

EXPOSURE TO FELINE AND CANINE PATHOGENS IN BOBCATS AND GRAY FOXES IN URBAN AND RURAL ZONES OF A NATIONAL PARK IN CALIFORNIA

Authors: Riley, Seth P. D., Foley, Janet, and Chomel, Bruno

Source: Journal of Wildlife Diseases, 40(1): 11-22

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-40.1.11

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

EXPOSURE TO FELINE AND CANINE PATHOGENS IN BOBCATS AND GRAY FOXES IN URBAN AND RURAL ZONES OF A NATIONAL PARK IN CALIFORNIA

Seth P. D. Riley,1,4,5 Janet Foley,2 and Bruno Chomel3

- ¹Department of Environmental Science and Policy, University of California, One Shields Ave., Davis, California 95616, USA
- ²Department of Medicine and Epidemiology, School of Veterinary Medicine, University of California, One Shields Ave., Davis, California 95616, USA
- ³ Department of Population Health and Reproduction, School of Veterinary Medicine, University of California, One Shields Ave., Davis, California 95616, USA
- ⁴Current address: Santa Monica Mountains National Recreation Area, National Park Service, 401 West Hillcrest Drive, Thousand Oaks, California 91360, USA
- ⁵ Corresponding author (email: seth_riley@nps.gov)

ABSTRACT: Exposure of bobcats (Lynx rufus) and gray foxes (Urocyon cinereoargenteus) to a range of common canine and feline pathogens was assessed in urban and rural zones of Golden Gate National Recreation Area, a National Park in the San Francisco Bay Area, (California, USA) from 1992 to 1995. Testing included serology for canine distemper virus, canine parvovirus (CPV), canine adenovirus, Leptospira interrogans, feline calicivirus (FCV), feline panleukopenia virus, feline herpesvirus, feline enteric coronavirus (FECV), feline immunodeficiency virus, feline leukemia virus, Toxoplasma gondii, and Bartonella henselae. Testing was also performed for Dirofilaria immitis. Significantly more gray foxes were seropositive for CPV in the urban zone than in the rural zone. In addition, radio-tracking of gray foxes in the rural zone indicated that all three of the rural CPV-seropositive foxes had traveled into adjoining small towns, whereas only one of the 11 seronegative animals had done so. Significantly more bobcats were seropositive for FCV in the rural zone than in the urban zone. Individual bobcats with positive FCV antibody titers had patterns of movement that intercepted park inholdings where domestic cats lived. Bobcat samples were seronegative for all five of the other viral feline pathogens, with the exception of a FEĈV-seropositive bobcat. High seroprevalence was detected for B. henselae and T. gondii in both zones. Variation in the seroprevalence for different pathogens might be related to differences in the exposure of bobcats and foxes to domestic animals: in the urban zone, gray foxes were located in residential areas outside the park, whereas bobcats were not. Although for most of the pathogens examined there was no relationship between urbanization and exposure, our results for CPV in foxes and FCV in bobcats indicated that proximity to urban areas or contact with humans can increase the risk of disease exposure for wild carnivore populations. Combining behavioral information from radio-tracking with data on pathogen exposure or disease incidence can provide valuable insights into the ecology of wildlife disease that might be missed with broadscale, population-level comparisons alone.

Key words: Canine parvovirus, disease ecology, feline calicivirus, Lynx rufus, nature reserve, urban wildlife, radio-tracking, Urocyon cinereoargenteus.

INTRODUCTION

Edge effects associated with humans can be an important cause of mortality for mammalian carnivores in nature reserves (e.g., Woodroffe and Ginsburg, 1998), and transmission of disease from domestic animals is one mechanism by which carnivore populations in reserves can be affected by humans (Deem et al., 2001). Disease has threatened many carnivore populations of conservation concern (MacDonald, 1996; Murray et al., 1999; Funk et al., 2001), including canine distemper in African wild

dogs (*Lycaon pictus*; Alexander et al., 1993; Laurenson et al., 1997), lions (*Panthera leo*; Harder et al., 1995; Roelke et al., 1996), and black-footed ferrets (*Mustela nigripes*; Thorne and Williams, 1988), and rabies in the Ethiopian wolf (*Canis sinensis*; Sillero-Zubiri et al., 1996). The prevalence of infectious diseases often is correlated with population size or associated with a threshold population size below which the disease goes extinct (e.g., Anderson and May, 1979; May and Anderson, 1979; Foley et al., 1999). The presence of domestic animals

among wild animals can result in a larger effective population of susceptible animals, leading to increased disease prevalence and more infections exceeding minimum threshold population sizes (Cleaveland et al., 2000, 2001). Although contact with domestic animals has been cited as a potential cause of disease in wild carnivore populations (e.g., Alexander and Appel, 1994; Packer et al., 1999; see review in Funk et al., 2001) with some possible transmission even to marine carnivores (Osterhaus et al., 1989), a direct link between domestic animals and increased disease exposure in wild carnivores has been difficult to establish.

The objective of this study was to determine whether proximity to humans and their pet cats and dogs was associated with higher seroprevalence in wild carnivores for pathogens occurring in populations of domestic dogs and cats. Seroprevalence of canine distemper virus (CDV), canine parvovirus (CPV), canine adenovirus (CAV), canine herpesvirus (CHV), Leptospira interrogans, and Dirofilaria immitis in gray foxes (Urocyon cinereoargenteus) and of feline calicivirus (FCV), feline panleukopenia virus (FPV), feline herpesvirus (FHV), feline enteric coronavirus (FECV), feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), Toxoplasma gondii, and Bartonella henselae in bobcats (Lynx rufus) was compared in urban versus rural zones of a national park in northern California (USA). A second objective was to use radio-tracking data to examine the patterns of movement of animals as they might relate to pathogen exposure. We hypothesized that pathogen exposure would be higher in the urban zone of the park and in individual animals that utilized areas of human development.

