A Model Analysis of Effects of Wolf Predation on Prevalence of Chronic Wasting Disease in Elk Populations of Rocky Mountain National Park

N. Thompson Hobbs
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Background

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 compress the time interval when animals are infectious, thereby reducing the number of infections produced per infected individual. The effect of reduced intervals of infectivity is amplified by reductions in population density that occur as mortality increases, reductions that cause declines in the number of contacts between infected and susceptible individuals. Both of these mechanisms retard the 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 eradicated from the population.

Any elevation in mortality rate has the potential to cause these effects, however, reductions in transmission rates and disease prevalence can be particularly large if mortality rates are elevated in the infected portion of the population to a greater extent than in the susceptible portion. This explains why diseases that cause rapid death fail to persist. However, other, non-disease 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 non-selective.

Here, I use a simple mathematical model to evaluate the potential for selective predation by wolves to reduce or eradicate chronic wasting disease in populations of elk in Rocky Mountain National Park.

Model Structure

Miller et al. (2004)showed that CWD can be transmitted to susceptible animals from residues of excreta from infected animals and their carcasses. Using data from two epidemics of CWD in a captive population of mule deer, Miller et al. (submitted) found that models of indirect transmission of CWD from excreta had almost 7 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:

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

where

S = number of susceptible animals,
I = number of infected animals,
E = the mass of infectious material in the environment,
a = the per capita birth rate,
m = the per capita death rate from causes other than CWD,
γ = the indirect transmission coefficient,
μ = the per capita death rate from CWD,
ε = the per capita rate of excretion of infectious material by infected animals,
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., $\frac{dI}{dtS} = \gamma E$, and that the rate of uptake of infectious material by elk has negligible effects on the pool size.

I modified this model to include density dependent effects on recruitment into the population and to include predation by wolves:

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

where K_a is the population density where recruitment = 0 and d_s is the number of susceptible elk killed by wolves per unit time and d_i is the number of infected elk killed by wolves. The total number of elk killed by wolves was calculated using a type III functional response (Coughenour 2002) assuming that wolves preyed exclusively on elk. Thus, the total kill rate (d) was calculated as

$$d = \frac{WF_{\max}\left(S+I\right)^2}{{w_0}^2+\left(S+I\right)^2} \qquad (3)$$

where W is the number of wolves, $F_{\max}$ is the maximum annual kill rate, and w_0 is the density of elk at the maximum rate of increase in the kill rate, that is, the inflection point in the functional response.

The total kill rate (d) was partitioned into infected animals killed (d_i) and susceptible animals killed (d_s) as

$$\begin{aligned} d_i &= p(1-c_i)d \\ d_s &= (1-p)(1-c_s)d \end{aligned} \tag{4}$$

where c_i represents the extent to which wolf predation is compensatory with natural mortality and disease mortality in infected animals and c_s represents the extent to which wolf predation is compensatory with natural mortality in susceptible animals. The values of the cs range from 0–1. When they are equal to 0, then wolf mortality is completely additive. When they are equal to 1, then kills by wolves are completely compensatory and do not add to other sources of mortality.

The p term in equation 4 represents the proportion of the total kill that is infected. It is calculated as

$$p = \frac{vI}{vI + S} \tag{5}$$

where v is the relative 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 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.

The number of wolves (W) is treated as a constant in the model under the assumption that management action, both culling and introductions, would be used to maintain a constant pack size.

Estimates of Model Parameters

Parameters for population dynamics in the absence of disease (a, m, K_a) were estimated from a time series of data on elk abundance using maximum likelihood techniques (Fig. 1) (Table 1). Estimates of parameters governing disease transmission have not been estimated for elk. In the absence of these estimates we modified parameter values for deer obtained by Miller et al. (in preparation). It appears that CWD transmission in elk populations occurs substantially more slowly than in deer (Miller et al. 1998, Miller and Wild 2004). Thus, as a first approximation, we estimated transmission rates (γ) in elk as an order of magnitude less rapid than those for deer and excretion rates (ε) as ½ the value for deer. Model experiments were conducted to examine sensitivity of predictions to variation in these approximations. The turnover rate of infectious material in the environment was estimated from Miller et al. (submitted) and compensation terms for susceptible animals (c_s) as well as parameters of functional response ($F_{\max}$, w_0) were obtained from Coughenour (2002). The values of vulnerabilities (v) and compensation

for infected animals (c_i) were not estimated, but instead were varied across a range of plausible values in model experiments.

Table 1. Estimates of model parameters.

