Survival and Population Growth of a Free-Ranging Elk Population With a Long History of Exposure to Chronic Wasting Disease

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ABSTRACT Investigations of chronic wasting disease (CWD), a fatal, contagious prion disease of free-ranging cervids, suggest the disease can cause long-term population declines in deer (Odocoileus spp.). However, the implications of CWD for elk (Cervus elaphus nelsoni) populations are less certain. During 2008–2010, we used rectal biopsies and telemetry to observe disease transmission and survival in adult female elk from a high-density herd in Rocky Mountain National Park (RMNP) that had been infected by CWD for over 25 years. We studied a cohort of 123 adult female elk that were determined to be free of CWD by rectal biopsy in 2008. Annual incidence of CWD was 0.08 [95% Bayesian credible interval (BCI) = 0.05, 0.12]. Annual survival probabilities of the cohort excluding harvest declined from 0.97 in 2008 (BCI = 0.93, 0.99) to 0.85 in 2010 (BCI = 0.75, 0.93). Declines in survival were attributed almost entirely to CWD; the proportion of radiocollared elk that died of CWD increased from 0.02 in 2008 (BCI = 0.00, 0.05) to 0.11 in 2010 (BCI = 0.04, 0.21). We attributed the increase to the time lag required for development of new CWD cases. We used survival rates of susceptible and infected elk to develop a projection matrix for a discrete time, female only model that estimated the intrinsic population growth rate ($\lambda$) of this elk herd to be 1.00 (BCI = 0.93, 1.05) using the prevalence of CWD (12.9%) and calf:cow ratios (24:100) observed during this study. Population declines were predicted to occur when prevalence of CWD exceeded 13% (BCI = 0, 35). However, this estimate was contingent on calf:cow ratios and harvest. Greater recruitment will offset some of the effects of CWD, whereas the inclusion of female harvest, which was excluded from this study, would likely result in lower $\lambda$ values than those observed in this study. We conclude that CWD can exceed natural rates of mortality, reduce survival of adult females, and decrease population growth of elk herds. Published 2014. This article is a U.S. Government work and is in the public domain in the USA.

KEY WORDS Cervus elaphus nelsoni, chronic wasting disease, Colorado, elk, incidence, mortality, prion, survival.

Decreases in adult survival have a disproportionate, negative impact on the population growth of ungulates (Nelson and Peek 1982, Eberhardt 2002). In many cases, adult survival tends to be relatively stable and has little influence on annual population trends because short-term variability and stochastic weather events primarily affect patterns of mortality in juveniles (e.g., Gaillard et al. 1998, Smith and Anderson 1998, Lubow et al. 2002). Elk (Cervus elaphus nelsoni) tend to exhibit this pattern; for example, spring population estimates are primarily correlated with variability in calf survival rather than female survival because calves are more susceptible to weather, predation, or disturbance (Singer et al. 1997). Adult survival is typically subject to variation on longer time scales (compared to juveniles), and in most populations is largely determined by harvest, which is relatively consistent or can be adjusted in response to population surveys (Raithel et al. 2007).

Declines in adult survival can occur when a virulent pathogen is introduced to a susceptible ungulate population (e.g., George et al. 2008). The duration and extent of disease-related impacts on wildlife populations are normally constrained by development of host immunity and resistance (Bonneaud et al. 2011) or by decelerating rates of pathogen 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 developed resistance to an introduced disease may be at particular risk because they are unable to overcome such pathogens or limit their spread. This can lead to disease-
induced declines in host populations and, in some cases, to local extinction (e.g., McCallum 2008).

Chronic wasting disease (CWD) is a contagious transmissible spongiform encephalopathy that infects members of the deer family (Cervidae) in North America. This emerging prion disease, which was first described in samples from 1967 (Williams and Young 1980, Williams and Miller 2002), illustrates the significant impacts that an introduced pathogen can exert on a population. No evidence suggests that infected individuals develop immunity or survive infection (Williams et al. 2001), and field studies have found that the annual survival and reproduction of infected mule deer (Odocoileus hemionus) is 30–40% lower than uninfected deer (Miller et al. 2008, Dulberger et al. 2010). Transmission of prions between ungulates has been demonstrated to occur through direct contact and presumably soils (Miller and Williams 2003; Miller et al. 2004, 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 accumulated over the past decade on the effectiveness of management strategies and importance of host and spatial metrics, such as the influence of land use, on prevalence of CWD (% infected) in free-ranging deer (Conner et al. 2008, Wasserberg et al. 2009, Lawson and Song 2010, Habib et al. 2011, Potapov et al. 2012a).

