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THE ROLE OF PREDATION IN DISEASE CONTROL: A COMPARISON OF SELECTIVE AND NONSELECTIVE REMOVAL ON PRION DISEASE DYNAMICS IN DEER

Margaret A. Wild,[1,5] N. Thompson Hobbs,[2] Mark S. Graham,[1,4] and Michael W. Miller[3]

[1] National Park Service, Biological Resource Management Division, 1201 Oak Ridge Drive, Suite 200, Fort Collins, Colorado 80525, USA
[2] Natural Resource Ecology Laboratory, Colorado State University, Fort Collins, Colorado 80523-1499, USA
[3] Colorado Division of Wildlife, Wildlife Research Center, 317 West Prospect Road, Fort Collins, Colorado 80526-2097, USA
[4] Current address: National Park Service, New River Gorge National River, PO Box 246, 104 Main Street, Glen Jean, West Virginia 25846, USA
[5] Corresponding author (email: margaret_wild@nps.gov)

ABSTRACT: Effective measures for controlling chronic wasting disease (CWD), a contagious prion disease of cervids, remain elusive. We review theoretic relationships between predation and host-parasite dynamics and describe a mathematical model to evaluate the potential influence of random removal through harvest or culling and selective predation by wolves (*Canis lupus*) upon CWD dynamics in deer (*Odocoileus* spp.) populations. Imposing nonselective mortality representing a 15% annual harvest or cull 51 yr after CWD introduction lowered both deer population size and steady state CWD. Selective (4×) mortality at the same 15% predation rate caused a more modest reduction in deer population size accompanied by a relatively rapid decline in CWD prevalence and elimination of the disease from a closed population. The impacts of selective predation on epidemic dynamics were sensitive to assumptions on parameter estimates; however, within expected ranges, the results of selective predation were consistent and robust. We suggest that as CWD distribution and wolf range overlap in the future, wolf predation may suppress disease emergence or limit prevalence.

Key words: *Canis lupus*, chronic wasting disease, deer, host-parasite, *Odocoileus* spp., predator-prey, selective predation, wolf.

INTRODUCTION

Disease emergence and reemergence threaten the abundance and viability of wildlife species worldwide (Daszak et al., 2000). Although a variety of factors appear to be contributing to the recent surges in diseases impacting natural populations, ecosystems altered by human activities seem particularly vulnerable to such effects (Harvell et al., 1999; Daszak et al., 2000, 2001; Kutz et al., 2005; Johnson et al., 2007; Pedersen et al., 2007). Ecologic imbalances can diminish the resilience of host species to natural fluctuations in pathogens and the host's capacity to resist or recover from pathogen introductions. Such impacts on resiliency can be observed with alterations to host-parasite relationships resulting in changes in host survival and contact rates among susceptible and infected individuals (Harvell et al., 1999; Daszak et al., 2001; Kutz et al., 2005; Johnson et al., 2007; Pedersen et al., 2007).

Changes in predation rates or predator-prey dynamics are among the factors that may affect patterns of disease emergence, reemergence, and persistence (Choo et al., 2003; Packer et al., 2003; Holt and Roy, 2007). The potential effects of predation on epidemic dynamics vary depending on both the nature of predation occurring upon hosts and attributes of the host-parasite relationship. Nonselective predation could dampen epidemic dynamics by reducing host densities and contact rates or by lowering the total number of infected individuals in a host population (Heesterbeek and Roberts, 1995; Barlow, 1996; Packer et al., 2003). Similarly, selective predation on infected individuals could eliminate pathogens or prevent their establishment under some circumstances (Heesterbeek and Roberts, 1995; Gross and Miller, 2001; Packer et al., 2003).

Alternatively, both nonselective and selective predation might facilitate pathogen emergence and persistence in cases where resistant individuals become less abundant (Choisy and Rohani, 2006; Holt and Roy, 2007) and in cases where infected individuals are avoided by predators (Packer et al., 2003). It follows that ecosystems altered by removal of natural predators by humans may respond differently to endemic or novel pathogens than intact systems.

In light of the potential influence of predation on host-parasite dynamics, the role of predators should be considered in devising strategies for control of emerging or reemerging pathogens in natural populations. We review the theoretic relationships between predation and host-parasite dynamics, using the term parasite broadly to describe any infectious agent capable of infecting a host, utilizing host resources, and spreading to new hosts (Altizer et al., 2003). We then describe a simple mathematical model developed to evaluate how dynamics of prion disease in deer (*Odocoileus* spp.) populations may respond to nonrandom removal resulting from selective predation by wolves (*Canis lupus*) and compare this outcome with effects of random removal through harvest or culling.

Predation and host-parasite dynamics

Nonselective predation: The interplay among host regulation, immune response, and the pattern of predator selectivity determines whether predation reduces or increases the prevalence of disease in a population (Holt and Roy, 2007). Under many scenarios, increasing mortality rates in diseased populations can retard disease transmission and reduce disease prevalence (Barlow, 1996; Lafferty and Holt, 2003; Packer et al., 2003; Ostfeld and Holt, 2004). Increasing mortality slows transmission via two mechanisms. First, it reduces the average lifetime of infected individuals. Reduced lifespan, in turn, can truncate the time interval when animals are infectious, thereby reducing the number of infections produced per infected individual. Second, the effect of reduced intervals of infectivity is amplified by reductions in population density that occur as mortality increases; such reductions cause declines in the number of contacts between infected and susceptible individuals. Both of these mechanisms slow rates of transmission of disease. If these mechanisms cause the number of new infections produced per infected individual to fall below one, then the disease will be eliminated from the population.

