania in 2010. Pennsylvania Department of Agriculture, Pennsylvania Veterinary Laboratory, Pennsylvania Bureau of Animal Health & Diagnostic Services, Pennsylvania, 3pp.

Hill, D., C. Coss, J. P. Dubey, K. Wroblewski, M. Sautter, T. Hosten, C. Muñoz-Zanzi, E. Mui, S. Withers, K. Boyer, G. . Hermes, J. Coyne, F. Jagdis, A. Burnett, P. McLeod, and H. Morton, 2011: Identification of a sporozoite-specific antigen from *Toxoplasma gondii*. *J. Parasitol.* 97, 328–337.

Holland, C. V., and Smith H. V., eds (2006) Toxocara: The Enigmatic Parasite, pp. 301. CABI Publishing, Cambridge, MA.

Horn, J., N. Mateus-Pinella, R. Warner, and E. Heske, 2011: Home range, habitat use and activity patterns of free-roaming domestic cats. *J. Wildl. Manage.* 75, 1177–1185.

Iwasaki, Y., 1991: Spread of rabies virus within the central nervous system. In: Baer, G. M. (ed.), The natural history of rabies, 2nd edn, pp. 122–132. CRC Press, Boca Raton, FL.

Jessup, D. E., 2004: The welfare of feral cats and wildlife. *J. Am. Vet. Med. Assoc.* 225, 1377–1383.

Jessup, D., and E. Stone, 2010: More on feral cats. *J. Am. Vet. Med. Assoc.* 235, 7.

Jessup, D., K. Pettan, and L. Lowenstine, 1993: Feline leukemia virus infection and secondary spirochetemia in a free-ranging cougar (*Felis Concolor*). *J. Am. Vet. Med. Assoc.* 214, 73–79.

Kazacos, K. R. 2001. *Baylisascaris procyonis* and related species. In: Samuel, W. M., M. J. Pybus, and A. A. Kocan (eds), Parasitic Diseases of Wild Mammals, 2nd edn, pp. 301–341. Iowa State University Press, Ames, IA.

Kuiken, T., G. Rimmelzwaan, D. V. Riel, G. V. Amerongen, M. Baars, R. Fouchier, and A. Osterhaus, 2004: Avian H5N1 in cats. *Science* 306, 241.

Kulasena, V. A., R. Rajapakse, J. P. Dubey, P. N. Dayawansa, and S. Premawansa, 2011: Seroprevalence of *Toxoplasma gondii* from cats in Colombo Sri Lanka. *J. Parasitol.* 97, 152.

Lappin, M. R., B. Griffin, J. Brunt, A. Riley, D. Burney, J. Hawley, M. Brewer, and W. Jensen, 2006: Prevalence of *Bartonella* species, haemoplasms species, *Ehrlichia* species, *Anaplasma phagocytophilum*, and *Neorickettsia risticii* DNA in the blood of cats and their fleas in the United States. *J. Feline Med. Surg.* 8, 85–90.

Lee, A., P. M. Schantz, K. R. Kazacos, S. P. Montgomery, and D. D. Bowman, 2010: Epidemiologic and zoonotic aspects of ascarid infections in dogs and cats. *Trends Parasitol.* 26, 155–161.

Lee, S. E., N. H. Kim, H. S. Chae, S. H. Cho, H. W. Nam, W. J. Lee, S. H. Kim, and J. H. Lee, 2011: Prevalence of Toxoplasma gondii from feral cats in Seoul, Korea. *J. Parasitol.* 97, 153–155.

Leschnik, M., J. Weikel, K. Mostl, S. Revilla-Fernandez, E. Wodak, Z. Bago, E. Vanek, V. Beneetka, M. Hess, and J. Thalhammer, 2007: Subclinical infection with avian influenza A (H5N1) virus in cats. *Emerg. Infect. Dis.* 13, 243–247.

Liles, W. C., and R. J. Burger, 1993: Tularemia from domestic cats. *West. J. Med.* 158, 619–622.

Lindsay, D. S., and J. P. Dubey, 2007. Toxoplasmosis in wild and domestic animals. In: Weiss, L. M., and K. Kim (eds), Toxoplasma Gondii the Model Apicomplexan: Perspectives and Methods, pp. 133–152. Elsevier, London.

Little, S., 2011: A review of feline leukemia virus and feline immunodeficiency virus seroprevalence in cats in Canada. *Vet. Immunol. Immunopathol.* 143, 243–245.

