

University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

USGS Northern Prairie Wildlife Research Center

US Geological Survey

2011

Infectious Diseases in Yellowstone's Canid Community

Emily S. Almberg

The Pennsylvania State University

Paul C. Cross

USGS Northern Rocky Mountain Science Center

L. David Mech

University of Missouri

Doug W. Smith

Yellowstone National Park

Jennifer W. Sheldon

Yellowstone Ecological Research Center

See next page for additional authors

Follow this and additional works at: <https://digitalcommons.unl.edu/usgsnpwrc>



Part of the [Animal Sciences Commons](#), [Behavior and Ethology Commons](#), [Biodiversity Commons](#), [Environmental Policy Commons](#), [Recreation, Parks and Tourism Administration Commons](#), and the [Terrestrial and Aquatic Ecology Commons](#)

Almberg, Emily S.; Cross, Paul C.; Mech, L. David; Smith, Doug W.; Sheldon, Jennifer W.; and Crabtree, Robert L., "Infectious Diseases in Yellowstone's Canid Community" (2011). *USGS Northern Prairie Wildlife Research Center*. 391.

<https://digitalcommons.unl.edu/usgsnpwrc/391>

This Article is brought to you for free and open access by the US Geological Survey at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in USGS Northern Prairie Wildlife Research Center by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

Authors

Emily S. Almberg, Paul C. Cross, L. David Mech, Doug W. Smith, Jennifer W. Sheldon, and Robert L. Crabtree



Infectious Diseases in Yellowstone's Canid Community

*Emily S. AlMBERG, Paul C. Cross, L. David Mech, Doug W. Smith,
Jennifer W. Sheldon, and Robert L. Crabtree*

EACH SUMMER Yellowstone Wolf Project staff visit den sites to monitor the success of wolf reproduction and pup rearing behavior. For the purposes of wolf monitoring, Yellowstone National Park (YNP) is divided into two study areas, the northern range and the interior, each distinguished by their ecological and physiological differences. The 1,000 square kilometer northern range, characterized by lower elevations (1,500–2,200 m), serves as prime winter habitat for ungulates and supports a higher density of wolves than the interior (20–99 wolves/1,000 km² versus 2–11 wolves/1,000 km²). The interior of the park encompasses 7,991 square kilometers, is higher in elevation, receives higher annual snowfall, and generally supports lower densities of wolves and ungulates.

During the Yellowstone Wolf Project's 2005 observations on the northern range, researchers noticed that some wolf pups were disappearing and those that remained were unusually listless. The Slough Creek pups, at first numbering 18, dwindled to three survivors. Similar findings were mirrored at other den sites across the northern range. When annual den surveys were conducted in late July, all that remained were scattered piles of bones and fur. Coyotes suffered similar setbacks in 2005, with many

of the survivors exhibiting neurological shakes and tremors. The park's canids had been affected by something, but what?

Prompted by what seemed to be a disease outbreak, the Yellowstone Wolf Project, the Yellowstone Ecological Research Center (YERC), and the University of Minnesota decided to take several collaborative approaches toward improving our understanding of the presence and role of infectious disease in Yellowstone's canid community. Several serological studies have been conducted in the past among the park's coyotes (Gese et al. 1997) and cougars (Biek 2006), providing a helpful foundation on which to build and compare. A serological survey was conducted, using serum samples collected during routine wolf and coyote captures over a period of 18 years (AlMBERG et al. 2009). Simulation models were used to explore the dynamics of canine distemper virus (AlMBERG et al. 2010)—one of the more prominent pathogens in terms of its effects on its hosts—and several long-term pathogen surveillance projects were initiated which are intended to someday provide a foundation for more advanced genetic-based analyses of pathogen dynamics. Since these initial efforts, the group has also expanded the research to include a study of sarcoptic mange, which began affecting wolves and coyotes in YNP in 2006 and 2007.

Serological survey

Serum is the component of blood that contains antibodies, which are protein molecules that recognize foreign objects in the body and flag them for destruction. Following exposure to a particular pathogen, the body produces millions of antibodies specific to that pathogen. In many cases, these antibodies circulate within the body for long periods and are detectable through laboratory assays as evidence of exposure to a specific pathogen. Although the timing of a previous exposure cannot be determined from a serological assay, with sufficient samples, particularly from young animals collected over time, it is often possible to obtain a useful picture of how a particular pathogen has been circulating in the wildlife population.

Since wolf reintroduction in 1995 and as part of a long-term ecological study of coyotes, the Yellowstone Wolf Project and YERC have collected serum from wolves and coyotes handled during routine capture and radio-collaring efforts. As a starting point, we sought to use these long-term

serological data to describe the spatial, temporal, and demographic patterns of wolf and coyote exposure to several common canid pathogens (table 1). We screened for exposure to canine parvovirus (CPV), canine adenovirus (CAV-1), canine distemper virus (CDV), and canine herpesvirus (CHV), all of which can cause morbidity and mortality in canids. Among wolves, we also screened for exposure to *Neospora caninum*, a protozoan parasite whose life cycle includes canids as the definitive hosts where sexual reproduction takes place, and ungulates as intermediate hosts where the parasite has been implicated in spontaneous abortions.