Selective predation: Any elevation in mortality rate has the potential to cause the foregoing effects. Reductions in transmission rates and disease prevalence can be particularly large if mortality rates are disproportionately higher in the infected portion of the population than in the susceptible portion (Heesterbeek and Roberts, 1995). This explains why diseases that cause rapid death fail to persist. However, other, nondisease, agents of selective mortality can exert the same beneficial effect. For example, if predators prey selectively on diseased individuals, it is reasonable to expect that they might reduce disease prevalence much more rapidly than would occur if mortality were nonselective.

Evidence that predators have a greater selectivity for diseased prey has been widely observed. Voříšek et al. (1998) found parasitized voles in buzzards' diets in a greater proportion than they occurred in the population. Birds with high blood parasite loads (Moller and Nielsen, 2007) and birds with weakened immune systems (Moller and Erritzoe, 2000) were preyed upon at higher rates than uncompromised birds. Murray et al. (1997) reported increased predation on snowshoe hares (*Lepus americanus*) with heavy burdens of the sublethal nematode *Obeliscoides cuniculi* during periods of limited food supplies.

It is logical to assume that predators' high success with diseased prey may be due to poorer body condition of the prey and consequently prey's slower avoidance behavior, decreased awareness, or re-

duced stamina. Studies have suggested that predators may also use visual pattern, scent, or behavioral cues to select compromised prey. Hudson et al. (1992) suggested that heavily parasitized female red grouse (*Lagopus lagopus scoticus*) emitted more scent, and were, as a result, more easily detected by mammalian predators. Larks (*Calandrella rufescens*) that were infected with poxvirus had shorter, lower-pitched distress calls than uninfected birds, indicating a behavioral change that could affect predation rates (Laiolo et al., 2007). Lafferty and Morris (1996) reported that parasitized killifish (*Fundulus parvipinnis*) exhibited more conspicuous behavior than uninfected killifish, and were also preyed upon more heavily by birds. Red-legged frog (*Rana aurora*) tadpoles also exhibited modified behavior when infected with yeast (*Candida humicola*), resulting in changes in thermoregulatory behavior, compromised predator avoidance behavior, and increases in being preyed upon (Lefcort and Blaustein, 1995). Examples of increased vulnerability to selective predation in large mammals are less numerous; however, diseased moose (*Alces alces*; Joly and Messier, 2004a) and bison (*Bison bison*; Joly and Messier, 2004b) appeared to be more susceptible to predation by wolves than apparently healthy animals. White-tailed deer (*Odocoileus virginianus*) killed by wolves may appear normal to human inspection, but subtle alterations may be present as demonstrated by the correlation of fawn and subadult survival to maternal and grand-maternal nutrition (Mech et al., 1991). Further, Krumm et al. (2009) recently reported that mountain lions (*Puma concolor*) prey selectively on prion-infected mule deer (*Odocoileus hemionus*) in Colorado, USA.

Wolves, selective predation, and prion disease dynamics

Chronic wasting disease (CWD; Williams and Young, 1980) is a contagious prion disease of at least four North American cervid species (Spraker et al., 1997; Baeten et al., 2007). The origins and evolutionary history of CWD are unclear, but uncontrolled epidemics have the potential to depress deer populations (Williams and Young, 1992; Miller et al., 2000, 2006; Gross and Miller, 2001; Williams et al., 2002) and to impact ecosystems dominated by these species (Hobbs, 1996). Epidemics of CWD are sustained naturally by horizontal transmission (Miller and Williams, 2003; Miller et al., 2006), with both infected animals and contaminated environments serving as sources of infection (Miller and Williams, 2003; Miller et al., 2004, 2006; Mathiason et al., 2006, 2009; Tamgüney et al., 2009). Under some conditions, the CWD agent persists in the environment for years in residues from excrement and infected carcasses (Miller et al., 2004). Mechanisms for both direct (animal-animal) and indirect (animal-environment-animal) prion transmission have been demonstrated empirically (Miller et al., 2004; Mathiason et al., 2006, 2009; Tamgüney et al., 2009), but models incorporating indirect transmission best represent epidemic dynamics in captive deer (Miller et al., 2006).

Effective measures for controlling CWD remain elusive. In the absence of vaccines or therapies, strategies undertaken to combat CWD have focused on depressing the abundance of host species either locally or regionally in an attempt to disrupt prion transmission (Williams et al., 2002; Grear et al., 2006; Conner et al., 2007). Thus far, control strategies relying on hunting or culling by humans to lower deer numbers and subsequently CWD prevalence have not yielded demonstrable effects (Conner et al., 2007). However, these results are not surprising given the limited duration of such management actions and because theory suggests that randomly removing individuals from an infected population should have less effect on epidemic dynamics than selectively removing infected individuals (Heesterbeek and Roberts, 1995; Gross and Miller,

2001). The protracted course of CWD in deer (Williams and Young, 1980, 1992; Fox et al., 2006) and occurrence of agent shedding well before the hallmark signs of emaciation and behavioral changes are discernable to human observers (Mathiason et al., 2009; Tamgüney et al., 2009) suggest that selectively removing only obviously ill deer from a population would not be an effective control strategy (Gross and Miller, 2001). If infected deer were detectable earlier in the disease course, however, selective removal might be more effective than random removal in controlling epidemics (Gross and Miller, 2001; Wolfe et al., 2004).

