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

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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.

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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.

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