Parameter	Definition	Units	Value
a	maximum per capita rate of recruitment	yr^{-1}	0.188
m	per capita natrual mortality rate	yr^{-1}	0.041
μ	per capita mortaltiy rate from CWD	yr^{-1}	0.57
γ	transmission rate	$mass^{-1}\ yr^{-1}$	0.0789
ε	excretion rate	mass yr^{-1}	0.055
τ	turnover rate of infectious material	yr^{-1}	2.55
Ka	population number where recruitment is 0	number	1287
S_0	initial number of susceptibles	number	300
E_0	initial mass of infected material	number	0
I_0	initial number of infecteds	number	10
W	number of wolves	number	20
Fmax	maximum kill rate	yr^{-1}	25
wo	inflection point of functional response	number	968
c_s	Compensation between predation and natural mortality	unitless	0.3

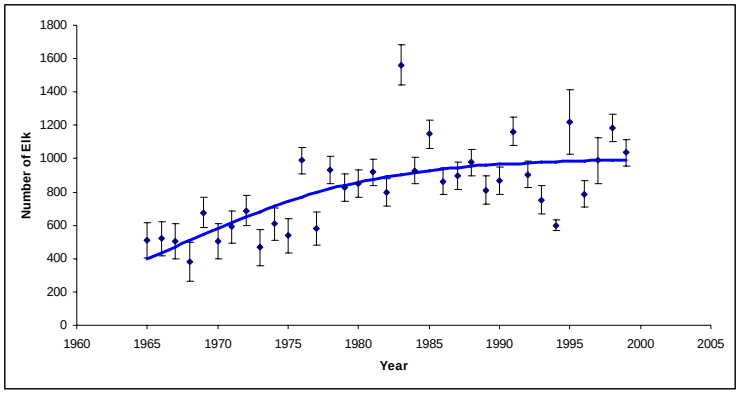
Model Experiments

Dynamics of infected and susceptible pools were estimated using numerical methods (4^{th} order Runge-Kutta). I ran the model without disease, with disease and without wolves, and with disease and with wolves. To add wolves to the simulation, I allowed the model to run for 49 years with disease and without wolves. Fourteen wolves were introduced at year 50. The number of years required to eradicate the diseases was recorded for 7 levels of vulnerability (v = 1, 1.5, 2.0, 2.5, 3, 3.5, 4) and four levels of compensation (c_i = 0, .25, .50, .75). In addition, we varied excretion rate of infectious material and the transmission rate as .1, .3 and .5 x values observed for deer while holding vulnerability constant at 2 and compensation constant at .5.

Results

In the absence of disease the model predicted that the elk population would reach an ecological carrying capacity of 1044 animals over about 35 years (Figure 1). Adding disease in year 1 produced oscillatory dynamics typical of epidemics (Figure 2 A, B). Adding wolves in year 50 caused relatively rapid decline in the prevalence of CWD, leading to eradication 19 years after introduction (Figure 3 A, B). The time required to eradicate the disease was sensitive to assumptions on vulnerability of infected animals and compensation between predation and mortality due to disease and natural causes (Figure 4). Increasing vulnerability of infected animals accelerated the rate of eradication but did not influence the number of susceptible elk at steady state, which was approximately 240 animals, regardless of vulnerability.

Figure 1. Model fit to data on elk population size in Rocky Mountain National Park assuming no influence of disease on population dynamics.



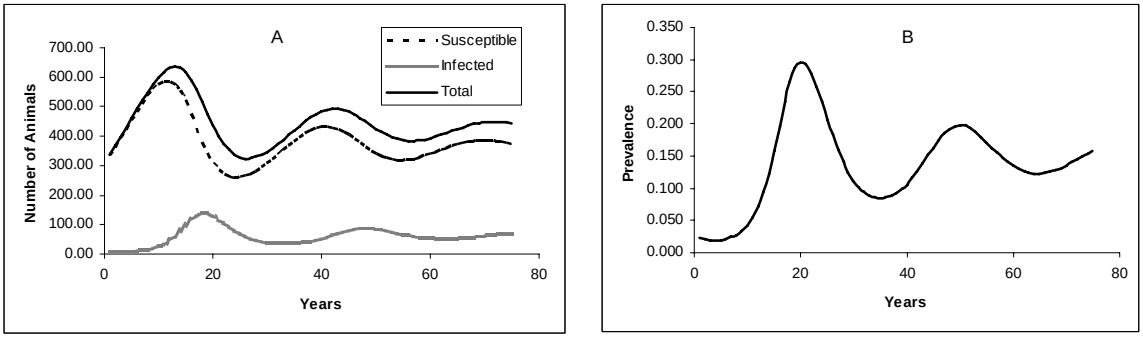


Figure 2. Model output for population sizes (A) and disease prevalence (B) assuming no wolves.