In contrast, little is known about the ecology of CWD in free-ranging elk. Limited data indicate some differences in the ecology of CWD between deer and elk, which have minimized the concern over the potential population-level effects of CWD on elk (Sargeant et al. 2011). Disease progression appears more variable and often longer in captive elk (12–40 months) than mule deer (20–25 months; Williams et al. 2002, Hamir et al. 2006). Prevalence is generally lower in free-ranging elk (1–3%) than mule deer (1–30%; Miller et al. 2000, 2008; Colorado Division of Wildlife 2009). However, similarities in the effects of the disease on elk populations may be greater than previously recognized. Recent work found prevalence of CWD to exceed 10% in elk from Rocky Mountain National Park (RMNP), Colorado (Monello et al. 2013), and the combined effects of CWD, predation, and low recruitment were found to prevent elk population growth at Wind Cave National Park in South Dakota (Sargeant et al. 2011).

Several facets of CWD may allow it to maintain a relatively constant rate of transmission, regardless of host population density, and lead to long-term population declines. First, CWD prions (\(\text{PrP}^{\text{CWD}}\)) can remain infectious for at least 2 years 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), incidence (proportion of susceptible animals infected per year) will either exhibit a delayed response or have no relationship with host population density (Schauber et al. 2007, Almberg et al. 2011). Second, research on captive animals indicates that \(\text{PrP}^{\text{CWD}}\) accumulation progressively increases in the nervous system and lymphoid tissues of infected deer and elk (Spraker et al. 2002, 2010; Williams and Miller 2002; Miller and Williams 2003). 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 once CWD is well established and exceeds 5% prevalence, it could affect short-term population growth rates and, under appropriate conditions, the long-term sustainability of deer populations (Gross and Miller 2001, Wasserberg et al. 2009, Almberg et al. 2011). Empirical efforts to date generally support these findings and have found prevalence of CWD did not decline in the presence of intensive culling efforts (Conner et al. 2007) or in the presence of population declines that exceeded 50% (Miller et al. 2008). However, our current understanding is still limited by the lack of long-term studies of CWD in free-ranging populations, and recent work suggests that population reductions may be able to limit the increase of CWD in deer when prevalence is low (e.g., approx. 1%; Mateus-Pinilla et al. 2013).

These findings raise concern for the effects of CWD on elk population viability. To clarify the potential impacts of this disease, we measured rates of transmission and effects of CWD on survival in free-ranging adult female elk from RMNP. We also collected opportunistic mortality data from this population over the course of 10 years to supplement this work. This population is ideally suited to study the full range of CWD-related impacts on elk because this herd had been exposed to \(\text{PrP}^{\text{CWD}}\) for over 25 years at the time of our study (Spraker et al. 1997). Our specific objectives were to 1) determine incidence of CWD in adult females, 2) estimate effects of CWD on elk survival, 3) determine sources of mortality and measure the annual proportion of elk that died of CWD, and 4) estimate the relationship between prevalence of CWD and the rate of population growth in this elk herd.

**STUDY AREA**

We conducted this study on the elk winter range in RMNP (105°58′W, 40°36′N), which encompassed 10,000 ha and ranged from 2,400 to 2,800 m in elevation. Estimates of elk population density from this area ranged from 15–110 elk/km² during 1995–2000 (Singer et al. 2002), when the population was at its largest size (Fig. 1). Multiple factors may have contributed to fewer elk on the park winter range over the last decade, including increased hunter harvest adjacent to the park and dispersal to adjacent winter range areas (National Park Service 2007). The potential effects of CWD have not been evaluated. Additional details on the winter range and elk dynamics have been previously described (Lubow et al. 2002, Singer et al. 2002). Chronic wasting disease was first found in this elk herd in 1981 (Spraker et al. 1997). Prevalence of CWD in our study
population of elk was estimated to be 12.9% in 2008 (Monello et al. 2013). Prevalence in harvested elk from areas within 10–15 km of the park winter range were estimated to be 2.4% (9 of 382) from 2006 to 2008 (Colorado Division of Wildlife 2009). Prevalence of CWD in mule deer from areas immediately adjacent to the elk winter range in RMNP (in and around the Town of Estes Park) was 8.3% (15 of 181) in 2002–2003 (Wolfe et al. 2004).

