



## **FACTORS ASSOCIATED WITH PATHOGEN SEROPREVALENCE AND INFECTION IN ROCKY MOUNTAIN COUGARS**

Authors: Biek, Roman, Ruth, Toni K., Murphy, Kerry M., Anderson, Charles R., Johnson, Mark, et al.

Source: Journal of Wildlife Diseases, 42(3) : 606-615

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-42.3.606>

---

BioOne Complete ([complete.BioOne.org](https://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](https://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.

## FACTORS ASSOCIATED WITH PATHOGEN SEROPREVALENCE AND INFECTION IN ROCKY MOUNTAIN COUGARS

Roman Biek,<sup>1,9</sup> Toni K. Ruth,<sup>2</sup> Kerry M. Murphy,<sup>2</sup> Charles R. Anderson, Jr.,<sup>3</sup> Mark Johnson,<sup>4</sup> Richard DeSimone,<sup>5</sup> Rachel Gray,<sup>6</sup> Maurice G. Hornocker,<sup>2</sup> Colin M. Gillin,<sup>7</sup> and Mary Poss<sup>1,8</sup>

<sup>1</sup> Wildlife Biology Program, University of Montana, Missoula, Montana 59812, USA

<sup>2</sup> Hornocker Wildlife Institute/Wildlife Conservation Society, Bozeman, Montana 59030, USA

<sup>3</sup> Wyoming Cooperative Fish and Wildlife Research Unit, University of Wyoming, Laramie, Wyoming 82017, USA

<sup>4</sup> Global Wildlife Resources, Inc., Bozeman, Montana 59719, USA

<sup>5</sup> Montana Fish, Wildlife, and Parks, Helena, Montana 59620, USA

<sup>6</sup> Beringia South, Kelly, Wyoming 83011, USA

<sup>7</sup> Center for Conservation Medicine, Tufts University, North Grafton, Massachusetts 01536, USA

<sup>8</sup> Division of Biological Sciences, University of Montana, Missoula, Montana 59812, USA

<sup>9</sup> Corresponding author (email: rbiek@emory.edu)

**ABSTRACT:** Serological and genetic material collected over 15 years (1990–2004) from 207 cougars (*Puma concolor*) in four populations in the Rocky Mountains were examined for evidence of current or prior exposure to feline immunodeficiency virus (FIV), feline parvovirus (FPV), feline coronavirus (FCoV), feline calicivirus (FCV), canine distemper virus (CDV), feline herpesvirus (FHV), and *Yersinia pestis*. Serologic data were analyzed for annual variation in seroconversions to assess whether these pathogens are epidemic or endemic in cougars, and to determine whether family membership, age, sex, or location influence risk of exposure. FIV and FPV were clearly endemic in the studied populations, whereas exposure to FCoV, FCV, CDV, and *Y. pestis* was more sporadic. No evidence was found for FHV. Age was the most consistent predictor of increased exposure risk, often with no other important factors emerging. Evidence for transmission within family groups was limited to FIV and FCoV, whereas some indication for host sex affecting exposure probability was found for FIV and *Y. pestis*. Overall, cougar populations exhibited few differences in terms of pathogen presence and prevalence, suggesting the presence of similar risk factors throughout the study region.

**Key words:** Canine distemper virus, feline calicivirus, feline coronavirus, feline herpesvirus, feline immunodeficiency virus, feline parvovirus, feline pathogens, *Puma concolor*, *Yersinia pestis*.

### INTRODUCTION

Infectious diseases have received particular attention in the context of large carnivores, a group that includes many species of conservation concern and that has provided some of the best-documented examples of disease-induced die-offs (Murray et al., 1999; Funk et al., 2001). However, our understanding of disease epidemiology in wild carnivores is still very limited compared to domestic species and captive populations. More effective conservation and management of carnivores clearly requires a better understanding of the natural history of these diseases (Murray et al., 1999; Cleaveland et al., 2002; Haydon et al., 2002).

Cougars (*Puma concolor*) are the most widely distributed carnivore species in the

Americas, occupying a range that stretches from southern Alaska to Patagonia. Among the New World felines they are second in size only to the jaguar (*Panthera onca*). In North America, cougars have been extirpated from the east almost entirely, but occur in most of the western states and provinces, where numbers are thought to have increased in recent decades, likely as a consequence of regulated hunting and an increased prey base (Logan and Swenor, 2001). Recovery of western populations has also led to eastward expansions, as evidenced by individual animals recently appearing in several midwestern and eastern states where cougars had not been recorded for decades (Tischendorf and Johnson, 2003). Despite their wide distribution, possibly increasing abundance, and important ecological role,

information about pathogen dynamics in cougars is limited and virtually absent for the Rocky Mountain region.