Specifically, we were interested in whether these pathogens were endemic (constant and relatively stable prevalence over time) or epidemic (periods of little or no prevalence punctuated by outbreaks) within YNP's canid populations. Among wolves, for which we had samples from both the northern range and the park interior, we sought to determine whether patterns of exposure varied by region in relation to local canid densities. Among coyotes, which were only

Table 1. Epidemiological characteristics of selected canid pathogens

Pathogen	Transmission	Symptoms	Course of infection	Mortality pattern*	Reference
Canine parvovirus (CPV)	Direct contact with oral and nasal exudates, and indirect fecal-oral contact	Immune depression, anemia, vomiting, diarrhea, and dehydration	Mild to acute gastrointestinal inflammation, followed by clearance or occasional carrier status	In unvaccinated populations, mortality is greatest in pups <1 year	Barker et al. 2001
Canine distemper virus (CDV)	Direct contact with respiratory exudates (aerosol)	Fever, nasal and conjunctival discharges, anorexia, vomiting, diarrhea, muscle tremors, encephalitis, immunosuppression	Acute infection is followed by complete clearance or subacute/persistent infection in the central nervous system	In unvaccinated populations, mortality is greatest in pups <1 year	Greene and Appel 2006
Canine adenovirus type-1 (CAV-1)	Direct contact with nasal and conjunctival secretions, urine, or feces; indirect through contact with contaminated fomites	Immune depression, fever, apathy, anorexia, vomiting, and diarrhea. May develop bronchopneumonia, conjunctivitis, photophobia and transient corneal opacity ("blue eye")	Virus is either quickly cleared or causes acute/chronic hepatitis. Following full recovery, immunity is likely lifelong	In unvaccinated populations, mortality is greatest in pups <1 year	Woods et al. 2001
Canine herpesvirus (CHV)	Direct contact with oral, nasal, and genital secretions; transplacental	<i>Adults</i> : Mild upper respiratory infection; genital lesions; abortion <i>Neonates</i> : Lethargy, anorexia, weight loss, rhinitis, and rash	Following initial clinical/sub-clinical infections, latent infection persists for months to years and is intermittently reactivated	Fetal and neonate mortality are greatest	Greene and Carmichael 2006
<i>Neospora caninum</i> (protozoan)	Canids consuming infected wild or domestic ungulate tissues; transplacental	Most infections are likely subclinical and asymptomatic. <i>Acute disease</i> : neurological and muscular disorder (paralysis in pups), hepatic, pulmonary, and myocardial dysfunction, fever and vomiting	Following initial clinical/sub-clinical infection, infection is either chronic or subclinical and can be reactivated during periods of stress or pregnancy	While mortality is generally uncommon, pups are most susceptible	Greene 2006

