

Destabilization of cow elk survival and population growth by chronic wasting disease

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Abstract

Investigations of chronic wasting disease (CWD), a contagious prion disease of cervids, have found it can cause long-term population declines in deer (*Odocoileus* spp.). However, almost no data exist on the individual or population-level effects of CWD on free-ranging elk (*Cervus elaphus nelsoni*). We conducted a radio-telemetry study on adult female elk from 2008-2010 and determined incidence (proportion of susceptible elk infected per year), sources of mortality, annual survival, and population growth in a high-density herd from Rocky Mountain National Park (RMNP) that has been infected by CWD for over 25 years. Annual incidence of CWD was 0.08 (95% credible limits (CL) = 0.05, 0.12). Following removal of elk that tested positive for CWD via rectal biopsy at project initiation, annual survival (excluding harvest) consistently

declined from 0.97 in 2008 (CL = 0.93, 0.99) to 0.85 in 2010 (CL = 0.75, 0.93). Declines in survival were due almost entirely to increases in the cause-specific mortality rate of CWD, which increased from 0.02 in 2008 (CL = 0.00, 0.05) to 0.11 in 2010 (CL = 0.04, 0.21). We used survival rates of susceptible and infected elk to develop a transition matrix, which estimated the intrinsic population growth rate (λ) of this elk herd to be 0.97 (CL = 0.90, 1.06) at the prevalence of CWD (12.9%) observed during this study. We conclude that CWD-caused mortality can exceed natural rates of mortality, destabilize cow elk survival, and cause population declines in high-density elk herds.

Key words: *Cervus elaphus*, chronic wasting disease, Colorado, elk, incidence, mortality, prion, survival

Introduction

The duration and extent of disease-related impacts on wildlife populations are normally constrained by a variety of factors, including development of host immunity or resistance (Bonneaud et al. 2011) and increasingly limited rates of disease transmission as contacts between susceptible hosts and pathogens decline (Haukisalmi and Henttonen 1990, Arneberg et al. 1998, Krasnov et al. 2002). However, hosts that have not evolved in the presence of an introduced or emerging disease frequently lack the ability to overcome such pathogens or limit their spread. This can lead to long-term, disease-induced host population declines or extirpation (e.g., McCallum 2008).

Chronic wasting disease (CWD) is a contagious transmissible spongiform encephalopathy that infects cervids in North America. It is an emerging prion disease that was first described in 1967 (Williams and Young 1980, Williams and Miller 2002) and first detected in free-ranging

cervids in an elk from Rocky Mountain National Park, Colorado, in 1981 (Spraker et al. 1997). There is no evidence that infected individuals develop immunity or survive infection (Williams et al. 2001).

Once established, there are several facets of CWD that may allow it to maintain a relatively constant rate of transmission, regardless of host population density, and depress the population size of ungulates. First, CWD prions (PrP^{CWD}) remain infectious for at least two years and likely longer in soil (Miller et al. 2004), and the closely-related prions that cause sheep scrapie are known to remain infectious for at least 16 years in farm pastures of Iceland (Georgsson et al. 2006). If environmental contamination plays an important role in the transmission process, as data suggest (Miller et al. 2006), rates of exposure will either exhibit a delayed response or have no relationship with host population density (Schuaber et al. 2007, Almberg et al. 2011). Second, research on captive animals indicates that PrP^{CWD} accumulation progressively increases in the nervous system and lymphoid tissues of infected deer (*Odocoileus* spp.) and elk (*Cervus elaphus nelsoni*) (Spraker et al. 2002, Williams and Miller 2002, Miller and Williams 2003, Spraker et al. 2010). The principle route of transmission and duration of prion shedding is unclear, but the potential exists for an individual to shed prions for months or years and across a variety of seasonal habitats before succumbing to clinical disease. Finally, modeling results indicate that, even with intensive management, it is difficult to ensure the long-term sustainability of mule deer (*Odocoileus hemionus*) populations once CWD is established and exceeds 1-5% prevalence (Gross and Miller 2001, Wasserberg et al. 2009, Almberg et al. 2011). Empirical efforts to date generally support these findings and have found CWD can be maintained at a relatively high level of prevalence (e.g., 20-30%), even in the presence of mule deer population declines that exceed 50% (Miller et al. 2008).

Extant knowledge of CWD in free-ranging ungulate populations is based almost entirely on studies of deer. Field studies have found that the annual survival and reproduction of infected mule deer is 30-40% lower than uninfected deer (Miller et al. 2008, Dulberger et al. 2010). Horizontal transmission has been demonstrated to occur through direct and indirect routes (Miller and Williams 2003, Miller et al. 2004, Miller et al. 2006), although the relative importance of host contact versus environmental contamination in free-ranging deer is unknown (Conner et al. 2008, Almberg et al. 2011). A significant body of work has amassed over the past decade on the effectiveness of management strategies and importance of host and spatial metrics on prevalence of CWD in free-ranging deer (Farnsworth et al. 2005, 2006, Joly et al. 2006, Conner et al. 2007, Habib et al. 2007, Wasserberg et al. 2009, Lawson and Song 2010).

Comparatively little is known about the ecology of CWD in free-ranging elk. Limited data suggest some differences in the ecology of CWD between deer and elk. Disease progression appears longer and more variable in captive elk (12-40 months) than mule deer (20-25 months) (Williams et al. 2002, Hamir et al. 2006); and prevalence is generally lower in elk (1-3%) than mule deer (1-30%) (Miller et al. 2000, Miller et al. 2008, Colorado Division of Wildlife (CDOW) 2009). However, similarities in population effects may be greater than previously recognized. Recent work found prevalence of CWD to exceed 10% in elk from RMNP (Monello et al. in review) and CWD-caused mortality can interact with predation and low recruitment to limit elk population growth (Sargeant et al. 2011). These findings raise concern for the effects of CWD on elk population viability.

We conducted a three-year radio-telemetry study to assess rates of transmission and effects of CWD in free-ranging elk from RMNP. Our specific objectives were to 1) determine incidence of CWD (proportion of susceptible elk infected per year), 2) measure effects of CWD on elk

survival, 3) assess the importance of CWD as a source of mortality, and 4) estimate the relationship between prevalence of CWD and the rate of population growth in this elk herd.

Materials and Methods

Study Area and Population

The elk winter range in RMNP occurs at 2,400-2,800 m in elevation and forms continuous winter habitat with the Town of Estes Park, Colorado (105°58'W, 40°36'N). Elk generally exhibit site fidelity to either the park or town areas (Lubow et al. 2002). Hunting is prohibited in RMNP and most of Estes Park, although elk from both areas occasionally move into locations where they are subject to harvest. The park and town sub-populations display independent population dynamics. At their peak in the winter of 1999-2000, the park sub-population size was approximately 1,000 elk with a density of 15-110 elk/km² (Singer et al. 2002) and the town sub-population size was approximately 2,400 elk (Lubow et al. 2002). Winter estimates have declined by ~30% since that time, with 600-700 elk inhabiting the park and 1,200-1,600 elk inhabiting the town during the course of our three year study (T. Johnson, unpublished data). There are likely multiple factors that have caused the recent population declines, including increased hunter harvest adjacent to the park (years 2004-2009), the implementation of a population reduction program in RMNP in 2008 (National Park Service 2007), dispersal to adjacent winter range areas, and effects of CWD (T. Johnson, personal communication; Monello et al. in review).