In the present study, we use extensive serologic and genetic data collected from four cougar (*Puma concolor*) populations in the central part of the Rocky Mountains to examine patterns of variation in antibody prevalence and exposure risk for six viruses and one bacterial agent. Predictions about the occurrence and dynamics of infectious diseases can be made based on cougar life history, which is similar to that of other solitary large carnivores. Typically, male home ranges overlap with those of several females and contact among individuals is limited to territorial fights, mating, and family groups, consisting of females and their offspring (Logan and Sweaner, 2001). Low contact rates would predict that few infectious diseases are endemic in cougars, whereas family groups should offer greater opportunities for transmission. Based on these predictions, our aim was to determine: 1) which pathogens can be classified as endemic or epidemic in Rocky Mountain cougar populations; 2) whether individuals within family groups share similar serologic status for particular pathogens; and 3) which additional factors (age, sex, and population) predispose individuals to higher risk of exposure.

## METHODS

We collected 277 blood samples from 207 cougars from four study sites in Montana and Wyoming (Table 1 and Fig. 1) as part of detailed population studies (Murphy, 1998; Anderson, 2003). The largest number of samples ( $n=150$ ) came from the Northern Yellowstone ecosystem, involving two consecutive cougar studies, referred to in the following as Yellowstone I (1990–94) and Yellowstone II (1998–2004).

Kittens (<1 yr) and most yearlings (1–2 yr) were aged within one day to one month based on denning behavior of females, denning dates predicted from male–female associations, or like adults ( $\geq 2$  yr; estimated to the nearest year), based on morphologic characteristics such as pelage and tooth characteristics (Ash-

man et al., 1983). Among our study sites, sport hunting was the primary source of cougar mortality in the Garnet and Snowy Range, whereas hunting was largely absent in Yellowstone and the Teton populations. None of the sampled cougar populations exhibited conspicuous levels of disease-related or unexplained mortality during the course of the study.

Six viruses and one bacteria, which are considered important in wild and domestic felines (Murray et al., 1999; Packer et al., 1999), were included in this study. Polymerase chain reaction (PCR) using DNA extracted from blood samples was used to test for feline immunodeficiency virus (FIV) infection (Biek et al., 2003). Serologic testing for feline parvovirus (FPV), feline coronavirus (FCoV), feline calicivirus (FCV), canine distemper virus (CDV), and feline herpesvirus (FHV) was performed at the Washington Disease Diagnostic Lab (WADDL) in Pullman, Washington, using assays and criteria from Biek et al. (2002) or at Tufts or Cornell University using equivalent assays. For all viruses, labs yielded similar proportions of positive cases as indicated by a chi-square analysis ( $P>0.304$ ) and results were pooled for further analysis. All tests for *Yersinia pestis* were performed at the Wyoming State Diagnostic Laboratory in Laramie, Wyoming as described (Biek et al., 2002).

Because we employed mainly serologic assays, it is most appropriate to interpret positive results as evidence of exposure rather than of past or current infection. The only exception to this was FIV, for which a PCR assay was used that provided evidence for current infection. For consistency however, we use the terms “exposure” throughout this manuscript, even when referring to FIV.

Temporal variation in exposure patterns was examined based on five years of data from the Snowy Range and eleven years in Yellowstone. Because the presence of antibodies in older individuals by itself provides little information about the actual time of exposure, this analysis focused on recent seroconversions only. For each year (1 July–30 June), we estimated the proportion of susceptible individuals that had recently seroconverted and tested for significant deviations in the proportion of new cases among years. Susceptible individuals were defined as those that were either born within the last two years or that had been seronegative for a given pathogen at a previous capture (usually 1–2 yr prior). This analysis was conducted for all pathogens except FIV, which causes chronic infections and is endemic in North American cougars in most areas (Carpenter et al., 1996).

TABLE 1. Factors considered for risk of pathogen exposure. For all pathogens other than FIV, the candidate models also contained FIV (infected/uninfected) as a factor. See Biek et al. (2006b) for full details on candidate models and model selection results.

Factor	Number of categories	Categories	Model notation
Age	3	Kittens: 1–12 mo, yearlings: 13–24 mo, adults: >24 mo	<i>Age</i> (3)
	continuous	Age in years as individual covariate	<i>Age</i> (cov)
Sex	2	male, female	<i>Sex</i>
Population	5	Garnet Range, Yellowstone I, Yellowstone II, Teton Range, Snowy Range,	<i>Pop</i> (5)
	4	Garnet Range, Yellowstone, Teton Range, Snowy Range	<i>Pop</i> (4)

Potential effects of family groups on exposure were examined in two ways. First, we looked at possible transmission between mothers and offspring by testing whether seropositive mothers were more likely to have at least one seropositive offspring in their litters ( $n=44$  recorded litters). Over 90% of litters were sampled when kittens were  $\geq 4$  months so that maternal antibodies were unlikely to be present, based on data from domestic cats (Ueland and Nesse, 1992; MacDonald et al., 2004). Second, we used a binomial distribution to assess correlation in serologic status among littermates by comparing the observed distribution of seropositive offspring in litters with more than one kitten to an expected distribution based on combined prevalence of seropositive kittens and yearlings.

Differences in serologic status among statistical groups (years, family groups) were evaluated using a chi-square or Fisher's exact test ( $P < 0.05$ ) with Bonferroni correction for multiple tests. In cases where the number of groups became too large for the exact calculation, or when cells in contingency tables had expected numbers  $< 5$ , Monte Carlo approximations were employed.