MATERIALS AND METHODS

Study area

The study was conducted in Golden Gate National Recreation Area (GGNRA) in Marin County, California. Golden Gate National Recreation Area comprises 30,000 ha of parkland in the San Francisco Bay area and is one of the most visited parks in the National Park System,

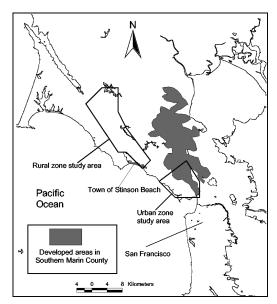


FIGURE 1. Urban and rural study sites and adjacent developed areas in Main County, California, LISA

receiving approximately 14 million visitors per year. Bobcats and gray foxes were studied in urban and rural zones of GGNRA (Fig. 1). The urban zone is in the southern part of the park, across the Golden Gate Bridge from San Francisco, and is adjacent to Highway 101, a major six- to eight-lane freeway, and the cities of Sausalito, Marin City, and Mill Valley. The rural zone is in the northern part of the park and extends from just north of the small town of Stinson Beach, northwest along Bolinas Lagoon, and through the Olema valley. The rural zone begins 15 km to the northwest of the urban zone, is 7-17 km from any dense human habitation, and borders to the east on large expanses of state park and county water district land. There are occasional dwellings and small settlements within and adjacent to the park in the rural zone, including the town of Stinson Beach and the small settlement of Jewel.

Trapping and handling

Bobcats and gray foxes were captured in homemade box traps (Zezulak, 1998) and Tomahawk live traps (Tomahawk Live Trap Co., Tomahawk, Wisconsin, USA). Captured animals were immobilized with a 5:1 (v/v) mixture of approximately 25 mg ketamine hydrochloride and 5 mg xylazine hydrochloride for foxes and 50 mg ketamine plus 10 mg xylazine for bobcats, injected intramuscularly. Monitoring of anesthesia included heart rate, respiration rate, and body temperature. Standard body

measurements and sex were recorded, and age was assessed on the basis of incisor wear and body size and weight. Animals were marked with eartags and fitted with radio collars if adult (Telonics, Inc., Mesa, Arizona, USA). When the fox or bobcat began to regain consciousness, an intramuscular injection of 0.2 to 0.4 mg yohimbine hydrochloride was given to antagonize the xylazine, and when fully recovered, the animal was released at the site of capture. Capture and handling procedures were approved by the Animal Care and Use Committee at the University of California at Davis (protocol 5328; May, 1992).

Radiotracking and home range estimation

Animals were radio-tracked from July 1992 to September 1995 in the urban zone and from July 1993 to September 1995 in the rural zone. Animals in both zones were intensively radiotracked for at least 12 mo (August 1992–March 1994 in the urban zone, January 1994-March 1995 in the rural zone), during which time at least three locations, two daytime and one nighttime, were obtained each week for each animal. Sequential radio-tracking sessions were conducted, during which three to five animals were located every hour for the full 24-hr cycle in two 12-hr periods. These sequential locations were not used to compute adaptive kernel home ranges because they were not independent; however, the home ranges computed from independent locations encompassed all of the locations obtained in sequential tracking sessions.

Locations were obtained by triangulation with an H-Adcock peak antenna (Telonics, Inc.). All bearings for a particular location were taken within 15 min, and when three reliable bearings could not be obtained within this period, two bearings were used (ca. 20% of locations). Bearings were taken from the same drainage as the radio-collared animal to minimize the distance between the observer and the target and the effects of intervening topography. Receiver locations were determined to within 2-5 m by a Global Positioning System (GPS; Pathfinder Plus, Trimble Inc., Sunnyvale, California). Animals were sometimes directly observed, and visual locations were determined with two bearings or one bearing and the distance (m).

Telemetry system accuracy was tested by direct measurement. Radio-collars were placed within the home range of an animal at locations unknown to the observer and were located as if they were that animal. The radio-collar location was determined by GPS and compared to the triangulated location. The mean distance

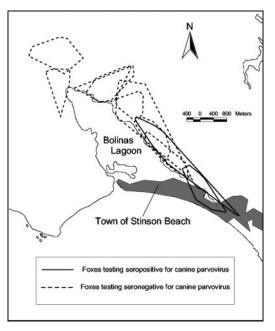


FIGURE 2. Minimum convex polygon (100%) home ranges of gray foxes testing seronegative and seropositive for canine parvovirus along Bolinas Lagoon at the rural study site, Marin County, California, USA.

between triangulated locations and test collars was 76.6 \pm 53.3 m (\pm SD; range, 17.7–287.1 m; n=37).

For each radio-collared animal, 100% and 95% minimum convex polygon home ranges (MCPs; Hayne, 1949) and 95% adaptive kernel home ranges (Worton, 1989) were computed as representations of the overall home range. Often, ecologically important events, such as the use of residential areas, were represented by a small number of points, so this was represented graphically by 100% MCPs (e.g., Fig. 2).

Pathogen exposure

Approximately 5–12 ml of blood was collected from each animal by jugular (foxes) or cephalic (bobcats) venipuncture. Blood was kept on ice in the field (generally 1–4 hr) and then centrifuged later that day for 20 min at 1,200XG. Serum was removed to separate tubes and stored at -20 C until tested.