Prevalence of CWD in our study elk was estimated to be 12.9% in 2008 (95% credible limits [CL] = 8.0, 19.1; Monello et al. in review). There are no formal estimates for elk that inhabit areas in and around the Town of Estes Park during winter, but test results from this general

region indicate that 2.4% (n = 9) of 382 harvested elk tested positive for CWD from 2006-2008 (CDOW 2009).

Study Design

Elk were initially captured, sampled, fitted with a radio transmitter, and released during the winter of 2007-2008 (hereafter referred to as initial captures). Elk that tested positive for CWD via immunohistochemistry (IHC) of rectal biopsies were recaptured within 55 days of their initial capture, euthanized, and necropsied. We resampled, euthanized, and necropsied 20-34 of the remaining radio-collared elk in each of the following three winters (hereafter referred to as resampled elk) in conjunction with elk management at RMNP and other research purposes (e.g., Monello et al. in review). Elk were monitored from 2008-2010 to assess annual survival and sources of mortality. Elk deaths that occurred during the course of this study were investigated in the field and collected to determine cause of mortality.

In a separate effort, we also collected 1) non-collared elk carcasses and 2) non-collared elk that were euthanized because they were exhibiting end-stage signs of CWD (emaciation, depression, excessive salivation; Miller et al. 1998). These elk were opportunistically collected on the winter range in RMNP from 2001-2009 and tested for CWD. These are included here because they provide additional insights into age- and sex-specific trends of CWD mortality in RMNP.

Survival and Cause-Specific Mortality

Elk capture and sampling techniques for this study have been previously described (Monello et al. in review) and are only briefly covered here. During initial capture efforts, we anesthetized

adult female elk that did not show end-stage signs of CWD, attached a radio-collar, collected two rectal biopsies that were stored in 10% neutral buffered formalin, and reversed the effects of immobilization drugs. Resampled elk were processed in the same manner, with the exception that once samples were collected, elk were euthanized and necropsied within eight hours of death at the Colorado State University (CSU) Veterinary Diagnostic Laboratory (Fort Collins, Colorado, USA).

Each elk was fitted with a very high frequency (VHF) radio collar (ATS, Isanti, Minnesota, USA) that activated a mortality signal after eight hours of non-movement. The status (alive, dead, not detected) of each elk was checked at least once per week from January 2008 to December 2010. We minimized harvest mortalities in the last two years of the study by sending letters to licensed hunters that requested them to not harvest radio-collared elk. When possible, we collected the entire carcass of elk that died during the study and necropsied them at the CSU Veterinary Diagnostic Laboratory. When we could not collect the entire carcass, we removed the obex and retropharyngeal lymph nodes and stored them in 10% neutral buffered formalin.

Causes of mortality were investigated in the field and during necropsy, and recorded as hunter harvest, CWD, predation, vehicle collision, or unknown. Harvested elk were identified when collars and available postmortem samples (e.g., obex) were returned to the National Park Service or Colorado Division of Wildlife by a hunter. Mountain lion (*Puma concolor*) kills were identified by cached carcasses and puncture wounds and hemorrhage on the throat or back of the neck (Shaw et al. 2007). We considered CWD to be cause of death when there were no other potential causes identified during field or lab necropsies, and the elk was infected with PrP^{CWD} and had an obex score ≥ 8 (described below). Carcasses that were too decomposed or scavenged to determine cause of death were classified as unknown, even if PrP^{CWD} was detected. Once an

elk was euthanized or found dead, a primary incisor was removed to determine age by cementum analysis (Matson's Lab, Milltown, Montana, USA).

Opportunistic Collections

Elk with end-stage signs of CWD (CWD-suspects) and carcasses found on the winter range were reported by park staff or visitors. None of these elk or carcasses were radio-collared and all CWD-suspects were euthanized by RMNP staff. Collection procedures were the same as for collared elk. In most cases, an incisor was collected to determine age by cementum analysis.

For carcasses, we only included dead elk that had usable diagnostic samples for CWD (i.e., obex or retropharyngeal lymph nodes). Causes of mortality were investigated in the field and during necropsy, and recorded as unknown, vehicle collision, predation, harvest (legal and illegal), or gored. Sources of mortality were classified in a similar manner as for collared elk, except that harvest was determined by the presence of gunshot wounds. We did not include CWD as a potential cause of death because carcasses were frequently too scavenged to rule out other sources of mortality and we did not score the obex.

CWD Diagnostics

The obex and medial retropharyngeal lymph nodes were removed and stored in 10% neutral buffered formalin for CWD diagnostics. Antemortem rectal biopsies and postmortem samples were assayed for PrP^{CWD} by IHC as previously described (Spraker et al. 2006). We considered IHC staining of any postmortem sample (obex or retropharyngeal lymph node) to be a definitive diagnosis of CWD. Obex samples that contained coarse, red, granular, or particulate deposits surrounding neurons and scattered in the neuropil were considered CWD positive. Obex samples

were scored on a scale from 0 (no PrP^{CWD} detected in brain stem) to 10 (elk in terminal stage of disease) as previously described (Spraker et al. 2010). Lymph nodes and rectal mucosa tissue were considered to have PrP^{CWD} if follicles or nerves had coarse, bright red, granular material (Spraker et al. 2009).

Analyses

We conducted all analyses in a Bayesian framework. To calculate incidence (annual probability of infection), we assumed all radio-collared elk were susceptible (uninfected) at the start of the study except for those that 1) tested positive for CWD via rectal biopsy or 2) were infected by PrP^{CWD} but misdiagnosed as susceptible during initial capture efforts (as previously described; Monello et al. in review). Incidence was estimated as

$$\Pr(\emptyset|y, n) \propto \prod_{t=1}^3 \text{binomial}(y_t|(1 - \emptyset)^t, n_t) \text{beta}(1,1)$$

where n_t = the number of elk tested for CWD in year t , y_t = the number of elk that tested negative for CWD in year t , and $\emptyset$ = the probability that an elk becomes infected by CWD during t to $t+1$. The number of elk tested per year (n_t) consisted of collared elk that were resampled and euthanized or died during the study and had viable postmortem samples that could be tested for CWD. Only elk that contained PrP^{CWD} in the obex or retropharyngeal lymph node were considered to be infected.