Logistic regression and model selection based on sample size adjusted Akaike's Information Criterion ( $AIC_C$ ; Burnham and Anderson, 2002; Johnson and Omland, 2004) were used to identify factors that best explained differences in exposure probability (Table 2). Support for a particular factor was determined based on its Akaike weight,  $w_i$  (Burnham and Anderson, 2002), which ranges from zero (no support) to one (maximum support relative to other factors considered). Where more than one factor received considerable support (here defined as  $w_i > 0.4$ ),

parameter estimates and variances were derived through model averaging (Burnham and Anderson, 2002), based on all appropriate models within four  $AIC_C$  units of the best model. Full details regarding the candidate set of models and the model selection results can be found in a complimentary paper, focusing on the effects of FIV on cougar fitness and pathogen susceptibility (Biek et al., 2006b).

## RESULTS

Positive serologic or PCR results were found for all selected pathogens except FHV; antibodies to FHV were not detected in any of 158 tested individuals (Fig. 2). Results indicated that all four cougar populations had been exposed to FIV, FPV, and FCoV. Prevalence was consistently high for FPV (antibody prevalence, 58–69%) and FIV (% positive by PCR, 19–50%); all other pathogens had an antibody prevalence of  $\leq 28\%$ . We found no seropositive cases of FCV in the Snowy Range, CDV in the Teton Range, or *Y. pestis* in the Garnet and Teton Ranges. Given low antibody prevalence for these pathogens (average: 9–17%), nondetection due to sampling error is a possibility for small samples ( $n < 20$  for Garnet and Teton Range) but probably does not explain why we did not find antibodies to FCV in the more extensively sampled Snowy Range ( $n=58$ ).

Antibody prevalence data do not provide information to determine whether

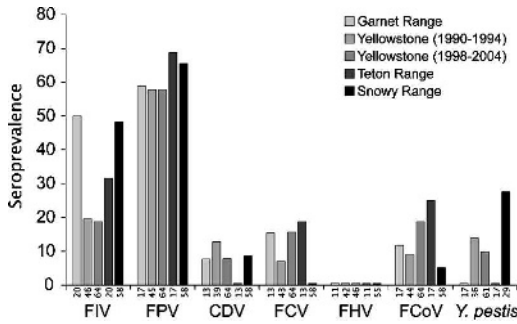


FIGURE 1. Cougar seroprevalences for six viral and one bacterial pathogen in five studies conducted at four Rocky Mountain locations. FIV = feline immunodeficiency virus, FPV = feline parvovirus, CDV = canine distemper virus, FCV = feline calicivirus, FHV = feline herpesvirus, FCoV = feline coronavirus, *Y. pestis* = *Yersinia pestis*. Numbers of individuals tested are shown below bars. Seroprevalence of zero is shown as 0.5% bars. Garnet Range: 46°40′–47°01′N, 112°57′–113°20′W; Yellowstone: 44°88′–45°45′N, 110°13′–110°90′W; Teton Range: 44°36′–43°77′N, 110°82′–111°17′W; Snowy Range: 40°46′–41°37′N, 105°50′–106°37′W.

cougars came into contact with a pathogen at a constant basis or whether contact was sporadic. For two populations with long-term data, Yellowstone and Snowy Range, we therefore estimated the annual proportion of susceptibles that seroconverted.

Seroconversions for FPV were observed in most years; however, the proportion of new cases tended to decrease following years of high incidence (Fig. 2a). New cases in most years were also characteristic for FCoV in Yellowstone, but not for the Snowy Range, where seroconversions were only detected in one out of six years (Fig. 2b). Exposure to CDV, FCV, and *Y. pestis* was sporadic, and noticeable only for short periods of time (Fig. 3a–c). Apart from FCV in Yellowstone, differences in the number of seroconversions were not significant among years after correcting for multiple tests (all  $P > 0.123$ ).

Among mothers and their offspring, antibody test results for FCoV were significantly correlated ( $P < 0.001$ ). All FIV-infected kittens were offspring of infected mothers ( $P < 0.001$ ). For all remaining pathogens, there was no correla-

tion between serologic status of mothers and offspring (all  $P > 0.091$ ). Antibody test results for littermates were not significantly correlated for any of the pathogens.

All best models for explaining exposure risk contained “age” as a factor, either in the form of age as a continuous variable (FIV, CDV, FCV, *Y. pestis*; Table 2) or as age groups (FPV, FCoV; Table 3). Differences in exposure risk among populations were supported for FIV (Table 2) and FCoV (Table 3). In the case of FIV, a strong difference for males and females was apparent in the Yellowstone population, where exposure probability rose quickly for males but much less so for females (Fig. 4b). In comparison, exposure probability increased in a much more similar fashion for both sexes in other populations (Fig. 4a, c, d). There was also some indication for differences in exposure risk between males and females for *Y. pestis* but model support for this effect was weak ( $w_i = 0.42$ , Table 2).

## DISCUSSION

High FPV prevalence, also reported from cougar populations in California and Florida (Roelke et al., 1993; Paul-Murphy et al., 1994), likely reflects the virus’ transmission mode. This virus remains infectious in the environment for months and exposure occurs through contact with feces of virus-shedding individuals (Barker and Parrish, 2001). Indirect transmission can therefore account for high levels of exposure even in a solitary species. Observed seroconversions for FPV in most years (Fig. 2a) further support that this virus is endemic in cougar populations. The fluctuations in the frequency of new cases are thereby suggestive of a cyclic pattern of FPV exposure, possibly associated with the changing proportion of susceptibles over time (Packer et al., 1999).