Antibodies to FIV, FECV, FPV, FCV, FHV, CDV, B. henselae, and CPV were detected in sera by indirect immunofluorescent antibody assays (IFAs). For FIV, the substrate was FIV-Petaluma in Crandell feline kidney cells (CRDK); for FHV and FPV, the cell line was also CRDK infected with field strain viruses; and for FECV, the substrate was FECV-UCD1

TABLE 1. Seroprevalence of pathogens in gray foxes (n=27 for urban zone, n=14 for rural zone) and bobcats (n=12 for urban zone, n=13 for rural zone) in Golden Gate National Recreation Area, Marin County, California (only pathogens with at least one positive result are listed, see text for all negative results).

Pathogen	% Positive urban	% Positive rural	% Positive total
Gray fox			
Canine parvovirus	63	21	49
Canine adenovirus	88	86	88
Canine heartworm	4	7	5
Leptospira interrogans ^a			
Serovar pomona	0-4	0–7	0-5
Serovar bratislava	4–7	0–7	4-7
Bobcat			
Feline infectious peritonitis/feline enteric coronavirus	0	8	4
Feline calicivirus	17	67	46
Toxoplasma gondii	100	77	88
Bartonella henselae	73	75	74

^a For two foxes that were seropositive for *L. interrogans*, we could not determine whether the serovar was pomona or bratislava.

in Felis catus whole fetal-4 (Fcwf-4) cells. Bartonella henselae was assayed with whole-cell preparations of strain U4 in Fcwf-4 cells. Canine distemper virus antibodies were detected with the use of CDV-infected ferret kidney cells (American Bioresearch, Milton, Tennessee, USA) and CPV and FCV antibodies were detected with commercially available substrate slides (Veterinary Medical Research and Development, Pullman, Washington, USA). Feline sera were serially diluted in 10 mM phosphatebuffered saline solution (138 mM NaCl, 2.7 mM KCl), pH 7.4, with a cutoff of 1:25 considered positive. Sera to be tested for CDV antibodies were screened at 1:8 and for CPV antibodies at 1:10. Titer was defined as the highest dilution that produced distinct fluorescence in foci of infected cells, and positive and negative control sera were included on each slide. Feline leukemia virus antigen was measured with an enzyme-linked immunosorbent assay (ELISA) antigen test kit (Synbiotics Corp., San Diego, California). Canine herpesvirus and CAV antibodies were detected by virus neutralization test with serial dilutions from 1:4 to 1:512, with neutralization at a dilution of > 1:4indicating a positive result. Microscopic agglutination was used to detect antibodies against L. interrogans serovars canicola, icterohaemorrhagiae, pomona, and bratislava. Adult canine heartworm antigen was assayed using a commercial ELISA (Dirochek, Synbiotics Corp.). Exposure to T. gondii was assessed by latex agglutination (Toxotest-MT "Eiken" kit, Eiken Chemical Co., Tokyo, Japan). Chi-squared contingency tests were used to test for differences

in disease seroprevalence between the urban and rural zones.

RESULTS

Thirty-two gray foxes and 12 bobcats were captured in the urban zone. Blood was collected from 27 foxes and all bobcats, and 20 foxes and 10 bobcats were radio-collared. In the rural zone, 16 gray foxes and 13 bobcats were captured. Blood was collected from 14 foxes and 13 bobcats, and 15 foxes and 12 bobcats were radio-collared. Although one bobcat and one fox exhibited long-distance dispersal movements, no radio-collared animals ever moved from the urban zone to the rural zone, or vice versa.

Seroprevalence varied by pathogen and, in some cases, by geographic zone (Table 1). Differences between the urban and rural zones were detected for CPV in foxes, for which seroprevalence was higher in the urban zone (63% [17/27] vs. 21% [3/14] in the rural zone; χ^2 =6.36, 1 df, P=0.012), and FCV in bobcats, for which seroprevalence was higher in the rural zone (67% [8/12] vs. 17% [2/12] in the urban zone; χ^2 =6.17, 1 df, P=0.013). All foxes were seronegative for CDV, CHV, and serovars icterohaemorrhagiae and canicola of L. in-

Developed area contact	n	No. positive	No. negative
Urban zone			
Contact with urban development outside the park	8	5	3
Contact with developed areas within the park	5	4	1
No contact with developed areas	5	3	2
Rural zone			
Contact with rural towns/settlements	4	3	1
No contact with rural towns/settlements	9	0	9

TABLE 2. Results of serology of radio-collared gray foxes for canine parvovirus, relative to their contact with developed areas, Golden Gate National Recreation Area, Marin County, California.

terrogans. All bobcats tested were seronegative for FeLV, FIV, FPV, FHV, and FECV, except for one animal in the rural zone, which was seropositive for FECV. For CAV, seroprevalence was higher in older foxes: four of seven young foxes (yearlings or young-of-the-year) but only one of 34 adult foxes were seronegative (Fisher's exact test, P=0.002). Three foxes were seropositive for L. interrogans: one fox in the urban zone was exposed to the serovar bratislava and one fox in the urban zone and one in the rural zone were exposed to either serovar pomona or bratislava.

Animal movements and seroprevalence

Radio-tracking data were used to examine the relationship between CPV exposure and travel by gray foxes into residential areas (Table 2). In the rural zone, 13 animals were radio-collared and tested for CPV antibodies, 11 of which inhabited an area adjacent to and just north of Bolinas Lagoon. Of these 11 foxes, the only two that were CPV-seropositive were also the only two foxes that were radio-located in the town of Stinson Beach (Fig. 2). The other two tested and radio-collared foxes in the rural zone utilized the small northern settlement of Jewel, which included houses and pet dogs. One of these animals was also CPV-seropositive.