METHODS

Study Design
We used 2 sources for assessing CWD and mortality in elk. First, we captured a sample of adult female elk, tested the elk for CWD, and released them with a radio collar during the winter of 2007–2008 (n = 136; hereafter referred to as initial captures). Thirteen of the initial captures were positive for PrP^CWD (CWD-positive) via immunohistochemistry (IHC) of rectal biopsies and were recaptured, euthanized, and necropsied within 55 days of their initial capture. These removals provided a design to investigate a cohort of elk (n = 123) that was free of detectable CWD at the start of the study. We then resampled, euthanized, and necropsied 20, 25, and 34 of the remaining cohort of elk in each of the 3 following winters, respectively (hereafter referred to as resampled elk, Colorado State University Institutional Animal Care and Use Protocol no. 07-231A). We monitored the status of each study elk (alive or dead) at least once per week using telemetry. If we found an animal dead, we necropsied the whole carcass in the field or laboratory to determine cause of death. We collected postmortem samples for CWD diagnostics from the brainstem (medulla oblongata at the level of the obex; hereafter obex) and retropharyngeal lymph nodes.

Second, prior work in Wind Cave National Park, South Dakota reported on the frequency of carcasses with CWD from 2005 to 2009 (Sargeant et al. 2011). For comparative purposes and to determine if CWD is common among dead elk, we tested for CWD in carcasses of non-collared elk found dead and non-collared elk that were euthanized because they were exhibiting end-stage signs of CWD (emaciation, depression, excessive salivation; Miller et al. 1998) on the winter range in RMNP from 2000 to 2009 using the same methods as above.

Mortality
We visually evaluated elk for clinical signs of CWD before we darted them from vehicles or on foot. After immobilizing an elk, we attached a radio collar and collected rectal biopsies for CWD diagnostic tests. We captured and biopsied resampled elk in the same manner, and euthanized and necropsied elk within 8 hr of death at the Colorado State University Veterinary Diagnostic Laboratory (Fort Collins, CO). Additional details on capture and sampling were described by Monello et al. (2013).

We fitted each elk with a very high frequency (VHF) radio collar (Advanced Telemetry Systems, Isanti, MN) that activated a mortality signal after 8 hr of non-movement. We minimized harvest mortalities in the last 2 years of the study (fall and winter of 2009–2010 and 2010–2011) by sending letters to licensed hunters that requested they avoid harvesting radiocollared elk that ranged outside RMNP. When possible, we collected the entire carcass of elk that died during the study and necropsied them at the Colorado State University Veterinary Diagnostic Laboratory. When we

Figure 1. The total population size of elk in Rocky Mountain National Park (RMNP) during winter (±2 standard errors, only available after 1993) using previously described methods (Lubow et al. 2002).
could not collect the entire carcass, we removed the obex and retropharyngeal lymph nodes and stored them in 10% neutral buffered formalin.

We investigated causes of mortality of collared elk in the field and during necropsy. We identified harvested elk when collars and postmortem samples (e.g., obex) were returned to the National Park Service or Colorado Division of Wildlife by a hunter. We identified mountain lion (Puma concolor) kills by cached carcasses, puncture wounds, and evidence of hemorrhaging on the throat or back of the neck (Shaw et al. 2007). We considered CWD to be the cause of death when we did not find signs of trauma or predation, the proximate cause of death was associated with CWD (e.g., pneumonia or emaciation), and the elk was infected with PrP<sub>CWD</sub> and had an obex score $\geq$8 (described below). We classified dead study elk that were too decomposed or scavenged to determine cause of death as unknown, even if PrP<sub>CWD</sub> was detected. We removed a primary incisor from all elk carcasses to determine age by cementum analysis (Matson’s Lab, Milltown, MT).

For carcasses of uncollared elk, we included only elk that had usable diagnostic samples for CWD (i.e., obex or retropharyngeal lymph nodes). We classified sources of mortality in a similar manner as for collared elk, with two exceptions. First, we determined harvest by the presence of gunshot wounds. Second, although we report the proportion of carcasses with prion infection, we did not include CWD as a potential cause of death because carcasses frequently were scavenged to rule out other sources of mortality and we did not score the obex.

To diagnose CWD in elk, we removed the obex and medial retropharyngeal lymph nodes and stored them in 10% neutral buffered formalin. We assayed antemortem rectal biopsies and postmortem samples for PrP<sub>CWD</sub> by IHC as previously described (Spraker et al. 2006). We considered positive IHC staining of any obex or retropharyngeal lymph node to be a definitive diagnosis of CWD (Spraker et al. 2009). We scored obex samples on a scale from 0 (no PrP<sub>CWD</sub> detected in brain stem) to 10 (terminal stage of disease) as previously described (Spraker et al. 2010).