Despite its lability in the environment, FIV can reach higher prevalence levels by causing persistent infections and by ex-

TABLE 2. Model selection results and parameter estimates of pathogen exposure probability in cougars. Shown are four pathogens for which probability of exposure increased continuously with age. Only factors supported by Akaike weights ( $w_i$ )  $>0.4$  are shown. Also shown are the best model and its  $AIC_C$  difference from the next best model ( $\Delta_i$  next). See Methods for further details and Table 1 for factors included in candidate models.

Pathogen	Factor	$w_i$	Best model	$\Delta_i$ next	Odds ratio of exposure probability per year of age (95% CI)
FIV <sup>a</sup>	Age	1.00	$Age(cov) \times Pop(4) \times Sex$	3.02	GR: Males 1.91 (0.36–10.03), Females 2.46 (0.91–6.66) <sup>b</sup>
	Population	0.99			YE: Males 8.04 (1.99–32.51), Females 1.32 (1.07–1.63)
	Sex	0.74			TR: Males 1.45 (0.78–2.68), Females 1.42 (0.87–2.33) SR: Males 1.62 (0.84–3.13), Females 1.86 (1.14–3.05)
CDV	Age	0.99	$Age(cov)$	1.49	1.36 (1.15–1.60)
FCV	Age	0.98	$Age(cov)$	1.95	1.24 (1.07–1.43)
<i>Y. pestis</i>	Age	0.99	$Age(3)$	0.75	Males 0.88 (0.43–1.76), Females 1.16 (0.98–1.38)
	Sex	0.42			

<sup>a</sup> Because PCR assay was used for detection of FIV, exposure in this case represents actual infection.

<sup>b</sup> GR = Garnet Range, YE = Yellowstone, TR = Teton Range, SR = Snowy Range.

exploiting the close association of mothers and kittens for frequent vertical transmission. All other pathogens evaluated in this study cause acute infections, have short infectious periods outside the host (hours or days), and require direct or close contact for transmission. Their lower seroprevalence might thus reflect the relative infrequency of cougar-to-cougar contact. Although FCoV appeared to be endemic in Yellowstone, based on frequent seroconversions (Fig. 2b), antibodies were observed too infrequently to be considered FCoV endemic in the Snowy Range. The dynamics of FCoV infections could be complicated by the occasional occurrence of persistently infected individuals (Kennedy et al., 2001), which would provide a mechanism for the virus to persist independently of the number of susceptibles.

The sporadic nature of CDV, FCV, and *Y. pestis* in these populations suggests that these pathogens are either maintained in other host species or are able to persist as a metapopulation, where the pathogen might be locally absent from some cougar populations but not from all simultaneous-

ly (Bolker and Grenfell, 1996). The latter could be the case for FCV, which occurred in all populations but the Snowy Range and for which the number of seroconversions varied significantly among years in Yellowstone ( $P=0.001$ ). Persistence in other species is most probable for CDV, which infects a large range of carnivore species (Munson, 2001), and for *Y. pestis*, which is primarily maintained in rodent populations (Gasper and Watson, 2001). For both, transmission from cougar to cougar should be rare. The increased incidence observed in certain years thus likely reflects higher transmission from an unknown reservoir host species, resulting in simultaneous exposure of multiple cougars. For Yellowstone, the proposed temporal fluctuations in incidence of CDV and *Y. pestis* fit observations based on serologic data in coyotes (*Canis latrans*), for which seroprevalence fell from 100% in 1989 to 33% in 1993 for CDV and from 57% in 1991 to 0% in 1993 for *Y. pestis* (Gese et al., 1997).

We found a positive correlation between exposure to FCoV in mothers and

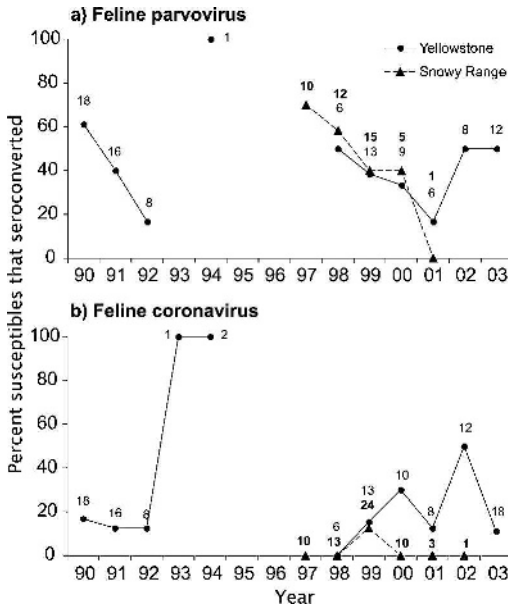


FIGURE 2. Annual proportion of susceptible cougars at two populations that seroconverted for (a) feline parvovirus and (b) feline coronavirus. Numbers indicate total number of susceptibles (newborns or previously seronegative) tested in that year. Yellowstone numbers are shown in regular font, Snowy Range numbers in bold. The year given refers to the start of the sampling season in the fall, sampling continued into spring of the subsequent year (i.e., “90” refers to season 1990–91).

in their offspring. This could be due to frequent transmission among individuals within family groups or due to spatial autocorrelation (a higher risk resulting from time spent in the same area). The short infective period for coronavirus outside the host makes exposure from direct contact among individuals more likely. For FIV, all positive kittens were associated with infected mothers, consistent with infection through vertical transmission (Biek et al., 2003).