In the urban zone, 18 gray foxes were radio-tracked and tested for CPV antibodies. Eight foxes had home ranges that overlapped residential areas outside the park; five animals were frequently located around developed areas within the park, such as horse stables, offices, and residences; and five foxes had home ranges that did not overlap developed areas either inside or outside the park, although two of their ranges bordered residential areas outside the park (Riley, 1999). Nine of the 13 animals with direct contact with human development were CPV-seropositive, and even among those without such contact, three of five were seropositive (Table 2).

In the urban zone, 10 bobcats were tested for FCV antibodies and radio-collared; two were seropositive. One of these animals was the only bobcat ever radio-located around the commercial stable in the park, and the other was a younger female searching for a home range, and the bobcat most frequently located among the offices and residences within the park. No bobcats were ever radio-located outside the park in the urban zone (Riley, 1999). In the rural zone, 11 bobcats tested for FCV antibodies were also radio-collared. Of these 11 animals, nine had home ranges that included ranch houses and private residences within the park; eight of these animals were seropositive. The only two bobcats whose home ranges did not include these ranches and residences were seronegative.

DISCUSSION

We found evidence of pathogens in wild felids and canids in a national park in Northern California, and for two pathogens seroprevalence was significantly increased when there was potential contact with people and their pets. Effective conservation of wild carnivores must take into account the potential for disease transmission from pets to wild carnivores and take steps to reduce this transmission. Although many authors have suggested a link between disease in wild carnivore populations and humans (Alexander and Appel, 1994; Cleaveland and Dye, 1995; Sillero-Zubiri et al., 1996; Laurenson et al., 1998; Rhodes et al., 1998), few studies have directly assessed carnivore populations that have varied in their amount of contact with people, and none have examined simultaneous radio-tracking data for a link to pathogen exposure. Truyen et al. (1998) did not find a significant difference in disease exposure among suburban and rural red foxes (Vulpes vulpes) in Germany, although Valenzuela et al. (2000) found that an epizootic of notoedric mange in coatis (Nasua narica) in Mexico began in the part of a reserve near a small town. Twenty-two coyotes (Canis latrans) in urban Tucson, Arizona, had high levels of exposure to four canine diseases, including 100% seroprevalence for CPV, although results were not compared with a nonurban population (Grinder and Krausman, 2001).

In this study, wild canids had significantly higher CPV seroprevalence in the urban zone than in the rural zone. Only three gray foxes in the rural zone (21%) had evidence of parvovirus exposure, and all three were known by radio-tracking to have traveled into residential areas. In the urban zone, even animals that did not move out of the park or inhabit park edges were seropositive for CPV, indicating that the virus was present throughout the fox population in the urban zone, not just in animals with direct contact with people. Canine parvovirus is shed in large quantities in the feces of infected animals and can survive for months or years in the environment (Appel and Parrish, 1987), potentially causing significant mortality in wild canids, including coyotes (Windberg,

1995) and wolves (Canis lupus; Johnson et al., 1994; Mech and Goyal, 1995; Peterson et al., 1998). Foxes might have been exposed to CPV by interacting with pet dogs visiting the park, pets from neighboring residential areas that sometimes roam unattended in the park, or dogs that live in park inholdings, either through direct contact with these dogs or more likely indirectly through contact with their feces. Foxes also might be exposed to parvovirus through exposure to other foxes carrying the virus. There is extensive home range overlap among foxes in the urban zone, and the home range of every radio-collared fox that did not actually utilize residential areas overlapped the home range of a fox that did (Riley, 1999).

Gray foxes also were exposed to CAV, a common respiratory pathogen in many wild canid populations independent of exposure to human development (e.g., Davidson et al., 1992). Canine adenovirus replicates in canine respiratory mucosal cells and is spread through exposure to respiratory secretions. Most immunocompetent animals suffer little morbidity and recover fully, but occasional dogs can develop signs of "kennel cough" or pneumonia (Ford and Vaden, 1998). The low prevalence of heartworm in gray foxes in this study is consistent with the low prevalence of the disease in dogs in Marin county (Theis et al., 1995) and in gray foxes generally (e.g., Wixsom et al., 1991), although Sacks (1998) found a heartworm prevalence of 76% in 33 coyotes just 137 km (85 miles) to the north of our study area.

Leptospirosis can be an acute disease in dogs, other animals, and humans worldwide, although most infections are mild or chronic, and is caused by the spirochete *L. interrogans* (Solomon, 1994), Gray foxes in GGNRA were exposed to serovars pomona and bratislava, consistent with results in dogs in northern California (Adin and Cowgill, 2000). Use of a vaccine containing the serovars icterohaemorrhagiae and canicola in dogs might have resulted

in reduced environmental contamination with these organisms and a shift in canine cases to other serovars (Rentko et al., 1992; Greene et al., 1998). Nevertheless, the San Francisco Bay area remains the location from which the majority (77%) of canine cases referred to the University of California Davis teaching hospital have originated over the last decade (Adin and Cowgill, 2000) and a location where foxes and other susceptible animals will likely continue to be infected.

Canine pathogens for which there was no evidence of exposure included CDV and CHV. Although every gray fox was seronegative for CDV exposure between 1992 and 1995, there were reports the following year of a widespread outbreak of canine distemper in foxes in Marin County, confirmed by elevated IgG and IgM titers (B. Puget, pers. comm.). Lack of herd immunity against distemper in foxes in this study might have predisposed the fox populations in GGNRA to an epidemic, although given the high virulence of this pathogen for gray foxes, population or even individual immunity might be rare or nonexistent.

Lack of evidence of CHV probably reflects the low transmissibility of the virus among species. Canine herpesvirus is relatively common in purebred dogs and is spread through respiratory excretions or genital contact with infected animals (Appel, 1987). However, CHV is not stable in the environment and the intimate contact necessary for transmission seems unlikely between domestic dogs and gray foxes.