Increased vulnerability of CWD-infected mule deer to vehicle collisions (Krumm et al., 2005) suggests that lowered vigilance also might make them more vulnerable to large predators. It follows that if natural predators were able to develop a search image for subtle behavioral changes of CWD infection in deer, then fostering predation upon CWD-infected deer populations might offer a viable adjunct or alternative to other control measures. Although mountain lions do appear to preferentially prey on mule deer infected with CWD (Krumm et al., 2009), epidemics persist in mule deer herds in the presence of mountain lion predation (Miller et al., 2008; Krumm et al., 2009). Based on the subtlety of the behavioral changes early in the course of CWD infection, we would expect coursing predators like wolves to show even greater potential selective capability than ambush predators like mountain lions; however, wolves were extirpated and packs are presently absent from the areas in North America where CWD is endemic in deer, so field data are not available for comparison. Consequently, to assess this possibility we developed and explored the behavior of models representing the effects of selective predation by wolves and compared these with nonselective predation, such as through harvest or culling, on CWD dynamics in deer.

MATERIALS AND METHODS

Model structure

We explored the potential impacts of predation on dynamics of deer populations using a simple model of interactions among infected animals, susceptible animals, and infectious residue in the environment in a closed population. We derived the model used here (Appendix A) from the indirect transmission model of Miller et al. (2006), which was the best approximating model of two CWD epidemics in a captive population of mule deer. Because of the similarities in CWD epidemiology between mule deer and white-tailed deer (Miller and Wild, 2004), here we generalize inferences to "deer." We modified the best approximating model found by Miller et al. (2006) to portray disease dynamics in free-ranging populations as follows:

1) We assumed that transmission rates were approximately 25 times lower in natural populations than in captive ones. This assumption was based on the elevated densities of deer in captive populations (Miller et al., 2006). Adjusting transmission rates for differences in density was plausible; however, the magnitude of the adjustment for transmission was uncertain. Therefore we targeted this adjustment as one of the variables to be explored in simulation studies through the use of a scaling coefficient.
2) Per-capita birth rates were assumed to decline linearly with increasing population density.
3) We added a term representing predation. This term could be adjusted to reflect selective predation, where predators favored infected animals over susceptible ones, or nonselective predation, as would occur with hunting or culling, where removals were assumed to be random. In the case of selective predation on diseased animals, we also included a term to represent the extent to which predation mortality was compensatory with CWD mortality.

We sought to use the simplest model possible to achieve the greatest generality of results (Levins, 1966) and to reduce the number of parameters that had to be estimated. We avoided the use of an age-structured model, which would have required estimating unknown transmission rates for several age classes. Dynamics of the prey population was not coupled to the dynamics of predators and predation intensity did not change with prey abundance. In the interest of parsimony, we

used a constant relative rate for predation. Preliminary modeling included a type II functional response and did not yield results that were qualitatively different than those presented here. More importantly, by holding predation constant, we could be sure that observed dynamics resulted from the interplay between CWD and deer, rather than between deer and wolves (analogous to choosing to hold one factor constant in a designed experiment). Although our model is simple, we believe it represents the essential interactions in the deer-CWD system. As knowledge of parameters improves, more detailed models will be justified.

Model experiments

We exercised the model to examine how selective and nonselective predation may influence CWD prevalence. We made three model runs using our most plausible estimates of model parameters to examine differences among trajectories of diseased populations in the presence and absence of predation. We first conducted a reference simulation introducing a single infected animal into a population of 1,000 deer at time=0 and allowed the model to equilibrate over 100 yr. In two experimental simulations, we introduced predation in year 51. In one of these simulations, predation was assumed to occur randomly; in the other, predators were assumed to favor infected individuals. In the case of selective predation, the modeled 15% predation rate was equivalent to about seven wolves removing 16 deer/wolf/yr (Mech and Peterson, 2003).

Although most parameters in the model were derived from Miller et al. (2006) or from reasonable assumptions on deer biology (See Table 1 in Appendix A for all parameter values), there was substantial uncertainty in our best guesses of the value of several parameters controlling the effects of the disease and of predation. Notable among these were the extent of predator selectivity for CWD-infected animals, the extent of compensation between CWD and predator mortality, and the adjustment for the rate of transmission in free-ranging populations. We explored consequences of these uncertainties by conducting model experiments varying these parameters singly and in pairs to examine the sensitivity of model predictions to uncertainty in their estimates.

RESULTS

In the absence of CWD and predation, the modeled deer population stabilized at an ecologic carrying capacity of about 1,000 animals. Adding a single infected deer in year 1 produced oscillatory dynamics typical of epidemics. With disease and no predation, the equilibrium density was 736 deer and disease prevalence was 29%. Thus, the disease reduced animal abundance in our model by almost a third (Fig. 1A, B). Our model resembles classic susceptible-infected (SI) models with an additional mortality source from predation and an environmental reservoir of infection. Models of this general type are known to have conditions that allow steady states (Miller et al., 2006), and the model used here shows that equilibrium.