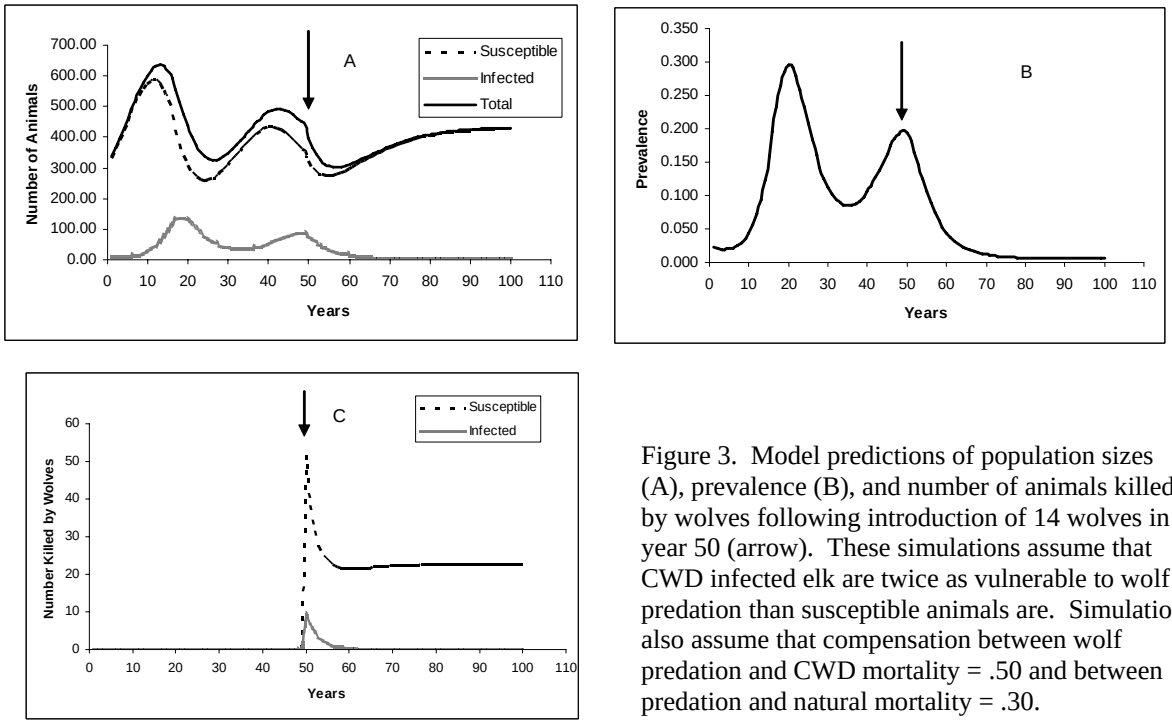


Figure 3. Model predictions of population sizes (A), prevalence (B), and number of animals killed by wolves following introduction of 14 wolves in year 50 (arrow). These simulations assume that CWD infected elk are twice as vulnerable to wolf predation than susceptible animals are. Simulations also assume that compensation between wolf predation and CWD mortality = .50 and between predation and natural mortality = .30.