The most common viral pathogen to which bobcats in this study were exposed was FCV. Feline calicivirus is highly transmissible via feline respiratory secretions. Despite being inactivated quickly in sunlight, the casual contact observed by one of the authors (SPDR) among bobcats and domestic cats in the field would probably have sufficed for transmission. The significantly higher FCV seroprevalence in the rural zone was opposite of what we expected. In contrast to gray foxes, bobcats

were never radio-located outside the park in the urban zone (Riley, 1999). Therefore, exposure to pathogens would likely come from contact with animals inside the park, and the only two animals that were seropositive in the urban zone were also the two bobcats with the most contact with the commercial stable and with residences within the park. Many of the bobcats in the rural zone occupied home ranges that included ranch houses and isolated homes within the park. Eight of the nine bobcats with such home ranges were seropositive for FCV, whereas both of the bobcats without contact with ranch houses were seronegative. These data suggest that FCV might have been endemic in domestic cats and not bobcats, given that urban zone bobcats did not have evidence of FCV infection. Further data regarding the health and infection status of domestic cats in GGNRA would be informative.

Antibodies to FECV were detected in one bobcat. Although FECV is highly infectious through exposure to virus in feces and is endemic in multiple-cat households (Pedersen, 1995; Foley et al., 1997), the virus does not persist in the environment for more than about 36 hr (Pedersen, 1987b). Maintaining FECV infection endemically requires dense cat populations (Foley et al., 1997b), so the very low exposure to FECV observed in the bobcats in this study was not surprising.

There was no evidence of bobcat exposure to FIV, FeLV, or FHV. These three viruses require direct or intimate contact for transmission. Lack of evidence of exposure of bobcats to FPV suggests that this population might be vulnerable to an outbreak in the future. During an epizootic in a Florida population, 11 of 18 radio-collared bobcats died of FPV over a 3-mo period (Wassmer et al., 1988).

Seroprevalence for *T. gondii* was high in bobcats in both the urban and rural zones. *Toxoplasma gondii* can encyst in humans, livestock, and carnivores (Dubey and Beattie, 1988; Dubey, 1994), but felids are the

definitive hosts. Although infection is common in felids, clinical toxoplasmosis is rare (Dubey et al., 1987). Serologic evidence of exposure of wild felids to *T. gondii* is common: in six other studies of bobcats, including two in California, the prevalence of antibodies to *T. gondii* ranged from 34% to 73% (Dubey and Beattie, 1988). It is likely that most infections in bobcats occur after exposure to infected prey.

Three quarters of the bobcats in both zones were seropositive for *B. henselae*, the agent of cat scratch disease, which is a flea-transmitted zoonosis with a feline reservoir. Exposure to *B. henselae* was reported in 20 species of captive wild felids, including bobcats, and from free-ranging bobcats and mountain lions (*Felis concolor*) from California (Yamamoto et al., 1998), where seroprevalence for bobcats was 53%. Generally, *Bartonella* is not associated with clinical disease in felids.

Management and conservation implications

Results of this study illustrated that dense human populations and increased contact with people and their pets can lead to increased pathogen exposure in wildlife. Results such as these can be important for managing rare carnivore populations. For example, population viability analysis incorporating disease for the highly endangered Ethiopian wolf suggests that exposure to large dog populations in urban centers next to a national park could have significant implications for this species' survival (Laurenson et al., 1998; Haydon et al., 2002). Interspecific variability in social structure might also contribute to variability in exposure to domestic animal diseases. In this study, exposure to infections transmitted between carnivores occurred more frequently in foxes than in bobcats, a difference that could be related to their greater sociality. In general, disease epizootics are reported more frequently in canids than in felids. In their review of disease in large carnivores, Murray et al. (1999) documented 16 epidemics that

caused a population change, of which 15 were in canids and only one in a felid, the African lion. Most members of the family Felidae, including bobcats, are solitary and territorial (Kleiman and Eisenberg, 1973; Sandell, 1989), rendering widespread disease epidemics less likely. African lions and domestic cats, more social felids (Schaller, 1972; MacDonald et al., 1987), also experience disease epizootics (e.g., FeLV and FIV in cats, canine distemper in lions). Canids, in contrast, are among the most social of mammals, and epizootics of contagious diseases are reported in many members of this family including red foxes (Anderson et al., 1981), gray foxes (Hoff et al., 1974), Ethiopian wolves (Sillero-Zubiri et al., 1996), African wild dogs (Ginsburg et al., 1995), coyotes (Windberg, 1995), and wolves (Petersen et al., 1998).

Variation in utilization of human development could also produce variation in disease exposure. In this study, foxes regularly used developed areas both inside and outside the park in the urban zone, whereas bobcats were never located there. In the rural zone, however, although small towns were visited by a few foxes, neither species was widely exposed to high-density human populations. However, the presence of ranches and horse stables within the park provided locations where wild carnivores could have been exposed to domestic animals. Movement and FCV seroprevalence data suggest that this might be the case for bobcats. Although bobcats, like gray foxes, are not an endangered species, small populations of other solitary cat species, such as reintroduced lynx (Lynx lynx) in Switzerland (Schmidt-Posthaus et al., 2002) and European wildcats Felis sylvestris in Scotland (Daniels et al., 1999) and mainland Europe (Leutenegger et al., 1999), could be at risk from diseases transmitted by domestic cats.