Simulated selective and nonselective predation affected epidemic dynamics to different degrees. Imposing nonselective mortality representing a 15% annual harvest or cull in year 51 lowered both deer population size and steady state CWD prevalence; however, under the assumptions of this simulation, the disease was able to persist in the population (Fig. 1C, D). Selective (4×) mortality at the same 15% predation rate beginning in year 51 caused a more modest reduction in deer population size accompanied by a relatively rapid decline in CWD prevalence and elimination of the disease from this closed population (Fig. 1E, F). The impacts of selective predation on epidemic dynamics were sensitive to assumptions on vulnerability of infected animals and compensation between predation and mortality due to CWD, as well as overall predation and CWD transmission rates (Fig. 2). Doubling the vulnerability of infected animals to selective predation accelerated the rate of decline in prevalence (Fig. 2A). Increasing the proportion of compensatory deaths among infected deer dampened the predicted decline in prevalence (Fig. 2B); when compensation exceeded ca. 60%, selective predation had less of a predicted effect on epidemic dynamics than nonselective predation. The overall predation rate also affected the rate and magnitude of decline in steady state prevalence (Fig. 2C). Epidemic dynamics also were sensitive to assumptions on values for the scaling of transmission rate (Fig. 2D), with asymptotic prevalence varying by a

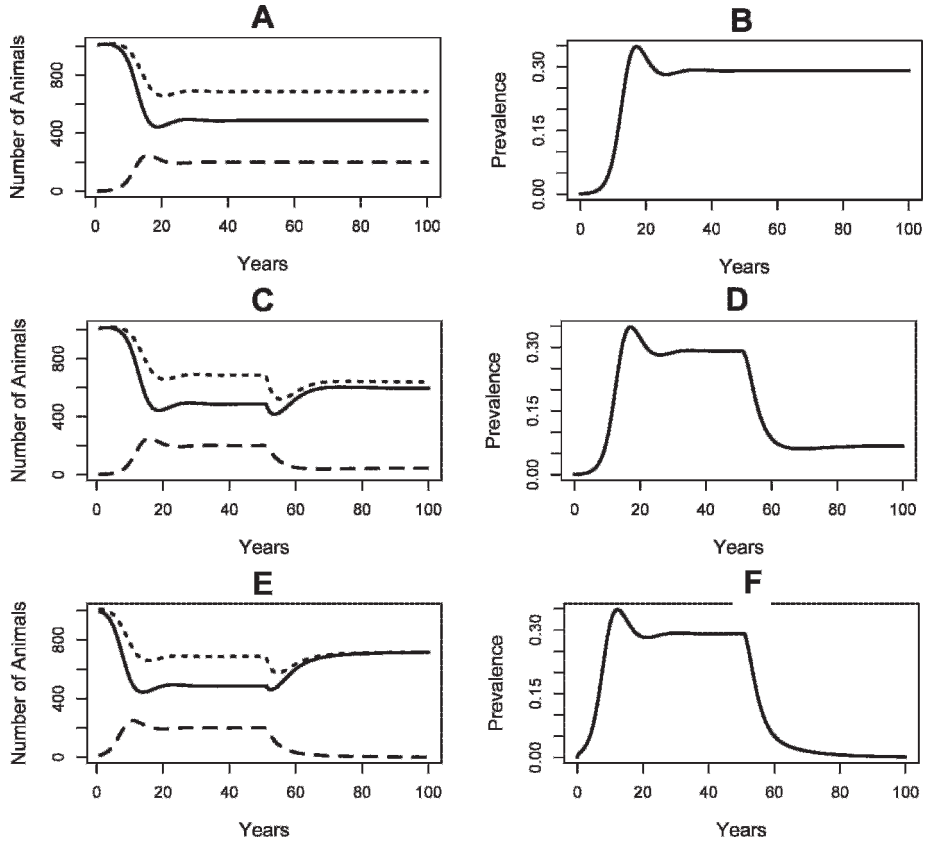

FIGURE 1. Simulations of deer abundance and disease prevalence in populations infected with CWD assuming no predation (A, B), nonselective predation (C, D), and selective predation (E, F). Lines in population-number graphs are number of susceptible deer (solid), number of infected deer (dashed), and total population (dotted). In the absence of CWD and predation, the population would reach equilibrium at 1,000 animals. We assumed that predators consumed four times more infected animals than would be expected by random selection among susceptible and infected deer and that compensation of predation for disease is 0.3. The predation rate was set at 0.15 beginning in year 51 and the scaling factor for transmission rate was 25 in all simulations.

factor of more than 10 when changing the scaling coefficient from 20 to 40.

Our models predicted that interactions between the relative selectivity of predation and the degree to which mortality in infected deer is compensatory also will influence epidemic dynamics in emergent CWD foci (Fig. 3). Predicted CWD prevalence 20 yr after introducing a single infected deer into simulated populations subjected to 15% annual predation under different combinations of selectivity and compensation varied from ~0 to 8%. In general, simulations suggested that even modest levels of selectivity might be expected to greatly diminish the persistence of CWD in a susceptible deer population provided that such pressure was largely additive; however, models predicted that sufficiently strong selection could still dampen the dynamics of emergent CWD epidemics even in cases where mortality among infected deer was largely compensatory.