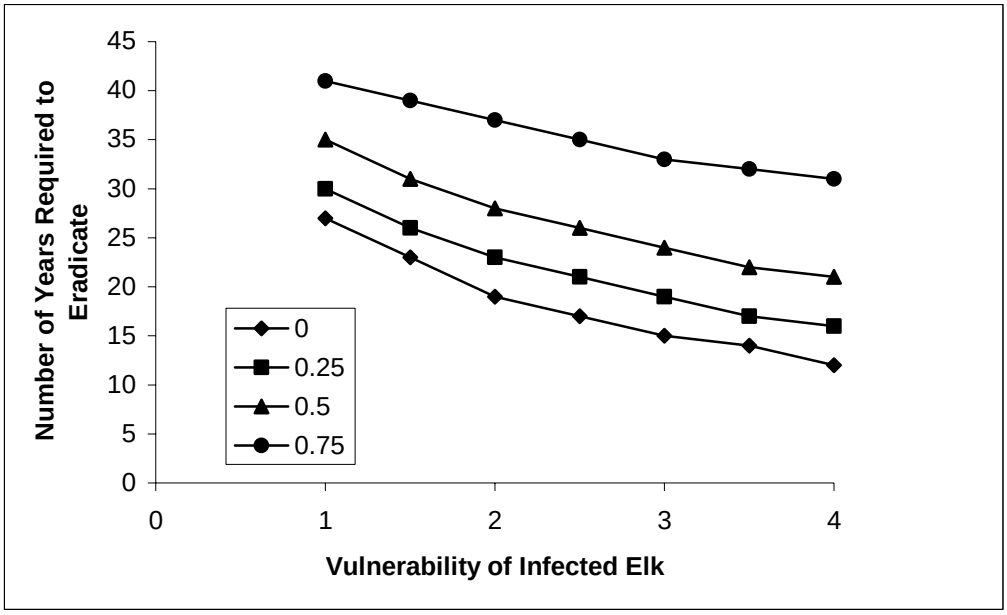


Figure 4. Model experiment varying relative vulnerability (x-axis) of infected elk to predation by wolves and level of compensation between predation and mortality from CWD (series). Relative vulnerability is a multiplier giving the number of infected animals in the total kill per susceptible animal assuming equal abundance of infected and susceptible. Thus, a value of $v = 2$ means that if susceptible and infected animals were equally abundant, wolves were 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. Compensation estimates the extent to which predation mortality fails to add to other sources. When compensation = 0, predation mortality is entirely additive.

Predictions of time to eradication were sensitive to assumptions on values for excretion rate and transmission rate (Figure 5), varying by more than 10 fold over a relatively narrow range of parameter values.

Time to eradication declined as the population size of wolves increased (Figure 6). For any given wolf population size, times to eradication increased with increases in the extent of compensation between predation and disease mortality. When compensation between disease mortality and predation mortality was high (i.e., .75), there was a threshold population size of wolves somewhere between 10 and 15 animals where eradication of CWD was infeasible (causing the truncation of the .75 compensation curve in Figure 6). When the wolf population size dropped below this threshold, predation simply pushed the prevalence of the disease to a new, lower steady state in the elk population. Near this threshold, time to eradication declined dramatically with increasing numbers of wolves. However, when the wolf population size exceeded 20, further increases in numbers of wolves had much less dramatic effects on time to eradication (Figure 6).

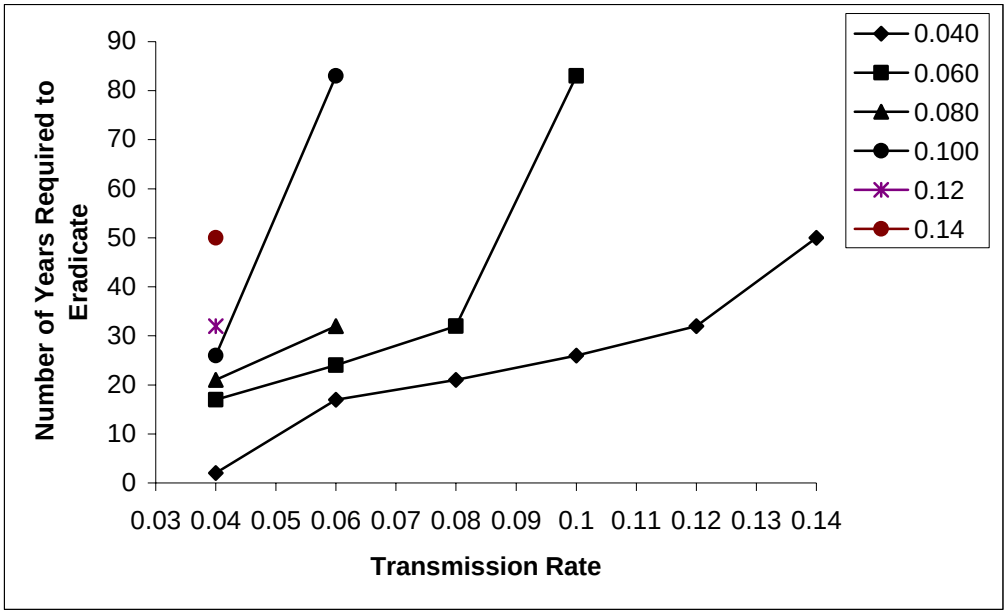


Figure 5. Sensitivity of predicted time to eradication to assumptions on rate of transmission of CWD (x-axis) and rate of excretion of infectious material (series). In theses simulations, I held constant parameter values for relative vulnerability of infected animals $(\nu = 2)$ and compensation of predation mortality for disease mortality (c_i = .5). Areas of the graph that lack points indicate combinations of parameters for which eradication was infeasible during 150 year simulations.