*In domestic carnivores

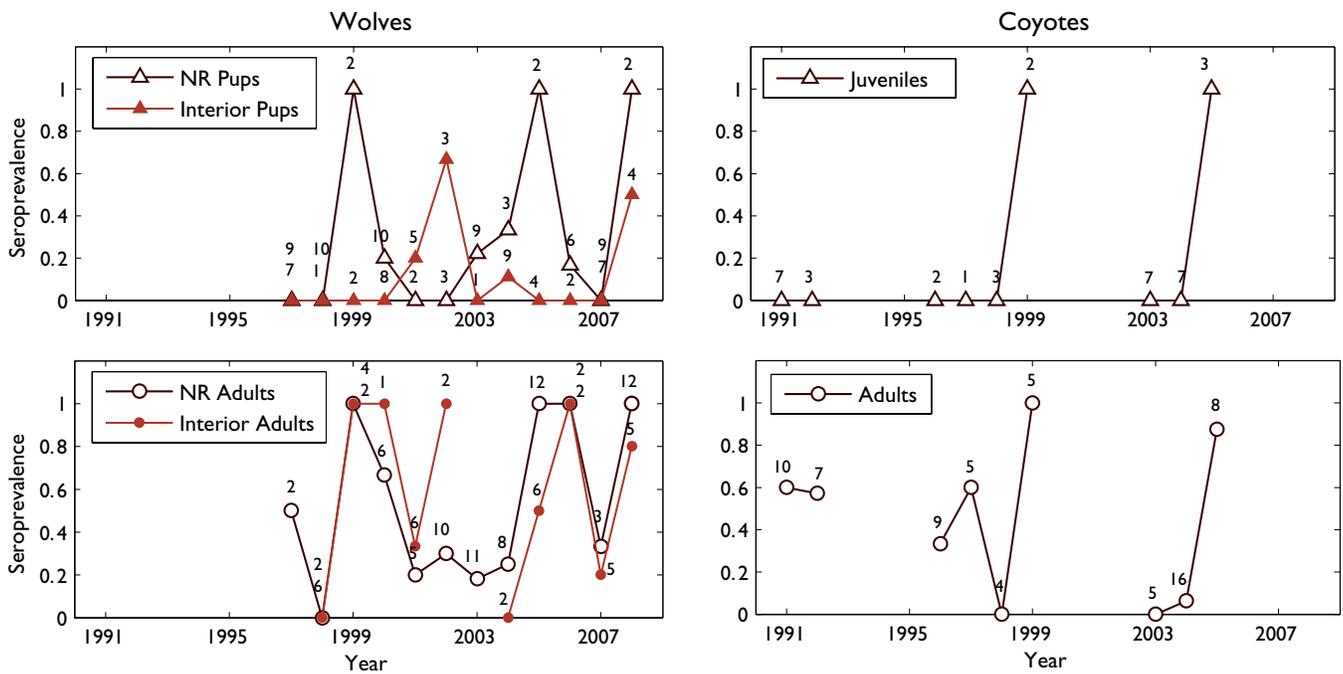


Figure 1. Annual canine distemper virus seroprevalence among wolves and coyotes in Yellowstone National Park, 1991–2008. Among wolves, data are divided by location. Coyotes were sampled only on the northern range (NR). Sample sizes are displayed above seroprevalences. Where points overlap, the top number refers to the northern range, the bottom to the interior. Small sample sizes among NR wolves in 1999, 2005, and 2008 reflect poor pup survival, which was likely the result of the CDV outbreaks. (Modified from AlMBERG et al. 2009.)

sampled on the northern range, we asked whether behavioral differences between pack residents and transients might contribute to differences in their risk of infection. We hypothesized that transients might be at greater risk of disease exposure because of their overlap in home range with multiple resident packs. We also evaluated age class as a risk factor for recent infection with CHV or *N. caninum*.

Although we did not have survival data for coyotes, we did have survival estimates for wolf pups, gathered through aerial and ground monitoring efforts from May through December. Motivated by a desire to understand whether disease had a role in the 1999, 2005, and 2008 wolf pup mortalities, we examined the relationship between pathogen exposure and wolf pup survival.

Methods

We had 262 wolf samples from 237 individuals collected from 1997 to 2008 and 110 coyote samples from 109 individuals collected from 1991–1992, 1996–1999, and 2003–2005). These sera were screened at the New York State Animal Diagnostic Center in Ithaca for antibodies to CPV, CAV-1, CDV, and CHV; due to insufficient quantities of coyote sera, only wolf samples were screened for *N. caninum*. We analyzed positive and negative serological test results using logistic, generalized-linear-mixed-models, and

candidate models were compared using Akaike's Information Criterion. This statistical approach allowed us to examine the evidence for the influence of year, spatial location, resident versus transient status (coyotes only), and age class on the probability of pathogen exposure. We also used a logistic, generalized-linear-mixed-model and model-selection procedures to evaluate the effect of year and location on wolf pup survival. We used regression analyses to examine the relationship between annual wolf pup survival and annual wolf pup seroprevalence.

Results

All wolves and 94% of both adult and juvenile coyotes tested positive for CPV, yielding no patterns of exposure with respect to year, location, age group, or resident status. Wolf exposure to CAV-1 was also high and constant (93%). However, both juvenile and adult resident coyotes had slightly greater (although non-significant) probabilities of CAV-1 exposure (juvenile seroprevalence: 23%; adult seroprevalence: 89%) than their transient counterparts (juvenile seroprevalence: 11%; adult seroprevalence: 71%).

By contrast, there was substantial temporal variation in wolf and coyote exposure to CDV (fig. 1). Young wolves and coyotes give the best picture of when various diseases are circulating because they have only been exposed for a short



Wolf pups of the Delta pack with two adults at the den site.

period. Adults, on the other hand, may have been exposed several years before capture. Exposure to CDV among wolf pups was highest in 1999, 2002, 2005, and 2008, a pattern less clearly mirrored in the adult data. Between these four outbreak years, we found evidence for a small amount of seroconversion (converting from negative to positive status) among pups (20%–33% in 2000, 2001, 2003, and 2004). In addition, both northern range pups and adults had greater, although non-significant, probabilities of exposure compared to their park interior counterparts.

Both juvenile and adult coyote seroprevalence mirrored the temporal patterns observed among northern range wolf pups; CDV seroprevalence was 100% in 1999 and 2005 among both age groups and 0% otherwise among juveniles (fig. 1; no coyote data available beyond 2005). Furthermore, adult resident coyotes were more likely to have been exposed to CDV than adult transients, although this difference was not statistically significant.

Wolf exposure to CHV was uniformly high (87%), but among coyotes, we found support for age class and resident status effects on the risk of CHV exposure. As is common for endemic pathogens, the probability of CHV exposure among coyotes significantly increased with age class (juvenile seroprevalence = 23%; young adult seroprevalence = 51%; and old adult seroprevalence = 87%). Although not statistically significant, resident coyotes had a higher probability of CHV exposure than did transients.