DISCUSSION

Results from these simulations suggest that predation could markedly decrease prevalence of CWD under certain condi-

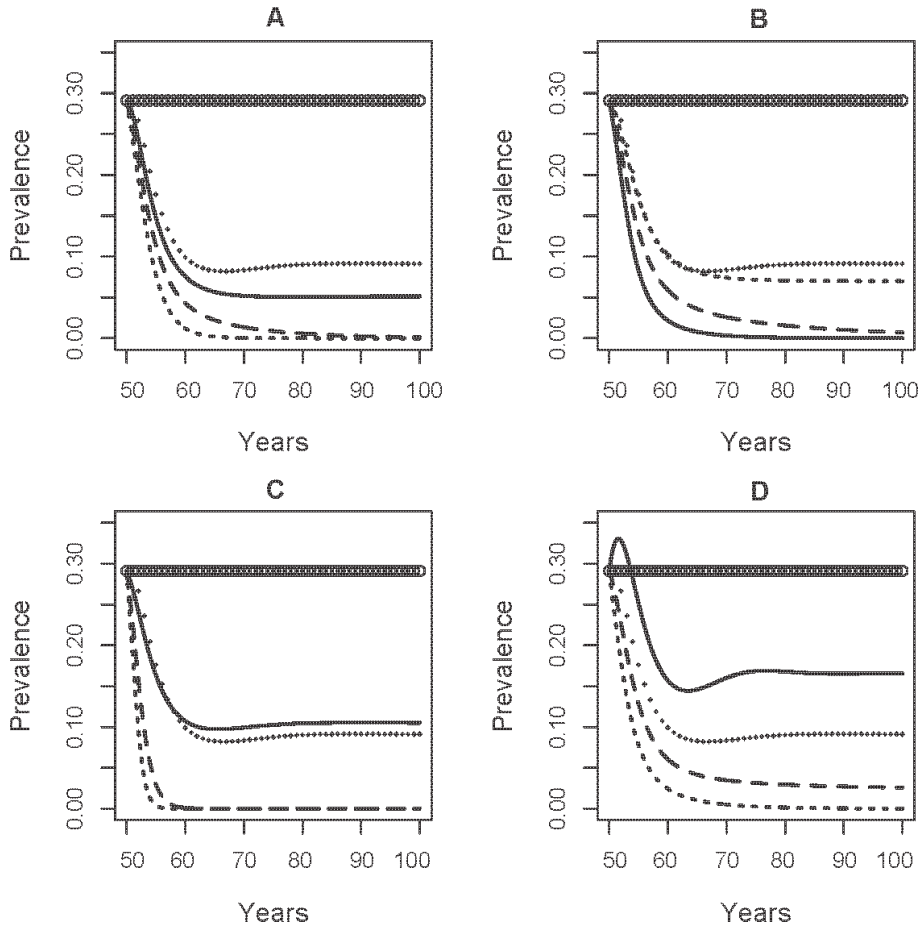

FIGURE 2. Results of model experiments to examine sensitivity of variation in uncertain model parameters. Open circles show results from simulations with no predation, diamonds show nonselective predation at a rate of 0.15. A. Effect of variation in prey vulnerability to selective predation (v=2, solid line; 4, dashed line; 8, dotted line). Increasing values of v indicate greater selection for infected over susceptible animals. In all cases, compensation was held constant at 0.3 and predation rate at 0.15. B. Effect of variation in the level of compensation between predation and CWD mortality (c=0.1, solid line; 0.3, dashed line; 0.6, dotted line). Increasing values of c indicate greater compensation between predation and CWD. In all cases, selectivity was held constant at four and the predation rate at 0.15. C. Effect of variation in predation rate (δ; solid line, 0.10; dashed line, 0.30; dotted line, 0.50). In all cases, vulnerability to selective predation was held constant at 4 and compensation at 0.3. D. Effect of variation in the scaling coefficient for the transmission rate (solid line, 20; dashed line, 30; dotted line, 40). The scaling coefficient reduces the transmission rate to account for differences between captive and free-ranging deer. A scaling coefficient of 20 indicates that transmission is 20 time more rapid in captivity than in the wild. For other parameter values, see Table 1 in Appendix A.

tions. Nonselective predation, as might occur with hunting or culling by humans, may decrease disease prevalence over time but the disease was not eliminated under modeled conditions (Fig. 1C, D). Alternatively, selective predation by wolves at the same rate would result in a more precipitous drop in CWD prevalence that would culminate in disease elimination in a closed system (Fig. 1E, F). Selective predation does not allow a larger population of susceptible animals to persist relative to the nonselective case because wolves are assumed to consume

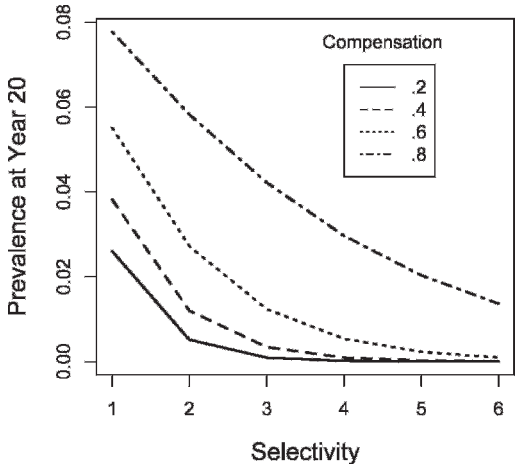

FIGURE 3. Predicted CWD prevalence 20 yr after introducing a single infected deer into simulated populations subjected to 15% annual predation. Results of this model experiment revealed interactions between selectivity and compensation: for example, low compensation and high selective predation result in inability of disease to emerge. The benefits of selectivity for reducing prevalence are opposed by increasing compensation.

more susceptible animals as infected ones become rare. Although the time required to achieve results depends in a fundamental way on assumptions about prey vulnerability to selective predation and the nature of compensation among different sources of mortality, as well as parameters regulating disease transmission, it appears that prevalence could be halved within a decade and eliminated within the century through sustained predation by a pack of wolves that removed 15% of deer per year in a closed population.