If decreasing mortality from disease is a priority for either of these populations, the best strategy for foxes might be to target domestic animals in developed areas outside the park, whereas for bobcats, the priority might be to reduce or vaccinate domestic pets within the park. In general, reserve or land ownership boundaries are not limits to animal movement or to ecological processes (Schonewald-Cox and Bayless, 1986; Knight and Landres, 1998), and in this case, pathogen exposure occurs far across the boundary of the national park, specifically for CPV and FCV. Managers of protected areas should consider reducing or eliminating access of domestic animals to reserves when species susceptible to the pathogens of domestic animals are present, especially if the species is of conservation concern. Requiring the collection of feces and reducing feral and unaccompanied domestic animals in reserves might also help reduce the risk of transmission of many diseases. More studies of wild populations in rural and urban areas are needed, but combining data on pathogen exposure with information on individual animal home ranges and movements relative to human development would improve our understanding of pathogen transmission between domestic and wild species.

ACKNOWLEDGMENTS

Some tests on gray fox sera were conducted by B. Puget (Veterinary Medicine Teaching Hospital, University of California, Davis, California; distemper, parvovirus, and herpesvirus), the Washington Animal Disease Diagnostic Laboratory of J. Evermann (Washington State University, Pullman, Washington; adenovirus), and C. Scott (University of California, Davis; heartworm). S. Riley thanks the Golden Gate National Recreation Area for extensive logistical and financial support throughout the field portion of this work; J. Howell, C. Schonewald, and D. Van Vuren for encouragement and assistance both in the field and after; Earthwatch for financial support; 18 teams of Earthwatch volunteers for their assistance and enthusiasm; and B. McKeon and G. Brooks for their excellent and tireless work in the field. We thank E. Kelley, B. Sacks, B. Lewison, J. Neale, M. Hoopes, J. Fordyce, M. Marchetti, D. Van Vuren, and three anonymous reviewers for comments on earlier versions of the manuscript.

LITERATURE CITED

- ADIN, C. A., AND L. D. COWGILL. 2000. Treatment and outcome of dogs with leptospirosis: 36 cases (1990–1998). Journal of the American Veterinary Medicine Association 216: 371–375.
- ALEXANDER, K. A., AND M. J. G. APPEL. 1994. African wild dogs (*Lycaon pictus*) endangered by a canine distemper epizootic among domestic dogs near the Masai Mara National Reserve, Kenya. Journal of Wildlife Diseases 30: 481–485.
- ——, P. A. CONRAD, I. A. GARDNER, C. PARISH, M. APPEL, M. G. LEVY, N. LERCHE, AND P. KAT. 1993. Serologic survey of selected canine pathogens in African wild dogs (*Lycaon pictus*) and sympatric domestic dogs in Masai Mara. Journal of Zoo and Wildlife Medicine 24: 140–144.
- ANDERSON, R. M., AND R. M. MAY. 1979. Population biology of infectious diseases, Part I. Nature 280: 361–367.
- ———, H. C. JACKSON, R. M. MAY, AND A. D. M. SMITH. 1981. Population dynamics of fox rabies in Europe. Nature 289: 765–771.
- APPEL, M. J. G. 1987. Canine herpesvirus. In Virus infections of carnivores, M. J. G. Appel (ed.). Elsevier, New York, New York, pp. 5–15.
- —, AND C. R. PARRISH. 1987. Canine parvovirus type 2. In Virus infections of carnivores, M. J. G. Appel (ed.). Elsevier, New York, New York, pp. 69–62.
- CLEAVELAND, S., AND C. DYE. 1995. Maintenance of a microparasite infecting several host species: Rabies in the Serengeti. Parasitology 111: S33—S47.
- ——, M. G. J. APPEL, W. S. K. CHALMERS, C. CHILLINGWORTH, M. KAARE, AND C. DYE. 2000. Serological and demographic evidence for domestic dogs as a source of canine distemper virus infection for Serengeti wildlife. Veterinary Microbiology 72: 217–227.
- ——, G. R. Hess, A. P. Dobson, M. K. Laurenson, H. I. McCallum, M. G. Roberts, and R. Woodroffe. 2001. The role of pathogens in biological conservation. *In* The ecology of wild-life diseases, P. J. Hudson, A. Rizzoli, B. T. Grenfell, H. Heesterbeek, and A. P. Dobson (eds.). Oxford University Press, Oxford, UK, pp. 139–150.
- DANIELS, M. J., M. C. GOLDER, O. JARRETT, AND D. W. MACDONALD. 1999. Feline viruses in wildcats from Scotland. Journal of Wildlife Diseases 35: 121–124.
- DAVIDSON, W. R., M. J. APPEL, G. L. DOSTER, O. E. BAKER, AND J. F. BROWN. 1992. Diseases and parasites of red foxes, gray foxes, and coyotes from commercial sources selling to fox-chasing