We found evidence suggesting that *N. caninum* exposure among wolves was influenced by age class, year, and location. Wolves' probability of exposure to *N. caninum* increased with age (old adult seroprevalence: 33%; young adult seroprevalence: 19%; and juvenile seroprevalence: 8%). There were no significant year or location effects.

Between 1995 and 2008, the Yellowstone Wolf Project annually monitored an average of 10 wolf dens, an average of 89% of reproducing packs. Our best supported models suggested that year and location were important factors influencing pup survival. Pup survival was significantly lower on the northern range than in the interior (fig. 2). The pup survival was also significantly lower on the northern range in 2005 and 2008 (13% and 10%, respectively) than in most years, and lower than average, but not significantly so, in 1999 (7%).

Annual wolf pup CDV seroprevalence was negatively correlated with annual pup survival on the northern range ($r^2 = 0.77$, $t = -5.8$, $df = 11$, $P < 0.001$), although this was not the case in the interior ($r^2 = 0.002$, $t = 0.15$, $df = 11$, $P = 0.88$). Our failure to detect a relationship between interior pup survival and CDV seroprevalence was most likely due to biases in the timing and quality of pup observations in the interior. None of the other pathogens (CPV, CAV-1, and CHV) exhibited significant temporal variation capable of explaining temporal patterns of pup survival, and annual wolf pup survival was independent of annual pup exposure to *N. caninum*.

Discussion

The (sero)prevalence of a pathogen is not always a very good indicator of its impact on its host. Deadly infections are rarely detected (or much more difficult to detect) because they kill their hosts before there is an opportunity to sample them, whereas we may frequently detect less pathogenic organisms. The consistently high levels of exposure to CPV, CAV-1, and CHV suggested that these pathogens are firmly

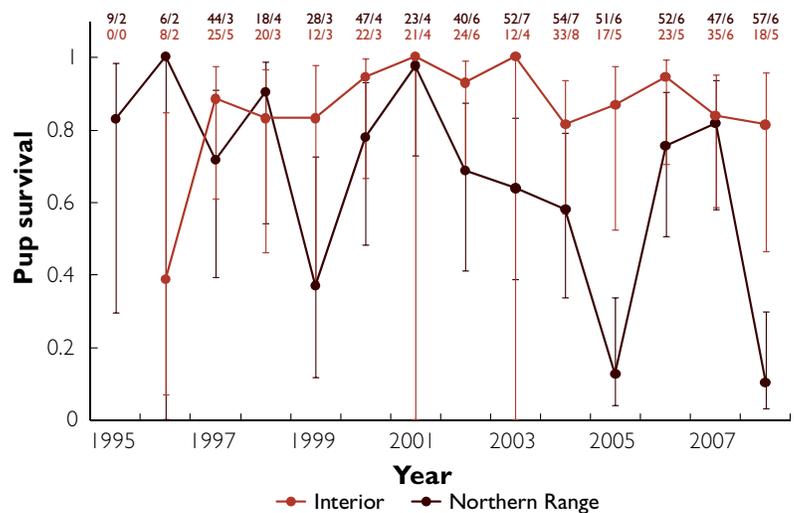


Figure 2. Annual wolf pup survival in Yellowstone National Park by location, 1995–2008. Survival = December high pup counts divided by May high pup counts at the den. Error bars represent a 95% confidence interval. Small numbers above the graph show the number of pups monitored/number of packs observed. (Modified from Almgren et al. 2009.)

established in YNP's wolf and coyote populations and that they are unlikely to be causing acute mortality in their hosts. Although this study was unable to detect mortality associated with CPV, CAV-1, and CHV, these pathogens may still cause occasional mortality among individuals during periods of nutritional stress or co-infection with other pathogens and parasites, or predispose their hosts to other forms of mortality (e.g., death during inter-pack strife). For example, CHV infections can flare up in response to stress during pregnancy, and although we do not have sufficient data on the CHV status of reproducing females, every so often we witness a pregnant female localize but then abandon her den early in the season. There are any number of possible explanations for this; however, neonatal mortality due to CHV infection would be a plausible hypothesis. Although *N. caninum* is unlikely to impact canid health, wolf exposure indicates that the parasite is circulating among canids and ungulates within the park, which may or may not be related to the parasite's dynamics among regional livestock.

Contrary to our hypothesis, resident coyotes exhibited a trend toward slightly higher risks of exposure to various pathogens than did transients. However, we also found that residents tended to be slightly older on average, and we were unable to determine whether this pattern was due to a behavior-driven difference in transmission or was simply a function of a bias in host age and hence opportunities for exposure (or even a spurious pattern driven by small sample sizes). Perhaps repeated opportunities for close contact within a pack are more important in pathogen transmission than fewer contacts distributed across a greater number of packs.