**Analyses**

We conducted all analyses in a Bayesian framework. To calculate incidence, we assumed all radiocollared elk were susceptible (uninfected) at the start of the study except for those that were CWD-positive via rectal biopsy or were infected by PrP<sub>CWD</sub> but misdiagnosed as susceptible during initial capture (Monello et al. 2013). The posterior distribution of incidence ($\phi$) was estimated as

$$
\Pr(\phi | y, n) \propto \prod_{t=1}^{\text{12}} \text{binomial}(y_t/(1 - \phi)^t, n_t)\beta(\phi | 1, 1)
$$

where $n_t$ is the number of elk tested for CWD in year $t$, $y_t$ is the number of elk that tested negative for CWD in year $t$, and $\phi$ is the annual probability that an elk becomes infected by CWD. 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. We considered only elk that contained PrP<sub>CWD</sub> in the obex or retropharyngeal lymph node to be infected. We raised the probability of remaining healthy to the power of $t$ in equation 1 because we resampled 3 groups for CWD, 1 during each year of the study. Because the probability of remaining healthy for 1 year was $1 - \phi$, the probability of remaining healthy for 2 years was $(1 - \phi)^2$ and for 3 years was $(1 - \phi)^3$.

We calculated annual survival and mortality from CWD from January to December for each year of the study. We did not include any elk that died within 2 weeks of initial capture ($n = 1$). We included harvested elk but censored them in the month they died because our request to licensed hunters to not harvest elk with radio collars prevented an accurate estimate of harvest mortality. We also censored resampled elk that were euthanized for research purposes. However, we included elk that were euthanized because they were exhibiting end stage signs of CWD (i.e., did not censor from survival analyses). Within each year of the study, we estimated the probability of monthly survival conditional on the data as

$$
\Pr(z | x, m) \propto \prod_{t=1}^{\text{12}} \text{binomial}(z_t|x_t, m_t)\beta(z | 1, 1)
$$

where $z_t$ is the number of elk alive at the end of month $t$, $x_t$ is the survival probability of elk, and $m_t$ is the number of elk that were alive at the beginning of month $t$. We rescaled estimates of monthly survival probabilities to annual probabilities as $p_t = s_t^{12}$.

We estimated the relationship between prevalence of CWD and the intrinsic rate of population growth ($\lambda$) in this elk herd using the dominant eigenvalue from a projection matrix for a discrete time, female only model with 2 age classes, calves (8 months at census) and reproductive females ($\geq$20 months at census). Prior work found no age-related differences among reproductive female elk in RMNP (Lubow et al. 2002), and we assumed adult survival did not differ due to age once elk were $\geq$20 months old. We used ground classifications of herd composition conducted from 2007 to 2009 (T. Johnson, National Park Service, unpublished data) to estimate calf:cow ratios (i.e., recruitment) and divided observations by 2 because we assumed a 1:1 sex ratio for calves. This estimated the recruitment of calves per reproductive female, which allowed us to not have to estimate separate reproductive parameters for yearling and older females. We estimated survival of infected and uninfected elk using the last 18 months of data from this study (Jun 2009 to Dec 2010) as described in Appendix A. We included only elk that were resampled and euthanized or died and had viable postmortem samples for CWD testing during the last 18 months. We considered any elk found to have PrP<sub>CWD</sub> in the obex or a retropharyngeal lymph node to be infected by PrP<sub>CWD</sub> for the entire 18-month period. We chose to use only 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, because we removed all known individuals infected with
PrPCWD at the outset of the study (i.e., elk infected with PrPCWD in the first 18 months of the study would all be in an early disease state). We conducted analyses with JAGS (version 3.1.0, www.mcmc-jags.sourceforge.net, accessed Oct 2012) using the rjags package (Plummer 2011) in the R computing environment (version 2.15.2, R Development Core Team 2011). Additional details on analyses are available in Appendix A.

RESULTS

Survival, Mortality, and Population Growth

None of the radiocollared elk displayed classical signs of CWD prior to initial capture. Mean survival probabilities of elk in our study cohort of radiocollared individuals, all of which were initially free of detectable CWD, declined from 0.97 in 2008 [95% Bayesian credible interval (BCI) BCI = 0.93, 0.99] to 0.90 in 2009 (BCI = 0.84, 0.95) to 0.85 in 2010 (BCI = 0.75, 0.93; Fig. 2). Although uncertainty increased annually because of the removal of elk for research and management purposes, the BCIs for survival in 2008 and 2010 did not overlap. The preponderance of deaths occurred from October to April, and we did not observe mortalities in July or August (Fig. 2).