Although uncertainty in parameter estimates limits our confidence in predicting the precise timeframe required for control or elimination of disease, these time estimates provide a basis for comparison of approaches. What is most clear is a consistent and robust trend toward decreasing CWD prevalence in populations subject to predation, particularly selective predation, over a range of parameter estimates (Fig. 2). A similar decreasing trend would be predicted in a population subject to predation where CWD was repeatedly introduced at low levels (i.e., an open population); however, the slope of decline would be variable and elimination might never be achieved because high rates of disease reintroduction may offset selective predation of CWD-positive individuals. Although they are not the most likely scenarios, other combinations of parameters, in particular high excretion rates leading to increased levels of transmission, also may result in an inability to eliminate the disease within a reasonable period of time.

Simulation results suggested that selective predation could also dampen or eliminate the emergence of CWD in new locations (Fig. 3), adding support to speculation that the absence of large predators presents an amplification risk factor for establishment of CWD (Samuel et al., 2003). Our prediction may prove testable in the future as geographic distribution of CWD expands to areas such as the Greater Yellowstone Ecosystem and northern Wisconsin, USA, and Prince Albert National Park, Canada, where wolves are present but adjacent areas lack wolves. The simulated influences of large predators on the outcomes of CWD epizootics also may lend insight into circumstances surrounding the original emergence of CWD in Colorado, where wolves have been absent since 1943 and where mountain lion populations were suppressed by bounty hunting at the time of likely CWD emergence in the mid-1900s (Barrows and Holmes, 1990; Miller et al., 2000). The origins of CWD are unknown but may have been a result of spillover of scrapie from domestic sheep or may represent a spontaneous, naturally occurring spongiform encephalopathy of cervids (Williams and Young, 1992; Spraker et al., 1997). Regardless, our simulations suggest that had selective predation by wolves been present during that period, CWD may never have been established or detected. In combination with influences of human-assisted movement of infected cervids (Williams et al., 2002) and land use

alterations (Farnsworth et al., 2005), the absence of large predators, particularly wolves, over much of their native range in the United States (Laliberte and Ripple, 2004) has likely played a significant role in the current unnatural distribution and prevalence of this disease.

The decrease in CWD prevalence observed in simulations with selective predation is most likely a result of removing infectious individuals earlier in the disease course. Chronic wasting disease exhibits a prolonged disease course of about 18–36 mo (Williams and Miller, 2002). Transmission models (Miller et al., 2006) reveal little support for a disease latency period and instead support early onset of prion shedding, potentially from peripheral lymphoid tissue. Accumulation of abnormal prion protein (PrP^{cwd}) in deer has been observed in alimentary tract–associated lymphoid tissues as early as 42 days following experimental oral inoculation (Sigurdson et al., 1999) and in tonsils as much as 20 mo prior to death from naturally occurring CWD (Wild et al., 2002). Moreover, orally inoculated deer shed infectious prions in saliva and feces 6–11 mo or more before the onset of clinical signs (Mathiason et al., 2009; Tamgüney et al., 2009). Therefore, early removal of infected individuals should markedly truncate CWD shedding and resultant opportunities for disease transmission.

The prolonged clinical course and type of clinical abnormalities associated with CWD make it the prototypic disease for selection by predators. Chronic wasting disease produces subtle changes in behavior and body condition that progress over weeks or months to overt signs of end-stage disease typified by loss of attentiveness or response to external stimuli, emaciation, and weakness (Williams and Young, 1980, 1992; Wild et al., 2002). Loss of attentiveness and cognitive function due to the neurodegenerative process likely account for the marked increase in risk for vehicle collision of CWD infected mule deer compared to hunter-harvested deer (Krumm et al., 2005). It follows that infected deer also would be less attentive to predators, and in later stages, that emaciation and weakness would decrease both their fight and flight response capabilities (Krumm et al., 2005, 2009; Miller et al., 2008); a nearly fourfold greater relative risk of infected mule deer succumbing to mountain lion predation (Miller et al., 2008) supports this notion. Furthermore, predators—particularly coursing predators such as wolves—focus on animals vulnerable due to odd behavior or compromised body condition (Temple, 1987; Mech et al., 1991). Field observations also suggest that predators can select CWD-infected deer: mule deer killed by mountain lions were much more likely (odds ratios $\geq$3.2) to be infected with CWD than same-sex deer killed in the vicinity by hunters (Krumm et al., 2009). Based on the prolonged course of CWD, the ability of wolves to detect vulnerable prey, and field observations of mountain lion predation patterns in a system where CWD occurs naturally, we believe that selective predation modeled at a rate four times higher than that of healthy deer is a reasonable, if not conservative, estimate.