That serologic status of littermates was uncorrelated for all pathogens tested was surprising, given that littermates lived in close proximity for several months and thus likely were nonindependent in their history of pathogen exposure. In addition to sampling issues (kittens from the same litter were not always sampled at the same time), occasional persistence of maternal

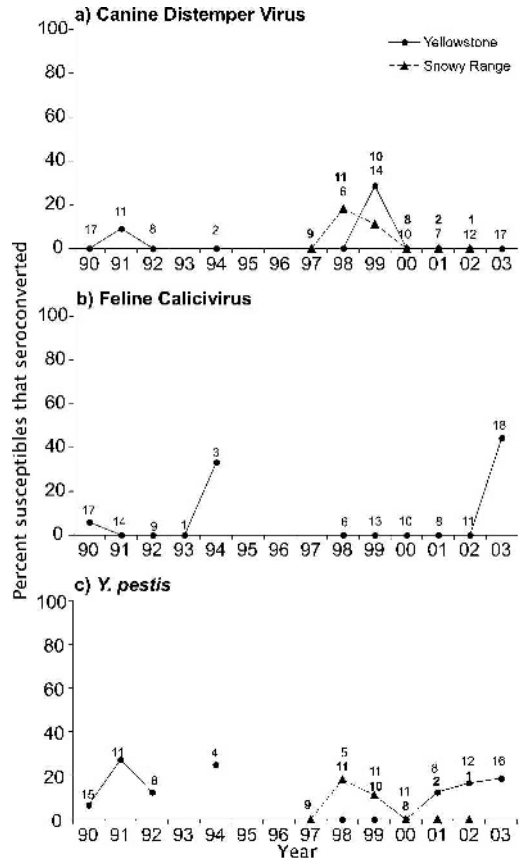


FIGURE 3. Annual proportion of susceptible cougars at two populations that seroconverted for (a) canine distemper virus, (b) feline calicivirus, and (c) *Y.pestis*. See Fig. 2 for further descriptions.

antibodies might have obscured such relationships.

Much of the variation in exposure among cougars could be explained by age (Table 2, Table 3). Age increases the probability of experiencing a local outbreak in the case of epidemic pathogens or might correlate with entering new life stages that are associated with higher exposure risk due to physiologic or behavioral changes. For example, FPV exposure probability more than doubled from about 0.42 for kittens and yearlings to 0.88 for adults (Table 3).

We found a much higher predisposition of males to FIV exposure in Yellowstone (Fig. 4b) but this strong bias was not

TABLE 3. Model selection results and parameter estimates of pathogen exposure probability in cougars. Shown are two pathogens for which probability of exposure increased with age group (kitten, yearling, adult). See Table 2 for further descriptions.

Pathogen	Factor	$w_i$	Best model	$\Delta_i$ next	Exposure probability (95% CI)
FPV	Age	1.00	<i>Age(3)</i>	4.56	Kit 0.42 (0.34–0.57), Yrl 0.43 (0.28–0.58), Ad 0.88 (0.79–0.93)
FCoV	Age	0.74	<i>Age(3) + Pop(4)</i>	0.35	GR: Kit 0.04 (0.01–0.16), Yrl 0.05 (0.01–0.19), Ad 0.08 (0.02–0.27) <sup>a</sup>
	Population	0.61			YE: Kit 0.11 (0.04–0.23), Yrl 0.12 (0.05–0.26), Ad 0.20 (0.09–0.37) TR: Kit 0.08 (0.02–0.28), Yrl 0.09 (0.02–0.32), Ad 0.15 (0.04–0.41) SR: Kit 0.15 (0.03–0.46), Yrl 0.17 (0.04–0.48), Ad 0.27 (0.10–0.54)

<sup>a</sup> GR = Garnet Range, YE = Yellowstone, TR = Teton Range, SR = Snowy Range.

repeated in other populations (Fig. 4a, c, d; see below). Among the remaining pathogens, only *Y. pestis* showed some model support for an effect of sex ( $w_1=0.42$ ; Table 2). Closer examination revealed that only two of nineteen individuals positive for plague were males, aged 1.6 and 2.0 yr. Consequently, the estimated increase in exposure probability with age only pertained to female cougars, 35% of which were plague positive as adults. Logan and Sweanor (2001) report three confirmed deaths, all females, caused by plague in a New Mexico population. Higher exposure risk in females could reflect a larger reliance on small prey compared to males, but we are unaware of any data to support or refute this.