Survival and cause-specific mortality of CWD was calculated from January to December for each year of the study. We did not include any elk that died within two weeks of their initial capture. Harvested elk were included but censored in the month they died because our request to licensed hunters to not harvest elk with radio-collars likely prevented an accurate representative

estimate of harvest mortality. Resampled elk that were euthanized for research purposes were also censored; however, we did include elk that were euthanized because they were exhibiting signs consistent with end-stage CWD. Within each year of the study, the probability of monthly survival (θ) was estimated as

$$P(\theta|y, n) \propto \prod_{i=1}^{12} \text{binomial}(y_i|\theta, n_i) \text{beta}(\theta|1,1),$$

where y_i is the number of elk that are alive in the i th month and n_i is the total number of elk (dead or alive) detected, excluding harvested and resampled/euthanized elk. Although seven elk were undetected for 1-4 months, none of the 136 radio-collared elk disappeared or died while they were unaccounted for during the three-year study. Thus, any individuals that were not detected were considered alive for the purposes of these analyses. Annual survival (β) for each year t was then calculated as $\beta_t = \exp(\log(\theta_t)*12)$.

We estimated the relationship between prevalence of CWD and the intrinsic rate of population growth (λ) in this elk herd using the dominant eigenvalue from a transition matrix (Dulberger et al. 2010). The matrix was a discrete-time, female-only model that requires annual survival rates for susceptible and infected elk. We estimated survival of these two groups using the last 18 months of data from this study (June 2009 to December 2010) and the same analyses as above. We only included elk that were resampled and euthanized or died and had viable postmortem samples during the last 18 months. Any elk found to have PrP^{CWD} in the obex or a retropharyngeal lymph node was considered to be infected by PrP^{CWD} for the entire 18 month period. We chose to only use the last 18 months to estimate survival because data from the first half of this study would have resulted in unrealistically high survival estimates for infected elk, due to removal of all known individuals infected with PrP^{CWD} at the outset of the study (i.e., elk

infected with PrP^{CWD} in the first 18 months of the study would all be in an early disease state).
These analyses are fully described in Appendix 1.

Results

Survival, Mortality, and Population Growth

We captured, sampled, and placed radio-collars on 136 cow elk from December 2007 to March 2008. Age at initial capture ranged from 2 to 21 years old. The majority ($n = 79$) of collared elk were resampled and euthanized for research and management purposes, 9 of which tested positive for CWD (Table 1). In addition, 13 elk were removed at the start of the study because they tested positive for CWD by rectal biopsy, 9 elk died of CWD during the study, and 1 harvested elk tested positive for CWD (Table 1). Of the 9 elk that died of CWD during the study, 4 were misdiagnosed as susceptible during initial capture efforts (Monello et al. in review). Overall, 32 of 136 elk tested positive for CWD during the course of the three-year study. Incidence of CWD in radio-collared elk was calculated to be 0.08 (CL = 0.05, 0.12).

Mean survival of adult female elk declined from 0.97 in 2008 (CL = 0.93, 0.99) to 0.85 in 2010 (CL = 0.75, 0.93) (Figs. 1, 2). Although uncertainty increased annually due to the removal of elk for research and management purposes, the 95% credible limits for survival in 2008 and 2010 did not overlap (Fig. 1). Elk primarily died from October to April, and no mortalities were observed in July or August (Fig. 2).

The decline in annual survival of study elk was concurrent with an increase in mortalities due to CWD; 1 of 121 elk died of CWD in 2008, but 5 of 56 elk died of CWD in 2010 (Fig. 3). Cause-specific rates of mortality for CWD were 0.02 (CL = 0.00, 0.05) in 2008, 0.06 (CL = 0.02, 0.11) in 2009, and 0.11 (CL = 0.04, 0.21) in 2010. Conversely, the number and proportion of elk

that were harvested declined after we requested hunters to not remove collared elk in 2009 and 2010. Other sources made up approximately 1/3 of annual mortality and included mountain lion predation, a vehicle collision, and unknown causes (Fig. 3). Four of seven elk with an unknown cause of death did not have a usable postmortem sample and could not be tested for CWD.

Using data from the last 18 months of our radio-telemetry study, annual survival of non-infected elk was estimated to be 0.95 (CL = 0.88, 0.99, $n=55$), while annual survival of infected elk was estimated to be 0.55 (CL = 0.33, 0.76, $n=15$). We estimated λ to be 0.97 (CL = 0.90, 1.06) using the 2008 estimate for prevalence of CWD (12.9%). The transition matrix predicted that the population will decline (i.e., $\lambda < 1.0$) once prevalence of CWD exceeds 5% in females (Fig. 4). However, the credible limits of this prediction were relatively large and prevalence may increase to 20% before CWD causes population declines in this elk herd (Fig. 4).

Opportunistic Collections

We opportunistically collected 78 non-collared elk carcasses on the winter range, 53 (68%) which had no identifiable proximate cause of death (Table 2). More than half of the carcasses in the unknown category (27 of 53) were infected with PrP^{CWD} , and all but one elk carcass that tested positive for CWD was in the unknown category. Overall, 4 of 25 (16%) male carcasses and 24 of 53 (45%) female carcasses tested positive for CWD. Among 20 CWD-suspects that were euthanized, 16 tested positive for CWD (15 females, 1 male) (Table 2).

CWD and Age

Among all elk, we did not detect any upper age limit to CWD infection; 4 of 9 collared elk that died of CWD and 4 of 26 opportunistically collected elk were ≥ 10 years old (Tables 1, 2).

274 Additionally, radio-collared adult elk that tested positive for CWD at the start of the study via
275 rectal biopsy (median = 3, mean age = 5.8, CL = 4.6, 7.2) were almost half the age of radio-
276 collared adult elk that died of CWD (median = 9, mean age = 10.2, CL = 8.3, 12.4). Non-
277 collared CWD-suspects that were euthanized did not tend to be older (Table 2); however, when
278 we restricted analyses to only include adult females (to be consistent with data from radio-
279 collared elk), median age at death increased (median = 8, mean age = 8.1, CL = 6.2, 10.4) and
280 none of the CWD-suspects were less than 5 years old.

281 282 **Discussion**

283 Our findings indicate cow elk survival - a metric that exhibits low variance but has the
284 greatest impact on elk population dynamics (Nelson and Peek 1982, Raithel et al. 2007) - can be
285 destabilized and negatively affected by CWD. Following removal of all radio-collared elk that
286 tested positive for CWD, we observed consistent annual increases in CWD-caused mortality and
287 corresponding declines in survival during each year of the telemetry study. The survival
288 estimate of 0.85 (CL = 0.75, 0.93) observed in the last year of our study is of particular concern
289 because range-wide research on elk indicates that the majority (>80%) of herds decline when
290 cow survival is ≤ 0.85 (Raithel et al. 2007). Markedly lower survival rates due to CWD appear
291 to be a recent phenomena in this population, as prior estimates of annual cow survival in RMNP
292 that included harvest averaged 0.93 ± 0.023 (SE) from 1995 to 1998 (Lubow et al. 2002). We
293 recognize that we have only one year of data where survival is < 0.90 , and thus our inferences
294 are limited with regard to future projections of CWD-related impacts on this elk herd. However,
295 we suggest our annual estimates of survival are likely too high versus too low because we
296 selectively removed elk that tested positive for CWD and censored hunter harvest mortalities. If

we did overestimate survival, effects of CWD on this elk population are even greater than our results indicate here.