Overall, our modeling results also are likely a conservative portrayal of the beneficial impacts that selective predation could have on damping prion epidemic dynamics in deer. The model we developed did not include carcasses of infected deer as a source of infectivity because necessary parameter estimates were not available (Miller et al., 2006). However, carcasses of CWD-infected deer would be an added source of environmental infectivity in natural systems (Miller et al., 2004), and thus their consumption by wolves or other carnivores either via selective predation or scavenging would be expected to reduce the contribution of carcass material to the overall pool of environmental infectivity through local dispersal and dilution (Krumm et al., 2009). Passage through the alimentary

tract of wolves likely markedly degrades infectivity of tissues. In sheep, in vitro incubation of a dilute scrapie brain inoculum with alimentary tract fluids resulted in almost complete degradation of PrP (Jeffrey et al., 2006). Moreover, changes in deer behavior due to the presence of predators, i.e., predation risk effects or what has been termed the ecology of fear (Brown et al., 1999; Ripple and Beschta, 2004), include changes in use of space through habitat preferences or foraging patterns within a given habitat, or both (Lima and Dill, 1990). If deer move more within established home ranges due to fear of predation, then contact rates with environmental deposits of infectivity also might diminish. Given the sensitivity of epidemic dynamics to such contact rates, even relatively small reductions would further dampen epidemic dynamics beyond effects arising from selective predation on infected deer alone.

Although here we modeled wolf predation on deer, similar outcomes would be expected for wolf predation on other species susceptible to CWD. Hobbs (2006) used CWD and elk (*Cervus elaphus nelsoni*) population data from Rocky Mountain National Park (Colorado, USA) to model the impact on CWD that may be achieved through maintaining a pack of wolves in the park. Results from these simulations supported the idea that predation could drive decreases in CWD prevalence over a range of parameter estimates. Impacts by predators other than wolves may also reduce CWD prevalence to varying degrees, as seen in our results from nonselective removal by humans. We consider the wolf, a large coursing predator, to be most effective in selective removal of deer vulnerable from CWD infection; however, opportunistic mountain lions (Krumm et al., 2009), and potentially coyote (*Canis latrans*) packs, would likely benefit from lack of vigilance by CWD-affected deer as well.

The potential impact on wolves and other native North American predators from consumption of CWD-positive ruminants is unknown; however, no evidence of naturally occurring CWD has been reported outside four species in the family Cervidae. Limited surveillance of predators and scavengers in CWD-affected areas (Jennelle et al., 2009; Miller and Wild, unpubl. data) has not revealed evidence of abnormal prion accumulation. Naturally occurring transmissible spongiform encephalopathies (TSE) other than CWD have been documented in domestic mink (transmissible mink encephalopathy), domestic sheep and goats (scrapie), and domestic cattle (bovine spongiform encephalopathy [BSE]), as well as in humans (variant Creutzfeldt-Jacob disease) and domestic and captive wild felids (feline spongiform encephalopathy) that consumed BSE-contaminated feed (Hörnlimann et al., 2007). Interestingly however, no TSE has been observed in a canid despite dietary challenge of BSE to dogs (Kirkwood and Cunningham, 1994). A species barrier is generally believed to be responsible for the specificity of prion diseases to their respective hosts, although some spillover, as with BSE, has been documented for at least one prion strain. Raymond et al. (2000) demonstrated a barrier at the molecular level that they suggest limits the susceptibility of noncervid species to CWD. The dog and wolf are very similar in PrP sequence and quite different from cattle, domestic cats, and elk (Schätzl, 2007).

We suggest that predation, particularly wolf predation, may be a useful tool for management of CWD. Currently, the range of wolves (Boitani, 2003) does not overlap with the distribution of CWD (Chronic Wasting Disease Alliance, 2009) so our predictions on the effects of wolves on CWD prevalence remain untested. However, as wolf range expands through Wyoming and Wisconsin, USA, and Alberta and Saskatchewan, Canada, and into Colorado and Utah, USA, the possibility for such evaluation may occur. Alternatively, CWD may be detected in a new

geographic location where wolves are present. Based on our simulations, disease may be difficult to detect in these areas unless unique methods of surveillance, such as monitoring of wolf-killed cervids for presence of PrP^{res}, are implemented. Beschta and Ripple (2009) suggest that restoration of large predators, such as wolves, provides a recovery strategy for native flora, functional predator-prey-scavenger food webs, and ecosystems degraded by overabundant wild ungulates. Wolf restoration also provides an opportunity to observe and evaluate the effects that selective predation may have on prevalence of an invariably fatal chronic disease in deer and elk. In areas where predator restoration is not possible, deployment of wolves as stewardship tools for the primary purpose of disease control could provide a novel approach to management.

Although somewhat novel, the concept of using wildlife species as stewardship tools to provide ecosystem services is not new. Restoration of bison to reestablish healthy landscapes of prairie vegetation in the United States (United States Department of the Interior, 2008) and large carnivore, (e.g., lion [*Panthera leo*]), translocations to restore ecologic integrity in fenced parks in Africa (Hayward et al., 2007) are occurring. Licht et al. (2010) propose use of small populations of wolves for ecosystem restoration in North America. Public tolerance of wildlife, particularly predators, may dictate intensive management in species used in such restoration efforts. Regardless of whether wolves are managed under natural regulation or primarily for fulfilling their ecologic role, they provide a promising approach for control of CWD that warrants further evaluation.