The decline in annual survival of radiocollared elk coincided with an estimated CWD annual incidence of 0.08 (BCI = 0.05, 0.12) from 2008 to 2010 and an increase in mortalities due to CWD. Among all radiocollared elk, the proportion that died because of CWD was 0.02 (BCI = 0.00, 0.05; 1 of 121) in 2008, 0.06 (BCI = 0.02, 0.11; 3 of 91) in 2009, and 0.11 (BCI = 0.04, 0.21; 5 of 56) in 2010. Conversely, the number and proportion of harvested elk declined after we requested hunters to avoid those with collars in 2009 and 2010 [6 (0.05%) were harvested in 2008, 0.11 (BCI = 0.05, 0.21) in 2009, and 0.02 (BCI = 0.00, 0.05) in 2010].

Uncollared Elk Carcasses

We opportunistically collected 78 non-collared elk carcasses on the winter range, 53 (68%) of 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 PrPCWD, and all but 1 CWD-positive elk carcass was in the unknown category. Overall, 4 of 25 (16%) male carcasses and 24 of 53 (45%) female carcasses were CWD-positive. Among 20 CWD-suspects that were euthanized, 16 were CWD-positive (15 females, 1 male; Table 2).

CWD and Age

We did not detect any upper age limit to CWD infection; 4 of 9 collared elk that died of CWD and 4 of 26 uncollared elk carcasses that were infected were >10 years old (Tables 1 and 2). Radiocollared adult elk that were CWD-positive at the start of the study via rectal biopsy (median = 3, mean age = 5.8, BCI = 4.6, 7.2) were almost half the age of radiocollared adult elk that died of CWD during the study (median = 9, mean age = 10.2, BCI = 8.3, 12.4).

DISCUSSION

Our findings indicate cow elk survival—a metric that exhibits low variance but has the greatest potential influence on elk population dynamics (Nelson and Peek 1982, Raithel et al. 2007)—can be reduced by CWD. Following removal of all radiocollared elk that were known to be CWD-positive at the start of the study, we observed consistent annual increases in CWD-caused mortality and corresponding declines in survival of collared elk during each year of the telemetry study. We attributed the increase to the time lag required for development of new CWD cases. The survival estimate of 0.85 (BCI = 0.75, 0.93) observed in the last year of our study is of particular concern because research on elk populations throughout North America indicated that the majority (>80%) of herds decline when cow survival is ≤0.85 (Raithel et al. 2007). Lower survival rates resulting from CWD appear to be a recent phenomena in this population, because prior estimates of annual cow survival in RMNP that included harvest averaged 0.93 ± 0.023 (SE) from 1995 to 1998 (Lubow et al. 2002). We recognize that we have only 1 year of data where survival is <0.90, and thus our inferences are limited with regard to future projections of CWD-related impacts on this elk herd. However, we suggest our annual estimates of survival are likely too high versus too low because we selectively removed radiocollared elk that tested positive for CWD at the start of the study and censored

![Figure 2](image-url). Cumulative survival rate of adult female elk in Rocky Mountain National Park (n = 121 in 2008, n = 91 in 2009, and n = 56 in 2010) following removal of all elk known to have chronic wasting disease at the start of the study (n = 13, disease status determined with rectal biopsies). Harvest mortality and removals for management or research purposes are censored.
hunter harvest mortalities. If we did overestimate survival, effects of CWD on this elk population are even greater than our results indicate here.

The posterior distribution of $\lambda$ shows that at current prevalence (12.9%) we cannot rule out growth rates that would lead to declines in our study population. We found $\lambda$ of elk in our study was less than it would have been if CWD were absent or occurred at lower prevalence. This indicates CWD may have contributed to the smaller elk population sizes recently observed in RMNP and warrants further research. Other empirical studies support this conclusion because CWD has been found to reduce adult survival and contribute to population declines that exceed 50% in mule deer (Miller et al. 2008) and to decrease population growth rates in elk (Sargeant et al. 2011). Results of the projection matrix predicts that, even in the absence of hunting or other sources of mortality, CWD alone could induce population declines once prevalence exceeds 13% ($\text{BCI} = 0$, 35). However, our predicted relationship between CWD and $\lambda$ is largely contingent on the survival rate of infected elk, which had a wide BCI in this study (0.33–0.76), and calf:cow ratios, which have been relatively low for the past 15 years (20–30 calves per 100 cows; Lubow et al. 2002). If the annual survival of infected elk is higher than we estimated or recruitment increases, as would be expected in a herd that is not limited by their forage base (Lubow et al. 2002), recruitment increases, as would be expected in a herd that is not limited by their forage base (Lubow et al. 2002) and calf:cow ratios may be able to sustain slightly higher rates of CWD without exhibiting population declines, due in part to larger numbers of uninfected animals entering the population annually (Potapov et al. 2012b).