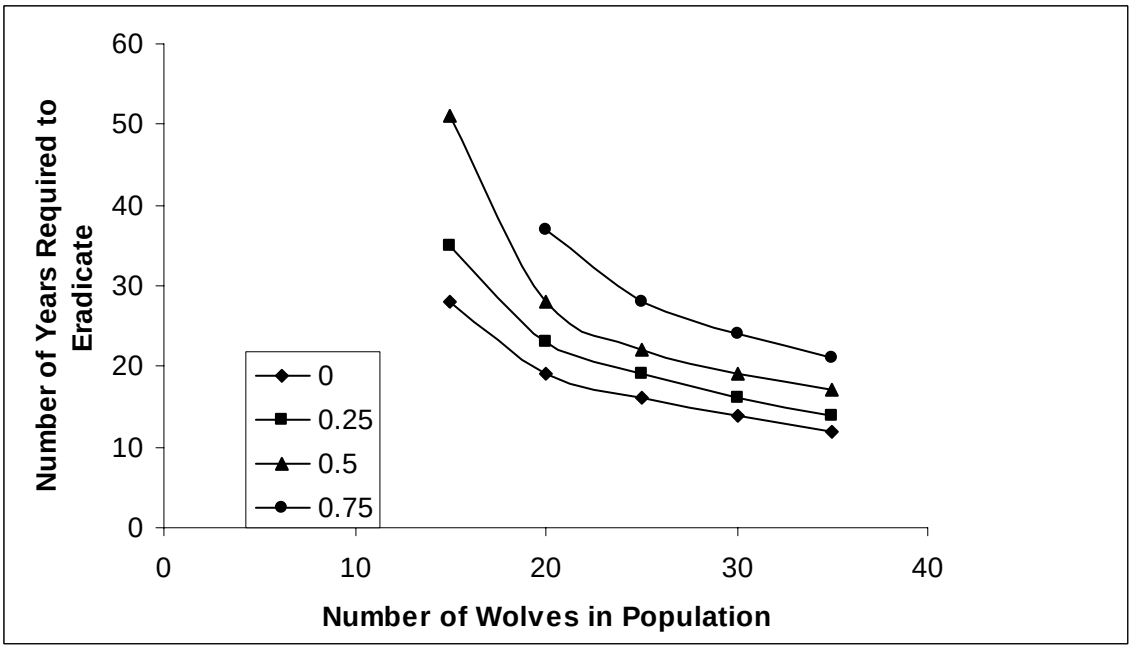


Figure 6. Sensitivity of predicted time to eradication to assumptions on the number of wolves maintained in the population (x-axis) and the extent of compensation between predation and disease mortality (series). These simulations assumed that vulnerability of infected elk to wolf predation was twice as great as vulnerability of susceptible elk and that the transmission rate was

.07879 yr^{-1}. The truncated curve indicates combinations of parameters where eradication is infeasible.

Discussion

Results from simulations suggest that predation by wolves has the potential to eliminate CWD from an infected elk population. Although the time required to achieve this result depends in a fundamental way on assumptions about prey vulnerability, the nature of compensation among different sources of mortality, as well as parameters regulating disease transmission, it appears that eradication within two or three decades would be feasible by maintaining a constant population of approximately 20 wolves.

This result must be tempered with the very important caveat that the elk population is closed to infection from outside sources. If infection is continually reintroduced, then eradication may not be feasible. However, model results suggest that even of open populations, wolf predation will substantially reduce prevalence. Moreover, the model does not represent a potentially large benefit of predation, the removal of carcasses as sources of infection. Although I did not have any way to estimate parameters for transmission from carcasses, such transmission is known to occur in mule deer.

Selective predation does not allow a larger population of susceptible animals relative to the non-selective case because wolves are assumed to consume more susceptible animals as infected ones become rare.

Model experiments reveal that uncertainties in estimates of values of parameters for transmission and excretion rates cause substantial uncertainty in estimates of time required to eradicate CWD from elk populations. Thus, the quantitative result from the simulations described here is not strong—we cannot specify with confidence how long it might take for predators to eliminate CWD from an elk population. Moreover, there are combinations of parameters for which eradication is infeasible. In particular, if transmission and excretion rates are high, it appears unlikely that predation by wolves will cause eradication within any reasonable period of time.

These results suggest that predation by wolves could have potent effects on disease prevalence under certain conditions. Although non-selective predation, as might occur with culling for example, may also be effective in eradicating the disease in a closed population, our results suggest that natural predation could substantially reduce the time required to eliminate the disease.

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