Long, P. L. 1990. Coccidiosis of Man and Domestic Animals. CRC Press, Boca Raton, FL, 356 pp.

Maggi, R. G., B. R. Mozayeni, E. L. Pultorak, B. C. Hegarty, J. M. Bradley, M. Correa, and E. B. Brietscherdt, 2012: *Bartonella* spp. bacteremia and rheumatic symptoms in patients from Lyme disease-endemic region. *Emerg. Infect. Dis.* 18, 783–791.

McElroy, K. M., B. L. Blagburn, E. B. Breitschwerdt, P. S. Mead, and J. H. McQuiston, 2010: Flea-associated zoonotic diseases of cats in the USA: bartonellosis, flea-borne rickettsioses, and plague. *Trends Parasitol.* 26, 197–204.

Murray, K. O., K. C. Holmes, and C. A. Hanlon, 2009: Rabies in unvaccinated dogs and cats in the United States, 1997-2001. *J. Am. Vet. Med. Assoc.* 235, 691–695.

Nutter, F. B., J. P. Dubey, J. F. Levine, E. B. Breitschwerdt, R. B. Ford, and M. K. Stoskopf, 2004: Seroprevalences of antibodies against *Bartonella henselae* and *Toxoplasma gondii* and fecal shedding of *Cryptosporidium* spp, *Giardia* spp, and *Toxocara catiin* feral and pet domestic cats. *J. Am. Vet. Med. Assoc.* 225, 1394–1398.

Prandota, J., 2010: Autism spectrum disorders may be due to cerebral toxoplasmosis associated with chronic neuroinflammation causing persistent hypercytokinemia that resulted in an increased lipid peroxidation, oxidative stress, and depressed metabolism of endogenous and exogenous substances. *Res. Autism Spectr. disord.* 4, 119–155.

Prociv, P. 1998. Zoonotic hookworm infections (Ancylostomosis). In: Palmer, S. R., E. J. L. Soulsby, and D. I. H. Smipson (eds), Zoonoses, 1st edn, pp. 803–822. Oxford Medical Publications.

Recuenco, S., B. Cherry, and M. Eidson, 2007: Potential cost savings with terrestrial rabies control. *BMC Public Health* 7, 47.

Roseveare, C. W., W. D. Goolsby, and I. M. Foppa, 2009: Potential and actual terrestrial rabies exposure in people and domestic animals, Upstate South Carolina, 1994–2004: a surveillance study. *BMC Public Health* 9, 65–70.

Rupprecht, C. E., 2002: Rabies: reemergence of the disease. *Compend. Contin. Educ. Vet.* 24, 57–60.

Rupprecht, C., K. Stohr, and C. Meredith, 2001. Rabies. In: Williams, E. S., and I. K. Barker, (ed.), Infectious Diseases of Wild Mammals, 2nd edn, pp. 3–36. Iowa State University Press, Ames, IA.

Songserm, T., A. Amonsin, R. Jam-on, N. Sae-Heng, N. Meemak, N. Pariyothorn, S. Payungporn, A. Theamboonlers, and Y. Poovorawan, 2006: Avian influenza H5N1 in naturally infected domestic cat. *Emerg. Infect. Dis.* 12, 681–683.

Sorvillo, F., B. Gondo, R. Emmons, P. Ryan, S. Waterman, and A. Tilzer, 1993: A suburban focus of endemic typhus in Los Angeles County: association with seropositive domestic cats and oppossums. *Am. J. Trop. Med. Hyg.* 48, 269–273.

Souza, M., E. Ramsey, S. Patton, and J. New, 2009: *Baylisascaris procyonis* in raccoons from Eastern Tennessee. *J. Wildl. Dis.* 45, 1231–1234.

Spagnoli, S., K. Kuroki, S. Schommer, T. Reilly, and W. Falls, 2011: Pathology in practice. *J. Am. Vet. Med. Assoc.* 238, 1271–1273.

Thiry, E., A. Zicola, D. Addie, H. Egberink, K. Hartmann, H. Lutz, H. Poulet, and M. C. Horzinek, 2007: Highly pathogenic avian influenza H5N1 virus in cats and other carnivores. *Vet. Microbiol.* 122, 25–31.

Yabsley, M., E. Blizzard, M. Beck, and S. Harsch, 2010: Geographical expansion of Baylisascaris procyonis roundworms, Florida, USA. *Emerg. Infect. Dis.* 16, 1803–1804.