CDV proved to be the most dynamic pathogen, and in combination with previous serological surveys from YNP's cougars (Biek 2006) and coyotes (Gese et al. 1997), our data suggested that these outbreaks were synchronized among multiple carnivores in YNP over time. CDV most likely contributed to the low wolf pup survival in 1999, 2005, and 2008 on the northern range. At present, CDV appears to cause short-term population declines of relevance to state and federal agencies responsible for meeting wolf population management goals; it does not appear to jeopardize the long-term population survival of YNP wolves. The combined effects of multiple pathogens on the wolf population remains an important area of research.

Young wolves and coyotes give the best picture of when various diseases are circulating because they have only been exposed for a short period.



Transient coyotes exhibited a slightly lower risk of exposure to various pathogens.

Canine distemper virus and critical community size

The serological survey found that outbreaks of canine distemper were periodic, synchronous across wolves, coyotes, and cougars, and highly correlated with years of very low wolf pup survival. This raised questions about where and how CDV was being maintained in YNP, and how often outbreaks were likely to occur in the future. CDV is a generalist pathogen capable of infecting a wide range of carnivore species. It is considered an acute, highly immunizing (inducing life-long immunity in its hosts) pathogen, requiring large populations and high densities of hosts for its persistence. It is a close relative of human measles, for which an estimated community size of 250,000 to 500,000 is needed for the virus to persist long-term. However, unlike measles, CDV manages to persist among carnivore hosts that tend to occur at relatively low densities, live in small social groups, tend to be territorial, and are patchily distributed. Thus, we posed a series of questions pertaining to the conditions under which CDV is likely to persist within the Greater Yellowstone Ecosystem (GYE):

- (1) Given plausible estimates of group size, host survival, and spatial connectivity between packs on the landscape, can GYE wolves alone support the persistence of CDV?
- (2) What is the critical community size (the threshold population size needed for a pathogen to persist long-term) of a plausible, alternate reservoir host, such as coyotes? What does this suggest about the geographic scale over which CDV is operating?
- (3) How would the addition of a second host affect our estimate of the critical community size within any one host species and the spatial scale over which the disease may be persisting?

In order to answer these questions, we developed a computer simulation model (a susceptible-exposed-infectious-recovered



During collaring, Wolf Project staff collect biological samples to assess health and determine genetics.

disease model) that allowed us to simulate the spread of CDV between packs of wolves or coyotes on the landscape over time. In this model, we were able to manipulate the total host population size, the social group size, disease characteristics (e.g., the transmission rate, the duration of the infection, the disease-induced mortality rate), host survival, and the degree of spatial connectivity between social groups. We also created a two-species disease model, whereby we simulated CDV transmission within and between species, examining how this affected the spatial scale and total carnivore population size necessary for disease persistence.

Using these simulation models, we found that recent estimates of the GYE's gray wolf population (453 wolves; US Fish and Wildlife et al. 2008) were too small to support the persistence of CDV. Even when we expanded the potential number of hosts to include the entire population of wolves in the Northern Rocky Mountains (~1,500 wolves in 192 packs; US Fish and Wildlife Service et al. 2008), long-term persistence was still very unlikely with wolves as the sole maintenance population.

This finding suggested that outbreaks of CDV observed in YNP wolves were being driven by spillover from another carnivore host species. We found that the probability and magnitude of subsequent CDV outbreaks among wolves increased with increasing inter-wolf-pack connectivity, time since the last CDV outbreak, and increasing demographic turnover (survival and reproduction) rates.

Assuming coyotes were the most likely alternate host, based on their relative abundance and sociality, we estimated that there would need to be a minimum of 5,000 to 10,000 packs of coyotes, or between 50,000 and 100,000 individuals, to support a 50% probability of pathogen persistence over ten years. This is likely a conservative estimate; lower levels of spatial connectivity or increased spatial heterogeneity (due to habitat, variable hunting pressure, etc.) is likely

to drive this estimate upward of 15,000 packs (150,000 individuals) to achieve a reasonable probability of long-term pathogen persistence.

We also found that the presence of a second host generally increased the probability of disease persistence at smaller geographic scales. Transmission among multiple host species improved CDV persistence by both increasing the local density of hosts and adding meta-population structuring, either by providing another dimension of space where multiple species represent vertical layers of space that take additional time to invade and infect, in effect “buying time” for the pathogen until the next birth pulse of susceptible hosts; or by facilitating “rescue effects” when CDV burned out in any one species.

If our assumptions about CDV in canids are correct, namely, that there are no long-term carrier states for the virus and that CDV induces life-long immunity, CDV cannot currently be maintained in the GYE wolf population alone. Coyotes, by virtue of their relative abundance and wide distribution, are much more likely to be part of the local maintenance community for CDV. However, the large population sizes and spatial scales needed to ensure CDV persistence suggest that it is much more likely to be persisting via transmission among multiple host species at more regional geographic scales. Using a simplified two-host model, we found that it is theoretically possible that CDV is persisting at a geographic scale roughly 0.5 to 1.5 times the size of the GYE (32,500–97,500 km²) encompassing 2,500–7,500 coyote territories with approximately 50,000–150,000 hosts.