This is the first study to demonstrate the potential for CWD to cause a population decline in elk. We found λ of elk in our study was lower than it would have been if CWD was absent or occurred at lower prevalence. This suggests CWD has contributed to the recent elk population declines observed in RMNP and warrants further research. Other empirical studies support this conclusion, as CWD has been found to reduce adult survival and contribute to population declines that exceed 50% in mule deer (Miller et al. 2008) and reduce λ in elk (Sargeant et al. 2011). The transition matrix predicts that, even in the absence of hunting or other sources of mortality, CWD alone will induce population declines once prevalence reaches 5-20%, although this relationship may be obscured by the dynamics and dispersal patterns of adjacent elk herds. This outcome is largely contingent on the survival rate of infected elk, which had a wide credible interval (0.33 - 0.76); and calf:cow ratios, which have been low for the past 15 years, including during recent declines (20-30 calves per 100 cows; Lubow et al. 2002, T. Johnson, unpublished data). If the annual survival of infected elk is higher or calf:cow ratios increase, as would be expected in a herd that is no longer limited by their forage base (Lubow et al. 2002), the population may be able to sustain higher rates of CWD without exhibiting population declines.

Primary routes of prion transmission and the relationship between incidence of CWD and population density in free-ranging ungulates remain unresolved (Conner et al. 2008). However, the long-term trajectory and impacts of CWD on population size will also depend on how rapidly the disease progresses in elk. We found elk that tested positive for CWD via rectal biopsy were on average five years younger than elk that died of CWD, and none of our collared elk died of CWD prior to six years of age. This difference was not due to age-related follicle counts, which

had relatively little bearing on CWD diagnostics (Monello et al. in review). Length of disease course cannot be inferred from our results, but it does suggest the possibility that free-ranging adult female elk that are naturally infected by PrP^{CWD} may live longer than captive elk that are orally inoculated with PrP^{CWD} (Williams et al. 2002, Hamir et al. 2006). A prolonged disease course in elk could lessen the impact of CWD on population size relative to deer herds (e.g., Gross and Miller 2001, Wasserburg 2009), but may also increase infectious contacts, environmental contamination, and long-term prevalence levels. Estimates and associated uncertainties of disease progression times and survival should be further refined with research that monitors free-ranging elk known to be infected with PrP^{CWD} via rectal biopsy.

The cause-specific mortality rate from CWD in the last year of the study (0.11) and proportion of opportunistically collected carcasses that tested positive for CWD (> 0.35) underscore the importance of disease in this elk population. Aside from human harvest, only the combined effects of predation by wolves (*Canis lupus*), mountain lions, and bears (*Ursus* spp.) has been found to cause a similar rate of mortality for elk (annual mortality due to combined predation = 0.09, Evans et al. 2006; other rates from Unsworth et al. 1993, Ballard et al. 2000, Eberhardt 2002, Sargeant et al. 2011, Webb et al. 2011). However, the potential effects of wolf predation and CWD on the age structure of elk herds are profoundly different; in healthy elk populations, wolves primarily kill calves or elk older than 12 years of age (Carbyn 1983, Wright et al. 2006), whereas CWD frequently infects and kills elk that are of prime breeding age (see also Sargeant et al. 2011). Hunting also tends to remove prime-age elk (Carbyn 1983, Wright et al. 2006), but harvest rates can be adjusted to allow for population recovery. Other sources of non-CWD mortality also vary due to annual weather patterns and predator-prey population dynamics (Carbyn 1983, Boyd et al. 1994, Mech et al. 2001). In contrast, the consistent effects

of CWD will not allow for periods of recovery and thus has a greater potential to cause long-term population declines relative to other sources of mortality, particularly if prion transmission rates are independent of population density (e.g., Almberg et al. 2011).

These results clearly indicate that CWD poses a risk, at least for high-density elk populations. We found CWD-caused mortality can exceed natural sources and, at the rates observed in our study, will likely result in either an unsustainable population or necessitate declines in harvest. Reductions in harvest will only be a temporary solution to prevent population declines if CWD continues to increase, as our modeling results indicate CWD alone is capable of causing large declines in λ . It is critical to monitor the trajectory of CWD in this population and further reduce the uncertainty of the metrics used in our study to determine management options and the full impact this disease may have on elk populations.

Management Implications

A critical need exists to examine if CWD displays similar patterns in other elk herds, for although we observed relatively high prevalence in a high-density elk population that is presumably lightly harvested, there is evidence that hunted elk populations in Southeastern Wyoming also display relatively high prevalence (<http://gf.state.wy.us/services/education/cwd>). Although data for elk are limited, empirical research and model simulations have consistently indicated that CWD will be difficult to eliminate or reduce in deer populations once it is established (e.g., Gross and Miller 2001, Conner et al. 2007, Almberg et al. 2011). Observations indicate that hunting or the absence of localized refugia and attractants may slow the increase of CWD in deer and reduce its overall prevalence (Wolfe et al. 2002, Farnsworth et al. 2005,

Wasserberg et al. 2009), but the cause of these differences are unknown and large scale reductions of deer have failed to reduce CWD (Conner et al. 2007).

Several potential differences between CWD ecology in elk and deer may allow a test-and-cull strategy to be just as or even more effective in elk versus deer (e.g., Wolfe et al. 2004), especially in our study location. First, cow elk occur in larger herds that are more easily found during winter than deer. This could facilitate capture and removal efforts, especially in herds that are habituated to humans. Second, our data (Table 2) and prior work (see CWD-caused mortality rates; Sargeant et al. 2011) found no evidence that male elk have higher prevalence or CWD-caused mortality rates than females. If confirmed, this means that management efforts can largely be concentrated on cow elk, as bull elk live for shorter periods of time and pose no increased risk of disease transmission relative to females. Finally, prevalence is typically lower in elk than deer and the length of disease course may be longer in elk than deer (Williams et al. 2002, Hamir et al. 2006). This suggests removal of infected elk will be more beneficial than removal of infected deer because each elk represents a greater per capita source of transmission that may remain on the landscape for a longer period of time. Similarly, selective removal of CWD-positive elk by wolves could be effective in truncating the period of prion shedding and resultant disease transmission opportunities (Wild et al 2011). However, a better estimate of the time to death after antemortem CWD detection is critical to estimating the potential benefits of any selective removal strategy.