Support for an effect of location on exposure risk was only found for FCoV and FIV. For the former, the evidence was in fact weak, given that confidence intervals for all populations were overlapping widely (Table 3). Spatial differences were more pronounced for FIV, largely due to exposure probability in Yellowstone rising particularly fast for males but much more slowly in females (Table 2 and Fig. 4). Genetic differences in susceptibility of cougars or transmission ability among virus strains could account for this observation. However, both seem unlikely because cougars throughout the study

area show little genetic distinction and genetic population structure in the virus does not correspond to high and low prevalence populations (Anderson et al., 2004; Biek et al., 2006a).

Vertical transmission of FIV from females to their offspring appeared to be equally common in the Snowy Range and Yellowstone: the probability of passing on an infection to offspring did not differ between infected mothers at both locations ( $P=0.528$ ). Differences among populations must therefore be related to horizontal transmission, which is thought to occur through fighting and possibly mating (Biek et al., 2003). Whatever causes the reduced rate of horizontal transmission to females in Yellowstone appears to be a persistent effect, given consistently low FIV prevalence in Yellowstone females over 15 years. Because cougars in the Garnet and Snowy Range sites experienced much higher levels of exploitation compared to the other populations, it is possible that the more rapid turnover of territorial adults is associated with higher rates of FIV transmission. A similar phenomenon has recently been documented in another carnivore–pathogen system (Donnelly et al., 2003) and could be related to higher intraspecific conflict associated with the constant spatial reorganization of home ranges in exploited populations.



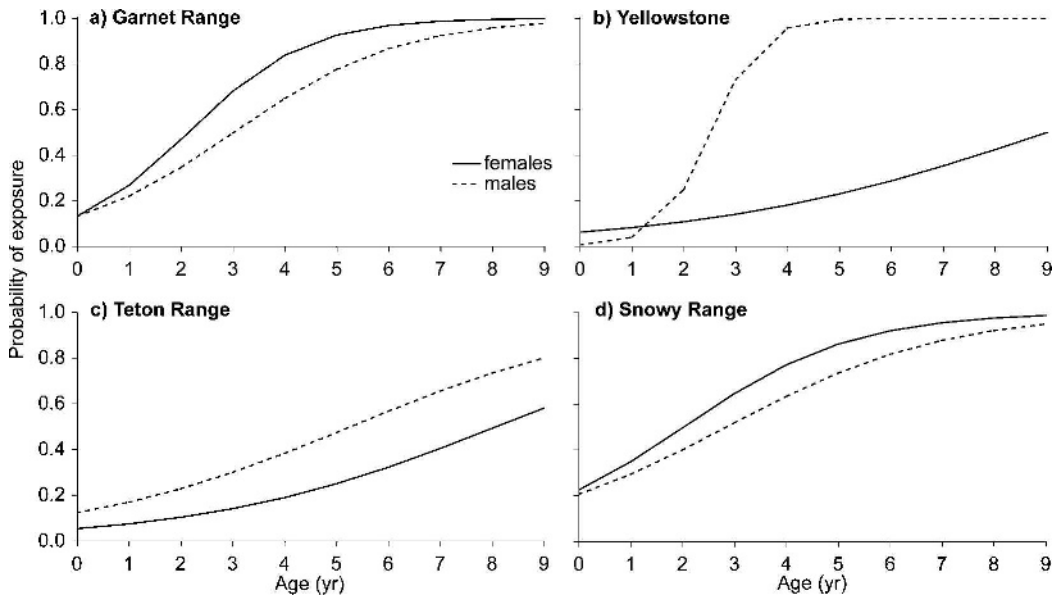


FIGURE 4. Estimated FIV exposure probability in relation to age for male and female cougars in four populations: (a) Garnet Range, (b) Yellowstone, (c) Teton Range, and (d) Snowy Range. See Table 2 for parameter estimates and measures of uncertainty.

Consistent with our predictions for a solitary carnivore, we identified only two pathogens, FIV and FPV, as endemic in cougars. In both cases, high antibody prevalence and continuous presence are probably related to prolonged opportunities for transmission. One of few studies to date that has examined long-term dynamics of infectious diseases in a large carnivore was done in a gregarious species, the African lion (Packer et al., 1999). Discrete epidemics for FPV, FCoV, CDV, and FCV at intervals of 4–12 yr characterized virus dynamics in Serengeti lions, in contrast to the shorter or nonexistent interepidemic intervals our data showed for these pathogens. Higher host contact rates in the group-living lion could be responsible for the more pronounced epidemics, but more comparative studies are needed to separate effects of social organization from other factors, including variability among pathogen strains and different ecological and climatic conditions.

Antibody prevalence was surprisingly similar between cougar populations. This lack of spatial heterogeneity was remark-

able not only because it occurred over distances of several hundred kilometers but also because the sampled populations differed in significant parameters such as presence/absence of cougar hunting. It suggests that pathogen presence and transmission in these cougar populations might be driven by certain unifying ecological factors. One such factor could be a comparable suite of mammalian prey and sympatric carnivore species present at each of our four sampling sites. For pathogens shared among multiple mammalian host species, such as CDV and *Y. pestis*, this could translate into similar levels of exposure in cougars. Considering their low density relative to other potential hosts, cougar numbers probably have little effect on the dynamics of these pathogens which can cause significant mortality in wild carnivore populations (Williams et al., 1994; Roelke Parker et al., 1996).