The large populations required for CDV persistence tend to refute the hypothesis that domestic dogs might constitute a viable CDV reservoir in and around the GYE. Unlike in much of sub-Saharan Africa where CDV, rabies, and other canid pathogens are thought to be maintained by extremely large populations of unvaccinated domestic dogs, the unvaccinated population of dogs in the United States is comparatively small. There are no published estimates of dog densities or vaccination compliance for the GYE. However,



COURTESY OF THE AUTHOR

Looking for *Sarcoptes scabiei*, the mite that causes mange.

even if we assume less-than-average vaccination coverage among local dogs, it is still unlikely that there are enough animals to maintain CDV. Although we cannot rule out the role of dogs visiting from all over the country, the likelihood of relevant contacts between these dogs and wildlife during the relatively short phase of infectiousness also seems low.

The exact combination of host species comprising the CDV maintenance community responsible for outbreaks among YNP wolves, coyotes, and cougars is unknown. Coyotes, raccoons (thought to be the dominant reservoir host for CDV in the eastern United States), and perhaps some of the mustelid species are the most likely candidates. Future research on these species could include serological work to determine whether CDV is circulating among them.

Since it is likely that CDV is persisting among multiple, wild host species and/or over a large geographic scale, any system-wide attempt at eradication or control would be both impractical and impossible. Instead, we have suggested that state managers pay particular attention to CDV and make corresponding adjustments to management activities so as to accommodate potentially sizeable and unpredictable population declines.

Pathogen monitoring and surveillance

To augment the information gained from serological surveys, in 2008 the group began to collect samples that could be directly screened for the presence of various viral pathogens using the molecular technique, polymerase chain reaction. We have since been collecting fecal samples as well as fecal, eye, and nasal swabs during necropsies and winter wolf capture operations. Fecal samples and swabs have been screened for CPV, CAV-1, and canine coronavirus (CCV), a pathogen that can cause severe gastrointestinal disease and mortality, particularly when coupled with a CPV infection. In addition to these enteric pathogens, we have screened for pathogens found in the respiratory tract, including CDV, CHV, canine adenovirus type-2, canine respiratory coronavirus, canine parainfluenza, and canine influenza type A, all of which are considered common or emerging among domestic dogs. Although the sampling window for this surveillance tool is brief (swabs are only taken during captures or necropsies and reflect active infections only), if we collect enough samples, we may be able to address questions about transmission and dynamics using the genetics of these pathogens.

Echinococcus granulosus is a tapeworm that requires both ungulates and canids to complete its life-cycle. The tapeworm's eggs, which are shed in canids' feces, are consumed by ungulates, where they mature into larvae that cause large cysts throughout the ungulate's liver and lungs. When canids consume these cysts, the larvae develop into adults that then sexually reproduce within the canid's small intestine. *E. granulosus* is considered a zoonotic pathogen and if humans

accidentally consume eggs shed in canid feces, the larvae can, in some cases, cause a potentially lethal disease. Although the park does not screen for *E. granulosus*, we would like to briefly comment on the public's recent concern over the perceived transmission risk to humans.

Some have suggested that wolves are increasing the risk to humans of contracting *E. granulosus* infections. We have no evidence to suggest that *E. granulosus* was not already present throughout the Northern Rockies well before the reintroduction of wolves; domestic dogs and coyotes are extremely competent definitive hosts. In fact, a domestic biotype of *E. granulosus* (one of the strains most lethal to humans) was circulating among domestic sheep and dogs in Idaho in the absence of wolves (Jenkins et al. 2005). Given the small number of wolves compared to domestic dogs and coyotes outside YNP, wolves probably have a minimal effect on the already small risk of humans contracting the disease. Basic precautions when handling dead canids or canid feces should be sufficient to prevent human infection. A number of years ago, several canid biologists (who had collectively handled thousands of wolves, coyotes, and canid scats throughout North America) were screened for *E. granulosus*, and none was positive (International Wolf Center 2010). The incidence of this disease in humans is low throughout North America, and as long as basic precautions are observed, it does not appear to be a major human health concern in the GYE.

Sarcoptic mange

Sarcoptic mange is an infectious disease of the skin caused by the mite *Sarcoptes scabiei*. The mite burrows into its mammalian host's epidermis to feed and lay eggs, which causes severe irritation and itchiness, skin lesions, secondary skin infections, and hair loss. Sarcoptic mange was introduced into the Northern Rockies in 1909 by state wildlife veterinarians in an attempt to help eradicate local wolf and coyote populations.



Skin lesion on a wolf infected with sarcoptic mange.



Wolf 625F, a female of the Leopold pack, was healthy during her collaring in 2009 (left). Less than a year later, she died from the effects of mange infection (right).