Primary routes of prion transmission and factors that influence incidence of CWD in free-ranging ungulates remain unresolved (Conner et al. 2008). However, our estimate of incidence (proportion infected per year; $\text{BCI} = 0.05$, 0.12) is greater than the total proportion of infected individuals found in most elk herds with CWD (Miller et al. 2000) and clearly demonstrates that prion transmission has the potential to increase over time in elk. We suggest that a long history of exposure to prions and high concentrations of elk from the 1970s to 1990s likely contributed to the rates of disease transmission and prevalence observed in this study population. We recognize that it is unknown if CWD is transmitted in a frequency- or density-dependent manner (Conner et al. 2008), but related work has suggested that greater concentrations of deer are correlated with higher CWD prevalence (Farnsworth et al. 2005) and that prions are transmitted through the environment (Miller et al. 2004, Georgsson et al. 2006). Demonstrating the existence or lack of a relationship between incidence and population density would help determine if culling programs are warranted. However, to be done effectively using our methods would require a long-term mark-recapture study that incorporates potential time lags between these metrics and includes a range of densities and herds.

In addition to incidence, the trajectory and impacts of CWD on population size will depend on how rapidly the disease progresses in elk. Although we found elk that tested positive for CWD via rectal biopsy were typically younger than elk that died of CWD, the overall range of ages was similar and duration of disease course cannot be inferred from our results. One reason for this is because the majority (10 of 13) of infected elk detected and removed at the start of the study had the genotype MM$_{132}$, whereas 5 of 9 that died of CWD during the study had the genotype ML$_{132}$ (Monello et al. 2013). Previous work found the genotype MM$_{132}$ to have the most rapid disease course (Hamir et al. 2006, O’Rourke et al. 2007). Further, the removal of 13 infected elk at the start of the study is likely to have biased the average age at death among elk that died of CWD. Relationships between genotype, disease progression, and annual survival should be further refined with research that monitors free-ranging elk known to be infected with PrP$^{\text{CWD}}$ via rectal biopsy.

The mortality rate attributed to CWD in the last year of the study (0.11 of the study cohort) and proportion of opportunistically collected carcasses of uncollared elk 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 ($\text{Canis lupus}$), mountain lions, and bears ($\text{Ursus}$ spp.) has been found to cause a similar rate of mortality for adult elk (annual mortality of elk due to predation $= 0.06–0.09$, Evans et al. 2006, Brodie et al. 2013; 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

### Table 1. Fate of 136 radiocollared adult female elk in Rocky Mountain National Park and median age at time of death. Five elk did not have usable postmortem samples for chronic wasting disease (CWD) diagnosis (1 in harvested category, 4 in unknown category) and 15 elk were still alive at the end of the study.

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>Total</th>
<th>Median age (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CWD-positive at start, euthanized</td>
<td>13</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>3 (2–17)</td>
</tr>
<tr>
<td>Resampled, euthanized</td>
<td>20</td>
<td>25</td>
<td>34</td>
<td>79</td>
<td>9 (3–19)</td>
</tr>
<tr>
<td>Harvested</td>
<td>6</td>
<td>3</td>
<td>1</td>
<td>10</td>
<td>9 (4–14)</td>
</tr>
<tr>
<td>Died of CWD*</td>
<td>1</td>
<td>3</td>
<td>5</td>
<td>9</td>
<td>9 (6–16)</td>
</tr>
<tr>
<td>Unknown cause of mortality*</td>
<td>0</td>
<td>5</td>
<td>2</td>
<td>7</td>
<td>11 (6–19)</td>
</tr>
<tr>
<td>Vehicle collision, predator</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>18 (8–21)</td>
</tr>
</tbody>
</table>

*Includes 1 of 2 elk that were euthanized because it was exhibiting end-stage signs of CWD (only 1 tested positive for CWD, the other was emaciated and unable to move).
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 in juvenile and adult elk also vary because of annual weather patterns and predator-prey population dynamics (Carbyn 1983, Boyd et al. 1994, Mech et al. 2001). In contrast, the consistent effect of CWD across age classes 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 PrP$^\text{CWD}$ transmission is independent of population density (e.g., Almberg et al. 2011, Storm et al. 2013).