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Literature Cited

- Almberg, E.S., P.C. Cross, C.J. Johnson, D.M. Heisey, and B.J. Richards. 2011. Modeling routes of chronic wasting disease transmission: environmental prion persistence promotes deer population declines and extinction. *PloS ONE* 6:e19896.
- Arneberg, P., A. Skorping, B. Grenfell, and A. F Read. 1998. Host densities as determinants of abundance in parasite communities. *Proceedings of the Royal Society of London B* 265:1283-1289.
- Ballard, W.B., H.A. Whitlaw, B.F. Wakeling, R.L. Brown, J.C. deVos, Jr., and M.C. Wallace. 2000. Survival of female elk in Northern Arizona. *Journal of Wildlife Management* 64:500-504.
- Bonneaud, C., S.L. Balenger, A.F. Russell, J. Zhang, G.E. Hill, and S.V. Edwards. 2011. Rapid evolution of disease resistance is accompanied by functional changes in gene expression in a wild bird. *Proceedings of the National Academy of Sciences* 108: 7866-7871.
- Boyd, D.K., R.R. Ream, D.H. Pletscher, and M.W. Fairchild. 1994. Prey taken by colonizing wolves and hunters in Glacier National Park area. *Journal of Wildlife Management* 58:289-295.

410 Carbyn, L.N. 1983. Wolf predation on elk in Riding Mountain National Park, Manitoba. *Journal*
 411 *of Wildlife Management* 47:963-976.

412 Colorado Division of Wildlife. 2009. Chronic wasting disease in Colorado, 2006-2008. Colorado
 413 Division of Wildlife Report. 5 pp. [<http://wildlife.state.co.us/hunting/biggame/cwd/>]

414 Conner, M.M., M.W. Miller, M.R. Ebinger, and K.P. Burnham. 2007. A meta-baci approach for
 415 evaluating management intervention on chronic wasting disease in mule deer. *Ecological*
 416 *Applications* 17:140-153.

417 Conner, M.M., M.R. Ebinger, J.A. Blanchong, and P.C. Cross. 2008. Infectious diseases in
 418 cervids of North America. *Annals of the New York Academy of Sciences* 1134:146-172.

419 Dulberger, J., N.T. Hobbs, H.M. Swanson, C.J. Bishop, and M.W. Miller. 2010. Estimating
 420 chronic wasting disease effects on mule deer recruitment and population growth. *Journal of*
 421 *Wildlife Diseases* 46:1086-1095.

422 Eberhardt, L.L. 2002. A paradigm for population analysis of long-lived vertebrates. *Ecology*
 423 83:2841-2854.

424 Evans, S., L.D. Mech, P.J. White, and G.A. Sargeant. 2006. Survival of adult female elk in
 425 Yellowstone following wolf restoration. *Journal of Wildlife Management* 70:1372-1378.

426 Farnsworth, M.L., L.L. Wolfe, N.T. Hobbs, K.P. Burnham, E.S. Williams, D.M. Theobald, M.
 427 M. Conner, and M.W. Miller. 2005. Human land use influences chronic wasting disease
 428 prevalence in mule deer. *Ecological Applications* 15:119–126.

429 Farnsworth, M.L., J.A. Hoeting, N.T. Hobbs, and M.W. Miller. 2006. Linking chronic wasting
 430 disease to mule deer movement scales: a hierarchical Bayesian approach. *Ecological*
 431 *Applications* 16:1026-1036.

432 Gross, J.E., and M.W. Miller. 2001. Chronic wasting disease in mule deer: disease dynamics and
 433 control. *Journal of Wildlife Management* 65:205-215.

434 Georgsson, G., S. Sigurdarson, and P. Brown. 2006. Infectious agent of sheep scrapie may
 435 persist in the environment for at least 16 years. *Journal of General Virology* 87:3737-3740.

436 Habib, T.J., E.H. Merrill, M.J. Pybus, and D.W. Coltman. Modelling landscape effects on
 437 density-contact rate relationships of deer in eastern Alberta: implications for chronic wasting
 438 disease. *Ecological Modelling* 222:2722-2732.

439 Hamir, A.N., T. Gidlewski, T.R. Spraker, J.M. Miller, L. Creekmore, M. Crocheck, T. Cline, and
 440 K.I. O'Rourke. 2006. Preliminary observations of genetic susceptibility of elk (*Cervus*
 441 *elaphus nelsoni*) to chronic wasting disease by experimental inoculation. *Journal of*
 442 *Veterinary Investigation* 18:110-114.

443 Haukisalmi, V., and H. Henttonen. 1990. The impact of climatic factors and host density on the
 444 long-term population dynamics of vole helminths. *Oecologia* 83:309-315.

445 Huwer, S. 2007. Elk management plan. Data analysis unit E-9. Colorado Division of Wildlife
 446 Report. 66 pp.

447 Joly, D.O., M.D. Samuel, J.A. Langenberg, J.A. Blanchong, C.A. Batha, R.E. Rolley, D.P.
 448 Keane, and C.A. Ribic. 2006. Spatial epidemiology of chronic wasting disease in Wisconsin
 449 white-tailed deer. *Journal of Wildlife Diseases* 42:578-588.

450 Krasnov, B., I. Khokhlova, and G. Shenbrot. 2002. The effect of host density on ectoparasite
 451 distribution: an example of a rodent parasitized by fleas. *Ecology* 83:164-175.

452 Lawson, A., and H. Song. 2010. Semiparametric space-time survival modeling of chronic
 453 wasting disease in deer. *Environmental and Ecological Statistics* 17:559-571.

454 Lubow, B.C., F.J. Singer, T.L. Johnson, and D.C. Bowden. 2002. Dynamics of interacting elk
 455 populations within and adjacent to Rocky Mountain National Park. *Journal of Wildlife*
 456 *Management* 66:757-775.

457 McCallum, H. 2008. Tasmanian devil facial tumour disease: lessons for conservation biology.
 458 *Trends in Ecology and Evolution* 23:631-637.

459 Mech, D.L., D.W. Smith, K.M. Murphy, and D.R. MacNulty. 2001. Winter severity and wolf
 460 predation on a formerly wolf-free elk herd. *Journal of Wildlife Management* 65:998-1003.

461 Miller, M.W., and E.S. Williams. 2003. Horizontal prion transmission in mule deer. *Nature*
 462 425:35-36.

463 Miller, M.W., E.S. Williams, C.W. McCarty, T.R. Spraker, T.J. Kreeger, C.T. Larsen, and E.T.
 464 Thorne. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado
 465 and Wyoming. *Journal of Wildlife Diseases* 36:676-690.

466 Miller, M.W., E.S. Williams, N.T. Hobbs, and L.L. Wolfe. 2004. Environmental sources of prion
 467 transmission in mule deer. *Emerging infectious diseases* 10:1003-1006.

468 Miller, M.W., H.W. Swanson, L.L. Wolfe, F.G. Quartarone, S.L. Huwer, C.H. Southwick, and
 469 P.M. Lukacs. 2008. Lions and prions and deer demise. *PloS One* 3:e4019.

470 Miller, M.W., M.A. Wild, and E.S. Williams. 1998. Epidemiology of chronic wasting disease in
 471 captive Rocky Mountain elk. *Journal of Wildlife Diseases* 34:532-538.

472 Miller, M.W., N.T. Hobbs, and S.J. Tavener. 2006. Dynamics of prion disease transmission in
 473 mule deer. *Ecological Applications* 16:2208-2214.