With the successful extirpation of wolves from the Northern Rockies, the mite is thought to have persisted among regional furbearers such as coyotes and foxes. The current epidemic among wolves in the GYE began about 2002 in southwest Montana and northwest Wyoming outside YNP (Jimenez et al. 2010). Mange was first officially detected in YNP in the winter of 2006–2007 among several wolves of Mollie’s pack in the park interior. It rapidly spread to the northern range, and has afflicted roughly half of the park’s packs, primarily those on the northern range. The number of infected packs/groups peaked at 8 of 16 during the fall and winter of 2009; as of the summer of 2010, only 3 of the 12 packs/groups in YNP were infected (Yellowstone Wolf Project, unpublished data).

Studies on coyotes and red foxes outside of the GYE have documented significant deleterious impacts of mange on host survival, reproduction, body condition, and social behavior, but conclusions regarding the effects of the disease at the population level are mixed. Several studies have found evidence for mange-induced population declines in foxes and coyotes (Forchhammer and Asferg 2000; Chronert et al. 2007), while Pence and Windberg (1994) believed that coyote mortality associated with mange in Texas was compensatory. Mange is hypothesized to have contributed to an 11% decline in wolf population growth in Wisconsin in 1993 and the reduction in the rate of expansion of wolves in Michigan (Michigan Department of Natural Resources 1997).

Beginning in 2008, the Yellowstone Wolf Project began a partnership with the US Geological Survey to rigorously address questions about how mange is affecting individual wolves and the overall population in the Yellowstone region. Since then, they have been monitoring individuals and their mange status over time, following their survival, reproductive status, and social status. The project has also been conducting population surveys to determine the prevalence

of infection across YNP over time. The aim is to compare the fates of infected and uninfected individuals in the current outbreak as well as population metrics before and after mange arrived in the park. We hypothesize that mange will negatively affect wolf survival, reproduction, and pack cohesion, and will increase the probability of dispersal for diseased individuals. Based on what has been observed in other wolf populations, we anticipate that the prevalence of mange will wax and wane over time, but will remain endemic in YNP for the foreseeable future. The impacts of mange may be more severe in YNP than in neighboring regions due to higher local wolf densities and consequently may be of particular concern with respect to how it affects the rate at which healthy YNP wolves disperse to Montana, Idaho, and Wyoming.

Conclusion and future direction

Parasites can play important roles in the ecology of a system. Despite the fact that they are so small and can go easily unnoticed, pathogens and parasites can make up a surprisingly large portion of an ecosystem’s biomass. One study showed that parasites outweighed the top predators of several estuary ecosystems (Kuris et al. 2008). Behind the scenes, these pathogens can affect important ecological processes. The challenge remains to identify these important pathogens, measure their impacts on their host populations, and relate these impacts to larger ecological processes. For example, how do CDV and/or mange-induced population declines in wolves and coyotes affect top-down processes like predation pressure on elk or small mammals? Are there measurable bottom-up drivers of disease, such as the effects of food stress on pathogen susceptibility? In the case of pathogens that affect multiple host species, are some species better equipped to handle infection, giving them a competitive advantage? As climate changes, are there detectable effects on pathogen abundance and

distribution, and therefore effects on host morbidity and mortality? These are the challenging questions.

As novel pathogens continue to emerge via jumps into new host species or new geographic regions, and as climates change, it is reasonable to anticipate the invasion of new pathogens into wildlife populations. For example, although canine heartworm, which is transmitted between canids via mosquitoes, had not previously been present in the Yellowstone area, it is now found in a number of urban centers throughout Montana, including the nearby Gallatin Valley. Climate change, particularly increases in the mean nighttime low temperatures during summer, combined with visiting dogs that carry the active parasite, may assist in its range expansion.

The reintroduction of wolves into the Northern Rocky Mountains has been a conservation success story. To ensure the long-term legacy of this historic effort, the regional states and YNP have voiced a commitment to monitoring the effect of infectious disease on wolf populations and making any necessary adjustments to management activities. For biologists and ecologists, Yellowstone National Park continues to provide an amazing place to study ecological interactions, of which pathogens and parasites are another integral part.