Aside from FIV, for which there is compelling evidence of a cougar-specific strain (Troyer et al., 2005), it is unclear whether the remaining feline viruses found in Rocky Mountain cougars (FPV,

FCoV, and FCV) are shared with other species. Exposure to all three viruses has been documented in the bobcat (*Lynx rufus*) and the Canada lynx (*L. canadensis*) (Wassmer et al., 1988; Biek et al., 2002; Riley et al., 2004), the other two wild cat species present in our study area. Because FPV, a virus occasionally associated with considerable mortality (Wassmer et al., 1988), is evidently endemic in cougars, the latter could conceivably act as a source of transmission to other species. This could be relevant for species of conservation concern, such as the Canada lynx, for which recovery efforts in the Rocky Mountain region are currently underway.

#### ACKNOWLEDGMENTS

Main funding for this research was provided by Morris Animal Foundation grant D01ZO-111. Additional funding came from the Fanwood Foundation, the Wilburforce Foundation, the J. Murdock Charitable Trust, National Fish and Wildlife Federation, Richard King Mellon Foundation, Charles Engelhard Foundation, Wildlife Conservation Society, Michael Cline Foundation, M. Comegys, The Laura Moore Cunningham Foundation, The Summerlee Foundation, Tim and Karen Hixon Foundation, The National Geographic Society, L. Westbrook, The Argosy Foundation, J. Hagenbuch, M. and A. Manship, The Bay Foundation, Montana Fish Wildlife and Parks, US Fish and Wildlife Service, and Yellowstone National Park. We thank the many people involved in cougar capturing and sampling. All sampling followed standard animal care and handling protocols. F. Allendorf, E. Crone, J. Graham, M. Kauffman, L. S. Mills, D. Pletscher, and two anonymous reviewers provided helpful comments or suggestions on an earlier version of this manuscript.

#### LITERATURE CITED

- ANDERSON, C. R., JR. 2003. Cougar ecology, management, and population genetics in Wyoming. PhD Dissertation, University of Wyoming, Laramie, Wyoming, 124 pp.
- , F. G. LINDZEY, AND D. B. McDONALD. 2004. Genetic structure of cougar populations across the Wyoming Basin: metapopulation or megapopulation. *Journal of Mammalogy* 85: 1207–1214.
- ASHMAN, D. L., G. C. CHRISTENSEN, M. L. HESS, G. K. TSUKAMOTO, AND M. S. WICKERSHAM. 1983. The mountain lion in Nevada. Nevada Department of Wildlife, W-048-15, Job 01, Study R-V. 83 pp.
- BARKER, I. K., AND C. R. PARRISH. 2001. Parvovirus infections. *In* Infectious diseases of wild mammals, E. S. Williams and I. K. Barker (eds.). Iowa State University Press, Ames, Iowa, pp. 131–146.
- BIEK, R., R. L. ZARNKE, C. GILLIN, M. WILD, J. R. SQUIRES, AND M. POSS. 2002. Serologic survey for viral and bacterial infections in western populations of Canada lynx (*Lynx canadensis*). *Journal of Wildlife Diseases* 38: 840–845.
- , A. G. RODRIGO, D. HOLLEY, A. DRUMMOND, C. R. ANDERSON, JR., H. A. ROSS, AND M. POSS. 2003. Epidemiology, genetic diversity, and evolution of endemic feline immunodeficiency virus in a population of wild cougars. *Journal of Virology* 77: 9578–9589.
- , A. J. DRUMMOND, AND M. POSS. 2006a. A virus reveals population structure and recent demographic history of its carnivore host. *Science* 311: 538–541.
- , T. K. RUTH, K. M. MURPHY, C. R. ANDERSON, JR., AND M. POSS. 2006b. Examining effects of persistent retrovirus infection on fitness and pathogen susceptibility in a natural feline host. *Canadian Journal of Zoology* 84: 365–373.
- BOLKER, B. M., AND B. T. GRENFELL. 1996. Impact of vaccination on the spatial correlation and persistence of measles dynamics. *Proceedings of the National Academy of Science USA* 93: 12648–12653.
- BURNHAM, K. P., AND D. R. ANDERSON. 2002. Model selection and multimodel inference. 2nd Edition. Springer, New York, New York, 488 pp.
- CARPENTER, M. A., E. W. BROWN, M. CULVER, W. E. JOHNSON, J. PECON-SLATTERY, D. BROUSSET, AND S. J. O'BRIEN. 1996. Genetic and phylogenetic divergence of feline immunodeficiency virus in the puma (*Puma concolor*). *Journal of Virology* 70: 6682–6693.
- CLEAVELAND, S., G. R. HESS, A. P. DOBSON, M. K. LAURENSEN, H. MCCALLUM, M. G. ROBERTS, AND R. WOODROFFE. 2002. The role of pathogens in biological conservation. *In* The ecology of wildlife diseases, P. J. Hudson, A. Rizzoli, B. T. Grenfell, H. Heesterbeek, and A. P. Dobson (eds.). Oxford University Press, Oxford, UK, pp. 139–150.
- DONNELLY, C. A., R. WOODROFFE, D. R. COX, J. BOURNE, G. GETTINBY, A. M. LE FEVRE, J. P. MCINERNEY, AND W. I. MORRISON. 2003. Impact of localized badger culling on tuberculosis incidence in British cattle. *Nature* 426: 834–837.
- 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. W. MacDonald and R. K. Wayne (eds.). Cambridge University Press, Cambridge, UK, pp. 443–466.