Observations indicate that harvest, the absence of localized hunting refugia and associated high densities of deer, or population reduction programs may help maintain lower prevalence values (Wolfe et al. 2002, Farnsworth et al. 2005, Wasserberg et al. 2009, Mateus-Pinilla et al. 2013), but there is still uncertainty about how to reduce prevalence, and large-scale reductions of deer have so far failed to reduce CWD (Conner et al. 2007, Mateus-Pinilla et al. 2013). Several potential differences between CWD ecology in elk and deer may allow a test-and-remove 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 testing 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 exposure within female groups. 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). Assuming a longer disease course results in more time to shed prions, removal of infected elk may be more beneficial than removal of infected deer because each elk represents a greater per capita source of transmission. Similarly, selective removal of CWD-positive elk by wolves could also 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.

We found CWD-caused mortality can exceed natural sources and, at the rates observed in our study, could result in either an unsustainable population or necessitate declines in harvest. Reductions in harvest will be only a temporary solution to prevent population declines if CWD continues to increase and other forms of mortality are additive, because our modeling results indicate that under these circumstances CWD alone is capable of causing large declines in λ. Natural selection may favor genotypes that have a longer disease course or are resistant to disease (Robinson et al. 2012). A prolonged disease course in elk could lessen the impact of CWD on population size, but may also increase infectious contacts, environmental contamination, incidence, and long-term prevalence levels. In addition, it is currently unknown why cervid genotypes that slow the progression or increase resistance of prion infection are underrepresented in natural

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**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 telemetry study and only elk that had a usable postmortem sample for diagnosis of chronic wasting disease (CWD) are included.

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Males</th>
<th>Females</th>
<th>Median age (range; n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carcasses (collected)</td>
<td>9</td>
<td>17</td>
<td>8 (1–18; n = 16)</td>
</tr>
<tr>
<td>Unknown, CWD-negative</td>
<td>3</td>
<td>24</td>
<td>5 (2–12; n = 16)</td>
</tr>
<tr>
<td>Vehicle collision (all CWD-negative)</td>
<td>4</td>
<td>8</td>
<td>2 (1–15; n = 6)</td>
</tr>
<tr>
<td>Predation (1 male was CWD-positive)</td>
<td>3</td>
<td>4</td>
<td>3 (1–8; n = 4)</td>
</tr>
<tr>
<td>Harvested or poached (all CWD-negative)</td>
<td>6</td>
<td>0</td>
<td>NA (1–5; n = 2)</td>
</tr>
<tr>
<td>CWD-suspects (euthanized)</td>
<td>1</td>
<td>15</td>
<td>6 (1–12; n = 10)</td>
</tr>
<tr>
<td>Euthanized CWD-suspect, CWD-positive</td>
<td>1</td>
<td>3</td>
<td>NA (6–15; n = 2)</td>
</tr>
</tbody>
</table>

---

Figure 3. Relationship between prevalence (% infected) of chronic wasting disease (CWD) and intrinsic rate of population growth (λ) of the elk population in Rocky Mountain National Park in 2008–2010 [dashed lines represent 95% Bayesian credible interval (BCI)]. In the absence of hunting, the projection matrix estimated λ to be 1.00 (BCI = 0.93, 1.05) using observed calf:cow ratios from 2007 to 2009 (approx. 24:100) and the 2008 estimate for prevalence of CWD (12.9%).
MANAGEMENT IMPLICATIONS

Our results indicate that the long-term exposure to PrP\textsuperscript{CWD} could negatively affect the survival and population growth of some elk populations. A critical need exists to determine 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, prevalence in hunted elk populations from the CWD endemic area in Wyoming has averaged 4.5–8.9% over the last decade (T. Kreeger, Wyoming Game and Fish Department, personal communication). Although data on the effects of CWD on 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). Therefore, preventative efforts to minimize the risk of CWD into new geographic areas remains the most effective approach to minimizing long-term population limiting effects of CWD.

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We estimated the effect of chronic wasting disease (CWD) on population growth rate 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 2 age classes. Age classes included calves, age 8 months at census, and adults, age ≥20 months at census. We assumed census in the model occurred in late January and the birth pulse occurred 4 months later. We recognize that yearling females produce calves at lower rates than older animals. However, we used data on ratios of calves per yearling and older females to estimate recruitment, and did not attempt to include a yearling age class because it could not be discerned in the ratio data.