- enclosures. Journal of Wildlife Diseases 28: 581–589.
- DEEM, S. L., W. B. KARESH, AND W. WEISMAN. 2001. Putting theory into practice: Wildlife health in conservation. Conservation Biology 15: 1224–1233.
- DUBEY, J. P. 1994. Toxoplasmosis. Journal of the American Veterinary Medicine Association 205: 1593–1598.
- —, AND C. P. BEATTIE. 1988. Toxoplasmosis of animals and man. CRC Press, Inc., Boca Raton, Florida, 220 pp.
- ——, W. J. QUINN, AND D. WEINANDY. 1987. Fatal neonatal toxoplasmosis in a bobcat (*Lynx rufus*). Journal of Wildlife Diseases 23: 324–327.
- FOLEY, J., P. FOLEY, AND N. PEDERSEN. 1999. The persistence of an SIS disease in a metapopulation. Journal of Applied Ecology 36: 555–563.
- FOLEY, J. E., A. POLAND, J. CARLSON, AND N. C. PEDERSEN. 1997a. Patterns of feline coronavirus infection and fecal shedding from cats in multiple-cat environments. Journal of the American Veterinary Medical Association 210: 1307–1312.
- , —, AND ——. 1997b. Risk factors for feline infectious peritonitis among cats in multiple-cat environments with endemic feline enteric coronavirus. Journal of the American Veterinary Medical Association 210: 1313–1318.
- FORD, R., AND S. VADEN. 1998. Canine infectious tracheobronchitis. *In* Infectious diseases of the dog and cat, C. Greene (ed.). Saunders, Philadelphia, Pennsylvania, pp. 33–38.
- FUNK, S. M., C. V. FIORELLO, S. CLEAVELAND, AND M. E. GOMPPER. 2001. The role of disease in carnivore ecology and conservation. *In Carnivore* conservation, J. L. Gittleman, S. M. Funk, D. Macdonald, and R. K. Wayne (eds.). Cambridge University Press, Cambridge, UK, pp. 441–466.
- GINSBURG, J. R., K. A. ALEXANDER, S. CREEL, P. W. KAT, J. W. MCNUTT, AND M. G. L. MILLS. 1995. Handling and survivorship of African wild dog (*Lycaon pictus*) in five ecosystems. Conservation Biology 9: 665–674.
- GREENE, C., M. MILLER, AND C. BROWN. 1998. Leptospirosis. In Infectious diseases of the dog and cat, C. Greene (ed.). Saunders, Philadelphia, Pennsylvania, pp. 273–281.
- GRINDER, M., AND P. R. KRAUSMAN. 2001. Morbidity-mortality factors and survival of an urban coyote population in Arizona. Journal of Wildlife Diseases 37: 312–317.
- HARDER, T. C., M. KENTER, M. J. G. APPEL, M. E. ROELKE-PARKER, T. BARRETT, AND A. D. M. E. OSTERHAUS. 1995. Phylogenetic evidence of canine distemper in Serengeti's lions. Vaccine 13: 521–523.
- HAYDON, D. T., M. K. LAURENSON, AND C. SILLERO-

- ZUBIRI. 2002. Integrating epidemiology into population viability analysis: Managing the risk posed by rabies and canine distemper to the Ethiopian wolf. Conservation Biology 16: 1372–1385.
- HAYNE, D. W. 1949. Calculation of size of home range. Journal of Mammalogy 30: 1–18.
- HOFF, G. L., W. J. BIGLER, S. J. PROCTOR, AND L. P. STALLING. 1974. Epizootic of canine distemper virus infection among urban raccoons and gray foxes. Journal of Wildlife Diseases 10: 423– 428.
- JOHNSON, M. R., D. K. BOYD, AND D. H. PLETSCH-ER. 1994. Serology of canine parvovirus and canine distemper in relation to wolf (*Canis lupus*) pup mortalities. Journal of Wildlife Diseases 30: 270–273.
- KLEIMAN, D. G., AND J. F. EISENBERG. 1973. Comparisons of canid and felid social systems from an evolutionary perspective. Animal Behavior 21: 637–659.
- KNIGHT, R. L., AND P. B. LANDRES. 1998. Stewardship across boundaries. Island Press, Washington, D.C., 366 pp.
- LAURENSON, K., J. VAN HEERDEN, P. STANDER, AND M. J. VAN VUREN. 1997. Seroepidemiological survey of sympatric domestic and wild dogs (*Ly-caon pictus*) in Tsumkwe District, north-eastern Namibia. Onderstepoort Journal of Veterinary Research 64: 313–316.
- ——, C. SILLERO-ZUBIRI, H. THOMPSON, F. SHI-FERAW, S. THIRGOOD, AND J. MALCOLM. 1998. Disease as a threat to endangered species: Ethiopian wolves, domestic dogs and canine pathogens. Animals Conservation 1: 273–280.
- Leutenegger, C. M., R. Hofmann-Lehmann, C. Riols, M. Liberak, G. Worel, P. Lups, D. Fehr, M. Hartmann, P. Weilenmann, and H. Lutz. 1999. Viral infections in free-living populations of the European wildcat. Journal of Wildlife Diseases 35: 678–686.
- MACDONALD, D. W. 1996. Dangerous liaisons and disease. Nature 379: 400–401.
- ———, P. J. APPS, G. M. CARR, AND G. KERBY. 1987. Social dynamics, nursing coalitions and infanticide among farm cats, *Felis catus*. Advances in Ethology 28.
- MAY, R. M., AND R. M. ANDERSON. 1979. Population biology of infectious diseases, Part II. Nature 280: 455–461.
- MECH, L. D., AND S. M. GOYAL. 1995. Effects of canine parvovirus on gray wolves in Minnesota. Journal of Wildlife Management 59: 565–570.
- MURRAY, D. L., C. A. KAPKE, J. F. EVERMANN, AND T. K. FULLER. 1999. Infectious disease and the conservation of free-ranging large carnivores. Animal Conservation 2: 241–254.