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LITERATURE CITED

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APPENDIX A—MODEL STRUCTURE

Using data from two epidemics of chronic wasting disease (CWD) in a captive population of mule deer (*Odocoileus hemionus*), Miller et al. (2006) found that models of indirect transmission of CWD from excreta had almost seven times more support in data than more traditional models of direct, animal-to-animal transmission. The best approximating model in their studies used three linked differential equations representing the number of infected and susceptible animals and the mass of infectious material in the environment:

$$\begin{aligned} \frac{dS}{dt} &= a(I+S) - S(\gamma E + m), \\ \frac{dI}{dt} &= \gamma SE - I(m+\mu), \\ \frac{dE}{dt} &= \varepsilon I - \tau E, \end{aligned} \tag{1}$$

where

S=number of susceptible (uninfected) animals, I=number of infected animals, E=the mass of infectious material in the environment, α=the per capita birth rate, m=the per

capita death rate from causes other than CWD, γ=the indirect transmission coefficient, μ=the additive, per capita death rate from CWD, ε=the per capita rate of excretion of infectious material by infected animals, and τ=the mass specific rate of loss of infectious material from the environment.

This model is based on two assumptions, that the instantaneous per capita rate of infection was directly proportionate to the mass of infectious material in the environment (i.e., $dI/dtS=\gamma E$) and that the rate of uptake of infectious material by deer has negligible effects on the pool size.

We modified this model to include density-dependent effects on recruitment into the population and to include selective and nonselective predation:

$$\begin{aligned}\frac{dS}{dt} &= a(S+I)\left(1-\frac{S+I}{K_a}\right)-S(\gamma E+m)\\ &\quad -(1-p)\delta(S+I),\\ \frac{dI}{dt} &= \gamma SE-I(m+\mu)-p(1-c)\delta(S+I),\\ \frac{dE}{dt} &= \varepsilon I-\tau E,\end{aligned} \tag{2}$$

where K_a is the population level where birth rate=0 and δ is the additive, instantaneous per capita rate of predation when predators select prey randomly. Predation rates were adjusted to account for selectivity by the term p, which represents the proportion of the total kill that was infected. We calculated p as

$$p=\frac{vI}{vI+S} \tag{3}$$

where v is the vulnerability of infected animals relative to susceptible ones. Relative vulnerability is a multiplier giving the number of infected animals in the total kill per susceptible animal, assuming equal abundance of infected and susceptible. Thus, a value of $v=2$ means that if susceptible and infected animals were equally abundant, wolves would selectively kill twice as many infected animals as susceptible ones. A value of $v=1$ indicates no vulnerability of infected animals and increasing values of v above 1 indicate increasing vulnerability to selective predation.

If predators select prey totally at random, then the probability of dying from CWD is independent of the probability of dying from predation, as δ is defined. In this case the probability that an infected animal will survive, φ, over an interval of time=Δt is

$$\phi=e^{-(m+\mu+\delta)\Delta t}. \tag{4}$$

However, when predators are selective, then it follows by definition that the probability of dying from predation is not independent of the probability of dying from the disease:

$$\phi=e^{-[m+\mu+\delta(1-c)]\Delta t} \tag{5}$$

The term c allows us to represent the extent to which predation mortality compensates for CWD mortality. Because $1/m+\mu+\delta$ is the average lifetime of an infected animal assuming that disease mortality and predation mortality are completely additive, it follows that $1/[m+\mu+\delta(1-c)]-1/(m+\mu+\delta)$ is the increase in the average lifetime of an infected animal that results because predation mortality may not fully add to disease mortality. The value of c ranges from 0 to 1. When $c=0$, then predation mortality is completely additive with CWD mortality, as in equation (4). When $c=1$, predation mortality is completely compensatory and does not add to deaths from disease (i.e., deer would have died from CWD within the year had they not been preyed upon).

To solve the system of equations in (2), we used numeric integration implemented in the lsoda package of the R computing environment (R Development Core Team, 2008). Values for parameters used in simulations are derived from Miller et al. (2006) and plausible assumptions about deer population dynamics in the absence of CWD (Table 1).

TABLE 1. Values for model parameters used in example simulations.

Parameter	Definition	Value[a]	Reference or source
a	Birth rate[b] at population=0	0.6	Medin and Anderson, 1979
m	Non-CWD[c] death rate[d]	0.1	White and Bartmann, 1998
K_a	Population at which birth rate=0	1,230	Assigned
γ	Transmission rate[e]	0.787	Miller et al., 2006
μ	CWD death rate	0.567	Miller et al., 2006
ε	Rate of excretion of infectious material	0.111	Miller et al., 2006
τ	Rate of loss of infectious material from the environment	2.55	Miller et al., 2006

[a] Units for all rates are per year.

[b] The birth rate in continuous time, which corresponds to a discrete time birth rate of 1.8 fawns per female.

[c] CWD=chronic wasting disease.

[d] The continuous-time death rate corresponds to an annual adult survival probability of 0.90.

[e] The transmission rate was scaled to account for differences in density between the wild and the captive setting where it was measured by Miller et al. (2006). The default scaling factor was allowed for densities in paddocks that were 25 times higher than in the wild, thus 0.787/25.