474 National Park Service. 2007. Elk and vegetation management plan, Rocky Mountain National
 475 Park, Colorado. Final Environmental Impact Statement, Estes Park, Colorado, 530 pp.

476 Nelson, L.J., and J.M. Peek. 1982. Effect of survival and fecundity on rate of increase of elk.
 477 Journal of Wildlife Management 46:535-540.

478 Quimby, D. C., and J. E. Gaab. 1957. Mandibular dentition as an age indicator in Rocky
 479 Mountain elk. Journal of Wildlife Management 21:134-153.

480 Raithel, J.D., M.J. Kauffman, and D.H. Pletscher. 2007. Impact of spatial and temporal variation
 481 in calf survival on the growth of elk populations. Journal of Wildlife Management 71:795–
 482 803.

483 Sargeant, G.A., D.C. Weber, D.E. Roddy. 2011. Implications of chronic wasting disease, cougar
 484 predation, and reduced recruitment for elk management. Journal of Wildlife Management
 485 75:171-177.

486 Schaubert, E.M., D.J. Storm, and C.K. Nielsen. 2007. Effects of joint space use and group
 487 membership on contact rates among white-tailed deer. Journal of Wildlife Management
 488 71:155-163.

489 Shaw, H.G., P. Beier, M. Culver, and M. Grigione. 2007. Puma field guide. The Cougar
 490 Network. [<http://www.cougarnet.org/Assets/pumafieldguide.pdf>]

491 Singer, F.J., L.C. Zeigenfuss, B. Lubow, and M.J. Rock. 2002. Ecological evaluation of potential
 492 overabundance of ungulates in U.S. National Parks: a case study. Pages 205-248 in F.J.
 493 Singer and L.C. Zeigenfuss, compilers. Ecological evaluation of the abundance and effects
 494 of elk herbivory in Rocky Mountain National Park, Colorado 1994-1999. United States
 495 Geological Survey Open File Report 02-208.

496 Spraker, T.R., K.C. VerCauteren, T. Gidlewski, D.A. Schneider, R. Munger, A. Balachandran,
 497 and K.I. O'Rourke. 2009. Antemortem detection of PrPCWD in preclinical, ranch-raised

498 Rocky Mountain elk (*Cervus elaphus nelsoni*) by biopsy of the rectal mucosa. Journal of
 499 Veterinary Diagnostic Investigation 21:15-24.

500 Spraker, T.R., K.I. O'Rourke, T. Gidlewski, J.G. Powers, J.J. Greenlee, and M.A. Wild. 2010.
 501 Detection of the abnormal isoform of the prion protein associated with chronic wasting
 502 disease in the optic pathways of the brain and retina of Rocky Mountain elk (*Cervus elaphus*
 503 *nelsoni*). Veterinary Pathology 47:536-546.

504 Spraker, T.R., M.W. Miller, E.S. Williams, D.M. Getzy, W.J. Adrian, G.G. Schoonveld, R.A.
 505 Spowart, K.I. O'Rourke, J.M. Miller, and P.A. Merz. 1997. Spongiform encephalopathy in
 506 free-ranging mule deer (*Odocoileus hemionus*), white-tailed deer (*Odocoileus virginianus*)
 507 and Rocky Mountain elk (*Cervus elaphus nelsoni*) in north central Colorado. Journal of
 508 Wildlife Diseases 33:1-6.

509 Spraker, T.R., R.R. Zink, B.A. Cummings, C.J. Sgurdson, M.W. Miller, and K.I. O'Rourke.
 510 2002. Distribution of protease-resistant prion protein and spongiform encephalopathy in free-
 511 ranging mule deer (*Odocoileus hemionus*) with chronic wasting disease. Veterinary
 512 Pathology 39:546-556.

513 Spraker, T.R., T.L. Gidlewski, A. Balachandran, K.C. VerCauteren, L. Creekmore, and R.D.
 514 Munger. 2006. Detection of PrPCWD in postmortem rectal lymphoid tissues in Rocky
 515 Mountain elk (*Cervus elaphus nelsoni*) infected with chronic wasting disease. Journal of
 516 Veterinary Diagnostic Investigation 18:553-557.

517 Unsworth, J.W., L. Kuck, M.D. Scott, and E.O. Garton. 1993. Elk mortality in the Clearwater
 518 Drainage of Northcentral Idaho. Journal of Wildlife Management 57:495-502.

519 Wasserberg, G., E.E. Osnas, R.E. Rolley, and M.D. Samuel. 2009. Host culling as an adaptive
 520 management tool for chronic wasting disease in white-tailed deer: a modelling study. *Journal*
 521 *of Applied Ecology* 46:457-466.

522 Webb, S.L., M.R., Dzialak, J.J. Wondzell, S.M. Harju, L.D. Hayden-Wing, and J.B. Winstead.
 523 2011. Survival and cause-specific mortality of female Rocky Mountain elk exposed to human
 524 activity. *Population Ecology* 53:483-493.

525 Wild, M.W., N.T. Hobbs, M.S. Graham, and M.W. Miller. 2011. The role of predation in disease
 526 control: a comparison of selective and nonselective removal on prion disease dynamics in
 527 deer. *Journal of Wildlife Diseases* 47:78-93.

528 Williams, E.S., and M.W. Miller. 2002. Chronic wasting disease in deer and elk in North
 529 America. *In* Bengis, R.G. (ed.): *Infectious diseases of wildlife: detection, diagnosis, and*
 530 *management*. Office of International Epizootics Scientific and Technical Review 21:305-316.

531 Williams, E.S., and S. Young. 1980. Chronic wasting disease of captive mule deer: a spongiform
 532 encephalopathy. *Journal of Wildlife Diseases* 16:89-98.

533 Williams, E.S., J.K. Kirkwood, and M.W. Miller. 2001. Transmissible spongiform
 534 encephalopathies. Pages 292-301 *in* Williams, E.S., and I.K. Barker (eds.): *Infectious*
 535 *diseases of wild mammals*. Iowa State University Press, Ames, IA.

536 Williams, E.S., M.W. Miller, T.J. Kreeger, R.H. Kahn, and E.T. Thorne. 2002. Chronic wasting
 537 disease of deer and elk: A review with recommendations for management. *Journal of*
 538 *Wildlife Management* 66:551-563.

539 Wright, G.J., R.O. Peterson, D.W. Smith, and T.O. Lemke. 2006. Selection of Northern
 540 Yellowstone elk by gray wolves and hunters. *Journal of Wildlife Management* 70:1070-1078.

541 Wolfe, L.L., M.M. Conner, T.H. Baker, V.J. Dreitz, K.P. Burnham, E.S. Williams, N.T. Hobbs,
542 and M.W. Miller. 2002. Evaluation of antemortem sampling to estimate chronic wasting
543 disease prevalence in free-ranging mule deer. *Journal of Wildlife Management* 66:564-573.
544 Wolfe, L.L., M.W. Miller, and E.S. Williams. 2004. Feasibility of “test-and-cull” for managing
545 chronic wasting disease in urban mule deer. *Wildlife Society Bulletin* 32:500-505.