YS



COURTESY OF THE AUTHOR

Literature Cited

- AlMBERG, E.S., P.C. Cross, and D.W. Smith. 2010. Persistence of canine distemper virus in the Greater Yellowstone Ecosystem's carnivore community. *Ecological Applications* 20(7):2058–2074.
- AlMBERG, E.S., L.D. Mech, D.W. Smith, J.W. Sheldon, R.L. Crabtree. 2009. A serological survey of infectious disease in Yellowstone National Park's canid community. *PLoS ONE* 4:e7042.
- International Wolf Center. 2010. Reality check: western wolves and parasites. Interview with L. David Mech. 3/12/2010. http://www.wolf.org/wolves/news/live_news_detail.asp?id=4768. Accessed 4/12/2011.
- Barker, I.K., C.R. Parrish, and E.S. Williams. 2001. Parvovirus infections. In *Infectious diseases of wild mammals*. E.S. Williams and J.K. Barker eds., 131–146. Iowa: Iowa State University Press.
- Biek, R., T.K. Ruth, K.M. Murphy, C.R. Anderson, Jr., M. Johnson, R. DeSimone, R. Gray M.G. Hornocker, C.M. Gillin, and M. Poss. 2006. Factors associated with pathogen seroprevalence and infection in rocky mountain cougars. *Journal of Wildlife Diseases* 42: 606–615.
- Chronert J.M., J.A. Jenks, Roddy D.E., Wild M.A., and J.G. Powers. 2007. Effects of sarcoptic mange on coyotes at Wind Cave National Park. *Journal of Wildlife Management* 71:1987–1992.
- Forchhammer M.C., and T. Asferg. 2000. Invading parasites cause a structural shift in red fox dynamics. *Proceedings of the Royal Society B: Biological Sciences* 267(1445):779–786.
- 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.
- Greene, C.E. 2006. Neosporosis *Neospora caninum* infection. In *Infectious diseases of the dog and cat*. Greene, ed., 768–774. Philadelphia: Saunders/Elsevier.
- Greene, C.E., and M.J. Appel. 2006. Canine Distemper. In *Infectious diseases of the dog and cat*. Greene, ed., 25–41. Philadelphia: Saunders/Elsevier.
- Greene, C.E., and L.E. Carmichael. 2006. Canine herpesvirus infection. In *Infectious diseases of the dog and cat*. Greene, ed., 47–53. Philadelphia: Saunders/Elsevier.
- Jenkins D.J., T. Romig T, and R.C.A. Thompson. 2005. Emergence/re-emergence of *Echinococcus* spp.—a global update. *International Journal for Parasitology* 35:1205–1219.
- Jimenez M.D., E.E. Bangs, C. Sime, and V.J. Asher. 2010. Sarcoptic mange found in wolves in the Rocky Mountains in western United States. *Journal of Wildlife Diseases* 46(4):1120–1125.
- Kuris, A.M., R.F. Hechinger, J.C. Shaw, K.L. Whitney, L. Aguirre-Macedo, C.A. Boch, A.P. Dobson, E.J. Dunham, B.L. Fredensborg, T.C. Huspeni, J. Lorda, L. Mababa, F.T. Mancini, A.B. Mora, M. Pickering, N.L. Talhouk, M.E. Torchin, and K.D. Lafferty. 2008. Ecosystem energetic implications of parasite and free-living biomass in three estuaries. *Nature* 454:515–518.
- Michigan Department of Natural Resources. 1997. Michigan gray wolf recovery and management plan. Lansing, MI: Michigan Department of Natural Resources, Wildlife Division.
- Pence, D.B., and L.A. Windberg. 1994. Impact of a sarcoptic mange epizootic on a coyote population. *Journal of Wildlife Management* 58: 624–633.
- Woods, L.W., E.S. Williams,, and I.K. Barker. 2001. Adenoviral diseases. In *Infectious diseases of wild mammals*. E.S. Williams and J.K. Barker eds., 202–211. Iowa: Iowa State University Press.
- US Fish and Wildlife Service, Nez Perce Tribe, National Park Service, Montana Fish, Wildlife and Parks, Blackfoot Nation, Confederated Salish and Kootenai Tribes, Idaho Fish and Game, and USDA Wildlife Services. 2008. Rocky Mountain Wolf Recovery 2007 Interagency Annual Report. C.A. Sime and E.E. Bangs, eds. USFWS, Ecological Services, Helena Montana. 275p.

Emily S. AlMBERG (left) is a PhD candidate at Pennsylvania State University pursuing the study of sarcoptic mange and its impacts on Yellowstone's wolf population. She has worked and collaborated with the Yellowstone Wolf Project since 2003.

Paul C. Cross is a disease ecologist at the USGS Northern Rocky Mountain Science Center. His research integrates field ecology, animal behavior, statistics, mathematical modeling, remote sensing, microbiology, virology, and genetics to address wildlife disease, conservation, and management issues.

L. David Mech is a senior scientist with the Biological Resources Division, US Geological Survey and an adjunct professor in the Department of Fisheries, Wildlife and Conservation Biology, and

Ecology, Evolution and Behavior at the University of Minnesota. He has studied wolves and their prey since 1958.

Doug W. Smith is the leader of Yellowstone National Park's Wolf Project and has been with the project since its beginning in 1994. He received his PhD from the University of Nevada, Reno in Ecology, Evolution and Conservation Biology.

Jennifer W. Sheldon is an ecologist with the Yellowstone Ecological Research Center, specializing in terrestrial ecology and canid behavioral ecology.

Robert L. Crabtree is chief scientist of the Yellowstone Ecological Research Center. His specialties include ecosystem and landscape ecology, and predator-prey relations and behavioral ecology.