- OSTERHAUS A. D. M. E., J. GROEN, F. G. C. M. UYTDEHAAG, G. VISSER, M. W. G. VAN DE BILT, A. BERGMANN, AND B. KLINGEBORN. 1989. Distemper virus in Baikal seals. Nature 338: 209–210.
- PACKER, C., S. ALTIZER, M. APPEL, E. BROWN, J. MARTENSON, S. J. O'BRIEN, M. ROELKE-PARKER, R. HOFMANN-LEHMANN, AND H. LUTZ. 1999. Viruses of the Serengeti: Patterns of infection and mortality in African lions. Journal of Animal Ecology 68: 1161–1178.
- PEDERSEN, N. C. 1987. Feline infectious peritonitis virus. In Virus infections of carnivores, M. J. G. Appel (ed.). Elsevier, New York, New York, pp. 267–286.
- 1995. An overview of feline enteric coronavirus and infectious peritonitis virus infections. Feline Practice 23: 7–22.
- Peterson, R. O., N. J. Thomas, J. M. Thurber, J. A. Vucetich, and T. A. Waite. 1998. Population limitation and the wolves of Isle Royale. Journal of Mammalogy 79: 828–841.
- RENTKO, V. T., N. CLARK, L. A. ROSS, AND S. H. SCHELLING. 1992. Canine leptospirosis. A retrospective study of 17 cases. Journal of Veterinary Internal Medicine 6: 235–244.
- RHODES, C. J., R. P. D. ATKINSON, R. M. ANDERSON, AND D. W. MACDONALD. 1998. Rabies in Zimbabwe: Reservoir dogs and the implications for disease control. Philosophical Transactions of the Royal Society of London B 353: 999–1010.
- RILEY, S. P. D. 1999. Spatial organization, food habits and disease ecology of bobcats (*Lynx rufus*) and gray foxes (*Urocyon cinereoargenteus*) in national park areas in urban and rural Marin County, California. Ph.D. Dissertation, University of California, Davis, California, 163 pp.
- ROELKE, M. E., L. MUNSON, C. PACKER, R. KOCK, S. CLEAVELAND, M. CARPENTER, S. J. O'BRIEN, A. POSPISCHIL, R. HOFMANN-LEHMANN, H. LUTZ, G. L. M. MWAMENGELE, M. N. MGASA, G. A. MACHANGE, B. A. SUMMERS, AND M. J. G. APPEL. 1996. A canine distemper virus epidemic in Serengeti lions (*Panthera leo*). Nature 379: 441–445.
- SACKS, B. 1998. Increasing prevalence of canine heartworm in coyotes from California. Journal of Wildlife Diseases 34: 386–389.
- SANDELL, M. 1989. The mating tactics and spacing patterns of solitary carnivores. *In* Carnivore behavior, ecology, and evolution, J. L. Gittleman (ed.). Cornell University Press, Ithaca, New York, pp. 164–182.
- SCHALLER, G. B. 1972. The Serengeti lion. University of Chicago Press, Chicago, Illinois, 480 pp.
- SCHMIDT-POSTHAUS, H., C. BREITENMOSER-WÜRSTEN, H. POSTHAUS, L. BACCIARINI, AND U.

- BREITENMOSER. 2002. Causes of mortality in reintroduced Eurasian lynx in Switzerland. Journal of Wildlife Diseases 38: 84–92.
- SCHONEWALD-COX, C. M., AND J. W. BAYLESS. 1986. The boundary model: A geographical analysis of design and conservation of nature reserves. Biological Conservation 38: 305–322.
- SILLERO-ZUBIRI, C., A. A. KING, AND D. W. MAC-DONALD. 1996. Rabies and mortality in Ethiopian wolves (*Canis sinensis*). Journal of Wildlife Diseases 32: 80–86.
- SOLOMON, F. 1994. Leptospira and leptospirosis. CRC Press, Inc., Boca Raton, Florida, 353 pp.
- THEIS, J. H., R. G. SCHWAB, F. STEVEN, AND C. E. FRANTI. 1995. Canine filariasis due to *Dirofilaria immitis* (heartworm). *In* Canine filariasis in California, J. H. Theis, R. G. Schwab, F. Steven, and C. E. Franti (eds.). Merck and Company, Inc., St. Louis, Missouri, pp. 1–55.
- THORNE, E. T., AND E. S. WILLIAMS. 1988. Disease and endangered species: The black-footed ferret as a recent example. Conservation Biology 2: 66–74.
- TRUYEN, U., T. MÜLLER, R. HEIDRICH, K. TACK-MANN, AND L. E. CARMICHAEL. 1998. Survey on viral pathogens in wild red foxes (*Vulpes vulpes*) in Germany with emphasis on parvoviruses and analysis of DNA sequence from a red fox parvovirus. Epidemiology and Infection 121: 433–440.
- Valenzuela, D., G. Ceballos, and A. Garcia. 2000. Mange epizootic in white-nosed coatis in western Mexico. Journal of Wildlife Diseases 36: 56–63.
- Wassmer, D. A., D. D. Guenther, and J. N. Layne. 1988. Ecology of the bobcat in south-central Florida. Bulletin of the Florida State Museum, Biological Sciences 3: 159–228.
- WINDBERG, L. A. 1995. Demography of a high-density coyote population. Canadian Journal of Zoology 73: 942–954.
- WIXSOM, M. J., S. P. GREEN, R. M. CORWIN, AND E. K. FRITZELL. 1991. *Dirofilaria immitis* in coyotes and foxes in Missouri. Journal of Wildlife Diseases 27: 166–169.
- WOODROFFE, R., AND J. R. GINSBERG. 1998. Edge effects and the extinction of populations inside protected areas. Science 280: 2126–2128.
- WORTON, B. J. 1989. Kernel methods for estimating the utilization distribution in home-range studies. Ecology 70: 164–168.
- Yamamoto, K. B., B. Chomel, L. J. Lowenstine, Y. Kikuchi, L. G. Phillips, B. C. Barr, P. K. Swift, K. R. Jones, S. P. D. Riley, R. W. Kasten, J. E. Foley, and N. C. Pedersen. 1998. *Bartonella henselae* antibody prevalence in free-

ranging and captive wild felids from California. Journal of Wildlife Diseases 34: 56–63.

ZEZULAK, D. S. 1998. Spatial, temporal, and population characteristics of two bobcat, *Lynx rufus*,

populations in California. PhD Dissertation, University of California, Davis, California, 165 pp.

Received for publication 4 November 2002.