- GASPER, P. W., AND R. P. WATSON. 2001. Plague and Yersiniosis. *In* Infectious diseases of wild mammals, E. S. Williams and I. K. Barker (eds.). Iowa State University Press, Ames, Iowa, pp. 313–329.
- GESE, E. M., R. D. SCHULTZ, M. R. JOHNSON, E. S. WILLIAMS, R. L. CRABTREE, AND R. L. RUFF. 1997. Serological survey for diseases in free-ranging coyotes (*Canis latrans*) in Yellowstone National Park, Wyoming. *Journal of Wildlife Diseases* 33: 47–56.
- HAYDON, D. T., M. K. LAURENSEN, AND Z. C. SILLERO. 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.
- JOHNSON, J. B., AND J. S. OMLAND. 2004. Model selection in ecology and evolution. *Trends in Ecology and Evolution* 19: 101–108.
- KENNEDY, M., N. BOEDEKER, P. GIBBS, AND S. KANIA. 2001. Deletions in the 7a ORF of feline coronavirus associated with an epidemic of feline infectious peritonitis. *Veterinary Microbiology* 81: 227–234.
- LOGAN, K. A., AND L. L. SWEANOR. 2001. Desert puma: evolutionary ecology and conservation of an enduring carnivore. Island Press, Washington, D.C., 390 pp.
- MACDONALD, K., J. K. LEVY, S. J. TUCKER, AND P. C. CRAWFORD. 2004. Effects of passive transfer of immunity on results of diagnostic tests for antibodies against feline immunodeficiency virus in kittens born to vaccinated queens. *Journal of the American Veterinary Medical Association* 225: 1554–1557.
- MUNSON, L. 2001. Feline morbillivirus infection. *In* Infectious diseases of wild mammals, E. S. Williams and I. K. Barker (eds.). Iowa State University Press, Ames, Iowa, pp. 59–62.
- MURPHY, K. M. 1998. The ecology of the cougar (*Puma concolor*) in the northern Yellowstone ecosystem: Interactions with prey, bears, and humans. PhD Dissertation, University of Idaho, Moscow, Idaho, 147 pp.
- 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.
- 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.
- PAUL-MURPHY, J., T. WORK, D. HUNTER, E. MCFIE, AND D. FJELLINE. 1994. Serologic survey and serum biochemical reference ranges of the free-ranging mountain lion (*Felis concolor*) in California. *Journal of Wildlife Diseases* 30: 205–215.
- RILEY, S. P., J. FOLEY, AND B. CHOMEL. 2004. Exposure to feline and canine pathogens in bobcats and gray foxes in urban and rural zones of a national park in California. *Journal of Wildlife Diseases* 40: 11–22.
- ROELKE, M. E., D. J. FORRESTER, E. R. JACOBSON, G. V. KOLLIAS, F. W. SCOTT, M. C. BARR, J. F. EVERMANN, AND E. C. PIRTLE. 1993. Seroprevalence of infectious disease agents in free-ranging Florida panthers (*Felis concolor coryi*). *Journal of Wildlife Diseases* 29: 36–49.
- ROELKE PARKER, M. E., L. MUNSON, C. PACKER, R. KOCK, S. CLEAVELAND, M. CARPENTER, S. J. O'BRIEN, A. POSPISCHIL, L. R. HOFMANN, H. LUTZ, L. M. MWAMENGELE GEORGE, 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.
- TISCHENDORF, J., AND K. JOHNSON. 2003. Prairie panthers and midwest mountain lions. *Cat News* 39: 8–9.
- TROYER, J. L., J. PECON-SLATTERY, M. E. ROELKE, W. JOHNSON, S. VANDEWOUDE, N. VASQUEZ-SALAT, M. BROWN, L. FRANK, R. WOODROFFE, C. WINTERBACH, H. WINTERBACH, G. HEMSON, M. BUSH, K. A. ALEXANDER, E. REVILLA, AND S. J. O'BRIEN. 2005. Seroprevalence and genomic divergence of circulating strains of feline immunodeficiency virus among Felidae and Hyaenidae species. *Journal of Virology* 79: 8282–8294.
- UELAND, K., AND L. L. NESSE. 1992. No evidence of vertical transmission of naturally acquired feline immunodeficiency virus infection. *Veterinary Immunology and Immunopathology* 33: 301–308.
- 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* 33: 159–228.
- WILLIAMS, E. S., K. MILLS, D. R. KWIATKOWSKI, E. T. THORNE, AND A. BOERGER-FIELDS. 1994. Plague in a black-footed ferret (*Mustela nigripes*). *Journal of Wildlife Diseases* 30: 581–585.

Received for publication 14 July 2005.