Table 1. Fate of 136 radio-collared adult female elk and median age at time of death. Five elk did not have any usable postmortem samples for chronic wasting disease (CWD) diagnosis (1 in harvested category, 4 in unknown category).

Cause of death	Number	Median age (range)
Resampled and euthanized	79	9 (3 – 19)
CWD-positive and euthanized at start of study	13	3 (2 – 17)
Harvested	10	9 (4 – 14)
Died of CWD ¹	9	9 (6 – 16)
Unknown ¹	7	11 (6 – 19)
Other (vehicle collision or predator kill)	3	18 (8 – 21)
Alive at end of three-year study	15	NA

¹Includes one of two elk that were euthanized because it was exhibiting end-stage signs of CWD (only one tested positive for CWD, the other was emaciated and unable to move)

Table 2. Sources of mortality and median age of 98 opportunistically collected elk from the winter range in Rocky Mountain National Park, 2001-2009. None of these were part of the radio-telemetry study and we only included elk that had a usable postmortem sample for diagnosis of chronic wasting disease (CWD).

Cause of death	Males	Females	Median age (range; <i>n</i>)
Unknown, CWD-negative	9	17	8 (1 – 18; <i>n</i> = 16)
Unknown, CWD-positive	3	24	5 (2 – 12; <i>n</i> = 16)
Vehicle collision	4	8	2 (1 – 15; <i>n</i> = 6)
Predation (one male was CWD-positive)	3	4	3 (1 – 8; <i>n</i> = 4)
Harvested or poached	6	0	NA (1, 5; <i>n</i> = 2)
Euthanized CWD-suspect, infected	1	15	6 (1 – 12; <i>n</i> = 10)
Euthanized CWD-suspect, not infected	1	3	NA (6, 15; <i>n</i> = 2)

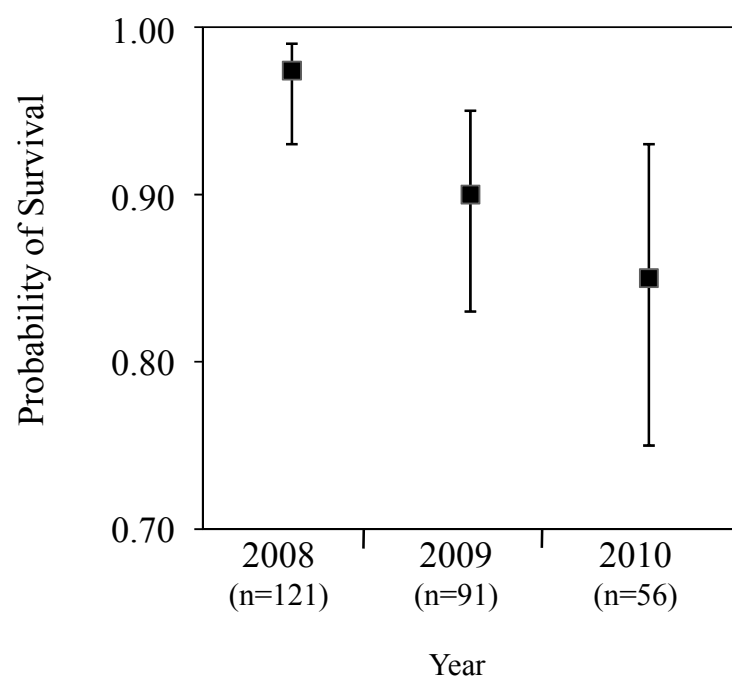
Figure Captions

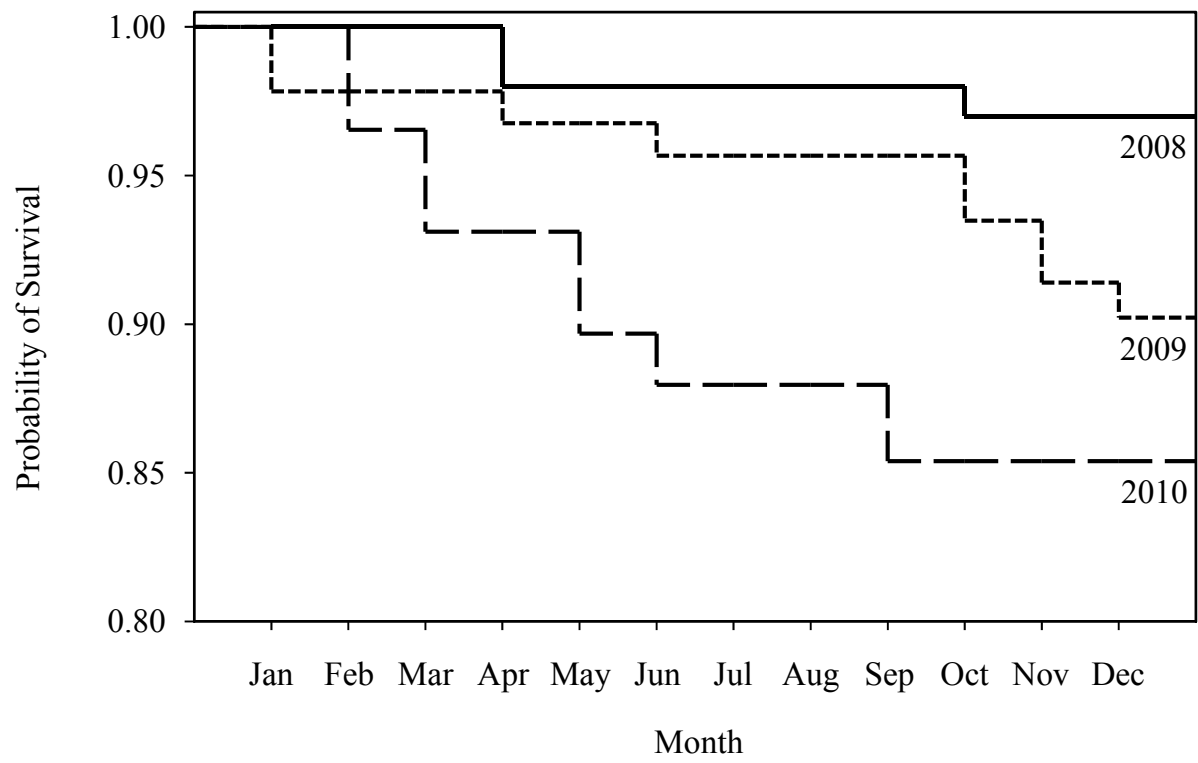
Figure 1. Annual survival rate ($\pm$ 95% credible limits) of adult female elk in Rocky Mountain National Park following removal of all elk known to have chronic wasting disease in 2008 ($n = 13$, disease status determined with rectal biopsies). Harvest mortality and management/research removals are censored ($n =$ number of elk included in analyses after management/research removals in January).