Our model was

\[
\begin{pmatrix}
N_1 \\
N_2
\end{pmatrix}
_{t+1} = \mathbf{M}
\begin{pmatrix}
N_1 \\
N_2
\end{pmatrix}
_t
\]

where \(N_1\) is the number of female calves, \(N_2\) is the number of adult females, and \(\mathbf{M}\) is the projection matrix,

\[
\mathbf{M} = \begin{pmatrix}
0 & p_{2,1}^{1/3} \cdot f \\
p_1 & p_2,1
\end{pmatrix}
\]

Parameters in \(\mathbf{M}\) are \(f\), the number of female calves that survive to their first census produced per adult female alive at time \(t\), \(p_1\), the probability that animal survives from age 8 months to 20 months; and \(p_{2,1}\), the annual survival probability of survival of adult females in 2 states, infected with CWD \((i = 1)\) or uninfected \((i = 2)\). Using a single value for juvenile survival requires the assumption that it is not influenced by CWD. We included the term \(p_{2,1}^{1/3}\) in the estimate of number of calves produced per female because females must survive from census to the birth pulse to produce offspring (Noon and Sauer 1992). We estimated the population growth rate for an entirely healthy and an entirely infected population as the dominant eigenvalue \(\lambda_i\) of the matrix \(\mathbf{M}\):

\[
\lambda_i = \frac{1}{2} p_{2,1} + \frac{1}{2} \sqrt{p_{2,1}^2 + 4 \sqrt{p_{2,1} / p_1}}
\]

(A1)

In the Bayesian framework, any quantity that is a function of random variables becomes a random variable. Thus, if we know the posterior distributions of the parameters in \(\mathbf{M}\) then we can obtain the posterior distribution of \(\lambda_i\) using equation A1. We estimated the posterior distribution of the population growth rate for hypothetical levels of prevalence by weighting adult survival probabilities by assumed values for the proportion of females infected with CWD varying from 0 to 1. We estimated the growth rate of the current population from the dominant eigenvalue of \(\mathbf{M}\) using adult survival probability weighted by observed prevalence \(\pi\). We included uncertainty in prevalence in our model by treating it as a beta distributed random variable. We computed the posterior distribution of prevalence estimates \(\pi\) using a beta-binomial conjugate prior relationship, \(\pi \sim \text{beta}(\mu + 1, \omega - \mu + 1)\), where \(\mu\) is the number of CWD positive individuals in our sample and \(\omega\) is the total number of animals sampled.

We estimated parameters in the projection matrix from data obtained in this study or from prior information. We estimated the posterior distribution of \(f\) as

\[
\Pr(f | y, n) \alpha \prod_{j=1}^{3} \text{binomial}(y_j | f, n_j) \text{beta}(1, 1)
\]

where \(y\) is a vector of the number of female calves observed during midwinter ground classification counts conducted during 2007–2009 (each observation = total calves observed/2 because we assumed a 1:1 sex ratio of calves) and \(n\) is a vector of the number of adult females observed. We estimated the posterior distributions of monthly survival probabilities \(s_i\) of infected and susceptible females using

\[
\Pr(s_i | z, m) \alpha \prod_{t=1}^{18} \text{binomial}(z_{t,i} | S_i, m_{t-1,i}) \beta(1, 1)
\]

where \(z_{t,i}\) is the number of animals in disease state \(i\) that were alive during month \(t\), \(s_i\) is the monthly survival probability of animals in susceptible \((i = 1)\) or infected \((i = 2)\) states, and \(m_{t-1,i}\) is the number of animals in state \(i\) that were alive at time \(t - 1\). We estimated annual survival of adults in each disease state from the monthly observations, \(p_{2,1,i} = s_i^{12}\). We estimated juvenile survival probabilities from an updated version of the model of Lubow et al. (2002) developed by N. T. Hobbs and J. A. Hoeting (Colorado State University, unpublished data):

\[
p_1 \sim \text{beta}(6.40, 4.31)
\]

Thus, we treated \(p_1\) as a beta distributed random variable with mean = 0.61 and standard deviation = 0.14.

We estimated posterior distributions of parameters in \(\mathbf{M}\) and quantities derived from \(\mathbf{M}\) using Markov chain Monte Carlo methods implemented in JAGS (version 3.1.0, www.mcmc-jags.sourceforge.net, accessed Oct 2012) using the rjags package (Plummer 2011) in the R computing environment (version 2.15.2, R Development Core Team 2011). We randomly chose initial values for 3 chains from vague priors. We assured convergence using Gelman–Rubin statistics (Brooks and Gelman 1997, Gelman and Rubin 1992), the upper confidence limits of which were uniformly 1. All chains passed the Heidelberger and Welch (1983) test for stationarity and half-width tests for accuracy of estimation of the mean of the posterior distribution.