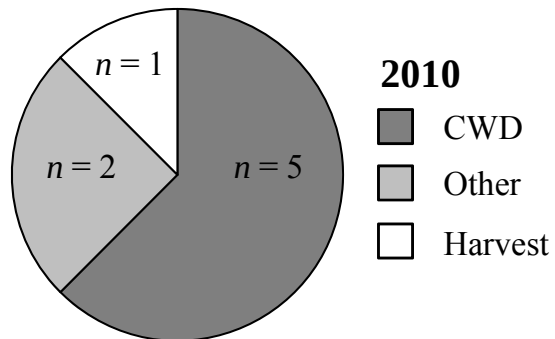
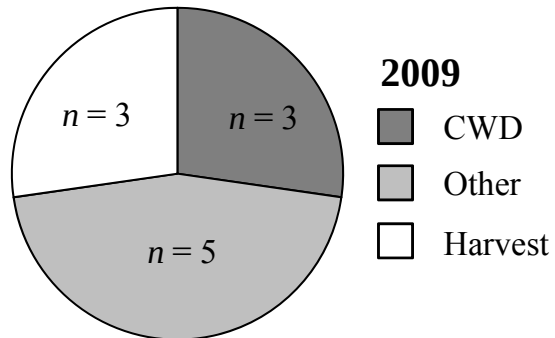
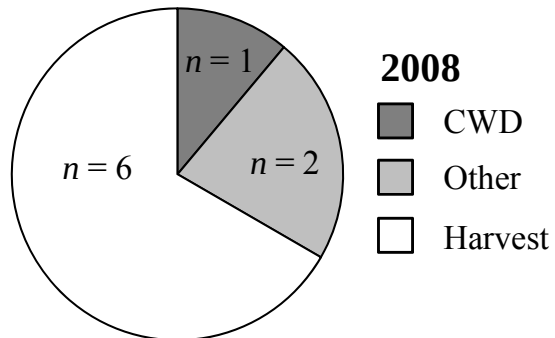
Figure 2. Cumulative survival rate of adult female elk in Rocky Mountain National Park following removal of all elk known to have chronic wasting disease in 2008 ($n = 13$, disease status determined with rectal biopsies). Harvest mortality and management/research removals are censored.

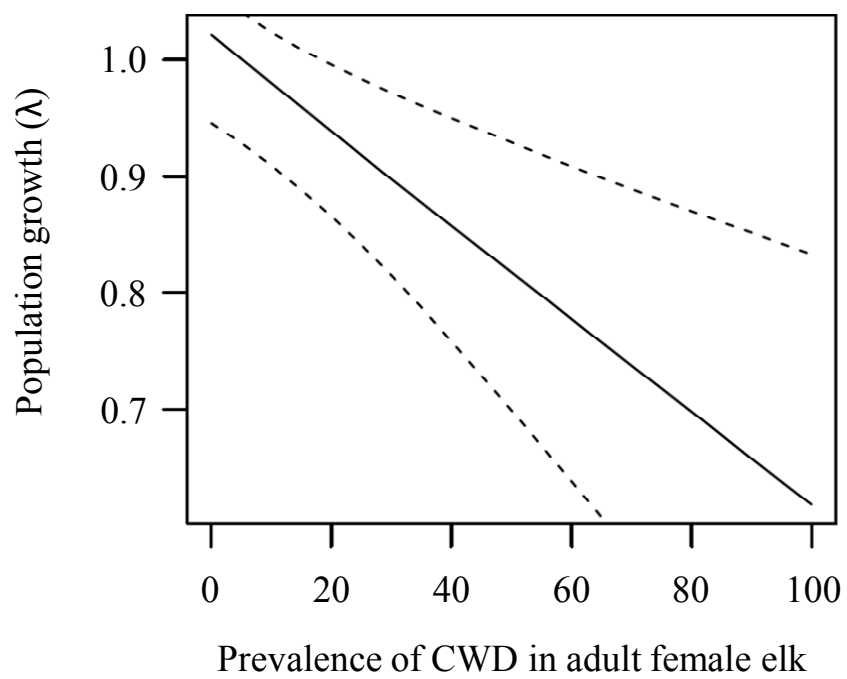
Figure 3. Mortality sources of adult female elk in Rocky Mountain National Park. Note: 13 elk found to have chronic wasting disease during initial capture efforts were removed at study initiation (January – March 2008) and hunters were requested to not harvest collared elk in 2009 and 2010.

Figure 4. Effect of prevalence (% infected) of chronic wasting disease (CWD) in adult female elk on the intrinsic rate of population growth (λ) of the elk population in Rocky Mountain National Park (dashed lines represent 95% credible limits [CL]). The transition matrix estimated λ to be 0.97 (CL = 0.90, 1.06) using the 2008 estimate for prevalence of CWD (12.9%).









Appendix 1

We estimated the effect of CWD on the intrinsic rate of elk population growth (λ) as follows. We assumed that the growth of the population could be reasonably represented using a discrete time model portraying the female segment of the population in two age classes. Age classes included calves, age 6 months at census, and adults, age ≥ 1.5 years at census. We recognize that yearling females produce calves at lower rates than elk 2.5 years and older. However, we used data on ratios of calves per yearling and older females to estimate recruitment to age 0.5 years, and did not attempt to include a yearling age class because it could not be discerned in the ratio data from Rocky Mountain National Park (RMNP). Our model was

$$\begin{pmatrix} N_1 \\ N_2 \end{pmatrix}_{t+1} = M \begin{pmatrix} N_1 \\ N_2 \end{pmatrix}_t,$$

where N_1 is the number of female calves, N_2 is the number of adult females; M is the projection matrix and $M = \begin{pmatrix} 0 & f \\ s_j & s_j \end{pmatrix}$, f is the number of female calves per adult female that survive to census, and s_j is the survival probability for adult females in two disease states, infected with PrP^{CWD} ($j = 1$) or susceptible ($j = 2$).

The posterior distribution of f was estimated as

$$\Pr(f|y, n) \propto \prod_{i=1}^5 \text{binomial}(y_i|f, n_i) \text{beta}(1,1),$$

where y is a vector of the number of female calves observed during midwinter classification counts from 2005-2010 assuming a 50/50 sex ratio of recruited calves and n is a vector of the number of adult females observed. All ratio data was obtained from RMNP (T. Johnson, unpublished data). We estimated the posterior distributions of monthly survival probabilities (s_j) of infected and susceptible females using

$$\Pr(s_j|z, m) \propto \prod_{i=1}^{18} \text{binomial}(z_{t,j}|s_j, m_{t-1,j})\text{beta}(1,1),$$

where $z_{t,j}$ is the number of elk in disease state j that were alive during month t , s_j is the survival probability of elk in susceptible or infected disease states, and $m_{t-1,j}$ is the number of elk in disease state j that were alive at time $t - 1$. Estimates of monthly survival probabilities were rescaled to annual probabilities as s_j^{12} .

The posterior distribution of the population growth rate was estimated as the dominant eigenvalue of the matrix M , where survival probabilities were weighted by assumed values for the proportion of females infected with PrP^{CWD} varying from 0 - 1.